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Role of cerebellum in learning postural tasks

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Abstract
For a long time, the cerebellum has been known to be a structure related to posture and equilibrium control. According to the anatomic structure of inputs and internal structure of the cerebellum, its role in learning was theoretically reasoned and experimentally proved. The hypothesis of an inverse internal model based on feedback-error learning mechanism combines feedforward control by the cerebellum and feedback control by the cerebral motor cortex. The cerebellar cortex is suggested to acquire internal models of the body and objects in the external world. During learning of a new tool the motor cortex receives feedback from the realized movement while the cerebellum produces only feedforward command. To realize a desired movement without feedback of the realized movement, the cerebellum needs to form an inverse model of the hand/ arm system. This suggestion was supported by FMRi data. The role of cerebellum in learning new postural tasks mainly concerns reorganization of natural synergies. A learned postural pattern in dogs has been shown to be disturbed after lesions of the cerebral motor cortex or cerebellar nuclei. In humans, learning voluntary control of center of pressure position is greatly disturbed after cerebellar lesions. However, motor cortex and basal ganglia are also involved in the feedback learning postural tasks.

Key words: *Motor learning, posture, cerebellum, motor cortex, basal ganglia*

Introduction: Cerebellum and postural control

The role of cerebellum in motor control and, particularly, in postural control (position of the supporting segments, i.e., trunk and limbs) and equilibrium was described in clinical and experimental studies (1–8). Luciani (8) pointed out the role of the cerebellum in control of postural tone and muscle force. He described the main results of cerebellar lesions: atonia, asthenia, astasia and dysmetria. Later on, these symptoms were detailed and role of the cerebellum in timing between movement components and coordination of the agonists and antagonists sequence was described (see 3). Babinski (9) demonstrated a characteristic balance deficit during backward body inclinations in patients with cerebellar lesions. Further studies showed that lesions of different parts of cerebellum may result in different postural deficits because of their various afferent and efferent connections (2,10,11). According to the contemporary clinical and 3D MRI data (12), limb ataxia is mainly correlated with lesions of the interposited and part of the dentate nuclei, ataxia of posture and gait with lesions of the fastigial nuclei including interposited

nucleus. Correlations with cortical lesions are less significant and present in the superior cerebellum only. Ataxia of posture and gait is correlated with lesions of vermal and paravermal lobules II, III and IV. Lesions involved in the cerebellar nuclei are shown to be not fully compensated. Massion (13) distinguishes two main functions of the postural control system: (i) antigravity and balance function, and (ii) orientation and position of segments as a reference frame for perception and action with respect to the external world. Concerning the balance control, two discrete strategies for the postural reactions to external perturbation in the sagittal plane have been described, namely, ankle and hip strategies (14). With regards to the role of sensory inputs in postural stability, the vestibular system has been shown to be involved in hip strategy (7,15,16). In patients with cerebellar anterior lobe pathology, somatosensory feedback could be used for scaling of the disturbance velocities while the scaling of initial postural response magnitude to expected displacement amplitudes disappeared (16).

An important point of postural control is anticipatory postural adjustments (APAs) which was described first in animals (17) and then in humans

(18). After cerebellar lesions, the basic pattern of APA was preserved but the temporal coordination of muscle activity and scaling of muscle force was found to be under cerebellar control (19). Temporal discoordination after cerebellar lesions was also observed in bimanual unloading reaction where anticipatory postural adjustments consisted of changes in the activity of a forearm flexor muscle prior to active unloading of the limb and acted to stabilize the forearm position (20).

Role of cerebellum in learning

In addition, the cerebellum is also regarded as a structure involved in learning. The theoretical background for this was elaborated by Brindley (21), Marr (22) and Albus (23). The theory is based on the anatomic structure of inputs and the internal structure of the cerebellum.

Brindley suggested that cerebellum adjusts cortical motor command with the context of the movement. According to Marr's theory, afferent information (context) reaches the cerebellum by activation of mossy fibers bringing it to many Purkinje cells through granule cells and parallel fibers, while an efferent command copy comes by climbing fibers from the inferior olive to individual Purkinje cells. By this way, the cerebellum can correct the inital motor command taking into account features of the context: 'The "learned" movement is distinguished from the "unlearned" conscious movement by its now being automatic, rapid, and stereotyped' (24). Albus (23) proposed that the climbing fiber discharge suppresses rather than activates the Purkinje cell response to the input activity of parallel fibers. Since Purkinje cells inhibit the cerebellar nuclei, the depression of their activity results in activation of the nuclei and an increase in the output command. The Marr-Albus hypothesis was supported in a number of experiments (11,25,26). Some changes to the model were brought by the adjustable pattern generator (APG) model with quasi-feedforward characteristics (27). Then the general theory on error-driven LTD-based reorganization of the neuronal circuit in a microcomplex, functional module of the cerebellum as a basic mechanism of motor learning was formulated and signal transduction for LTD was analyzed by Ito (28,29). According to the Marr-Albus-Ito hypothesis, this error-driven reorganization is promoted by climbing fibers and what is reorganized is the efficacy of the coupling between parallel fibers and P-cells. However, Llinas and Welsh (30) suggested that the climbing fiber input is directly involved in eliciting the motor response, rather than in modulating the strength of parallel fiber/P-cells coupling. Thus, the olivocerebellar system is mainly involved in the coordination of movements. Thach (24,31) has reviewed the role of cerebellum in motor coordination. He paid attention to activation of varied combination of muscles in precise time-amplitude specification so as to produce coordinated movements appropriate to specific contexts. Particularly, he discussed the influence of cerebellum on frontal planning area in order to take part in planning movements that are to be executed, and movements that are not to be executed (24).

It appears that an important function of the cerebellum is storing motor memory (28). Miles and Lisberger (32) proposed, however, that the role of the cerebellum was not to store the motor memory but rather to compute the instructive signal guiding the induction of plasticity. This position has been supported by Boyden et al. (33).

Classical conception of motor learning is based on the main role of feedback afferent control for error corrections. However, fast and coordinated movements cannot be performed solely under feedback control, since biological feedback loops are very slow and have small gains. A popular hypothesis suggests that, during motor learning, the brain uses feedback error control to acquire neural internal models of the motor apparatus and environment for planning and executing movements. The hypothesis of inverse internal model (34–36) is based on feedback-error learning mechanism combining feedforward control by the cerebellum and feedback control by the cerebral motor cortex. This suggests that the cerebellar cortex can acquire internal models of the body and objects in the external world. During learning a new task the motor cortex receives feedback afferent signal from the realized movement while the cerebellum produces only feedforward command. So, to realize a desired movement without feedback of the realized movement, the cerebellum needs to form an inverse model of the hand/arm system. This suggestion was supported by FMRi data (35). Multiple internal models have been proposed and shown to be able to control different movements in different situations (37).

A relative contribution of the cerebellar cortex and nuclei to motor learning was suggested to depend on the characteristics of training (38). The cerebellum has been shown to be important for making movement more automatic (requiring less attention to the details of a movement) (39). The subjects with cerebellar lesions could improve the movement being learned to a very limited extend with practice. The motor performance decreased to prepractice level when attention was focused away from the performed movement. Stimulation of cerebellar nuclei during performimg a learned movement in animals interferes with the movement program and disorganizes it (40). The high frequency stimulation (100–500 Hz) produced postural disturbances, and the strengthening of the stimulation resulted in the loss of the equilibrium (41).

Cerebellum and learning postural tasks

The role of the cerebellum in the reorganization of posture and in learning new postural tasks has also been studied. The anterior cerebellum has been shown to play a critical role in modifying the magnitude of automatic postural responses to a platform displacement to anticipated displacement conditions based on prior experience (16). The cerebellum appears to be important for predictive adaptation of anticipatory postural adjustment during step initiation (42). Distinct parts of the medial and lateral cerebellum are shown to be involved in long-term habituation of the acoustic startle response (implicit learning of unspecific postural aversive reactions) (43,44). However, no difference in changes of postural response size to repeated perturbations during training have been found between groups of patients with cerebellar lesions and control subjects (45). Meanwhile, classical conditioning (tilting of the platform preceded by an auditory conditioning stimulus) has been shown to be remarkably disturbed in cerebellar patients (46). As was mentioned above, temporal organization of the learned anticipation of the forearm flexion in bimanual unloading task is also disturbed in patients with cerebellar lesions (20).

The presented data are somewhat controversial but in general they show that the cerebellum is involved into control of learned automatic postural reactions, particularly in their temporal and magnitude structure. Let us consider now a role of cerebellum in reorganization of postural coordination by learning.

Reorganization of innate postural pattern in animals

When motor coordination has to be changed, it needs time for retraining (27,31). The cerebellum plays an important role in such reorganization of motor coordination. Cerebellar nuclei lesions disturb the performance of learned coordination in animals (40). Moreover, a learned postural pattern in dogs has been shown to be disturbed after lesions of the cerebral motor cortex (47,48) or cerebellar nuclei (49). When dogs lift a forelimb in a conditioned avoidance task, they normally exhibit a diagonal pattern of postural adjustment: the limb diagonally opposite to the lifted one is unloaded while the other pair of diagonal limbs is loaded. In the mentioned experiments, dogs were trained to suppress this natural pattern and to perform the so-called unilateral pattern: to unload the hindlimb ipsilateral to the limb being lifted, and to load the two contralateral limbs (Fig. 1). The diagonality coefficient was calculated using the formula

$$D = 1 - \frac{|\Delta F_1 - \Delta F_4| + |\Delta F_2 - \Delta F_3|}{|\Delta F_1| + |\Delta F_2| + |\Delta F_3| + |\Delta F_4|}$$
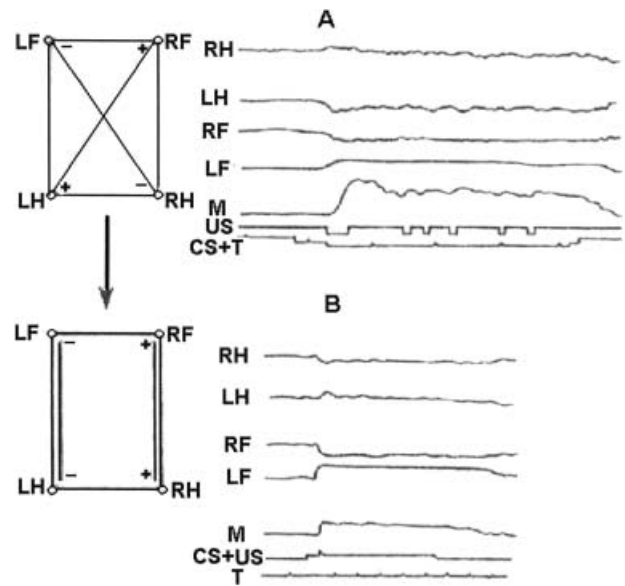

Figure 1. Rearrangement of the diagonal pattern of postural adjustment accompanying the avoidance reaction into a unilateral one. (A) Diagonal pattern of postural adjustment (simultaneous unloading vs. loading in pairs of the diagonal limbs); (B) Unilateral pattern of postural adjustment (simultaneous unloading vs. loading in pairs of the ipsilateral limbs). On the left, schemes of the support forces changes, on the right, fragments of the recordings: LF, RF, LH, RH, force traces of the left and right forelimbs and left and right hindlimbs, respectively (+ and downward force traces displacement correspond to loading, − and upward displacement correspond to unloading), M, trace of the limb movement, CS, US, marks of the conditioned and unconditioned stimuli, T, time marks, s.

where D is the value of the coefficient of diagonality, F1, F2, F3, F4, values of suppport forces of left and right forelimbs and left and right hindlimbs, respectively. Maximum D value equal to 1 corresponds to the lifting of two diagonal limbs (50). The trajectories of limb movements, center of gravity displacement and the diagonality coefficient were assessed (Fig. 2).

A lesion of the motor cortex in the hemisphere contralateral to the limb being lifted results in temporary disturbances of the reorganized pattern of postural adjustment which may be recovered by retraining for 3–4 weeks. However, a subsequent lesion in the ipsilateral motor cortex causes stable disappearance of the learned postural pattern, and only diagonal pattern of postural adjustment can be elicited. No recovery of the unilateral pattern may be obtained by retraining. It was shown earlier (51), that the learned head-forelimb coordination (maintaining a cup with food lifted by the lifted forelimb during eating with lowered head) in dogs disappeared after lesion of the motor cortex and only natural coordination (lowering the forelimb during lowering the head) was possible. Meantime, the motor cortex lesion did not prevent the forelimb lifting itself since the eating with maintaining the forelimb lifted was still possible after motor cortex lesion if the food was given from above and the dog

could eat with lifted but not lowered head. This suggested that during learning the motor cortex inhibits motor patterns interfering with the pattern being learned. The above experiments with learning a new postural pattern suggest that the motor cortex inhibits interfering postural coordination during their rearrangement as well as it does for limb movements (47,48).

Some interesting results were seen in studies of the role of cerebellum in such reorganization of the natural postural pattern (49). Learning began after bilateral electrolytic lesions of cerebellar nuclei in two groups of dogs. Both the dentate and inter-positus were lesioned in two dogs (Group 1) and only the dentate nucleus in two others (Group 2).

Training results were different in the two groups of animals (Fig. 3). After combined bilateral lesions of dentate and interpositus (Group 1), the dogs had dramatic difficulties in maintaining position of the lifted limb, stabilization of the center of gravity trajectory, and its final position. Learning the new unilateral postural pattern was impossible despite three months of postoperative training. By contrast, when the dentate alone was lesioned (Group 2), the dogs learned to keep the limb lifted, to perform ballistic and stereotyped center of gravity displacement, and to produce the new unilateral pattern. The results show that the cerebellum plays a role in reorganization of the natural postural pattern. In particular, the role of the interpositus is very important. Some possibilities can be considered. The first is that interpositus itself plays a role in learning a new program that results in reorganization of the natural coordination. The other suggestion is that interpositus can compensate for the deficit of posture and movement after the dentate lesion. A basis for this might be axonal sprouting, particularly, changes of the location of cortico-rubral or inter-posito-rubral terminals on rubral neurons (52). Also, neural plasticity in cerebellum, manly based on LTD of parallel fiber synapses onto Purkinje cells or on multiple mechanisms (11,27,28,33) might be a mechanism of the reorganization. However, there were no selective lesions of the interpositus in the described experiments, so we can not exclude the possibility that, in normal conditions, both nuclei take part in the reorganization of the natural postural pattern.

Supervised learning of postural tasks in humans

However, other brain structures besides the cere-bellum play important roles in motor learning. According to the 'computational hypothesis of learning' (53), three kinds of learning can be distinguished: supervised learning, reinforcement learning and unsupervised learning. Supervised learning (feedback learning) is based on the
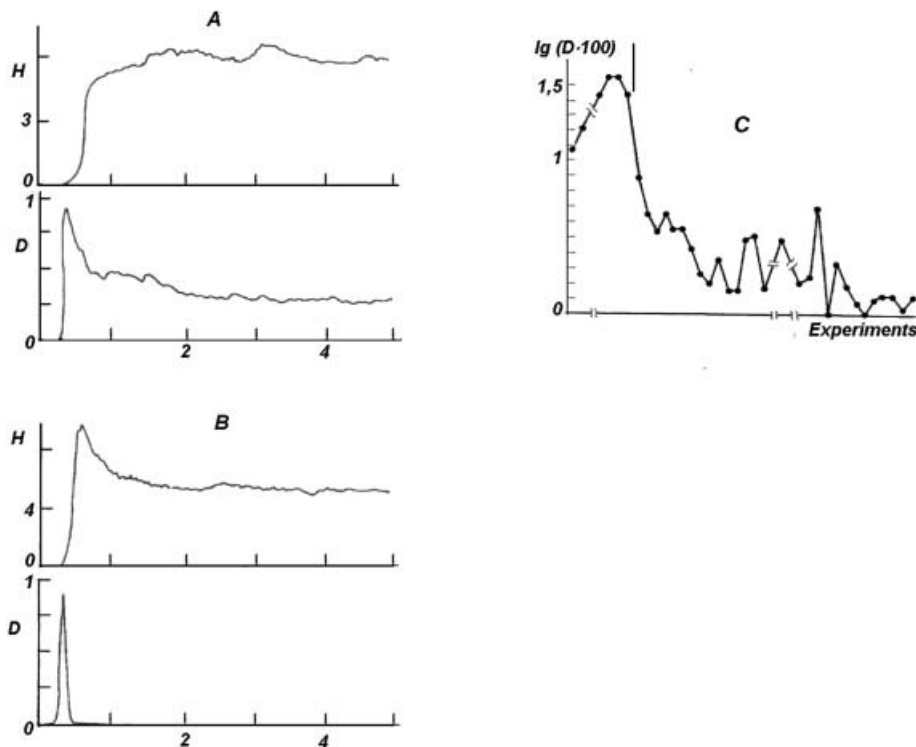
Figure 2. Changes of the diagonality coefficient (D) in the course of the rearrangement of the diagonal pattern of postural adjustment into a unilateral one. (A) Changes of the limb movement amplitude (H, cm) and diagonality coefficient (D) in the well trained avoidance reflex. Average of 10 trials; (B) The same, after the rearrangement of diagonal pattern of postural adjustment into unilateral. Average of 10 trials. Abscissa, time marks, s; (C) Dynamics of D in the course of rearrangement of diagonal postural pattern into unilateral; abscissa, successive sessions, ordinate, logarithm of mean values of D for a session, per cent.
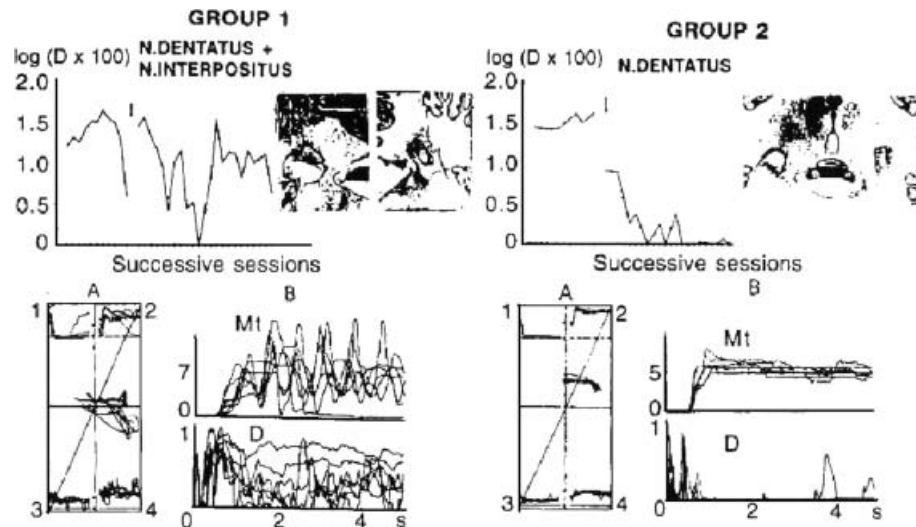
Figure 3. Rearrangement of the diagonal postural pattern in two groups of animals. *Top*, dynamics of diagonality coefficient. Abscissa, successive sessions (3 sessions per week), ordinate, logarithm of mean values of D for a session, per cent. Arrows: beginning of the rearrangement. *Bottom*, (A) force traces in the corners and trajectories of the center of gravity in the center; (B) time course of the limb movement (Mt) and diagonality (D). Abscissa, time, s; ordinate, amplitude of the limb lifting (cm) and value of diagonality. Superposition of eight trials. The traces on the bottom belong to the last period of training.

evaluation of error during performing a reaction and correction of it for improving the result. Feedback learning is usually involved in motor learning when an error signal is available. The cerebellum estimates an error signal and is considered to play a pivotal role in supervised learning. Reinforcement learning is learning what to do in a given situation to maximize the probability and value of reward. The basal ganglia are shown to be involved in the estimation of the reinforcement value and selection of the action to reach it, thus they play the main role in reinforcement learning. The cerebral cortex is considered to work by the principle of unsupervised learning, i.e., purely in an associative manner based on Hebbian plasticity (35,53,54). Further, different neuromodulators are thought to have roles in different kinds of learning. According to the above-mentioned hypothesis of multiple internal models, various 'responsibility' signals have been suggested for gating the learning of the internal models (37). It is proposed that serotonin controls the 'responsibility' of each cerebellar unit (or microcomplex) in cerebellar learning and control; norepinephrine is assumed to play 'gating of learning' role in the cerebellar cortex; dopamine enhances goal-oriented cerebellar learning; and, finally, acetylcholine controls the speed of supervised learning in Purkinje cells (37).

Since there are a lot of data concerning the role of the motor cortex in learning, including error estimation, it seemed important to study the roles of cerebellum, basal ganglia, and motor cortex in supervised learning. A particular question concerned the role of these structures in learning new postural tasks. This was studied by learning voluntary control of center of pressure (CP) position in visual feedback training in patients with

lesions of the motor cortex or pyramidal system (poststroke hemiparesis), nigro-striatal system (Parkinson's disease) and cerebellum (spinocerebellar ataxia) (55). The subjects stood on a force platform and were trained to change the position of the center of pressure presented as a cursor on a monitor screen in front of the patient (Fig. 4). Subjects were instructed to align the CP with the target and then move the target by shifting the CP in the indicated direction. Two different tasks were used. In 'Balls', the target (a ball) position varied randomly, so the subject learned a general strategy of voluntary CP control. In 'Bricks', the subject had to always move the target in a single direction (downward) from the top to the bottom of the screen, so that a precise postural coordination had to be learned. The training consisted of 10 sessions for each task. The number of correctly performed trials for a session (2 min for each task) was scored. The voluntary control of the CP position was initially impaired in all groups of patients in both tasks. In 'Balls', there were no differences between the groups of the patients on the first day. The learning course was somewhat better in hemiparetic patients than in the other groups while in the group of cerebellar patients the learning course was the worst. In 'Bricks', the initial deficit was greater in the groups of parkinsonian and cerebellar patients than in hemiparetic patients. However, learning was more efficient in parkinsonian than in hemiparetic and cerebellar patients. In the group of cerebellar patients the learning course in 'Bricks' was the worst as well. After 10 days of training, the hemiparetic and cerebellar patients had reached a plateau with no further acquisition (the lowest level was in the cerebellar patients group) whereas the parkinsonian patients showed the potential for
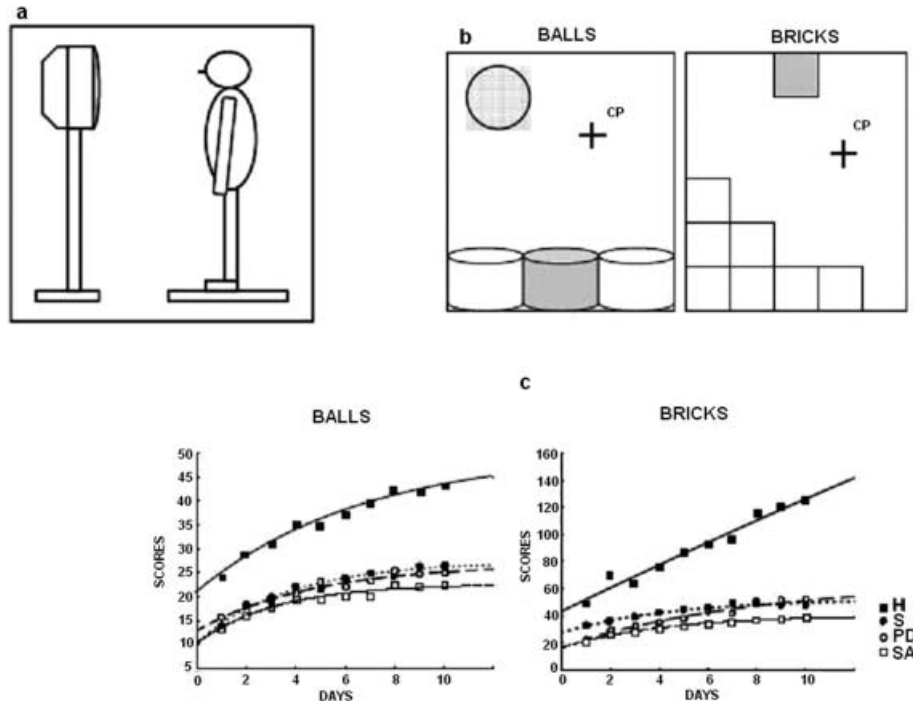
Figure 4. Feedback learning two postural tasks in patients with different cerebral lesions. (A) Sketch of the experimental paradigm; (B) Representation of the computer games: balls (left) and bricks (right). The cross corresponds to the position of CP. A ball (left) appeared randomly in different parts of the screen and the subjects learned to catch the ball by the cursor and to move the CP to put the ball into a randomly illuminated (shaded) basket. A brick (right) appeared always on the top of the screen and the subject had to put it down to arrange rows of bricks. The number of correct trials for two minutes of the session was scored; (C) Regression curves corresponding to the learning course in different investigated groups. Exponential model: S, stroke group; PD, Parkinson's disease group; SA, spinocerebellar ataxia group; H, group of healthy subjects (55).

further improvement. The results suggest that the motor cortex, cerebellum, and basal ganglia are all involved in voluntary control of posture and learning different postural tasks. However, these structures play different roles in postural control and learning: the basal ganglia are mainly involved in learning a general strategy of CP control while the function of the motor cortex chiefly concerns learning a specific CP trajectory. The cerebellum is involved in both kinds of learning. Both learning a general strategy of CP control and CP trajectory are more disturbed after cerebellar lesions than after isolated basal ganglia or motor cortex lesions.

Conclusion

Thus, the cerebellum plays a very important role in learning new postural tasks. Though other brain systems such as the basal ganglia and the motor cortex-pyramidal system are specifically involved in this process as well, the cerebellum seems to be one of the main structures providing learning voluntary control of posture. The above mentioned cerebellar mechanisms of feedforward-feedback learning could be a basis of this process. Particularly, the motor cortex might be involved in feedback control whereas the cerebellum might play a role in feedforward control by acquiring internal models in new postural tasks.

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