

Adaptive vector integration to endpoint: Self-organizing neural circuits for control of planned movement trajectories

Paolo Gaudiano * and Stephen Grossberg **

Boston University, Boston, USA

Abstract

Gaudiano, P. and S. Grossberg, 1992. Adaptive vector integration to endpoint: Self-organizing neural circuits for control of planned movement trajectories. *Human Movement Science* 11, 141–155.

A neural network model is described for adaptive control of arm movement trajectories during visually guided reaching. The model clarifies how a child, or infant robot, can learn to reach for objects that it sees. Piaget has provided basic insights with his concept of a *circular reaction*. As an infant makes internally generated hand movements, the eyes automatically follow this motion. A transformation is learned between the visual representation of hand position and the motor representation of hand position. Learning of this transformation eventually enables the child to accurately reach for visually detected targets. Grossberg and Kuperstein (1989) have shown how the eye movement system can use visual error signals to correct movement parameters via cerebellar learning. Here it is shown how the arm movement system can endogenously generate movements which lead to adaptive tuning of arm control parameters. These movements also activate the target position representations that are used to learn the visuo-motor transformation that controls visually guided reaching. The arm movement properties obtain in the Adaptive Vector Integration to Endpoint (AVITE) model an adaptive neural circuit based on the VITE model for arm and speech trajectory generation of Bullock and Grossberg (1988a).

* Supported in part by the National Science Foundation (NSF IRI-87-16960).

** Supported in part by the Air Force Office of Scientific Research (AFOSR, 90-0175), DARPA (AFOSR 90-0083) and the National Science Foundation (NSF IRI-87-6960).

We wish to thank Carol Yanakakis Jefferson and Cynthia E. Bradford for their valuable assistance in the preparation of the manuscript.

Requests for reprints should be sent to S. Grossberg, Center for Adaptive Systems, Boston University, 111 Cummington Street, Boston, MA 02215, USA.

Self-organization of intermodal and intramodal maps for visually guided reaching

This article describes self-organizing neural circuits for the control of planned arm movements during visually guided reaching. The problem that motivates our results concerns the issue: how does a child learn to reach for objects that it sees? This problem requires understanding the interactions between two distinct modalities: vision (seeing an object) and motor control (moving a limb). In particular, we need to characterize the self-regulating mechanisms whereby an individual can stably learn transformations within and between the two different modalities that are capable of controlling accurate goal-oriented movements. The behavioral events that enable such learning to occur were called a *circular reaction* by the Swiss psychologist Jean Piaget (1963).

The circular reaction is an autonomously controlled behavioral cycle with two components: *production* and *perception*, with learning linking the two modalities to enable sensory-guided action to occur. Such a circular reaction is *intermodal*; that is, it consists of the coupling of two systems operating in different modalities. In order for the intermodal circular reaction to generate stable learning of the parameters that couple the two systems, the control parameters within each system must already be capable of accurate performance. Otherwise, performance may not be consistent across trials and a stable mapping could not be learned between different modalities. Thus it is necessary to self-organize the correct *intramodal* control parameters before a stable *intermodal* mapping can be learned.

Grossberg and Kuperstein (1986, 1989) have modelled how such intramodal control parameters can be learned within the eye movement system. During early development, eye movements are made reactively in response to visual inputs. When these eye movements do not lead to foveation of the visual target, the nonfoveated position of the target generates a visual error signal. The model suggests how such error signals can be used by the cerebellum to learn eye movement control parameters that lead to accurate foveations.

Here we show how the arm movement system can endogenously generate movements during a 'motor babbling' phase. 'Motor babbling' describes the spontaneous arm movements of an infant during an early developmental phase. These movements help to generate the data needed to learn correct arm movement control parameters. These

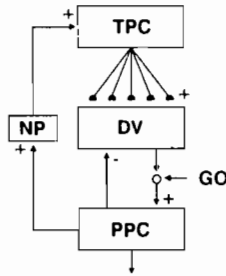


Fig. 1 A schematic diagram of the Adaptive VITE (AVITE) circuit. The Now Print (NP) gate copies the PPC into the TPC when the arm is stationary and the plastic synapses (semicircles in the $TPC \rightarrow DV$ pathways) learn to transform target commands into correctly calibrated outflow signals at the PPC.

endogenously generated arm movements also activate the target position representations that are used to learn the visuo-motor transformation that controls visually guided reaching.

Our results are developed within a model that we call the AVITE model (fig. 1) for variable-speed adaptive control of multi-joint limb trajectories.

Trajectory formation as an emergent invariant

Bullock and Grossberg (1988a) have suggested that trajectory formation is an emergent invariant that arises through interactions among two broad types of control mechanisms: planned control and automatic control. Planned control variables include (1) target position, or where we want to move; and (2) speed of movement, or how fast we want to move to the desired position, and the 'will' to move at all. Automatic control variables compensate for (3) the present position of the arm; (4) unexpected inertial forces and external loads, and (5) changes in the physiognomy of the motor plant, say due to growth, injury, exercise, and aging.

The VITE model of Bullock and Grossberg implements such a strategy of trajectory control and has been used to explain a large behavioral and neurobiological data base (see Bullock and Grossberg 1988a, 1988b, 1989, 1991). The model clarifies how motor synergies can be dynamically bound and unbound in real-time, and how multiple joints within a synergy can be synchronously moved at variable speeds.

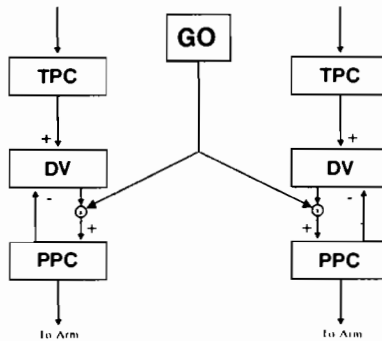


Fig. 2. The VITE model, adapted from Bullock and Grossberg (1988a). TPC = Target Position Command, DV = Difference Vector, PPC = Present Position Command. The GO signal acts as a nonspecific multiplicative gate that can control the overall speed of a movement, or the will to move at all. Use of a single GO signal insures synchronous activation of all muscles in the synergies involved in a coordinated movement.

The synchrony with which different muscles of a synergy contract by different amounts in equal time emerges from the interactive dynamics of the network; it is not externally controlled or programmed into the network.

The VITE model

Fig. 2 summarizes the main components of the VITE circuit. At the top of the figure, inputs to the Target Position Command (TPC) populations represent the desired final position of the arm. At the bottom of the figure, the Present Position Command (PPC) populations code an internal representation of where the arm actually is. Outflow movement commands to the arm are generated by the PPC. These outflow signals, supplemented by spinal circuitry and cerebellar learning (Bullock and Grossberg 1989, 1991) move the hand to the location relative to the body that is coded by the PPC.

Signals from the TPC and the PPC enable the Difference Vector (DV) populations to continuously compute the discrepancy between present position (PPC) and desired position (TPC). DV activation is integrated by the PPC until the latter becomes equal to the TPC, at which time the DV will be equal to zero and PPC integration stops. Hence the VITE circuit embodies an automatic process that moves the

PPC continuously to the TPC. The Adaptive VITE (AVITE) model presented herein explains how 'motor babbling' endogenously generates PPC representations that move the arm through a full range of positions, and activate TPCs whose signals to the DV are adaptively tuned to be dimensionally consistent with the corresponding PPCs, by using the DVs as source of error signals during learning.

Coding movement speed and intentionality: The GO signal

If the PPC were always allowed to integrate the DV, then a movement would begin as soon as the TPC becomes active. Somehow it must be possible to 'prime' a target position without moving the arm until another signal indicates the intent to carry out the movement. A related issue concerns how the overall speed of a movement can be varied without changing the desired TPC. 'Priming' denotes the limiting case of zero speed.

Trajectory-preserving speed control can be achieved by multiplying the output of the DV with a nonspecific gating signal. This is the GO signal depicted in fig. 2. Because of its location within the VITE model, the GO signal affects the rate at which the PPC is continuously moved toward the TPC, without altering the resulting trajectory.

For example, as long as the GO signal is zero, instatement of a TPC generates a nonzero DV, but the PPC remains unaltered. This 'primed' DV codes the difference between the arm's present position and desired position. When the GO signal is nonzero, the DV is integrated by the PPC at a rate proportional to the product $(DV) \cdot (GO)$. Integration ceases when the PPC equals the TPC and the DV equals zero, even if the GO signal remains positive. Other things being equal, a larger GO signal causes the PPC to integrate at a faster rate, so the same target is reached in a shorter time.

The synchrony of synergetic movement control by a VITE circuit is preserved in response to an arbitrary GO signal, and the main qualitative properties of VITE-controlled velocity profiles are preserved in response to a wide class of increasing GO signals (Bullock and Grossberg, 1988a). The model's prediction of a reversal in the direction of velocity profile asymmetry with increasing speed was confirmed in an explicit test by Nagasaki (1989), and its prediction of a late-acting

execution-gating signal was confirmed in an explicit test by DeJong et al. (1990).

Autonomous learning of VITE coordinates

In order for the VITE model to generate correct arm trajectories, the TPC and PPC must be able to activate dimensionally consistent signals $TPC \rightarrow DV$ and $PPC \rightarrow DV$ for comparison at the DV. There is no reason to assume that the gains, or even the coordinates, of these signals are initially correctly matched. Learning of an adaptive coordinate transformation is needed to achieve self-consistent matching of TPC- and PPC-generated signals at the DV.

In order to learn such a transformation, TPCs and PPCs must simultaneously be activated that represent the same target positions. This cannot be accomplished by activating a TPC and then letting the VITE circuit integrate the corresponding PPC. Such a scheme would beg the problem being posed; namely, to discover how $TPC \rightarrow DV$ and $PPC \rightarrow DV$ signals are calibrated so that a TPC *can* generate the corresponding PPC. An analysis of all the possibilities that are consistent with VITE constraints suggests that PPCs are generated by internal, or endogenous, activation sources during a motor babbling phase. After such a babbled PPC is generated and a corresponding action taken, the PPC is itself used to activate a TPC which *a fortiori* represents the same target position. This occurs via pathway $PPC \rightarrow NP \rightarrow TPC$ in fig. 1 (NP = Now Print gate). Thus motor babbling samples the work space and, in so doing, generates a representative set of pairs (TPC, PPC) for learning the VITE coordinate transformation.

Associative learning from parietal cortex to motor cortex during the motor babbling phase

Further analysis suggests that the only site where an adaptive coordinate change can take place is at the synaptic junctions that connect the TPC to the DV. These junctions are represented as semi-circular synapses in fig. 1. Moreover, the DV represents an internal measure of error, in the sense that miscalibrated signals $TPC \rightarrow DV$ and $PPC \rightarrow DV$ of TPCs and PPCs that correspond to the same target

position will generate a nonzero DV. Learning is designed to change the synaptic weights in the pathways $TPC \rightarrow DV$ in a way that drives the DV to zero. After learning is complete, the DV can only equal zero if the TPC and PPC represent the same target position. If we accept the neural interpretation of the TPC as being computed in the parietal cortex (Anderson et al. 1985; Grossberg and Kuperstein 1986, 1989) and the PPC as being computed in the motor cortex (Bullock and Grossberg 1988a; Georgopoulos et al. 1982, 1984, 1986), then we are led to predict that associative learning from parietal cortex to motor cortex takes place during motor babbling along a possibly multi-synaptic pathway, and attenuates activation of the difference vector cells in the motor cortex during postural intervals. The GO signal shares properties with cells in the globus pallidus (Bullock and Grossberg 1989, 1991; Horak and Anderson 1984a, 1984b).

Vector associative map: On-line DV-mediated learning and performance

When such a learning law is embedded within a complete AVITE circuit, the DV can be used for on-line regulation of both learning and performance. During a performance phase, a new TPC is read into the VITE circuit from elsewhere in the network, such as when a reaching movement is initiated by a visual representation of a target. The new DV is used to integrate a new PPC that represents the same target position as the TPC. Zeroing the DV here creates a new PPC while the TPC is held constant. During the learning phase, the DV is used to drive a coordinate change in the $TPC \rightarrow DV$ synapses. Zeroing the DV here creates new adaptive weights while both the PPC and TPC are held fixed.

Both the learning and the performance phases use the same AVITE circuitry, notably the same DV, for their respective functions. Thus learning and performance can be carried out on-line in a real-time setting, unlike schemes like back propagation. The operation whereby an endogenously generated PPC activates a corresponding TPC, as in fig. 1, 'back propagates' information for use in learning, but does so using local operations without the intervention of an external teacher or a break in on-line processing.

Autonomous control, or gating, of the learning and performance phases is needed to achieve effective on-line dynamics. For example,

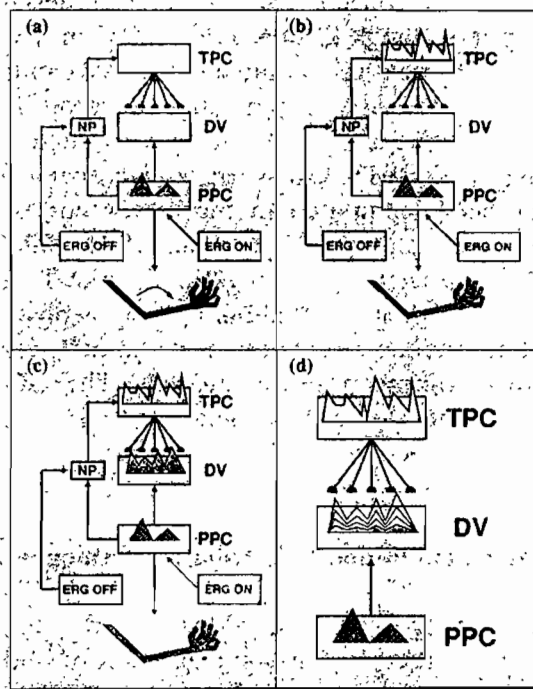


Fig. 3. A diagrammatic illustration of a single babbling cycle in the AVITE. (a) The Endogenous Random Generator ON channel output (ERG ON) is integrated at the PPC, giving rise to random outflow signals that move the arm. (b) When the arm stops moving at ERG ON offset, a complementary ERG OFF signal opens the Now Print (NP) gate, copying the current PPC into the TPC through an arbitrary transformation. (c) The filtered TPC activation is compared to the PPC at the DV stage. DV activation would be zero in a properly calibrated AVITE. (d) The learning law changes $TPC \rightarrow DV$ synapses to eliminate any nonzero DV activation, thus learning the reverse of the $PPC \rightarrow NP \rightarrow TPC$ transformation.

the network needs to distinguish whether $DV \neq 0$ because the TPC and PPC represent different target positions, or because the $TPC \rightarrow DV$ synapses are improperly calibrated. In the former case, learning should not occur; in the latter case, it should occur. Thus some type of learning gate is needed to prevent spurious associations from forming between TPCs and PPCs that represent different target positions. The design of the total AVITE network shows how such distinctions are computed and used for real-time control of the learning and performance phases. We now explain how this is accomplished.

The motor babbling cycle

During the motor babbling stage, an Endogenous Random Generator (ERG) of random vectors is activated. These vectors are input to the PPC stage, which integrates them, thereby giving rise to outflow signals that move the arm through the workspace (fig. 3a). After each interval of ERG activation and PPC integration, the ERG *automatically* shuts off, so that the arm stops at a particular target position in space.

Offset of the ERG opens a Now Print (NP) gate that copies the PPC into the TPC through some fixed transformation (fig. 3b). The top-down adaptive filter from TPC to DV learns the correct reverse transformation (fig. 3c) by driving the DV toward zero while the NP gate is open (fig. 3d).

Then the cycle repeats itself automatically. When the ERG becomes active again, it shuts off the NP gate and thus inhibits learning. A new PPC vector is integrated and another arm movement is elicited. The ERG is designed such that, across the set of all movement trials, its output vectors generate a set of PPCs that form an unbiased sample of the workspace. This sample of PPCs generates the set of (TPC, PPC) pairs that is used to learn the adaptive coordinate change $TPC \rightarrow DV$ via a vector associative map.

The endogenous random generator of workspace sampling bursts

The ERG design embodies an example of opponent interactions (fig. 4). The motor babbling cycle is controlled by two complementary phases in the ERG mechanism: an *active* (ERG ON) and a *quiet* (ERG OFF) phase. The active phase generates random vectors to the PPC. During the quiet phase, input to the PPC from the ERG is zero, thereby providing the opportunity to learn a stable (TPC, PPC) relationship. In addition, there must be a way for the ERG to signal onset of the quiet phase, so that the NP gate can open and copy the PPC into the TPC (fig. 3b). The NP gate must not be open at other times: if it were always open, any incoming commands to the TPC could be distorted by contradictory inputs from the PPC. Offset of the active ERG phase is accompanied by onset of a complementary mechanism whose output energizes opening of the NP gate. The signal that opens

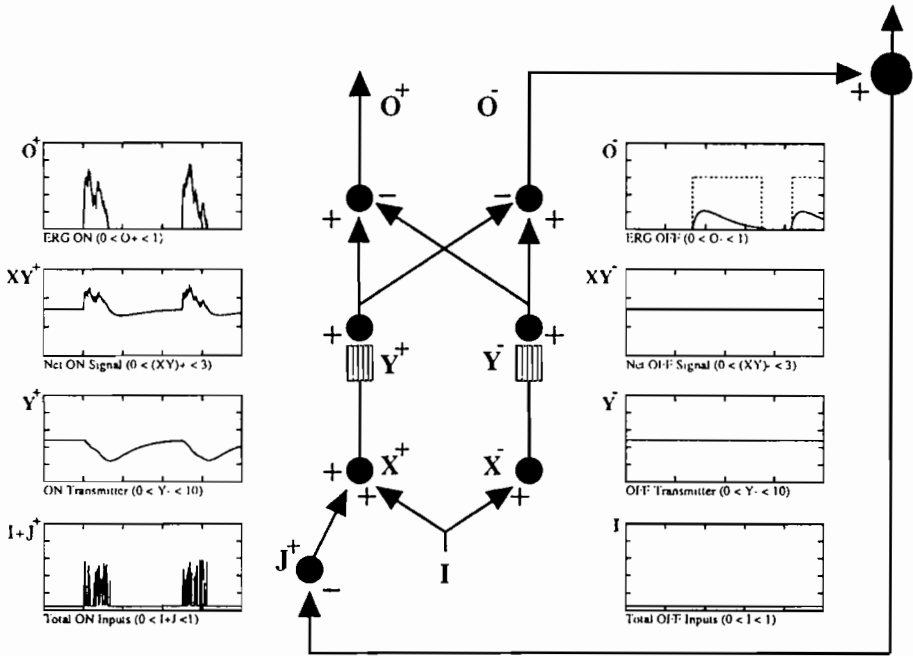


Fig. 4. Response of various ERG levels to a continuous differential input J^+ . An inverted-U transfer function through the chemical gates (rectangular synapses) leads to a transient ON response (O^+), followed by activation of the OFF channel (O^-). Sufficient OFF channel activation energizes the Pauser Gate (PG), which shuts off phasic input J^+ to the ON channel, causing a larger, transient rebound in the OFF channel. Removal of the phasic input allows ON channel transmitter Y^+ to replenish, eventually shutting off the PG and starting a new cycle. The ON channel output is choppy due to noisiness of phasic input J^+ . Dashed lines in upper right-hand plot represent PG activation (not drawn to scale).

the NP gate can also be used to modulate learning in the adaptive filter. No learning should occur except when the PPC and TPC encode the same position.

Fig. 4 provides a schematic diagram of the ERG circuit. The design is a specialized gated dipole (Grossberg 1972, 1982, 1984). A gated dipole is a neural network model for the type of opponent processing during which a sudden offset of one channel can trigger activation, or antagonistic rebound, within the opponent channel. Habituating transmitters in each opponent channel regulate the rebound property by multiplying, or gating, the signals in their respective channel.

In the present application, note the complementary time intervals during which the ON and OFF channels of the ERG are active. The

ON channel output is different during each active phase, leading to integration of PPCs that sample the workspace. In contrast, OFF channel activation is fairly uniform across trials, thereby providing intervals during which learning can stably occur.

The basic gated dipole circuit needs to be specialized to design an effective ERG circuit. Such an ERG circuit needs to convert an undifferentiated stream of random inputs to the ON channel into cyclic output bursts from the ON channel, interspersed with OFF intervals whose duration is relatively stable across trials, as in the computer simulation illustrated in fig. 4. In order to convert a stream of random inputs into a series of output bursts, activation of the ON channel must initiate a process that spontaneously terminates ON channel output due to transmitter habituation even while the random inputs remain on. In addition, after the ON channel shuts off, it must be able to spontaneously recover, even while its random inputs remain on, so that it can generate the next output burst.

In order to achieve cyclic output bursts from the ON channel, its chemical transmitter must be allowed to recover from habituation during its quiet phase. In order for this to happen, the random input stream to the ON channel must be blocked during its quiet phase. Our solution is to let the OFF activation actively gate shut the source of phasic input, so that the ON transmitter can recover from habituation. This process is represented in fig. 5 as a feedback pathway from the OFF channels of the ERG to the input source through a gate labelled PG that sums all OFF inputs.

Some results: correct parameter learning through motor babbling

This section provides a qualitative overview of the major results obtained through computer simulation of the AVITE model during its movement and learning phases. Fig. 5 is a schematic diagram of the complete system used in the simulations described below to control a two-jointed arm. Each AVITE module consists of one agonist channel and one antagonist channel, coupled in a push-pull fashion. Each channel receives inputs from its own ERG circuit. All ERG OFF channels cooperate to activate the PG gate. Fig. 6 summarizes a computer simulation that illustrates the relatively uniform distribution of endogenously generated arm positions. Each dot on the scatterplot

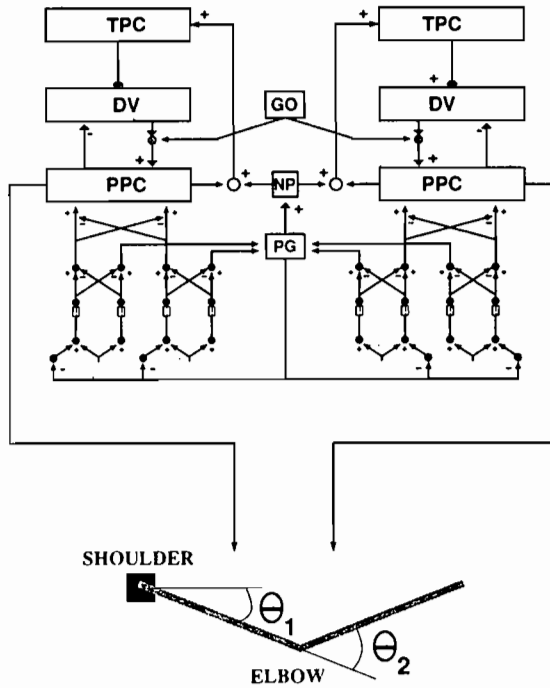
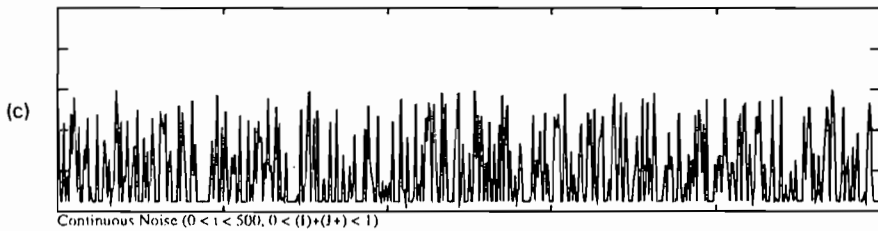
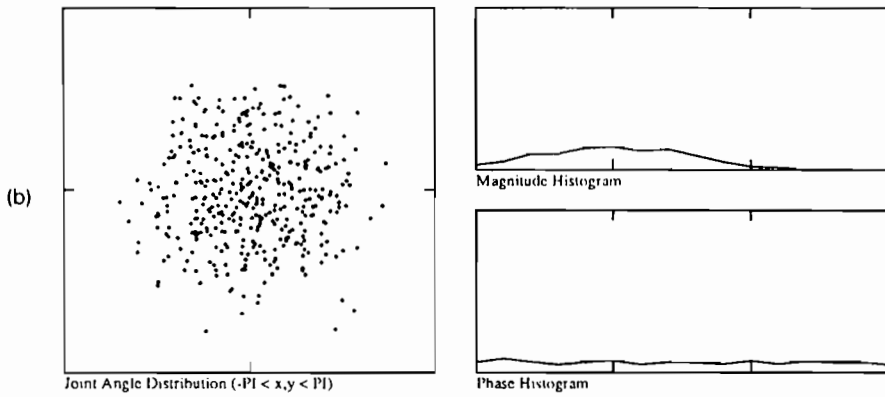
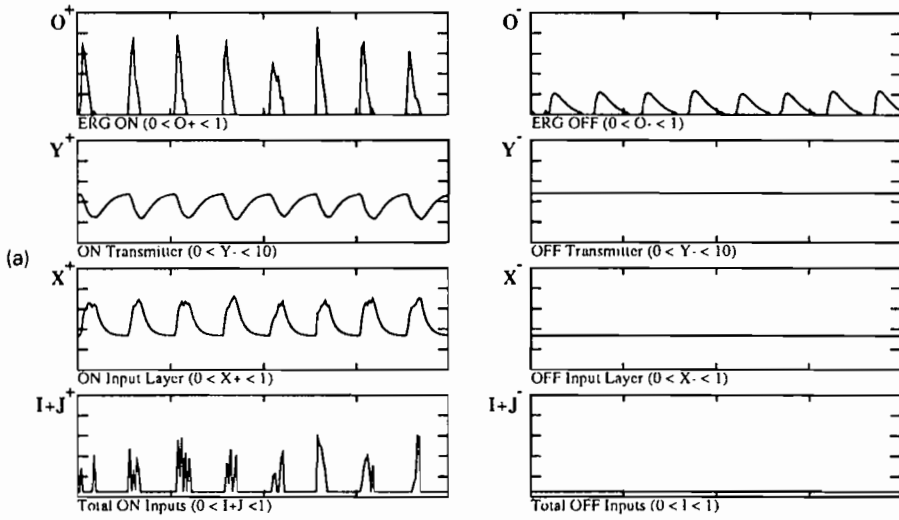


Fig. 5. Diagram of the complete ERG-AVITE system used for the two-joint simulations. Each AVITE agonist-antagonist module is driven by two ERG modules. AVITE outflow commands control movement of a simulated two-joint system. The GO signal, NP gate and PG are the same for all modules to ensure synchronous movement and learning for both synergies.

Fig. 6. Computer simulation of ERG-AVITE dynamics: (a) The time course behavior of four state variables in the ON (left) and OFF (right) ERG channels during 2,000 steps of the simulation. The range of each plot is indicated in parentheses under the abscissa. From top to bottom: ERG outputs (O^+ , O^-), available transmitter (Y^+ , Y^-), input layer activation (X^+ , X^-) and total input signal (I^+ , J^+ , I). Note that bottom three plots for OFF channel (right) are always constant. This is due to lack of phasic input to OFF channel. However, baseline activation is necessary to energize OFF channel rebounds (top right plot). (b) Distribution of joint angles (between $-\pi$ and π radians) attained during about 400 babbling movements (100,000 steps of the simulation). Left: each dot in the scatterplot represents the angle of the two joints (θ_1 , θ_2) during each quiet phase. Center point represents resting position. Right: histograms of the distribution of joint angles around resting position. Magnitude histogram represents the number of dots falling within each of 16 evenly spaced concentric rings about the center; the unimodal distribution toward the left side of the histogram indicates a tendency for less extreme joint angles. Phase histogram represents the number of dots at each of sixteen evenly spaced quadrants about the resting position; a flat phase histogram indicates a uniform distribution of joint angle combinations. (c) Representative sample of uninterrupted total input $I + J^+$ for 500 steps.



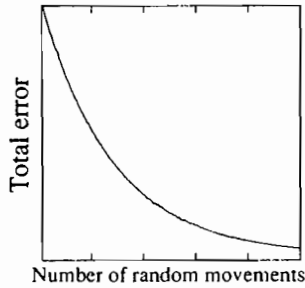


Fig. 7. Total DV error as a function of time.

of fig. 6b represents a particular arm configuration attained at the end of a single ERG active phase. The axes represent joint angle between $-\pi$ and π for each joint. Fig. 6b also provides histograms of the radial and angular distributions, respectively, of dots around the center of the scatterplot. Fig. 6c shows the random input that is converted into input bursts (fig. 6a) by the ERG. Fig. 7 illustrates a computer simulation showing the convergence of the learning process as motor babbling progresses. The plot computes the DV at the end of successive quiet phases, when the PPC equals the TPC. Learning successfully drives the DV to zero at an approximately exponential rate, that can be chosen very fast.

Concluding remarks

The AVITE model clarifies how motor babbling can sample the work space while generating Target Position Commands that can be used to learn correct *intramodal* arm trajectory control parameters and *intermodal* maps to control visually guided reaching. The mathematical analysis of how this is achieved will be described elsewhere (Gaudiano and Grossberg 1991). The Vector Associative Map, or VAM, by which the AVITE adaptive coordinate transformation is learned on-line promises to be useful in solving other problems of adaptive sensory-motor control and beyond.

References

- Anderson, R.A., G.K. Essick and R.M. Siegel, 1985. Encoding of spatial location by posterior parietal neurons. *Science* 230, 456-458.

- Bullock, D. and S. Grossberg, 1988a. Neural dynamics of planned arm movements: Emergent invariants and speed-accuracy properties during trajectory formation. *Psychological Review* 85, 49-90.
- Bullock, D. and S. Grossberg, 1988b. 'The VITE model: A neural command circuit for generating arm and articulator trajectories'. In: J.A.S. Kelso, A.J. Mandell and M.F. Shlesinger (eds.), *Dynamic patterns in complex systems*. Singapore: World Scientific Publishers.
- Bullock, D. and S. Grossberg, 1989. 'VITE and FLETE: Neural modules for trajectory formation and postural control'. In: W. Hershberger (ed.), *Volitional action*. Amsterdam: North-Holland. pp. 253-297.
- Bullock, D. and S. Grossberg, 1991. Adaptive neural networks for control of movement trajectories invariant under speed and force rescaling. *Human Movement Science* 10, 3-53.
- DeJong, R., M.G.H. Coles, G.D. Logan and G. Gratton, 1990. In search of the point of no return: The control of response processes. *Journal of Experimental Psychology: Human Perception and Performance* 16, 164-182.
- Gaudiano, P. and S. Grossberg, 1991. Vector associative maps: Unsupervised real-time error-based learning and control of movement trajectories. *Neural Networks* 4, 147-183.
- Georgopoulos, A.P., J.F. Kalaska, R. Caminiti and J.T. Massey, 1982. On the relations between the direction of two-dimensional arm movements and cell discharge in primate cortex. *Journal of Neuroscience* 2, 1527-1537.
- Georgopoulos, A.P., J.F. Kalaska, M.D. Crutcher, R. Caminiti and J.T. Massey, 1984. 'The representation of movement direction in the motor cortex: Single cell and population studies' In: G.M. Edelman, W.E. Goll and W.M. Cowan (eds.), *Dynamic aspects of neocortical function*. Neurosciences Research Foundations. pp. 501-524.
- Georgopoulos, A.P., A.B. Schwartz and R.E. Kettner, 1986. Neuronal population coding of movement direction. *Science* 233, 1416-1419.
- Grossberg, S., 1972. A neural theory of punishment and avoidance, II: Quantitative theory. *Mathematical Biosciences* 15, 253-285.
- Grossberg, S., 1982. *Studies of mind and brain: Neural principles of learning, perception, development, cognition and motor control*. Boston, MA: Reidel Press.
- Grossberg, S., 1984. Some normal and abnormal behavioral syndromes due to transmitter gating of opponent processes. *Biological Psychiatry* 19, 1075-1118.
- Grossberg, S. and M. Kuperstein, 1986. *Neural dynamics of adaptive sensory-motor control: Ballistic eye movements*. Amsterdam: Elsevier.
- Grossberg, S. and M. Kuperstein, 1989. *Neural dynamics of sensory-motor control: Expanded edition*. Elmsford, NY: Pergamon Press.
- Horak, F.B. and M.E. Anderson, 1984a. Influence of globus pallidus on arm movements in monkeys, I: Effects of kainic acid-induced lesions. *Journal of Neurophysiology* 52, 290-304.
- Horak, F.B. and M.E. Anderson, 1984b. Influence of globus pallidus on arm movements in monkeys, II: Effects of stimulation. *Journal of Neurophysiology* 52, 305-322.
- Nagasaki, H., 1989. Asymmetric velocity and acceleration profiles of human arm movements. *Experimental Brain Research* 74, 319-326.
- Piaget, J., 1963. *The origins of intelligence in children*. New York: Norton.