2003-09

Adaptive Neural Models of Queuing and Timing in Fluent Action

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September, 2003

Technical Report CAS/CNS-2003-018

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Submitted to *Trends in Cognitive Sciences*
24 September 2003

Boston University Technical Report CAS/CNS-2003-018

Acknowledgements: Preparation of this article was partially supported by NIMH R01 DC02852.

Teaser Sentence: Fifty years after Lashley inferred *parallel* cerebral representations of *serial* plans, and twenty-five years after Grossberg proposed a parallel *competitive queuing* model, confirmatory neurophysiological data arrive – but other new data cast doubt on network-delay models of cerebellar adaptive timing.

Abstract. Temporal structure in skilled, fluent action exists at several nested levels. At the largest scale considered here, short sequences of actions that are planned collectively in prefrontal cortex appear to be queued for performance by a cyclic competitive process that operates in concert with a parallel analog representation that implicitly specifies the relative priority of elements of the sequence. At an intermediate scale, single acts, like reaching to grasp, depend on coordinated scaling of the rates at which many muscles shorten or lengthen in parallel. To ensure success of acts such as catching an approaching ball, such parallel rate scaling, which appears to be one function of the basal ganglia, must be coupled to perceptual variables, such as time-to-contact. At a finer scale, within each act, desired rate scaling can be realized only if precisely timed muscle activations first accelerate and then decelerate the limbs, to ensure that muscle length changes do not under- or over-shoot the amounts needed for precise acts. Each context of action may require a much different timed muscle activation pattern than similar contexts. Because context differences that require different treatment cannot be known in advance, a formidable adaptive engine – the cerebellum – is needed to amplify differences within, and continuously search, a vast parallel signal flow, in order to discover contextual "leading indicators" of when to generate distinctive parallel patterns of analog signals. From some parts of the cerebellum, such signals control muscles. But a recent model shows how the lateral cerebellum may serve the competitive queuing system (in frontal cortex) as a repository of quickly accessed long-term sequence memories. Thus different parts of the cerebellum may use the same adaptive engine design to serve the lowest and the highest of the three levels of temporal structure treated. If so, no one-to-one mapping exists between levels of temporal structure and major parts of the brain. Finally, recent data cast doubt on network-delay models of cerebellar adaptive timing.

Keywords: adaptive timing, competitive queuing, neural network, prehension, motor coordination, time-to-contact, cerebellum, prefrontal cortex, basal ganglia

Introduction: Three levels of temporal structure in skilled performance.

Skilled behavior emerges in temporally structured episodes, and different parts of the brain play distinct roles in temporal structuring. This review treats computational models of brain bases of three nested levels of temporal structuring in skilled behavior. The first is the *fluent succession of acts* that have been planned together as a sequence. This feature is most noticeable during breakdowns, e.g., stuttering, in which a highly practiced sequence fails to emerge with the expected fluency. The second level is *coordination of rates and completion times* across parallel processes. Consider reach-grasp coordination. To catch a thrown ball, the rate, and thus the duration, of an interceptive reach is adjusted to the approach time of the ball. The reach dynamics are coordinated with the event dynamics. Moreover, within the reach time, the hand first opens to an aperture larger than the ball and then closes, just as the reach completes. One breakdown of this competence is seen in Parkinson's patients' inability to scale movement rate. The third level is *timed anticipatory responses*, such as the ubiquitous braking contractions that our muscles generate to pre-empt movement 'overshoots'. Loss of this ability is apparent in the severely dysmetric movements generated by patients with damage to the cerebellum. What adaptive neural mechanisms enable us to achieve fluent succession of acts, coordination of event-act rates and completions, and timed anticipatory responses?

Fluent succession of acts via competitive queuing.

Fifty years ago, in a seminal article for cognitive neuroscience, Lashley[1] used data on *Spoonerisms* – in which early and late elements of a speech sequence mistakenly exchange positions (e.g., "seech spequence" is spoken instead of the planned "speech sequence") – as one basis for inferring that neural representations for all elements of a planned sequence are simultaneously active, and capable of interacting (e.g., exchanging positions), prior to production of the sequence. This radical proposal, that sequences are represented by simultaneous *parallel* activation of representations of their elements, is fundamentally different from many classical (e.g., behaviorist) and contemporary proposals. For example, in most recurrent-state network models[2,3,4], 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 both guides that element's performance and updates the state representation to create the distinctive context needed to recall the next element. In such models, both the sequence and the sequence representation are emergent and *serial*, not parallel as Lashley proposed.

State-dependent emergent sequencing is unavoidable if an adaptive cognitive system is to remain responsive to the evolving context, which is rarely predictable for long intervals. But in many cases of the type examined by Lashley, e.g., phrase-level speech or cursive letterform production, the elements of a sequence are routine and known in advance. In such cases, it is possible for the brain to treat the problem of sequencing as a problem of *relative timing* of the onsets and the *degree of overlap* between successive elements. Some recurrent neural network models take this approach and are compatible with Lashley's 1951 inference. In 1978, Grossberg[5,6] constructed the first of this class of neural network models (see Figure 1), which Houghton[7] later dubbed *competitive queuing* (**CQ**) models[8].

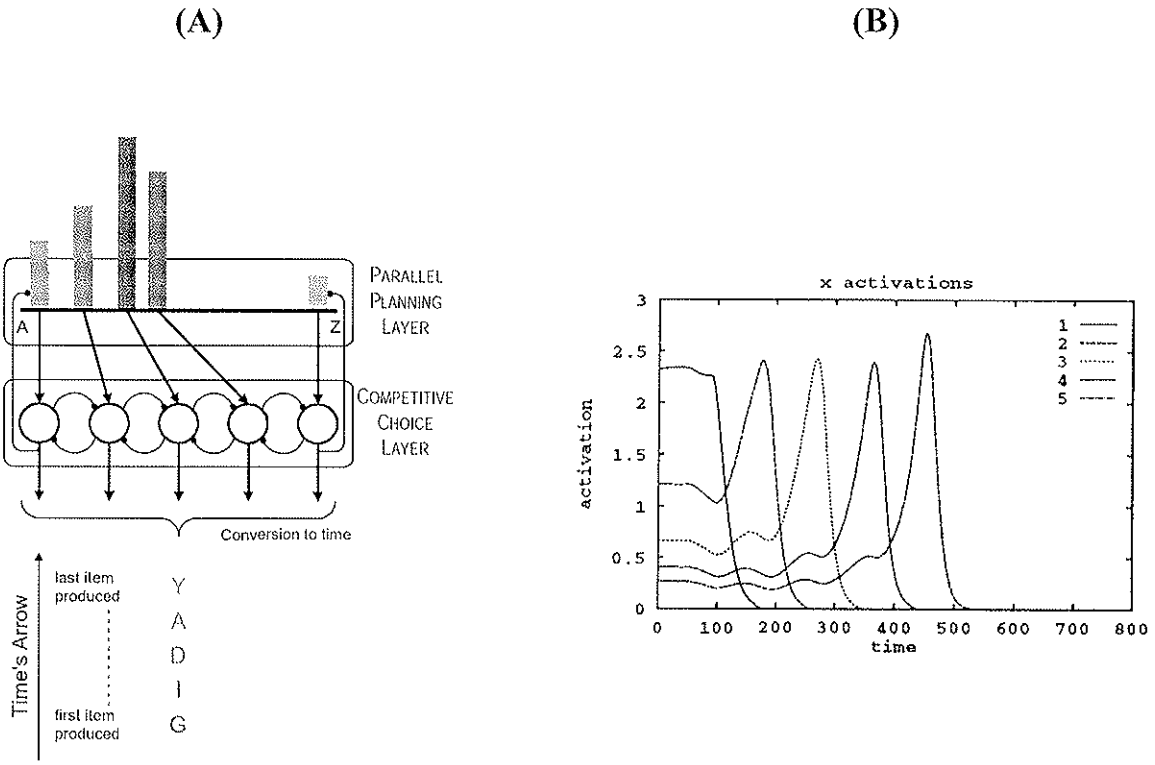

Figure 1: A competitive queuing (CQ) network and associated cellular dynamics. A: All CQ models have at least two layers, a parallel planning layer and a competitive choice layer. The parallel planning layer contains nodes representing possible sequence elements, such as letters of the alphabet A ... Z. To prepare a planned sequence, a desired subset of these nodes is activated in parallel (such as nodes representing the letters that spell the Australian greeting "GIDAY") and the relative *degree* of activation is used to control 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, then the most active plan layer node will always win the competition, and thereby generate a corresponding output from the choice layer, which initiates the chosen action. A second effect of this output, mediated by a recurrent inhibitory pathway from each output node to its corresponding plan layer node, is deletion of activity at whatever plan layer node has just won. For a two item sequence, 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 second choice, the plan layer will be empty, and thus ready for preparation of further sequences. For use with sequences longer than two, the planning layer must be designed so that deletion of any node's activity leaves invariant the rank ordering of the remaining node activations. If nothing interrupts the feedback and iterated choice processes, then production of a planned sequence can be very fluid. **B:** A simulation of cellular dynamics in the plan layer of a normalized CQ model[12] during production of a 5-item sequence, such as "GIDAY". These simulation traces correspond remarkably well with empirical observations made a decade later[9]. Each simulation trace marks the activation history of one of the sequence element representations during the interval from just before element one's performance to just after production of the entire sequence.

Neurophysiological confirmation of the CQ model.

Until 2002 – almost twenty-five years after Grossberg proposed the first CQ model and fifty years after Lashley inferred parallel activation – there was no compelling electrophysiological evidence that the brain used both the parallel sequence code and the iterative choice cycle postulated by CQ theorists. New cell recordings by Averbeck, Chafee, Crowe and Georgopoulos[9,10] plug that evidential gap. They trained monkeys to respond to presentation of a geometric form by using a joystick to draw a copy of the same form. To make their drawings, the monkeys were required to use a prescribed stroke sequence that had become routine via extensive practice. Thus on each trial, a geometric form, which contained no visible indication of sequence, served as a cue for recall of sequence-representing information from long term memory. Recordings from prefrontal cortex showed that just before the monkey began the stroke sequence, there existed a parallel representation of exactly the type proposed in CQ theory (see Figure 1). As the 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 the final representation deleted. These data strikingly confirm one of the most dramatic and counter-intuitive hypotheses of cognitive neuroscience: that the brain uses *parallel* activation patterns to represent, plan, and control the execution of short routine movement sequences. The same results *dis*confirm the recent conjecture[11] that monkeys might be incapable of using such a "collective" planning strategy.

Averbeck's data support the type of CQ model[5,12-14] in which the total activity allocated to plans is approximately normalized and dynamically redistributed to the remaining element representations on each iteration of the competition. For example, by the time the last element was to be chosen, its activity had grown to a much larger value than it had initially. Such normalization (which also figures prominently in the explanatory successes of a recent 'neuralized' production system model[15]) implies a simple prediction. When more elements are represented in a neural planning layer, the average activation of each representation must be less. This neurophysiological prediction has been confirmed in recent experiments that systematically varied the number of alternative response options in a deferred choice task[16,17].

Progress of CQ models in explaining sequencing and timing.

When he introduced the normalized CQ model, Grossberg[5,6] stressed that nodes in real neural networks are subject to finite activation level ranges and some inevitable noise. Such factors constrain the ability of a neural circuit to use relative activation level to *reliably* code the relative priority of *many* sequence elements. Brains using this analog basis for prioritization should exhibit a small upper bound on the number of elements that can be reliably recalled in correct sequential order without use of secondary strategies, such as reloading chunks from long-term memory[5]. Cowan[18] shows that such a small upper bound has been convincingly demonstrated by research on the capacity of working memory as assessed in tasks that require immediate recall of novel sequences in correct order. Recently, Page and Norris[19] 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 take the form of transpositions of adjacent elements in the planned sequence.

In all CQ models, the latency to produce a sequence element is a function of the time needed for the activation level of the corresponding plan to win the competition, by being the first to exceed the threshold for execution. Because more simultaneously active plans imply a lower activation level for each (normalization), the latency to initiate the first element of a sequence should increase with sequence length. Such a *sequence length effect on latency* was reported by Sternberg and colleagues[20] and successfully simulated by a CQ model[12] in 1991. However, recent data[21,22] show that this latency effect does not persist at high levels of practice. Some practice-dependent process makes it possible to overcome the latency penalty initially associated with preparing sequences. Consistent with new neuroanatomical evidence[23] of projections from lateral zones of the deep cerebellar nuclei to the premotor and prefrontal areas of the cerebral cortex, Brad Rhodes[14,24] recently constructed an adaptive neural model to explain how cerebellar outputs learned during extensive practice could effect a "parallel analog load" of sequence element activations into the plan layer of a frontal working memory that obeys CQ principles (Figure 1). Rhodes's self-regulating adaptive network model shows how a cortico-cerebello-cortical circuit can learn and recall long-term (procedural) sequence memories. After enough practice, the cerebellar output becomes strong enough to force a pre-commitment to the first sequence element by the choice layer of the CQ system. Thereafter, initiation latency is independent of the number of elements in the practiced sequence.

The past decade has been a rich period for extensions of CQ-compatible sequence learning and control models to a wide range of phenomena. These include sequences with repeating elements[7,25], sequences with overlapping performance of successive elements, as in speech coarticulation[7] and cursive handwriting[26,27], pitch-duration sequences in melody learning[28,29], and grammatical language production[30,31]. Given the dramatic recent neurophysiological confirmations[9,10,16,17] of key CQ predictions, a sustained burst of further CQ model elaborations can be expected in the next few years.

Coordination of rates and completion times in voluntary action.

Many movement models, such as *equilibrium point* models (Text Box 1), treat the temporal structure of voluntary actions from a biomechanical perspective. In contrast, *vector integration to endpoint* (VITE) models (Text Box 1) treat timing from a cognitive perspective, with a focus on voluntary gating of plan execution and willed modulation of movement rates. Since their inception, VITE models have successfully predicted the discharge patterns of a diversity of motor cortical cell types[33,40], and have been applied to numerous movement timing problems[14,26-28,41], including adapting rate to the size of a load[40]. Recently, VITE-consistent models have been applied to explain timing properties of interceptive reaching and reach-grasp coordination. In 1994, Peper and colleagues[42] noted that if an interceptive reach is to succeed at getting the hand to an approaching ball before it passes the plane of the torso, then the global scaling of the reaching rate should be coupled to a perceptual variable specifying the ball's changing relationship to the actor. This led to the Required Velocity (RV) model[42], in which a continuously evolving perceptual variable, the ball's declining *time-to-contact* (TTC) with the catcher, adjusts the reaching rate to that velocity required for a successful interception. Because 1/TTC is continuously increasing during the reach, it can function in the same way as the increasing GO signal of the VITE model[33]. Comparing predictions of the VITE and RV models with interceptive performances revealed a common weakness[43]. Whereas both models used the evolving relative *positions* of hand and ball to guide movement, they ignored their relative

velocities. (Anyone who has considered the analogous problem of intercepting one missile with another will recognize why both variables are critical!) With the addition of sensitivity to relative velocity, and appropriate coupling to TTC, the new velocity integration to endpoint model of Dessing and colleagues[43] explains a range of human reaching data that is – literally – beyond the reach of prior models. Neural evidence for the model's assumption of TTC cells is abundant[43,44], and TTC is prominent in other sensory-motor timing models, including VITE-based models of viapoint movements[45] and legato articulation[46,47] by pianists (see Figure 2).

Text Box 1: Equilibrium point models contrasted with vector integration to endpoint models. 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 muscles 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 toward 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 quadriped trying to stand abruptly instates the muscle forces used to hold standing posture. This fails because standing quadripeds support most of their weight by columns of bone – not by active muscle forces. More elaborate EP models have been empirically refuted[32], and none are well suited to explain overwhelming evidence for continuous movement vector and postural computations in motor cortex. In contrast, such evidence inspired the *vector integration to endpoint* (**VITE**) circuit model[33], which incorporates a *nexus* of brain adaptations believed to lie at the core of the system for deliberative planning and volition-modulated execution. Most acts require parallel contractions by many synergistic muscles, which often contract by markedly different lengths during the act. Suppose that all contributing muscles began to contract at the same instant and thereafter contracted at the same fixed rate until finishing their contribution. Then the muscles that had short-length contractions would complete their transits much earlier than those with long contractions. The result would be very jerky – and utterly unlike our fluid skeletal movements, in which the rate of a contraction is proportional to its desired size[34]. Moreover, when we want to slow or speed an act, multiplicatively scaling all these proportionate rates with a single volitional GO signal ensures synchronization of contractions, whatever the overall movement duration set by the global scaling factor. Even if contraction onsets are asynchronous, *temporal equifinality* (synchronized terminations) of contractions is greatly facilitated if the value of the GO signal (volitional rate scaling signal) increases as the act unfolds. Onset and offset of a GO signal serve to initiate or halt plan execution – a basic requirement for voluntary action. Finally, vector plans can be cognitively prepared for several alternative effectors – such as the right and the left arm – until a deliberative process reaches a decision that selectively gates realization of one alternative. One prediction of the VITE model is therefore a brain site that acts as both a gate for execution of movements and a modulator of movement speed. Stimulation at such a site should affect movement rate while leaving movement direction unaffected. Evidence confirming this prediction[35-38], together with much additional evidence[39], supports the hypothesis that the basal ganglia serve the gating/scaling function for locomotion, reaching and eye movements. An adaptive basal ganglia gating model, consistent with both VITE and CQ principles, has recently appeared[39].

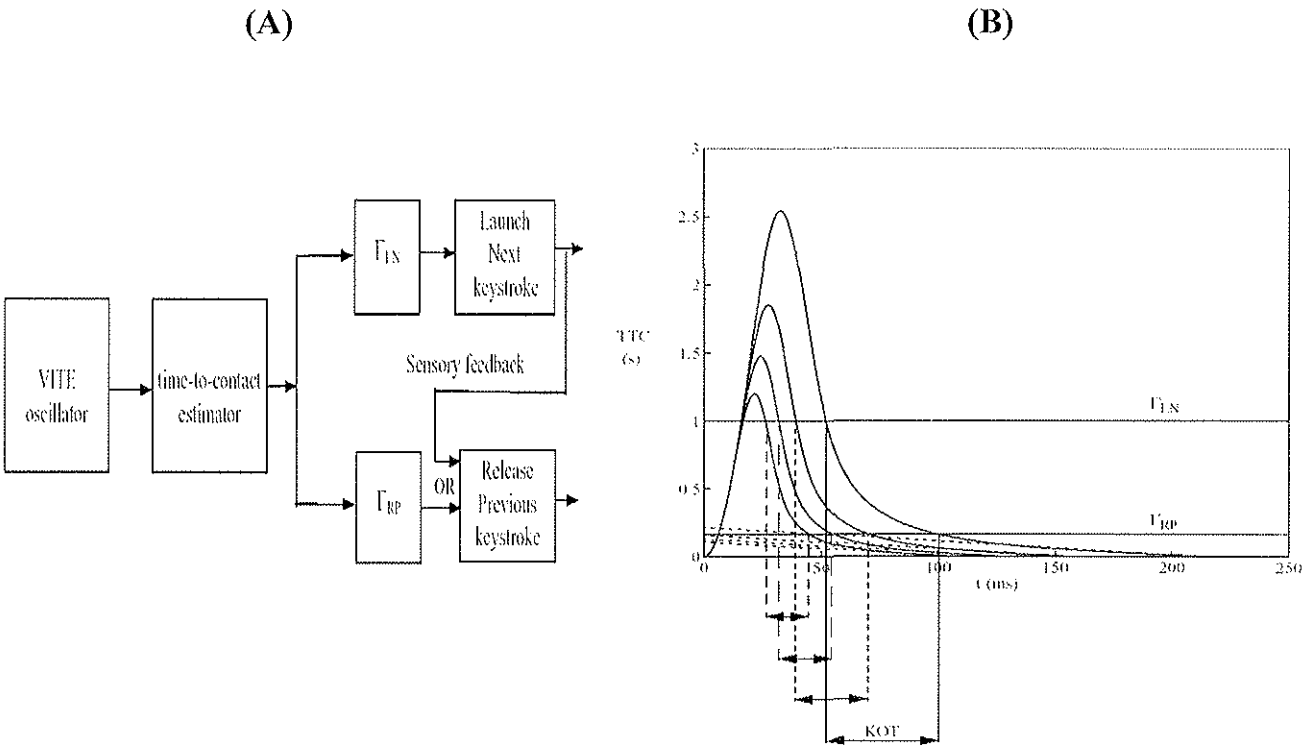

Figure 2. How do pianists produce legato articulation? Part A schematizes a dual-process model for *legato* control of key depressions and key releases during performance of isochronous note sequences. Successive piano tones fall into two classes: unconnected tones and connected tones. A pianist can use finger articulation to produce connected tones, referred to as *legato*, by waiting to release key *n* at or after the time that key *n+1* is depressed. The duration of simultaneous depression is called the *key overlap time* or KOT. The KOTs measured during experts' *legato* articulation vary markedly with tempo. For scales performed at inter-note onset intervals (IOIs) of 100-1000 msec., there is an increasing non-linear functional dependence of KOT on IOI. Because the non-linearity appears in the long IOI (slow tempo) region, it is not attributable to gross biomechanical factors, such as finger inertias. Concert pianist (and engineer) Pieter Jacobs showed[46] that the dependence can arise from a neural circuit (schematized here in part A) in which a prospective central process and a slow sensory feedback process cooperate to control articulation. The vector integration to endpoint model was extended to create a neural circuit that exhibits volition-controlled oscillation rates and simulates 'mental foot tapping'. It also affords prospective control by continuously computing an internal first order estimate of the remaining time-to-contact (TTC) with a targeted integration level, reaching of which triggers the oscillator's next half cycle. At fixed successive threshold values of the estimate of time remaining in the current half cycle, the performer first launches keystroke *n+1* and then lifts keystroke *n*. The higher of these thresholds, and the first crossed by the declining value of TTC, is G_{LN} (LN for "Launch Next") and the lower is G_{RP} (RP for "Raise Prior"). As tempo slows (see part B), the time required for the internal estimate of TTC to pass between threshold crossings elongates, and KOT adapts automatically (increases). If performers used only this mental process to control articulation, they would not show the bend seen in the slow tempo region of the KOT vs. IOI function[46]. The bend emerges if performers lift keystroke *n* whenever the TTC estimate crosses threshold G_{RP} *or* the brain receives tactile feedback from the finger-key contact associated with stroke *n+1*, whichever comes earlier. Tactile feedback delay times are consistent with this interpretation. Why use the mental process at all? At high playing speeds, there is not enough time to wait for the delayed feedback!

Another recent focus of timing research is reach-grasp coordination. A computational model by Hoff & Arbib[48] utilized a complex internal timing scheme to ensure that the durations of hand opening and closing were adjusted to both the expected duration of the reach and the maximum expected hand *aperture* (the thumb to finger distance). This model assumed that component durations are known in advance and that the maximal aperture is solely a function of object size. Yet reach duration often *emerges* from a dynamic coupling between actor and object motions[42,43], and maximum hand aperture is strongly dependent on reach rate[49], not just on object size. Thus neither the component durations nor the maximum aperture are known in advance. Recent VITE-based models of Ulloa and colleagues[50,51] show how reach-grasp coordination can be achieved without the advance knowledge and internal accounting of durations assumed in the Hoff-Arbib model. Key timing data trends[52,53] emerge dynamically. Although there is a single *pre*-planned hand aperture goal (equivalent to perceived 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 models is unimodal and near its maximum midway through the reach's duration. Therefore, the transient increment to hand aperture automatically begins to fade just after mid-time of the reach. Under this condition, synchronous completions of the reach and the hand closure on the object are enabled by the temporal equifinality property of a VITE circuit (Text Box 1), provided that a single rate-scaling signal coordinates the reach and grasp components.

Timed anticipatory responses.

In a successful catch, the arm flicks out and 'stops on a dime' at whatever degree of arm extension is needed to allow the hand to catch the ball. Newtonian mechanics implies that an arm set in motion by synergistic extensor muscles A, B, C would (disastrously) continue 'past the mark' unless braked by precisely timed, anticipatory action of opposing muscles D, E, F. When driving a car, stomping the accelerator and hitting the brake are separate voluntary actions. When 'driving' our bodies, the braking contractions are largely automatic and of sub-cortical origin. The timed anticipatory components of the motor cortex signals that shape braking reactions disappear if the deep cerebellar nuclei are cooled[54]. This observation fits with a huge body of data that implicates the cerebellum as an engine for the learning and performance of timed, anticipatory, context-dependent responses[55-57] that fit the following formula: In context C generate signals in output channel A after waiting interval T. Furthermore, Perrett, Ruiz and Mauk[58] showed that a restricted cerebellar cortical lesion, which spares the deep cerebellar nuclei, produces an animal that makes its anticipatory responses *too early*. This and subsequent experiments have created a consensus that *some mechanism* in the cerebellar cortex confers the ability to learn 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 Figure 3).

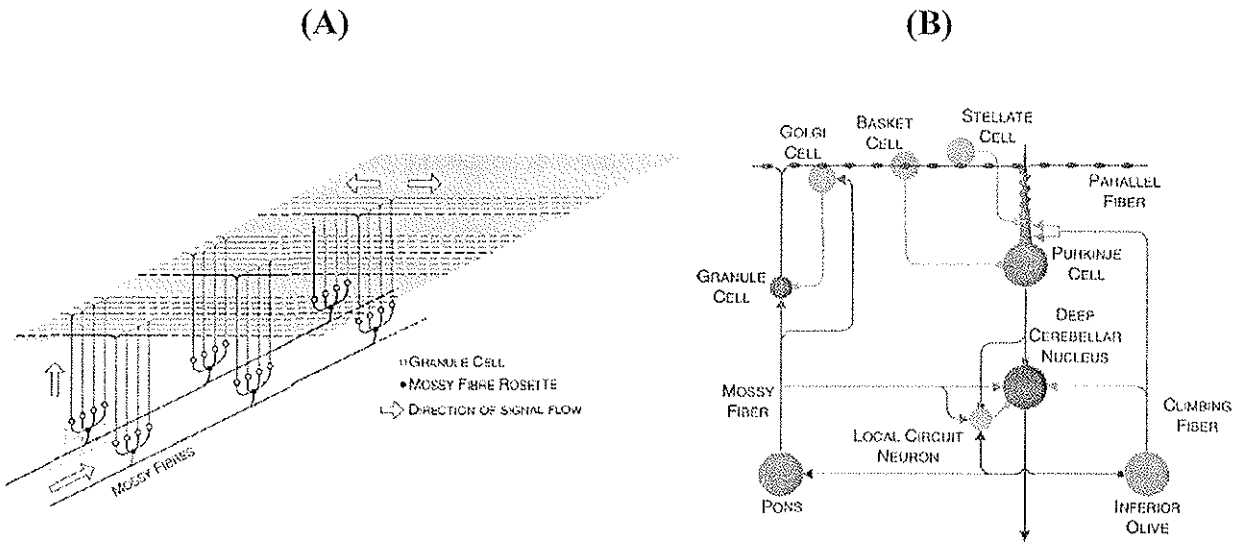

Figure 3: Network-delay versus synaptic-delay models of cerebellar adaptive timing. (A) The passage of signals from mossy fibers through granule cells to parallel fibers within the cortex of the cerebellum. (B) A highly schematic view of the basic cerebellar circuit, including the pons, source of mossy fibers, and the inferior olive, source of the climbing fibers whose discharges gate cerebellar learning. Two similar cerebellar **network-delay** models[59,60] were proposed independently in 1994 and both borrowed ideas from earlier adaptive timing models[61,62]. 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. This enables any CS to generate a spectrum of transient, time-lagged granule cell activations that is 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. Even the latest network-delay models[63,64] use whole cells and network interactions to create the temporal basis. In contrast, **synaptic-delay** models[65] postulate that the spectrum of delays needed for adaptive timing emerge in synapse-specific elements, namely the tiny spines[65,66] (not shown) 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 whole 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 population 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 attributed to the granule cell stage in the Marr-Albus model[67-69]. In contrast, all variants of the synaptic-delay model introduced by Fiala and colleagues[65,70,4] are compatible with granule cell performance of the spatial pattern separation function (see Text Box 2).

Text 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[67-69], the first stage of cerebellar processing uses the granule cells (Figure 3) to perform *spatial pattern separation* (also called *expansive recoding*). In particular, each of millions of mossy fibers (MFs) distributes its signal, which constitutes one component of a massive state/context vector, across the cerebellar cortex (Figure 3). Billions of cerebellar granule cells each use 5 or 6 dendrites to sample a partially distinctive subset of the MF context vector. This design allows granule cells to detect highly specific event or state-subset *combinations*. It is the creation of these combination representations, together with inhibitory suppression of granule cells excited by only a small fraction of their potential inputs, that makes the representations of any two contexts more dissimilar ('farther apart' whence 'separation') in the very high dimensional space defined by granule cell outputs than in the much lower dimensional space defined by MF signals. Each granule cell axon branches to form a parallel fiber (Figure 3) that sends the current result of its sampling operation (if any) to hundreds of Purkinje cells, and each Purkinje cell receives inputs from many thousands of parallel fibers. *Synaptic-delay* models (Figure 3) 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. This makes it easy for spatial pattern separation and adaptive timing to efficiently co-exist and co-operate in the same cerebellar model. In contrast, *network-delay* models (Figure 3) 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. However that issue resolves, the two operations imply that the cerebellum performs a massively parallel real-time search for highly context-dependent leading indicators (event combinations) that will allow its control actions to be both optimally apt and precisely timed.

Although further computational challenges to *some* network-delay models exist – e.g., poor signal processing repeatability due to intrinsic oversensitivity to network fluctuations – recent empirical results cast strong doubt on the sufficiency of *any* cerebellar network-delay model. Three separate laboratories have recently shown that cerebellar adaptive timing occurs under conditions where it should be *impossible* if adaptive timing requires the network-delay mechanism. In contrast, the synaptic-delay model correctly predicts that adaptive timing should be preserved in all three cases. In the earliest experiment, Shinkman, Swain and Thompson[55] 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[71], was explicitly designed to test between network-delay models and synaptic-delay models. They showed that the information needed to control the output had *disappeared* from the granule cell discharges approximately 100 milliseconds 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 msec – an inference since confirmed by direct observations on Purkinje cell dendritic spines[66]. In the third experiment, Svensson and Ivarsson[72] used decerebrate ferrets to eliminate any hippocampal or other cerebral contributions to adaptive timing. They then demonstrated 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. In contrast, the synaptic delay model[65] predicts robust learning, provided that the trace interval is no longer that 1-2 sec. Thus all three experimental results are consistent with the synaptic-delay model, but inconsistent with the network-delay model.

Conclusions.

Some recurrent-state network theorists adhere to a neo-behaviorist and reductionist view of sequencing, in which *all* sequence representation is fundamentally sequential. Modern competitive queuing theorists follow Lashley's radical cognitivist inference that some sequence planning involves "co-temporal" or *parallel* activation of all sequence elements. Early reductionist EP models tried to explain movements as mere biomechanical transitions between postures made without *any* central trajectory generation. Vector integration to endpoint models treat central trajectory-related planning and volitional control of timing as signatures of the nexus of adaptations that made deliberative action possible. Network-delay models of adaptive timing conform to the strict Hebbian doctrine that only cells that fire together – *within a few msec* – can wire together. Synaptic-delay models are more radical. They imply that the brain long ago transcended Hebbian co-incidence strictures by adding an efficient way to detect predictive relationships between events (and cell firings) separated by any interval from tens of msec. to a few seconds. Thus, the trend in real-time adaptive network models of action queuing and timing is decidedly cognitive and anti-reductionist. At the same time, new models exemplifying this trend have begun to show how subcortical structures heretofore associated with simple cases of operant conditioning (the basal ganglia) and classical conditioning (the cerebellum) can also participate in high level cognitive functions, such as decision making[39] and serial plan preparation[14]. If correct, these results imply that no one-to-one mapping exists between levels of temporal structure and major parts of the brain.

An outstanding question: How efficient should a biologically accurate model of adaptive timing be? The synaptic-delay model of adaptive timing proposed by Fiala et al.[65] is much more efficient than the network-delay model (Figure 3 and Text Box 2), but at first glance it does not appear to be *maximally* efficient. This was shown in two recent variants[4,70] of the model, both of which replaced the original model's *population* of dendritic spines (and associated pre-existing spectrum of delays) with a *single* dendritic element that possesses an *experience-trained* time delay. In both variants, a single granule cell output signal can be transformed into a separately learnable time delay within each Purkinje dendritic spine that it contacts. No more efficient model would seem to be possible, if efficiency is measured solely in number of synapses. However, in both variants it is less easy to explain the learning of multiple, distinctively timed responses to a single conditional stimulus. Although for typical cases, this difficulty can be resolved by using the cerebellum's known output-to-input recurrence[4] to iteratively update the cerebellum's state representation as each timed output is generated, it is important to remember that confidence in judgments of relative efficiency must await a far better understanding of the full range of learning cases and ontogenetic infelicities that a biological mechanism has evolved to cope with. In addition to efficiency of representation, the brain needs enough redundancy to afford robust mean statistics that are relatively insensitive to noise or cell death. And a less efficient representation might be favored (over an optimally efficient one) if it supports a faster learning rate.

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