Some General Effects of Brain Damage

FRONTAL LOBE
If you would have been using it you wouldn't be about to lose it
Frontal Lobe Lesions

- exact character depends on location of lesion
- posterior lesions (in the motor areas) result in movement disorders
- anterior lesions (in the prefrontal cortex) result in much more complex changes
Frontal Lobe Lesions

[Diagram of the brain with labeled regions: Anterior premotor cortex (BA 8), Premotor cortex (BA 6), Primary motor cortex (BA 4), Dorsolateral prefrontal cortex (BA 9/46), Lateral frontopolar cortex (BA 10), Ventrolateral prefrontal cortex (BA 47/45/44), Ventral anterior premotor cortex (BA 44/6).]
Frontal Lobe Lesions

• disturbances of motor functions and movement programming up to and including hemiplegia (upper motor neuron disease)

• disturbances of voluntary gaze (patients don’t “look at the right places” to answer questions)
Frontal Lobe Lesions

- speech production - Broca’s aphasia (Broca’s area and supplementary speech area in left hemisphere)
Frontal Lobe Lesions

- disturbance in intelligence
  - but not in IQ test taking ability - convergent thinking (just one answer)
  - divergent thinking impaired - ability to come up with multiple answers
  - failure of abstract thinking - thinking becomes concrete
Frontal Lobe Lesions

example of divergent thinking
Frontal Lobe Lesions

- loss of behavioral spontaneity
- impaired strategy formation and decision making - esp. in novel situations
- impaired response inhibition
- perseveration
Frontal Lobe Lesions

Brain maps reveal where decisions happen

CALTECH / USC (US)
— By mapping the brains of patients with frontal lobe damage, scientists show that reasoning and behavioral control depend on a different area from decision-making.

MRI scans of a human brain show the regions significantly associated with decision-making in blue, and the regions significantly associated with behavioral control in red. On the left is an intact brain seen from the front—the colored regions are both in the frontal lobes. The image on the right is that same brain with a portion of the frontal lobes cut away to show how the lesion map looks in the interior. (Credit: CalTech)
Frontal Lobe Lesions

circuits responsible for impulsive behaviors (red, yellow, and orange)
Frontal Lobe Lesions

- distractibility and poor attention
- failure to attend to novel situations because of distraction

Figure 3
Viewing duration on novel and background stimuli (mean (SEM) in ms) for normal controls and frontal lobe patients. There was a significant stimulus type by group interaction ($p<0.009$). Normal controls spent significantly more time than frontal lobe patients looking at novel stimuli ($p<0.05$); however, the two groups did not differ in their viewing duration on background stimuli.

Effects of frontal lobe damage due to stroke on attention to novel stimuli.
Frontal Lobe Lesions

- failure to comply with instructions, rule breaking, risk taking, failure to learn from experience (impulsiveness)
- impaired autobiographical memory - e.g., patients can remember going to HS, they can tell what HS was like, but they can’t relate personal stories about HS
Frontal Lobe Lesions

some brain areas associated with memory
(notice prefrontal cx right in the middle of it all)
Frontal Lobe Lesions

• impaired social and sexual behavior - personality changes

• pseudodepression - apathy, indifference, loss of initiative, reduced sexual interest, reduced verbal output (more common with left hemisphere damage)
Frontal Lobe Lesions
(remember this guy)

Astrocytoma

History
This 35 male, non-smoker had a 2-year history of loss of initiative, depression, and rejection of his personal relationships. His wife noticed that he had slowly lost his drive to win all the big deals he always done so well at work. 3 months ago he began to experience headache, which did not respond to acetaminophen or aspirin. His wife noticed that his lethargic state had increased in the past few months. 3 days ago his right arm began to convulse uncontrollably for 1 minute. The patient shrugged the incident off as some aberrant behavior and did not reveal this to wife. 1 day ago the patient began again violently shaking his right arm, and the right side of face began to twitch at the dinner table. His wife panicked and called 911. He presents to this hospital without fever, change in appetite, or fatigue.

Physical
Remarkable findings are bilateral papilledema, increased deep tendon reflexes of the right bicep, tricep, and, Babinski sign on the right foot, reduced leg strength on the right.
Frontal Lobe Lesions

- impaired social and sexual behavior - personality changes (continued)
  - pseudopsychopathy - immature behavior, lack of tact and restraint, profanity, promiscuity, lack of social grace (often said to be more common with right hemisphere damage, but may be due to damage in the orbitofrontal cortex)
Frontal Lobe Lesions

(remember this guy)

Phineas Gage

It is uncertain what the extent of damage was in Gage’s frontal lobes. Some sources say both frontal lobes must have been damaged, but others (including Harlow, who cared for him after the accident) maintain that the right frontal lobe remained largely in tact.
Temporal Lobe Lesions

- disturbances in auditory sensation and perception
- difficulty discriminating speech sounds and “tone of voice” (prosody)
- difficulty with music perception (loss of melody)
- auditory hallucinations - due to spontaneous activity in the auditory cortex
Temporal Lobe Lesions

Auditory Functional MR Imaging

fMRI of auditory cortex responding to music

Fig. 2. —Auditory functional MR imaging with music. Axial plane is parallel to sylvian fissure at Heschl’s gyrus level. Intensity of activation varies from high (yellow) to low (blue). Bilateral activation of primary auditory cortex is seen, with extension to secondary auditory cortex more pronounced on left.
Temporal Lobe Lesions

fMRI of patient having auditory hallucinations
Temporal Lobe Lesions

- Wernicke’s aphasia - inability to comprehend or produce meaningful speech (damage to left posterior superior temporal gyrus)
Temporal Lobe Lesions

- disturbances in visual perception
- visual agnosia
- prosopagnosia
Temporal Lobe Lesions

Cecilia Burman has always had a problem with faces. As a child, she struggled to pick out her own face in school photos, and she is hard-pressed today to describe her mother's features. Over the years she has offended countless friends, passing them on neighborhood streets or in office hallways like strangers. "People think I'm just snobby," says Burman, 38, a computer consultant in Stockholm. "It makes me really, really sad to lose new friends because they think I couldn't bother to say hello."

prosopagnosia
prosopon = face, agnosia = no knowledge
Temporal Lobe Lesions

- anterograde amnesia - due to ventral temporal and hippocampal damage

H.M. (Henry Molaison)

Clearly evident here is necrosis of the left temporal lobe following infection by Herpes Simplex Virus Type 1.
Temporal Lobe Lesions

- temporal lobe personality (very few patients display all of this syndrome) - sometimes seen in temporal lobe epilepsy (Geschwind)
  - overemphasis on trivia and petty details of daily life
  - pedantic speech and egocentricity
  - “stickiness” - person persists in discussing personal problems, and you “get stuck listening to him”
  - paranoia
  - obsessive preoccupation with religion
  - proneness to aggressive outbursts
  - hypersexuality
Occipital Lobe Lesions

- blindness for all or part of the visual field - cortical blindness
- visual agnosia - failure of object recognition (occipitotemporal)

pictures drawn by a patient with visual agnosia
Occipital Lobe Lesions

- visuospatial agnosia - topographical disorientation (occipitoparietal)

- prosopagnosia - facial agnosia (including for their own face in severe cases); lesions tend to be occipital and ventral temporal

Cortical abnormalities in congenital prosopagnosia
Occipital Lobe Lesions

- alexia - inability to read (left hemisphere damage)
Occipital Lobe Lesions

- failure of visual imagery

Parietal Lobe Lesions

- somatoperceptual disorders
- loss of tactile sensation
- astereognosia - inability to recognize objects by touch
- asomatognosia - loss of body sense and condition
Parietal Lobe Lesions

• contralateral neglect (esp. after right hemisphere damage)
Parietal Lobe Lesions

- optic ataxia - failure of visually guided movements
- apraxia - a peculiar loss of skilled movements in response to directions (esp. after left hemisphere damage, but some forms after right as well); a “disconnection syndrome”
Explanation of the limb apraxias

verbal command

TACx (left)
interpretation of speech

PACx (left)
position of limbs and body

posterior corpus callosum

FACx (left)
planning of movements

to control of right hand and arm

lateral corticospinal tract

alpha motor neurons

muscles of left hand and arm

PACx (right)
location of objects in 3D space

FACx (right)
planning of movements w/ left hand

FMCx (right)
execution of movements w/ left hand

LESIONS

1 = left limb apraxia
2 = left limb apraxia w/right limb paralysis
3 = bilateral limb apraxia
Parietal Lobe Lesions

- failure of spatial cognition - e.g., mental rotation

Figure 1: Based on Shepard & Metzler's 'Mental Rotation Task'

Figure 2: Mental Rotation Task Based on Canonical Orientations
Parietal Lobe Lesions

- impaired IQ testing ability - failure of convergent thinking
Parietal Lobe Lesions

• agraphia - inability to write (left hemisphere damage)

meningioma resulting in apraxia, agraphia, and right foot paralysis
Parietal Lobe Lesions

- acalculia - inability to do arithmetic (left hemisphere damage)
- right-left confusion (esp. after left hemisphere damage)
- language disturbances - alexia and aphasia (left hemisphere damage)
Parietal Lobe Lesions

- language disturbances - angular gyrus
Basal Ganglia Lesions

• movement disorders
  • akinesia - no movement
  • bradykinesia - slowed movement
  • hypokinesia - deficiency in movement (includes above)
  • dyskinesia - abnormal, involuntary movements
    • athetosis - slow, writhing movements, esp. in the hands
    • chorea (choreiform movements) - rapid, jerky movements
    • dystonia - writhing movements often involving large parts of the body, resulting in strange postures and body positions that can be very painful
  • hyperkinesia - too much movement (including dyskinesias)
Basal Ganglia Lesions

- tremor - uncontrollable shaking
  - essential (familial) tremor
  - drug-induced
- resting tremor - as in Parkinson’s disease
- intention tremor - more characteristic of cerebellar damage
Basal Ganglia Lesions

- palsy (paralysis)
- tics - as in Tourette’s disease
- obsessions and compulsions
Limbic System Lesions

- hippocampus
- seizures
- anterograde amnesia
- disorientation (loss of spatial memory)
- amygdala
  - emotional changes - particularly loss of fear responses
  - changes in social behavior - overfriendliness, inappropriate comments, lack of awareness of personal space (observed in S.M.)
Limbic System Lesions

Amygdala contribution to selective dimensions of emotion

Click on image to view larger version.

A. Amygdala lesion

B. Contrast lesion

Fig. 3
Illustrative lesions. (A) Bilateral lesion of the amygdala secondary to Herpes Simplex Encephalitis. Although only two of the six patients in the amygdala group had bilateral lesions, the lesions of this patient illustrate the range of completeness of unilateral damage to the amygdala in other patients. (B) Illustration of one of the smaller lesion in the lesion contrast group.
Limbic System Lesions

The Human Amygdala and the Induction and Experience of Fear

Although clinical observations suggest that humans with amygdala damage have abnormal fear reactions and a reduced experience of fear [1,2,3], these impressions have not been systematically investigated. To address this gap, we conducted a new study in a rare human patient, SM, who has focal bilateral amygdala lesions [4]. To provoke fear in SM, we exposed her to live snakes and spiders, took her on a tour of a haunted house, and showed her emotionally evocative films. On no occasion did SM exhibit fear, and she never endorsed feeling more than minimal levels of fear. Likewise, across a large battery of self-report questionnaires, 3 months of real-life experience sampling, and a life history replete with traumatic events, SM repeatedly demonstrated an absence of overt fear manifestations and an overall impoverished experience of fear. Despite her lack of fear, SM is able to exhibit other basic emotions and experience the respective feelings. The findings support the conclusion that the human amygdala plays a pivotal role in triggering a state of fear and that the absence of such a state precludes the experience of fear itself.

- Case study of patient SM, a rare human patient with focal bilateral amygdala lesions
- First investigation of the induction and experience of fear in such a patient
- SM failed to exhibit fear behaviors, and her fear experience was highly impoverished
- The human amygdala plays a pivotal role in triggering a state of fear