Gross Structure of Cerebral Hemispheres

You have already looked at the blood supply of the cerebral hemispheres in lab, and will be dissecting and slicing the cerebral hemispheres to study deeper structures in a later lab. A detailed understanding of the blood supply of the cerebral cortex and its fiber systems is extremely important since blockage and rupture of these vessels accounts for a large proportion of the pathology of the brain. An extremely important point is that even though there usually are small anastomoses between branches of the different cerebral arteries or between cerebral arteries and dural arteries, these anastomoses are inadequate to supply sufficient blood to prevent loss of function. Typically, blockage of any one of the three arteries supplying the cerebral cortex (distal to their origin at the Circle of Willis) produces a rapid and irreversible destruction of a large region of cortex, although the exact extent of destruction is somewhat variable in different individuals as a result of differences in the extent of anastomoses.

The pattern of distribution of the three arteries to the cerebral cortex (anterior, middle, and posterior cerebral) is seen in figures 2-4. The arterial supply for some of the more important parts of the cortex is as follows:

- Visual cortex (areas 17, 18, 19) in occipital lobe: posterior cerebral
- Somatic sensory (areas 3, 1, 2) and motor cortex (areas 4 & 6): anterior cerebral and middle cerebral. The foot and leg representations which are found medially are supplied by the anterior cerebral; the remainder of the body is supplied by the middle cerebral.
- Auditory cortex (areas 41, 42): middle cerebral
- Wernicke’s (area 22) and Broca’s (area 45) speech areas: middle cerebral supplies the cortex, but anterior cerebral supplies some of the underlying white matter so that blockage of this artery can also interfere with speech.
- Medial aspect of temporal lobe, including much of the hippocampus (limbic system structure to be discussed in later lecture): posterior cerebral.

Brodmann’s areas

The numbers given to visual, somatic sensory, motor and auditory cortex listed above were assigned to these areas in the early 1900’s by a researcher named Brodmann. Amazingly, many of these boundaries that were delineated on the basis of the cellular appearance of the cortex (called cytoarchitecture) still have functional significance today. It is important that you learn the numbers for those areas that I have listed. Use the following figure (Fig 1) to learn their locations. Although the major lobes (frontal, parietal, temporal, and occipital) of the brain do not serve as functional areas, many clinicians speak of these zones so it is important to know the boundaries between frontal and parietal (the central sulcus) and between temporal and frontal (the Sylvian or lateral fissure). I would also like you to know the positions of the major areas of Brodmann as they relate to these lobes.
Venous Drainage

There are names for many of the veins that drain the cerebral cortex, but they are not really important because unlike the cerebral arteries anastomoses between these veins are so extensive that blockage of even large ones typically does not result in infarction of the brain (although it can occur). Rupture of large veins can be of significant clinical importance but we will not discuss it here.

Fig. 1: Brodmann’s map of the cerebral cortex. By the end of the course you should be familiar with areas 3, 1, 2 (somatic sensory); 4, 6 (motor); 5, 7 (parietal association); 8 (frontal eye fields); 17, 18, 19 (vision); 41, 42 (auditory); and 44, 45 (Broca’s area).
Fig. 2: Distribution of the cerebral arteries on the lateral surface of the brain. (note that the middle cerebral supplies the white, unshaded area)

Fig 3: Areas of distribution of the anterior, middle and posterior cerebral arteries on the ventral side of the brain.
Structure and blood supply of the internal capsule. All of the fibers entering and leaving the cerebral cortex from the thalamus, brainstem, and spinal cord pass through the internal capsule (Figs. 5 and 6). Only the fibers to the striatum bypass the internal capsule.

The descending fibers of the internal capsule consist of the corticospinal, corticobulbar, corticopontine, and corticothalamic fibers. All of these fibers collect in the corona radiata of the cerebral hemispheres (Figs. 5 and 6) before converging onto the internal capsule. Corticothalamic fibers pass directly from the internal capsule into the thalamus while corticospinal, corticobulbar, and corticopontine fibers continue into the cerebral peduncles (Figs. 5 and 6).

Ascending fibers that pass through the internal capsule consist almost entirely of thalamocortical axons. The exception is the projection from the small cell groups in the brainstem that use monoamines as neurotransmitters. These cells project directly to the cerebral cortex without relaying in the thalamus. Like descending fibers, ascending fibers in the internal capsule are distributed to the cortex via the corona radiata.

As seen in Fig. 5, the main body of the internal capsule radiates into the frontal and parietal lobes. The internal capsule also connects with the temporal and occipital lobes by its so-called sublenticular and retrolenticular parts. These portions of the internal capsule get their names because they pass ventral and posterior, respectively, to the lenticular nucleus, as illustrated in Fig. 7A and B. Note that at the level of the cross section in Fig. 5, the path between the main body of the internal capsule and temporal lobe is blocked by the lenticular nucleus, forcing the connection to occur at a more posterior level (as seen in Figs. 7A and 9, the retrolenticular part of the internal capsule passes ventral to the posterior part of the lenticular nucleus). An important point that you should remember is that the visual radiations are contained in the retrolenticular part, and the auditory radiations in the sublenticular part (Fig. 9).
Fig. 5: Cross section through the brain showing the main body of the internal capsule and the corona radiata.

Fig. 6: Drawing of a dissection demonstrating the continuity of the corona radiata, internal capsule, cerebral peduncle, and medullary pyramid.
Fig. 7: A. Level 12 showing the retrolenticular portion of the Internal Capsule (ICr). The ICr is the portion of the Internal Capsule that courses posterior (retro) to the lenticular nucleus (Putamen and Globus pallidus). B. Moving one level rostral, Weigert stained Level 13 shows the sublenticular portion of the Internal Capsule (ICs). Note that the ICs courses ventral to the lenticular nucleus which is represented here by a caudal segment of the Putamen.

Fig. 8: A. Drawing of a brain dissection showing gross relationships of thalamus, internal capsule, putamen, and the caudate nucleus. B. Horizontal section through thalamus, internal capsule, lenticular nucleus (putamen and globus pallidus) and caudate. ICr = retrolenticular portion of the internal capsule. Recall that the putamen and globus pallidus are together termed the lenticular nucleus because of the lens shape of their collective structure.
In horizontal sections, the internal capsule is V-shaped, with an anterior limb interposed between the caudate nucleus and lenticular nucleus, and a posterior limb between the thalamus and lenticular nucleus (Figs 8B and 9). The different fiber systems of the internal capsule are not randomly intermixed but rather organized in a consistent fashion (Fig. 9). Fibers destined for the brainstem and spinal cord are organized within the internal capsule as follows (Fig. 9): corticopontine fibers are located in the anterior limb, corticobulbar fibers at the genu (bend between anterior and posterior limbs) and corticospinal fibers in the posterior limb. Corticospinal fibers are topographically arranged with the upper body represented near the genu and the lower body posteriorly. As a consequence of this organization, lesions of particular parts of the internal capsule will result in loss of function restricted to part of the body.

Corticothalamic and thalamocortical axons that connect VPL and VPM with the cerebral cortex also pass through the posterior limb of the internal capsule. However, effects of lesions of this structure on somatic sensation are difficult to predict, perhaps as a consequence of a more diffuse organization of sensory than motor fibers. Remember that lesions of the internal capsule can result in combined motor and sensory deficits, although a pure motor deficit (contralateral hemiparesis) can also occur.

The internal capsule is supplied by the anterior and middle cerebral arteries via their medial and lateral striate (also termed lenticulostriate) branches, respectively, and from branches of the internal carotid, especially the anterior choroidal (Figs. 10 and 11). From a neurological standpoint the blood supply of the posterior limb and genu is most important since lesions in this region result in paralysis from interruption of corticospinal and corticobulbar fibers. The dorsal part of the posterior

Fig. 9: Schematic of a horizontal section through the internal capsule (right side of brain) showing its component fiber tracts and relationship to the thalamus, lenticular nucleus (putamen and globus pallidus) and the caudate nucleus. Horizontal shading denotes descending fibers (except for corticobulbar and corticospinal fibers which are unshaded), stippling denotes ascending fibers.

Compare to Fig. 8B for orientation.
limb and genu is supplied by the lateral striate arteries from the middle cerebral; the ventral part is supplied by the anterior choroidal and small unnamed branches from the internal carotid (Fig. 11). Since the visual radiations course through the retrolenticular part of the internal capsule, lesions of the internal capsule that produce a contralateral hemiplegia often result in a visual deficit as well (homonymous hemianopsia).
An extremely important point to remember is that because of the small size of the internal capsule a very small lesion can produce the same deficit as a very large lesion in the cerebral hemisphere. Lesions in the internal capsule are very common, especially from blockage of the internal carotid and middle cerebral arteries or their branches, and from hemorrhage from the striate arteries. Hemorrhage from the striate (lenticulostriate) arteries damages a larger area of the internal capsule and adjacent structures than supplied by these arteries. Because the striate/lenticulostriate arteries are so susceptible to hemorrhage (primarily in people with hypertension), they have been referred to as the “arteries of stroke”.

**Fiber Systems of Cerebral Cortex**

Much of the interior of the cerebral hemispheres is taken up by the fiber systems of the cerebral cortex. In addition to the 1) ascending and 2) descending fibers that have already been discussed, there are two other massive fiber systems within the cerebral hemispheres: 3) association fibers that connect different cortical areas on the same side of the brain, and 4) commissural fibers that connect the two halves of the cortex. The commissural fibers run in the corpus callosum and to a lesser extent in the anterior commissure.

**Association Fibers**

Numerous association fiber bundles are illustrated in Figure 12. The superior longitudinal fasciculus, which is the largest of the association bundles, connects the cortex of the frontal, parietal, occipital and temporal lobes. The inferior longitudinal fasciculus connects the temporal and occipital lobes. The uncinate fasciculus runs deep to the Sylvian fissure to connect the frontal lobe with the rostral part of the temporal lobe. The cingulum, which runs within the cingulate and parahippocampal gyri, will be discussed in the limbic system lecture. In addition to these long association bundles, short association fibers connect the cortex of adjacent gyri.
**Function of the Corpus Callosum**

The corpus callosum is sometimes cut in patients with epilepsy to prevent seizures from spreading to both sides of the brain. In everyday situations, deficits resulting from these sections are very subtle, but cleverly devised experiments have produced some remarkable findings. These experiments have demonstrated the problem that you would predict—basically that the two halves of the brain cannot communicate with each other when the fibers connecting them are severed. Since sensory information normally impinges on both ears, both eyes, etc., this does not usually present a problem. In experimental situations where information is presented only to one hemisphere, however, these people are found to have essentially two independent brains that differ markedly in many respects. Normally only one hemisphere (usually the left) is capable of speech, but the other hemisphere is much better at perceiving and manipulating spatial relationships. The two hemispheres even appear to have different “personalities.” These people sometimes demonstrate this latter finding in non-experimental situations since their facial expressions can contradict their verbal messages (when the verbal hemisphere is talking and the non-verbal hemisphere is controlling the facial expressions).

**Association areas of neocortex**

So far we have discussed areas of the brain, which had either a specific motor or sensory function. Areas without predominantly sensory or motor functions are referred to as association cortex. Such cortex is found in the frontal, temporal, parietal, and occipital lobes. Within these association areas, information from a variety of sources is assembled to allow very complex tasks to be carried out. Association areas receive inputs from thalamic nuclei and from other cortical areas by way of association axons.

In an earlier lecture you learned that there is considerable overlap in function between sensory and motor areas (e.g., for somatic sensation and movement). The distinction between association and sensory-motor areas is similarly blurred. This is exemplified by the secondary sensory and motor areas that are closely associated with a particular sensory modality or motor cortex, but whose functions are clearly more complex and “global” in nature than the primary areas.

Most of what is presently known about the function of association areas is derived from the effects of lesions. Functions that have been assigned to association areas in this fashion include: recognition of complex spatial patterns such as faces, language functions, perception of spatial relationships, integration of complex movements, decision making, control of social interactions, etc.

The cerebral hemispheres comprise a large fraction of the total volume of the brain and, as a result, are commonly affected by disease processes as well as by trauma. Cortical pathology is characterized by the complexity of symptoms. Pure sensory or motor deficits of a specific nature are rarely seen following cortical damage but, rather, sensory and motor problems tend to be combined with “higher order” dysfunctions involving thought processes, speech, emotions, or memory. This probably reflects the fact that primary sensory and motor areas cover only a relatively small fraction of the cortex and are immediately surrounded by association areas mediating more complex functions, which are usually damaged with the primary areas.

There is also a great deal of variability in the expression of deficits resulting from damage to apparently the same area of cortex in different patients. These variations may be due in part to a difference in the location of the representation of different functions in different individuals. Differences in the extent to which undamaged areas take over the function of damaged areas. Difficulties in comparing the locations and extents lesions in different clinical cases also presumably contribute to the apparent differences. You will encounter very few patients with small, well-localized lesions in the cerebral cortex. There is also great variation in the extent of involvement of deeper structures, and multiple lesions are commonly encountered.
Another striking feature of cortical pathology that is unique to the cortex is an asymmetry in the effects of damage to the two sides of the brain, reflecting an asymmetry in localization of function. The best-known example is speech. In most people, speech and other language functions are localized only in the left cerebral hemisphere, although in approximately 4% of right-handed and 17% of left-handed individuals, speech is represented predominantly in the right hemisphere. Speech is also represented bilaterally in some people, especially those with left-handedness. It is obviously important for a neurosurgeon to establish which side of the brain is the verbal side before performing surgery to remove tumors, epileptic foci, etc.

The two best-known areas that are concerned with speech and other language functions are named after the neurologists who first described them: Broca’s area in the frontal lobe and Wernicke’s area at the junction of parietal, temporal and occipital lobes (Fig. 13). Broca’s area corresponds to area 45 of Brodmann. Wernicke’s area corresponds to area 22 of Brodmann. The effects of lesions in these areas will be covered in the next lecture, but for now you should begin to associate damage to these areas with a deficit known as aphasia (see below).

![Fig. 13: The lateral surface of the left cerebral hemisphere showing the speech areas of Broca and Wernicke (to be considered in an upcoming lecture and discussions).](image)

An important aspect of cortical pathology that can be unexpected and devastating for relatives and friends of patients is an alteration in emotional expression and personality. Such changes are usually associated with damage to the prefrontal cortex, although they can also occur from damage to other cortical areas or subcortical structures (see olfactory-limbic system lecture notes). The prefrontal cortex is a large, somewhat ill-defined region that is anterior to the motor areas in the frontal lobe. It includes cortex on the medial and ventral aspects of this lobe as well as lateral. People with prefrontal damage can lose social awareness and complex abilities that are necessary for leading productive lives such as judgment, initiative, and concentration. Emotional responses and personality can be so profoundly altered that they can be very difficult to live with. In your small groups you will discuss the celebrated case of Phineas Gage who sustained damage to his prefrontal cortex in a mining accident.

An extensive and often confusing terminology has been generated to describe deficits resulting from
damage to association areas of cerebral cortex. Some of these terms will be introduced in the upcoming “clinical” lectures and case history discussions. In the way of a preview, three categories of deficits that you will consider are: **aphasia**, the disruption of speech functions; **agnosia**, loss of the ability to recognize objects or perceive spatial relationships in the absence of significant sensory impairment; and **apraxia**, loss of the ability to perform complex motor tasks in the absence of paralysis.

**SUMMARY OF IMPORTANT POINTS FROM CEREBRAL CORTEX LECTURES:**

1) **BLOOD SUPPLY.** You will see a very large number of patients with deficits resulting from problems with the blood supply of the cerebral hemispheres (cortex, internal capsule, and basal ganglia) even if you don’t become a neurologist or neurosurgeon. You should overlearn this material to the point where you won’t forget it. Don’t forget that beyond the Circle of Willis, anastomoses between the three cerebral arteries are usually insufficient to maintain more than a small part of the blood supply when one of these arteries is occluded.

2) Locations of primary and secondary or supplementary sensory and motor areas (3, 1, 2, 4, 6, 8, 41, 42, 17, 18, 19 of Brodmann), and Broca’s (area 45) and Wernicke’s (area 22) speech areas. Don’t forget that areas 3, 1, 2, and 4 extend onto the medial aspect of the hemisphere (foot and lower leg representation), which is supplied by the anterior cerebral artery. Brodmann’s numbers for the association cortex in the parietal lobe (5 & 7) are also commonly used. Comments concerning the importance of the blood supply also apply to this material.

3) **INTERNAL CAPSULE.** This material is also extremely important because lesions in this area are very common. Again, try to learn this material sufficiently well so that you won’t forget it! Individual points to concentrate on: (a) Spatial relationships between the different parts of the internal capsule (anterior and posterior limbs and genu) and the thalamus, caudate, and lenticular nucleus. You will have to have a clear picture of these relationships in your mind for both frontal and horizontal planes or you will not be able to interpret C-T scans (C-T scans are now made in both planes). (b) Positions of corticospinal and corticobulbar fibers, and topographical organization of corticospinal fibers in the posterior limb. (c) Blood supply for the genu and posterior limb. (d) Remember that very small lesions of the internal capsule can cause loss of function of large areas of cerebral cortex by severing their ascending and descending connections with subcortical structures. However, an important point is that unilateral lesions confined to the internal capsule usually do not compromise functions of association areas of cortex. For example, loss of motor and visual functions on the right with no aphasia could result from a lesion localized in the internal capsule.

4) In addition to ascending and descending fiber systems of the cerebral cortex, you should also know the commissural and the major association fiber systems.

5) The material on the association cortex can be considered to be a brief introduction to the upcoming lectures and small group discussions in the “clinical block” to follow.
OPTIONAL READING

PREVIEW OF ALZHEIMER’S DISEASE AND EPILEPSY

Two common neurological problems involving the cerebral cortex are Alzheimer’s disease and epilepsy. While discussion of the pathophysiology of these diseases is beyond the scope of this course, a brief description of Alzheimer’s disease with emphasis on recent discoveries follows:

Alzheimer’s dementia is a severe, progressive mental deterioration that can occur in relatively young individuals (40s or 50s) although it is more commonly a disease of old age. A great deal of excitement has been generated by the recent finding of an associated selective loss of large cholinergic neurons that project diffusely to virtually the entire cerebral cortex. These cells are widely dispersed in the basal forebrain but concentrated in the nucleus basalis, which is located lateral to the hypothalamus and ventral to the corpus striatum (see below). It should be noted, however, that it has not yet been demonstrated that loss of these cells causes the dementia. Since there are histological changes in neurons throughout the cerebral cortex in Alzheimer’s disease, and since cholinergic drugs have limited effectiveness in treatment of this condition, the role of the nucleus basalis is still an open question.