LETTER

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Computational Studies of Lateralization of Phoneme Sequence Generation

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The mechanisms underlying cerebral lateralization of language are poorly understood. Asymmetries in the size of hemispheric regions and other factors have been suggested as possible underlying causal factors, and the corpus callosum (interhemispheric connections) has also been postulated to play a role. To examine these issues, we created a neural model consisting of paired cerebral hemispheric regions interacting via the corpus callosum. The model was trained to generate the correct sequence of phonemes for 50 monosyllabic words (simulated reading aloud) under a variety of assumptions about hemispheric asymmetries and callosal effects. After training, the ability of the full model and each hemisphere acting alone to perform this task was measured. Lateralization occurred readily toward the side having larger size, higher excitability, or higher learning-rate parameter. Lateralization appeared most readily and intensely with strongly inhibitory callosal connections, supporting past arguments that the effective functionality of the corpus callosum is inhibitory. Many of the results are interpretable as the outcome of a “race to learn” between the model’s two hemispheric regions, leading to the concept that asymmetric hemispheric plasticity is a critical common causative factor in lateralization. To our knowledge, this is the first computational model to demonstrate spontaneous lateralization of function, and it suggests that such models can be useful for understanding the mechanisms of cerebral lateralization.

1 Introduction

A great number of functional cerebral asymmetries are currently recognized to exist in humans (Hellige, 1993; Kinsbourne, 1978; Springer & Deutsch, 1993). These cognitive and behavioral lateralizations include language, handedness, visuospatial processing, emotion and its facial expression, olfaction, and attention. For example, over 90% of the population has dominance of the left cerebral hemisphere for language. Much of the evidence for known qualitative and quantitative asymmetries comes from studies of deficits

in stroke and trauma patients, from special procedures applied to normal subjects and “split-brain” patients following commissurotomy (Gazzaniga, 1987), and from functional imaging studies of higher cortical functions and perceptual tasks (Mazziotta & Phelps, 1984; Peterson, Fox, Posner, Mintun, & Raichle, 1988; Reivich et al., 1984). There is also substantial plasticity of the brain with respect to functional asymmetries. For example, left hemispherectomy in infants or even children as old as 9 years of age can result in the right hemisphere’s becoming fairly skilled in language functions (Dennis & Whitaker, 1976; Vargha-Khadem et al., 1997). Lateralization of various functional brain asymmetries (motor preference, vocalizations, perception, etc.) is not uniquely human; it has been found repeatedly in numerous studies with rodents, birds, and primates (Hauser, 1993; Hellige, 1993; Ward & Hopkins, 1993).

Although such hemispheric specializations are well documented, the underlying causes of hemispheric function lateralization are not well understood and have been the subject of intense scientific investigation for over a century. Perhaps the most widely accepted theory is that hemispheric anatomical asymmetries are a critical factor in function lateralization. (Galaburda & Habib, 1987; Geschwind & Galaburda, 1987; Hellige, 1993). Anatomic and cytoarchitectonic asymmetries include, for example, a larger left temporal plane in 65% of subjects, an asymmetry that is present in newborns (Galaburda, Sanides, & Geschwind, 1978; Geschwind & Levitsky, 1968; Loftus et al., 1993; Witelson & Pallie, 1973). However, other significant asymmetries exist in the hemispheres. For example, higher-order dendritic branching is greater in speech areas of the left hemisphere (Scheibel, 1985), and evidence exists that there is more gray matter relative to white matter in the left hemisphere than in the right (Gur et al., 1980). Important neurotransmitters such as dopamine and norepinephrine are asymmetrically distributed between the hemispheres (Tucker & Williamson, 1984), while the threshold for motor-evoked potentials is lower for the left hemisphere (Macdonell et al., 1991). Substantial arguments have been advanced that it is unlikely that a single underlying hemispheric asymmetry will be able to account for language and other hemispheric specializations (Hellige, 1993).

Besides intrinsic hemispheric differences, another potential factor in function lateralization is hemispheric interactions via pathways such as the corpus callosum (Zaidel, 1983). Corpus callosum fibers are mostly homotopic; each hemisphere projects to the other in a topographic fashion so that roughly mirror-symmetric points are connected to each other (Innocenti, 1986; Pandya & Seltzer, 1986). Most neurons sending axons through the corpus callosum are pyramidal cells, and these synapse mainly on contralateral pyramidal cells over a cortical region of diameter up to 2 mm (Hartenstein & Innocenti, 1981; Innocenti, 1986). Such cellular components, as well as transcallosal diaschisis and split-brain experiments, suggest that transcallosal interhemispheric interactions are mainly excitatory in nature (Berlucchi, 1983), but this hypothesis is quite controversial (Denenberg, 1983). Tran-
scallosal monosynaptic excitatory postsynaptic potentials are subthreshold and of low amplitude, and are followed by stronger, more prolonged inhibition (Toyama, Tokashiki, & Matsunami, 1969), suggesting to some that transcallosal inhibitory interactions are much more important (Cook, 1986; Kinsbourne, 1978). The case for transcallosal inhibition has been strengthened recently by transcranial magnetic stimulation studies indicating that activation of one primary motor cortex inhibits the contralateral one (Ferbert et al., 1992; Meyer, Roricht, von Einseidel, Kruggel, & Weindl, 1995).

In spite of this wealth of information about hemispheric asymmetries and interactions, it is not yet clear what physiological mechanisms are responsible for individual or population lateralization of function (Reggia, 1995). While it is often argued that anatomical asymmetries underlie functional asymmetries such as language lateralization to the (usually) left hemisphere (Geschwind & Galaburda, 1987), the mechanisms by which this might occur are not known. Further, it is difficult to see how such anatomical asymmetries, favoring the left in roughly 65% of the population, could account for left-hemisphere dominance for language in over 90% of the population.

It seems likely that computational modeling could be useful for refining, extending, and examining the plausibility of various hypotheses concerning the mechanisms of hemispheric specialization. Although a few neural models of interacting hemispheric regions have been studied previously (Anninos, Argyrakis, & Skouras, 1984; Cook & Beech, 1990; Ringo et al., 1994), none of these past studies has directly looked at how lateralization of functions can arise spontaneously. In this article, our goal is to use computational modeling to investigate possible causes for lateralization of a single task. We do this by studying a recurrently connected neural model consisting of two simulated hemispheric regions interacting via a corpus callosum. In a series of simulations, the model was repeatedly trained to generate a temporal sequence of outputs when given a fixed input pattern (a simple caricature of single-word reading aloud). Each time the model was trained, we varied its assumptions about hemispheric asymmetry and callosal excitatory and inhibitory strength, determining which conditions resulted in function lateralization.

Our initial hypothesis was that each of the hemispheric asymmetries introduced into the model would produce functional lateralization when and only when inhibitory interhemispheric interactions were present. The actual situation turned out to be more interesting. Although functional lateralization usually occurred with underlying hemispheric asymmetries if the callosal connections were inhibitory, under certain conditions it would also develop with excitatory callosal connections. Further, lateralization secondary to hemispheric size asymmetry was found to have multiple causes. Many of the results we observed can be interpreted as a “race to learn” between the model’s two hemispheric regions, supporting the concept that asymmetric hemispheric plasticity is a common causative factor in lateralization.
The neural model is trained to take three-letter words (CAD, MOP, SIT, etc.) as input and to produce the correct temporal sequence of phonemes for the pronunciation of each word as output. For example, the single fixed input pattern MAT, the trained model’s output goes through a sequence of three states representing the phonemes for M, A, and T, one at a time. The 50 words used as inputs are listed in the appendix.

2.1 The Model. The architecture of the model is summarized schematically in Figure 1. While this model captures the direct transformation of input to output suggested by functional imaging studies of single-word reading (Peterson et al., 1988), the intent is not to create a veridical model of underlying neocortical structures. Rather, our goal is to represent the functionality of two interacting, recurrently connected pathways (left and right) as sequential output is generated. The model’s input elements (I) are divided into three groups, each corresponding to the possible input characters at one of the three input character positions. Input elements are fully connected to two sets of neural elements representing corresponding regions of the left (LH) and right (RH) hemisphere cortex. These regions are fully connected to each other via a simulated corpus callosum (CC), and they are also fully connected to a set of output neural elements (O) representing individual phonemes. A set of state elements (S) provides delayed feedback to the hemispheric regions via recurrent connections, similar to those used in Jordan (1986). These recurrent feedback connections are motivated in part by the recurrent or “backward” neuroanatomic connections between cortical regions (Felleman & Van Essen, 1991). The two hemispheric regions are taken to represent roughly mirror-image cortical regions, consistent with the fact that such regions are generally specialized for the same or a similar function (Heilman & Valenstein, 1979; Kupfermann, 1991).

During simulations, activation patterns representing written words are clamped on the input elements and held fixed while the network generates a sequence of outputs, as follows. With the onset of input, activation propagates forward to the two hemispheric regions, and then to the output elements, where ideally only the single element representing the correct first phoneme of the input word is activated. This output pattern activate the set of state elements (S), and then the network recomputes its output, generating the second phoneme in the word’s pronunciation sequence. In doing this, the activation levels of the elements in the hemispheric layers are now determined not only by the input activation pattern but also by the activation pattern of the state elements. The state elements provide one time unit of delay between output activity and its feedback via recurrent connections to the hemispheric regions. This process repeats, and a third output phoneme is produced.

The activation level $a_i$ of each neural element $i$ is set to zero at the begin-
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Figure 1: Model architecture. Individual neural elements are indicated by small circles; sets of related elements are indicated by boxes. I = inputs, O = outputs, LH/RH = left/right hemisphere regions, CC = corpus callosum, S = state elements.

The activation level of hemispheric element $i$ is governed by

$$\frac{da_i}{dt} = -a_i + M\sigma(h_i),$$

(2.1)

where $M$ is a constant maximum activation level, $h_i$ represents the linear weighted sum of input activation to element $i$ from inputs I, state elements S, and the opposite hemispheric region, and $\sigma$ is the logistic equation $\sigma(x) = (1 + e^{-x})^{-1}$. At equilibrium $a_i = M\sigma(h_i)$, so each hemispheric element effectively computes a sigmoid function of its input. The reason for iteratively computing $a_i$ (25 iterations, 0.1 time step, Euler method), rather than just assigning it a value $M\sigma(h_i)$, is to allow the hemispheric regions time to influence each other during a simulation via the corpus callosum. Output element activation levels are simply computed directly as $a_i = \sigma(h_i)$. State element $i$ is assigned the value of the corresponding output element plus a fraction of its previous value, that is,

$$a_i^s = \mu a_i^o + a_i^o$$

(2.2)

where superscript $s$ designates a state element and $o$ designates an output element.

The supervised learning rule used in the model is a variant of recurrent error backpropagation specifically designed for networks transforming a
single, fixed input pattern into a sequence of outputs (Jordan, 1986). The equations governing learning are briefly stated in the appendix. Learning occurs on all connections pictured in Figure 1 except those forming the corpus callosum (CC), and also on connections from a single bias unit to hemispheric and output elements (not shown in Figure 1). Learning was incremental, with weight updates occurring after each individual output. Weights modified by learning are initialized with uniform random real values between \(-1\) and \(+1\). Callosal weights are uniform, usually all having a constant value \(c\) during any given simulation. When \(c > 0\), positive feedback via the excitatory callosal connections can excessively activate hemispheric elements (i.e., all or most elements nearly maximally activated, greatly slowing learning). To avoid this, hemispheric elements have a “self-connection” that contributes \(-2c(1 + 2a_i)\) to \(h_i\) when \(c > 0\).

2.2 Experimental Methods. Using the model described, we undertook a series of simulations in which the assumptions about hemispheric asymmetries and the excitatory and inhibitory effects of the corpus callosum were systematically altered with each simulation. Five hemispheric asymmetries were examined: relative size, maximum activation level, sensitivity to input stimuli, learning-rate parameter, and amount of feedback. Asymmetries were examined one at a time in isolation. In addition, a model with symmetric hemispheric regions was used as a control. In most simulations, each hemispheric region was sufficiently large (six or more elements) that it could independently learn the input-output mapping. This was motivated by experimental evidence that either human hemisphere alone can acquire language (Dennis & Whitaker, 1976; Vargha-Khadra et al., 1997), and that function lateralization would trivially occur if one hemispheric region was too small to learn the mapping. For each hemispheric asymmetry examined and the control model, the uniform value \(c\) of callosal weights was varied over 17 values between \(-3.0\) and \(+3.0\). In this manner, over a thousand simulations were done, each involving training the model and then testing its performance.

Training data consisted of 50 three-letter words with their associated pronunciation sequence. These words were designed so that any single-output phoneme alone could never unambiguously predict the subsequent phoneme (e.g., an L in the first position could be followed by an A, I, or O). Thus, the feedback from state elements to hemispheric regions alone could not predict uniquely the subsequent correct output state. The baseline parameters, used in all simulations described below unless explicitly noted otherwise, are \(M = 1.0\), \(\mu = 0.5\), a learning rate of 0.05, and 10 elements per hemispheric region. All software is implemented in C, and most simulations were run on DEC Alphas.

In each simulation, model performance was measured as root mean square error \(E\). Training consisted of repeated passes through the training data until either \(E\) was reduced to 0.05 or 10,000 passes through the
data occurred. Error was measured prior to training and under three conditions after training: with both hemispheric regions connected to outputs (E), and with each of the left and right hemisphere regions alone connected to outputs (E_L and E_R, respectively). When only one hemispheric region was connected to the outputs, the other still had an indirect, albeit limited, influence on output via the corpus callosum. Lateralization was measured using an asymmetry coefficient $\rho_c$ (Lezak, 1995), specifically:

$$\rho_c = \frac{E_L - E_R}{1 - \frac{1}{2}(E_L + E_R)},$$

(2.3)

where $c$ indicates corpus callosum strength. Negative values of $\rho_c$ indicate left lateralization of function, positive values indicate right lateralization, and for the specific simulations done here, $\rho_c = \pm 0.6$ roughly corresponds to maximal or complete lateralization. We use $\bar{\rho}$ ($\bar{\rho}^+$, $\bar{\rho}^-$) to designate the mean value of $\rho_c$ for all $c$ (for $c \geq 1$, $c \leq -1$, respectively).

3 Results

3.1 Symmetrical Hemispheric Regions. Simulations were done with a symmetrical, control version of the model in which both hemispheric regions were identical except for random differences in initial connection weights. Figure 2a shows a representative example of the results using the baseline parameters (see above) but different random initial weights on each run. Lateralization is typically more pronounced for negative callosal strengths (mean $|\rho|$ of 0.21 for $c \leq -1$) than for positive callosal strengths (mean $|\rho|$ of 0.06 for $c \geq 1$). However, the direction of lateralization is arbitrary ($\bar{\rho} = 0.0001$, $\bar{\rho}^- = -0.01$, $\bar{\rho}^+ = 0.01$). Similar results occur with other size hemispheric regions and other random weight sets.

Lateralization in this case is due to initial random asymmetries in weight values. These weight asymmetries, when accentuated by the iterative process by which hemispheric activation values are determined, can lead to significant pretraining mean activation asymmetries, especially with inhibitory callosal connections, as shown in Figure 2b. Comparing Figures 2b and 2a shows that these initial activation-level asymmetries are predictive of the direction of lateralization with inhibitory callosal connections. With backpropagation learning, higher mean hemispheric activation levels closer to 0.5 on one side cause faster weight changes on that side for both the incoming and outgoing connections of hemispheric elements. Thus, the more highly activated side of the model learns the task more quickly. Once one hemisphere region has largely learned to generate the correct outputs, the output error values drop to near zero, and weight changes throughout the entire model effectively end. The opposite hemispheric region is therefore never driven to learn an adequate set of weights.

It is possible to find random seeds for which, by chance, pretraining
Figure 2: Symmetric control model. (a) Error versus corpus callosum weights. The top dashed line indicates pretraining error; the thick line at the bottom indicates posttraining error, with both RH and LH jointly determining model output. The middle lines between .15 and .35 are the error when the left (dashed line) or right (solid line) hemispheric region alone sends activation to output elements. For example, when all callosal connections had an inhibitory weight of $-1.5$, the left hemispheric region ($E_L \approx .16$) was more accurate than the right hemispheric region ($E_R \approx .32$) in generating correct outputs, giving a substantial asymmetry coefficient value $\rho_{-1.5} = -0.21$. (b) Corresponding mean pretraining activation levels for left (dashed line) and right (solid line) hemispheric regions.

mean activation levels are essentially equal in the two hemispheric regions for all callosal strengths. If one uses such a seed in all simulations (unlike in Figure 2a, where a different random seed was used in each simulation), significant lateralization does not occur with the symmetrical model for any callosal strengths. However, even with arbitrary seeds, the lateralization
occurring in the symmetric model is limited in magnitude relative to that seen when various asymmetries are introduced, as described below.

### 3.2 Asymmetric Hemisphere Sizes

Larger left hemisphere language regions are often suggested as a causative factor for left language lateralization (Geschwind & Galaburda, 1987; Hellige, 1993). To examine this, we studied simulations where hemispheric sizes $|LH| > |RH|$, that is, models with more left hemispheric elements. Figure 3a shows a representative set of results with $|LH| = 14$ and $|RH| = 10$. Consistent lateralization to the left occurs for $c \leq -0.5$, and the amount of lateralization tends to increase as $c$ becomes increasingly negative (e.g., in Figure 3a, $\rho_{-0.5} = -0.15$, $\rho_{-0.39} = -0.39$, $\rho_{-0.57} = -0.57$). Lateralization also increases with increasing ratio of hemispheric sizes $|LH| / |RH|$. For example, $\rho_{-0.21}$ is $-0.21$ at $14/12$, $-0.39$ at $14/10$, and $-0.49$ at $14/8$.

While lateralization to the larger side is a robust finding with this version of the model, it is associated with increased pretraining mean hemispheric activation levels on the larger side for negative callosal strengths. This occurs because the larger hemispheric region more effectively inhibits the smaller when there are equally strong inhibitory callosal connections to both hemispheres. As the control model showed, an asymmetry in pretraining activation levels by itself can lead to lateralization. To control for this factor, we modified the model so that the uniform strength of inhibitory left-to-right callosal connections could differ from that of right-to-left connections. In this case, the strength of each left-to-right callosal connection was taken as $10c / |LH|$, and each right-to-left callosal connection $10c / |RH|$. This tended to produce pretraining hemispheric activation levels that were approximately the same.

With this modification, consistent lateralization to the larger hemispheric region with negative callosal strengths largely disappeared in many cases. For example, this effect can be seen in Figure 3b, where $|LH| / |RH| = 14/10$ as in Figure 3a, but now symmetric pretraining mean hemispheric activation is present. Some lateralization still occurs here for negative callosal strengths, but its direction is arbitrary ($\rho^- = -0.01$). Mild but inconsistent lateralization still occurred with positive callosal strengths in this case (e.g., $\rho^+ = -0.07$ in Figure 3b). Even when the ratio $|LH| / |RH|$ is quite large (e.g., $24/6$), in general less lateralization occurred. Figure 4a shows an example where some lateralization did occur, favoring the left side, mainly in the range $-0.5 \leq c \leq 1.0$ (i.e., when callosal connections are relatively weak). More consistent and pronounced lateralization for all values of $c$ could still be elicited when the number of hemispheric elements on the smaller side was so low that the smaller side could not learn the task in isolation, for example, $|LH| / |RH| = 20/4$.

In section 4, we will consider the explanation that it is the relative speed at which a hemisphere learns that is often the critical factor in determining whether lateralization occurs. In that context, the results in Figure 4b are very
important. They show that for a single hemispheric region operating alone, the rate at which learning occurs increases as the number of hemispheric elements increases.

3.3 Asymmetric Cortical Excitability. Asymmetric cortical excitability has been associated experimentally with functional lateralization (e.g., Macdonell et al., 1991) and regionally may also be implied by asymmetries in various neurotransmitter levels (Tucker & Williamson, 1984). Asymmetric excitability in the model should lead to relatively higher mean pretraining...
Figure 4: (a) Error versus callosal strength for asymmetric size cortical regions after adjusting to equilibrate pretraining mean activation levels. Same notation as in Figure 2a; 20 elements on the left, 6 on the right ($\bar{\rho} = -0.10$). (b) Time for a single hemispheric region to learn this task versus number of hemispheric elements.

We modeled asymmetric cortical excitability in two ways. First, we let the maximum activation level $M$ in equation 2.1 be asymmetric. For example, keeping $M_L = 1.0$ on the left and considering progressively lower values of $M_R$ on the right, we found significant lateralization toward the left first occurred for simulations with callosal strengths $\epsilon < 0$ at about $M_R = 0.8$. 

activation levels in the more excitable hemispheric region, and this would be expected to result in better independent performance by that hemispheric region.
At $M_R = 0.7$, lateralization increased and became present for all callosal strengths, although it was progressively more pronounced for increasingly inhibitory callosal connections (see Figure 5a). Lower and lower $M_R$ led to progressively increased lateralization, reaching the point where the left hemispheric region alone was almost always as good as the two hemispheric regions together when $M_R = 0.3$ (see Figure 5b). This progressive lateralization with increasing $M$ asymmetry can be summarized by plotting $\rho^+$ and $\rho^-$ for various $M_R$ values (see Figure 5c).

The second way asymmetric cortical excitability was produced was by allowing hemispheric sensitivity (i.e., input gain) to input received from input elements and state elements to differ on the two sides. This produced results similar to those seen with asymmetric maximum activation levels. As the ratio of left to right input sensitivity progressively increased, lateralization to the left first appeared with negative callosal strengths, being more pronounced with increasingly strong negative callosal strengths. With greater and greater asymmetry in input sensitivity ratio, this trend became increasingly more pronounced and was eventually accompanied by left lateralization of lesser magnitudes even when callosal strengths were positive.

### 3.4 Lateralization Without Asymmetric Pretraining Mean Activation Levels

Another possible cause of functional lateralization is asymmetric synaptic plasticity, which might also be implied by regional asymmetries in various neurotransmitters. If one hemispheric region is more modifiable and better able to learn new information, then one could hypothesize that it would assume a greater portion of a task. We simulated asymmetric synaptic plasticity by allowing one hemisphere to have a larger learning-rate parameter than the other. In this situation, functional lateralization tended to occur readily and consistently toward the side with the larger learning-rate parameter. This effect increased with increasing learning-rate parameter asymmetry. Unlike the asymmetries already described, lateralization was largely independent of whether the corpus callosum was excitatory or inhibitory. Figure 6a illustrates these points, where the learning-rate parameters are 0.05 (left) and 0.02 (right), and there is consistent lateralization to the left hemisphere regardless of the sign of $c$ ($\bar{\rho}^- = -0.29$, $\bar{\rho}^+ = -0.28$). Pretraining mean activation levels of the hemispheric regions had only a secondary, modulating effect on this lateralization. For example, lateralization to the left still occurs at $c = -2.5$ in this case (see Figure 6a; $\rho_{-2.5} = -0.23$) in spite of higher pretraining activation levels in the right hemisphere when $c = -2.5$ (see Figure 6b).

Asymmetric feedback to the hemispheric regions (via state elements; see Figure 1) might also be conjectured to lead to lateralization. However, this was difficult to elicit with our model unless the feedback to one side was so small as to preclude that side from learning the task at all. For example, when feedback activation from state elements to the right hemi-
Figure 5: Error versus callosal strength with $M_L = 1.0$, but (a) $M_R = 0.7$, or (b) $M_R = 0.3$. (c) Values of $\rho^+$ (solid line) and $\rho^-$ (dashed line) for various $M_R$ values when $M_L = 1.0$. 
Figure 6: Effects of asymmetric learning-rate parameters of 0.05 on the left and 0.02 on the right. (a) Error versus callosal strength; same notation as Figure 2a. (b) Mean pretraining activation levels for left (dashed line) and right (solid line) hemispheric regions.

A hemispheric region was scaled to be 0.2 of that on the left, very mild but consistent lateralization to the left could occur. Independent simulations with a single hemispheric region showed that the number of training epochs required to learn the task was inversely related to the feedback level (e.g., 950 epochs for baseline feedback level versus 1500 epochs for 0.2 scaled feedback).
4 Discussion

While there have been many previous neural models of cerebral cortex, to our knowledge only three have examined aspects of hemispheric interactions. The earliest of these demonstrated that oscillatory activity in one simulated hemisphere could be transferred to the other hemisphere via interhemispheric connections (Anninos et al., 1984; Anninos & Cook, 1988). A second model established that inhibitory callosal connections produce slower convergence and different activity patterns in the two simulated hemispheres (Cook & Beech, 1990). Finally, a pair of recurrent error backpropagation networks were trained to learn a small set of input-output associations simultaneously, and it was shown that slow “interhemispheric” connections were not critical for short output times (Ringo et al., 1994). None of these previous neural models of hemispheric interactions examined lateralization of functionality through synaptic weight changes (learning). In addition, there has been one symbol-processing model of interacting hemispheres (van Kleek & Kosslyn, 1991). This latter study is not a neural model; it represents the cognitive processes in high-level vision as numerous complex modules. This algorithmic model assumes a priori the lateralization of speech and other functions and then examines how such preexisting lateralizations could cause lateralization of other functions. This work does not demonstrate “spontaneous” lateralization of functionality (i.e., it starts with assumed lateralizations of functions).

The work described here is also related to past studies involving the gating of multiple performance networks, such as mixture of experts architectures (Jacobs, Jordan, Nowlan, & Hinton, 1991; Jordan & Jacobs, 1994) and modular neural networks (Ronco & Gawthrop, 1997). The architecture of these models typically includes two or more expert networks analogous to our hemispheric regions. Unlike our model and other past models incorporating callosal connections (Anninos et al., 1984; Cook & Beech, 1990; Ringo et al., 1994), the expert networks in mixture-of-experts models have their output multiplicatively filtered by a gating network. Such gating networks have not been intended as a model of the corpus callosum, and it is difficult to see how they could be considered as such. Typically the gating networks directly receive and process input patterns through their own separate weighted network, unlike the real corpus callosum. Neither expert network (hemisphere) determines the activation levels of neurons in the gating network, in contrast to our model and the biological corpus callosum. Thus, the expert networks do not directly influence each other as occurs biologically, nor do they do so indirectly by activating the gating network. In addition, the output connectivity of a gating network would suggest the existence of callosal fiber synapses on cortical neuron axons or subcortical structures, something that appears to be inconsistent with experimental data showing that callosal fibers synapse primarily (about 97%) on cortical dendrites and dendritic spines (White, 1989). Finally, in mixture-of-expert
models the different networks become experts on different domains of the function being learned, whereas in our model the direct interhemispheric interactions generally produce a network (hemisphere) that is better than the other over all of the function domain, capturing more closely the essence of hemispheric dominance. While gated architectures have been applied to neuropsychological questions recently (Dailey, Cottrell, & Padgett, 1997), they have not been used to study the specific issues of function lateralization or callosal functionality. Thus, to our knowledge, the research reported here is the first systematic study of computer-simulated emergence of function lateralization.

Our simulations show that within the limitations of the model we studied, it is easy to produce lateralization of function with several hemispheric asymmetries. Lateralization tended to occur most readily when callosal connections were inhibitory, supporting previous arguments that the primary functionality of the corpus callosum is inhibitory. However, when hemispheric asymmetries were sufficiently pronounced or directly affected learning rate, some lateralization could occur with all callosal strengths. For example, almost complete lateralization occurred even with strongly excitatory callosal connections when the hemispheric regions were asymmetrically excitable or had asymmetric learning rates.

Many of our results can be intuitively understood if one views learning in the model as a “race” between the two hemispheric regions to acquire an input-output mapping via weight changes. The “race-to-learn” ends when the model as a whole has acquired the mapping and output error values approximate zero. In this situation, weight changes stop. Thus, any factor that increases the learning speed on one side of the model causes that side to learn the input-output associations better than the other side, leading to function lateralization. Specifically, asymmetries in such different factors as initial random weights, hemispheric size, maximum activation level, and input sensitivity all could lead to asymmetric pretraining activation levels when callosal strengths were negative, and via this to more rapid learning on the eventually dominant side. Asymmetries in hemisphere size and feedback intensity were found to be associated with asymmetric learning speeds, and these could lead to lateralization without asymmetries in pretraining hemispheric activation levels. Finally, directly accelerating weight changes in one hemisphere by increasing its learning-rate parameter led to function lateralization for all callosal strengths.

Of special interest is that lateralization could occur with asymmetry in size of cortical regions under three conditions. First, if asymmetric-size regions led to asymmetric pretraining activation levels, marked lateralization could occur. Second, even if one prevented secondary asymmetry in hemispheric activation levels (and thus in the rate at which individual synapses changed), some lateralization could still occur if the size asymmetry was
sufficiently dramatic (e.g., 4 to 1). Such large asymmetries in size have been observed in cortical cytoarchitectonic areas related to language (Galaburda et al., 1978). In this case, the increased numbers of synaptic weights in the model’s larger hemispheric region resulted in the larger region’s having a faster learning rate (e.g., Figure 4b). Finally, if one hemispheric region was too small to learn the associative input-output mapping while the other was not, significant lateralization could occur.

Overall, these simulations also suggest an asymmetric synaptic plasticity hypothesis for biological lateralization of language and other associative functions: asymmetric synaptic plasticity in hemispheric regions forms a common causative mechanism for many cortical factors that result in behavioral lateralization. Among other things, this hypothesis leads to the testable and potentially refutable prediction that lateralization of a function to one cerebral region will be associated with increased synaptic plasticity in that region relative to its mirror-image region in the opposite hemisphere. Experimental evidence for or against this expectation could be provided in many ways, such as immunochemical or electron microscopic measures of synaptic density, or total synaptic area per unit volume, or physiological assessment of long-term potentiation. To our knowledge, the asymmetric synaptic plasticity hypothesis has not been suggested previously, and relative measures of synaptic plasticity have not been made in paired regions of cerebral cortex. This hypothesis seems particularly intriguing to us in the context of suggestions, made by others independent of any considerations of function lateralization, that the peak period of synaptogenesis during the first three years of life is an important neural correlate of language acquisition (Bates, Thal, & Janowsky, 1992).

While this work clearly demonstrates that computational models can be used to investigate the emergence of function lateralization, much further work is needed to assess the generality of the results reported here. For example, we have studied only error backpropagation in this work, selecting it as an initial learning method to examine because it is effective in learning input-output associations, it has been used successfully in the past to simulate aspects of cortical neuron responses (Lehky & Sejnowski, 1988; Zipser & Anderson, 1988), it has produced interesting results from learning involving interacting hemispheric regions (Ringo et al., 1994), and there is recent experimental evidence supporting its biological relevance (Fitzsimonds, Song, & Poo, 1997). Further, we examined only diffuse and not homotopic callosal connections. Finally, we emphasize that we have considered only lateralization of a single task in this work, as a logical first step before examining more general situations. There are multiple functional hemispheric asymmetries, and the question of how these are allocated to multiple cortical areas is an important direction for future research. Work is underway to address several of these issues.
Appendix

A.1 Words Used for Training.
CAD CAP CAT COD COP COT HAD HAP HAT HIP HIT HOD HOP HOT
LAD LAP LID LIP LIT LOP MAD MAP MAT MID MIP MOP PAD PAP
PAT PIP PIT POD POP POT SAD SAP SAT SIP SIT SOD SOP SOT TAP
TAT TIP TIT TOP TOT

A.2 Learning Rule Equations. Symbols $o, h, i, s,$ and $t$ are superscripts that denote output, hidden, input, state, and target values, respectively; $\eta$ is the learning rate and $M$ the maximum activation level.

- Error at the $i$th output unit:
  \[ e_o^i = (a_o^i - a_t^i). \]

- Weight change for connection from $j$th hidden unit to $i$th output unit:
  \[ \Delta w_{ij}^o = \eta_o a_h^j e_o^i [a_o^i (1 - a_o^i)]. \]

- Error at the $j$th hidden unit:
  \[ e_h^j = \sum_i w_{ij}^o e_o^i [a_o^i (1 - a_o^i)]. \]

- Weight change for connection from $k$th input unit to $j$th hidden unit:
  \[ \Delta w_{ik}^h = \eta_i a_i^k e_h^j [a_h^j (1 - a_h^j / M)]. \]

- Weight change for connection from $l$th state unit to $j$th hidden unit:
  \[ \Delta w_{ls}^h = \eta_s a_s^l e_h^j [a_h^j (1 - a_h^j / M)]. \]

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