Review TRENDS in Cognitive Sciences Vol.8 No.9 September 2004

Adaptive neural models of queuing and timing in fluent action

Daniel Bullock

Cognitive & Neural Systems Department, Boston University, 677 Beacon Street, Boston, MA 02215, USA

In biological cognition, specialized representations and associated control processes solve the temporal problems inherent in skilled action. Recent data and neural circuit models highlight three distinct levels of temporal structure: sequence preparation, velocity scaling, and state-sensitive timing. Short sequences of actions are prepared collectively in prefrontal cortex, then queued for performance by a cyclic competitive process that operates on a parallel analog representation. Successful acts like ball-catching depend on coordinated scaling of effector velocities, and velocity scaling, mediated by the basal ganglia, may be coupled to perceived time-to-contact. Making acts accurate at high speeds requires state-sensitive and precisely timed activations of muscle forces in patterns that accelerate and decelerate the effectors. The cerebellum may provide a maximally efficient representational basis for learning to generate such timed activation patterns.

An important strategy in cognitive science is to choose the representation and associated algorithms/heuristics that are best suited to rapid solution of a problem type. A cognitive system that solves many types of problems needs many kinds of representations. In cognitive neuroscience, there is a consensus that the brain uses many types of representations and associated heuristics. Brain theorists seek to characterize these types and explain how they cooperate to solve animals' cognitive problems.

Three levels of temporal structure in skilled performance

A surprisingly demanding problem is the genesis of skilled behavior using complex effectors like a human's arm or speech articulators. Skilled behavior emerges in temporally structured episodes, and brain areas that use distinct representations contribute to this temporal structuring. This review examines computational models of neural circuits contributing to three levels of temporal structure in behavior. Level one is the *fluent succession of acts* prepared collectively as a sequence. This feature is noticeable during breakdowns, for example, stuttering, in which familiar sequences fail to emerge fluently. Level two is *coordination of rates* across parallel processes, such as planned joint rotations contributing to a reach. To catch a ball, reach dynamics are coordinated with event dynamics: the global rate, and thus the duration, of an interceptive reach is scaled to the ball's approach time. Such scaling is compromised in basal ganglia disease. Level three is *timed anticipatory responses*, such as the braking contractions that our muscles generate to preempt movement 'overshoots'. Loss of such responses in cerebellar patients severely degrades movements. What neural representations and processes enable fluent succession of acts, act-event coordination, and timed anticipatory responses? And what is the proper analysis of particular cases? In reaching to grasp, the hand opens to an aperture larger than the object and then closes, just as the reach completes. Does this require explicit preparation of a sequence of apertures in prefrontal cortex (level one), or may the sequence emerge at lower levels?

Fluent succession of acts via competitive queuing

Fifty years ago, Lashley [1] used data on sequencing errors – in which early and later elements of a sequence mistakenly exchange positions – to infer that neural representations for all elements of a planned sequence are simultaneously active before sequence production. The proposal that sequences are represented by simultaneous *parallel* activation of representations of their elements differs from many classical and contemporary proposals. In most recurrent-state network models [2–4], representations of *all* the elements of a well-learned sequence are never simultaneously active. Instead, the generating system traverses a series of context-states, each of which activates a representation of just the current element. This transiently active representation guides that element's performance and updates the state representation to create the distinctive context needed to recall the next element. The sequence and the sequence representation are emergent and *serial*, not parallel as Lashley proposed.

In many cases examined by Lashley, for example, typing familiar words, the elements of a sequence are few, routine, and knowable in advance. Thus, parallel activation is feasible. Some neural models that take this approach exemplify Lashley's inference that links between successive elements are unnecessary in a sequence representation. Grossberg [5] constructed the first of this class of neural network models (see Fig. 1), now often called 'competitive queuing' (CQ) models [6,7].

Neurophysiological evidence for the CQ model

Until 2002 there was no compelling electrophysiological evidence that the brain used the parallel sequence code and iterative choice cycle postulated by CQ theorists. New

Corresponding author: Daniel Bullock (danb@cns.bu.edu).
Available online 3 August 2004

 1364-6613/$ - see front matter doi:10.1016/j.tics.2004.07.003

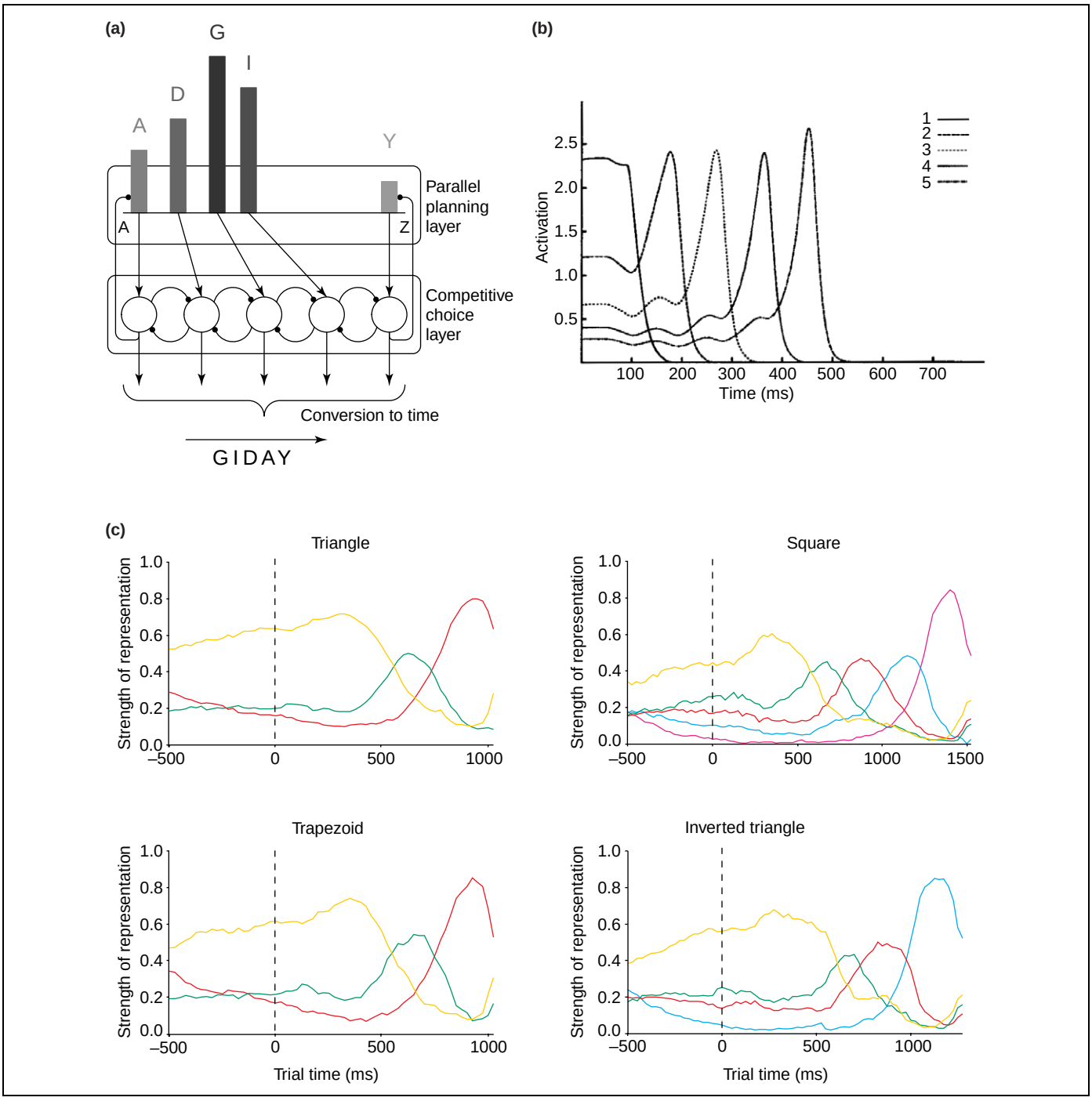


Figure 1. A competitive queuing (CQ) network and a comparison of simulated CQ dynamics with cellular data from area 46 of prefrontal cortex. **(a)** All CQ models have at least two layers, a parallel planning layer and a competitive choice layer. The planning layer contains nodes representing possible sequence elements, such as letters of the alphabet A…Z. To plan a sequence, a desired subset of these nodes is activated in parallel (e.g. nodes representing letters that spell the Australian greeting 'GIDAY') and the relative *degree* of activation (signaled by the relative heights of bars placed above the nodes) is used to specify the relative *priority* of performance. At the onset of a gating signal, the active representations begin to compete for output via the choice layer. If the competition is fair, the most active plan-layer node will win the competition and thereby generate a corresponding output from the choice layer, which initiates the action. A second effect of this output, mediated by an inhibitory pathway from each output node to its corresponding plan-layer node, is deletion of activity at whatever plan-layer node has just won. Iteration of this choose–perform–delete cycle assures that an element's initial relative activation level in the planning layer implicitly codes its relative priority in the forthcoming sequence, and that after the iteration, the plan layer will be empty, and ready for preparation of further sequences. If nothing interrupts the feedback and iterated choice processes, then production of a planned sequence is very fluid. **(b)** A simulation of cellular dynamics in the plan layer of a normalized CQ model [10] during production of a 5-letter sequence, such as 'GIDAY', or a 5-stroke drawing [8]. Each simulation trace depicts the activation history of one of the sequence element representations (1–5) during the interval from just before initiation of sequence performance to just after production of element 5. These simulation traces correspond remarkably well with empirical observations made a decade later [8] and shown in (c). [However, the data-processing steps [8] used to generate (c) preclude using it directly as evidence for neural normalization.] **(c)** Each colored data trace represents the relative activation level in area 46 (of a monkey's prefrontal cortex) of a small neural ensemble that represents one element of a 3-, 4-, or 5-element sequence used to draw a geometric form. (a) Adapted with permission from [7]; (c) Reproduced with permission from [8].

cell recordings by Averbeck *et al.* [8] plugged that evidential gap. They trained monkeys to draw a copy of a static geometric form using a routine, prescribed stroke sequence. Thus a form cued recall of sequence-representing information from long term memory. Recordings from area 46 of prefrontal cortex showed that before the monkey began the stroke sequence, there existed a parallel representation, as proposed in CQ theory (see Fig. 1b,c). As strokes were produced, deletions occurred as expected from this representation, with the most active representation being deleted first, and so on, until the final stroke was made and its representation deleted. These data buttress the hypothesis that the brain uses *parallel* activation patterns to represent, plan and control the execution of short sequences. The same results disconfirm the conjecture [9] that monkeys don't use a 'collective' planning strategy.

In some CQ models [5,10–12], the total activity allocated to plans is normalized and redistributed to the remaining representations on each iteration. By the time the last element is chosen, its activity will have grown to a much larger value than it had initially (Fig. 1b). Such normalization (which abets the explanatory successes of a recent production system model [13]) predicts that the average activation of each representation varies inversely with sequence length. Experiments that varied the number of alternative response options in deferred choice tasks [14–16] confirmed this neurophysiological prediction.

Progress of CQ models in explaining sequencing and timing

To motivate normalized CQ models, Grossberg [5] stressed that neurons exhibit finite activation ranges and noise. Both constrain the ability of neurons to use relative activations to *reliably* code the relative priority of a large number of sequence elements. Brains using this analog code should exhibit a small upper bound on the number of elements that can be reliably recalled in correct sequential order without secondary strategies, such as reloading chunks from long-term memory [5,6,12]. Cowan [17] showed that such a small upper bound has been found for working memory capacity in tasks requiring immediate recall of novel sequences in correct order. Page and Norris [18] showed that a CQ model with noisy choice predicts additional data from immediate serial recall tasks, such as the overwhelming tendency for exchange errors to be transpositions of adjacent elements in the planned sequence.

In all CQ models, the latency to produce a sequence element depends on the time needed for the activation level of the corresponding plan to win the competition. Because more simultaneously active plans imply a lower activation level for each (normalization), the latency to initiate the first element should increase with sequence length. Such effects, measured by Sternberg and colleagues [19], were successfully simulated [10] in 1991. However, more recent data [20,21] showed that for well-practiced sequences, some practice-dependent process overcomes the latency penalty initially associated with preparing sequences. Neurophysiological data [22] implicate the lateral cerebellum in recall of well-learned sequences, and recent neuroanatomical data [23] show projections to prefrontal cortical area 46 from the cerebellum's dentate nucleus. Consistent with these data, Rhodes and Bullock [12] constructed an adaptive neural model to explain how practice-dependent cerebellar outputs could mediate 'parallel analog loading' of sequence element activations into the plan layer of a working memory that obeys CQ principles (Fig. 1a). This model shows how a cortico-cerebello-cortical circuit can learn and recall long-term (procedural) sequence memories. After practice, strong cerebellar output forces the choice layer of the CQ system to pre-commit to the first sequence element. Thereafter, initiation latency is independent of the number of elements in the practiced sequence.

The past decade has seen many extensions of CQ-compatible sequence learning and control models to cognitive phenomena. These include sequences with repeating elements [6,11,24], sequences with overlapping performance of successive elements, as in speech coarticulation [6] and cursive handwriting [25,26], pitch-duration sequences in melody learning [27], and language production [28,29]. Notably, Ward's connectionist language generator illustrates how to link CQ theory to 'construction grammar' [30]. If recent neurophysiological evidence [8,14–16] for CQ assumptions and predictions survives scrutiny, many further CQ model elaborations can be expected.

Coordination of rates and completion times in voluntary action

Many movement models, such as Equilibrium Point (EP) models ([31], Box 1), treat the temporal structure of actions from a biomechanical perspective. By contrast, some central pattern generation models, such as Vector Integration To Endpoint (VITE) models ([32], Box 1), treat timing from a cognitive dynamics perspective, with a focus on voluntary gating of plan execution and voluntary control of movement rates. VITE models have successfully simulated both the discharge patterns of diverse motor cortical cell types [32,39] and numerous movement timing phenomena [12,25,26,39,40]. Recently, VITE-consistent models have explained timing properties of interceptive reaching and reach-grasp coordination. Peper and colleagues [41] noted that if an interceptive reach is to succeed at getting the hand to an approaching ball before it passes by, then global scaling of the reaching rate should be coupled to a relational percept. In their Required Velocity (RV) model [41], an evolving perceptual variable, the ball's declining time-to-contact (TC) with the catcher, continuously adjusts the reaching rate to that velocity required for a successful interception. Because TC^{-1} increases during the reach, it can function in the same way as the increasing GO signal in VITE models [32]. Comparing predictions of the VITE and RV models with data revealed a common weakness [42], which was corrected by allowing ball velocity to affect both rate (via TC) and hand direction. The resulting 'prospective' VITE model of Dessing and colleagues [42] explains many interceptive reaching phenomena (Fig. 2a). Evidence for

Box 1. Equilibrium point models contrasted with central pattern generators

Some neurobiologists once entertained the hypothesis that actions might not require *any* internal trajectory planning, because movements might be treated as mere transitions between postures. If the balance of muscle forces needed to *hold* a goal posture B were abruptly instated, then the body's existing posture, A, would suddenly be in a biomechanical disequilibrium created by muscles acting as spring-like force generators. Movement would ensue, with the body's configuration attracted towards goal posture B as a mechanical equilibrium point in the space of possible body configurations. So emerged 'equilibrium point' (**EP**) models. Simple versions of EP theory fall to logical counterexamples. Suppose a kneeling quadriped hoping to stand were to abruptly activate its muscles to the levels needed to generate the pattern of forces typically used to hold standing posture. This fails because standing quadripeds support most of their weight by columns of bone – not by muscle forces. Elaborated EP models have failed empirical tests [31], and none explains overwhelming evidence (e.g. [15,16,32]) for continuous movement vector computations in motor cortex. By contrast, such evidence inspired central pattern generator (CPG) models such as the 'Vector Integration To Endpoint' (**VITE**) model [32]. A CPG model is also preferable to sensory feedback control models, because simple voluntary movements in primates survive removal of all sensory feedback regarding the controlled limb. The VITE CPG models a nexus of brain adaptations needed for deliberative planning and voluntary movement. Thus, most acts require parallel contributions by many muscles contracting by markedly different lengths. To avoid jerky actions, the rate of each contraction must be proportional to its desired length. When we slow or speed an act, multiplicatively scaling all these proportionate rates with a single GO signal (see Fig. 2 in main text) ensures synchronous contractions. Even if onsets are asynchronous, temporal equifinality (synchronized terminations) of contractions occurs if the volitional rate scaling signal's amplitude increases as the act unfolds. Onset (or offset) of such a GO signal can initiate (or halt) plan execution – a basic requirement for voluntary action. Finally, vector plans can be cognitively prepared for alternative effectors – such as the right and the left arm – until a decision process selectively gates performance by one alternative. The VITE theory predicts a brain site that is both a gate for plan execution and a modulator of movement speed. Stimulation at such a site should affect movement rate but leave movement direction unaffected. Evidence supports the hypothesis that the basal ganglia and related parts of thalamus serve the gating/scaling function for locomotion, reaching, handwriting, speaking and eye movements [26,33–37]. An adaptive model of basal ganglia gating of cortical plans (consistent with VITE and CQ) recently appeared [38].

TC neurons is abundant [42–44], and TC is prominent in other timing models, including models of viapoint movements [44] and legato articulation [45,46] by pianists.

Another focus of timing research is reach-grasp coordination. The Hoff–Arbibmodel [47] showed how internal duration computations could ensure that hand opening and closing were adjusted to both the planned duration of a reach and the maximum expected hand aperture (the thumb to finger distance), which was solely a function of object size. Yet reach duration often emerges from a dynamic coupling between actor and object motions [41,42], and maximum hand aperture depends on reach rate [48]. Thus neither component durations nor maximum apertures are known in advance. The VITE-based model (Fig. 2b) of Ulloa and Bullock [49] generates reach-grasp coordination without such advance knowledge. Key timing-data trends [50,51] emerge dynamically. Thus, although there is a single *pre*-planned hand aperture goal (object size), a cross-coupling between the reach and grasp circuits allows the aperture to be transiently incremented during the reach by an amount proportional to reach velocity, which in VITE trajectories is maximal midway through the duration of a typical (unperturbed) reach. Because the transient increment to aperture fades after mid-time of the reach, the model generates an aperture sequence without explicit sequence preparation. Under typical (and perturbed, see Fig. 2b) conditions, the reach and hand closure finish synchronously. This exemplifies the temporal equifinality property (Box 1 and Fig. 2b) afforded by using a single rate-scaling signal to coordinate parallel processes.

To students of control theory, VITE circuits (Fig. 2) invite comparisons with engineering approaches such as PID (Proportional, Integral, Derivative) control, in which motor command components are proportional to the fedback error (between the desired and current sensory states), its integral, and its time derivative. However, whereas PID control requires sensory feedback, a central pattern generator (CPG) like VITE can operate without sensory feedback. Our analysis [39] concluded that primates use flexible CPGs of desired kinematics, which operate in cascade with a spinal PID controller and a cerebellar feedforward controller. The VITE circuit models only some properties of the inferred CPG. Equally important as voluntary rate scaling to the success of coordinated movements is flexible recruitment and de-recruitment of the multiple effectors that contribute motion components in different directions and amplitudes as an act unfolds. Guenther and colleagues [52,53] have developed adaptive neural models called DIRECT and DIVA to explain effector recruitment in arm and speech movements, respectively. Fiala [54] and Barreca and Guenther [53] each demonstrated how to preserve principles of the VITE model within complex, DIRECT-type, models. In DIRECT, learning creates inverse kinematic mappings and predictive forward kinematic mappings that transform between sensory and motor coordinates. Such networks enable 'motor-equivalence', i.e. equivalent results from variable means, because as the movement unfolds, the learned internal mappings recruit novel combinations of joint rotations adequate to produce desired visual motions of a hand, or a tool held in the hand. The DIRECT model clarifies how adaptive circuits can achieve performances similar to abstract, but behaviorally predictive, models like the 'optimal feedback' model [55] of Todorov and Jordan. In particular, DIRECT and DIVA respectively enable on-line corrections to unexpected perturbations of movements by arms and speech articulators.

Timed anticipatory responses

In a successful ball catch, the arm flicks out and 'stops on a dime' at whatever degree of arm extension enables the hand to catch the ball. Newtonian mechanics implies that an arm set in motion by extensor muscles would (disastrously) continue 'past the mark' unless braked by precisely timed, anticipatory action of opposing muscles. When driving a car, stomping the accelerator and hitting

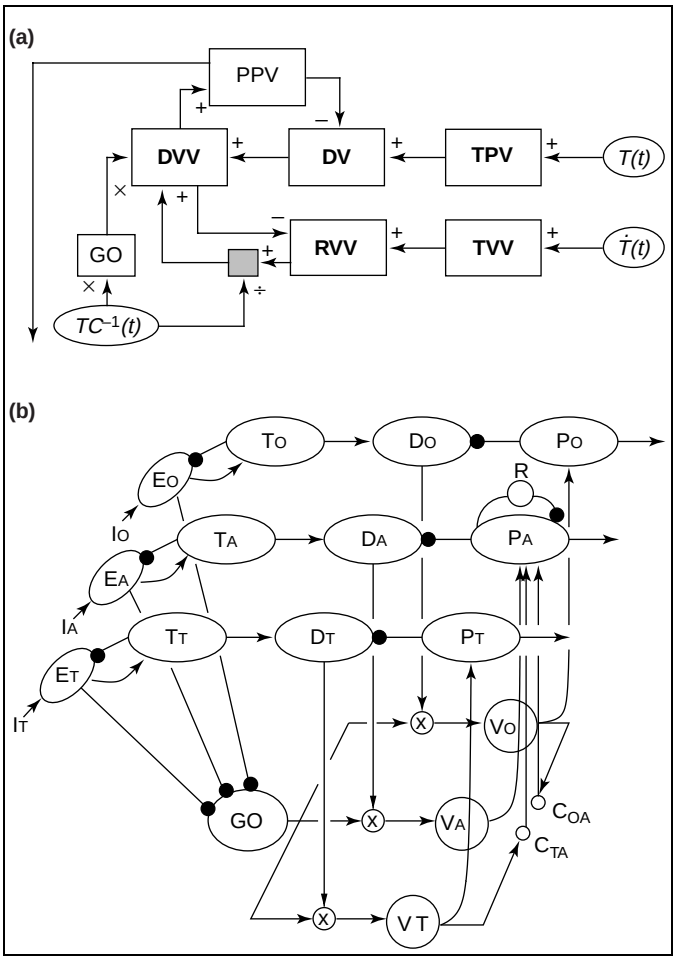


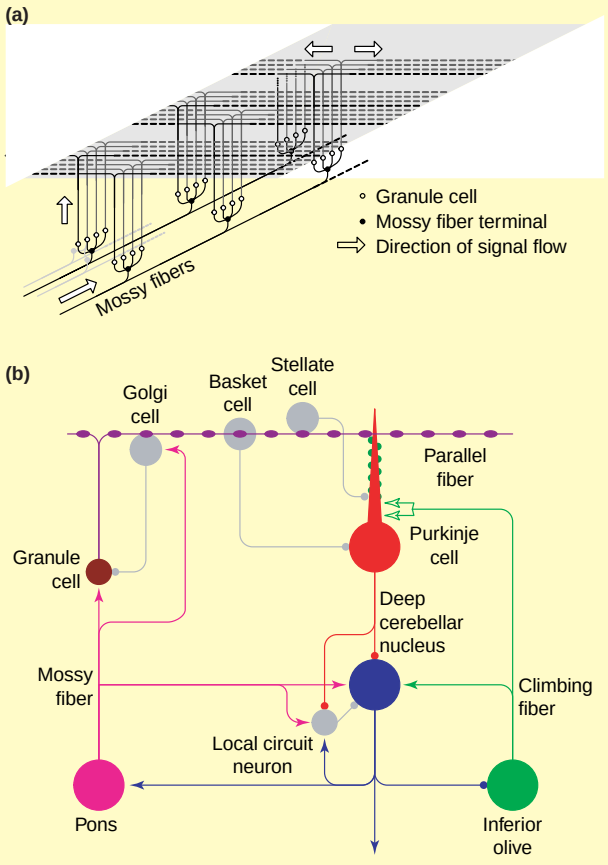
Figure 2. Vector integration to endpoint models for catching and grasping. **(a)** The prospective VITE model [42] of interceptive reaching, a basic component of catching. Symbols $T(t)$ and $\dot{T}(t)$ are the position and velocity of an approaching object (e.g. a ball). In all VITE models, DV is the difference vector computed between representations of the target position vector (TPV) and the hand's present position vector (PPV). Output from the PPV continuously specifies desired hand position. The desired velocity vector (DVV) for the hand arises as a product of the DV and an internally generated GO signal. Because of its multiplicative control of the DVV, the GO signal can be used to initiate movement, scale its overall velocity, and halt movement. The PPV is generated internally by continuously integrating the DVV. The prospective VITE model augments the basic VITE circuit (just described) by using target velocity both to compute TC (time-to-contact) and to refine the effective DVV by computing a relative velocity vector (RVV). The inverse, $TC^{-1}(t)$, an 'urgency to complete' signal, refines the effective GO signal. These interactions adjust movement velocities to values needed to intercept in the time available. This VITE model is called 'prospective' rather than 'predictive' because it anticipates without ever predicting the locus of interception. If a model were to predict and move by the shortest path to the locus of interception, it could not explain the systematic reversals of movement direction exhibited by human catchers. **(b)** A neural model [49] for temporal coordination of three component processes – hand orientation control, control of hand transport by the arm, and hand aperture (distance between thumb and index finger) control – that operate in parallel during reaches that culminate in a precision grasp of an object at rest. The symbols T_i, D_i, and P_i stand for internally computed target, difference, and present position vectors, respectively; the V_i stand for desired velocity vectors. The values of the subscript i indicate either transport (T), aperture (A), or orientation (O), for example, T_T is the target object's location, T_A is targeted hand aperture, and T_O is targeted hand orientation. To prepare a movement plan, initial values of the D_is are computed before GO signal activation initiates movement. I_i is a perceptual representation of the target for each component. E_i is the discrepancy between the perceptual (I_i) and plan (T_i) representations of the target for each component. Discrepancies arise if the object suddenly changes in size, orientation or distance. Such discrepancies inhibit the GO signal and slow all movement components until planning variables again agree with current percepts. C_{TA} and C_{OA} are inputs to aperture control from velocity of transport and velocity of orientation, respectively. These inputs cause maximum aperture transiently to exceed object size by an amount that scales with movement speed – useful for avoiding collision of the fingers with the object during the hand's approach [48]. Node R represents a process mediating delayed self-inhibition of hand aperture. (a) and (b) Reproduced with permission from [42] and [49], respectively.

the brake are separate voluntary actions. When 'driving' our bodies, the braking contractions are automatic and of subcortical origin. The timed anticipatory components of the motor cortex signals that shape braking reactions disappear if part of the cerebellum's dentate nucleus is cooled [56]. Many such observations implicate the cerebellum as an engine for the learning and performance of anticipatory responses [56–59] that fit the following formula: *In context C generate signals in output channel A after waiting time interval T.* Furthermore, Perrett, Ruiz and Mauk [60] showed that a restricted cerebellar cortical lesion, which spares the cerebellum's deep nuclei, produces an animal that makes its anticipatory responses *too early.* Such experiments have created a consensus that some mechanism in the cerebellar cortex learns to withhold an anticipatory response until the optimal time T after onset of the conditional stimulus (CS) that indicates the state/context within which the response should be generated.

The network and cells in the cerebellar cortex are complex, and several models have been proposed to explain cerebellar adaptive timing. These models fall into two broad classes: *network-delay* models and *synaptic-delay* models (see Box 2). Two similar cerebellar network-delay models [61,62] were proposed independently in 1994 and both borrowed ideas from earlier adaptive timing models (e.g. [63]). Both postulated that a CS (conditional stimulus) carried to the cerebellum by mossy-fibers induces local interactions between Golgi cells and large populations of granule cells. Such interactions might enable any CS to generate a spectrum of transient, time-lagged granule cell activations that was specific to that CS. Given such a temporal basis, whichever granule cell activation has the appropriate time delay to coincide with a climbing fiber signal can, over the course of repeated experiential trials, become able to control cerebellar output, provided that an associative learning process operates to change the synaptic weight between that granule cell's parallel fiber and those Purkinje (output) cells excited by the climbing fiber signal. (Signal propagation along parallel fibers does *not* provide a behaviorally significant delay.) Even the latest network-delay models [64,65] use whole cells and network interactions to create the temporal basis. By contrast, synaptic-delay models [66] postulate that the spectrum of delays needed for adaptive timing emerge in synapse-specific elements, namely the tiny spines [66,67] that populate the branchlets of Purkinje cells' dendritic trees. It is with these spines that parallel fibers actually synapse.

Relative to synaptic-delay models, network-delay models are very inefficient. They are metabolically inefficient because they use a cellular network to do what may be done within tiny dendritic spines; and they are computationally inefficient because each granule cell population dedicated to creating a spectrum of time-lagged responses to one CS cannot readily serve other roles attributed to cerebellar cortex. In particular, no network-delay model has shown how purported temporal-basis granule cells could simultaneously fulfill the 'spatial pattern separation' role (Box 2) attributed to the granule cell stage in Marr–Albus models [68–70]. By contrast, all

Box 2. A two-stage cerebellar adaptive engine?



If the cerebellum performs both of the major functions most commonly attributed to it – spatial pattern separation and adaptive timing – then cerebellar learning constitutes a parallel search, through masses of potentially predictive recent signal sets, for reliable leading indicators that will allow the animal to make timed anticipatory responses. According to Marr–Albus theory [68–70], the first stage of cerebellar processing uses the granule cells (Figure Ia,b) to perform spatial pattern separation. Each of millions of mossy fibers (MFs) distributes its signal, one component of a massive state/context vector, across the cerebellar cortex (Figure Ia). Billions of cerebellar granule cells each use four or five dendrites to sample a partially distinctive subset of the MF context representation (Figure Ia). This allows granule cells to detect highly specific event or state-subset combinations. The creation of these combination representations, abetted by inhibitory suppression of granule cells excited by only one or two of their potential inputs, makes the representations of any two contexts more dissimilar ('farther apart' whence 'separation') in the higher-dimensional space defined by granule cell outputs than in the lower-dimensional space defined by MF signals. Each granule cell's parallel fiber (Figure Ia,b) sends its output (if any) to hundreds of Purkinje cells, and each Purkinje cell receives inputs from many thousands of parallel fibers. **Synaptic-delay** models propose that adaptive timing is achieved after the granule stage, by a spectrum of delays within the *population* of Purkinje dendritic spines contacted by each granule cell via its long parallel fiber. Thus spatial pattern separation and adaptive timing can efficiently co-exist and co-operate in each cerebellar compartment. By contrast, **network-delay** models propose that the spectrum of delays emerges in the population of granule cells itself. It remains to be shown in a model how the granule stage could perform both functions.

Figure I. Circuit properties of the mammalian cerebellum. **(a)** Within the lattice-like cortex ('outer rind') of the cerebellum ('little brain'), signals pass along mossy fibers to mossy fiber terminals (rosettes), then to granule cells, then to granule cell axons, which rise and then bifurcate (T-branch) to form parallel fibers. For many mammals, there are as many granule cells in the cerebellum as there are neurons in the remainder of the brain. Each branch of each parallel fiber can excite up to hundreds of Golgi, basket, stellate and Purkinje cells, groups of which are linked in a highly conserved connectivity pattern, one token of which is shown in (b). **(b)** A very highly schematic depiction of the cell types and connections found in the basic cerebellar circuit, which is replicated millions of times, albeit with different inputs and output destinations, in each cerebellum of large mammals. Also shown is the pons, source of a large proportion of mossy fibers, and the inferior olive, sole source of the climbing fibers whose discharges gate one type of cerebellar learning. The convergence onto each Purkinje cell of *thousands* of parallel fibers (only one shown here) but just *one* climbing fiber (as shown) is a striking connectional asymmetry that has figured prominently in most theories of cerebellar learning, including adaptive timing. (A) and (B) reprinted with permission from [4].

variants [4,71] of the synaptic-delay model introduced by Fiala and colleagues [66] are compatible with granule-based spatial pattern separation.

Although further computational challenges to some network-delay models exist – for example, poor signal-processing repeatability – recent empirical results cast strong doubt on the sufficiency of *any* cerebellar network-delay model. Three separate laboratories [57,72,73] have discovered that cerebellar adaptive timing occurs under conditions in which it should be *impossible* if adaptive timing requires the network-delay mechanism. By contrast, the synaptic-delay model correctly predicts successful adaptive timing in all three cases. In the earliest experiment, Shinkman, Swain and Thompson [57] substituted prolonged direct stimulation of parallel fibers (granule cell axons) for CS presentation, and demonstrated normal delayed response learning and performance. For this protocol, the network-delay model instead predicts a learned response that begins much too early and persists until CS offset. The second experiment, by Raymond and Lisberger [72], was explicitly designed to pit network-delay models against synaptic-delay models. They found that the information needed to control behavior had disappeared from the granule cell discharges ~100 ms before the signal that initiated associative learning. Under such conditions, the network-delay model predicts no response learning – again, contrary to the data. Raymond and Lisberger inferred that there must be a synapse-specific delay of at least 100 ms – an inference since confirmed by direct observations on Purkinje cell dendritic spines [67]. In the third experiment, Svensson and Ivarsson [73] used decerebrate ferrets to eliminate any hippocampal or other cerebral contributions to adaptive timing. They then showed successful temporal conditioning using a 'trace' protocol, in which there is a gap between CS offset and the time the conditioned response should be generated. For this protocol, the network-delay model predicts no learning, because there is no stimulus to drive granule cell activations once the CS goes off. By contrast, synaptic delay models predict robust learning, provided that the trace interval is not too long. Available data suggest that

the cerebellum is sufficient for trace intervals up to at least 600 ms [73,74] Learning across longer trace intervals requires hippocampal assistance during training. Thus all three experimental results are consistent with the synaptic-delay model, but inconsistent with the network-delay model.

How efficient should a biologically accurate model of adaptive timing be? The synaptic-delay model proposed by Fiala *et al.* [66] is much more efficient than the network-delay model (Box 2), but it is not maximally efficient. Two offspring [4,71] of the synaptic-delay model have proposed distinct ways to replace each of the original model's populations of dendritic spines (and associated pre-existing spectra of delays) with a *single* adaptive element. In these untested models, a granule cell's output is transformed into a separate, experience-trained time delay within each Purkinje dendritic spine contacted.

Conclusions

This review has focused on neural circuit models of fluent performance of discrete actions, and the implicit claim was that *at least* three kinds of temporal structuring must be acknowledged to exist as distinct factors in most episodes of skilled action. Competitive queuing theorists who endorsed Lashley's inference that some sequence planning involves parallel activation of all sequence elements can now point to compelling electrophysiological support, but this does not imply that other mechanisms play no role in sequence representations important for fluent action. Vector-integration-to-endpoint models explain many electrophysiological and behavioral patterns as signatures of a nexus of circuit adaptations that made flexible voluntary action possible, but many ideas from the literatures on equilibrium point control and feedback control are usefully incorporated in extensions of CPG models like VITE. Synaptic-delay data and models indicate that the brain has an efficient intracellular mechanism for detecting predictive relationships between event onsets separated by intervals from tens of milliseconds to at least half a second, but this may say little about interval timing on significantly longer time scales [75]. Finally, it is exciting to see emerging data and computer models that highlight the embedding of subcortical circuits, in the basal ganglia and cerebellum, within segregated loops that include the frontal cortex [23,36,37]. These subcortical circuits, heretofore often associated with operant conditioning (basal ganglia) and classical conditioning (cerebellum), have emerged as key to cognitive functions such as decision making (e.g. [38]) and serial plan preparation (e.g. [12]). Clarifying the functional architecture of these extended circuits is a priority of most brain modelers interested in the temporal structure of behavior.

Acknowledgements

Preparation of this article was partially supported by NIH R01 DC02852.

References

1 Lashley, K.S. (1951) The problem of serial order in behavior. In *Cerebral Mechanisms In Behavior* (Jeffress, L.A. ed.), Wiley
2 Elman, J. (1995) Language processing. In *The Handbook Of Brain Theory And Neural Networks* (Arbib, M.A. ed.), pp. 508–512, MIT Press, MA
3 Dominey, P.F. (1998) Influences of temporal organization on sequence learning and transfer: Comments on Stadler (1995) and Curran and Keele (1993). *J. Exp. Psychol. Learn. Mem. Cogn.* 24, 234–248
4 Rhodes, B. and Bullock, D. (2002) A scalable model of cerebellar adaptive timing and sequencing: The recurrent slide and latch (RSL) model. *Appl. Intell.* 17, 35–48
5 Grossberg, S. (1978) A theory of human memory: Self-organization and performance of sensory-motor codes, maps, and plans. In *Progress in Theoretical Biology* (Vol. 5) (Rosen, R. and Snell, F., eds), pp. 233–374, Academic Press
6 Houghton, G. (1990) The problem of serial order: A neural network model of sequence learning and recall. In *Current Research In Natural Language Generation* (Dale, R. *et al*, eds), pp. 287–319, Academic Press
7 Bullock, D. and Rhodes, B. (2003) Competitive queuing for serial planning and performance. In *Handbook of Brain Theory And Neural Networks* (Arbib, M. ed.), pp. 241–244, MIT Press
8 Averbeck, B.B. *et al*. (2002) Parallel processing of serial movements in prefrontal cortex. *Proc. Natl. Acad. Sci. U. S. A.* 99, 13172–13177
9 Conway, C.C. and Christiansen, M.H. (2001) Sequential learning in non-human primates. *Trends Cogn. Sci.* 5, 539–546
10 Boardman, I. and Bullock, D. (1991). A neural network model of serial order recall from short-term memory. In *Proceedings of the International Joint Conference on Neural Networks*, II, pp. 879–884, IEEE
11 Bradski, G. *et al*. (1994) STORE working memory networks for storage and recall of arbitrary temporal sequences. *Biol. Cybern.* 71, 469–480
12 Rhodes, B. and Bullock, D. (2002). Neural dynamics of learning and performance of fixed sequences: Latency pattern reorganizations and the N-STREAMS model. *Boston University Technical Report CAS/CNS-02-007*
13 Byrne, M.D. and Anderson, J.R. (2001) Serial modules in parallel: The psychological refractory period and perfect time sharing. *Psychol. Rev.* 108, 847–869
14 Basso, M.A. and Wurtz, R.H. (1998) Modulation of neuronal activity in superior colliculus by changes in target probability. *J. Neurosci.* 18, 7519–7534
15 Cisek, P. and Kalaska, J.F. (2002) Simultaneous encoding of multiple potential reach directions in dorsal premotor cortex. *J. Neurophysiol.* 87, 1149–1154
16 Pellizzer, G. and Hedges, J.H. (2003) Motor planning: Effect of directional uncertainty with discrete spatial cues. *Exp. Brain Res.* 150, 276–289
17 Cowan, N. (2001) The magical number 4 in short-term memory: A reconsideration of mental storage capacity. *Behav. Brain Sci.* 24, 87–185
18 Page, M.P.A. and Norris, D. (1998) The primacy model: A new model of immediate serial recall. *Psychol. Rev.* 105, 761–781
19 Sternberg, S. *et al*. (1978) The latency and duration of rapid movement sequences: Comparisons of speech and typewriting. In *Information Processing In Motor Control And Learning* (Stelmach, G.E. ed.), pp. 117–152, Academic Press
20 Klapp, S.T. (1996) Reaction time analysis of central motor control. In *Advances in Motor Learning and Control* (Zelaznik, H. ed.), pp. 13–35, Human Kinetics, Champaign, IL
21 Verwey, W.B. (1996) Buffer loading and chunking in sequential keypressing. *J. Exp. Psychol. Hum. Percept. Perform.* 22, 544–562
22 Lu, X. *et al*. (1998) Role of monkey cerebellar nuclei in skill for sequential movement. *J. Neurophysiol.* 79, 2245–2254
23 Dum, R.P. and Strick, P.L. (2003) An unfolded map of the cerebellar dentate nucleus and its projections to the cerebral cortex. *J. Neurophysiol.* 89, 634–639
24 Hartley, T.A. and Houghton, G. (1996) A linguistically constrained model of short-term memory for nonwords. *J. Mem. Lang.* 35, 1–31
25 Bullock, D. *et al*. (1993) A neural network model for cursive script production. *Biol. Cybern.* 70, 15–28
26 Contreras-Vidal, J.L. *et al*. (1998) Neural dynamics of short and medium-term motor control effects of levodopa therapy in parkinson's disease. *Artif. Intell. Med.* 13, 57–79
27 Page, M.P.A. (1999) Modeling the perception of musical sequences with self-organizing neural networks. In *Musical Networks* (Griffith, N. and Todd P.M. eds), pp. 175–198, MIT Press
28 Ward, N. (1994) *A Connectionist Language Generator*, Ablex Publishing

29 Dell, G.S. *et al*. (1997) Language production and serial order: a functional analysis and a model. *Psychol. Rev.* 104, 123–147
30 Goldberg, A.E. (1995) *Constructions: A Construction Grammar Approach to Argument Structure*, University of Chicago Press
31 Shadmehr, R. (2003) Equilibrium point hypothesis. In *Handbook Of Brain Theory And Neural Networks* (Arbib, M. ed.), pp. 409–412, MIT Press
32 Bullock, D. and Grossberg, S. (1988) Neural dynamics of planned arm movements: Emergent invariants and speed-accuracy properties during trajectory formation. *Psychol. Rev.* 95, 49–90
33 Horak, F.B. and Anderson, M.E. (1984) Influence of globus pallidus on arm movements in monkeys, II. Effects of stimulation. *J. Neurophysiol.* 52, 305–322
34 Skinner, R.D. and Garcia-Rill, E. (1990) Brainstem modulation of rhythmic functions and behaviors. In *Brainstem Mechanisms Of Behavior* (Klemm, W.R. and Vertes, R.P. eds), pp. 465–496, John Wiley
35 Mateer, C. (1978) Asymmetric effects of thalamic stimulation on rate of speech. *Neuropsychologia* 16, 497–499
36 Turner, R.S. *et al*. (1998) Motor subcircuits mediating the control of movement velocity: a PET study. *J. Neurophysiol.* 80, 2162–2176
37 Hikosaka, O. *et al*. (2000) Role of the basal ganglia in the control of purposive saccadic eye movements. *Physiol. Rev.* 80, 953–978
38 Brown, J. *et al*. (2004) How laminar frontal cortex and basal ganglia circuits interact to control planned and reactive saccades. *Neural Netw.* 17, 471–510
39 Bullock, D. *et al*. (1998) Cortical networks for control of voluntary arm movements under variable force conditions. *Cereb. Cortex* 8, 48–62
40 Sternad, D. *et al*. (2000) Interaction of rhythmic and discrete pattern generators in single joint movements. *Hum. Mov. Sci.* 19, 627–665
41 Peper, C.E. *et al*. (1994) Catching balls: How to get the hand to the right place at the right time. *J. Exp. Psychol. Hum. Percept. Perform.* 20, 591–612
42 Dessing, J. *et al*. (2002) Prospective control of manual interceptive actions: Comparative simulations of extant and new model constructs. *Neural Netw.* 15, 163–179
43 Sun, H. and Frost, B.J. (1998) Computation of different optical variables of looming objects in pigeon nucleus rotundus neurons. *Nat. Neurosci.* 1, 296–303
44 Bullock, D. *et al*. (1999) A vector-integration-to-endpoint model for performance of viapoint movements. *Neural Netw.* 12, 1–29
45 Jacobs, J.P. and Bullock, D. (1998) A two-process model for control of *legato* articulation across a wide range of tempos during piano performance. *Music Perception* 16, 169–199
46 Repp, B.H. (1997) Acoustics, perception and production of *legato* articulation on a computer-controlled grand piano. *J. Acoust. Soc. Am.* 102, 1878–1890
47 Hoff, B. and Arbib, M.A. (1993) Models of trajectory formation and temporal interaction of reach and grasp. *J. Mot. Behav.* 25, 175–192
48 Wing, A. *et al*. (1986) Grasp size and accuracy of approach in reaching. *J. Mot. Behav.* 18, 245–260
49 Ulloa, A. and Bullock, D. (2003) A neural network simulating human reach-grasp coordination by continuous updating of vector positioning commands. *Neural Netw.* 16, 1141–1160
50 Paulignan, Y. and Jeannerod, M. (1996). Prehension movements. The visuomotor channels hypothesis revisited. In *Hand and Brain: The Neurophysiology and Psychology of Hand Movements* (Chap. 13) (Wing, A., Haggard, P., *et al*., eds), pp. 265–282, Academic Press
51 Saling, M. *et al*. (1996) Grip reorganization during wrist transport: the influence of an altered aperture. *Exp. Brain Res.* 108, 493–500
52 Guenther, F.H. *et al*. (1998) A theoretical investigation of reference frames for the planning of speech movements. *Psychol. Rev.* 105, 611–633
53 Barreca, D.M. and Guenther, F.H. (2001) A modeling study of potential sources of curvature in human reaching movements. *J. Mot. Behav.* 33, 387–400
54 Fiala, J.C. (1994). A network for learning kinematics with application to human reaching models. *Proceedings of the 5th IEEE International Conference on Neural Networks*, pp. 2759–2764, IEEE Press
55 Todorov, E. and Jordan, M.I. (2002) Optimal feedback control as a theory of motor coordination. *Nat. Neurosci.* 5, 1226–1235
56 Vilis, T. and Hore, J. (1980) Central neural mechanisms contributing to cerebellar tremor produced by perturbations. *J. Neurophysiol.* 43, 279–291
57 Shinkman, P.G. *et al*. (1996) Classical conditioning with electrical stimulation of cerebellum as both conditoned and unconditioned stimulus. *Behav. Neurosci.* 110, 914–921
58 Spencer, R.M. *et al*. (2003) Disrupted timing of discontinuous but not continuous movements by cerebellar lesions. *Science* 300, 1437–1439
59 Thompson, R.F. *et al*. (1997) Associative learning. In *The Cerebellum and Cognition (International Review of Neurobiology, Vol. 41)* (Schmahmann, J.D. ed.), pp. 152–190, Academic Press
60 Perrett, S.P. *et al*. (1993) Cerebellar cortex lesions disrupt learning-dependent timing of conditioned eyelid responses. *J. Neurosci.* 13, 1708–1718
61 Bullock, D. *et al*. (1994) A neural model of timed response learning in the cerebellum. *Neural Netw.* 7, 1101–1114
62 Buonomano, D.V. and Mauk, M.D. (1994) Neural network model of the cerebellum: temporal discrimination and the timing of motor responses. *Neural Comput.* 6, 38–55
63 Fujita, M. (1982) Adaptive filter model of the cerebellum. *Biol. Cybern.* 45, 195–206
64 Medina, J.F. *et al*. (2000) Timing mechanisms in the cerebellum: Testing predictions of a large-scale computer simulation. *J. Neurosci.* 20, 5516–5525
65 Tieu, K.H. *et al*. (1999) Perirhinal-amygdala circuit-level computational model of temporal encoding in fear conditioning. *Psychobiology* 27, 1–25
66 Fiala, J.C. *et al*. (1996) Metabotropic glutamate receptor activation in cerebellar Purkinje cells as substrate for adaptive timing of the classically conditioned eye blink response. *J. Neurosci.* 16, 3760–3774
67 Wang, S.S-H. *et al*. (2000) Coincidence detection in single dendritic spines mediated by calcium release. *Nat. Neurosci.* 3, 1266–1273
68 Marr, D. (1969) A theory of cerebellar cortex. *J. Physiol.* 202, 437–470
69 Albus, J.S. (1971) A theory of cerebellar function. *Math. Biosci.* 10, 25–61
70 Schweighofer, N. *et al*. (2001) Unsupervised learning of granule cell sparse codes enhances cerebellar adaptive control. *Neuroscience* 103, 35–50
71 Steuber, V. and Willshaw, D.J. (1997) How a single Purkinje cell could learn the adaptive timing of the classically conditioned eye-blink response. *Lecture Notes Comput. Sci.* 1327, 115–120
72 Raymond, J. and Lisberger, S. (1998) Neural learning rules for the vestibular-ocular reflex. *J. Neurosci.* 18, 9112–9129
73 Svensson, P. and Ivarsson, M. (1999) Short-lasting conditioned stimulus applied to the middle cerebellar peduncle elicits delayed conditioned eyeblink responses in decerebrate ferret. *Eur. J. Neurosci.* 11, 4333–4340
74 Clark, R.E. *et al*. (2002) Classical conditioning, awareness, and brain systems. *Trends Cogn. Sci.* 6, 524–531
75 Gallistel, C.R. and Gibbon, J. (2000) Time, rate, and conditioning. *Psychol. Rev.* 107, 289–344