

# **Modeling the Flow of Information in Cognition: An Account of Optic Aphasia**

**Michael Mozer**  
**University of Colorado**

**Mark Sitton**  
**University of Colorado**

**Martha Farah**  
**University of Pennsylvania**

# Optic Aphasia

- **Deficit in naming visually presented objects**

- **Not visual agnosia**

Nonverbal indications of recognition: sorting, gesturing

- **Not general anomia**

Naming possible given verbal definition, tactile stimulation, object sounds

- **Visual system roughly intact**

Insensitivity to visual quality; can copy drawings; normal interaction with world

- **No prosopagnosia**

- **Alexia**

- **Neuropathology: unilateral left posterior lesions, including occipital cortex and white matter**

## Data to Explain

1. Naming from visual presentation ( $V \rightarrow N$ ) severely impaired.
2. Naming from verbal description ( $A \rightarrow N$ ) relatively unimpaired.
3. Gesturing from visual presentation ( $V \rightarrow G$ ) relatively normal.
4. Gesturing from verbal descriptions ( $A \rightarrow G$ ) normal.
5. Many naming errors are semantic and perseverative, few visual.
6. When given unlimited time to produce name of visual stimulus, patients tend to “home in” on correct name.
7.  $V \rightarrow G$  has higher error rate than  $A \rightarrow N$ .
8.  $V \rightarrow G$  errors tend to increase as  $V \rightarrow N$  errors increase.

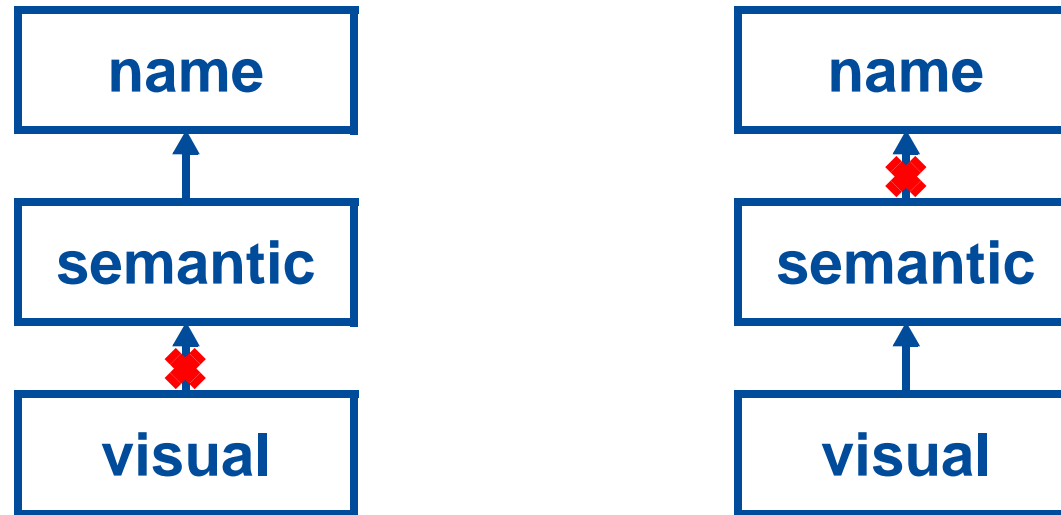
# Error Rates for Selected Optic Aphasia Patients

	V→N (visual stimulus, naming response)	V→G (visual stimulus, nonverbal response)	A→N (auditory stimulus, naming response)
Lhermitte & Beauvois (1973)	27%	0%	4%
Gil et al. (1985)	36%	0%	low
Riddoch & Humphreys (1987)	54%	25%	low
Manning & Campbell (1991)	58%	25%	0%
Larrabee et al. (1985)	70%	low	low
Ferro & Santos (1984)	77%	low	10%
Coslett & Saffran (1992)	79%	0%	32%
Assal & Regli (1980)	97%	75%	low
Poeck (1984)	100%	25%	10%
Coslett & Saffran (1989)	100%	50%	low

**low = no quantitative data, but presumably few errors**

# Models to Explain Optic Aphasia

## 1. Disconnection syndrome

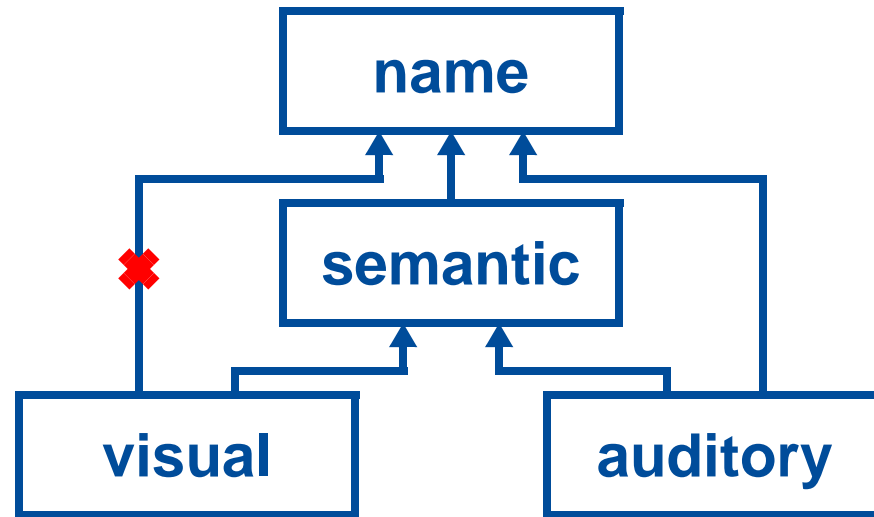


Can't have impairment in vision, semantics, or path between them because patients can respond to object nonverbally.

Can't have impairment in semantics, naming operations, or path between them because patients can name verbally presented material.

# Models to Explain Optic Aphasia

## 2. Direct route for object naming (Ratcliff & Newcombe, 1982)



**Visual percepts can produce their name directly.**

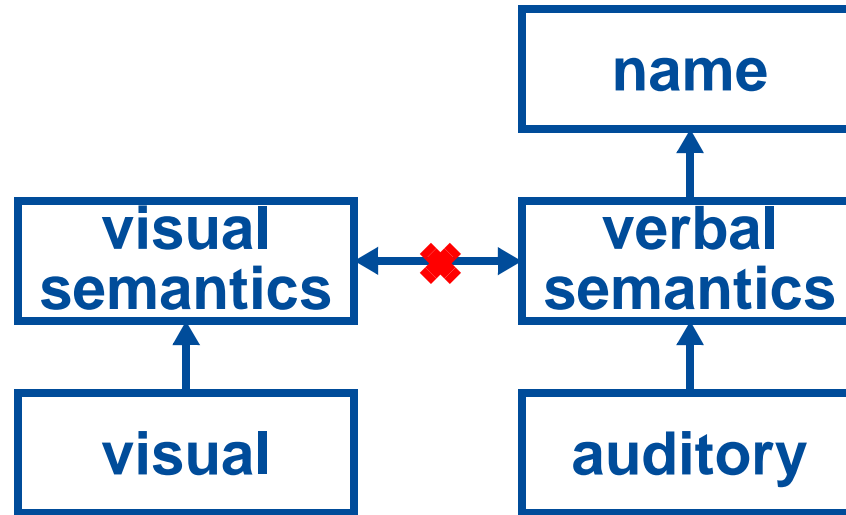
**Normal individuals use two routes in parallel.**

**Damage to direct route in optic aphasia leads to unreliability.**

**Problem: patient who can name visually presented object without knowing what it is?**

# Models to Explain Optic Aphasia

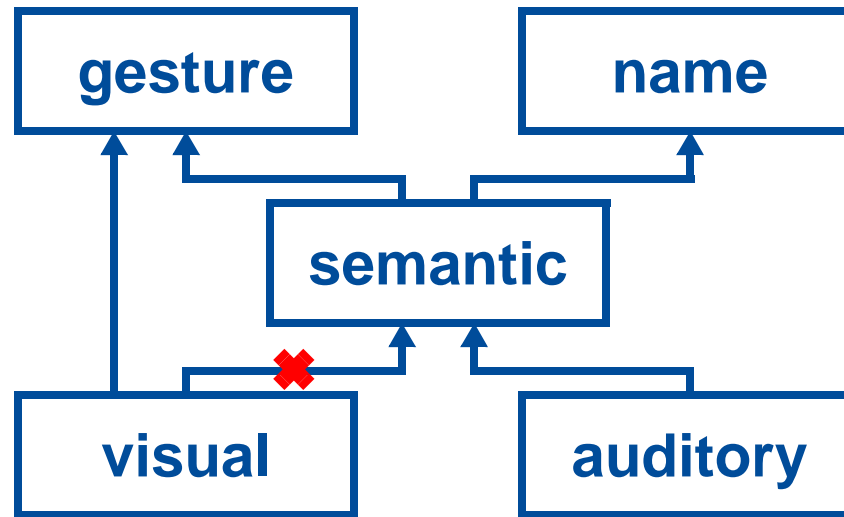
## 3. Modality-specific semantic systems (Beauvois, 1982)



**Problem: unparsimonious to suppose multiple copies of our entire stock of semantic knowledge**

# Models to Explain Optic Aphasia

## 4. Semantic access agnosia (Riddoch & Humphreys, 1987)

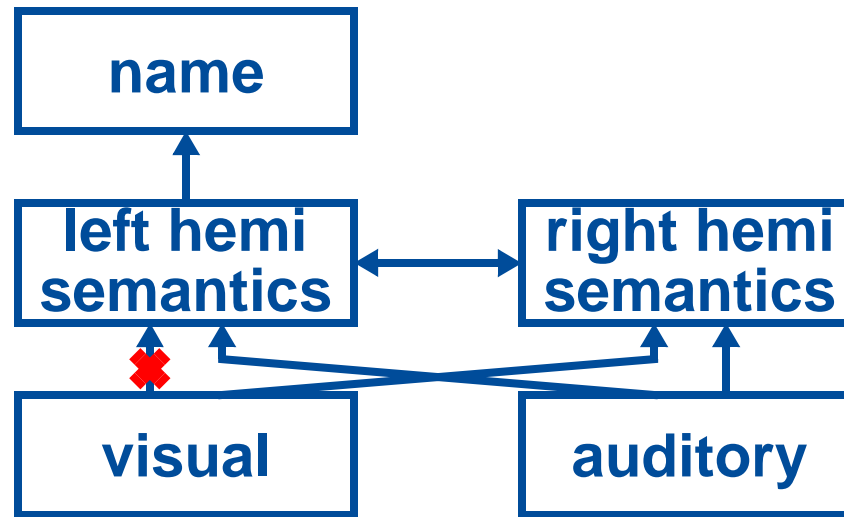


**Problem: how to explain categorization ability?**

**Problem: can gesturing really be done on the basis of visual appearance alone?**

# Models to Explain Optic Aphasia

## 5. Hemispheric-specific semantic systems (Coslett & Saffran, 1989)

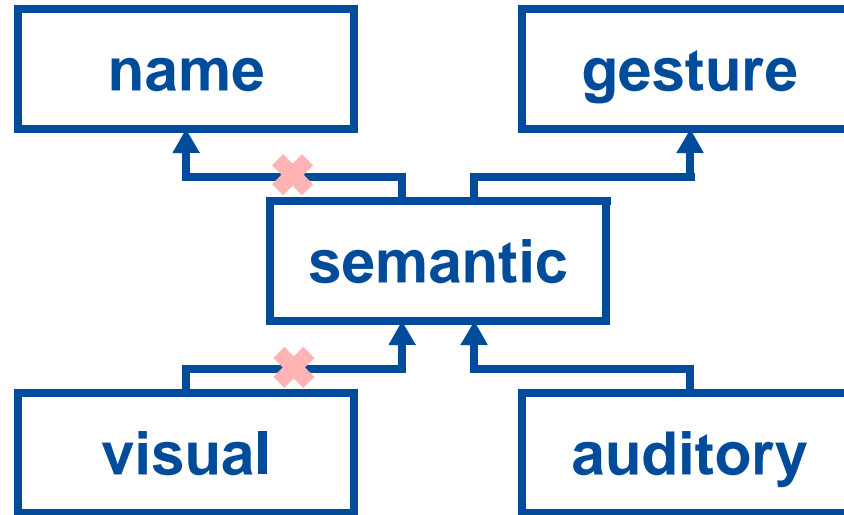


**Semantics is divided by hemisphere.**

**Right hemisphere semantics less finely differentiated than left hemisphere semantics, which leads to naming errors.**

# Models to Explain Optic Aphasia

## 6. Superadditive impairments in vision and naming (Farah, 1990)



# Modular Architecture

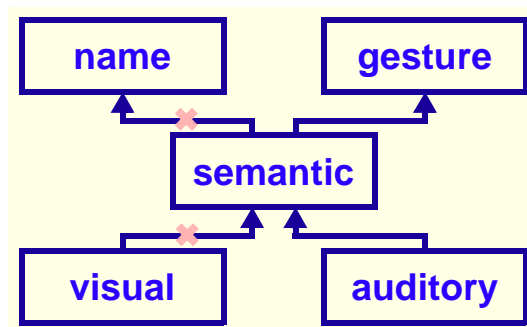
Each arrow represents a processing pathway

Pathway act as associative memories

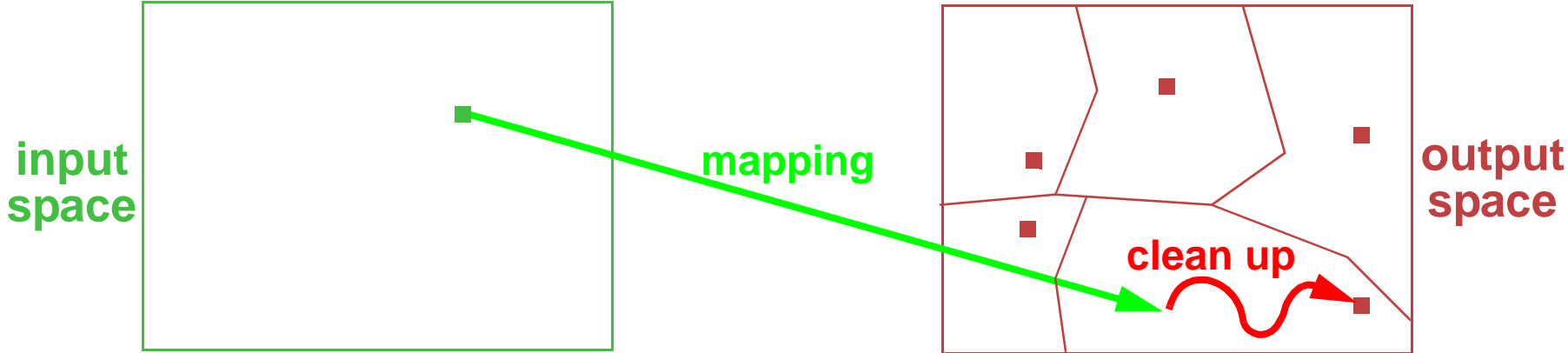
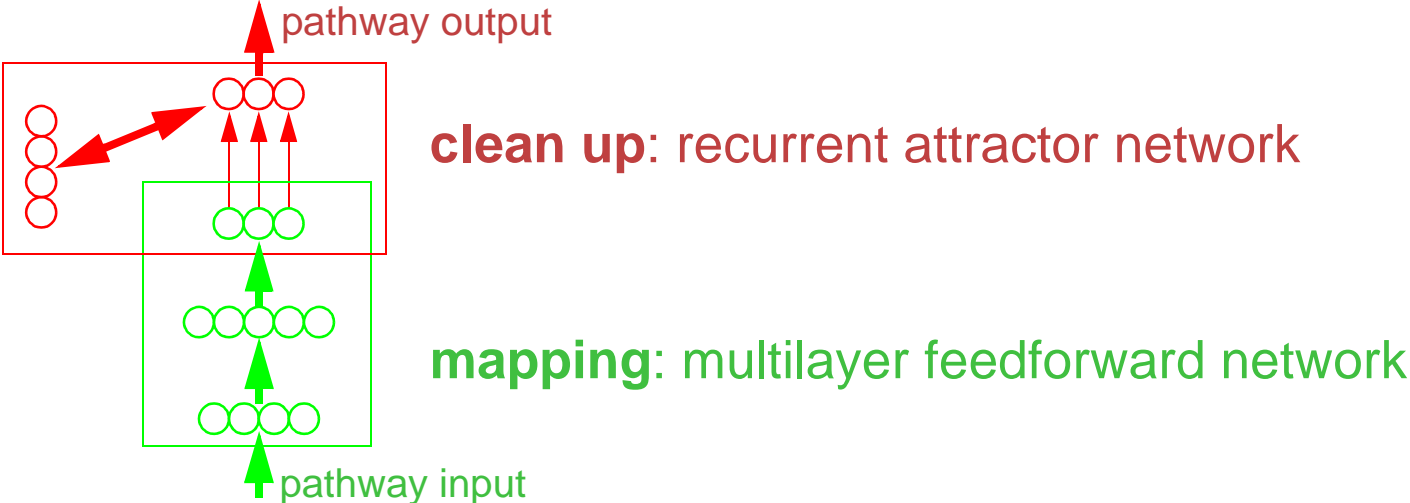
In this architecture, pathway operation shows speed-accuracy trade off

Initial output of pathway appears rapidly, but may be inaccurate.

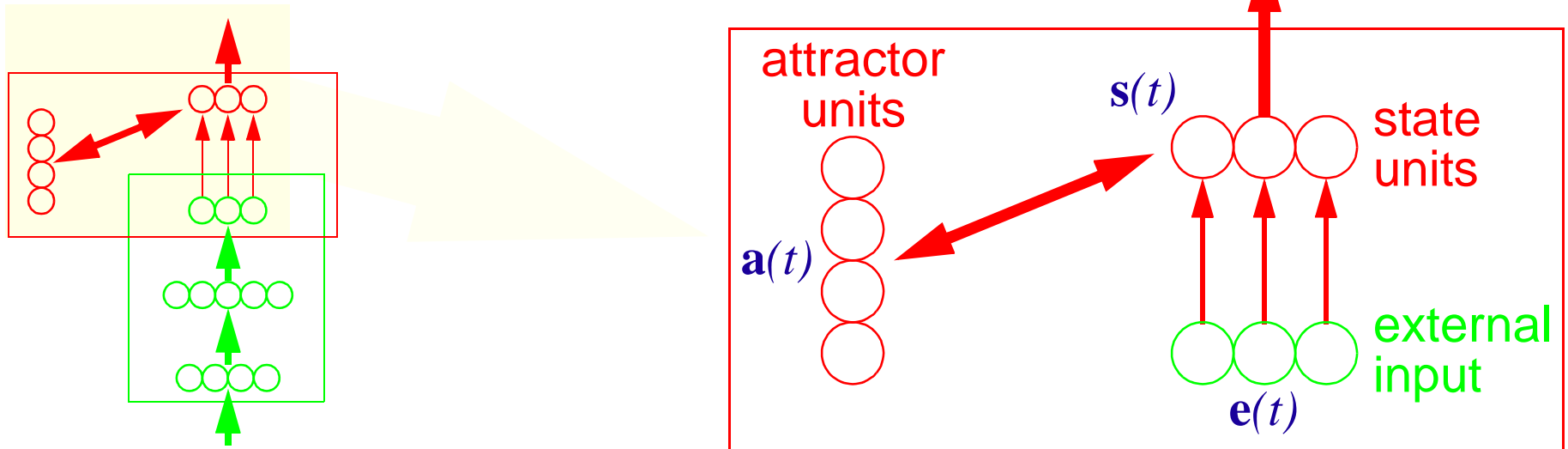
This “quick and dirty” guess is refined over time, and pathway asymptotically converges on the best interpretation.



# Connectionist Implementation of Pathway



# Model Dynamics



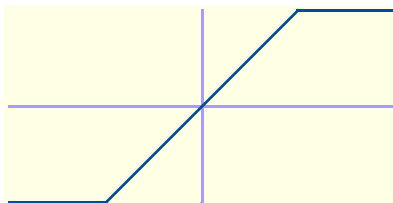
attractor unit update equation:

$$\hat{a}_j(t) = \exp(-\|s(t) - \mu_j\|^2 / \beta_j)$$

$$a_j(t) = \hat{a}_j(t) / \sum_i \hat{a}_i(t)$$

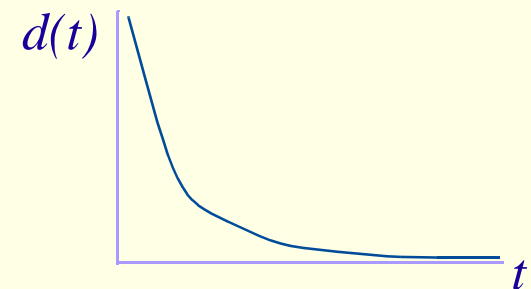
state unit update equation:

$$s_i(t) = h \left[ d_i(t) e_i(t) + (1 - d_i(t)) \sum_j a_j(t-1) \mu_{ji} \right]$$



$\mu_j$ : center of attractor  $j$

$\beta_j$ : width of attractor  $j$



$$d_i(t) = 1 - e_i(t-1) / e_i(t)$$

$$e_i(t) = \alpha e_i(t) + (1 - \alpha) e_i(t-1)$$

# Pattern Generation

Five representational spaces: **visual input, auditory input, semantic, name, and gesture**

Each space is 200 dimensional

Generated 200 random binary-valued ( $-1, +1$ ) patterns for each space

For **visual, auditory, and semantic** spaces:

50 clusters with 4 patterns per cluster

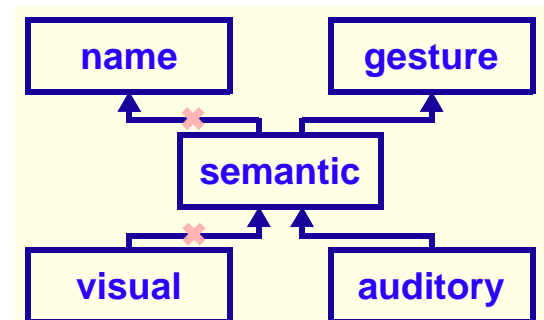
patterns in different clusters at least  $80^\circ$  apart

patterns in same cluster between  $25^\circ$  and  $50^\circ$  apart

For **name and gesture** spaces:

all patterns at least  $60^\circ$  apart

Patterns paired randomly



# Training Procedure

Four pathways:  $V \rightarrow S$ ,  $A \rightarrow S$ ,  $S \rightarrow N$ ,  $S \rightarrow G$

Each pathway trained separately

Mapping (feedforward) nets

trained on all 200 associates

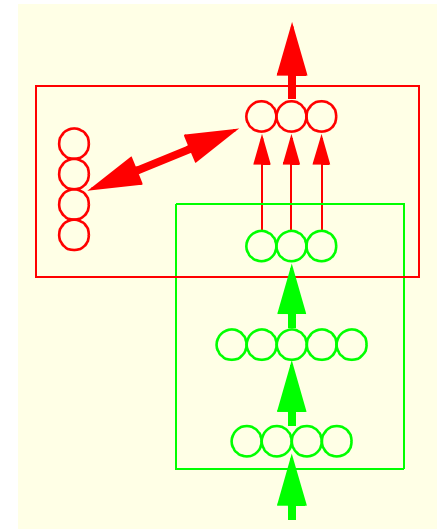
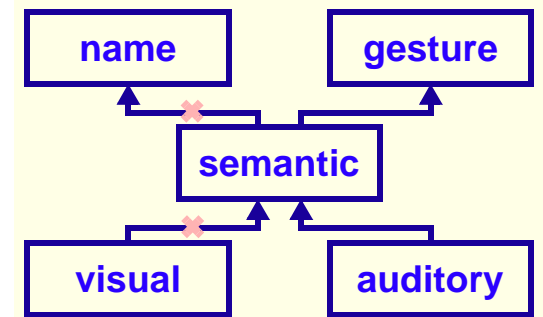
150 hidden units

training not perfect; 1 or 2 bits error

Attractor nets

hardwired with 200 attractors + 1 for rest state

all attractors had equal widths ( $\beta$ )



# Simulation Methodology

## For each of 25 simulated patients:

Damage model

Loop through all tasks ( $V \rightarrow N$ ,  $V \rightarrow G$ ,  $A \rightarrow N$ )

    Loop through all 200 input patterns

        Present input, run model until output settles

        Classify output

        Update attractor widths based on priming mechanism

    Repeat

Repeat

## Priming mechanism

Temporarily increase width ( $\beta$ ) of attractor into which each pathway settled

In undamaged model, causes model to settle more rapidly when same input is presented a second time

Used by Becker et al. (1993) and Mathis and Mozer (1996) to model priming

## Damaging model

Remove  $\gamma\%$  of input-to-hidden and hidden-to-output connections in the mapping networks of  $V \rightarrow S$  and  $S \rightarrow N$  pathways

No damage to attractor net

Other sorts of damage had qualitatively similar effects

## Response classification

*correct*: response (output attractor) corresponds to input pattern

*no response*: response is the rest state

*perseveration*: response same as that produced on any of three immediately preceding trials

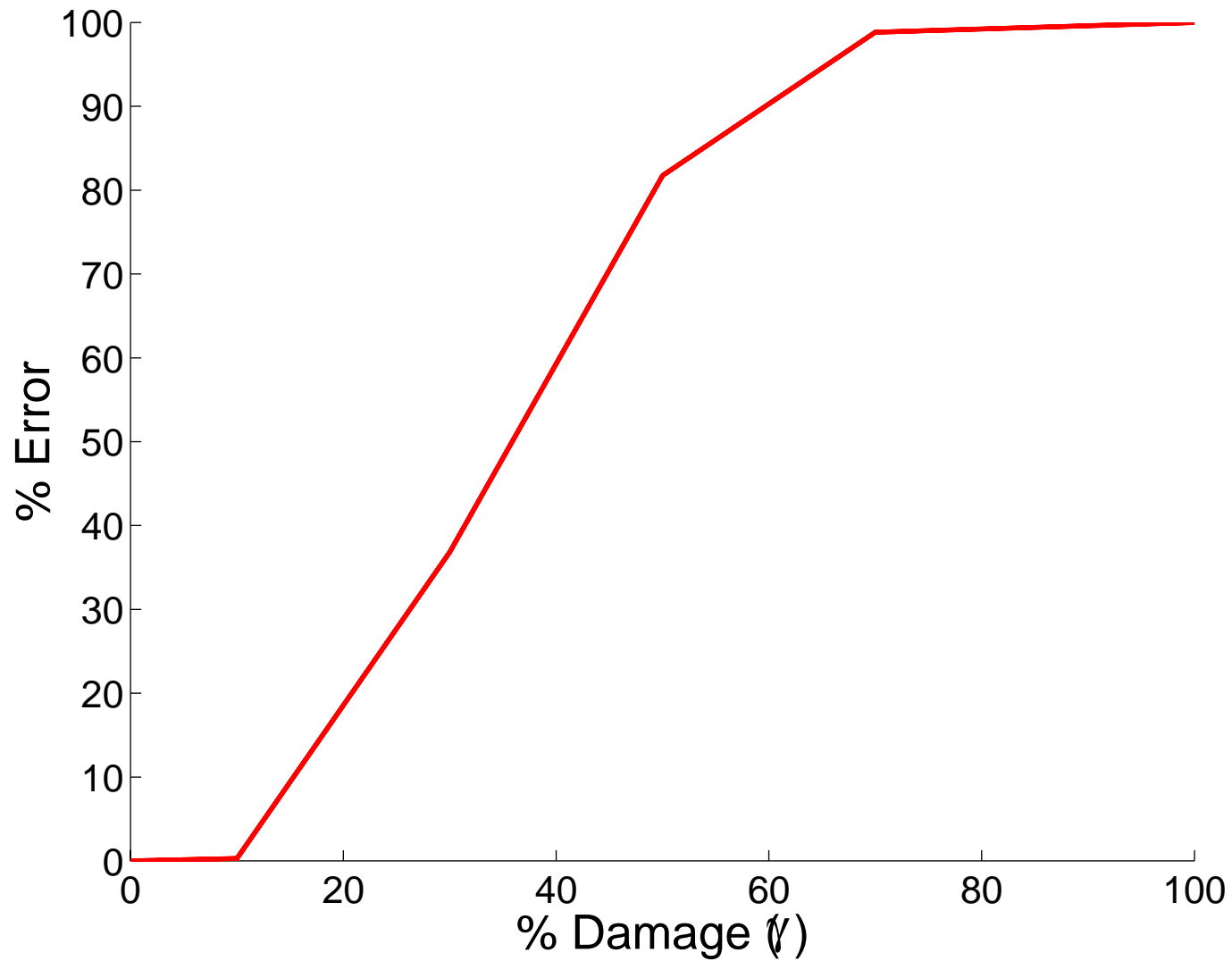
*visual*: visual pattern corresponding to incorrect response is sibling of visual pattern for correct response

*semantic*: semantic pattern corresponding to incorrect response is sibling of semantic pattern for correct response

*visual+semantic*: an error response that is both visual and semantic

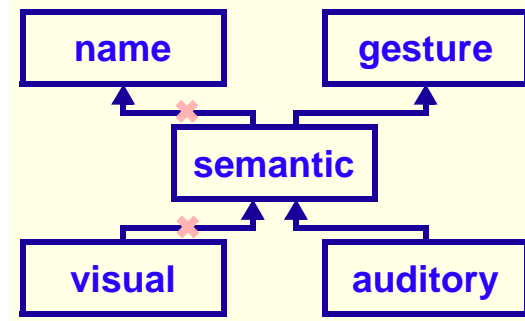
*other*: all other errors

# Error Rate for Visual Object Naming as a Function of Amount of Damage



# Error Rates by Task

<i>task</i>	<i>error rate</i>	<i>damaged pathways</i>
<b>A→G</b>	<b>0.0%</b>	
<b>A→N</b>	<b>0.5%</b>	<b>S→N</b>
<b>V→G</b>	<b>8.7%</b>	<b>V→S</b>
<b>V→N</b>	<b>36.8%</b>	<b>V→S, S→N</b>



**A→N: clean up compensates for S→N pathway damage**

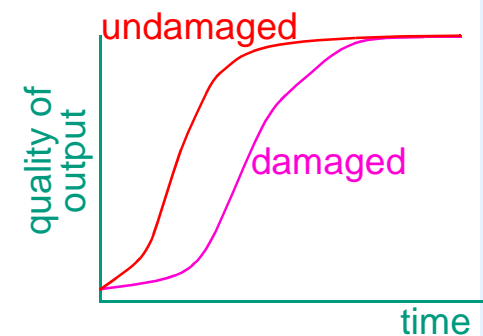
**V→G: clean up compensates for V→S pathway damage**

**V→N: effects of damage to V→S and S→N pathways interact**

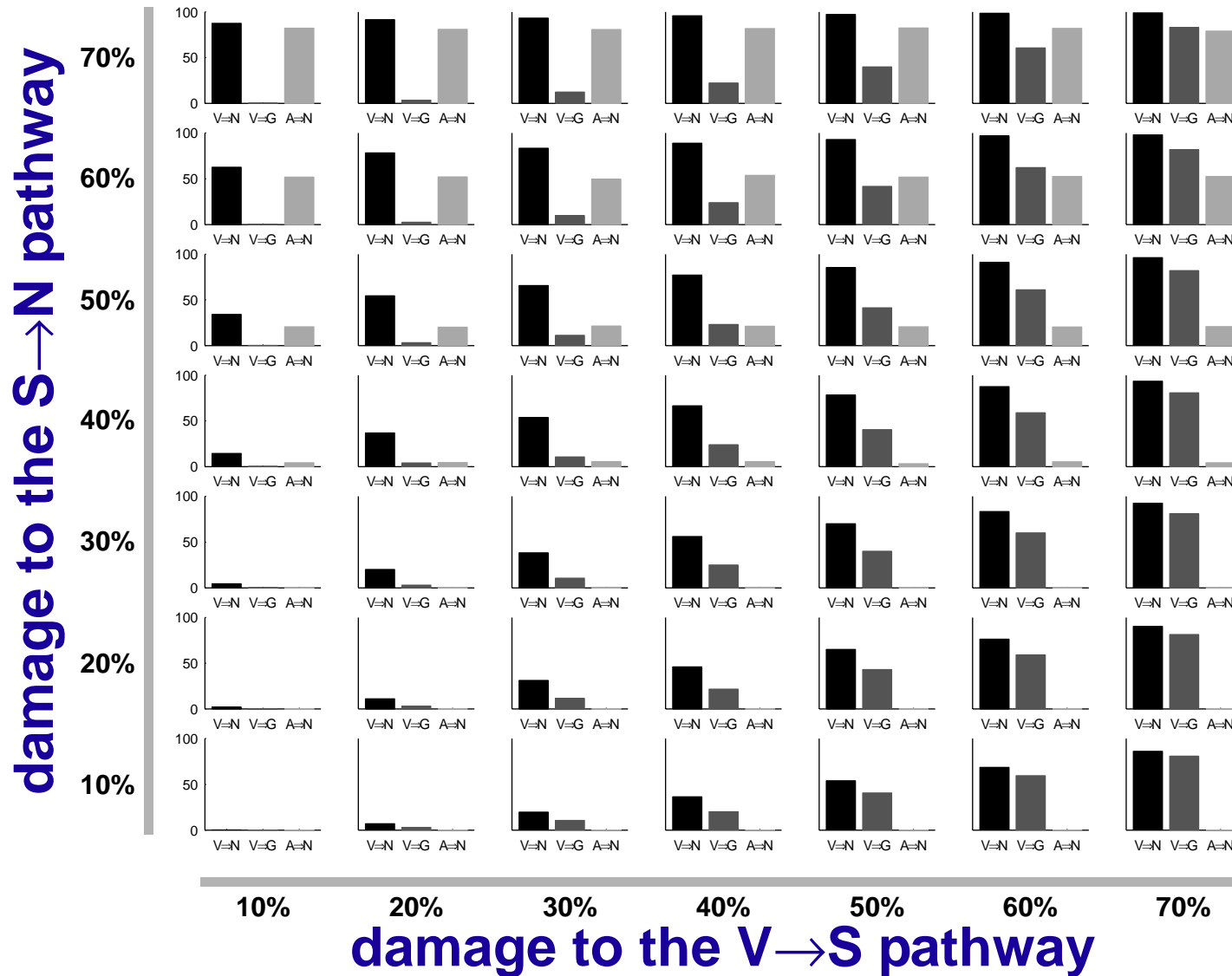
noisy input + internal damage to S→N pathway

Interaction would not occur if

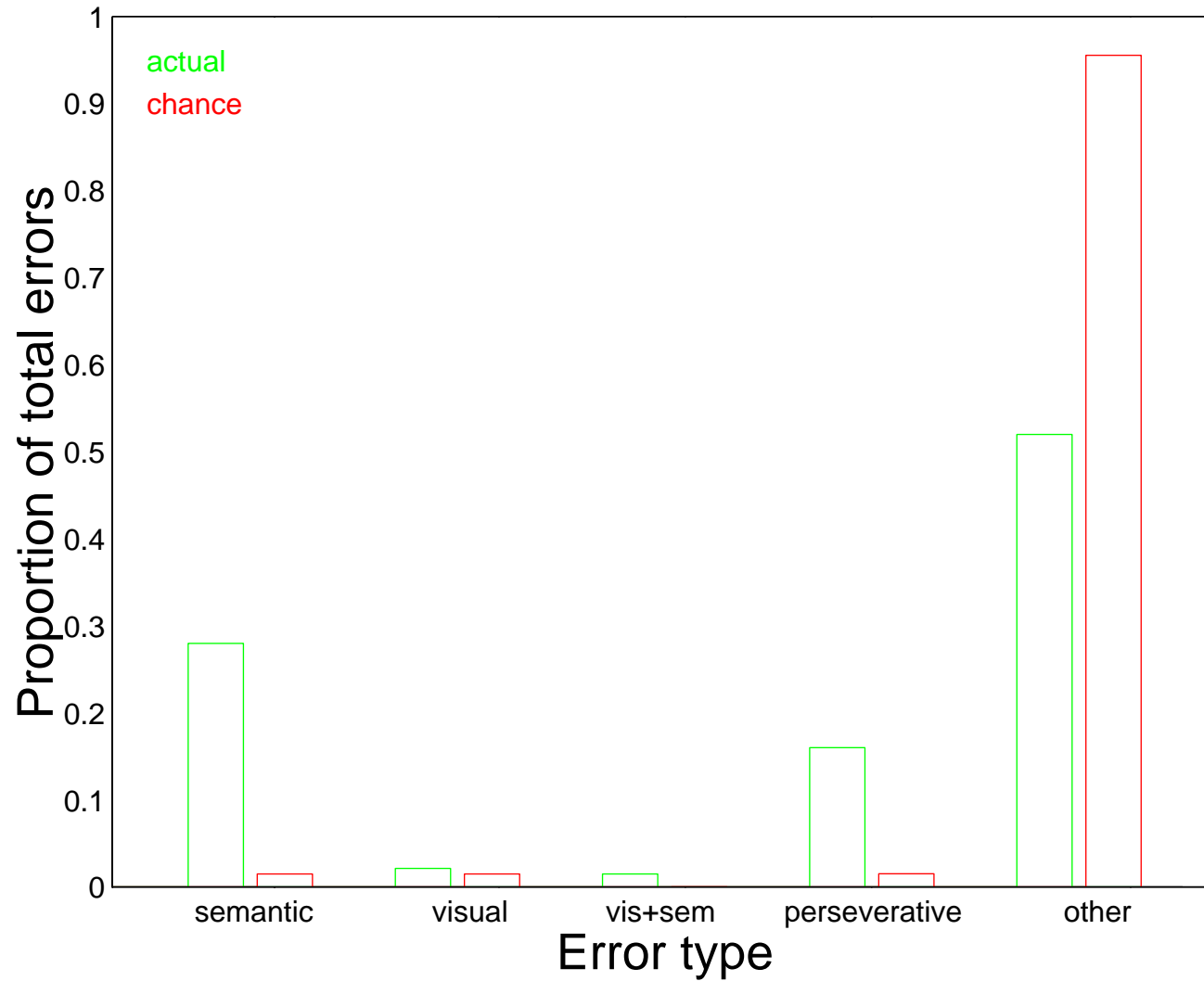
- (a) pathways operated sequentially, or
- (b) pathways showed no hysteresis



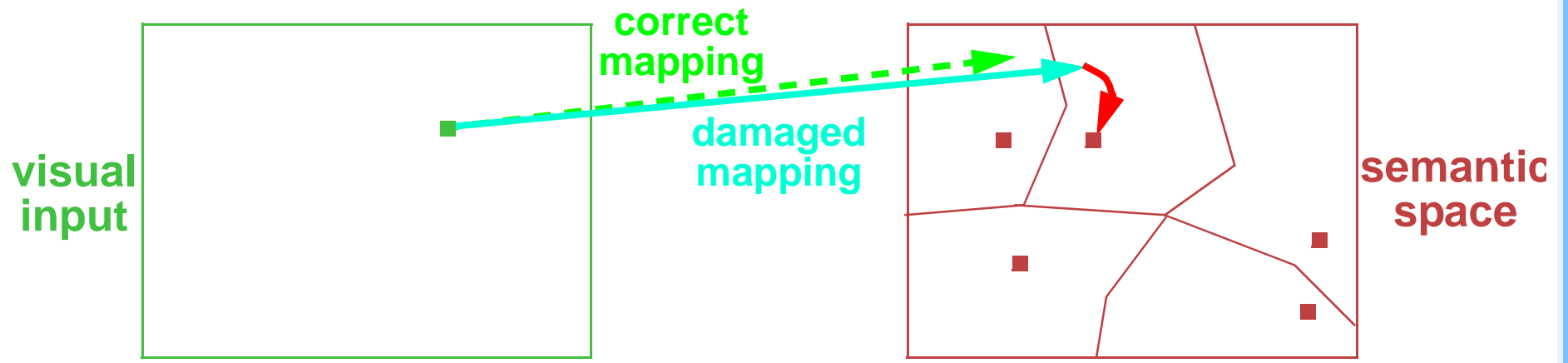
# Error Rates Based on Relative Damage



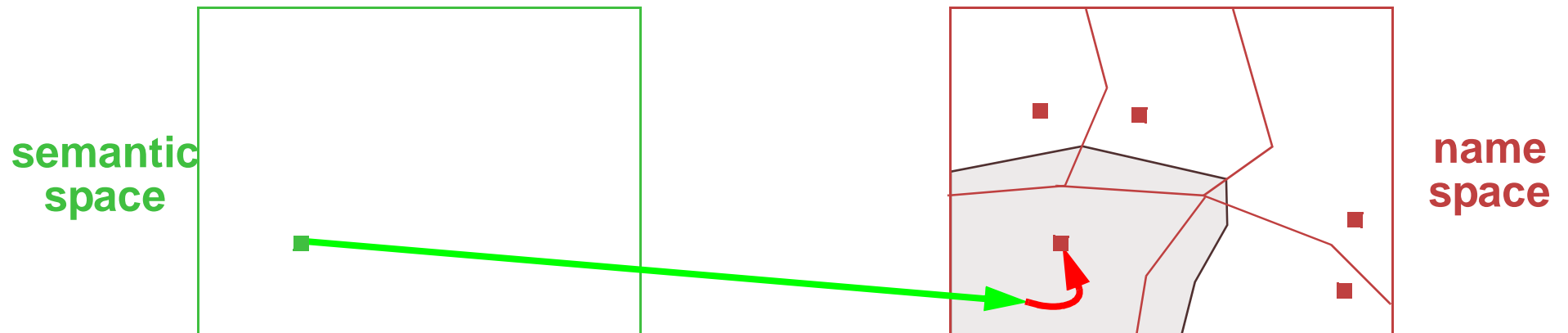
# Distribution of Errors for Visual Object Naming



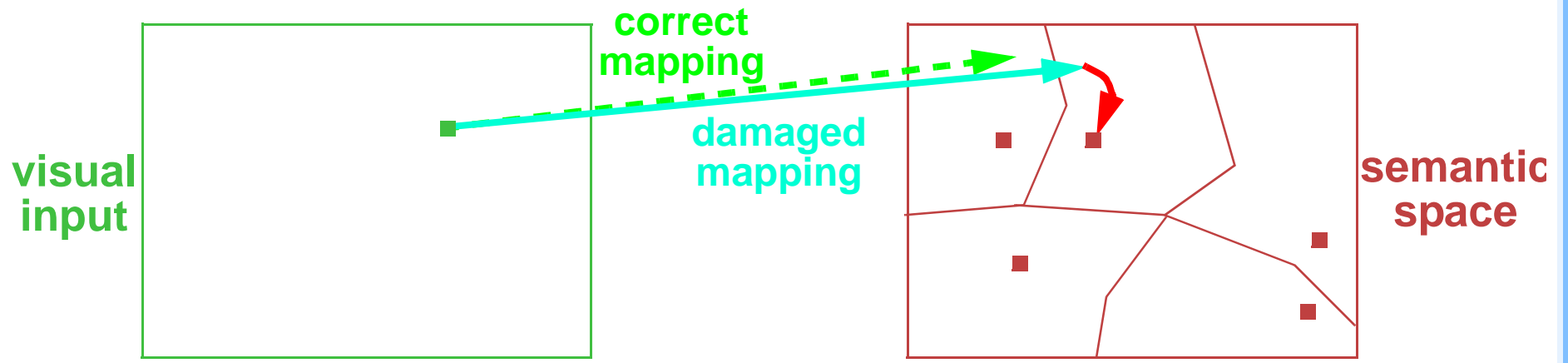
## Explanation for frequency of semantic errors:



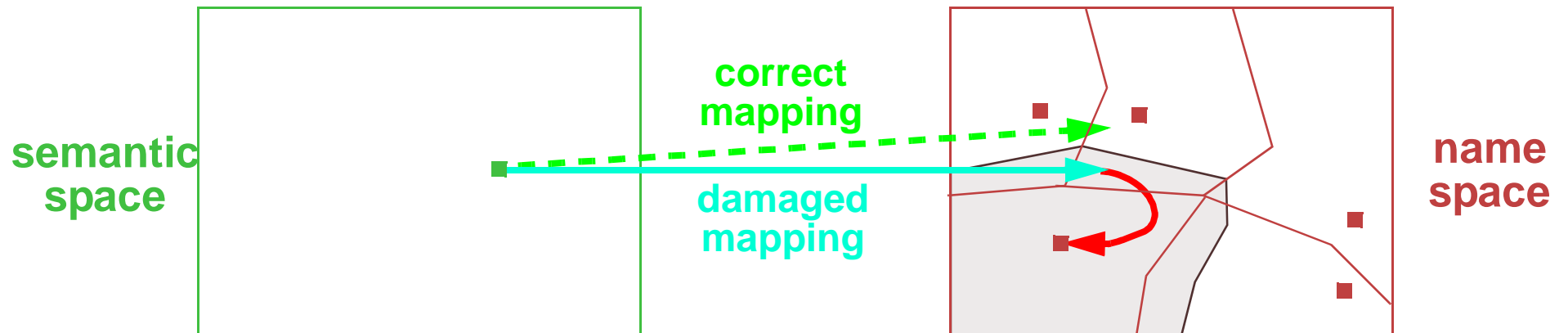
## Explanation for frequency of perseverative errors:



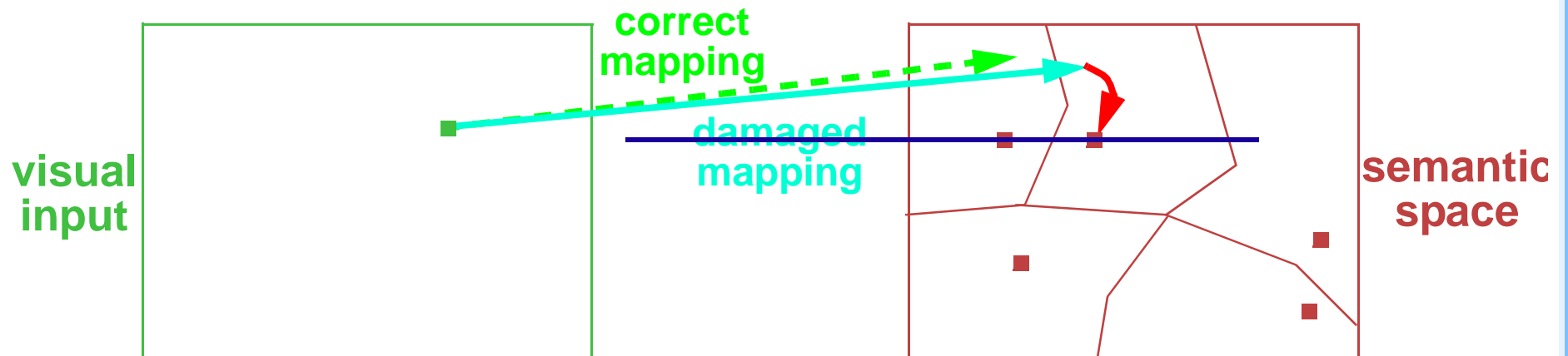
## Explanation for frequency of semantic errors:



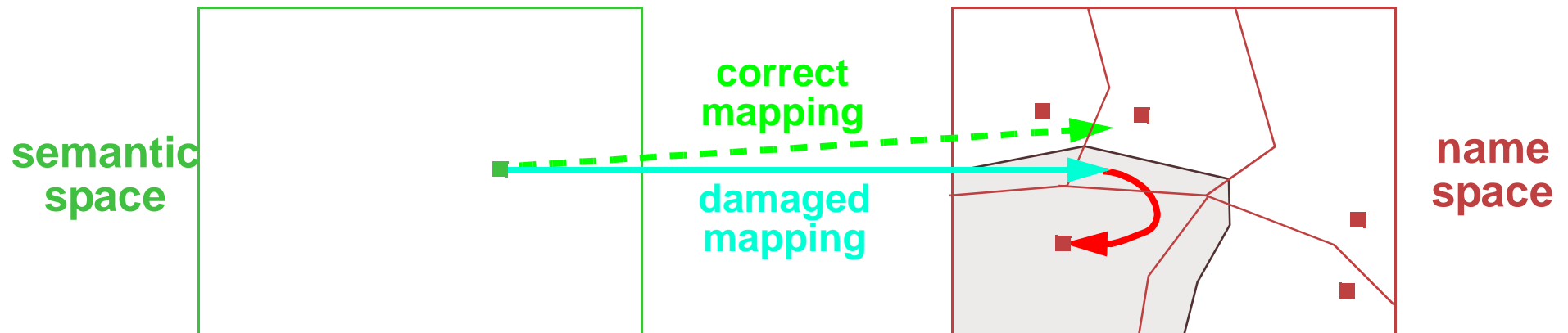
## Explanation for frequency of perseverative errors:



## Explanation for frequency of semantic errors:



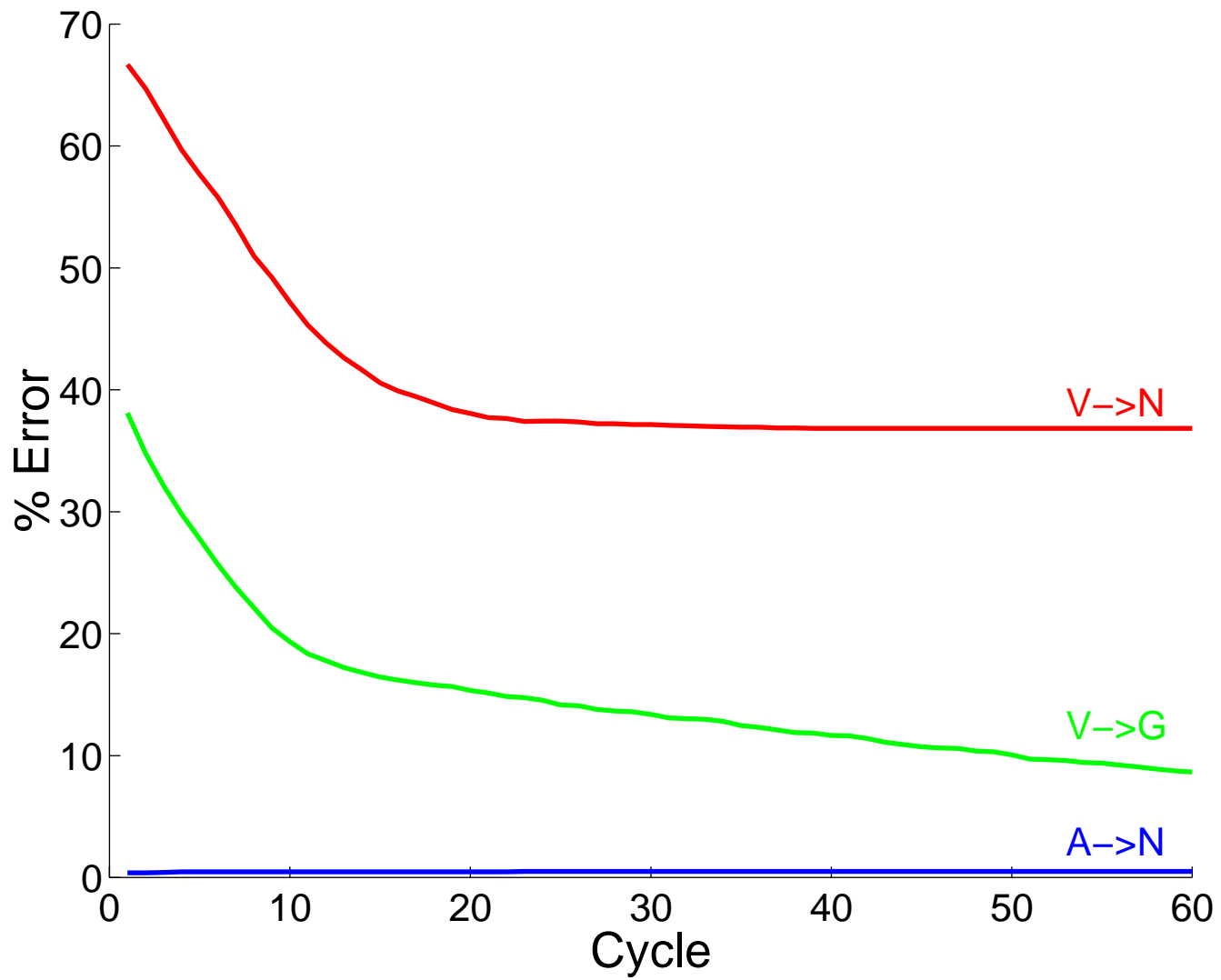
## Explanation for frequency of perseverative errors:



## Plaut & Shallice (1993) model also explains these errors

- obtained perseverative errors using short-term weight modification
- obtained semantic but not visual errors by careful construction of representations

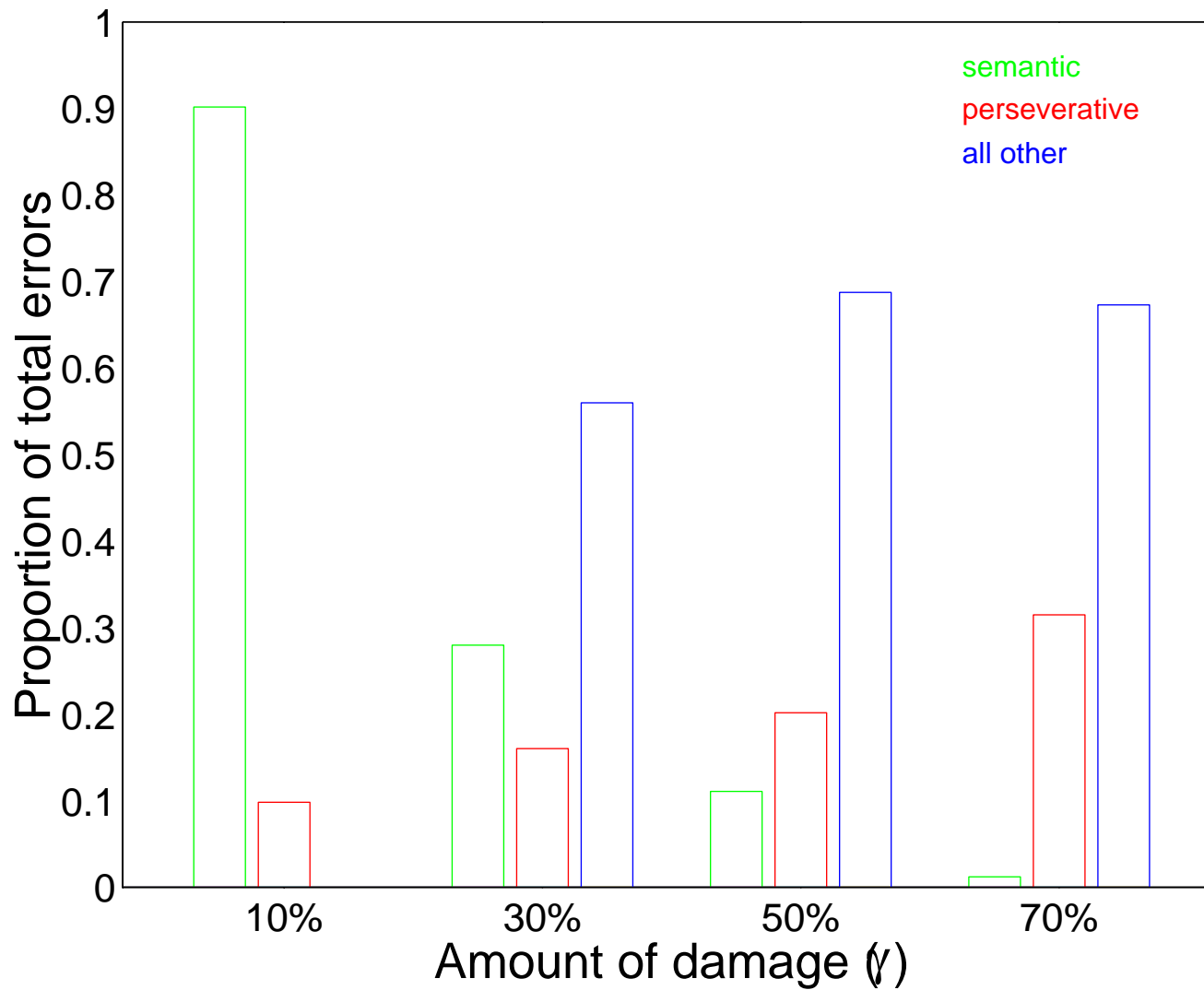
# Homing In



## Predictions of Model

- “Homing in” behavior with cued report
- $V \rightarrow G$  homing in, but no  $A \rightarrow N$  homing in
- With increased damage, fewer semantic errors and more perseverative errors as a proportion of total errors
- With increased damage, decreased superadditivity
- Might find patients with superadditive damage to two other pathways (e.g.,  $V \rightarrow S$  and  $S \rightarrow G$ )

# Error Distribution as a Function of Amount of Damage

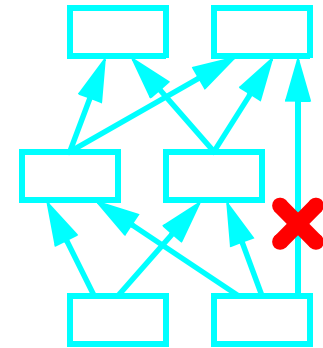


# Superadditivity

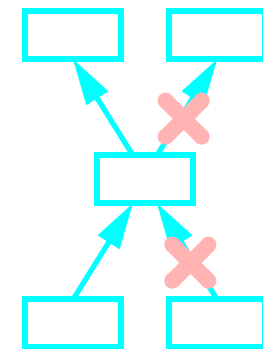
<b>Amount of Damage (<math>\gamma</math>)</b>	<b><math>\frac{\text{error}_{V \rightarrow N}}{\text{error}_{V \rightarrow G} + \text{error}_{A \rightarrow N}}</math></b>
<b>10%</b>	<b>3.00</b>
<b>30%</b>	<b>4.02</b>
<b>50%</b>	<b>1.68</b>
<b>70%</b>	<b>0.61</b>

# Summary of Optic Aphasia Model

Neuropsychological disorders have traditionally been explained by a focal lesion to a single processing pathway.



Farah (1990) argued that certain highly-selective deficits might have a parsimonious account in terms of multiple lesions with *superadditive* or *synergistic* effects.



We illustrated the viability of this account via a connectionist model of *optic aphasia*.

Next, we show the robustness of this account by applying it to an unrelated deficit using a different modeling substrate.

# Other Highly Selective Cognitive Impairments

## Language

Deficit in verb naming and reading aloud, versus deficit in writing responses involving nouns

Selectivity along two dimensions associated with different components of a cognitive architecture: grammatical category and response mode

→ separate orthographic output lexicon for nouns?

## Content-specific neglect

Unilateral neglect to just faces, human bodies, number, or words

Selectivity to both side of space and visual content

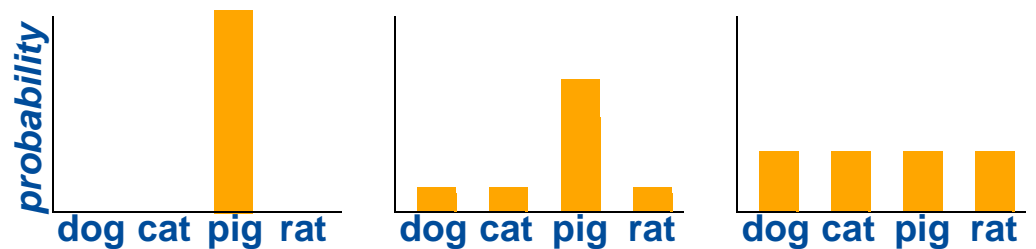
→ category-specific spatial attention systems?

# Probabilistic Modeling Framework I

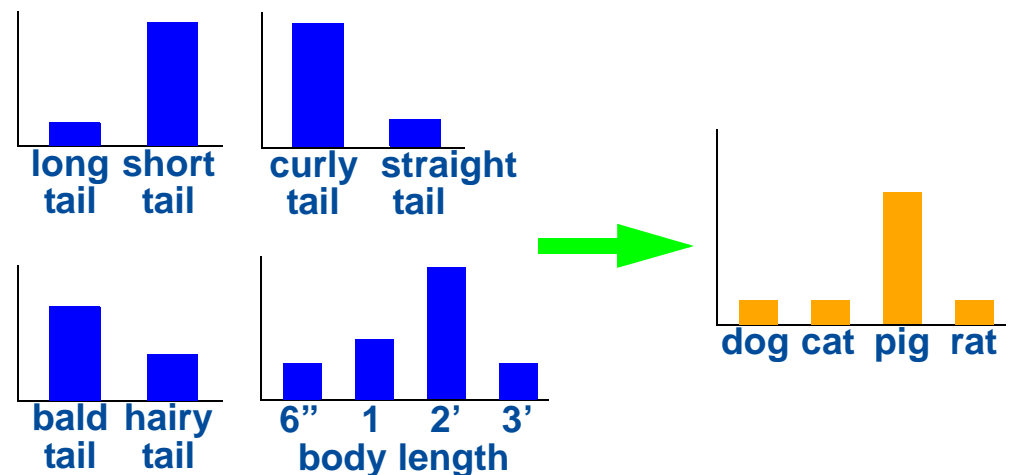
- Cortical computation is performed by a set of functionally specialized *pathways*.



- Internal representations characterized by *probability distributions* over alternatives.



- Pathways perform *probabilistic inference* (Bayesian belief revision) to transform input distribution to output distribution, based on previously acquired knowledge.



# Probabilistic Modeling Framework II

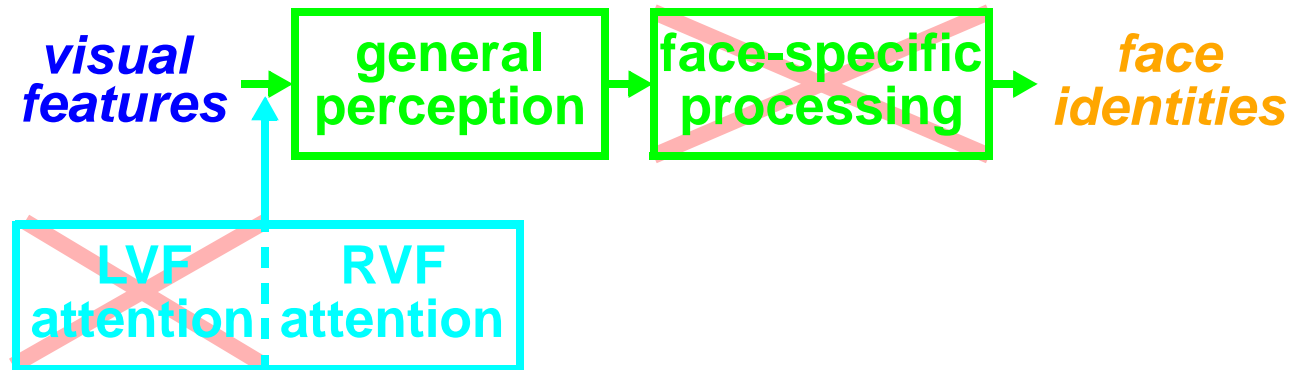
Model cognitive tasks via several pathways in cascade.



Framework is powerful tool for understanding cortical information transmission, yet makes only weak assumptions:

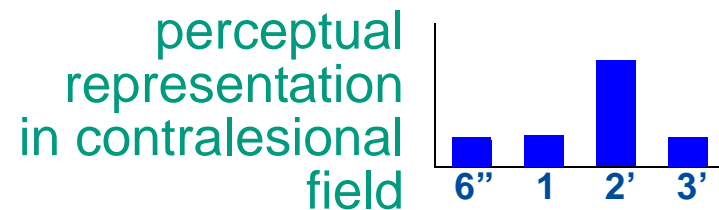
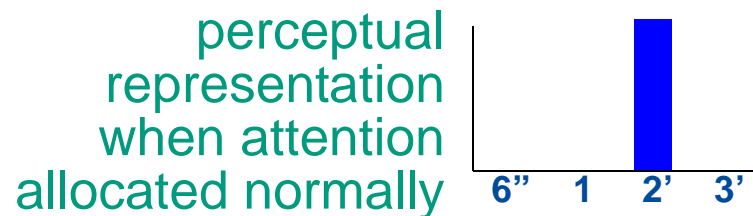
- speed-accuracy trade off
- continuous communication between pathways
- optimal probabilistic inference given prior learning

# Modeling Content-Specific Neglect



## Two focal lesions

- Attentional deficit in one hemifield, which gives rise to degradation of perceptual representations



- Distortion of mapping in face-specific processing i.e., noise added to  $p(x|y)$  conditional distribution

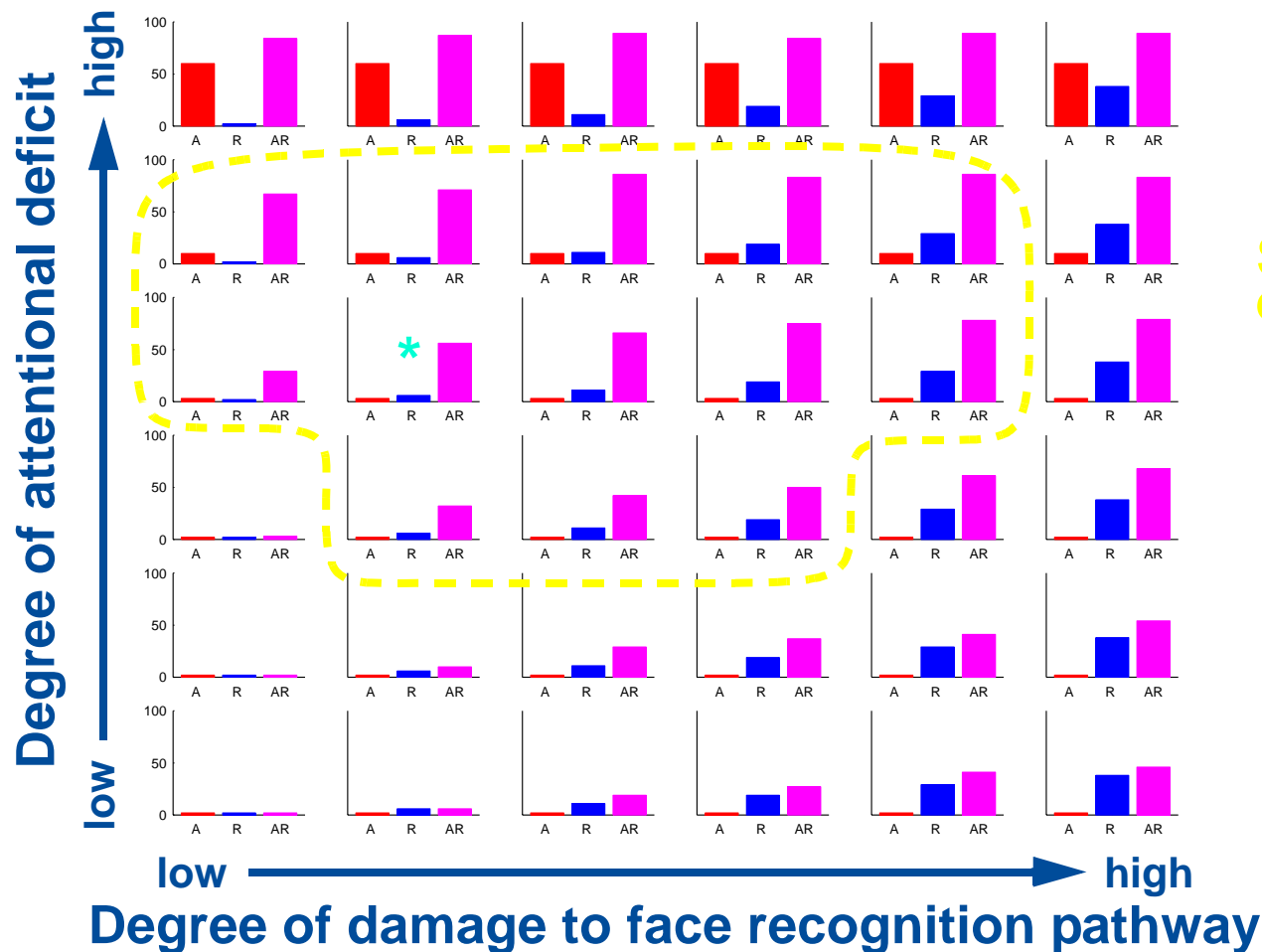
# Results

**Strong synergistic effects were observed. For example\***,

Error rate on objects other than faces in neglected region of space was 3%.

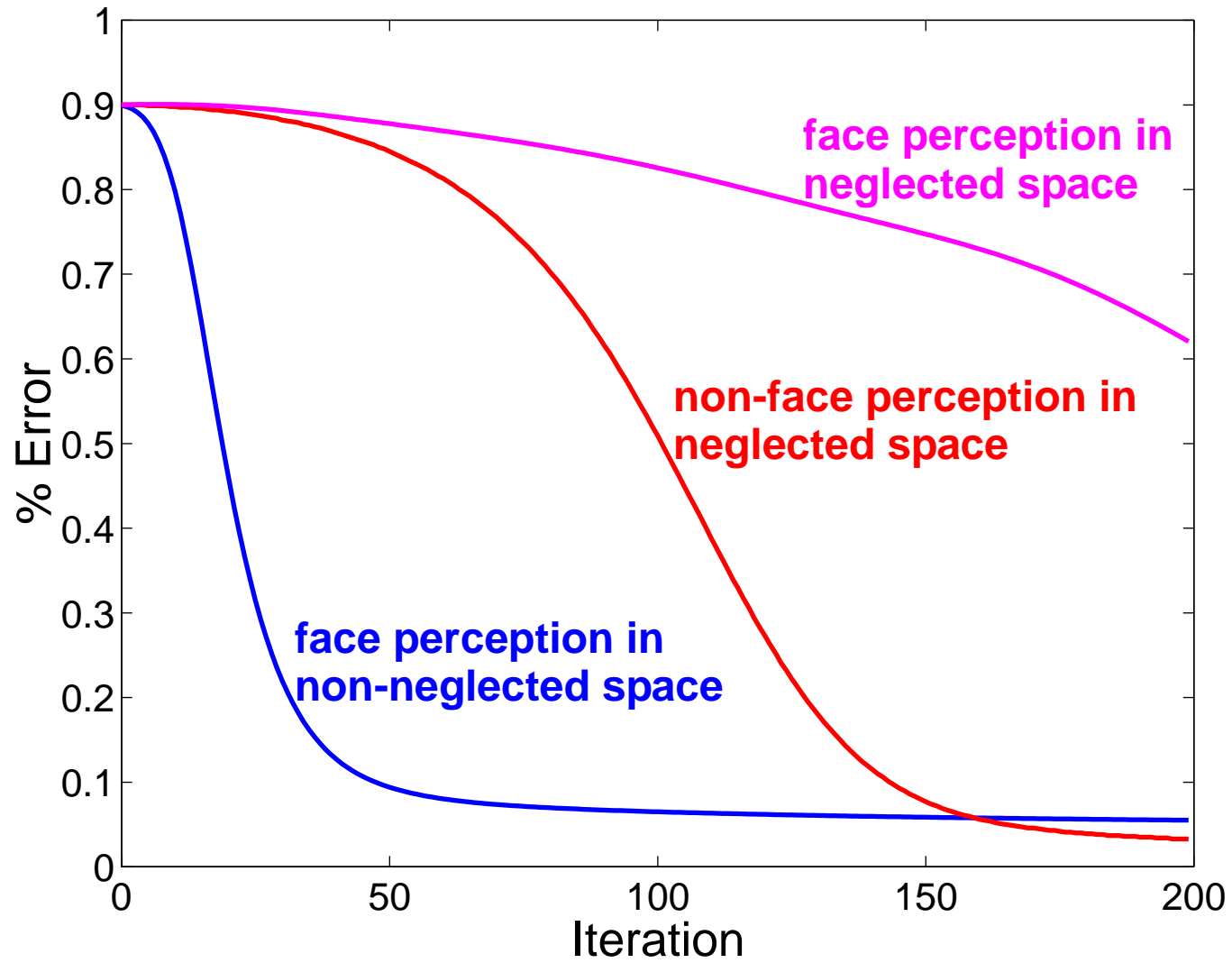
Error rate on perception of faces in non-neglected region of space was 6%.

Error rate on face perception in neglected region of space was 56%.



Synergistic effects

# Results



# Discussion

**Models show significant performance deficit when task requires two lesioned components, but little or no deficit when task requires just one of the lesioned components.**

**By hypothesizing multiple lesions, each with a single dimension of selectivity, we can account for highly selective deficits without positing implausible, counterintuitive, and unparsimonious cognitive architectures.**

**The “synergistic effect of multiple lesions” account**

- can serve to explain a range of neuropsychological syndromes
- can be instantiated in a variety of models, from feedforward neural networks to attractor neural networks, to abstract probabilistic networks.

**Key properties of these models**

- gradual convergence of pathway output on best interpretation over time
- continuous availability of information from other pathways