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Role of the cerebellum in reaching movements in humans. I. Distributed inverse dynamics control

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Abstract

This study focuses on the role of the motor cortex, the spinal cord and the cerebellum in the dynamics stage of the control of arm movement. Currently, two classes of models have been proposed for the neural control of movements, namely the virtual trajectory control hypothesis and the acquisition of internal models of the motor apparatus hypothesis. In the present study, we expand the virtual trajectory model to whole arm reaching movements. This expanded model accurately reproduced slow movements, but faster reaching movements deviated significantly from the planned trajectories, indicating that for fast movements, this model was not sufficient. These results led us to propose a new distributed functional model consistent with behavioural, anatomical and neurophysiological data, which takes into account arm muscles, spinal cord, motor cortex and cerebellum and is consistent with the view that the central nervous system acquires a distributed inverse dynamics model of the arm. Previous studies indicated that the cerebellum compensates for the interaction forces that arise during reaching movements. We show here how the cerebellum may increase the accuracy of reaching movements by compensating for the interaction torques by learning a portion of an inverse dynamics model that refines a basic inverse model in the motor cortex and spinal cord.

Introduction

Human hand trajectories are relatively straight for point to point movement, have bell-shaped velocity profiles, and are relatively precise. To translate the spatial characteristics of the target of a movement into an appropriate pattern of muscle activation, three subprocesses are required: (i) coordinate transformation from extracorporeal space to intrinsic body coordinates, (ii) trajectory planning and (iii) motor command generation. The first two steps are concerned primarily with the desired kinematics, that is, position, velocity, and acceleration. The last step deals with dynamics, that is, forces and torques applied to specific joints. Dynamics control is critical for reaching movements because of the different constraints put on moving masses, as described by the laws of mechanics; when a multijoint limb is accelerating, movement in one joint causes motion in all other joints and this leads to inertial and velocity torques. Thus, there may be a mechanism in the central nervous system (CNS) that compensates for these interaction forces during movements. Indeed, recent studies (Topka *et al.*, 1994; Bastian *et al.*, 1996) indicate that the cerebellum is involved in compensating for the interaction forces that arise during reaching movements.

Basic muscle motor commands can be generated by feedback control, feedforward control, or both. Feedback control is limited by long delays and the dynamic properties of muscles and proprioceptors. In addition, movement time is of the same order as the delays limiting the capabilities of the feedback controller, thus, the system can be driven into an undesirable state of oscillation at high gains (see Table 1 for a summary of delay values). In contrast, feedforward control is not affected by loop delays and operates more quickly. Experimental evidence supports the notion that feedforward control is an important component of biological motor control. First, control mechanisms using only feedback cannot explain how deafferented monkeys or patients can move their arm to a target without concurrent visual and somatosensory information (Polit & Bizzi, 1979; Ghez *et al.*, 1990). Second, if an intact monkey arm is perturbed while moving toward a target, it returns to an intermediate point on the predetermined trajectory (Bizzi *et al.*, 1984). Finally, Ghez *et al.* (1990) found that patients with sensory neuropathies were impaired in the feedforward control of movement. This suggests that feedforward control in the motor control system is required for relatively fast movements, while feedback control is important for unskilled movements, interaction with the environment, and unpredictable interactions.

To generate accurate multiarticulate reaching movements, two broad classes of biologically plausible control systems have been proposed, the first based on virtual trajectory and the second on internal models of the motor apparatus. In the virtual trajectory control hypothesis, the dynamics are not computed explicitly in the CNS, but motor information describing a desired trajectory is transferred through the spinal cord. In the internal model hypothesis,

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TABLE 1. Estimated delays in afferent and efferent pathways

Anatomical pathway	Nature of information	Average time delay and species	Delays modelled	References
Spinal reflexes (afferent + efferent)	velocity	25 ms (humans)	10 m	Dufresne *et al*. (1979)
	motor commands and afferent	110–150 ms (humans)	60 ms	
Transcortical reflex loop	sensory		(30 ms eff. + 30 ms aff.)	Flanders & Cordo (1989)
Cortex–cerebellum	desired trajectory	3–6 ms (cat)	0 ms	Allen & Tsukahara (1974)
Cerebellar–cerebral tract	Motor command	1.5–2 ms (cat)	0 ms	Allen & Tsukahara (1974)
Spino-cerebellar tract	muscle state (muscle spindles)	9 ms (cat)	10 ms	Murphy *et al*. (1973)
	muscle tension, muscle state	28–35 ms (cat)		
Spino-olivary tract	(Golgi tendons, muscle spindles)		20 ms	Gellman *et al*. (1985)
Sensorimotor–olivary tract	desired trajectory??	12–25 ms (cat)	20 ms	Allen & Tsukuhara (1974)

models of the dynamics are learned and stored in adaptive neural networks and the dynamics and non-linearities of the plant (i.e. the arm, the leg, the eye. . .) are compensated for, such that the actual movement is similar to the desired movement. An advantage of the virtual trajectory model over internal models lies in its simplicity compared with the complexity of the dynamics equations necessary for an internal representation of the human arm.

The present study addresses the following questions:

- what is the nature of the motor commands required to move the hand quickly and accurately along a desired trajectory?;
- how does the CNS generate these motor commands?;
- what are the roles of the different brain regions involved in dynamics control?

The virtual trajectory control hypothesis

The virtual trajectory control hypothesis states that the dynamics are not computed explicitly in the CNS, but the planned trajectory (issued by a feedforward controller) is fed directly to the muscles in terms of desired muscle equilibrium lengths. The joint torques required to move the arm are then generated as the product of mechanical stiffness and the difference between the desired and actual muscle lengths, that is, the virtual and actual trajectories. However, Bennett *et al*. (1992) and Gomi & Kawato (1996) found that dynamic stiffness during movement was much less than was previously assumed by Flash (1987). Katayama & Kawato (1993) showed that to reproduce relatively straight hand movements, the virtual trajectory must have a complicated shape that is as difficult to plan as computing the inverse dynamics. Moreover, Koike & Kawato (1995) found that although virtual and actual trajectories for slow movements are similar, those for medium speed movements vary considerably.

McIntyre & Bizzi (1993) proposed an expanded equilibrium–point control model incorporating both position and velocity that effectively implements a position plus derivative feedback controller that increases the command-following capability of the system. The velocity feedback loop can have a relatively larger velocity gain than the position gain since the velocity feedback loop has smaller delay than the position feedback loop. The velocity loop introduces phase advance and the position loop increases muscle stiffness. Simulation for single joint movements showed that the system is stable where a pure position feedback would not be, and the model produced fast movements at stiffness levels below those required by the equilibrium point hypothesis alone. Because the response gives good results for fast speed single joint movements, McIntyre and Bizzi concluded that the theory according to which the descending motor command specifies the positions and the velocities, without the need for explicit knowledge of system dynamics, seems a viable option for the control of movements.

To expand the virtual trajectory hypothesis to reaching movements, a planar two-joint arm movement was implemented. The detailed inverse dynamics equation of the arm are:

$$\tau_s = (I_1 + I_2 + 2M_2L_1L_{g2}\cos(\theta_e) + M_2L_1^2)\ddot{\theta}_s + (I_2 + M_2L_1L_{g2}\cos(\theta_e))\ddot{\theta}_e - M_2L_1L_{g2}\sin(\theta_e)\dot{\theta}_e^2 - 2M_2L_1L_{g2}\sin(\theta_e)\dot{\theta}_e\dot{\theta}_e$$
$$\tau_e = I_2\ddot{\theta}_e + (I_2 + M_2L_1L_{g2}\cos(\theta_e))\ddot{\theta}_s + M_2L_1L_{g2}\sin(\theta_e)\dot{\theta}_s^2 \quad (1)$$

The joint torque consists of the shoulder joint torque $\tau_s$and the elbow joint torque τ_e. M_i is the mass of the *i*th link, I_i, its moment of inertia around the *i*th joint, L_i, its length, and L_{gi}, the distance to the centre of gravity of the *i*th link from the proximal end of the link. In each of the torque equations, the first term corresponds to the normal inertial term, which represents a single-joint movement, the second term corresponds to the inertial torque due to movement of another joint, the third term corresponds to the centripetal forces, and the fourth term corresponds to the Coriolis force, which exists only for the shoulder torque.

Simulation results

The expanded virtual trajectory hypothesis was tested using the two-joint arm described above to simulate the reaching movements previously described by Koike & Kawato (1995); the hand paths recorded for five different movements (T1 => T3, T2 => T5, T3 => T5, T4 => T1 and T4 => T5) with durations between 500 ms and 750 ms were usually straight, with radial paths being significantly straighter than transverse paths. The arm parameters used in the model were described by Katayama & Kawato (1993). The set of control parameters used were those that gave the best results in McIntyre & Bizzi (1993). The position gain was 2.50 with a delay of 65 ms and the velocity gain was 0.60 with a delay of 25 ms. The desired trajectory was generated by a minimum-jerk trajectory generator in extra-personal space.

When the individual movement duration was 1 s, reaching movements were accurate and followed the straight, desired trajectory as shown in Figure 1(a). When the movement duration was decreased to 500 ms, however, the reaching movements deviated significantly from straight trajectories (Fig. 1b).

The results of the expanded virtual trajectory model presented here suggest that specification of the positions and velocities by the descending motor command is a viable mechanism for the control of slow movements. However, because the controllers operating for each joint are not coupled, this control system does not generate straight trajectories for rapid movements with large interaction forces. Thus,

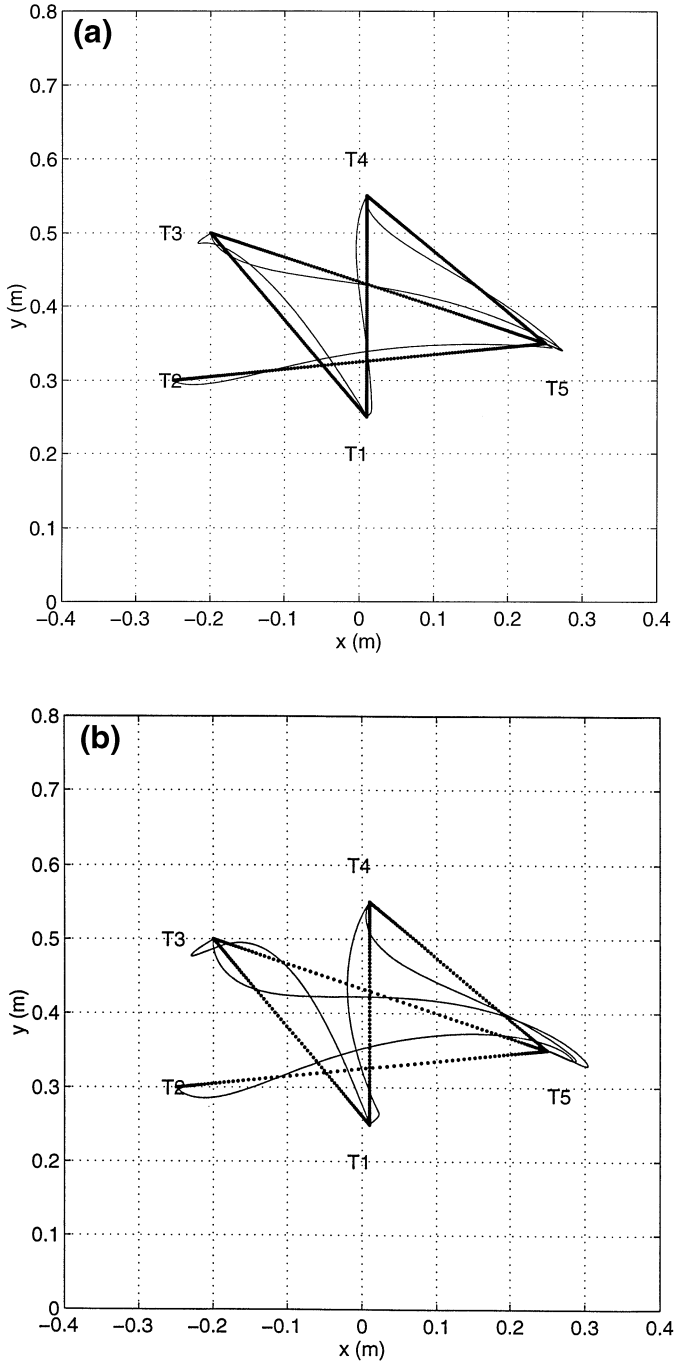

FIG. 1. Simulation of the model proposed by McIntyre & Bizzi (1993) for reaching movements when a two-joint planar arm is used for the musculoskeletal system (T1 => T3, T2 => T5, T3 => T5, T4 => T1, and T4 => T5). (a) Slow movements (duration for each movement 1 s). The origin is the shoulder position, the *x*-axis is toward the right, and the positive direction of the *y*-axis is forward away from the body. (b) Fast movements (duration for each movement: 500 ms)

for proper execution of fast movements, it appears that the CNS should possess a priori knowledge of the arm's dynamics.

The inverse dynamics control hypothesis

An inverse model is a neural representation of the transformation from the desired movement of the controlled object to motor commands serving to attain these movement goals. For instance, the inverse neural model of a two-link planar arm is a neural network that implements equation 1. Kawato *et al*. (1987) proposed the feedback error learning hypothesis as an alternative to the virtual trajectory hypothesis and Kawato & Gomi (1993) suggested that the cerebellum might be the locus of the inverse models. A feedback controller acts simultaneously to control movement and for the acquisition of an internal inverse dynamics neural model; the feedback error approximates the directions and magnitudes of the necessary modifications to the internal neural model. During learning, the feedback generated torque is slowly replaced by the feedforward torque, and movements gradually become more ballistic in nature.

If an internal inverse dynamics neural model underlies the control of reaching movements and if its acquisition relates to feedback error learning, the CNS should have the properties listed below.

1 In the virtual trajectory hypothesis, either the desired joint positions, or the desired positions and velocities, are directly sent to the muscles. Computation of the inverse dynamics also requires a desired joint acceleration component as part of its input, as shown in equation 1. As such, neurones carrying acceleration-type signals should exist.

2 The outputs of the inverse dynamics model are dynamics-related signals. As such, neurones carrying torque information should exist close to the efferent peripheral system. This definition accommodates outputs in any coordinate system, such as joint torques or muscle or 'synergy' coordinates.

3 As arm movements are controlled by both feedforward and feedback control schemes, a lesion of the loci of an inverse model should yield performance akin to that of a feedback controller with low gains and (very) large delays. This would result in (i) movement inaccuracy due to poor following of the desired response; (ii) overshoot and endpoint tremor at the end of movements due to the delays, and (iii) poor coordination of reaching movements due to the uncompensated interaction torques.

4 The inputs to an internal inverse dynamics neural model should be the desired accelerations, desired velocities and/or actual velocities, and desired positions and/or actual positions (Slotine & Li, 1991).

5 Computation of the torque for one joint necessitates the convergence of the kinematic variables of that joint and also other joints involved (see eqn 1). Therefore, the area of the brain where the computation occurs for one joint should be a point of convergence of information for other joints.

6 Acquisition of an internal inverse dynamics neural model involving feedback error learning requires a central error detector that should carry information in motor coordinates.

7 Because an internal inverse dynamics neural model cannot be entirely genetically predetermined due to its extreme complexity and to changes in arm characteristics during the life-span, synaptic plasticity (i.e. long-term depression or potentiation) should occur in the regions of the CNS mediating this process.

Biological substrate

Cortex

Kalaska *et al*. (1990) reported that cellular responses in Area 5 during arm movements were unaltered by external loads applied to the arm in different directions. Several other pieces of evidence indicate that these cells encode not hand paths but joint varying variables. These results suggest that Area 5 cells encode joint-centred kinematics parameters of reaching movement. Chapman *et al*. (1984) reported that the discharge of some Area 5 cells correlates with peak movement velocity in an elbow movement task. No other variables were tested, but cells recorded by Kalaska 'behave' like cells correlated with

position (their Fig. 6a), velocity (their Fig. 6b), and acceleration (their Fig. 4a; however, this cell seems to encode both position and acceleration). Taken together, these results suggest that Area 5 cells act as a desired trajectory generator in the CNS, providing desired kinematics of reaching movement in joint coordinates, including acceleration, as required by condition 1.*

In contrast, a large population of cortical motor cells controlling the proximal arm encode reaching movements in a reference frame that reflects movement dynamics (Thach, 1978; Evarts *et al.*, 1983; Kalaska *et al.*, 1992). The transformation from kinematics to dynamics can be achieved by two means, either by an internal inverse dynamics neural model or by a feedback controller. The motor cortex receives direct projections from the sensory cortex and responds to limb perturbations, suggesting that feedback signals are involved. A feedforward component also exists because deafferentation does not yield paralysis. The presence of projections from Area 5 to the motor cortex (Ghez, 1991) suggest that a basic inverse dynamics neural model exists in the cerebral cortex. Thus, condition 2 appears to be fulfilled by the motor cortex.

Intermediate cerebellum†

It has been proposed that the role of the cerebellum is to synthesize compound movements from simpler components (Flourens, 1824), to tune its downstream targets so that their functions are performed optimally (Holmes, 1939), or provide feedforward control (Arbib, 1981). Following cerebellar damage, simple reaching movements are observed to be relatively normal, however, a decomposition of compound movements with increased asynergy and intention tremor occurs (Goodkin *et al.*, 1993). These results suggest that the cerebellum modulates downstream movement generators and synthesizes compound movements from simpler components. In addition, classic kinematic cerebellar reaching deficits, such as poor coordination between the shoulder and the elbow, curved trajectory, and overshoot, has been shown to result from an inability to compensate for interaction torques (Topka *et al.*, 1994; Bastian *et al.*, 1996). These data strongly suggest that the cerebellum provides feedforward motor commands necessary for the proper execution of multijoint movement (condition 3).

The intermediate cerebellum receives spinal afferents, which carry information regarding the state of the arm, and reafferents, which combine afferents and copies of descending commands and projections from the primary motor, somatosensory, and posterior parietal cortex (area 5) (Brodal, 1978). It is important to note that these cortical projections are spatially related to the periphery; consequently specific groups of cerebellar neurones receive both sensory inputs and cortical inputs. During reaching movements, the firing rate of 80% arm related mossy fibres correlates with joint angle and ⊤ 33% correlates with velocity (some correlate with both; Van Kan *et al.*, 1993a). Several characteristics of the signals (especially the lead and lag times) indicate that some fibres contain information derived from the muscle spindles and others, information of central origin. Interestingly, the activity of some cells appears to be related to acceleration and had a lead time that could originate in Area 5 via the pontine nuclei (Brodal, 1978). These results are consistent with the information needed to compute the inverse dynamics; desired acceleration, velocity, and position are provided by a central planner (lead-time cells) and the actual velocity and position are provided by the muscle spindles (lag-time cells). Therefore, the cerebellum receives the necessary inputs to compute an inverse model of the arm (satisfying conditions 1 and 4).

The responses of interpositus neurones are correlated with movements of specific joints (Thach *et al.*, 1982) and responses to proprioceptive inputs are phasic in nature, and inputs from both the agonist and antagonist muscle affect the excitability of the same cell (McKay & Murphy, 1974). Also, the cells that fire during single-joint movements, fire more rapidly during a reach (Van Kan *et al.*, 1993b). Finally, many interpositus neurones respond to stimulation of the sensorimotor cortex, whereas fewer neurones respond to stimulation of Area 6 (Bloedel & Courville, 1981) and modulate ongoing activities of both the motor cortex and the magnocellular red nucleus. Thus, the interpositus forms a side path strongly activated during reaching movements which appears to transform kinematics variables into phasic motor commands in body coordinates. The results of these studies are in agreement with condition 2.

It has been proposed that arm coordination during reaching movements may depend on the cytoarchitecture of the intermediate cerebellum itself (Thach *et al.*, 1992; Garwicz & Anderson, 1992; Goodkin *et al.*, 1993). Purkinje cell dendrites are linked by parallel fibres, forming functionally coupled, task-specific subgroups that may be the basis for cerebellar coordination of movement. Such groups of Purkinje cells project to discrete areas of the cerebellar nuclei, possibly influencing synergetic muscles across several joints in the limb. The longer a parallel fibre is, the more cells in the nuclei are linked by Purkinje cell control. Mugnaini (1983) found that on the average, monkey parallel fibres are about 6 mm long. Garwicz & Anderson (1992) showed that activation of a single mossy fibre results in spread of activity along the parallel fibres as far as 1.5 mm beyond the mossy fibre termination area. In addition, the ascending branch of the granule cell axon makes numerous synapses with overlying Purkinje cells, before bifurcating into the parallel fibre portion of the axon (Pichitpornchai *et al.*, 1994). Similarly, Bower & Woolston (1983) stimulated a small group of mossy fibres and showed that the detectable response is limited to a small group of Purkinje cells somewhat overlying the stimulated area. Thus, if each granule cell contacts one Purkinje cell several times, the granule cell influence on the overlying Purkinje cell is quite strong and more focused than the information carried by the parallel fibres. This evidence suggests that each Purkinje cell receives strong localized information from a small set of underlying mossy fibres corresponding to the joint that the Purkinje cell controls and substantial information from other mossy fibres carrying information from other joints. Therefore, the Purkinje cells receive signals necessary to compute the different terms of the inverse dynamics (see eqn 1) fulfilling condition 5.

The inferior olive is often considered to be an error detector (Simpson & Alley, 1974). According to this hypothesis, the inferior olive receives an efferent copy signal and delayed feedback and acts as a comparator (Oscarsson, 1980). Any discrepancies between the two signals activates the inferior olive neurones and climbing fibres would signal a mismatch between intended movement and actual movement. Ojakangas & Ebner (1992) recorded complex spikes from Purkinje cells in the intermediate cerebellum in response to errors related to changes needed in velocity or acceleration during a motor

*As Kalaska (1991) noted, however, this distinction between the roles of Area 5 and motor cortex is not so straightforward, and neuronal mechanisms operating at several hierarchical levels may coexist simultaneously in any given cortical region. It is also apparent that these regions are not sequentially activated, but are active in parallel. Moreover, Kalaska noted that these results do not exclude the possibility that the time varying kinematics signals observed in Area 5 could be generated in another brain area projecting to Area 5.

†The following discussion is restricted to the intermediate part of the cerebellar hemispheres (if not otherwise mentioned) because it is known to be concerned with control of the evolving movement (Ghez, 1991) and to receive information regarding the state of the arm, in contrast to the lateral part of the cerebellar hemispheres, which may be involved in the planning of the movement.

TABLE 2. Cell groups and putative modalities

Cell group or area	Coordinate system	Modality	References
Visual system	Extra-personal	Target	
Area 5	Joint	Desired trajectory	Kalaska *et al*. (1990)
Sensory areas	Joint	Position and velocity	
Motor cortex output	Synergy motor commands	Torque-like	Thach (1978) Evarts (1983) Kalaska *et al*. (1992)
Nuclear cells (cerebellar output)	Joint	Torque-like	Van Kan *et al*. (1993b)
Inferior olive input	Joint (but also one limb, two, etc.)	Error in velocity; acceleration?? position??	Ojakangas & Ebner (1992)
Mossy fibers (cortical inputs)	Joint	Desired trajectory (acceleration)	Van Kan *et al*. (1993a)
Mossy fibers (sensory inputs)	Muscles	Muscle length and velocity	Van Kan *et al*. (1993b)
C3–C4	Synergy motor commands	Torque-like	
Motor neurons	Muscles	Muscle motor command	
Spindles	Muscles	Muscle length and velocity	
Golgi	Muscles	Muscle tension	

learning task involving visually guided arm movements. These authors suggested that the observed complex spike activity is related to an error between the desired and actual velocity. These results suggest that the inferior olive detects torque-like signal errors in performance, thus partially fulfilling condition 6.

Finally, the cerebellar cortex is commonly considered to be an array of perceptrons (Marr, 1969; Albus, 1971) consisting of a Purkinje cell, with the inferior olive providing the error signal necessary for learning. According to this hypothesis, the granule cell input to a Purkinje cell provides 'context' for current sensorimotor actions that is tuneable by experience. Ito *et al*. (1982) report that the pairing of parallel fibre and climbing fibre activity at Purkinje cells results in long-term depression at the parallel fibre synapse. Ekerot & Kano (1985) further showed this long-term depression to be associative and input specific. Under this hypothesis, the final condition 7 is fulfilled.

Functional model

Based on the data reviewed above (summarized in Tables 1 and 2), we now propose a functional model of motor control that incorporates a modified version of feedback error learning and is consistent with feedforward control, reflex functions, low stiffness values, and the existence of a distributed internal inverse dynamics neural model.

The motor cortex inverse dynamics model

The information about joint position and velocity is used to modify the inverse dynamics models arising in both the motor cortex and the cerebellum. The motor cortex, from a desired acceleration vector provided by a trajectory generator in joint coordinates presumably located in Area 5, produces a basic feedforward torque vector that accounts for the anisotropy in limb inertia, but not for the inertial interaction, centripetal, or Coriolis forces.* An approximate, linearized inertia matrix dependent on the arm configuration may be acquired by the motor cortex. The following is derived from equation 1:

$$\begin{pmatrix} \tau^{f}_{s\_mc} \\ \tau^{f}_{e\_mc} \end{pmatrix} = \begin{pmatrix} \alpha + \beta\theta_e & 0 \\ 0 & \lambda \end{pmatrix} \begin{pmatrix} \ddot{\theta}_s \\ \ddot{\theta}_e \end{pmatrix}, \quad (2)$$

where α, β and λ are positive constants, and the super-script *f* stands for feedforward. The inertia matrix depends on the elbow angle, which was provided by either a forward model or directly by proprioception or vision. The matrix is given zero terms for the off-diagonal components, because these terms correspond to inertial interaction torques.

The total torque vector computed by the motor cortex is the sum of the feedforward and feedback terms. In the model, the total delays of the transcortical loop are set to 60 ms: 30 ms for the afferent sensory information and 30 ms for the efferent motor commands. As the muscle spindles do not carry a significant amount of acceleration information, acceleration is not present in the feedback controller. The total torque vector computed by the motor cortex is:

$$\tau_{mc} = \tau^{f}_{mc} + \tau^{f}_{mc}, \quad (3)$$

where the superscripts *f* and *b* stand for feedforward and feedback, respectively. The left-hand side of Figure 2 illustrates the detail of the cortical control system.

The spinal cord inverse model

The following procedure addresses the question of how torque-like signals generated in joint coordinates in the motor cortex and the cerebellum are transformed into muscle commands. A two-link arm with six muscles, four single-joint muscles and two double-joint muscles is implemented (Katayama *et al*. 1993). The tension vector *T* is computed by:

$$\boldsymbol{T(l,i,u) = K(u)\{l_r(u) - l\} - B(u)i}, \quad (4)$$

where *l* is the muscle length vector and is the contraction velocity vector. $K(u)$, $B(u)$ and $l_r(u)$ are the muscle stiffness, muscle viscosity, and rest length of the muscle, respectively, and depend linearly on the activation of the motor neurones, *u*. The muscles produce a torque vector:

$$\boldsymbol{\tau(\theta,\dot{\theta},u) = A(\theta)^T T(l,\dot{l},u)}, \quad (5)$$

where $A(\theta)$ is the moment arm matrix that depends on the joint angles and *T*, as before, is the tension vector for the six muscles. By assuming

*A functional account rather than the actual neuronal activity of the motor cortex is modelled. It is also assumed that the motor cortex produces torque signals, however, the torque representation adopted in the model is partly for convenience. Thus, in the following discussion, the term 'torque' may be replaced by 'synergy motor command.' Moreover, for simplification, it is assumed that the motor cortex operates in joint coordinates (although the activity of some cells appears to correlate with muscle activity or direction of hand movement.) Note that for planar single-joint reaching movements, acceleration and torques are proportional; so an 'inverse model' for single-joint movements is quite straightforward (if there is no viscosity).

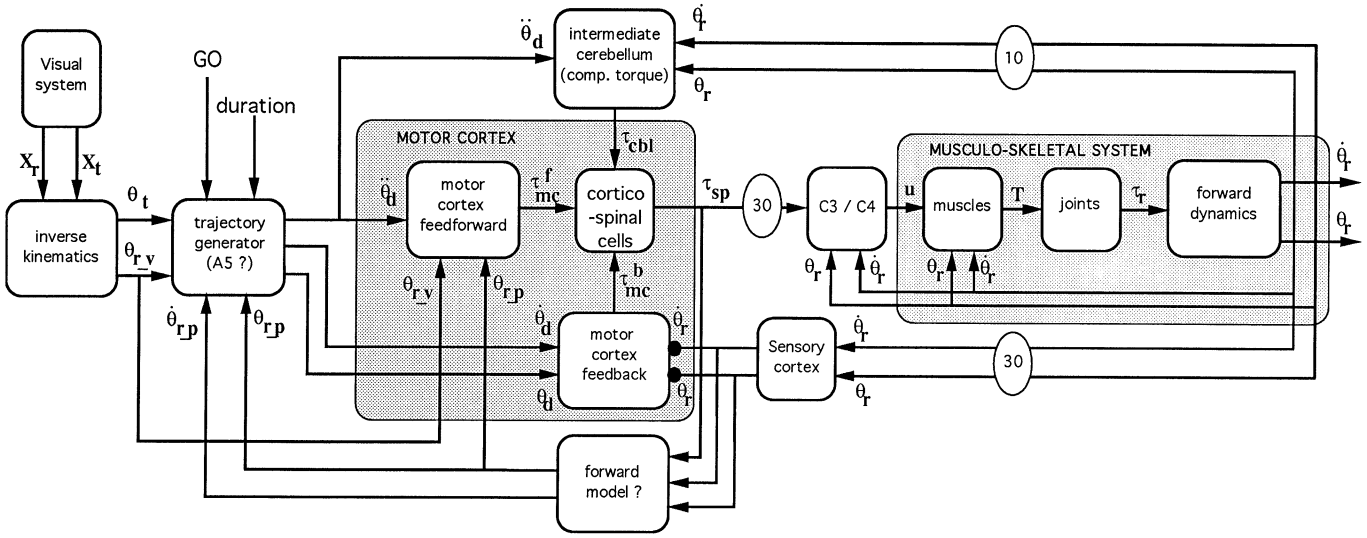

FIG. 2. Left-hand side: the cortical control system. The visual system provides the target location and initial hand posture to the CNS. An inverse kinematics model provides the trajectory generator with the target position in joint coordinates, as well as the hand position. The hand position can also be provided by the sensory system. Hand position is then sent to the trajectory generator (presumably in A5) which also receives the joint state as well as duration and GO signals. The desired acceleration is sent to a group of cells in the motor cortex. This cell group, when provided with the hand position (either from vision or proprioception), computes a basic feedforward motor command τ^f_{mc}. The trajectory generator also computes a feedforward desired velocity and position vector, which is sent to another group of cells in the motor cortex. These cells receive, via sensory cortex, joint position and velocity, and can compute a feedback torque τ^b_{mc}. The two torques (feedforward and feedback) are then summed in a group of cortico-spinal cells. Right hand side: The cerebellar/peripheral control system. C3/C4 takes the delayed signal τ_{sp} received from cortico-spinal cells and computes the motor command vector u, which is sent to the muscles, where it creates a tension vector T. Top, the cerebellum compensates for errors, providing the cortico-spinal cells with a compensatory torque τ_{cbl}. To compute the torque, the cerebellum receives the desired kinematics variables and the delayed arm state $\dot{\theta}_r$ and θ_r. Bottom: in addition, a forward model could be added that provides the cerebral cortex with the predicted current state, in contrast to the delayed state.

constant moment arms that do not depend on joint angles, $A(\theta) = A$, the muscle length vector is given by:

$$l = l_m - A\theta, \tag{6}$$

where l_m is the muscle length when the joint angle is zero, and A is the constant moment arm matrix.

The arm is redundant at the dynamics level because joint torque is generated by agonist and antagonist muscles, and, during either posture maintenance or a movement execution, there is an infinite number of combinations of muscle tensions, which correspond to varying stiffness, that produce the desired movement. Assuming constant stiffness and viscosity, equation 4 can be inverted giving the motor command vector as a function of the tension vector. By calculating the Moore-Penrose pseudo-inverse matrix $A^{T\#}$ of A^T, muscle tension can be uniquely determined from joint torques in a way that minimizes muscle tensions (Katayama & Kawato, 1993)

$$T = A(\theta)^{T\#}\,\tau. \tag{7}$$

Thus, the descending motor command vector is given in muscle coordinates by:

$$u = \frac{A^{T\#}\tau - k'\{l_m - l_o - A\theta) + b'\,A\dot{\theta}}{k'r}\,. \tag{8}$$

This transformation, presumably located in a C3/C4 network (Alstermark *et al*., 1981), depends on the actual muscle lengths and velocity, and therefore realises an integration of motor commands arising from the motor cortex (Kuypers, 1981)* and proprioception. The right hand-side of Figure 2 illustrates that C3/C4 is assumed to implement the transformation corresponding to equation 8 from the 'torque command signal' τ_{sp} to u. The muscles then convert u to tensions T, and joint dynamics in turn convert this into the torques τ_r, which yield the actual angular trajectory. Note that if this transformation is perfectly accurate (i.e. if the spinal cord implements a perfect inverse model of the muscles), $\tau_r = \tau_{sp}$.

*For simplicity, and because humans do not have a distinct rubrospinal tract (Kennedy, 1990), the red nucleus is not discussed or included in the model. Again for simplicity, the role of the premotor cortex is not discussed.

The cerebellar inverse dynamics model

At this stage of the model, τ_{sp} is determined by the motor cortex by the sum of its feedforward and feedback commands. As reviewed above, the cerebellum could learn to provide a third contribution, τ_{cbl} to τ_{sp} so that the total comes as close as possible to the 'true' torque given by equation 1. After learning, the cerebellum would provide a difference signal between the ideal torques and the torques generated by the basic system to the motor cortex, via the thalamus. The cerebellum would therefore learn the torque error due to the imperfect inertia matrix represented in the motor cortex, the velocity torques, and the error in torques due to the spinal cord controller. This does not suggest that the cerebellum knows the origin of these errors, but rather produces an approximate correction through a learning process. The top of Figure 2 shows that the cerebellum computes the compensatory torques and then adds its output to the feedback and the feedforward torques. In the companion paper (Schweighofer *et al*., 1998) we carefully describe and show how a realistic cerebellar neural network can acquire the part of the inverse model necessary to cancel these errors.

Simulation results

Because at this stage the model does not possess a cerebellum, the simulation results should be akin to behavioural deficits as found in

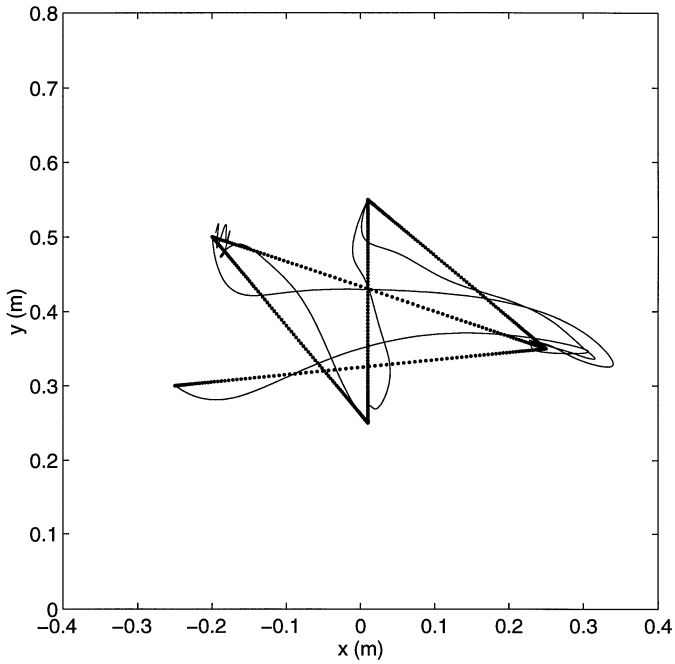

FIG. 3. Reaching movements (T1 => T3, T2 => T5, T3 => T5, T4 => T1, and T4 => T5, duration for each movement: 1.2 s) the trajectories generated by the model (solid curve) deviates significantly from the desired movement (dotted curve) and is significantly curved, even for a relatively small movement. Note the pronounced overshoot for movements to the right and the end point tremor for the T1 => T3 movement.

cerebellar patients. In simulation experiments, the following parameters were used for the motor cortex model: $\alpha = 0.4$, $\beta = 0.06$ and $\lambda = 0.01$. These values are close (but not equal) to the desired values given by equation 2, and therefore produced a basic inertia matrix. The position feedback gain was $K_p = 4$, the velocity feedback gain was $K_v = 1$, and the total loop delay was 60 ms (30 ms for the efferent delay and 30 ms for the afferent delay, for both the position and the velocity). The arm and muscle parameters were described previously by Katayama & Kawato (1993). The desired trajectory was generated by a minimum-jerk trajectory generator in extrapersonal space and the time step was 5 ms.

We performed the same reaching experiments as those shown in Figure 1 with a duration of 1.2 s (Fig. 3). As Holmes (1939) showed for cerebellar patients, asymmetrial and cerebellar tremor at slow speed can be observed. Note that the overshoot of T5 is quite significant. Moreover, oscillations are very pronounced in the movement T1 => T3. The end-point tremor was an oscillation of ⊤ 3 Hz, similar to cerebellar tremor, which has a frequency of ⊤ 3–5 Hz (Vilis & Hore, 1980). Finally, all the trajectories (except for T1 => T3) exhibits deviation from both sides the straight trajectories. In our simulations, we tried to make faster movements: the performance of the movements were greatly degraded compared to Figure 3. However, even with much slowing, the movements are still greatly impaired. Note the great similarities between the trajectories shown in Figure 1(b) and on Figure 3. The only notable differences were the T4 => T1 trajectories and the end-point tremor in T3. The similarities occurred in spite of different movement durations (0.5 vs. 1.2 s) and different delays (65 ms and 25 ms for the position and velocity feedback loops, respectively, in the virtual trajectory model, and 60 ms and 60 ms, respectively, in the present model).

To show that the basic feedforward controller located in the motor cortex increases movement accuracy, elbow movements (amplitude and 20° duration 1 s) for different values of the elbow feedforward gain (λ) were performed. For single-joint movement simulations, the system operated in a purely feedback mode when the gain was zero. Due to the large delays and the low gains, the error was large (mean square error = 13 cm^2). When the gain was increased, the error first decreased to a minimum (mean square error = 5.5 cm^2 for λ = 0.075) and then increased again when the feedforward controller was excessively active (mean square error = 11.0 cm^2 for $\lambda = 0.2$).

Discussion

Increasing evidence and models support the theory that an inverse dynamics model in the cerebellum compensates for imperfections in the inverse models in both the motor cortex and the spinal cord. Whereas the motor cortex provides each joint with both a feedback and a feedforward motor command, the cerebellum transforms information about the position, velocity, and acceleration of the controlled object to the necessary torques at the joint, so that the error between the resulting trajectory and the planned trajectory is minimal. Furthermore, because the cerebellum has a large number of neurones, is highly plastic, has significant access to proprioceptive information and feedforward motor commands, and has access to appropriate motor error from the climbing fibres, a neural equivalent of the inverse dynamics equations may arise in the cerebellum. Moreover, the granule cell input distribution to the Purkinje cells is both local and distributed, allowing the computation of the coupled terms of the inverse dynamics equation.

The results of the expanded virtual trajectory model presented here suggest that specification of the positions and velocities by the descending motor command is a viable mechanism for the control of slow movements; however, for rapid movements with large interaction forces, this control system does not generate straight trajectories, in contrast to those observed in human movements, because the controllers for each joint are not coupled, the controller cannot compensate for the interaction torques. The results of the expanded virtual trajectory model and the basic inverse model (i.e. without cerebellum) were similar because the models are both primarily position and velocity feedback controllers assisting the correction of basic feedforward controllers. In both simulations, the deviations from the straight trajectories primarily arose from the actions of the feedback controllers. When the velocity feedback delay was much shorter, movements were executed more accurately at faster speeds and the end-point tremor was not so pronounced.

The present model is based on feedback error learning but is different in that the inverse dynamics model is distributed: the motor cortex provides a basic command appropriate for slow single-joint movements and the cerebellum provides the correction necessary for the execution of multijoint movements. Schweighofer *et al.* (1996) showed that a similar principle applies to the saccade generator. A basic motor command generated in the brainstem is refined by the cerebellum so that the actual movements match the desired change in eye position, in spite of the orbital non-linearities. The role of the cerebellum in reaching movements is even more crucial in that, in addition to correcting for the muscle non-linearities and the motor cortical/spinal controller inaccuracy, it also allows coordination by compensating for the interaction torques. The companion paper (Schweighofer *et al.*, 1998) shows that a detailed, realistic model of the cerebellum embedded in the present functional model can learn to compensate for the imperfections of the basic inverse neural models. This strongly supports the hypothesis that inverse dynamics can be computed and learned in the brain.

The present study focuses on evidence for a distributed inverse model of the dynamics of the limb. However, as Miall *et al*. (1993) point out, the motor system could also benefit from a forward model of the plant. Ghez *et al*. (1990) suggest that proprioceptive information updates a model of the limb, which would provide the motor cortex with positional information. The results of these previous studies suggest that a forward model of the limb exists in the CNS and provides both Area 5 (for trajectory generation) and the motor cortex (for dynamic compensation) with the current state of the arm (see Fig. 2, bottom).

Predictions and proposed experiments

Computer models represent one possible solution to a given problem. In order to provide generalized significance, computer models have to be experimentally validated. Consequently, in the following, we provide testable predictions that arise from the analysis and the simulation results of the present study.

1 The trajectories of reaching movements generated by patients with cerebellar dysfunction would be similar to those shown in Figure 3, if each movement duration was $\top$ 1.2 s. Note that rightward movements have a tendency to largely overshoot the target, while the upward movement (T1 =$>$ T3) shows end point tremor perpendicular to the direction of the trajectory. These results indicate that to perform reasonably well, patients with cerebellar dysfunction must use slower movements, so that the ratio of the feedback delays to the movement time becomes smaller. After cerebellar injury, patients must rely heavily on the feedback controller because their inverse dynamics internal model is no longer accurate.

2 One of the primary predictions of the distributed inverse model hypothesis is that parallel fibres provide Purkinje cells with the kinematics of associated joints not directly controlled by those cells, whereas ascending granule cells provide the kinematics of the directly controlled joint. This raises the possibility that if the parallel fibres between two cerebellar 'controllers' (i.e. the group of cerebellar cells controlling the elbow and that controlling the shoulder) were lesioned, then only a basic approximation of the interaction torques could be computed.

3 The complex temporal pattern of the Purkinje cell firing frequency that occurs during ocular following responses elicited by movements of a large visual scene can be reconstructed by an inverse dynamics representation using the position, velocity, and acceleration of eye movements (Shidara *et al.*, 1993). These results support the hypothesis that the cerebellum may represent a primary site for inverse dynamics control of eye movements. We predict that Purkinje cell and nuclear cell firing rates recorded during arm movements could be reconstructed by an inverse dynamics representation. The nuclear cells coefficient should correspond to those of the inverse dynamics equation (except for the diagonal terms of the inertia matrix).

4 The model is consistent with the map found in the motor cortex, each small cortical region controlling a single joint. Our results indicate that the performance of the motor cortical feedforward controller was quite sensitive to the feedforward gains, and thus, synaptic plasticity in the motor cortex may allow the acquisition of these gains. Moreover, a complex transformation probably occurs in the spinal cord to transform synergy motor commands into individual motor commands. Thus, there may be learning mechanisms in the spinal cord; descending modulatory pathways may modulate the spinal network to affect this transformation.

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Abbreviations

CNS central nervous system

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