REVIEW

Gait Ataxia—Specific Cerebellar Influences and Their Rehabilitation

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ABSTRACT: It is well known that the cerebellum is important for movement control and plays a critical role in balance and locomotion. As such, one of the most characteristic and sensitive signs of cerebellar damage is gait ataxia. However, characterizing ataxic gait is no easy task, because gait patterns are highly variable. This variability seems to result from the interaction of different factors, namely, (1) the primary motor deficits in balance control and multi-joint coordination and oculomotor dysfunction, (2) the safety strategies used, and (3) inaccurate adjustments in patients with loss of balance. In this report, we review different approaches to analyzing ataxic gait and studies to identify and quantify the different factors contributing to this movement disorder. We also discuss the influence of the cerebellum in adaptive locomotor control, the interaction between cognitive load and gait in dual-task paradigms, and the recent advances in rehabilitation of gait and posture for patients with cerebellar degeneration. In the second part, we discuss open questions concerning cerebellar mechanisms in multi-joint coordination during different walking conditions. Furthermore, we point out potential future directions in motor rehabilitation, with the objective of identifying predictors of rehabilitation outcome and the development of individualized training programs that potentially involve rehabilitation technology. © 2013 International Parkinson and Movement Disorder Society

Key Words: cerebellum; ataxia; gait; rehabilitation; locomotion

The functional role of the cerebellum in motor control is not the generation but more in the shaping and fine-tuning of movements. Therefore, cerebellar damage does not cause loss of movement but, instead, leads to abnormalities in movement characterized by increased variability and poor accuracy.[1] Typical ataxia symptoms (Greek *ataxia*: without order, incoordination) are dysmetria (hypermetria and hypometria), cerebellar tremor, and dyssynergia—the loss of simultaneous joint movements.[2,3]

Because the cerebellum is involved in the control of various kinds of motor behavior in speech, oculomotor control, limb movements, and balance, the described abnormalities—like dysmetria and dyssynergia—occur in various movement modalities. Consistently, ataxic gait appears to be influenced by deficits like dyssynergia (eg disordered coordination between head, trunk, and legs) and dysmetria (eg impaired predictive postural adjustmends[4]) in balance control and multi-joint leg coordination, acting on a whole-body control problem with complex dynamics.[5]

Symptomatically, ataxic gait is typically characterized by an increased step width, variable foot placement, irregular foot trajectories, and a resulting instable, stumbling walking path with very high movement variability[6–8] and a high risk of falling.[9] Causes for ataxic gait can be various, including focal cerebellar disease like stroke, cerebellar tumors, multiple sclerosis, and degenerative diseases, affecting the cerebellum or its afferent pathways (for a more detailed description, see Ilg and Timmann[10]). In this review, we focus on ataxic gait caused by cerebellar dysfunction and, in particular, by cerebellar degeneration.

Typical Signs of Ataxic Gait

Various studies have compared the gait from cerebellar patients and healthy controls. These studies reported partially conflicting results, which can be

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TABLE 1. Overview of studies examining cerebellar ataxic gait in different conditions

Reference	No. of Patients: Description	Condition	Main result
Palliyath et al.[11]	10: OPCA, CCA	Gait	Reduced step length, impairments in multi-joint coordination, increased variability of several gait measures
Morton and Bastian[13]	20: IDCA, SCA3, SCA6, PVS	Gait	Balance deficits are more closely related to cerebellar gait ataxia than leg-placement deficits
Stolze et al.[12]	12: CD with various causes	Gait	Reduced cadence, increased balance related variables; tandem gait paradigm accentuates gait ataxia
Ilg et al.[8]	13: IDCA, SCA6	Gait	Temporal variability in intra-limb coordination is specific for cerebellar dysfunction
Serreo et al.[17]	16: SCA1, SCA2, FA	Gait	Irregular alternating joint behavior, increased variability of global and segmental gait parameters
Mitoma et al.[14]	14: OPCA, LCCA	Gait	EMG reveals high muscle activity during periods when these muscles were not active in normal walking
Wuehr et al.[18]	11: IDCA, SCA1, SCA2, SCA6	Gait	Alterations in the speed dependency of stride time and stride length variability, whereas base width variability remained unaffected
Bastian et al.[74]	5: PVS	Gait	Transection of the posterior inferior cerebellar vermis affects tandem fundamentally more than regular gait
Crowdy et al.[34]	8: IDCA, ADCA, SCA1, SCA7	Visually guided walking	Similarities between oculomotor deficits during visual fixation task and during walking
Crowdy et al.[35]	2: ADCA	Visually guided walking	Foot placement accuracy could be improved by prior practice in making saccadic eye movements toward the targets
Timmann and Horak[75]	8: ADCA, IDCA, EA-type 2	Gait initiation	Modification of postural adjustments
Conte et al.[76]	10: IDCA, SCA1, SCA2	Gait termination	Compensatory strategies during gait termination, eg increasing their step width and number of steps
Mari et al.[77]	10: SCA1, SCA2, SAOA	Turning	Compensatory strategies: enlarge the base of support, shorten step length, increase number of steps, and use the "multi-step" rather than the "spin-turn" strategy
Earhart and Bastian[32]	8: PVS, SCA7, OPCA, CAI	Stepping on inclined surfaces	Abnormal relative movement of hip, knee, and ankle joints; decomposition of joint movement
Morton et al.[64]	8: IDCA, SCA6, SCA8	Obstacle avoidance	Dynamic abnormalities associated with leg hypermetria is context-specific, voluntary compensatory strategies
Earhart et al.[48]	8: IDCA, ADCA, SCA6	Gait adaptation	Cerebellum is important for regulation of the amplitude of podokinetic adaptation
Morton and Bastian[47]	9: IDCA, ADCA, SCA6, SCA8	Gait adaptation	Specific impairments at making predictive changes in temporal aspects of intra-limb coordination
Ilg et al.[46]	12: Focal lesions after cerebellar tumor resection	Gait adaptation	Specific abnormalities in temporal aspects of intra-limb coordination for leg placement and adaptive locomotion
Ilg et al.[59]	16: IDCA, FA ADCA, SCA2, SCA6, SANDO	Gait rehab	Intensive coordination training improves gait in terms of velocity, lateral sway, and variability of intra-limb coordination pattern
Miyai et al.[61]	42: IDCA, SCA31, SCA6	Gait rehab	Physiotherapy in combination with occupational therapy improves gait speed and fall frequency
Ilg et al.[78]	10: FA, ADCA, arCA, AOA2	Gait rehab	Video-gamed–based coordination training in children improves velocity, step-length variability, and dynamic balance

OPCA, olivopontocerebellar atrophy; CCA, cerebellar cortical atrophy; IDCA, idiopathic cerebellar ataxia; SCA3, SCA6, spinocerebellar ataxia types 3 and 6, respectively; PVS posterior vermal split; CD, cerebellar disease; FA, Fredreich ataxia; LCCA, late cerebellar cortical atrophy; EMG, electromyelography; SCA1, SCA2, spinocerebellar ataxia types 1 and 2, respectively; ADCA, autosomal dominant cerebellar ataxia; SCA7, spinocerebellar ataxia type 7; EA-type 2, episodic ataxia type 2; SAOA, sporadic adult-onset ataxia of unknown etiology; CAI, cerebellar artery infarct; SCA8, spinocerebellar ataxia type 8; rehab, rehabilitation; SANDO, sensory ataxic neuropathy with dysarthria and ophthalmoparesis caused by mutations in the polymerase gamma gene (POLG); arCA, autosomal-recessive ataxia; AOA2, ataxia with oculomotor ataxia type 2.

explained by the heterogeneity of examined patient populations in terms of underlying diseases and particularly in terms of the severity of ataxia symptoms.

Palliyath et al.[11] reported significant changes for the averages in velocity and stride length, whereas Stolze et al.[12] observed almost no differences for the same gait parameters. Similarly, several studies demonstrated significant differences for the step width,[12] whereas others did not.[11,13–15] Mitoma et al.,[14] who divided their patient population into 2 groups with moderate ataxia and severe ataxia, observed significant changes in step width, step length, speed, and the durations of the double and single support periods only for the group with severe ataxia. Differences between patients and controls also have been reported for other gait parameters (see Table 1), eg angle ranges, cadence, stance time, the time of maximum flexion during the swing phase,[11,12] as well as increased body sway in different directions.[8,16]

Analyzing these features reveals that many of the parameters that measure average values of velocity, step length, or step width may reflect predominantly

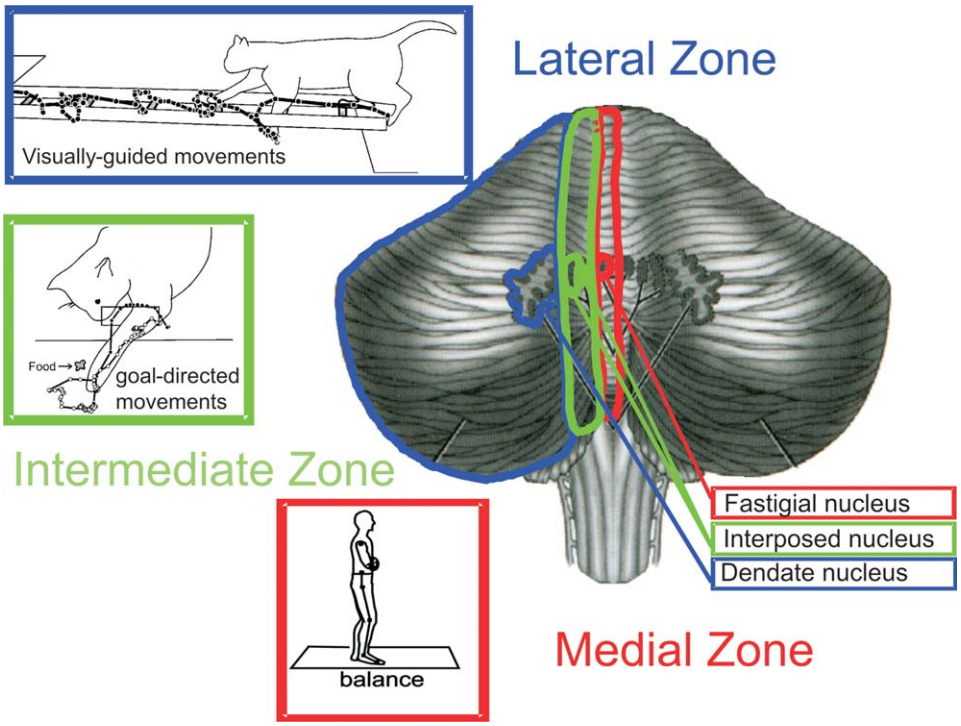

FIG. 1. Localization of cerebellar function in gait control is illustrated. The medial region plays a central role in locomotion by sustaining upright stance and dynamic balance control. It is suggested that the intermediate cerebellar regions are important for performing precise limb movements and that lateral cerebellar regions play a significant role in adjusting locomotor patterns to novel contexts or when strong visual guidance is required (adapted from Morton and Bastian,[13] Cooper et al.,[25] Grodd et al.,[72] and Marple-Horvat and Criado[73]).

cerebellar-unspecific safety strategies rather than specific deficits in control.[8,11,17] In contrast, the most striking and distinctive features of ataxic gait seem to be the high variability in several spatial and temporal gait features (eg variability in step length, step width, and gait cycle time).[8,11,17,18] This characteristic high variability of walking patterns most likely is because of the complex interaction between cerebellar-induced deficits in balance control and multi-joint coordination, the safety strategies used, and inaccurate adjustments to apparent losses of balance. This results in a continuous, step-by-step adjustment of the gait strategy, making it difficult to quantitatively describe the actual walking behavior of patients with ataxia.[17]

Factors Contributing to Ataxic Gait

Localization of Cerebellar Function in Gait Control

The cerebellum can be divided into distinct functional zones based on its afferent and efferent connections (Fig. 1).[19] Results, predominantly from animal studies, suggest that all these regions of the cerebellum play an important role in control and adjustment of balance and gait, each in different ways (see review by Morton and Bastian[20]).

In brief, the medial region (vermis), beyond its important involvement in eye movement control, plays a central role in locomotion by regulating extensor tone, sustaining upright stance and dynamic balance control, and modulating rhythmic flexor and extensor muscle activity (eg see Chambers and Sprague[21] and Sprague and Chambers[22]). The intermediate cerebellar regions seem to play an important role when performing precise limb movements, which involve directed limb placement and regulating agonist-antagonist muscle pairs to control the relative timing and movement amplitude (eg see Udo et al.,[23] Yu and Eidelberg,[24] and Cooper et al.[25]). The lateral cerebellum seems to be less important for the control of uninterrupted level walking. In contrast, it is suggested that lateral cerebellar regions play a significant role in adjusting locomotor patterns to novel contexts or when strong visual guidance is required[26,27] (for reviews, see Morton and Bastian[20] and Cerminara et al.[28]).

However, transferring these results to humans, it is important to keep in mind that human locomotion is bipedal and, thus, inherently less stable than quadrupedal locomotion. Human gait, therefore, likely requires additional supraspinal input for dynamic balance control,[7] potentially involving to a greater extent the intermediate and lateral cerebellar regions.

Balance Impairments in Posture and Gait

Some of the most reliable and most thoroughly documented deficits in cerebellar ataxia are postural

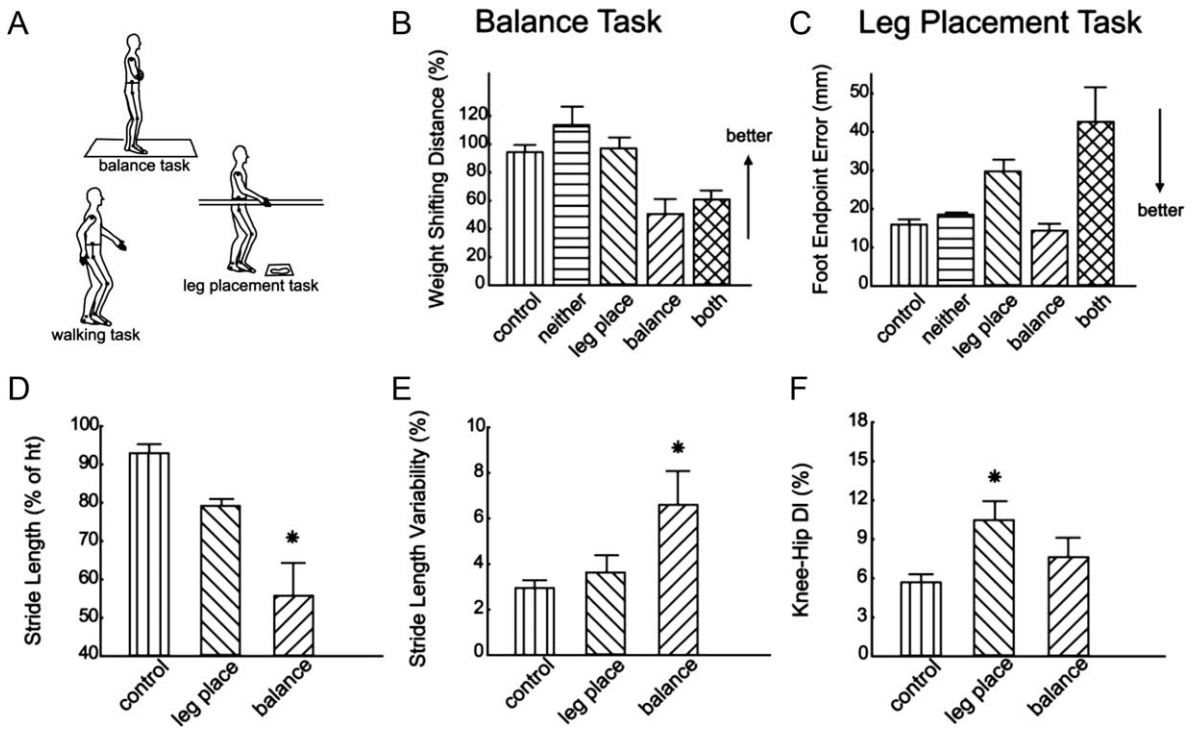

FIG. 2. (**A**) The experimental setup for the study by Morton and Bastian[13] is illustrated. During the balance task, the participant stood on the force plate with arms across the chest and feet shoulder-width apart. During the leg-placement task, the participant held onto parallel bars. During the walking task, participants walked across an uninterrupted, level platform. (**B**) Weight-shifting distances, normalized to foot spread, during the balance task are illustrated for all groups (see text). Participants with cerebellar disease in the balance deficit subgroup (balance) and in the both-deficits subgroup (both) had reduced lateral weight-shifting distances compared with the other groups. (**C**) Foot endpoint errors during the visually guided leg-placement task are illustrated. Patients with cerebellar disease in the leg-placement deficit and both-deficits subgroups had greater errors than all other groups. (**D–F**) Group means of walking variables are illustrated for the control group (n = 20), for patients with cerebellar disease in the leg-placement deficit subgroup, and for patients with cerebellar disease in the balance deficit subgroup, including (**D**) average stride lengths normalized to height ($P = 0.0005$); (**E**) stride-length variability (the coefficient of variability; $P = 0.0245$), and (**F**) decomposition at the knee-hip joint pair ($P = 0.0420$) (reproduced with permission from the American Physiological Society).

deficits in quiet stance, in response to balance disturbances,[29] and before voluntary limb movements, such as gait initiation.[4] Dysfunctions in postural control mechanisms have been identified, including increased gain and prolonged duration of long latency reflexes[30]; a disruption of sensorimotor processing for balance control[31]; disordered adaptation of postural responses; disordered coordination between head, trunk, and legs (asynergia); a loss of spatial orientation versus gravity; and impaired predictive postural adjustments.[4,29]

Accordingly, balance-related abnormalities are crucial and are often most obvious in ataxic gait, reflecting safety strategies like increasing step width or symptoms of impaired balance control, such as increased body sway. Consequently, a study by Morton and Bastian[13] suggested that cerebellar ataxic gait on level ground may be influenced predominantly by balance impairments, whereas leg coordination impairments may have only a minor influence. In that study, the authors tried to separate the influences of balance deficits and voluntary leg coordination deficits on cerebellar ataxic gait (Fig. 2). Based on patients' performance on a balance task and a leg-placement task, patients were assigned to 2 groups with dominantly balance-related and coordination-related impairments. Patients who had dominant balance-related deficits had significant deviations from normal controls for several gait parameters, including stride length and its variability and the peak angles of hip, knee, and ankle joints. Patients who had coordination-related impairments did not have significant deviations from normal controls for all tested gait parameters except for a movement decomposition index.[13,32]

Influences of Impairments on Intra-limb Coordination

However, Ilg et al.[8] have provided evidence that abnormalities in gait, like dynamic balance and irregular foot trajectories in ataxic gait, also are influenced by deficits in the control of intra-limb coordination, rather than solely being by-products of balance impairments. Walking patterns were quantitatively analyzed by combining standard gait measures and novel measures for the characterization of the spatial and the temporal variability of intra-joint coordination patterns (see Fig. 3). The temporal variability of gait patterns was significantly correlated with a subscale of

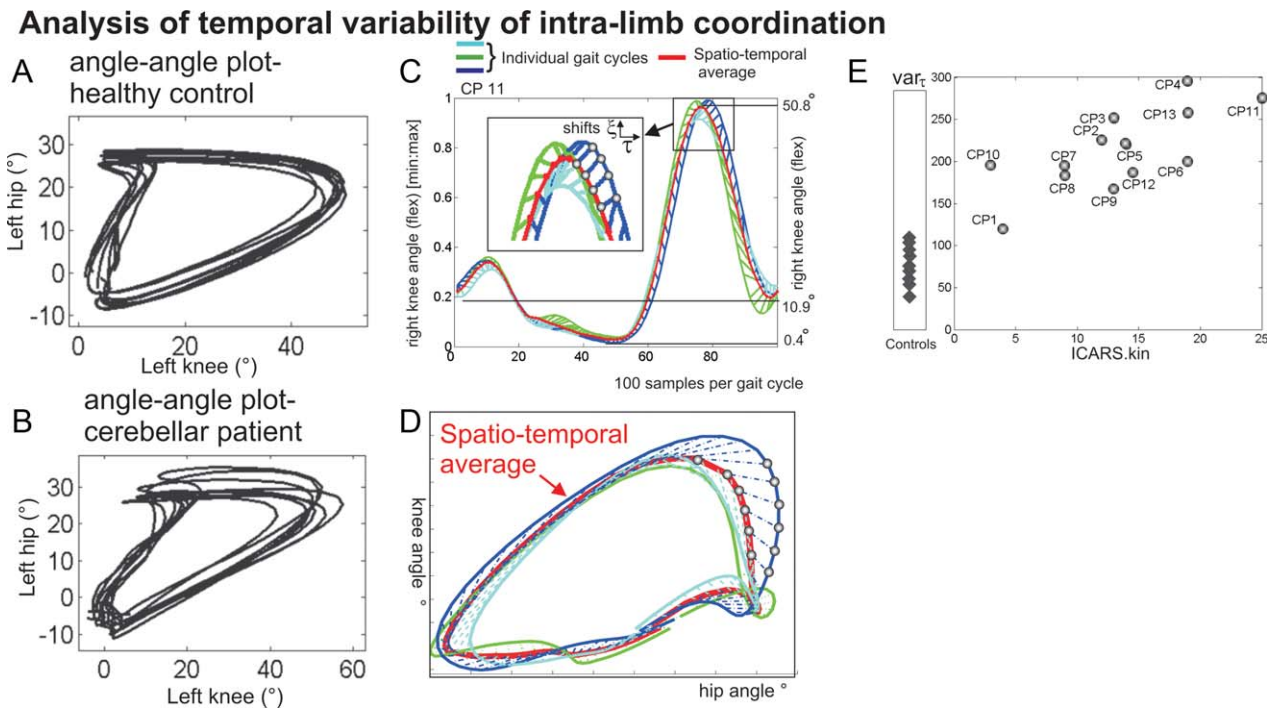

FIG. 3. Angle-angle plots illustrate the joint coordination patterns for the right and left leg in (**A**) a healthy control individual and (**B**) a patient (CP-11) with pancerebellar atrophy. (C) This is an illustration of spatial and temporal variability for real data and computation of the average movement for the gait trajectories of a patient with cerebellar disease. Joint angle trajectories (100 time samples) are indicated for the right knee flexion angle for 3 subsequent gait cycles. Thin lines between the trajectories connect points that are in spatio-temporal correspondence. (**D**) An analysis of the spatial and temporal inter-subject variability of 2-dimensional joint angle trajectories is illustrated. Trajectories are illustrated as angle-angle plots. In the 3 trajectories from individual gait cycles in the same patient (left), a spatio-temporal average trajectory is computed (right). (**E**) Temporal variability measures var_t are plotted against International Cooperative Ataxia Rating Scale (ICARS) kinetic subscores for 13 cerebellar patients and 9 controls. The variability measures (var) were determined for 1 exemplary set of joints, including flexion angles of the left hip and the left knee (L-HK)[8] (reproduced with permission from Oxford University Press).

the International Cooperative Ataxia Rating Scale (ICARS) clinical ataxia scale,[33] which rates deficits of the control of limb dynamics and intra-limb coordination for goal-directed movements. Conversely, gait parameters like step width and lateral sway were correlated predominantly with clinical measures of balance-related abnormalities. These results suggested that ataxic gait is influenced by both balance-related impairments and deficits related to limb control and intra-limb coordination. Applying the same analysis to patients with peripheral vestibular failure and Parkinson's disease indicates that increased temporal variability of intra-limb coordination is a specific characteristic of cerebellar dysfunction.

Influences of Oculomotor Dysfunction on Goal-directed Walking

The existence of links between these locomotor and oculomotor deficits has been provided by a study of Crowdy et al.[34] Eight patients suffering from primary cerebellar degenerative diseases undertook a walkway task, which demanded precise foot placement at each step, and a visual fixation task, which required only eye movements. Step cycle and horizontal eye movements were recorded throughout the tasks and were compared with those of healthy adults. Similarities between oculomotor deficits displayed by patients during the visual fixation task and during walking indicated that the latter were not merely a consequence of ataxic gait. In a subsequent study with a goal-directed foot placement task,[35] the same investigators observed that saccadic and foot placement accuracy could be improved by prior practice in making saccadic eye movements toward the targets.

Interactions of Cognitive and Motor Tasks—Dual-task Walking

In general, gait disorders in neurological patients often are accentuated when they perform concurrent cognitive tasks during walking (for review, see Woollacot and Shumway-Cook[36]). This has been extensively examined, for instance, in Parkinson's disease.[37,38] To date, there is still a lack of dual-task studies for cerebellar patients. However, the interaction of cognitive tasks and motor tasks may be especially relevant in cerebellar dysfunctions; because, along with the importance of the cerebellum on motor control walking, there is evidence for the involvement of the lateral cerebellum in cognitive processes like working memory.[39,40]

In a recent study, we examined the influence of focal cerebellar lesions on working memory (*N*-back task)

and on the interaction between working memory and gait tasks with different complexities in a dual task paradigm.[41] Seventeen young patients with who had chronic focal lesions after undergoing resection of cerebellar tumors and 17 age-matched controls were analyzed. Patients had mild to moderate ataxia. *N*-back tasks were executed with different levels of difficulty during sitting (baseline), treadmill walking, and treadmill tandem walking. For patients, gait analysis revealed that patients increased gait variability in the walking and tandem walking tasks. This gait variability scaled with *N*-back task difficulty only for tandem gait. These results indicate that, in patients with predominantly mild ataxia, additional cognitive load causes increased gait impairments only for more complex gait tasks like tandem walking. Lesion-symptom mapping analyses of regions associated with impairments in both the *N*-back task (baseline) and the walking tasks revealed no overlaps for walking but did find overlapping regions in the lateral hemisphere and the dentate for tandem gait, reflecting the visually guided, precise foot placement. These findings suggest that the interaction effects that have been specifically identified for difficult dual-task conditions may be caused, along with the prioritization of gait for safety reason, by a common involvement of lateral cerebellar regions in working memory as well as in complex motor tasks. This hypothesis implies that the clinical relevance of gait disturbances and falls may be accentuated in demanding cognitive tasks.

Adaptation of Walking Pattern

The cerebellum is also known to play a functional role in motor learning and motor adaptation (see reviews by Bastian[1,42]). Impairments of cerebellar patients in (short-term) practice-dependent motor learning have been demonstrated in various motor tasks on arm movements[43–45] and walking movements.[46–48] Such short-term adaptations occur in many situations that are relevant for everyday life, like the adjustment of leg control to changes in leg dynamics caused by wearing heavy winter shoes. This means that patients are impaired in finding optimal calibrations for movement control, which makes movements inaccurate and unsafe.

Such impairments in the adaptation of movement patterns to changed leg dynamics and its relation to goal-directed leg movements has been demonstrated.[46] Cerebellar patients performed 3 tasks: goal-directed leg placement, walking, and walking with additional weights on the shanks. Based on the performance on the first 2 tasks, patients were categorized as impaired or unimpaired for leg placement and for dynamic balance control in gait. The subgroup with impaired leg placement demonstrated abnormalities in the adaptation of locomotion to additional loads, whereas the subgroup with impaired balance did not. A detailed analysis revealed specific abnormalities in the temporal aspects of intra-limb coordination for leg placement and adaptive locomotion. Lesion-based magnetic resonance imaging subtraction analysis revealed that the interposed and adjacent dentate nuclei were more frequently affected in patients with impaired versus unimpaired leg placement. Thus, these results indicated, for the first time in humans, an influence of the intermediate zone for multi-joint limb control both in goal-directed leg movements and in adaptive locomotion.

The importance of the cerebellum in the adaptation of temporal aspects of walking pattern has been systematically examined by split-belt treadmill walking.[47] Participants walked on a split-belt treadmill in which one belt was suddenly driven at twice the speed of the other. Along with rapid reactive changes to account for this speed difference, healthy controls also made gradual, adaptive changes in the phasing of motion between the legs to optimize the inter-limb relation. The new phase relation was stored as a predictive calibration, as evidenced by slowly decaying after effects that appeared when the treadmill belts were returned to the same speed. Cerebellar patients exhibited normal reactive changes but impairments in making the predictive changes in temporal aspects of interlimb coordination.

Rehabilitation of Ataxic Gait

Functional recovery of cerebellar impairments heavily depends on the cause and site of the lesion. Ataxia after stroke, neurosurgery, or trauma affects only circumscribed regions of the cerebellum while leaving other regions intact. These unaffected regions may compensate for the defective parts. In addition, in the case of focal lesions, effects of neural plasticity are likely more effective, because there is no competition with ongoing progressive neurodegeneration. Whereas patients with focal lesions clearly improve in motor functions over time, patients with degeneration slowly deteriorate.[49] In focal cerebellar lesions, caused either by tumor surgery or stroke, lesion site appears to be more important than extent, and lesions affecting the deep cerebellar nuclei are not fully compensated at any age in humans.[50] Furthermore, additional extracerebellar stroke lesions involving the cerebral hemispheres or brainstem also negatively affect recovery of walking and other functional capacities after stroke.[51]

Within the spectrum of cerebellar diseases, degenerative diseases are especially difficult to treat because of their progressive nature and their effect on virtually all parts of the cerebellum. Possibilities for medical interventions are rare and are limited to specific diseases and symptoms (reviewed by Ilg and Timmann[10]). Furthermore, motor rehabilitation is also

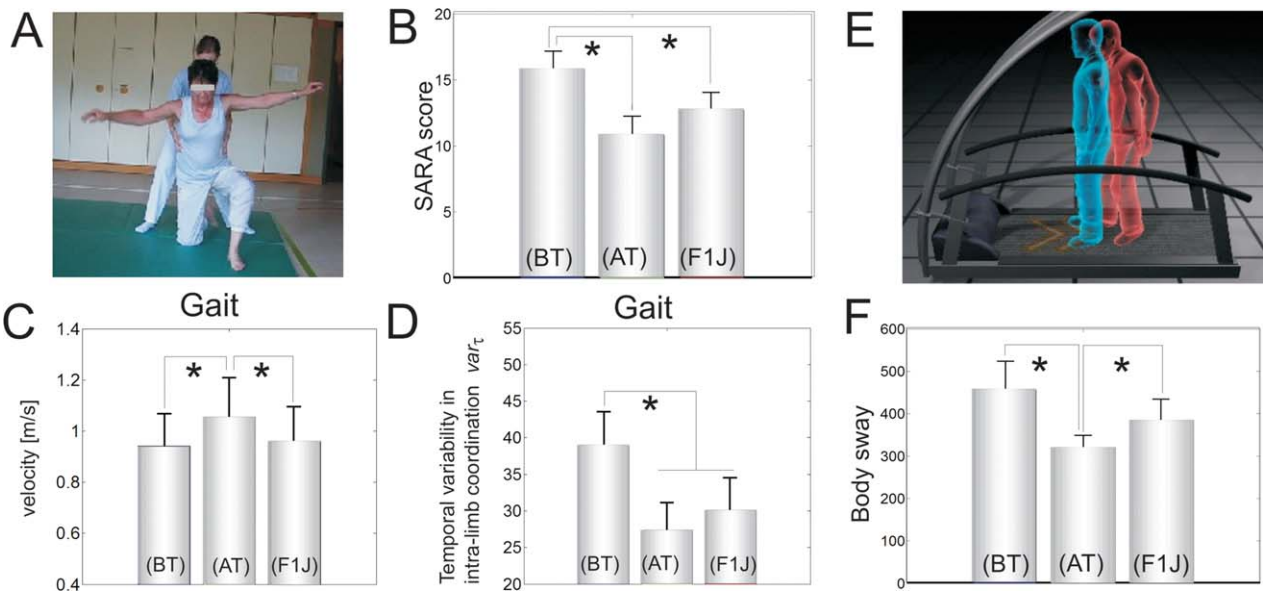

FIG. 4. (**A**) This snapshot of a demanding exercise illustrates training of dynamic balance and multi-joint coordination. (**B**) Group data from the clinical ataxia scores on the Scale for the Assessment and Rating of Ataxia (SARA) are illustrated before the training intervention (BT), after the 4-week training intervention (AT), and for follow-up assessment after 1 year. (**C,D**) Group comparisons of measures from quantitative gait analysis are provided. Groups of 3 bars indicate the examinations BT, AT, and FU; asterisks indicate significant differences between examinations ($P < 0.05$); and error bars denote standard errors. (**E**) This is an illustration of an experiment testing dynamic balance capacities. Patients have to compensate for the perturbation of the accelerating treadmill (accelerating phase, 1 second) by anterior-directed steps. The red and blue characters show one and the same participant before (red) and after (blue) the intervention period. After the intervention, participants were able to compensate for the perturbation more efficiently and robustly. (**F**) Quantitative measurement for the body sway in the perturbation experiment is illustrated for the 3 assessment points[60] (reproduced with permission).

challenging for this patient population because of the aforementioned functional role of the cerebellum in motor learning and motor adaptation. Therefore, poor recovery or low benefit of physiotherapeutic training may be a consequence of damaging structures critically involved in relearning of motor skills.[1,52]

Relatively few clinical studies have evaluated physiotherapeutic interventions for patients with cerebellar ataxia, most of them consisting of single cases or very small patient populations with different types of cerebellar disease and severity of ataxia (for reviews, see Ilg and Timmann[10] and Marsden and Harris[53]). Using increasingly demanding balance and gait tasks, improvements were reached in postural stability and in terms of less dependency on walking aids in everyday life.[54,55] Locomotion training on treadmills with[56,57] or without[58] body-weight support have been proposed, in particular for patients with more severe ataxia, who are not able to walk freely.

Recently, the benefits of intensive coordinative training on posture and gait in degenerative cerebellar disease were examined systematically in an intra-individual case-control design.[59,60] Sixteen patients suffering from progressive ataxia due to cerebellar degeneration (n = 10) or degeneration of afferent pathways (n = 6) were tested. The results indicated that, despite a gradual decline in motor performance and a gradual increase in ataxia symptoms because of the progression of underlying neurodegeneration, patient benefits could be meaningful for everyday life and persisted after 1 year. The strategy of the physiotherapeutic intervention was to activate and demand control mechanisms for balance control and multi-joint coordination. Furthermore, the intervention trained the patients' ability to select and use visual, somatosensory, and vestibular inputs to preserve and retrain patients' capability for reacting to unforeseen situations and for avoiding falls as much as possible. The physiotherapeutic program consisted of a 4-week course of intensive training with 3 sessions of 1 hour per week. Exercises included the following categories: 1) static balance, eg standing on 1 leg; 2) dynamic balance, eg sidesteps, climbing stairs; 3) complex, whole-body movements to train trunk-limb coordination; and 4) steps to prevent falling and falling strategies (for more details, see Ilg et al.)[59] An important principle of the motor training was to train increasingly demanding movements (from static to dynamic balance; from slow to fast movements; and from single joint movements to complex, multi-joint coordination). The specificity of motor improvements was demonstrated by distinct measures reflecting multi-joint coordination, like the temporal variability in intra-limb coordination in gait (see Fig. 4C,D), as well as by the quantification of dynamic balance capacities for the compensation of a perturbation in stance.

A related study[61] that combined physiotherapy with occupational therapy in 42 patients with degenerative

cerebellar ataxia revealed improvements of ataxia severity, gait speed, fall frequency, and activities of daily living. The improvement was more prominent in trunk ataxia than in limb ataxia, and patients with mild ataxia severity experienced a more sustained improvement.

Open Questions and Future Directions

Mechanisms of Multi-joint Coordination in Gait

Although the influence of cerebellar dysfunctions on intra-limb coordination during walking has been suggested by several clinical studies on a behavioral level,[8,17,46] our knowledge about the specific involvement of the cerebellum in control mechanisms for different walking conditions is still limited. Animal studies suggest an involvement in the control of limb dynamics as well as the modulation of rhythmic movements,[26,62] because lesions in the intermediate region of the cerebellum cause significant hypermetria in ipsilateral joints[21,24] and impairments of timing of the touch-down and lift-off events.[23] For a more detailed review of animal studies, see Morton and Bastian.[20]

Open questions include whether mechanisms like the predictive compensation of interaction torques in multi-joint movements—which is assumed to be a substantial cerebellar function in goal-directed arm movements[2]—involve the cerebellum for straight walking or only in special conditions when compensating for gait perturbations[63] and obstacles,[64] in which visual guidance play a stronger role. It has been suggested that the nervous system utilizes intersegmental dynamics during the reactive control of locomotion.[63] However, it remains largely unclear the extent to which and under which conditions forward models in the cerebellum contribute to this by prediction of passive limb dynamics. Future studies in this direction would strengthen our knowledge about the functional role of the cerebellum in walking and would help to design more efficient motor rehabilitation programs.

Gait Rehabilitation

Gait rehabilitation will remain a challenge for patients, physicians, and therapists, especially for degenerative cerebellar disease. However, the aforementioned recent advances will provoke further studies and hopefully lead to broader knowledge in this challenging field of motor rehabilitation.

Future studies should include imaging analyses to clarify whether the degenerating cerebellum is still able to adapt motor coordination or whether the learning deficit is compensated by other brain structures, which is suggested by a first study examining brain changes associated with postural training in patients with cerebellar degeneration.[65] In addition, studies on animal models of cerebellar ataxia have shown that intensive motor training could even slow down the process of degeneration.[66,67] Long-term rehabilitation studies in humans are needed to determine whether these promising results also hold for cerebellar patients.

Furthermore, studies are needed to examine whether patients with more severe impairments would also benefit from physiotherapeutic training or whether the capacity to improve motor performance relies on a specific level of residual cerebellar integrity. The aim is to find adequate predictors for the potential benefits an individual patient can expect from a specific motor training and, thus, the possibility of providing efficient training programs for different stages of cerebellar impairment. One such predictor could be the capability of short-term motor adaptation. Hatakenaka et al.[68] have demonstrated that, for ataxic patients with infratentorial stroke, the degree of impaired motor learning is correlated with reduced long-term rehabilitation gains.

Conclusions

Although a general description of ataxic gait has existed for centuries,[5] we do not fully understand the underlying mechanisms. Functionally, ataxic movement patterns emerge from the interactions between primary cerebellar control deficits, such as balance control and multi-joint coordination, influence of oculomotor impairments, safety strategies, and dysmetric compensatory behaviors. Therefore, the most characteristic and specific aspect of ataxic gait seems to be high variability in both temporal and spatial gait parameters.

A further understanding of the different influences in ataxic gait also will enable the development of individualized training concepts, which—along with pure motor training—should involve multiple training modalities (dual-task training, oculomotor) and also potentially rehabilitation technology, such as biofeedback[69,70] and noninvasive stimulation,[71] to further support rehabilitation.

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