

Cerebro-cerebellar Implementation of Gainscheduled Feedback Control

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I. Introduction

1.1 *The cerebellum as a motor controller*

Clarifying the way in which the central nervous system (CNS) controls motor behavior is an area of ongoing investigation in biology and medicine. Many of the neurophysiological details of how even the simplest voluntary movements are organized and executed remain to be established. Part of the motivation is basic scientific interest in achieving a better understanding of the brain, spinal cord and neuromuscular system. Another part relates to the neurological interest in understanding disorders of movement and, hopefully, eventually means of their management through neuro-compatible prosthetic and orthotic devices. At the same time, there is growing interest in possibly using neurophysiological principles in robot control. The fluid, stable movement of many animals in open, highly diverse environments is generally not achievable by current robots. This is especially with respect to stability and performance robustness to uncertainty and/or variability in body dynamics, and in terms of behavioral flexibility and adaptivity. The motor performance of animals is all the more impressive when it is considered that control computation is performed by neurons that are in general very much slower, noisier, and much less precise than electronic circuits and microprocessors. Nature has somehow learned to leverage architectural hierarchy and massive parallelism to achieve control that is in important ways unrivaled by artificial

devices. It is thus possible that control engineering may both inform and be informed by neurobiology.

Although no consensus has been achieved about many neurophysiological motor control mechanisms, a wealth of experimental data has been accumulated, and agreement is building on many points. Most investigators view the cerebrum as the primary driver of voluntary movements. The brainstem and spinal cord are concerned with reflex and simple patterned behaviors such as breathing or sneezing. The cerebellum, which is strongly interconnected with the cerebrum, brainstem and spinal cord, which is a structure long felt to be important for motor "coordination", has become viewed as some type of adaptive controller (Barlow 2002; Massaquoi and Topka 2002) in the engineering sense. The principal connections of the anterior lobe of the cerebellum, which is the principal motor control portion, are shown in Figure 1. These are involved in an extremely large number of feedback loops traversing pathways that are among the most rapidly conducting in the nervous system.

Characteristic motor control deficits associated with damage to the cerebellum include *ataxia*, tremors and impaired motor learning (Massaquoi and Hallett 2002). Ataxia comprises sluggishness (reduced acceleration and jerk), sloppiness with frequent target overshoot, increased variability in the temporal evolution of multi-joint and multi-stage movements (impaired "coordination") and degraded balance (inability to stabilize otherwise unstable postures). Tremors (oscillations related to

reduced stability of otherwise stable postures) take the form of underdamped oscillations that gradually decay near the movement target, or, when severe, grow in amplitude reflecting frank instability. A number of neurophysiological studies have demonstrated that the cerebellum is normally the site of rapid and potent changes in neural synaptic connection strength (Ito 1984). Correspondingly, experiments in humans (Thach, Goodkin et al. 1992a; Topka, Massaquoi et al. 1998; Smith and Shadmehr 2004) have verified that cerebellar damage impairs the ability to improve motor performance over time. Importantly, cerebellar dysfunction is not associated with frank paralysis. Thus, the motor cerebellum appears to modulate actions that can be driven at least crudely by parallel channels elsewhere.

These gross neuroanatomical and functional findings, coupled with the observation that the neural micro-circuitry of the cerebellar cortex in all vertebrates is highly regular (Eccles, Ito et al. 1967), almost crystalline, has for years spurred considerable theorizing about its precise role and mechanism of action. This effort has spawned a number of computational models. Most recent proposals suggest that a major function of the motor cerebellum is the appropriate adaptive compensation for body dynamics to enable effective execution of the commands specified by the cerebrum.

1.2 Stabilized feedback-dependent control

A current debate in motor control concerns the degree to which the cerebellar circuitry implements internal models of body dynamics to facilitate its operation as a compensator. The considerable value of such internal models in engineering systems raises the question of whether neural analogues may exist within or closely associated with cerebellum (Kawato and Gomi 1992; Paulin 1993b; Wolpert, Miall et al. 1998). On the other hand, other considerations suggest that the presence of internal dynamics models – namely neural circuits whose input-output relationship mimics in fair detail that of body components – is unlikely.

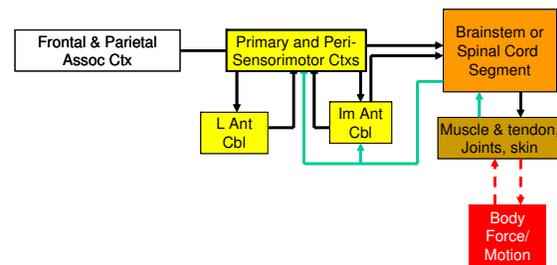


Figure 1. Basic motor control system architecture showing “long loop” neural feedback pathways between cerebral cortical areas (Ctxs), lateral and intermediate anterior cerebellum, brainstem, spinal cord and neuromuscular plant.

One concern is that for most animals there are frequent, large and sudden changes in dynamics due to changing environmental contact. Moreover, the dynamics are often nonlinear and may be complex. There are also different operating modes ranging from those that emphasize low-accuracy, low-power and energy conservation, to those needed for high-accuracy, high performance tasks. Finally, motor learning seems to specifically concern certain movement locations and directions, rather than more global solutions. To many investigators, but certainly not all, it seems less plausible that an animal would be designed to learn and rapidly invoke innumerable internal dynamics models, or a single extremely complex global dynamics model, to represent and manage these many operating conditions. Alternatively, context appropriate modulation of body impedance and, more generally, of feedback-dependent servo-like control, would appear to be a practical and much simpler means of managing both movement and posture under contact and non-contact conditions.

Strongly feedback-dependent motor control has been championed for many years as “lambda control” (Feldman 1986) “equilibrium point” and equilibrium trajectory control (Bizzi, Accornero et al. 1984; Flash 1987), servo control (McIntyre and Bizzi 1993), and “impedance control” (Hogan 1985). Included from this perspective are both the instantaneous feedback represented by physical stiffness, viscosity and mass that

can be controlled by adjusting muscular coactivation and body positioning, as well as the slightly delayed reflexive muscle activation mediated by neural feedback circuits. The general feedback-dependent view has been attractive chiefly because of its inherent flexibility, robustness and potential for obviating internal modeling of dynamics.

The frequent criticism that non-trivial delays in neural feedback transmission preclude loop gains large enough to be of practical value in feedback control has been addressed by specific formulations. For example, models that posit internal (forward) dynamics models for state estimation (e.g. (Paulin 1993b; Wolpert, Miall et al. 1998)) are in principle capable of state prediction and thereby delay compensation. Moreover, the “wave-variable” control model suggests how stable delayed feedback control could be without internal dynamics models (Massaquoi and Slotine 1996b). More recent “RIPID” (Recurrent Integrator PID) models (Figures 5 and 6) propose that certain cerebellar pathways mediate classical model-free phase-lead compensation that stabilizes long feedback loops (Massaquoi 1999). Such loops could contribute strongly to all types of movement control and postural regulation.

A second concern about strongly feedback-dependent control is that some simulations, e.g. (Flash 1987), have depended upon muscle impedance values that are larger than those measured *in vivo*. Gomi and Kawato measured elbow stiffnesses at less than 10 N-m/rad during point-to-point reaching movements (Gomi and Kawato 1996) while Flash used values on the order of 30 N-m/rad or higher. Importantly, however, the speeds of the movements studied by these investigators were significantly different. In any case, the RIPID model can produce well damped, high speed movements without requiring unduly high plant stiffness. This is particularly the case for the gainscheduled version explored below.

The RIPID model argues that cerebellar control might be implemented by an extremely large number of linear Proportional-Derivative and Integral signal processing modules (Massaquoi 1999; Jo and

Massaquoi 2004). These might be recruited to perform flexible PID control of body parts, as well as other signal processing functions. However, almost all studies of servo-type models have applied them to dynamically simple plants (McIntyre and Bizzi 1993; Miall, Weir et al. 1993; Massaquoi and Slotine 1996b). It is not clear *a priori* that complex multi-joint control can be achieved using only modulation of muscular impedance and simple neural feedback signals.

1.3 Control gain scheduling

A popular engineering approach to managing more complex and variable dynamics is to change, or *schedule*, the gains in feedback-dependent controllers as a function of some variable that changes across the operating region of the plant’s state space. Typically, plant dynamics are linearized at various nominal operating points and an appropriate controller is computed at each such point using linear quadratic regulator (LQR) or other control synthesis methods. Next one determines a scheme by which the gains should be transitioned as the system moves between the nominal operating points. If the plant state can be sensed quickly enough, the appropriate control gains can be chosen from a look-up table. This is a simple and often effective procedure. However, there are considerations regarding implementation. Schedules that are coarse in state-space suffer comparative limitation in control performance, while fine grained schedules call for an extremely large look up table to cover the operating region. One alternative is to use simple interpolation to derive intermediate gain values in a sparse look up table. Another proposal is to fit offline a possibly complex continuous gain function (Jarrah and Al-Jarrah 1999). Such continuous functions can be evaluated with arbitrarily great resolution and obviate a lookup table. Still, they require computation that may or may not be practical in a given circumstance. Although there remain issues regarding the suitability of various gain scheduling schemes, in practice, they often work well in applications. Because of the

simplicity, flexibility and power of the gainscheduling approach, it is of interest to try to determine whether the motor cerebellum may take advantage of some similar type of processing.

2. Methods: Scheduled RIPID Models

2.1 The motor cerebellum as an adaptable, locally linear PID controller

The RIPID model asserts that the basic functional modules of the motor portions of the cerebellum are linear and implement derivative, proportional or integral processing of its input. The proposals are shown in Figure 2. Briefly, the cerebellar cortical neurocircuitry is organized as a large lattice consisting principally of long transverse parallel fibers (PF, PFsig) that contact thin vertical (superficial to deep) fan-like Purkinje cells (PC). Virtually all output from the cerebellum leaves through the deep cerebellar nuclear cells (DCN, Dn, Ip) which receive inhibition from the PC. The two types of cerebellar input fibers are Mossy Fibers (MF) and Climbing Fibers (CF). Many MF enter and excite both the PFsig and the DCN. MF convey an extremely large variety of signals from throughout the nervous system, including importantly state information of the body as measured by neural sensors in muscles, joints and skin. Because the PC project to the DCN, two principal input-output signal processing paths are formed: Net excitatory direct (MF \rightarrow DCN), and net inhibitory indirect (MF \rightarrow PFsig \rightarrow PC \rightarrow DCN). CF project chiefly to the PC and are usually considered to relay signals that indicate the occurrence of behavioral error. Much evidence indicates that when CF and PF excite the same PC, the connection between the PF and PC changes strength. Thus, in response to the detection of behavioral error elsewhere in the nervous system, the weighting between the direct and indirect transcerebellar pathways can be adapted. The details and properties of this process remain under considerable investigation.

A simple analysis shown in Figure 2a indicates that whenever the indirect pathway suffers nontrivial delay or phase lag relative to the direct pathway, here through a dentate nuclear cell (Dn), then the output is some mixture of proportional and derivative scaling of the input. Further, it is known that the interpositus DCN (Ip), participates in a so called “reverberatory” (self-exciting) circuit involving neurons in the brainstem (magnocellular red nucleus (RNmc); lateral reticular nucleus (LRN)) (Allen and Tsukahara 1974). This circuit has been noted to have slowly decaying responses to pulses of excitation, consistent with the behavior of a “leaky” integrator. If RNmc response has decay constant α , then augmentation of circuit gain, as could be accomplished by a module

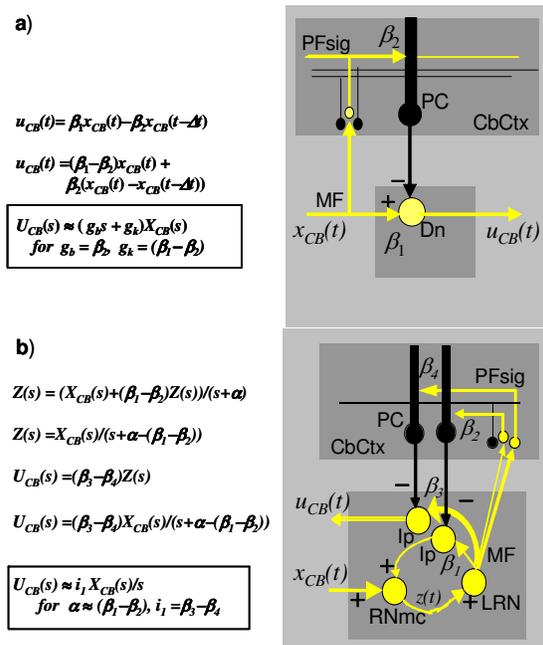


Figure 2. Proposed cerebellar cortical (CbCtx) and nuclear (Dn, Ip) participation in (a) approximate differentiation and proportional scaling, and (b) approximate integration. PC are shown on edge, β_i represent synaptic connection strengths. Adapted from (Jo and Massaquoi 2004)

such as in Figure 2a could increase the decay time and afford better integration (Figure 2b). To be sure, perfect integration is not needed for low frequency control. Leaky integration has been identified in the control of eye movements with a cerebellum-dependent time

constant on the order of 20 seconds (Leigh and Zee 1991). Thus, it is arguable that PID-like signal processing can be implemented by known cerebellar and brainstem neurocircuits.

2.2 Gainscheduling in the cerebellum?

The uniform lattice of PF and PC is punctuated by numerous basket cells (BC). These are excited by PF and inhibit PC located a small distance from the PF. As a result, it appears that depending upon the activity pattern in PF, certain PC are activated while many others are inhibited. Figure 3 proposes how this feature can be used to implement state-dependent, or, more generally, context-sensitive selection of active PC. More specifically, gainscheduled RIPID (GS-RIPID) models propose that there are at least two functional classes of PF: “Signal PF” (PFsig) are shown in Figure 3 arising from MF entering at left. These convey important state information to be scaled, differentiated and/or integrated to effect dynamic control. In addition, we consider that “Selector PF” (PFsel), shown arising from other MF entering at right, may convey a much wider variety of context information such as indicators of environmental contact, command “intent”, etc. Neuroanatomical support for two classes of PF includes the fact that while many MF send branches to the DCN, many others, arguably those destined for PFsel, do not. In Figure 3, the cross-connections with strength γ from PFsel to adjacent PC represent the lateral inhibition mediated by BC. Thus, depending upon which PFsel is active, $x_{cb}(t)$ will be processed through to $u_{cb}(t)$ via different pathways. Thus, it can be argued that PID modules may be scheduled according to context presumably including plant state measurements.

Based on analyses of spinal signals ascending to the cerebellum (Osborn and Poppele 1992), we may easily conjecture that PFsel fibers could transmit affine combinations of state variable signals. If so, the mechanism of Figure 3 could be used to define hyperplanes that separate the state space into regions in which different gainsets are dominant (Figure 4). A slightly modified

version of this architecture is described in greater detail in (Takahashi 2007).

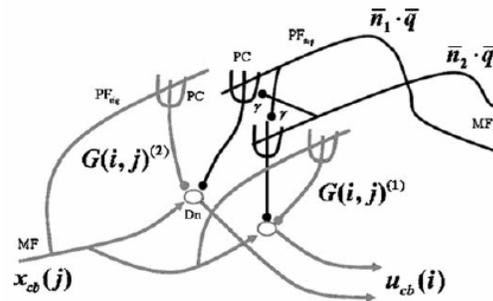


Figure 3. One proposed implementation of gainscheduling by cerebellar cortex. Gray: input-output paths including PFsig. Black: output selecting path including PFsel (here, PFsup). From (Jo and Massaquoi 2004).

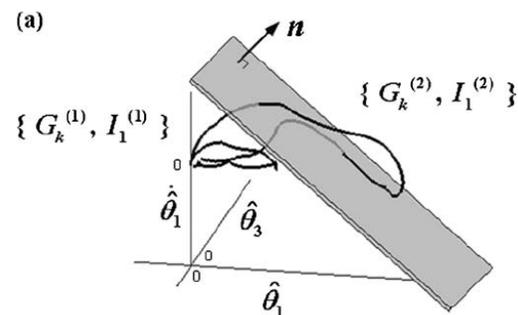


Figure 4. Typical low and high speed state balance control trajectories and a scheduling plane separating $\theta_1 \times \theta_3 \times d\theta/dt$ into two regions, each associated with a different matrix gainset. Carets in figure indicate sensed (measured and delayed) variables. From (Jo and Massaquoi 2004).

2.3 Pitch and catch switching control

Before examining applications to multi-joint control, it is useful to examine a possible use of gainscheduling in single-joint movements. Figure 5 shows a basic GS-RIPID model used to simulate point-to-point elbow movement control. The system is driven by a slightly smoothed position step input θ_{ref} (shown in Figure 7, below). The plant is a linear representation of human elbow neuromusculo-skeletal dynamics $P(s)$. This consists of a linear damped mass-spring system preceded by the low-pass filtering associated with the dynamics of neural excitation-to-force conversion (EC).

muscular coactivation (*CA*), excitation-contraction low pass filtering (*EC*) and neural one-way delays T_{spr} of 40, 35, 30 ms from cortex to ankle, knee and hip, respectively.

The free parameters of the cerebrocerebellar control model adjusted manually to refine performance were:

Cerebellum: $G^{(i)} = \{G_k^{(i)}, I_1^{(i)}\}$ for $i = 1, 2$

$G_k^{(i)}$: 3x3 proportional gain matrices

$I_1^{(i)}$: 3x3 integrator gain matrices

I_2 : 3x3 diagonal integrator gain matrix

Cerebrum: all 3x3 diagonal gain matrices

I_a : position error integrator

I_r : torque error integrator

CA: muscle coactivation control

MC: intra motor cortical direct path

F_2 : cortical sensory input

Once determined, these matrices were held fixed for all disturbances. Empirically, $G_b^{(i)}$ was not needed. Scheduling was performed by a planar boundary as shown in Figure 4. Gains were transitioned using a function similar to $S(\bullet)$ in section 2.3. Physiological delays were included in the transmission of the scheduling signals. Parameter values were found by trial and error.

3. Results

3.1 Robust point-to-point movement control

Figure 7 shows the simulated performance in a 1 radian movement and disturbance rejection of a single-d.o.f. GS-RIPID model of the human elbow using two gainsets scheduled by ε . Effective scalar gainsets and scheduling threshold found empirically were: $G^{(1)} = \{0, 190, 1630\}$, $G^{(2)} = \{9, 400, 1900\}$ and $e_0 = 0.11$. This schedule affords an almost ideally damped, fairly rapid elbow movement with a physiologically realistic bell-shaped velocity profile in response to a command that is essentially a position step. Here, the plant has appropriately low effective angular stiffness and viscosity. These are estimated by fitting the plant's responses to two 0.1 sec, rectangular, 1 N-m impulsive torque

disturbances to second-order damped mass-spring systems having the same moment of inertia as the elbow. The arrival gainset $G^{(2)}$ is made active during the first disturbance applied at $t=2.1$, and the launch gainset $G^{(1)}$ is active during the second disturbance applied at $t=4.1$. The RIPID model disturbance responses are

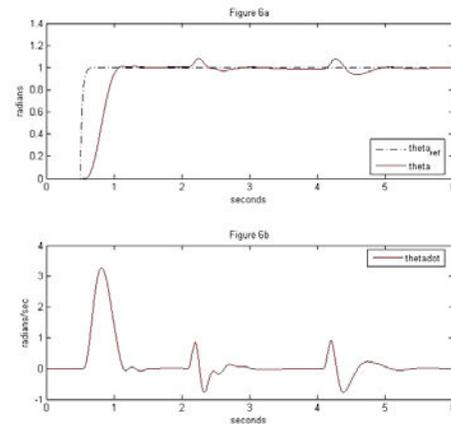


Figure 7. Simulated elbow motion (top) angular position (solid), reference command (dashed), (bottom) angular velocity.

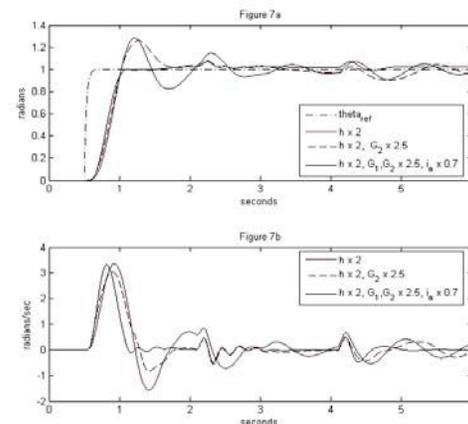


Figure 8. Effect of doubling inertia and adjusting cerebrocerebellar control gains progressively.

not precisely second order. However, the fit using second order systems with, respectively, $k = 11$ N-m/rad and $b = 0.75$ N-m/rad/sec and $k = 11$ N-m/rad and $b = 0.5$ N-m/rad/sec is fairly good (not shown). This indicates that the cerebrocerebellar system can more than double the effective (angular) stiffness of the native plant, and can increase effective viscosity by at least 50% near the target. Thus, this combination maintains the damping ratio

near 0.7, while reducing nominal settling time by a third. The step response is nonetheless almost critically damped owing to the switching control. This performance is achieved without muscle co-activation.

Figure 8 shows the effect of doubling the inertia of the plant to $h = 0.2 \text{ kg}\cdot\text{m}^2$. This causes some oscillation, but does not render the system frankly unstable (solid red line). Repeating the movement with the cerebellar arrival gainset $G^{(2)}$ scaled by 2.5 stabilizes the limb at the target, but leaves significant initial overshoot (dashed black line). Further scaling of the launch gainset $G^{(1)}$ by 2.5 and reduction of the forward integrator gain i_a by 25% restores a smooth, rapid performance (solid black line). Thus, control is regained by simple, gross gain changes without the learning of a new dynamic model. Moreover, the management of overshoot and oscillation is substantially independent.

As expected, weakening the cerebellar gains diffusely yields very sluggish movement. Attempting to speed command response by increasing the direct gain mc results in ataxic overshoot and decaying oscillations, and eventually in persistent tremor (not shown).

3.2 Robust balance control

Figures 4 and 9 show the simulated response of the GS-RIPID cerebrocerebellar balance control model (Figure 6) to backward platform disturbances of various speeds. Figure 4 indicates that (only) trajectories of high-speed disturbance responses (larger closed path) pass through upper operating region and thus invoke the second control gainset. Figure 9 shows the projections of body configuration trajectories onto the $\theta_1 \times \theta_3$ plane. All projections remain within the region bounded by dotted lines that corresponds to body configurations that maintain the center of mass above base of support. It is noteworthy that a very simple plane-separated schedule of two matrix gains, together with a somewhat arbitrary smooth interpolation function at the boundary is sufficient to produce successful balancing for a wide range of platform disturbance velocities. The action of each nominal control

gainset $G^{(i)}$ corresponds to a basic mode of balance control. Human balancing on a translating platform typically involves little motion at the knees. Rather, ankle movement, with or without the hips, maintains the center of mass above the base of support. For slow disturbances, ankle control with minor in-phase hip motion is sufficient. However, for fast translations, the ankle and hip must bend rapidly out of phase with each other. Thus, the control response varies significantly as a function of disturbance speed. The former coordination pattern has been termed the “ankle strategy” while the latter has been termed the “mixed ankle-hip strategy” (Horak and Nashner 1986). These “strategies” have been recognized as instinctive postural reactions that humans develop and/or hone through everyday living. The GS-RIPID simulation shows that this “instinct” could be implemented very simply by cerebrocerebellar circuits.

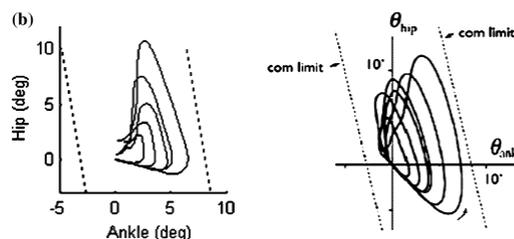


Figure 9. Ankle-hip trajectories for platform disturbances of varying speeds and region of balance compatible body configurations. Left: simulation. Right: human data adapted from (Park 2004) From (Jo and Massaquoi 2004).

4. Summary and discussion

The pitch and catch switching and multi-joint balance control examples presented here demonstrate some of the potential value of simply scheduled feedback-dependent linear control as might be implemented by the interaction between the cerebrum, cerebellum and spinomuscular system. The GS-RIPID models attempt to reconcile neuroanatomical and neurophysiological observations with a recognized engineering approach to nonlinear control. The fundamental compatibility of

gainscheduling with the CNS architecture relates to the use of multiple simple, general purpose modules to effect control. The CNS apparently makes available an extremely large number of such modules and lookup is not time consuming because module access is inherently parallel rather than serial. The robustness of performance derives from the use of strongly feedback-dependent control modules, instead of designs seeking direct cancellation of plant dynamics. Modules can therefore be linear, of low dynamic order, potentially capable of quick adaptation, and the resulting performance is free of sensitivity to errors in internal models.

The GS-RIPID models provide a possible functional interpretation of cerebellar neuronal architecture. Whether this view will be confirmed through neurophysiological studies remains to be seen. However, there are already further data that are consistent with this model. Takahashi (Takahashi 2007) has developed a considerably more detailed model of the neuroanatomical connections between cerebral cortex and cerebellum and has related these to the GS-RIPID model. From this, a Recurrent Integrator Cerebellar Single Spike (RICSS) model (Takahashi 2007) was derived to attempt to describe firing patterns in Purkinje cells of behaving monkeys. These data (Roitman, Pasalar et al. 2005) confirm that Purkinje cells are differentially sensitive to limb motion velocity and limb configuration consistent with the GS-RIPID model. The RICSS model is able to account for the single spike activity patterns of many of the Purkinje cells as a function of limb kinematics.

Further indirect support for the GS-RIPID models has been obtained through use of the Linear Parameter Varying (LPV) method to design a set of gainscheduled linear controllers for modeling human reaching containing sudden direction changes (Takahashi 2007; Takahashi and Massaquoi 2007). Such trajectories tend to be quite sensitive to the effects of the variation in limb inertia with changing limb configuration. The LPV technique afforded gainscheduled linear controllers that closely reproduced a number of recorded human horizontal planar

trajectories. Not surprisingly, the combination of scheduled gainsets outperformed any single, fixed-gain feedback control scheme. LPV may become a practical analytical method of characterizing human motor control.

A cerebellar role in switching control in point-to-point has been considered for years in terms of coordinating “move and hold” (Brooks 1985). Switching is known to be operative as pulse-step eye control and it is consistent with recent investigation of arm reaching (Scheidt and Ghez 2007). As shown here, when temporal switching is implemented implicitly via gainscheduling as a function of regulation or tracking error, eye and limb control can be also unified conceptually and mechanistically with balance control. In each situation, customizable postural settling automatically follows independently customizable movement trajectory formation.

The movement simulations shown in this report represent, to be sure, very preliminary explorations. Further simulations of more demanding control must be attempted before concluding that the GS-RIPID models are sufficiently powerful and flexible to account for the wide range of cerebellar control. One potentially promising recent investigation (Jo and Massaquoi 2006) demonstrates that a RIPID-based control architecture can control simple bipedal walking. By obviating internal models of body and of contact dynamics, the RIPID architecture may provide a simpler approach to robotic control. This example however, highlights the fact that none of the current RIPID models is self-adapting. Adaptation is clearly a major feature of the nervous system in general and of the cerebero-cerebellar system in particular. Therefore, relating cerebro-cerebellar function to *adaptive* gainscheduled control remains an essential direction for future study.

5. References

- Allen, G. I. and N. Tsukahara (1974). "Cerebrocerebellar communication systems." *Physiol Rev* **54**: 957-1006.
- Barlow, J. S. (2002). The Cerebellum and Adaptive Control, Cambridge University Press.

- Bizzi, E., N. Accornero, et al. (1984). "Posture control and trajectory formation during arm movement." *J Neurosci* **4**(11): 2738-2744.
- Brooks, V. B. (1985). How are "move" and "hold" programs matched? Cerebellar Functions. J. R. Bloedel, J. Dichgans and W. Precht. Berlin, Springer-Verlag: 1-21.
- Eccles, J. C., M. Ito, et al. (1967). The cerebellum as a neuronal machine. New York, Springer-Verlag.
- Feldman, A. G. (1986). "Once more on the equilibrium-point hypothesis (lambda model) for motor control." *J Mot Behav* **18**(1): 17-54.
- Flash, T. (1987). "The control of hand equilibrium trajectories in multi-joint arm movements." *Biol Cybern* **57**: 257-274.
- Gomi, H. and M. Kawato (1996). "Equilibrium-point control hypothesis examined by measured arm stiffness during multijoint movement." *Science* **272**(5): 117-120.
- Hogan, N. (1985). "The mechanics of multi-joint posture and movement control." *Biol Cybern* **52**: 315-331.
- Horak, F. and L. M. Nashner (1986). "Central programming of postural movements: adaptation to altered support-surface configurations." *J. Neurophysiol.* **55**(6): 1369-81.
- Ito, M. (1984). The Cerebellum and Neural Control. New York, Raven Press.
- Jarrah, M. A. and O. M. Al-Jarrah (1999). Position Control of a Robot Manipulator Using Continuous Gain Scheduling. IEEE International Conference on Robotics and Automation, Detroit, MI.
- Jo, S. and S. G. Massaquoi (2004). "A model of cerebellum stabilized and scheduled hybrid long-loop control of human balance." *Biol. Cybern.* **91**(3): 188-202.
- Jo, S. and S. G. Massaquoi (2006). "A model of cerebrocerebello-spinomuscular interaction in the sagittal control of human walking." *Biol Cybern* **96**(3): 279-307.
- Kawato, M. and H. Gomi (1992). "A computational model of four regions of the cerebellum based on feedback-error learning." *Biol Cybern* **68**: 95-103.
- Kuo, A. (1995). "An optimal control model for analyzing human postural balance." *IEEE Trans Biomed Eng* **42**(1): 87-101.
- Leigh, J. R. and D. S. Zee (1991). The Neurology of Eye Movements. Philadelphia, F.A. Davis.
- Massaquoi, S. G. (1999). Modelling the function of the cerebellum in scheduled linear servo control of simple horizontal planar arm movements. Electrical Engineering and Computer Science. Cambridge, MA, Massachusetts Institute of Technology: 240.
- Massaquoi, S. G. and M. Hallett (2002). Ataxia and other cerebellar syndromes. Parkinson's Disease and Movement Disorders. J. Jankovic and E. Tolosa. Baltimore, Williams and Wilkins: 523-686.
- Massaquoi, S. G. and J.-J. E. Slotine (1996b). "The intermediate cerebellum may function as a wave-variable processor." *Neuroscience Letters* **215**: 60-64.
- Massaquoi, S. G. and H. Topka (2002). Models of Cerebellar Function. The Cerebellum and its Disorders. M. Pandolfo and M. Manto. Cambridge, U.K., Cambridge University Press: 69-94.
- McIntyre, J. and E. Bizzi (1993). "Servo hypotheses for the biological control of movement." *J Mot Behav* **25**(3): 193-202.
- Miall, R. C., D. J. Weir, et al. (1993). "Is the cerebellum a Smith predictor?" *J Mot Behav* **25**: 203-216.
- Osborn, C. E. and R. E. Poppele (1992). "Parallel distributed network characteristics of the DSCT." *J Neurophysiol* **68**(4): 1100-1112.
- Park, S. e. a. (2004). "Postural feedback responses scale with biomechanical constraints in human standing." *Exp Brain Res.* **154**: 417-427.
- Paulin, M. G. (1993b). "A model for the role of the cerebellum in motor control." *Hum Movement Sci* **12**: 5-16.
- Roitman, A. V., S. Pasalar, et al. (2005). "Position, direction of movement, and speed tuning of cerebellar purkinje cells during circular manual tracking in the monkey." *J. Neurosci* **25**(40): 9244-9257.
- Scheidt, R. A. and C. Ghez (2007) "Separate adaptive mechanisms for controlling trajectory and final position in reaching." *J Neurophysiol* **online** DOI: 10.1152/jn.00121.2007v1
- Smith, M. A. and R. Shadmehr (2004). "Intact ability to learn intact models of arm dynamics in Huntington's Disease, but not Cerebellar Degeneration." *J Neurophysiol* **93**: 2809-2821.
- Takahashi, K. (2007). Modeling cerebrocerebellar control in horizontal planar arm movements of humans and the monkey. Aeronautics and Astronautics. Cambridge, MA, MIT. **Ph.D.:** 236.
- Takahashi, K. and S. G. Massaquoi (2007). Neuroengineering Model of the Human Limb -- Gainscheduled feedback control approach. 46th IEEE Conference on Decision and Control, New Orleans, LA.
- Thach, W. T., H. P. Goodkin, et al. (1992a). "The cerebellum and the adaptive coordination of movement." *Annual Review of Neuroscience* **15**: 403-42.
- Topka, H., S. G. Massaquoi, et al. (1998). "Motor skill learning in patients with cerebellar degeneration." *J Neurol Sci* **158**: 164-172.
- Wolpert, D. M., R. C. Miall, et al. (1998). "Internal models in the cerebellum." *Trend Cog Sci* **2**(9): 338-347.