FOCAL LESIONS
Language-Aphasias
Functional Localization in the Cerebral Cortex-Focal Lesions

A. Representation of movement

**Apraxia:** inability to execute a normal volitional act, even though the motor system and mental status are relatively intact and the person is not paralyzed. The lesions affect cerebral areas around or distant from the primary motor area but do not involve it. The apraxias differ from the well-categorized lower motor neuron, pyramidal, cerebellar and basal ganglia syndromes. The patient behaves as if the motor engrams or templates for movement have been lost.

B. Representation of primary sensation

C. Sensory association areas

**Agnosia** is the inability to recognize the symbolic significance or meaning of a sensory stimulus, even though the sensory pathway and primary sensory cortex are sufficiently intact to register the stimulus and the mental status is relatively intact. Patient with agnosia acknowledge the presence of a stimulus, but are unable to report exactly what it is. Agnosias can have both a lexical aspect – a mismatching of verbal or cognitive symbols with sensory stimuli – and a mnemonic component- a failure to recall stimuli when confronted with them again.
Somatosensory agnosia: astereoagnosia, astatognosia (loss of position sense)

Visual agnosia: prosopagnosia, color agnosia, color blindness (acromatopsia)

Auditory agnosia: word deafness

Dyslexia is the inability to recognize written words or the meaning of words (word blindness) in the presence of intact visual pathways and primary visual cortex.

Neglect: affected individuals deny awareness of sensory information in the affected field, even though sensation remains intact (an individual with contralateral neglect syndrome responds when his left arm is pinched, even though he/she may deny the arm’s existence)

D. Representation of the language

Aphasias: the inability to understand or express words as symbols for communication, even though the primary sensory systems, motor mechanisms or phonation, and mental state (sensorium) are relatively intact
Model of the neural regions associated with the production of skilled actions. The premotor area of the contralateral hemisphere are essential for skilled movements of the limbs. These areas receive input from the parietal lobe of the left hemisphere, an area, assumed to store the representations of the actions. Thus, a lesion in the posterior parietal area will lead to apraxic movements with both contra-and ipsilesional limbs (Gazzaniga et al., 2002).

(a): Motor; (b) perception test. (c): Patients with either anterior or posterior lesions who produced apraxic gestures on the motor task were selected. Only the patients with posterior lesions showed impairment on the perception test. The apraxic patients with anterior lesions performed as well as nonapraxic (Heilman et al., 1982; Gazzaniga).
Deficits in motion perception: Akinetopsia

For the patient with motion blindness, the world appears as static images; there is no continuity from one image to the other.
Lesions in the M System lead to Akinetopsia

Temporary brain inactivations using transcranial magnetic stimulation (TMS) over area MT can produce deficits in motion perception. Results are rates of accuracy in determining the direction of motion as a function of the time between stimulus onset and the TMS pulses applied over area MT just prior to the onset of the stimulus(a), V1 (c) and an extrastriate region between these two (b). When the TMS was delayed until 40 msec after stimulus onset, the performance was perfect. Note that much longer delays did TMS disrupt performance over V1, suggesting that MT may receive direct input from LGN M bypassing V1. Beckers and Zeki, 1995, From Gazzaniga et al., 2002
Damage to Temporal Cortex Leads to Visual Agnosia

- Patients can describe the visual features of an object, but cannot name the object. The patient insisted that the object is a telephone.
- Patients can easily name objects presented through other modalities (auditory description, smell, etc.)
- A particular type of visual agnosia, called prosopagnosia involves impaired recognition of faces (Gazzaniga et al., 2002)
DF, a 34-year old patient, suffered carbon monoxide intoxication, and the MRI revealed bilateral occipital lesions. She had severe disorder of object recognition. When asked to name household items, she made errors such as labeling a cup an ashtray or a fork a knife. She usually gave crude description of an object; for example a screwdriver was ‘long, black and thin’. Her deficit was not anomia (a problem with naming objects); whenever an object was placed in her hand, she identified it. Her visual acuity was normal. (a) DF was asked to view a circular block into which a slot has been cut. In the recognition task DF was given a card and asked to orient her hand so that the card would fit into the slot. She failed, she oriented the card vertically even the slot was horizontal. (b) When asked to insert the card into the slot, DF quickly reached forward and inserted the card. (c) The patient also did not show any impairment in the memory condition, verifying that her knowledge of orientation was intact. Df-s performance shows that processing systems make use of different sources of perceptual information. From the first task, it is clear that DF could not recognize the orientation of a 3D object (‘what’ deficit). She was OK with the where, accordingly the where system appears to be essential for more than determining the location of the different objects; it is also critical for guiding interactions with these objects. Indeed, Goodale and Milner (1995) suggested to replace the ‘where’ system with the ‘how’, to emphasize that the dorsal visual system provides a strong input to the motor system to compute how a movement should be produced (From Gazzaniga et al., 2003).
Alexia is Caused by Damage to the Left Angular Gyrus

- Alexia is a deficit in the perception of words
- Ball may be misread as doll or snake as stale

Alexia and Prosopagnosia do not occur together, but agnosia for objects is always accompanied by a deficit in either word or face perception (Gazzaniga et al., 2002)
Diagram from Dejerine’s 1892 paper showing the lesion that results in pure alexia. The lesion is shown from the inferior surface of the brain. It has destroyed the left visual cortex and interrupted fibers from the right visual cortex on their way to language centers in the left hemisphere.
Optic ataxia: problem with the where (how) system

Patient with optic ataxia can recognize the object yet cannot use visual information to guide their action (From Rafal, 2003)

Figure 2.5
Optic ataxia in Bálint’s syndrome.
Unilateral Damage to Parietal Cortex Leads to Hemispatial Neglect

(Gazzaniga et al., 2002)
LANGUAGE AND APAHASIAS
A depiction of the left hemisphere of the brain showing the main language areas. The area in the inferior frontal lobe is known as Broca’s area, and the area posterior tri-lobe area (temporal-parietal-occipital) is known as Wernicke’s area. Broca’s area is adjacent to the motor cortex and is involved in planning speech gestures. It also serves other language functions, such as assigning syntactic structure. Wernicke’s area is adjacent to the primary auditory cortex and is involved in representing and recognizing the sound patterns of words.
PLANUM TEMPORALE-Language lateralization

Upper surface of the human temporal lobes exposed by a cut on each side in the plane of the Sylvian fissure. The posterior margin (PM) of the planum temporale (PT) slopes backward more sharply on the left than on the right, so that end ‘y’ of the left Sylvian fissure lies posterior to the corresponding point on the right. The anterior margin of the planum formed by the sulcus of Heschl (SH) slopes forward more sharply on the left. In this brain there is a single transverse gyrus of Heschl (TG) on the left, but two on the right (TG1, TG2). T-temporal pole, OP-occipital pole (Geschwind and Levitsky, 1968)
Depiction of a horizontal slice through the brain showing asymmetry in the size of the planum temporale related to lateralization of language.
Connections involved in naming a seen object and in reading. The visual pattern is transferred from the visual cortex and association areas to the angular gyrus, which arouses the auditory pattern in the Wernicke area. The auditory pattern is transmitted to the Broca area where the articulatory form is aroused and transferred to the contiguous face area of the motor cortex. With destruction of the left visual cortex and splenium words perceived in the right visual cortex cannot cross over to the language areas and the patient cannot read.
Passively viewing words (occipital lobe)

Listening to words (temporal lobe)

Reading aloud
Bilateral in the motor ctx, insular ctx and middle cerebellum

Generating words
Left frontal, anterior cingulate, posterior temp, right cerebellum
Increased blood flow in Broca’s area when subjects are processing complex relative to SIMPLE syntactic structures (Caplan et al., 2000; Gazanigga et al., 2002)
Heschl’ gyri, the site of primary auditory ctx is indicate in purple. Blue are areas in the dorsal STG that are activated by frequency-modulated tones than by random noise. Yellow areas in the STS are speech specific, that is they show more activation for speech sounds (words, pseudowords, or reverse speech) than for nonspeech sounds. Finally red areas include regions of the MTG, ITG, angular gyrus and temporal pole and are more active for words than for pseudowords or nonwords. (Binder et al., 2000; Gazzaniga et al., 2002)
HIERARCHICAL BRAINS SYSTEMS FOR WORD RECOGNITION:

First, the stream of auditory information proceeds from auditory cortex in Heschl’s gyri to the superior temporal gyrus (STG). Here, no distinction is made between speech and non-speech sounds.

Distinction is made between speech and non-speech sounds in the adjacent superior temporal sulcus (STS), but no lexical-semantic information is processed in this area. From the STS, the information proceeds to the middle and inferior temporal gyri, where phonological and lexical-semantic aspect of word is processed. The next stage involves analysis in the angular gyrus.

Broca area may be important for processing syntactic information. Another area for syntactic processing is area 22 in STG.

SUBSTRATES OF SPEECH PRODUCTION:

Basal temporal regions of the left hemisphere, left frontal operculum (Broca). The articulation of words involves the posterior part of Broca (area 44), bilateral activation of motor cortex, the SMA and the insula.
(a) PET activations in the anterior portion of the Superior temporal gyrus related to syntactic processing. (b) Lesions in the anterior STG that lead to deficits in syntactic processing (Gazzaniga)
Wernicke-Lichtheim-Geschwind model of language processing. The area that stores permanent information about word sounds is represented by A (Wernicke area). The speech planning and programming area is represented by M (Broca area). Conceptual information is stored in area B (supramarginal, angular gyri). From this model it was predicted that lesions in the three main areas, or in the connections between the areas, or the inputs to or outputs from these areas, could account for seven main aphasic syndromes (Caplan et al., 1994; Gazzaniga, 2002).
Broca’s aphasia

The speech output of this patient is slow and effortful, and it lacks function words. It resembles a telegram (top). Such patients may have a hard time understanding reversible sentences, where a full understanding of the sentences depends on correct syntactic assignment of the thematic roles (who hit whom?) (middle). Finally, these patients will have problems with speech articulation because of deficits in the regulation of articulatory apparatus (muscle of the tongue) (bottom) Gazanigga, et al 2002
Wernicke’s aphasia.

Such patients can speak fluently. If one is not a native speaker of a language, one might not detect there was anything wrong with the speech. The message in Dutch in the lower part of the figure might sound perfectly normal unless you speak Dutch. However, as the English translation in the top indicates, the speech output is often meaningless. They will also make semantic errors (television for telephone). Sometimes these patients will produce utterances that do not exist in the language (jargon: rommer).
<table>
<thead>
<tr>
<th>Aphasia Type</th>
<th>Symptoms</th>
<th>Disturbance</th>
<th>Location</th>
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<tbody>
<tr>
<td><strong>Broca's aphasia</strong></td>
<td>Speech output is slow, effortful, often misarticulated, missing function words, agrammatism</td>
<td>Disturbance in the speech planning and production mechanism</td>
<td>Posterior aspect of the IFG, insula, portions of the basal ganglia</td>
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<td>Wernicke's aphasia</td>
<td>Fluent-sounding speech, composed of meaningless strings of words, sounds and jargon, the inability to name objects</td>
<td>Disturbance of the permanent representations of the sound structures of word</td>
<td>Posterior half of the STG, junction between the parietal and temp. lobes, including supramarginal and angular gyri, the white matter underlying W's area</td>
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<td>Conduction aphasia</td>
<td>Disturbance of repetition and spontaneous speech (phonemic paraphasia)</td>
<td>Disconnection between the sound patterns of words and the speech production mechanism</td>
<td>Lesion in the arcuate fasciculus and/or cortico-cortical connections between W's and B's areas</td>
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<td>Transcortical sensory aphasia</td>
<td>Disturbance of single word comprehension with relatively intact repetition</td>
<td>Disturbance in activation of word meanings despite normal recognition of auditory presented words</td>
<td>White matter tracts connecting parietal lobe to temporal lobe or portions of inferior parietal lobule</td>
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<tr>
<td>Transcortical motor aphasia</td>
<td>Disturbance of spontaneous speech, similar to Broca's aphasia with relatively preserved repetition, comprehension</td>
<td>Disconnection between conceptual representations of words and sentences and the motor speech production system</td>
<td>White matter tracts deep to Broca's area connecting to parietal lobe</td>
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<tr>
<td>Disorder</td>
<td>Symptom</td>
<td>Mechanism</td>
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<tr>
<td>Pure motor speech disorder</td>
<td>Disturbance of articulation, apraxia of speech, dysarthria, anarthria, aphemia</td>
<td>Disturbance of articulatory mechanisms</td>
<td>Outflow tracts from motor cortex</td>
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<tr>
<td>Pure word deafness</td>
<td>Disturbance of spoken word comprehension repetition</td>
<td>Failure to access spoken words impaired</td>
<td>Input tracks from auditory system to Wernicke's area</td>
</tr>
<tr>
<td>Anomic aphasia</td>
<td>Disturbance in the production of single words, nouns. Intact comprehension, repetition</td>
<td>Disturbance of concepts, and or the sound pattern of words</td>
<td>Inferior parietal lobe or connections between parietal lobe and temporal lobe</td>
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<tr>
<td>Global aphasia</td>
<td>Major disturbance in all language functions</td>
<td>Disrupting of all language processing components</td>
<td>Large portion of the perisylvian association cortex</td>
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<td>Isolation of the language zone</td>
<td>Disturbance of both spontaneous speech (sparse, halting speech) and comprehension, with some preservation of repetition, echolalia</td>
<td>Disconnection between concepts and both representations of word sounds and the speech production mechanisms</td>
<td>Cortex just outside the perisylvian association cortex</td>
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