NEURAL NETWORK MODELS OF THE PRIMATE MOTOR SYSTEM

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Dynamic neural network models that incorporate time-varying activity and allow unrestricted connectivity were trained by back-propagation to generate discharge patterns of cells previously observed in behaving monkeys. Neuronal recordings in monkeys performing a simple alternating step-tracking task have shown that forearm motor units and connected premotoneuronal cells fire with characteristic patterns: phasic-tonic, tonic, decrementing, etc.. To investigate the properties of networks that could transform a step input of target position to the four observed discharge patterns of flexor and extensor motor units we trained dynamic network models to generate these firing patterns as outputs. These networks have hidden units with either excitatory or inhibitory connections to each other and to the output "motor units". Network solutions have been found for a variety of connection matrices corresponding to different network topologies. The activity of many hidden units resembles the discharge patterns that have been observed in physiological recordings of neurons in motor cortex and red nucleus. In networks receiving both sustained (step) input and transient input signals, preferential connections can develop within subsets of phasic and tonic units.

The function of specific hidden units in the network can be tested by making selective lesions of particular units and determining the behavior of the remaining network. When relatively few hidden units with similar activations are strongly interconnected, removing a particular unit can have appreciable consequences in eliminating corresponding components of activity in other units. The output effects of a given unit can also be tested by delivering a simulated stimulus and analyzing the propagated network response. Delivering the stimulus pulse during various phases of the ongoing task shows how the impulse response is modulated by the changing activation patterns.

1. INTRODUCTION: PHYSIOLOGICAL NETWORKS

The neural circuitry controlling forelimb muscles of the primate has been elucidated by experiments on the anatomical connections and physiological discharge patterns of the neurons during movements. Particularly revealing are experiments in which both the activity and output connections of the same cells can be determined. The premotoneuronal cells which affect muscle activity can be identified in behaving

monkeys by spike-triggered averaging of EMG recordings, which can reveal post-spike facilitation or suppression of target muscles [2,3]. The response patterns of corticomotoneuronal (CM) cells and rubromotoneuronal (RM) cells have been documented during simple alternating flexion/extension task designed to relate activity to changes in force and to sustained static force [1,2]. The types of discharge patterns observed in CM and RM cells, as well as in single motor units (MU) of agonist muscles, fall into specific classes [1-3,5]. During a ramp-and-hold movement, all three groups include cells that show phasic-tonic discharge. The phasic component is related to the changing force and the tonic component is proportional to the amount of static force exerted. All groups also include tonic cells which show steady discharge throughout the hold period in proportion to the active force. Each region also has cells with unique firing properties. A large proportion of motor units show decrementing discharge, which decreases gradually through the hold period. The RM population is unique in having cells that fire during both flexion and extension. In particular, an unmodulated group of RM cells shows steady discharge during active movement that is unmodulated with the task [2]. Other response patterns have been observed in additional cells that do not facilitate motoneurons. In cerebral cortex many non-CM cells fire phasically at onset of movement and many of these fire bidirectionally with flexion and extension.

2. DYNAMIC NETWORK MODELS

To investigate the possible functional role of these cells and to determine whether other types of discharge patterns might be required to transform a step signal to the observed output of motoneurons, we developed a model that can incorporate these firing patterns. The network utilizes the back-propagation algorithm of the temporal flow model developed by Watrous [7], and is similar to the dynamic network algorithms recently described by Williams and Zipser [8]. These networks allow arbitrary interconnections between all elements; thus, in addition to feed-forward connections, they also permit cross-connections within layers as well as feedback connections. Secondly, the networks incorporate dynamic time-varying activity of units, representing the firing rates of neurons. In our simulations we have used as outputs the averaged firing rates of motor units recorded in monkeys performing a step-tracking task [5].

The input-output function of each unit is the standard sigmoidal function, with an offset to assure that units generate negligible output in the absence of input to the unit. The input consists of the summed activation of all other cells connected to the unit times their synaptic weight; a source of steady input may also be derived from a bias element. The synaptic weights are initially assigned randomly, and then modified by the back-propagation algorithm to reduce the error for the specified target output activations.

Figure 1 shows a neural network which simulates the input-output transformation performed by a monkey during the step-tracking task. The monkey sees a step change in target position, alternating between flexion and extension target zones. This is represented by sustained step input to the network for both flexion (fs) and extension (es). Since many visual cells discharge transiently, we also provided brief transient input at the onset of each target change (fb & eb). The network transforms these input signals to the observed response patterns of motor units at the output. The four types of motor unit patterns observed experimentally -- tonic, phasic-tonic, decrementing and phasic -- are generated for both flexor and extensor movements (ft to ep). The intervening hidden units consist of twelve excitatory and twelve inhibitory neurons (a and b units, respectively). The squares in the matrix symbolize the strength of the

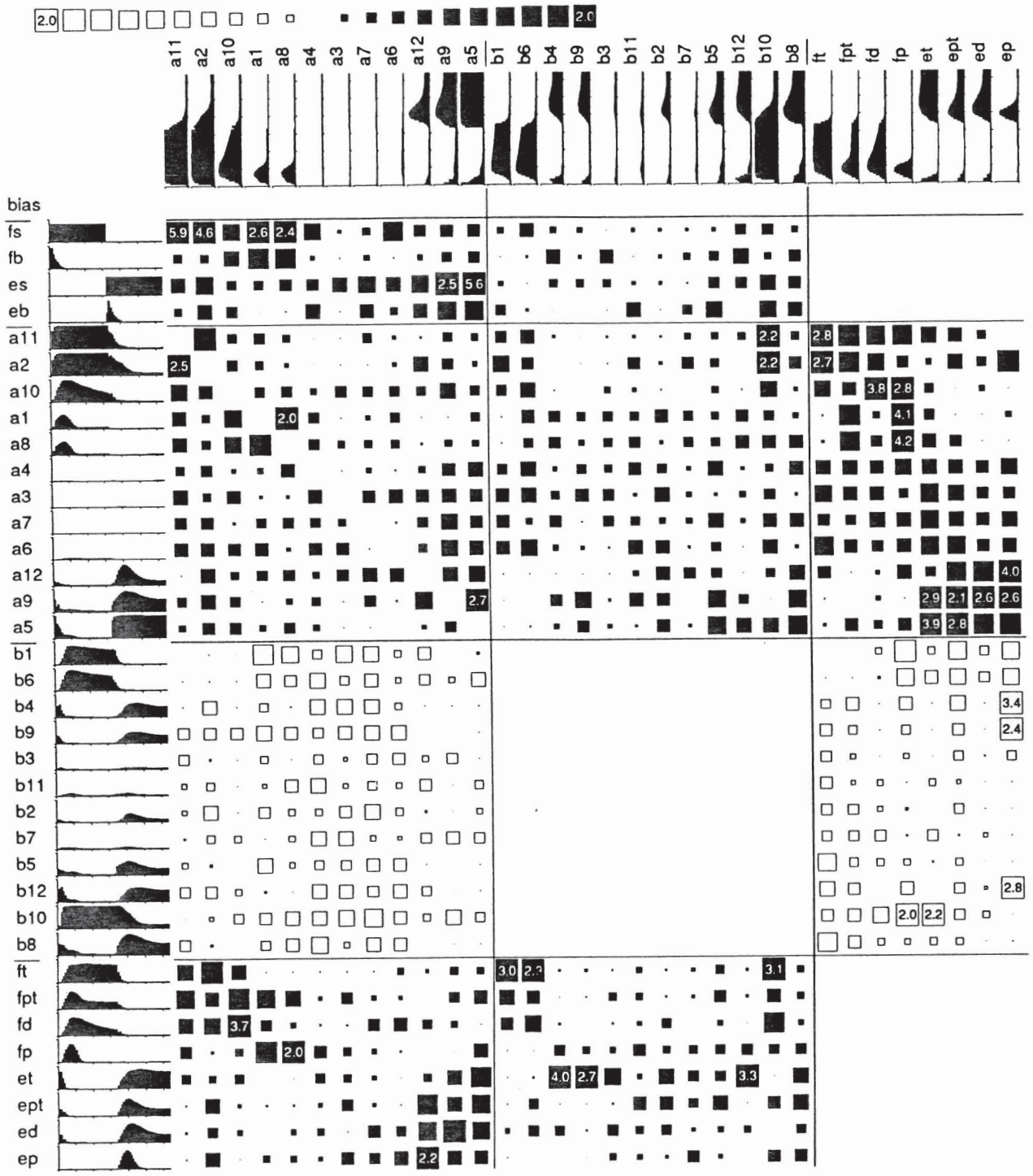

Fig. 1. Network transforming step and transient inputs to firing patterns of motor units at output. Unit activations during a flexion-extension cycle are shown at left and along the top. The weight matrix gives the strength of connections from the row to column units (scale at top). The rows represent, from top to bottom, the bias (which was eliminated for this simulation), the inputs (fs to eb), the excitatory hidden units (a's), inhibitory hidden units (b's) and the output flexor and extensor motor units (ft to ep). The target output patterns are tonic (ft & et), phasic-tonic (fpt & ept), decrementing (fd & ed) and phasic (fp & ep).

synaptic connection from the row unit to the column unit. Black squares designate excitatory weights and open squares represent inhibitory weights. The size of each square is proportional to the strength of the connection, except those which exceed the calibrated scale are designated numerically. Self-recurrent connections (corresponding to weights on the diagonal) were excluded. The excitatory hidden units are connected to each other, to the inhibitory group and to the output cells. The inhibitory hidden units connect to the excitatory units and to the output cells. There are also feedback connections from the output units back to the hidden units.

Figure 1 illustrates the weight matrix and the activation patterns of all units after 2000 training iterations, which was sufficient to produce the eight different output patterns. The discharge pattern of each unit is shown along the left, next to the row of output weights of that unit, and is shown again at the top of the column of weights representing the input connections to that unit. To better visualize the relationships between units, the hidden units were further sorted in order of the strength of their contribution to the phasic-tonic output units. The sorting algorithm used the product of activation and weight to the flexion phasic-tonic motor unit minus activation times weight to the extension phasic-tonic unit. Thus, the first hidden unit (a11) makes the largest relative contribution to the flexion phasic-tonic output unit (fpt). This hidden unit also developed the strongest weights to the flexion tonic output unit (ft), and also contributed connections to the other flexor units. Such divergent connections to different motor units, as well as to synergist muscles are representative of CM cells [1].

The activation patterns of the hidden units show several interesting features. The discharge patterns in the hidden units involve some recognizable variants of the output patterns, i.e., tonic, phasic, phasic-tonic, and decrementing patterns. Secondly, although the activation profiles of the target motor units are identical for the flexion and the extension groups, the network solution involves a different assignment of hidden units devoted to each. There are more excitatory and fewer inhibitory hidden unit activations related to flexion than to extension, yet they produce essentially identical output effects. Physiological experiments have revealed similar asymmetries between flexor and extensor related premotoneuronal cells [2].

Another striking feature of the connections are the preferentially strong connections within the sets of units with sustained and transient activity. Thus, the first two flexor hidden units exhibiting tonic activity (a11 & a2) are strongly interconnected with each other and receive potent input from the flexion step and connect strongly to the tonic output unit. Similarly, the transient input (fb) is most strongly connected to the phasic hidden units (a1 & a8), which are strongly interconnected with each other and which also have strong reciprocal connections with the phasic output unit (fp).

Despite this tendency for sustained and transient signals to propagate through segregated pathways, most cells receive a mixture of phasic and tonic input signals. For example, the phasic flexor motor unit (fp) receives input not only from the phasic hidden units (a1 & a8), but also from cells with sustained activity (a11 & a10); indeed, some of its phasic activity is derived from the difference between excitatory and inhibitory tonic cells with different onset times (e.g., a11 and b1). Such a subtraction of a delayed tonic pattern is used to produce phasic output in network simulations which receive only step inputs [4].

We expected the network to develop reciprocal inhibition between the flexion and extension groups. Although a few inhibitory units activated during one phase of movement (e.g. extension) do affect primarily reciprocally activated motor units (e.g., b8 & b5), just as many inhibit coactivated motor units (e.g., b12). Interestingly, the major drive on the tonic inhibitory units with connections on synergistic motor units is derived

from recurrent feedback from the motor units (e.g., et to b4 & b9, which in turn inhibit ep). This recurrent feedback makes these inhibitory hidden units more analogous to the Renshaw cells of the spinal cord than to the Ia inhibitory neurons. Another group of inhibitory hidden units develop connections to both flexor and extensor motor units (b1 & b10), a patterns that has no known correlate in physiological studies.

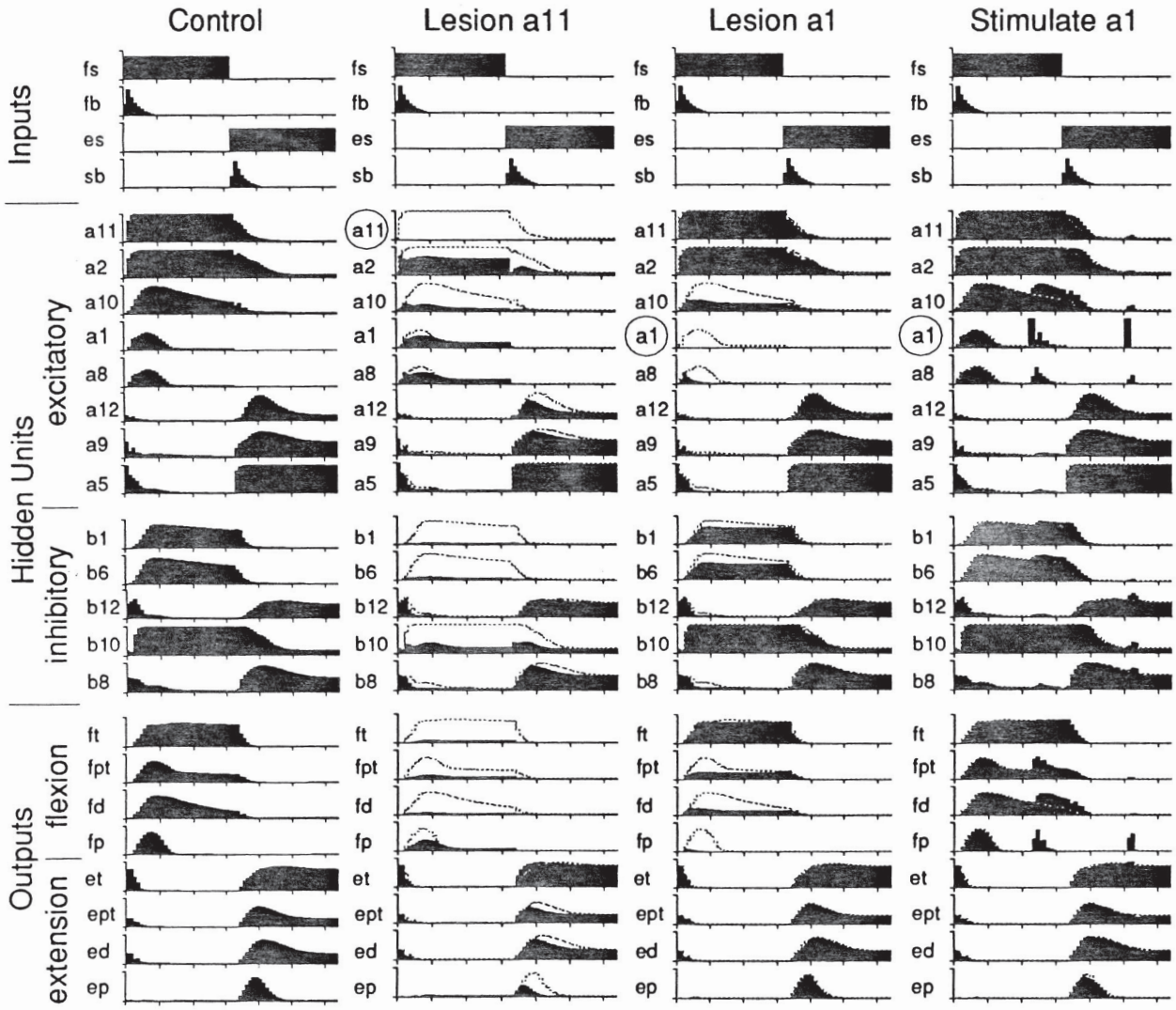

Fig. 2. Effect of lesioning and stimulating specific hidden units in the network in Fig. 1. The activations of representative units are shown for the unmanipulated, intact network (control, first column); after lesioning the tonic excitatory hidden unit a11 (second column); after lesioning the phasic excitatory hidden unit a1 (third column); and after stimulating the phasic excitatory hidden unit a1 (fourth column). The modified activations are shown superimposed on a stippled profile of the control activations.

3. MANIPULATION OF HIDDEN UNITS

3.1 LESIONS

The function of hidden units in the network can be tested by making selective lesions -- i.e., by eliminating the activation of particular units and analyzing the behavior of the remaining network. Fig 2 illustrates the effects of removing one of the tonic hidden units (a11) and one of the phasic hidden units (a1). The control activations of the units in the intact network are shown in the first column. A lesion of the tonic hidden unit a11 (second column) eliminates much of the tonic activity of the output and of the other hidden units. In contrast, lesioning the phasic unit (third column) eliminates the phasic activity of the output flexor units (fpt, fd and fp), as well as eliminating activity of the other phasic hidden unit (e8). The effects of these lesions are quite substantial because the network has relatively few units carrying a particular pattern, and these are strongly interconnected.

3.2 STIMULATION

The output effects of a given unit can also be tested by delivering a simulated stimulus and analyzing the propagated network response. The fourth column in Fig. 2 shows the effect of brief activation pulses delivered to a1 during the flexion and extension phase of the activity. The second pulse, during extension, evokes a response in the phasic output unit as well as in some of the active inhibitory hidden units.

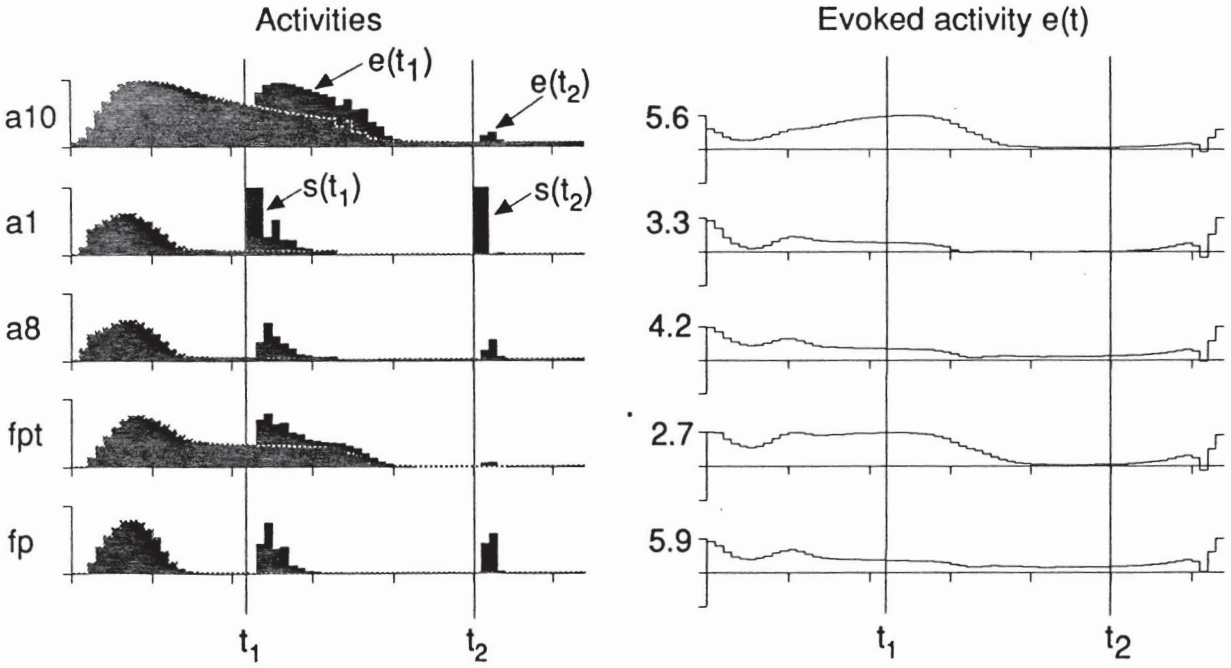

Fig. 3 Modulation of stimulus-evoked activity during the movement cycle. Left: unit a1 stimulated with a pulse during flexion (at time t_1) and extension (t_2). Response in three representative hidden units (a10, a1 & a8) and two flexion motor units (fpt & fp). The evoked response e(t) was calculated as the difference between stimulated and control activation. Right: Magnitude of evoked response plotted as a function of stimulus delay in the cycle.

In contrast, the first pulse, delivered during the flexion phase, evokes a stronger response in the phasic output unit, as well as more prolonged responses in the phasic-tonic and decrementing units; it also evokes activity in some of the excitatory hidden units.

The appearance of larger evoked responses in certain phases of the movement than in others is clearly related to the gating function of the activation of the intervening units. Such modulation of evoked responses is well-known in motor systems physiology [6]. To investigate this phenomenon more systematically, we delivered stimulus pulses at successive times during the movement cycle and measured the evoked response, defined as the difference between the stimulated and control activation (Fig. 3). Plotting the evoked response as a function of time in the flexion-extension cycle revealed modulations that were not simply proportional to the activation of the stimulated or target cell, but were more complex functions of the activations of the intervening units. The units with sustained activation patterns, such as a10, tended to exhibit more prolonged responses to the pulse than units with transient activity. In all cases the magnitude of the response evoked from this flexion unit was enhanced during the flexion period.

4. CONCLUDING COMMENTS

These preliminary observations suggest that dynamic network models may provide a useful tool for simulating neural mechanisms that generate patterns of activity in motor systems. Although these networks are still quite artificial in their connectivity, they develop many of the properties of biological networks [4]. Among the less realistic features of the present simulation are the ubiquitous recurrent interconnections of the hidden units. Such connectivity can lead to solutions in which relatively few interconnected units carry representative signals. It should be noted that such units could be interpreted as representing a larger population of neurons, and their connection weights interpreted as being proportional to the size of the population. Although these networks are highly abstracted, by incorporating profiles of physiological activity and by observing more realistic anatomical connectivity, we believe these models can be constrained to produce plausible simulations of sensorimotor integration.

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