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A Model of Cerebellar Adaptation of Grip Forces During Lifting

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Abstract - We investigated adaptive neural control of precision grip forces during object lifting. A model is presented that adjusts reactive and anticipatory grip forces to a level just above that needed to stabilize lifted objects in the hand. The model obeys principles of cerebellar structure and function by using slip sensations as error signals to adapt phasic motor commands to tonic force generators associated with output synergies controlling grip aperture. The learned phasic commands are weight- and texture-dependent. Simulations of the new circuit model reproduce key aspects of experimental observations of force application. After a data review, this paper presents simulations of a new mathematical model of the neural circuit that enables actors to learn to generate appropriate grip forces to prevent object slippage during lifting. Such learning involves a transition from reactive to primarily anticipatory application of grip forces that reflect the weight and texture of the object. Also addressed are the problems of reactive load force generation and temporal coordination between load and grip force generation.

I. INTRODUCTION

Grasping, lifting, and replacing an object require timed application of grip forces (to stabilize the object in the hand during object transport) and load forces (to elevate/lower the arm-object system to desired heights in the gravity field). An episode of lifting and lowering an object from and to a table top involves [1,2,3]: prelifting, using the fingers to apply force perpendicular to the object's surface at the points of contact of the fingers with the object; lifting, which involves continuing increase of grip force and simultaneous application of load forces sufficient to vertically displace the arm/object system, and to halt its motion at the desired height; holding by maintaining grip and load forces; controlled lowering, by reducing load forces below the value needed to counteract gravity; and release, by rapid simultaneous decrease of grip and load forces following object contact with the table.

After a data review, this paper presents simulations of a new mathematical model of the neural circuit that enables actors to learn to generate appropriate grip forces to prevent object slippage during lifting. Such learning involves a transition from reactive to primarily anticipatory application of grip forces that reflect the weight and texture of the object. Also addressed are the problems of reactive load force generation and temporal coordination between load and grip force generation.

II. DATA ON PRECISION GRIP

This section outlines roles of motor cortex and cerebellum in precision grip control, and trends in the relative timing of the exertion of load force versus grip force and in the dependence of grip force on object weight and texture.

A. Motor cortex and cerebellum in precision grip

Cell recordings and functional imaging of activity in primary motor cortex (MI) have established close links between MI activity and precision grip force [e.g., 4, 5]. Passingham [6] reviewed experiments in which a complete lesion of MI and somatosensory cortex impaired monkeys' ability to pick up food that could only be accessed with precision grip. Whereas pre-lesion monkeys used precision grip, post-lesion monkeys tried to retrieve the food using whole-hand prehension. Reversible inactivation of MI by injection of a GABA agonist produced a similar deficit [7]. Such results exemplify the principle [6] that MI enables selective activation of one or a few effectors, e.g., single joints or fingers, when many effectors could contribute.

Inactivation of the dentate nucleus of the cerebellum, which projects to MI via the thalamus, severely impairs precision grip. After GABA agonist injection into dentate, monkeys used only one finger to retrieve food from a hole, instead of the thumb-index strategy used before [cf. 8]. This effect probably depends partly on disrupted dentate input to MI. Inactivation of the dentate leads to a loss of anticipatory phasic components of MI cell discharges [9]. Loss of anticipatory components of precision grip, which requires two-finger coordination, may have so degraded the grip that the animal chose the simpler, one-finger, strategy. Consistently [10], patients with unilateral cerebellar damage showed timed, ramp-like anticipatory grip force adjustments on the unaffected side, but maintained high grip forces on the affected side. Switching from an efficient, phasic strategy to a costly tonic strategy may be necessitated by loss of the cerebellar adaptive timing needed for the more efficient strategy.

The intermediate zone of cerebellar cortex, which dominates the nucleus interpositus (NIP), also shows strong activity modulation during precision grip. A majority of Purkinje cells in this zone responded with a decrease in tonic activity during maintained grasping [11]. This decrease would disinhibit the NIP cells, whose

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resultant excitatory responses could act via the red nucleus, or via motor thalamus and MI, to generate a force increase.

Sufficient repetition of predictable slip events generates anticipatory discharges in NIP-controlling cerebellar cortex neurons. Dugas and Smith [12] trained monkeys to grasp an object and hold it in a fixed vertical position for 1 s. During a block of trials called slip perturbation trials, a downward force was briefly applied to the object after it had been kept at the correct vertical position for 750 ms. The monkey prevented the object from moving outside a narrow range of vertical heights by phasically stiffening its wrist and firming its grip. Objects of different weights and textures were used. On slip perturbation trials, there was a reflex response evidenced by increases in hand muscle activity and by modulation of neuronal discharge in Purkinje and unidentified cells in the paravermal anterior lobe of the cerebellum. Activity increased in muscles with a 30-50 ms latency, and peaked at 50-100 ms after the perturbation. About half of the recorded Purkinje cells increased or decreased their simple spike discharges at about 45 ms after the perturbation. Most of the Purkinje cells that responded to the perturbation had cutaneous receptive fields.

After a series of perturbations, a grip force increase, and an increase in Purkinje cell activity, developed in anticipation of the perturbation, which occurred reliably 750 ms after the cue tone. Grip force began to diverge upward relative to control levels 450 ms before the expected perturbation, and nearly half of these Purkinje cells increased their discharge at least 50 ms before the grip force divergence. As the anticipatory discharges developed, the same cells decreased their reactive, post-perturbation discharge (rendered unnecessary by the effectiveness of the anticipatory response). None of the Purkinje cells exhibited perturbation-related complex spikes, which if present would indicate excitation of Purkinje cells by climbing fiber (CF) discharges. The absence of slip-related CF discharges (in the Purkinje cells studied) may explain why anticipatory increases were observed in these Purkinje cell responses rather than anticipatory reductions. Many other studies of cerebellar activity have indicated that learned increments in some Purkinje cells’ activities typically coincide with learned decrements in others [e.g. 13]. Long-term depression (LTD) of excitatory parallel fiber (PF) inputs to Purkinje cells depends on coincidence between two inputs to Purkinje cells: predictive state/context signals carried by PFs and (putative error) signals carried by perturbation-locked CF discharges [14]. Long-term potentiation (LTP) occurs when predictive stimuli excite Purkinje cells in the absence of coincident CF discharges [14]. Both LTD and LTP can promote grip force increments if they occur in separate command pathways for opponent muscles. CF discharges in response to cutaneous slip have been reported [15].

B. Timing and variation of precision grip force

Timing of grip force with respect to load force. In [16], subjects grasped and lifted a 400 g object to about 2 cm above a table top, held it suspended for 10 s, and then replaced it. On some trials, the subjects (Ss) were asked to slowly let the object fall, in order to measure that force level, called the slip force, at which the object would slip from the fingers. On typical lift-hold-replace trials, the following phases were observed: (1) One of the fingers first touched the object ~50 ms before the first application of grip force. (2) Grip force increased but not load force. This period lasted 80-40 ms. (3) Grip and load forces increased in parallel. (4) Gravity force was overcome, and the object lifted, until it reached the intended height. In this period, grip force reached its maximum value during a transient overshoot of its steady state. (5) Grip and load forces stabilized while Ss held the object in the air. (6) A reduction of load force allowed the object’s position to slowly approach the table top. (7) At contact, grip and load forces were synchronously terminated.

Rate of rise of grip force during pre-lifting as a function of object weight. The rate of rise of grip force during prelifting is greater for heavier objects [17].

Rate of rise of grip force during pre-lifting as a function of object texture. More slippery objects induced faster rates of pre-lift grip force development. In marked contrast, the rate of rise of load force was the same for all textures [16].

Grip force as a function of texture and weight of the object handled. Applied grip force during lifting is a joint function of the surface texture and weight of the object lifted [18]. The static grip force (grip force maintained during the holding stage) was an increasing function of object weight, as was (of course) the minimal force required to prevent slipping (slip force). A greater grip force was used when the material was more slippery.

Time to maximum grip force across different weights and textures. The time to attain a level of grip force adequate for a given weight/texture is nearly constant for all weights and textures [16, 17]. Such constancy can be expected to greatly reduce variability of behavioral timing.

III. A NEW MODEL OF GRIP FORCE CONTROL

Prior treatments of grip force control [e.g., 1,19,20] have not modeled the neural substrates of adaptive control. This section introduces a new, neurobiologically interpretable, model that formalizes the role of MI and the cerebellum in learned transitions from reactive to anticipatory application of grip forces whose magnitudes are texture- and weight-dependent. Control is exercised in aperture coordinates because once the fingers enclose and touch the object, the targeted hand aperture can be voluntarily decreased by a further amount. Decreasing the targeted aperture to a value less than object width would cause the fingers to try to move beneath the object’s surface, thereby building up a force on it. The size of the
joint stiffness, control of which has been modeled.

Figure shows that current net adjustment $S$ is the integral of the error; applied force would be a function of the size of the decrement (below object weight) of target aperture, and of joint stiffness, control of which has been modeled elsewhere [e.g., 21].

**A. Model circuit and its operation during learning**

A model circuit that learns to generate and apply context-dependent grip forces in anticipation of load force application is shown in Fig. 1. It works as follows. Before learning, there is a significant slip error, $e_s$, the magnitude of which is needed as an input for the model. As shown at the top of Fig. 1, $e_s$ was computed as the difference between $GF_a$, the minimal grip force necessary to prevent slip, and the current net adjustment, $S$, to grip force. Input $e_s$ is tracked by cell activation variable $e_s$, to form an internal estimate of slip error in the frame of the hand. The figure shows that current net adjustment $S$ is the integral of two phasic inputs: a reactive input from cell $e_s$ and a learned anticipatory input from cell $n$. The net adjustment $S$ acts on the hand via aperture/force command $O_A$.

A cerebellar circuit (left side of Fig. 1) enables the model to learn how to pre-empt performance errors. In addition to providing phasic feedback to charge $O_A$, cell $e_s$ sends an error signal to the inferior olive, $IO$, to phasically activate climbing fiber (CF) signals, $cf$. Because CF branches reach both the cerebellar cortex and the deep cerebellar nuclear (DCN) cells, the $cf$ signals excite the Purkinje cell dendritic tree ($p$) and the DCN cells ($n$) inhibited by the Purkinje cell. A context signal, $C_i$, from the pons, corresponds to the decision to lift the object with the weight-texture combination indexed by $i$. Signal $C_i$ activates mossy fibers (MF) $m_i$, each of which in turn generates a spectrum of granule cell activations, $g_i$. This spectrum of activations, inhibited by Golgi interneuron activities $f_j$, generates phasic parallel fiber (PF) activities $f_j$ with different rise times and amplitudes [22]. Adaptive synapses $z_j$ from PFs to Purkinje cells undergo long-term depression (LTD) when PFs are repeatedly paired with CF activations $cf$. In addition, these synapses undergo long-term potentiation (LTP, slower than LTD) when PF signals $f_j$ are present but there are no correlated CF signals. There are also adaptive synapses, $w_p$, from MFs to nuclear cells; these synapses undergo LTD when MF activation is paired with CF activation $h(e_s)$; LTD (slower than LTP in these synapses $w_i$) occurs when MFs are activated without coincident CF activation. Purkinje cells have a baseline activation that normally inhibits the DCN cell. The DCN cell is gradually, and context-dependently, released from this inhibition as the PF-Purkinje cell synapses $z_j$ undergo LTD, because this reduces excitatory inputs to Purkinje cells while inhibitory inputs are maintained. Whenever the $C_i$ cue is presented, the resultant phasic reduction of Purkinje cell inhibition of DCN cells allows the DCN cell activation to express a learned compensation for (what would otherwise be) a forthcoming error. The signal $n$ from the DCN cell reaches the command stage $O_A$, following summation with $e_s$ and integration to form net compensation $S$. When a command is sent to increase the hand aperture $T_A$ and thus release the grip on the object, the integrators must be reset in order to zero the grip force adjustments. Fig. 1 shows that reset in the model is mediated by inhibition of the integrator $S$ whenever there is a positive (opening) aperture velocity command, $V_A$.

In order to generate a load force that depends on weight-related movement errors in the transport component, a corresponding outflow force position vector in the transport component ($O_T$ in the figure) was introduced, which also receives force adjustments from an integrator, $U$. As in the Vector Integration To Endpoint model [23] of MI contributions in arm movement control, $O_T$ cells provide graded force application modulated by integrated feedback of movement error based on signals arising in muscle spindles. Fig. 1 shows that $U$ integrates $T_A$ and $V_A$ to track the movement error, $e_T$. The input $e_T$ is computed as the difference between the minimal load...
force, \(LF_s\), adequate for the given object weight, and the current load force, \(U\).

**B. Technical specifications of the model**

This section specifies the dynamics of the Fig. 1 circuit.

**Arm transport component.** The arm transport component obeys the following system of equations (see [24, 23] for similar treatments):

\[
\dot{D}_c = \alpha (-D_c + T_r - P_f) \tag{1}
\]

\[
\dot{V}_c = \alpha_r (-V_c + G[D_r]) \tag{2}
\]

\[
\dot{P}_r = V_r \tag{3}
\]

and

\[
O_r = P_r + U \tag{4}
\]

where \(D_r\) is the transport difference vector (positive values only when rectified via \([D_r]^+\)), \(D_c\) is its time derivative of \(D_r\), \(T_r\) is the internal representation of the position of the target, \(P_r\) is the transport present position vector, \(V_r\) is a velocity command vector, and GO signal \(G\) initiates movement. Parameters \(\alpha\) and \(\alpha_r\) were set to 30 and 300, respectively. \(O_r\) is the outflow force-position vector for the transport component, and \(U\) integrates the load error:

\[
\dot{U} = \alpha U \tag{5}
\]

where \(\alpha\) was set to 40. In this study, 1-D vectors were sufficient to represent stages in the control of the elbow flexion needed to lift the object.

**Grip aperture component.** The grip aperture component obeyed the following system of equations:

\[
\dot{D}_a = \alpha (-D_a + T_a - P_a) \tag{6}
\]

\[
\dot{V}_a = \alpha_r (-V_a + GD_a) \tag{7}
\]

\[
\dot{P}_a = V_a \tag{8}
\]

and

\[
O_a = P_a - S \tag{9}
\]

where \(D_a\) is the difference vector for hand aperture, \(T_a\) is the internal vector representation of the target aperture, \(P_a\) is the aperture present position vector, \(V_a\) is the aperture velocity vector; \(O_a\) is the outflow force-position vector; \(S\) is defined by

\[
S = \alpha_s [-e_a + \beta_s [V_a] S] \tag{10}
\]

where \(e_a\) is the slip error signal (which tracks \(e_\text{f} \)) and \(n\) is the nuclear (DCN) cell activity. Here again, 1-D vectors sufficed for current purposes. The term \(-\beta_s [V_a] S\) resets the integration of the reactive and predictive adjustments to grip force, by causing a decay in cell \(S\) whenever the aperture velocity cell, \(V_a\), is positive. This reset is needed to implement releases of grip force, by eliminating the slip-preventing grip adjustments and allowing the aperture to relax. The rate term \(\alpha_s\) was set to 40; \(\beta_s = 3\).

**GO signal.** The GO signal generator is defined as:

\[
\dot{G} = \alpha_g (-G + G_o(t)) \tag{11}
\]

where \(G_o(t) = g_o \cdot a(t)\) \(a(t)\) and \(a_0 = 30\). \(G\) is the GO signal multiplying the difference vector of each component (i.e., (2), (7)), and \(g_o\) is a step input from a decision center in the brain.

**Slip error.** The slip error, \(e_\text{f}\), starts being integrated by \(e_\text{f}\) at 0.050 s after the onset of the GO signal, to account for the delay between onset of muscle activation and onset of slip signals from mechanoreceptors. The delayed slip error is defined by

\[
\dot{e}_\text{f} = \alpha_\text{r} [-e_\text{f} + \gamma_s [e_\text{f}]] \tag{12}
\]

where \(\alpha_\text{r} = 50\). Factor \(\gamma_s = 0.08\) scales the error, \(e_\text{f}\), which is approximated by

\[
e_\text{f} \approx GF_o(u, \nu) - S \tag{13}
\]

where \(GF_o\) is the minimal grip force needed to prevent slip of an object of weight \(\nu\) and texture \(\nu\).

**Load error.** The load force error for the transport component, \(\epsilon_r\), starts being integrated by \(\epsilon_r\) at 0.050 s after the onset of the GO signal, to account for the delay in detecting load error. The delayed load error is defined by

\[
\dot{\epsilon}_r = \alpha_r [-\epsilon_r + \gamma_r [\epsilon_r]] \tag{14}
\]

where \(\alpha_r = 50\); \(\gamma_r = 0.25\) scales the error, \(\epsilon_r\), which is approximated by

\[
\epsilon_r \approx LF_s(u) - U \tag{15}
\]

where \(LF_s\) is a load force adequate for an object of weight \(u\). \(a(t)\)

**Cerebellar component.** This component follows the cerebellar timing model developed by Bullock et al. [22], for eye blink conditioning [cf. also 25]. This is one of the simplest models that give the needed computations, viz.: learning with any interstimulus interval (ISI) in the range \([0.1, 4]\) s and timed generation of a context-specific response of sufficient size to preempt the expected error.

A phasic context signal, \(C_j\), activated at \(t = -0.200\) s, excites cell activities, \(m_j\), carried by mossy fibers:

\[
m_j = -\alpha_m n_j + \beta_m (1-m_j)(C_j + n_j) \tag{16}
\]

where \(\alpha_m = 0.2\) and \(\beta_m = 10\); the positive feedback in (17) allows the network to keep a trace of \(C_j\) in short-term memory. Mossy fibers are directed to two classes of cells, granule cells and deep cerebellar nuclear cells. Granule cells were defined as

\[
g_j = \alpha_f (1-g_j) m_j - \beta_g (g_j + \gamma_g j_j) \tag{17}
\]

where \(\alpha_f\) is the rate of activation, drawn from the interval \([1.3, 12]\), of the \(j\)th granule cell, where \(j = 1,2,...,40\), \(\beta_g = 4\), and \(\gamma_g = 0.1\). Signal \(j_j\) is a feedback inhibition of granule cells by Golgi cells:

\[
I_j = -\alpha_f j_j + \beta_f (1-I_j)(\gamma_f m_j + \gamma_f j_j) \tag{18}
\]

where \(\alpha_f = 0.1\), \(\beta_f = 5\), and \(\gamma_f = 0.02\). The use of \(j_j\) in (19) and \(I_j\) in (18) together imply recurrent signal processing in the cerebellar cortex. The signals \(j_j\) conducted by PFs were

\[
f_j = \frac{b_j [g_j - I_j]}{1 + c_j [g_j - I_j]^2} \tag{19}
\]

where \(b = 12\), \(c = 4\), and \(\lambda_j = 5\).
Signals $f_j$ are directed to a Purkinje cell through synapses, $z_{ij}$, which adapt according to
\[
\dot{z}_{ij} = f_j ((1 - z_{ij}) - \beta_i h(e_j) z_{ij})
\]  
where the learning rate $\beta_i = 10$. Thus weights $z_{ij}$ can exhibit slow LTP via term $f_j (1 - z_{ij})$ and faster LTD via term $f_j \beta_i h(e_j) z_{ij}$. The LTD process is gated by CF signal
\[
h(e_j) = \begin{cases} 
1 & \text{if } \dot{e}_j < 0, \\
0 & \text{otherwise} 
\end{cases}
\]  
This function provides a means to use only the leading edge of the slip error signal $e_A$. This is justified by evidence that the IO provides this type of filtering [e.g., 26]. The Purkinje cell firing rate was defined as
\[
p = 1 + \frac{1.5 \text{sign}(b) b^2}{1 + b^2}
\]  
where $b = J^+ - J^-$ is the net activity on the dendrites of the Purkinje cells. Here the excitatory term
\[
J^+ = \sum_{i=1}^N f_i z_{iA}
\]  
and the inhibitory term
\[
J^- = \sum_{i=1}^N f_i z_{iA}
\]  
where $N = 40$. Term $J^+$ represents the influence of basket and stellate cells on the Purkinje cell. For present purposes, the sum in (25) had a constant value of 1.0. Mossy fibers are also directed to the DCN cell, whose activity, $n$, was defined by
\[
n = \alpha_n (- n + \sum_{i=1}^M m_i w_i - p)
\]  
where $M$ is the number of different context cues and $\alpha_n = 100$. The adaptive synapses, $w_i$, from MFs to nuclear cells were adapted according to
\[
w_i = m_i (\alpha_n w_i + \beta_i (1 - w_i) h(e_j))
\]  
where the forgetting rate $\alpha_n = -0.001$ and the learning rate $\beta_i = 10$.

IV. Results

To show activation dynamics of key internal variables, simulations of initial learning (Fig. 2A) and asymptotic phases (Figs. 2B) are shown. The behavioral effect of learning can be seen by comparing the early learning plots in 2A with the asymptotic performance plots in 2B. Note that although the GO signal takes off at $t = 0$, load force and grip force take off (during early learning) with a lag corresponding to the delay in the error signals ($r = 0.050$ s). During early learning, grip force is increased in reaction to load force, whereas after learning grip force onset precedes load force onset. Grip aperture, which equals object width, remains the same in all stages of learning. The slip error ($e_A$), which is large during learning, becomes small after learning. The panel labeled IO shows how the model processes the slip error to use only its leading edge to gate cerebellar learning. The functional generation of the

![Figure 2](image-url)

Figure 2. A: Single trial evolution of model variables during early phase of learning adequate grip forces for a 400 g silk-covered object. Horizontal axes give time in s; the horizontal line in the grip force plot shows the minimal grip force necessary to prevent slip. Key: $e_A$, delayed slip error; IO, inferior olive discharge $h(e_A)$; $m_i$, mossy fiber activity; $z_{ij}$, adaptive parallel fibers-Purkinje cell synapses; $w_i$, adaptive mossy fiber-nuclear cell synapse; $f_j$, activities in parallel fibers; $p$, Purkinje cell activity; $n$, nuclear cell activity. In the $f_j$ and $z_{ij}$ plots, there are many separate traces that partially superpose. Separation of the $z_{ij}$ values begins to occur just after the IO discharge. B: Evolution of model variables during one trial following asymptotic learning of grip force generation for same object as in A.

adaptive response is seen by an evolution from no pause in activity to a deep pause in the Purkinje cell activity ($p$), which controls the DCN cell activation ($n$). The DCN cell is released from inhibition at the moment of the Purkinje cell pause and in proportion to the depth of the pause. The learning that enables the adaptive response can be appreciated by looking at the weights of the PF-Purkinje cell adaptive synapses ($z$) and the weights of the MF-
nuclear cell adaptive synapses ($w_i$). In each case, $w_i = 1.0$
after learning, whereas the below 1.0 deviations of those $z_{ij}$
associated with appropriately timed PF signals scaled
directly with the magnitude of the required grip force, and
and with the depth of the Purkinje cell pause. That the learned
$w_i$ were constant while the learned $z_{ij}$ values varied with
object weight and texture (not shown) indicates that the $z_{ij}$
changes were causative for both adaptive timing and
scaling of the cerebellar response.

V. DISCUSSION AND CONCLUSIONS

Other models [e.g., 27], have attributed the asymptotic
load in cerebellar learning to the plastic synapse between
MFs and DCN cells, after a transient phase in which the
PF-Purkinje cell synapse carried more of the adaptive load.
Our results show how the PF-Purkinje cell synapse could
control both timing and amplitude of predictive responses.
Further simulations (not shown) show model compliance
with the major properties of human grip force adjustment,
namely: grip force onset precedes load force onset, grip
force and its rate of increase during lifting are functions of
object texture and weight, and time to maximum grip force
is constant across different weights and textures.

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