Some General Effects of Brain Damage

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

- lesions in the primary visual cortex cause blindness for all or part of the visual field - cortical blindness

- lesions in the visual association cortex or ventral pathway into the temporal lobe cause visual agnosia - failure of object recognition (ventral occipitotemporal cortex)

pictures drawn by a patient with visual agnosia
Occipital Lobe Lesions

- lesions in the dorsal visual pathway into the parietal lobe cause visuospatial agnosia - topographical disorientation (dorsal occipitoparietal cortex)

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

Cortical abnormalities in congenital prosopagnosia occur in the fusiform gyrus.
Occipital Lobe Lesions

- alexia - inability to read (left hemisphere damage) due to interruption of pathways between visual cortex and language areas in the parietal and temporal lobes (inferior parietal lobule, Wernicke’s area)

Tumor in the left occipital lobe resulting in alexia.
Occipital Lobe Lesions

- failure of visual imagery

This diagram shows the methodology of an experiment designed to show that visual imagery (ability to imagine a visual stimulus) results from activation in the visual cortex similar to what would happen if the person were actually looking at the real thing. The subject looked at either an X or an O. A recording of electrical activity in the visual cortex was fed into a neural network, which was trained to recognize the activity associated with each stimulus. The subject was then asked just to imagine an X or an O, and the neural network was still able to recognize the imagined letter from the electrical activity recorded in the visual cortex.

Parietal Lobe Lesions

- Somatoperceptual disorders result from lesions in the somatosensory cortex (in the postcentral gyrus) and somatosensory association cortex
- Loss of tactile sensation
- Astereognosia - inability to recognize objects by touch
- Asomatognosia - loss of body sense and condition (for example, as in the coma video, the inability to realize that one is in a wheelchair and unable to walk)
Parietal Lobe Lesions

- contralateral neglect (esp. after right hemisphere damage) - the patient ignores the left half of the world, does not eat food on the left side of his plate, does not dress the left side of his body, etc. (in the most severe cases)

Patient is asked to draw a copy of the image on the left, producing the drawing on the right.

Tumor in the right parietal lobe resulting in contralateral neglect.
Parietal Lobe Lesions

• optic ataxia - failure of visually guided movements due to damage in the visual areas of the parietal lobe that are responsible for spatial perception

• 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” (meaning it results from disconnection of two brain areas that perform related functions)
Parietal Lobe Lesions

The right frontal motor cortex (FMCx) controls movement on the left side of the body. The right frontal motor association cortex (FACx) plans those movements. If a person is given a verbal instruction to make a movement with his left hand, that instruction will be interpreted and understood in the left temporal association cortex (TACx), or Wernicke’s area. That information must then be relayed forward to the areas that control movement in the opposite (right) hemisphere. Lesions in this pathway will result in various kinds of limb apraxias.

If the lesion is in the anterior corpus callosum (1), that will produce a left limb apraxia. If the lesion is in the left frontal motor association cortex (FACx) (2), that will produce a left limb apraxia but will also impair movement of the right hand and arm (right limb paralysis). If the lesion is in the left parietal association cortex (PACx) (3), both the right and left motor control areas will be in tact, thus no paralysis, but the instructions will not be able to get through to either of those motor areas, thus creating a bilateral limb apraxia.
Parietal Lobe Lesions

- failure of spatial cognition - e.g., mental rotation tasks are failed, in which the patient has to imagine how an object would look if rotated in space.

Are these two objects the same?

Do these two objects look the same?

Which of the remaining three objects matches the object on the left?
Parietal Lobe Lesions

- impaired IQ testing ability - failure of convergent thinking (in which the test taker has to come up with the one right answer to a problem)

This quote has nothing to do with the topic, so far as I know. I just like it.
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 in which the person has difficult telling right from left (especially after left hemisphere damage)

• language disturbances - alexia and aphasia (left hemisphere damage, see next slide)
Parietal Lobe Lesions

- language disturbances - angular gyrus (which is part of the inferior parietal lobule - see the aphasia video)

- disconnection aphasias result due to damage here because this structure is in the pathway between Wernicke’s area and Broca’s area as well as being in the pathways from sensory cortices to language areas
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

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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.
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