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REVIEW

Does the Cerebellum Initiate Movement?

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Abstract Opinion is divided on what the exact function of the cerebellum is. Experiments are summarized that support the following views: (1) the cerebellum is a combiner of multiple movement factors; (2) it contains anatomically fixed permanent focal representation of individual body parts (muscles and segments) and movement modes (e.g., vestibular driven vs. cognitive driven); (3) it contains flexible changing representations/memory of physical properties of the body parts including muscle strength, segment inertia, joint viscosity, and segmental interaction torques (dynamics); (4) it contains mechanisms for learning and storage of the properties in item no. 3 through trial-and-error practice; (5) it provides for linkage of body parts, motor modes, and motordynamics via the parallel fiber system; (6) it combines and integrates the many factors so as to initiate coordinated movements of the many body parts; (7) it is thus enabled to play the unique role of initiating coordinated movements; and (8) this unique causative role is evidenced by the fact that: (a) electrical stimulation of the cerebellum can initiate compound coordinated movements; (b) in naturally initiated compound movements, cerebellar discharge precedes that in downstream target structures such as motor cerebral cortex; and (c) cerebellar ablation abolishes the natural production of compound movements in the awake alert individuals.

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Introduction

Despite over 100 years of physiological research in lower animals and humans on the cerebellum, opinion is still divided on what its exact functions really are. This is especially true of the question of whether it modulates movements (some or all) through tonic excitatory discharge that provides fine tuning so as to permit other downstream target structures to initiate and coordinate them [19] or whether it instead initiates compound coordinated movements through programs contained and integrated entirely and exclusively within the cerebellum itself [20]. In this review, we summarize experiments that argue for the quintessential cerebellar role in the initiation and the coordination of compound movements.

To help appreciate the differences between these two proposed modes of operation, some metaphors may be used to illustrate them:

1. Tonic facilitatory mode:

   The caboose as contrasted with the locomotive of a freight train: the locomotive is the executive command center of the train, controlling start/stop, speed, and switching tracks. The caboose contains brakes that are tonically applied to prevent "slack" between the coupling of individual cars. Braking may be tonically increased to some fixed level to slow travel downhill. The caboose contains constantly operating flashing red light to warn other trains that might follow.

   A second common metaphor is that of an electric battery whose constant current drives and facilitates executive functions by downstream target structures (see below).

2. Initiatory role of compound movements:

   Eccles first likened the operation of the cerebellum to that of a computer, in its ability to store, learn, combine, and integrate the many different motor programs so as to send the complex command to downstream target structures (computers in their own right for different operations) [20].

Current Opinion

Wikipedia, 28 July 2012: "The cerebellum (Latin for little brain) is a region of the brain that plays an important role in motor control… The cerebellum does not initiate movement (sic), but it contributes to coordination, precision, and accurate timing… It receives input from sensory systems of the spinal cord and from other parts of the brain, and integrates these inputs to fine tune motor activity… Because of this fine-tuning function (sic), damage to the cerebellum does not cause paralysis, but instead produces disorders in fine movement, equilibrium, and posture…" [14].

Old History

Marie Jean Pierre Flourens (1794–1867)

In 1825, Flourens pioneered the experimental method of making localized lesions of the brain in living rabbits and pigeons and carefully observing their effects on motricity, sensibility, and behavior. His intention was to investigate localizationism, i.e., whether different parts of the brain had different functions, as the Austrian physician Franz Joseph Gall, the founder of phrenology, was then proposing… Flourens was able to demonstrate convincingly for the first time that the main divisions of the brain were indeed responsible for largely different functions. By removing the cerebral hemispheres, for instance, all perceptions, motricity, and judgment were abolished. The removal of the cerebellum affected the animal's equilibrium and motor coordination, while the destruction of the brainstem (medulla oblongata) caused death. These experiments led Flourens to the conclusion that the cerebral hemispheres are responsible for higher cognitive functions, that the cerebellum regulates and integrates movements, and that the medulla controls vital functions, such as circulation, respiration, and general bodily stability… (redacted from Ref. [34]).

Luigi Luciani (1842–1919)

In 1891, Luigi Luciani formulated his—triad of the cerebellar symptoms

1. "Atonia," a reduction in resting muscle tone;
2. "Asthenia," a weakness of voluntary muscle contraction;
3. "Astasia," an inability to maintain over time a posture of limb or body.

These cardinal deficits, he argued, explained all the troubles provoked by cerebellar lesions. Later, he added a fourth sign, "dysmetria," an inability to accurately reach to a target in space. He asserted that the one underlying fundamental principle of the function of the cerebellum was to modulate activity in downstream structures through tonic excitatory discharge; this in turn was interpreted to provide fine tuning so as to permit these structures (including cerebral cortex) to initiate and coordinate posture and movement. Luciani's interpretation of the cerebellar symptoms survives to today: his terminology has entered both the clinical vernacular and the routine of the neurological examination: the cerebellum is viewed as an—"excitatory tonic reinforcer" [39]; the "electric battery" is the commonly used analogy among current clinical neurologists. While one can agree that Luciani rightly pointed out the role of the cerebellum in contributing to postural tone and muscular force, was he correct in denying Babinski's proposed unique cerebellar control of coordination of multijoint postures and movements? Luciani specifically rejected such a role in muscle coordination because (in his experiments) he opined that cerebellar-lesioned dogs could swim correctly with perfect coordination (sic)… Therefore, he argued that his triad was sufficient to explain all the other observed cerebellar symptoms. In his view, there were far too many "ad hoc special properties" that would be required of the "simple little hind brain" for it to coordinate posture and movement, as proposed at about the same time by Babinski. Indeed, there would have to be a special representation of single muscles within the cerebellum (none such then known, cf. [38]), and a means of linking them together, and an ability to learn and store them as separate compound assemblies through trial-and-error learning. Thus, he argued, it was not necessary to call into action these hypothetical special properties of coordination. Indeed, he regarded the notion as unscientific and dismissed it as "a fictitious entity, obscure, imperfect, and unintelligible!" Luciani's dictum based upon the scientifically respectable principle of parsimonious reasoning was taken up by many thereafter, who maintained that atonia, astasia, and asthenia were indeed sufficient to explain all the movement irregularities constituting cerebellar ataxia. It was therefore left up to Babinski to actually demonstrate in cerebellar patients the existence of a specific decomposition of the movement. He called this deficit asynergia (Redacted from Martin et al. [37, 38]).

Joseph Babinski (1857–1932)

In 1899 [5], Babinski (from Ref. [6]) first published his work on the movements of cerebellar-damaged patients. His triad of cardinal deficits included "hypermetria," "adiadochokinesis," and "asynergia [7–9]." "Hypermetria" was illustrated in the patient's finger to nose to examiner's finger pointing test, in

which the patient's finger overshot the examiner's target finger ("past pointing"). Babinski showed that in contrast to locomotor ataxia (as in syphilitic tabes dorsalis), where the limb movements were misdirected, in cerebellar hypermetria the direction of movement was preserved. He opined the overshoot was due to an exaggerated "agonist impulsion" (i.e., the first agonist muscle burst), which could not be checked by a timely and efficient "antagonist braking action" (i.e., the first antagonist muscle burst). This was only much later confirmed in the monkey by Flament et al. in 1984 [22].

"Adiadochokinesis" was described in 1902 as an impairment in performing rapid successive alternating movements such as pronation/supination of the hand. Babinski's interpretation was that the cerebellar lesion "can, without diminishing the muscle energy, provoke a kind of inertia which reflects the difficulty of mobilizing and arresting the movement in time."

As for "asynergia," described in 1899, Babinski first defined "synergy" as "an association of movements that constitute a single act" [5]. Babinski illustrated asynergia with two examples. The first was the failure of the trunk to lean forward at the onset of walking, which resulted in staggering or falling when the first step was initiated. A second example was the absence of a forward displacement of the hip and knee when the standing patient was asked to look upward by tilting the head and trunk backward (resulting in falling). Other examples of the lack of synergy included cerebellar patients being unable to sit up from a supine position: the hip flexion was not associated with knee extension (such that the legs were raised above the bed). He also showed several examples illustrating the lack of combined simultaneous flexion of the hip and knee: e.g., the supine patient, in attempting to place one heel on the opposite knee, made the tow movements seriatim: the hip flexed first, and then the knee flexed with the heel then touching the knee, thus demonstrating the absence of a harmonious synergy involving the hip and knee flexors.

In sum, the main characteristic of asynergia was the execution one-at-a-time (decomposition) of the movements of the different body segments that were normally combined in a compound movement that were instead performed seriatim instead of being combined simultaneously in the single act.

As seen in the current Wikipedia article cited above [14], the concept of cerebellar asynergia/decomposition is still disputed. Whereas hypermetria and adiadochokinesis are accepted as main cerebellar symptoms, such is not the case for asynergia.

From the start, strong objections came from Déjerine (cf. [16], see also [15]) and Andre-Thomas in France and Holmes and Walshe in England; they continue to this day. Holmes, an English neurologist, treated many soldiers with traumatic head injuries in the battlefield hospitals in France during the First World War and systematically studied the effects of acute cerebellar injuries in 40 men. In his original article the *Brain* in 1917, Holmes very definitely and succinctly reported finding Babinski's asynergia [29]. However, later, he expressed some skepticism, so that in 1939, he stated that the term asynergie in Babinski's original sense to signify a lack of co-ordination between wider groups of muscles, including those which should fix segments of a moving limb, to be "unnecessary because it would include symptoms of different origin," and would require the long list of very special functions noted above which were not then known to reside within the cerebellum [30].

Notwithstanding these views that were critical of Babinski's cerebellar asynergia, Tilney, an American neurologist, showed that muscles are functionally arranged in "synergic units" of antagonist pairs that were more frequently co-activated than they were activated reciprocally (cf. [52]). Furthermore, they suggested that the synergic activity of these units was indeed controlled by the cerebellum. At a joint meeting of the American Neurological Association and the neurology section of the Royal Society of Medicine in 1927, Tilney and Pike's suggestion was vigorously opposed by both Walshe and Holmes, Walshe objecting that cerebellum could not be involved in the control of muscle synergies because none other than Sir Charles Scott Sherrington had clearly shown that this function was already performed by the spinal cord! Holmes, also in agreement with his mentor, also felt that no such specific disturbance as asynergia of antagonist muscles existed in sufficient degree to account for cerebellar dysfunction [30]. Finally, in his review published in the *Revue Neurologique* in 1958, on cerebellar syndrome, François Lhermitte (1921–1998) [37] similarly dismissed Babinski's definition of asynergia as a specific cerebellar deficit: "If we understand by the term synergy to be that neural organization which presides over a set of several muscles that accomplish an act, this function is certainly not included in the cerebellum … however, the cerebellum is indispensable to its correct execution" [37]. As a result, asynergia is not included in Dow and Moruzzi's scholarly and authoritative 1958 work "The Physiology and Pathology of the Cerebellum" [18] nor in Fine et al.'s introduction to the clinical examination for cerebellar deficits [21].

Babinski's Asnergia Revisited

Two main lines of evolution in understanding the central control of movement paved the way for reconsidering cerebellar synergy and asynergia.

A first line was proposed by Whachholder in 1928 [53]. According to him, a goal-directed movement is the result not only of the central control of muscles but also of the interactions with the viscoelastic forces and the inertia of the musculoskeletal system and with the external forces such as the gravity forces.

A second line was the suggestion that the cerebellum must play a particular role in motor learning. Bernstein in 1967 insisted on the redundancy or the excessive degrees of freedom for the achieving a goal-directed movement and he stressed the role of motor learning in building up the most effective coordination scheme to achieve the goal [12]. Similarly, the concept of dynamic internal model of movements built up by learning was proposed by Wolpert et al. [54, 55]. These models were used to simulate the dynamic disturbances caused by movement execution (direct dynamic models) and to anticipate the appropriate corrections (inverse dynamic models). Wolpert et al. [55] suggested that these internal models might be stored in the cerebellum.

Thus, re-emerged the old hypothesis of the cerebellum as the center for coordinating movement, now newly charged with the storing of learned internal dynamic (and inverse dynamic) models, which might then be used to anticipate the disturbances associated with movement performance. These ideas gave new meaning and life to Babinski's concept of asynergia. The loss of direct and inverse dynamic commands compensating for disturbances of posture and equilibrium resulting from movements suggests a possible unifying mechanism for explaining the functional diversity of the various mutijoint synergies and their loss after cerebellar damage.

"With his typical perspicacity for clinical observation, Babinski may be credited for noticing that a large variety of functionally different mutlijoint movements associated in a single act were consistently disturbed or absent in cerebellar patients. In this sense, Babinski was really a prescient prophet for what could only be explained much later after a long empirical study of cerebellar function." (redacted from Ref. [15]).

Experiments Suggesting that the Cerebellum May Indeed have a Unique Executive Function in Combining and Initiating Compound Bodily Movements

These studies in awake performing rats, macaques, and humans were inspired by the prior cited open questions and more immediately by the theories of Brindley [13] and his student, Marr [40], and independently of Albus [1] and by the work in reduced preparations of Ito et al. [31] on motor learning.

Inactivation/Ablation of the Cerebellum May Indeed Allow Simple Movements to be Performed Almost Normally While Impairing or Eliminating Compound Movements Entirely, as Babinski had Maintained (Fig. 1)

Rhesus macaques were trained [25, 26] to make flexion/extension movements of a single joint, the wrist, with and against torque loads with the forearm constrained in a cast. The movements were made in response to an oscillographic display; the animals were trained to track the horizontally moving target across the screen. Five different movements consisted of "Jerk" (move a.s.a.p. offscreen in the direction of the suddenly moved target), "Jump" (move a.s.a.p. ON screen to the NEW location of the suddenly displaced target, "Pert" (maintain position of the wrist despite sudden reversal=-perturbation of the torque load), "Ramp" (tracking of the target as it moved slowly across the screen), and "RAM" (self-paced rapidly alternating movements). After having learned and performed these movements over long practice, injections of muscimol were on separate days injected into each of fastigial, interposed, and dentate nuclei, and the animal allowed to repeat the movements.

Surprisingly, the wrist movements continued without interruption: an occasional inconsistent delay was see in the onsets of "Jerk" and "Jump; a slight 3–5 Hz action tremor on "Ramp"; a few beats of oscillation at the end of "Pert"; and a slight slowing of "RAM." Nevertheless, the movements were all otherwise successfully performed.

By contrast, marked deficits were noted when the animals were released to climb out of the chair, the nature depending on the site of the inactivation;

> Fastigius: the animal *fell to the side of the injection and could not right itself for the duration of the inactivation*,
> Interpositus: the animal *overreached the target in reaching for a food reward*,
> Dentate: the animal could no longer retrieve a food reward from a slot in a Lucite block as previously with a thumb/forefinger precision pinch. Instead, the animal reverted to a single-digit forefinger "winkling" strategy of raking the food bit out of the slot.

In sum, these results suggested a categorical difference in cerebellar control of "simple movements" and "compound movements" [24, 25, 51]. A similar deficit in compound movements with preservation of simple movements was also seen in human patients with infarcts of the cerebellar cortex [11].

A mechanism for the Trial-and-Error Learning of Such Large Combinations Required for Compound Mopvementt

Ito and colleagues [31, 32] had shown in 1972 in anesthetized animals that electric stimulation of the inferior olive climbing fibers paired with stimulation of the mossy fiber/parallel fiber system over multiple stimulations progressively changed the response to the stimulation mossy fiber/parallel fiber system. The alteration was private to that particular mossy fiber/parallel fiber system that had been stimulated. The climbing fiber was said to have been the teacher, the mossy fiber/parallel fibre/Purkinje cell, and the learner [32].

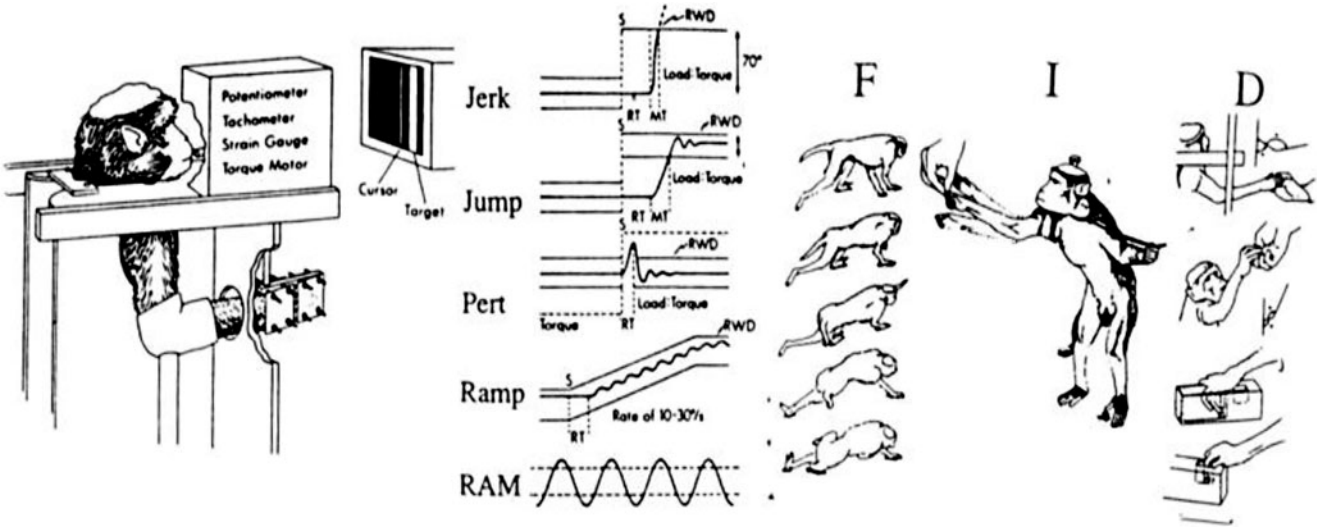

Fig. 1 Cerebellar inactivation impairs compound movements more than simple movements. Fastigial: falls to the side of the lesion; wide base. Interpositus: antagonist delay; action tremor. Dentate: reach overshoot, X–Y error, and finger incoordination (cf. Kane et al., unpublished data) [50]

Gilbert and Thach in 1977 recorded simple and complex [23] spikes from Purkinje cells [48] in macaques learning to adapt a motor response of the wrist to hold a steady position despite sudden perturbations of held position by novel torque pulses. At the first appearance of the novel torque pulse, the climbing fiber complex spike commenced firing in synchrony with the perturbation, and persisted over ensuing trials as the animal gradually adapted. The simple spike began to change frequency at the first novel trial and continued to do so until the adaptation was complete. By which time, the complex spike returned to its previous sporadic firing and the simple spike remained at its new level. These changes were consistent with the Albus/Marr/Ito ideas that the novel perturbation had caused the erroneous behavior, which in turn had caused the change in complex spike firing, the gradual change in simple spike firing, and the behavioral adaptation [23] (Figs. 2 and 3); compare also consistent experiments on eyeblink conditioning [45] and vestibular ocular reflex [44].

In human studies of Martin et al. (Fig. 4), subjects threw balls of clay at a visual target while looking through wedge prism spectacles. During the short-term adjustment, subjects threw in the direction of their prism-bent gaze, missing the target to that side. Within 10–30 throws, they gradually adapted with a wider gaze-throw angle so as to hit the target. Immediately after removal of the prisms, the widened gaze-throw angle persisted and throws missed the target to the opposite side, the so-called "negative after effect." Repeated throws were required to adapt back to the normal gaze-throw angle and hit the target. In a study of long-term adjustment, two subjects threw with the same hand (right) and the same type of throw (overhand) alternately, with and without prisms, over a period of 6 weeks. They gradually learned to hit the target on the first throw, both with and without prisms. The two gaze-throw calibrations (prism and no-prism) were retained for >27 months [41, 42]. The long-term adjustment was shown to consist of a coordinated relationship of eye-in-head, head-on-trunk and trunk-on-arm angles. Subjects with injury of the cerebellum or of the inferior olive were not able to adapt.

Coordination across body parts was then studied during adaptation to prisms. In humans [43], positions of head, shoulders, arm, and ball were video-recorded continuously. Body angles of eyes-in-head, head-on-trunk, trunk-on-arm, and arm-on-ball were then computed. In each subject, the gaze-throw adjustment during adaptation was distributed across all sets of coupled body parts. The distribution of coupling changed unpredictably from throw to throw within a single session. The angular variation among coupled body parts was typically significantly larger than angular variation of on-target hits. Thus, coupled body parts changed interdependently to account for the high accuracy of ball-on-target hits. Principal components and Monte Carlo analyses showed variability in body angles across throws with a wide range of variability/stereotypy across subjects. The data supported a model of a dynamic and generalized solution as evidenced by the distribution of the gaze-throw adjustment across body parts [43].

To further test coordination across body parts, three monkeys [46] performed a visually guided reach-touch task with and without laterally displacing prisms. The prisms offset the normally aligned gaze/reach and subsequent touch. As with humans, naïve monkeys showed adaptation, such that on repeated prism trials the gaze-reach angle widened and touches hit nearer the target. On the first subsequent no-prism trial, the monkeys exhibited an aftereffect, such that

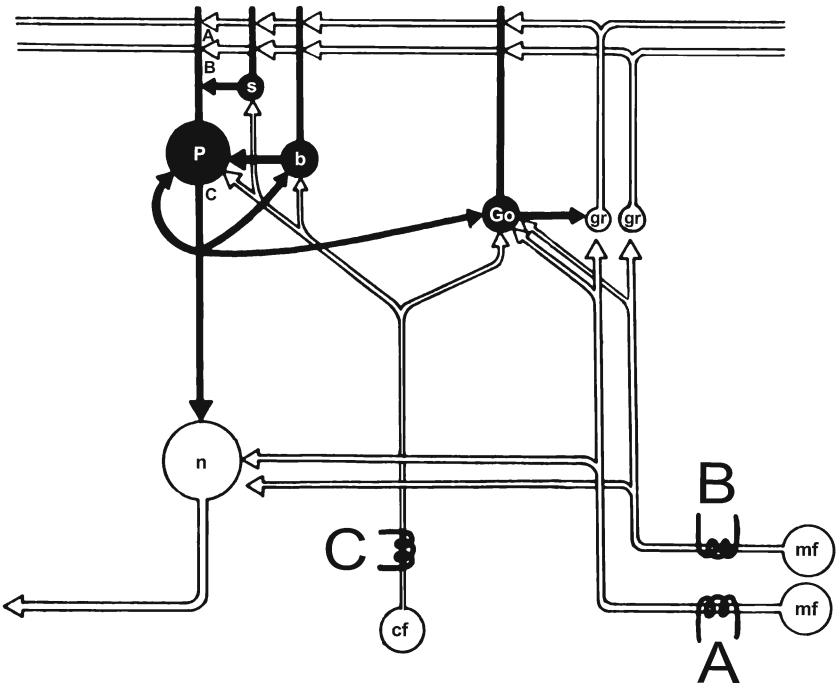

Fig. 2 Pairing climbing fiber (*CF*) shock with mossy fiber (*MF*) shock depresses parallel fiber (PF) excitation of the Purkinje cell [31]

the widened gaze-reach angle persisted and touches missed the target in the direction opposite that of initial prism-induced error. After 20–30 days of training, monkeys showed long-term learning and storage of the prism gaze-reach calibration: they switched between prism and no-prism and touched the target on the first trials without adaptation or aftereffect.

Lidocaine was then injected into the posterolateral cerebellar cortex or muscimol or lidocaine into dentate nucleus. The injections temporarily inactivated these structures. Immediately after injections into cortex or dentate, learned prism reaches were displaced in the direction of prism-displaced gaze, but the naïve no-prism reaches were relatively unimpaired. A single permanent lesion (kainic acid) in the lateral dentate nucleus of one monkey immediately impaired only the learned prism gaze-reach calibration and in subsequent persisted for the 18 days of observation, with little or no adaptation [46].

Single Muscles are Indeed Topographically Represented and Accessible as such Within the Cerebellar Deep Nuclei [2–4] (Fig. 5)

Patterns of termination of the cerebellothalamic pathway were investigated using anterograde tracing techniques. The thalamic projections from each of the deep cerebellar nuclei are topographically organized in two and possibly in three dimensions. First, the caudo-rostral cerebellar nuclear dimension is mapped onto the mediolateral dimension within the cell-sparse ventral lateral thalamic region (VPLo, VLc, VLps, and nucleus X). By correlating this topographic ordering with the previously established lamellar organization of the cell sparse thalamic region [2–4], a somatotopy was inferred within the deep cerebellar nuclei, with caudal body parts represented anterior and rostral body parts represented posteriorly in each nucleus. A second topography consists of the mapping of the mediolateral dimension of the dentate and interpositus nuclei onto the ventrodorsal dimension of the lamellae in the thalamus.

Since the thalamic connections with motor cortex predict a somatotopic organization with distal body parts ventral and axial parts dorsal in thalamus, each cerebellar nucleus should, therefore, represent axial body parts laterally and distal parts medially.

A third mapping dimension is shown for the dentatothalamic projection: dorsal parts of the dentate nucleus project posteriorly within the cell-sparse thalamic region, and ventral parts project anteriorly. The significance of this as regards representation of the body is not known. Subsidiary foci of terminations within the cell-sparse thalamic region are visible following tritiated amino acid injections into each of the deep cerebellar nuclei. Following dentate injections, these foci appear as anteroposteriorly elongated, rod-like aggregations of terminations which are similar to the rod-like aggregations of thalamocortical relay cells which have been demonstrated following focal injections of horseradish peroxidase into the motor cortex [4]. The interpositothalamic and the fastigiothalamic terminations are elongated and appear as focal clusters in all planes of section. The interpositothalamic clusters are distributed within posterodorsally curving planar sheets. An anterograde double-labeling technique, using a combination of the autoradiographic

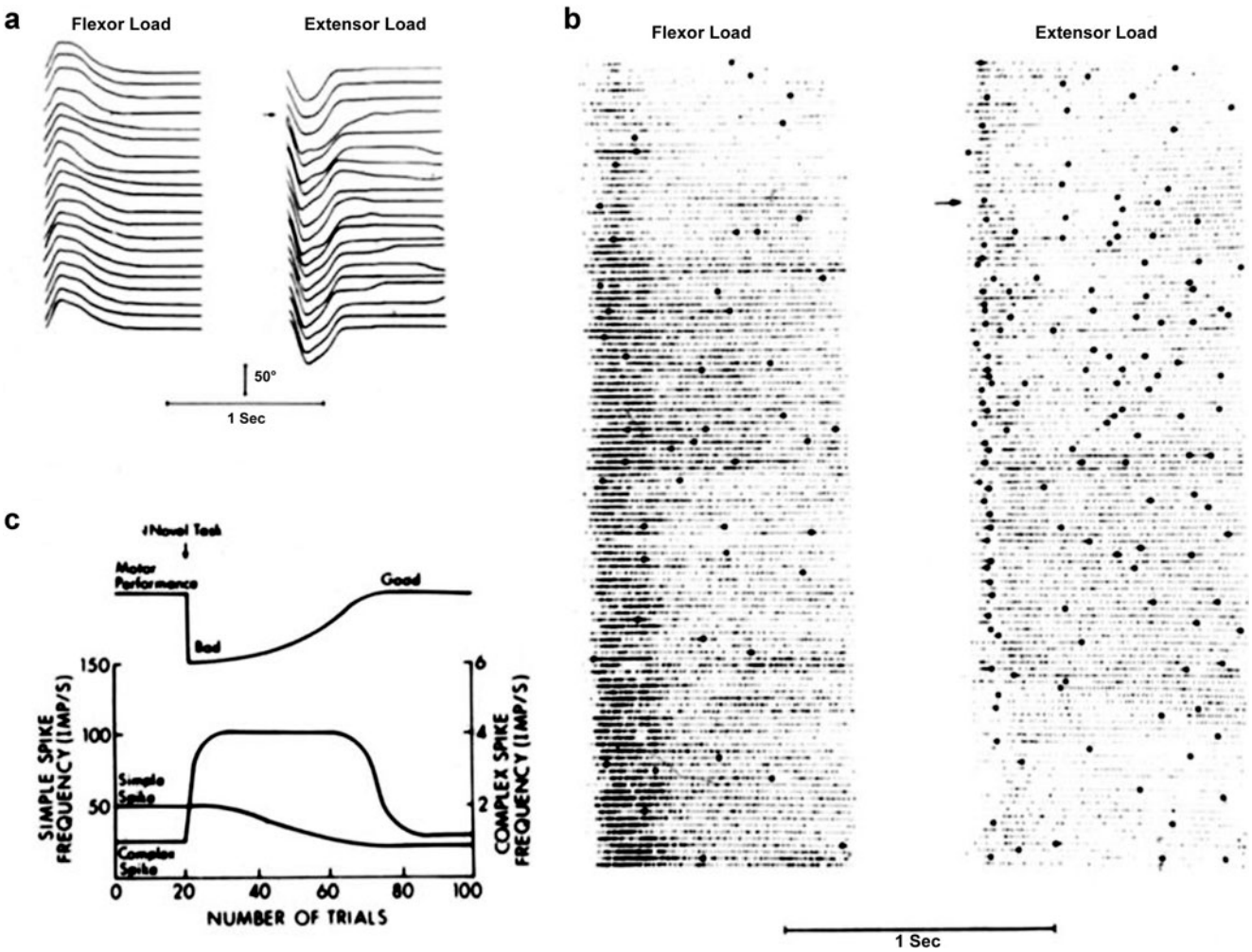

Fig. 3 When adapting to a novel perturbation, CF complex spikes increase and PF complex spikes decrease [23]

technique with the axonal degeneration technique, was used to investigate the interrelations of the terminations from different nuclei and from different parts of the same nucleus. Rods from different parts of the dentate nucleus terminate independently of one another. Dentatothalamic rods and interpositothalamic clusters, though interdigitating within the same thalamic region, do not overlap. This topographic and modular organization of the cerebellothalamic pathway suggests that the cerebellar input may reflect both the somatotopic and the columnar organization of the motor cortex. [2–4].

The Parallel Fibers Provide a Mechanism for Linking Purkinje Cells and Those Deep Nuclear Muscle Representations to Which They Project Together into Compound Many-muscled Movements

In trained reaching rats [28], simple spikes were recorded from pairs of Purkinje cells that, with respect to each other, were either aligned on a beam of shared parallel fibers or instead were located off beam. Rates of simple spike firing in both on-beam and off-beam Purkinje cell pairs commonly showed great variety in depth of modulation during reaching behavior, but with respect to timing, on-beam Purkinje cell pairs had simple spikes that were tightly time-locked to each other (either delayed or simultaneous) and to movement, despite the variability in rate. By contrast, off-beam Purkinje cell pairs had simple spikes that were not time-locked to each other, neither delayed nor simultaneous. On-beam Pcs in the paramedian lobe fired precisely synchronized SSs time-locked to behavior. Each plot represents the time-resolved cross-correlogram of Pc SS activity recorded at two different electrodes during reaching–grasping movements.

The method used to investigate and quantify whether and how on-beam synchrony was related to the reaching and grasping behavior was that of time-resolved cross-correlation analysis. The results revealed that the on-beam synchronous activity was a common and highly statistically significant phenomenon. Of 32 Pc on-beam pairs that showed movement-related SS

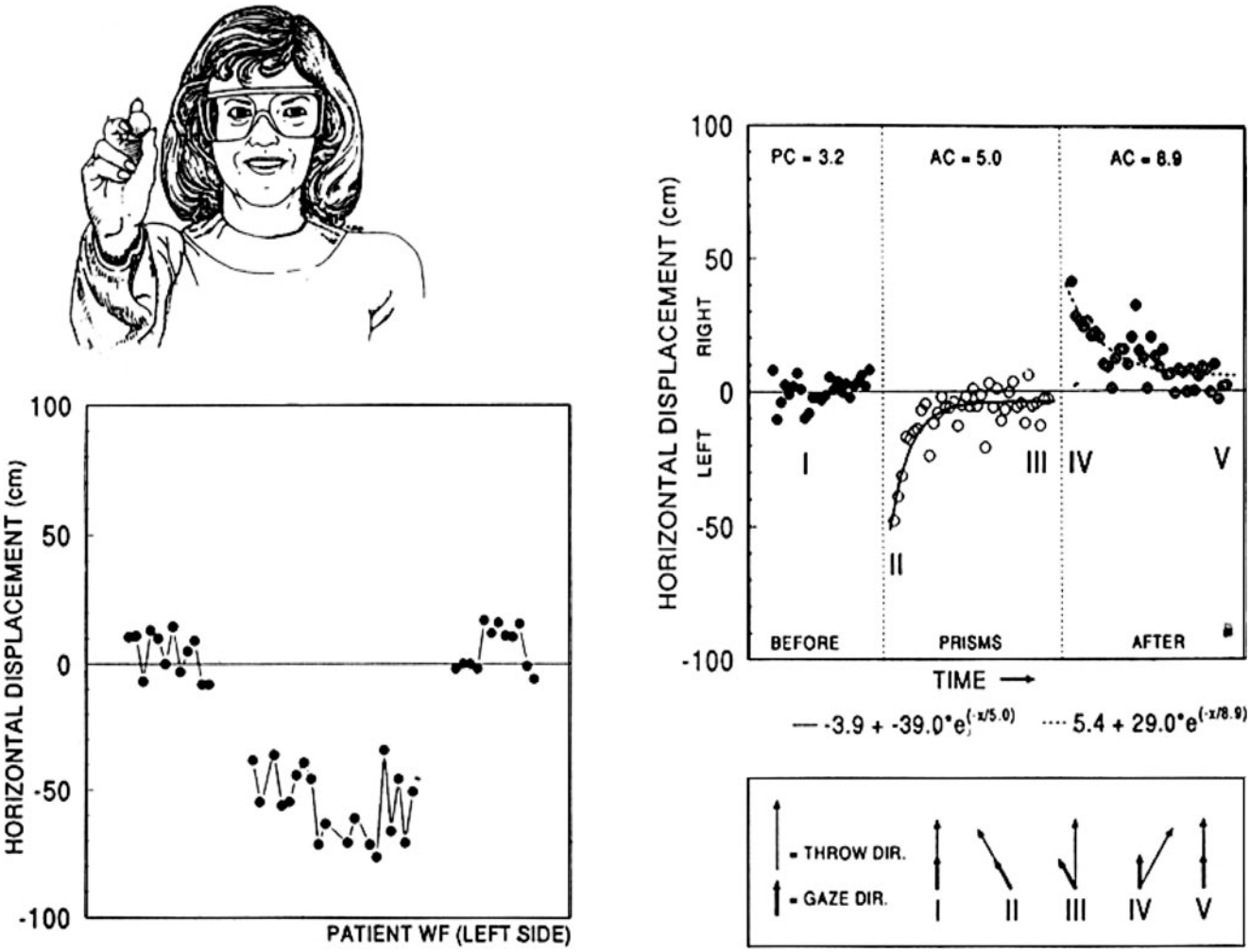

Fig. 4 Does the cerebellum contribute to motor learning? [41, 42]

changes, 31 (97 %) also showed epochs of significant synchronous activity during the movement. On-beam synchronous firing occurred preferentially during the extension phase of the reaching movement (Fig. 5). By contrast, changes in firing rates occurred during both the extension and retraction [28].

Fig. 5 PF are long enough to link together Purkinje cells projecting to different body parts within one deep nuclear map and across multiple maps on-beam PCs fire in synchrony [28]; surgical vermal split impairs tandem gait but not one-legged hopping [11]

Finally, studies were conducted in children who had undergone neurosurgical splitting of the cerebellar vermis in the midline to approach and remove lethal tumors in the fourth ventricle [11]. This procedure divided the parallel fibers spanning and linking the two sides of the cerebellum. These children could hop as successfully as did controls on one leg. Nevertheless, they were unable to coordinate the two legs in tandem heel-to-toe walking and would otherwise fall unless supported [11].

Even a Catalog (Presumably also Learned) of the Dynamic Features of the Body Parts, Including Mass, Inertia, Gravitational Weight, and Interaction Torques Required to be Compensated if Movement is to be Performed Correctly, is Stored in the Normal Cerebellum, and is Impaired or Lost After Damage (Fig. 6)

Seven human subjects with cerebellar lesions and seven normal controls were studied as they made reaching movements in the sagittal plane to a target directly in front of them

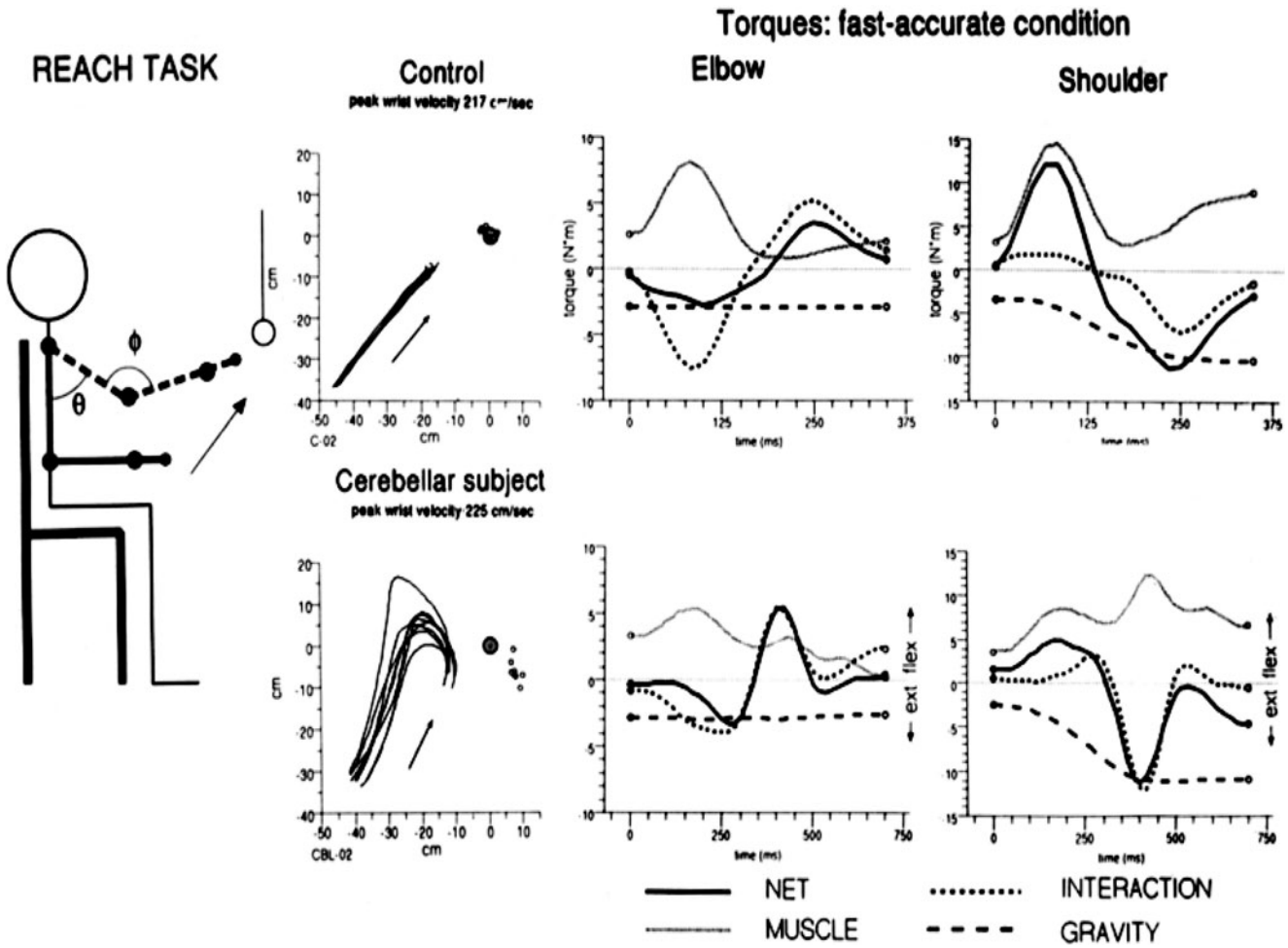

Fig. 6 Ataxic reach: inability to compensate for interaction torques in multijoint movements [10]

[10]. Reaches were made under three different conditions: (1) "slow-accurate," (2) "fast-accurate," and (3) "fast as possible." All subjects were videotaped moving in a sagittal plane with markers on the index finger, wrist, elbow, and shoulder. Marker positions were digitized and then used to calculate joint angles. For each of the shoulder, elbow and wrist joints, inverse dynamics equations based on a three-segment limb model were used to estimate the net torque (sum of components) and each of the component torques. The component torques consisted of the torque due to gravity, the dynamic interaction torques induced passively by the movement of the adjacent joint, and the torque produced by the muscles and passive tissue elements (sometimes called "residual" torque). (2) A kinematic analysis of the movement trajectory and the change in joint angles showed that the reaches of subjects with cerebellar lesions were abnormal compared with reaches of control subjects. In both the slow-accurate and fast-accurate conditions the cerebellar subjects made abnormally curved wrist paths; the curvature was greater in the slow-accurate condition. During the slow-accurate condition, cerebellar subjects showed target undershoot and tended to move one joint at a time (decomposition). During the fast-accurate reaches, the cerebellar subjects showed target overshoot. Additionally, in the fast-accurate condition, cerebellar subjects moved the joints at abnormal rates relative to one another, but the movements were less decomposed. In the fast as possible condition; cerebellar subjects moved more slowly than controls. (3) A kinetic analysis of torques generated at each joint during the slow-accurate reaches and the fast-accurate reaches revealed that subjects with cerebellar lesions produced very different torque profiles compared with control subjects. (4) The inability to produce muscle torques that predict, accommodate, and compensate for the dynamic interaction torques appears to be an important cause of the classic kinematic deficits shown by cerebellar subjects during attempted reaching. Given this, interaction torques often determined the pattern of incoordination of the elbow and shoulder that produced the curved trajectory and target overshoot for this defect, cerebellar subjects often resorted to a decomposition strategy so as to simplify the movement and not have to control both joints simultaneously [10].

This preserved ability to make simple movements has been used in rehabilitation training for humans with cerebellar injury [51].

Electrical Stimulation of the Deep Cerebellar Nuclei in Awake Baboons Causes Either and/or Both Simplex and Complex/Compound Movements

In alert baboons with chronically implanted electrodes, movements elicited by the stimulation of the cerebellar nuclei were studied. Two types of motor effects were observed: (1) Simple movements that concerned the unidirectional displacement of a limb segment. (2) Complex movements that involved distinct and frequently noncontiguous muscles were stereotyped and could not be dissociated. These movements were defined as motor synergies. Electromyographic study revealed the response latencies and the modality of cerebellar control on musculature. Simple movements were due to the activation of muscles within the involved segment in addition to the co-contraction of muscles of a nearby segment. Thus, they could have been due to a cerebellar control over muscular synergies. Complex movements would correspond to the simultaneous activation of distinct muscular groups and could also have been the outcome of a cerebellar control on motor synergies. The effects of the interposed nucleus concern preferably flexor muscles whereas the effects of the dentate nucleus appear to be equally distributed among flexor and extensor muscles. Somatotopic motor localization was evidenced both in the interposed and dentate nuclei: there are somatotopic relations between every region of the interposed nucleus and musculature. As regards the dentate nucleus, two subdivisions were distinguished according to the complexity of elicited motor effects: (a) an antero-medial region from which complex motor synergies were elicited. (b) a postero-lateral region giving rise to simple movements, mainly hand movements [47].

The Timing of Discharge in Deep Cerebellar Neurons Precedes the Discharge in Motor Cortex Neurons, Muscles, and Movement

The discharge of neurons in cerebellar dentate nucleus and cerebral motor cortex was recorded on alternate days in each of three monkeys in association with prompt arm movement in response to a light signal [49]. The time of change of the discharge of each neuron in relation to arm movement was computed. The distributions of the time of change for cerebellum and cerebrum overlapped, but the cerebellar distribution was shifted significantly earlier [49].

Discussion

Parenthetically, it has been suggested that the cerebellum may serve as a "clock" in pacing sequential elements of compound movements [33]. However, several studies of Purkinje and deep nuclear cell discharge have looked for and failed to find any periodicity in the firing patterns ([35, 36], cf. also [27]).

Moreover, experiments on eyeblink conditioning and VOR adaptation (cited above) support a cerebellar role in motor learning. It remains to be seen whether further experiments with these paradigms support the herein proposed roles in combination of body parts, representation of dynamic components, and integration of the above in a single unique cerebellar executive command signal.

Summary

1. Inactivation/ablation of the cerebellum may allow simple movements to be performed while impairing or eliminating compound movements;
2. A cerebellar mechanism for the trial and error learning of such large combinations required for compound movements;
3. The cerebellum has been shown to have a somatotopic representation of single muscles within the deep nuclei;
4. A cerebellar parallel fiber mechanism for linking Purkinje cells and deep nuclear muscle representations to which they project appear to combine these muscles together into compound many-muscled movements;
5. A catalog within the cerebellum (presumably also learned) of the interaction torques required to be compensated if movement is to be performed correctly;
6. Electrical stimulation of the deep cerebellar nuclei causes muscle contraction and movement;
7. Discharge in deep cerebellar neurons precedes discharge in motor cortex neurons, muscles, and movement.

In sum, it therefore seems to us likely that the cerebellum may not only modulate "simple" movements, but also uniquely initiates compound movements. These theories of simple modulation of all movements versus synthesis and initiation of compound movements by combination of simple motor components go back to those of Flourens, Luciani, and Babinski of over 100 years ago, and are still vigorously argued and debated.

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