

Answer to Case History XII

- 1). Weakness in this case is probably due to cortical dysfunction and particularly cortico-spinal neuron dysfunction in the pre-rolandic motor strip. Pressure from the subdural fluid collection can lead to dysfunction of cortical neurons.

- 2). Inattention is a non-specific finding that can lead to apparent dysfunction of other higher cognitive processes. In general, any condition that has a diffuse effect on brain function can lead to inattention. Examples would be drug induced states, metabolic encephalopathies, or increased intracranial pressure. Depression can also lead to inattentiveness. In this example, the inattention and confusional episodes may relate to the mass effect of the subdural hematoma. Subdural hematomas are often associated with mental status changes that fluctuate, presumably because of fluctuation of intracranial pressure. Increased intracranial pressure leads to a variety of potential effects that may cause impaired cognition. These include dysfunction of frontal lobe systems, pressure on the diencephalon with impaired thalamo-cortical interactions, and decreased blood flow. Cerebral blood flow is autoregulated, but when intracranial pressure rises, cerebral blood flow is compromised. The ultimate level of dysfunction occurs when the intracranial pressure exceeds the mean arterial pressure. Then there is no cerebral blood flow and brain death ensues.

- 3). The brain does not feel pain so that increased intracranial pressure produces headache by other means. The dura and the cerebral blood vessels are heavily innervated by nociceptive nerve endings. Pain from increased intracranial pressure is from traction on these pain sensitive structures. Above the tentorium, these fibers are part of C.N. V distribution and pain is often referred to the area innervated by the ophthalmic division of the trigeminal nerve. The posterior fossa dura and vessels are innervated by C2-3 and cranial nerves IX and X so pain is referred to the posterior head and neck and occasionally the ear or throat.

- 4). The subdural space normal holds a thin film of fluid and is a space that can expand when bridging veins rupture and bleed into the space. The dura is plastered to the periosteum of the skull on one side and the arachnoid on the other. Venous sinuses are formed by the dura and drain bridging veins that pierce the dura. Tearing of these veins is the mechanism of most subdural hematomas. Because the flow pressure in the venous system is low, subdural hematomas may be insidious in their development and may not be symptomatic for days or weeks after an initial injury. As the hematoma changes in osmotic composition, the hematoma may expand by absorbing CSF.

- 5). As we get older our brains have some degree of normal atrophy. This creates a larger subarachnoid space and more tension on bridging veins. Some degenerative diseases like Alzheimers disease are associated with greater than normal cerebral atrophy. Relatively minor trauma can cause subdural hematomas in older people.

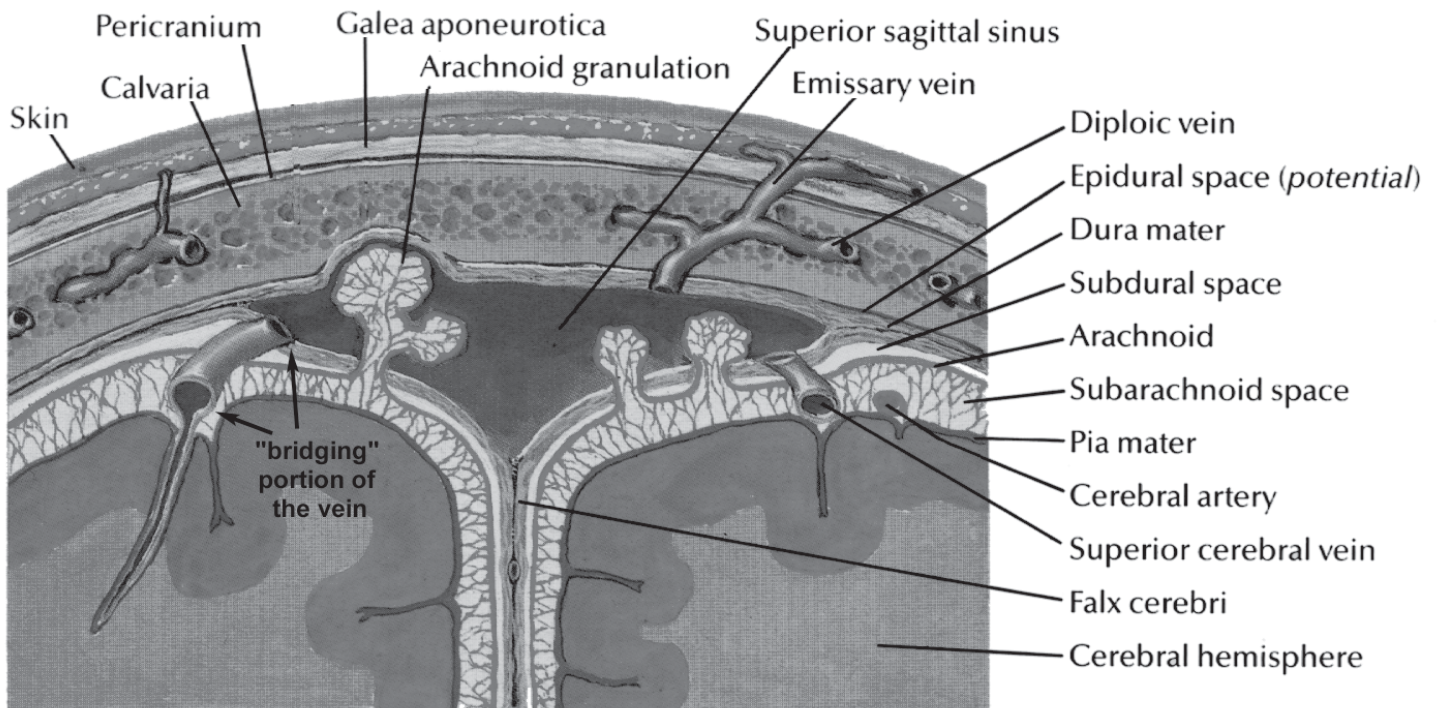
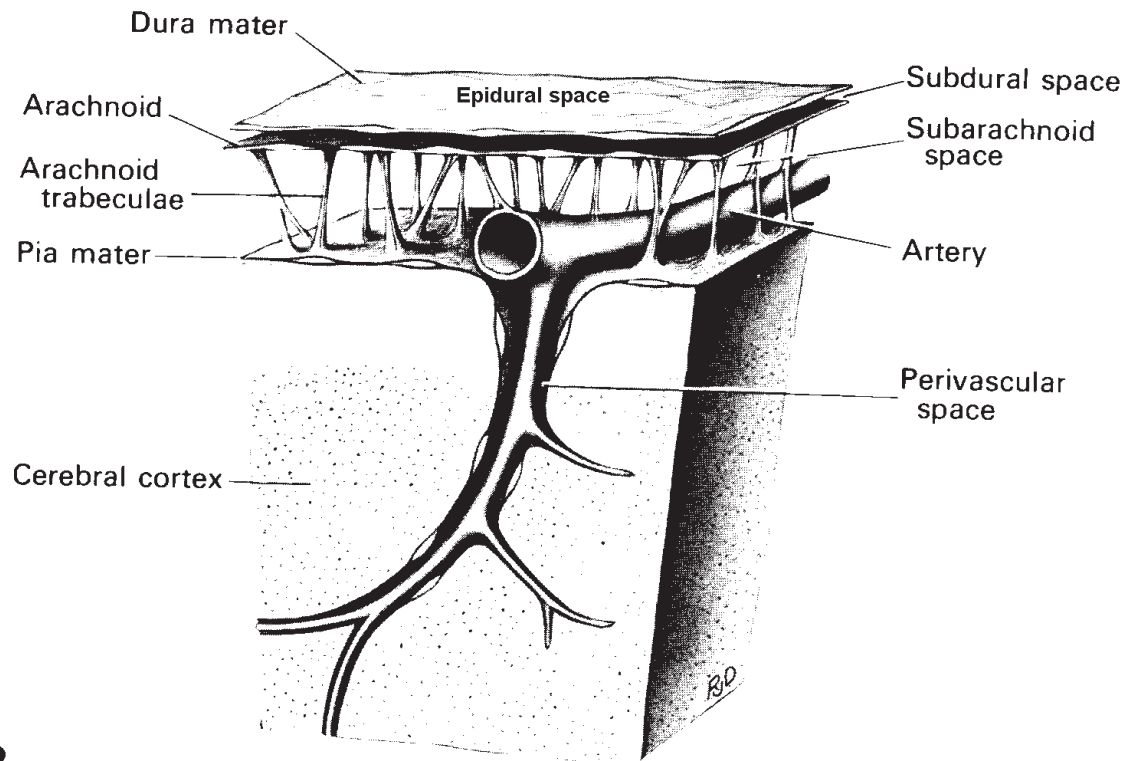


Figure 1: Two schematic diagrams of the relationship of the dura and subarchnoid space. A:

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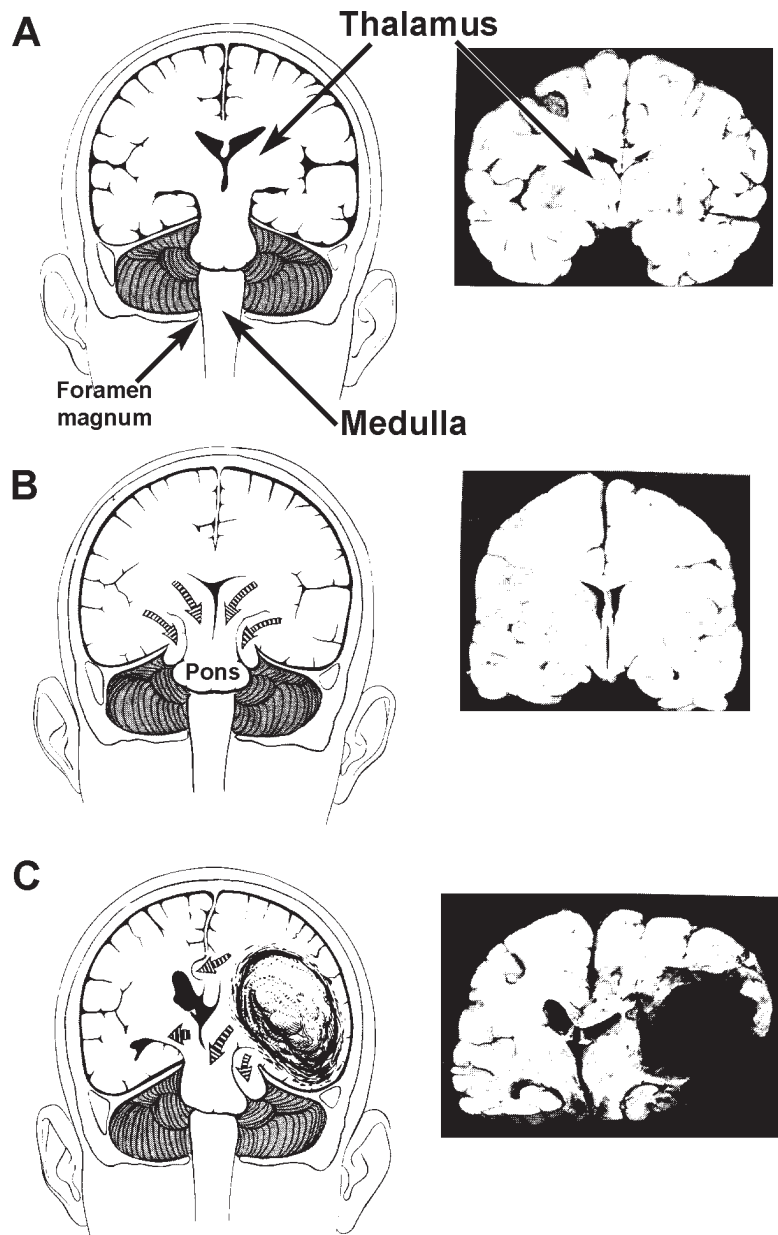
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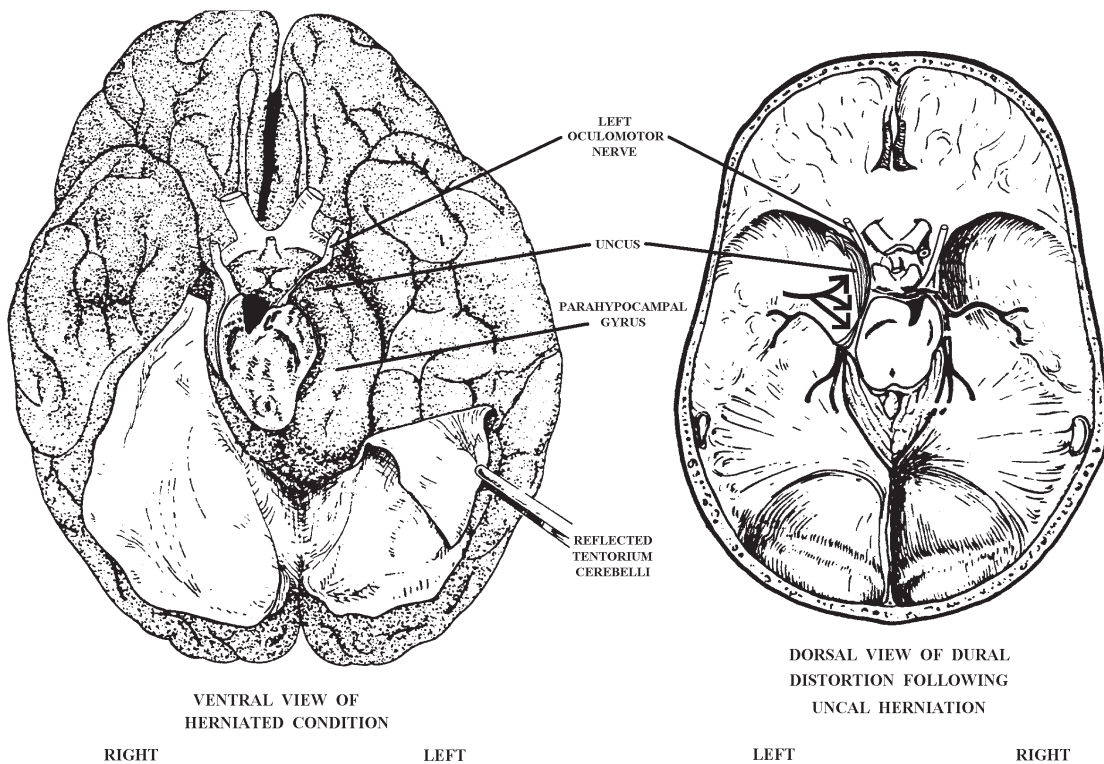
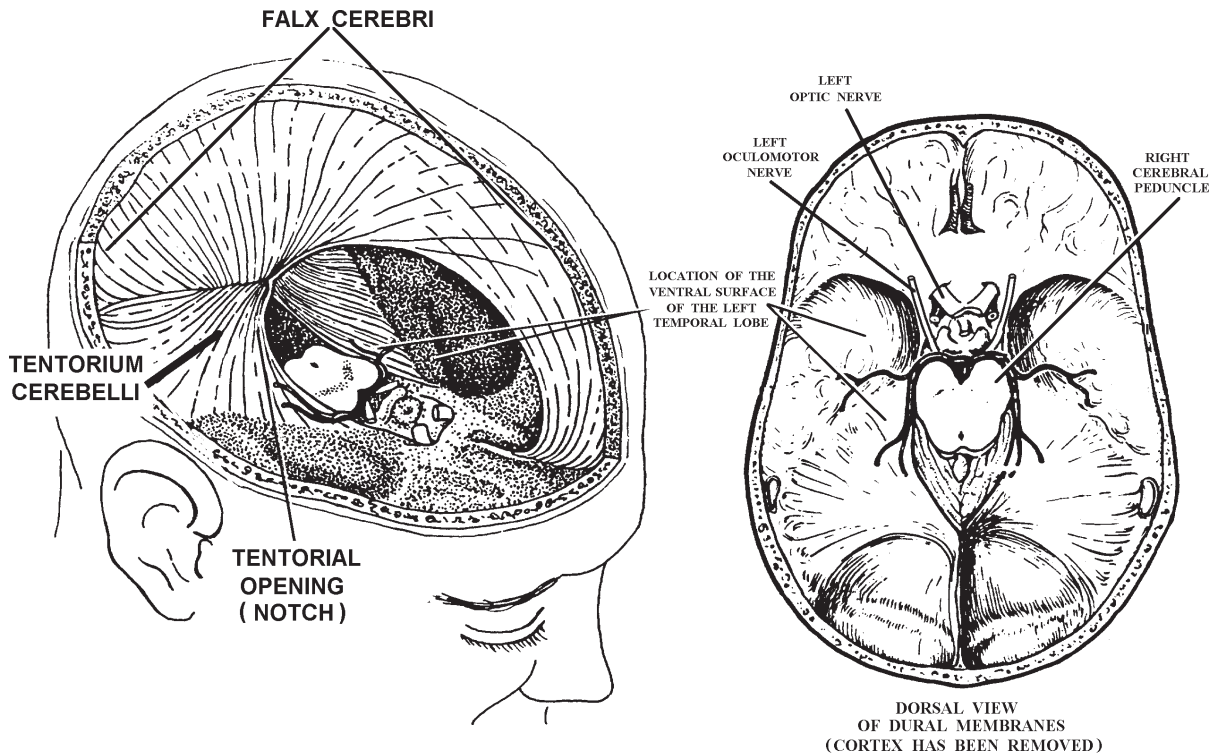
Relationship between the cortex and meninges showing how the dura forms venous sinuses that ultimately empty into the internal jugular vein. The arachnoid granulations drain CSF into the venous system. There are also veins that empty into the dural sinuses. B: Diagram to illustrate the subdural and subarchnoid spaces. The pia mater encompasses vessels as they penetrate the brain. The epidural space is at the top of the diagram and represents the space between the dura and skull. From Carpenter, M.B. **Human Neuroanatomy**, 7th edition.

Answer to Case History XIII

1). The CT scan showed a mass in the left middle fossa that was extraparenchymal and had a convex appearance. It was pushing on the left temporal lobe and the left ambient cistern (the CSF space adjacent to the midbrain) was obliterated. There was also a linear fracture of the left temporal bone. This fracture caused a laceration of the left middle meningeal artery and the formation of an epidural hematoma that formed between the periosteum and the dura. The pressure in the artery is much higher than the veins, and the result is a more rapidly expanding mass with significant increased intracranial pressure. Initially, however, there may be a "lucid interval" with minimal symptomatology before a mass effect develops.

2). The examination findings are due to compression of the left CN III. The pupillary fibers run on the outside of the nerve and are most susceptible to pressure. The weakness results from corticospinal tract involvement, mostly likely from cortical compression, but could also represent the ipsilateral cerebral peduncle being compressed. The patient's obtundation is produced by the increased intracranial pressure and its transmission to the diencephalon. The slow pulse rate and high blood pressure is in response to increased intracranial pressure and is termed **Cushing's** response. The response tries to maintain mean arterial pressure above intracranial pressure so that cerebral blood flow is preserved. The caloric response represents tonic deviation of the eyes toward the ear irrigated with cold water. There is the loss of the fast phase of nystagmus in coma. The fast phase of nystagmus is produced by the frontal eye fields trying to correct the slow deviation of the eyes produced by stimulation of the vestibular apparatus. The loss of left eye adduction in combination with pupillary abnormalities is consistent with a lesion of left CN III. If the pupil was normal, the caloric response would be consistent with a lesion of the third nerve nucleus or the left medial longitudinal fasciculus.





LEFT rapidly expanding cortical mass = LEFT uncus herniation = dilated pupil LEFT eye.
Also, the LEFT cerebral peduncle is compressed = damage to the LEFT corticospinal fibers
= RIGHT hemiplegia etc.

3). As the mass expands, the contralateral cerebral peduncle is compressed against the tentorium cerebelli causing corticospinal signs and is referred to as the syndrome of **Kernohan's notch**. A CN III palsy with ipsilateral hemiparesis from the cerebral peduncle compression opposite CN III is often termed a false localizing sign.

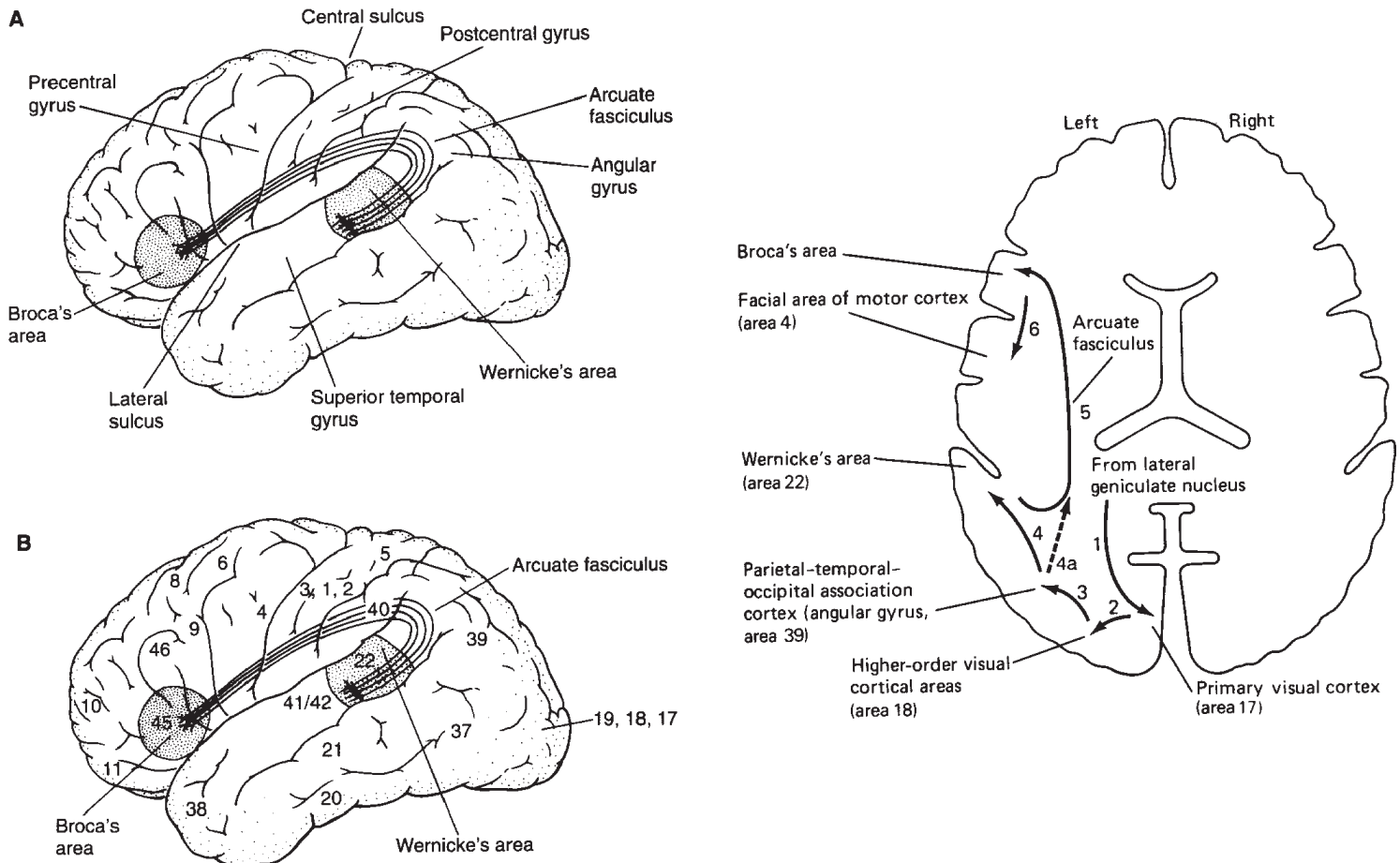
Answer to Case History XIV

This case demonstrates one of the most distinct forms of aphasia, known as Broca's aphasia. The case is taken nearly verbatim from Goodglass & Kaplan (1983).

BROCA'S APHASIA - This condition which often is referred to as nonfluent aphasia. The essential characteristics include: awkward articulation, restricted vocabulary, restriction of grammar to the simplest, most over-learned forms, and relative preservation of auditory comprehension. Utterances often are restricted to short telegraphic phrases consisting of nouns and verbs only, or even to single words. Reading is only mildly affected while written language is usually impaired at least as severely as oral language. Nonspeech oral movements often are affected. Often the individual may have awareness of the deficit.

Broca's aphasia arises from a lesion involving the third (inferior) frontal convolution of the left hemisphere, the subcortical white matter and extending posteriorly to the inferior portion of the motor strip (precentral gyrus). This condition often is seen with hemiplegia due to involvement of adjacent motor cortex. (from Goodglass & Kaplan, 1983, pg. 76).

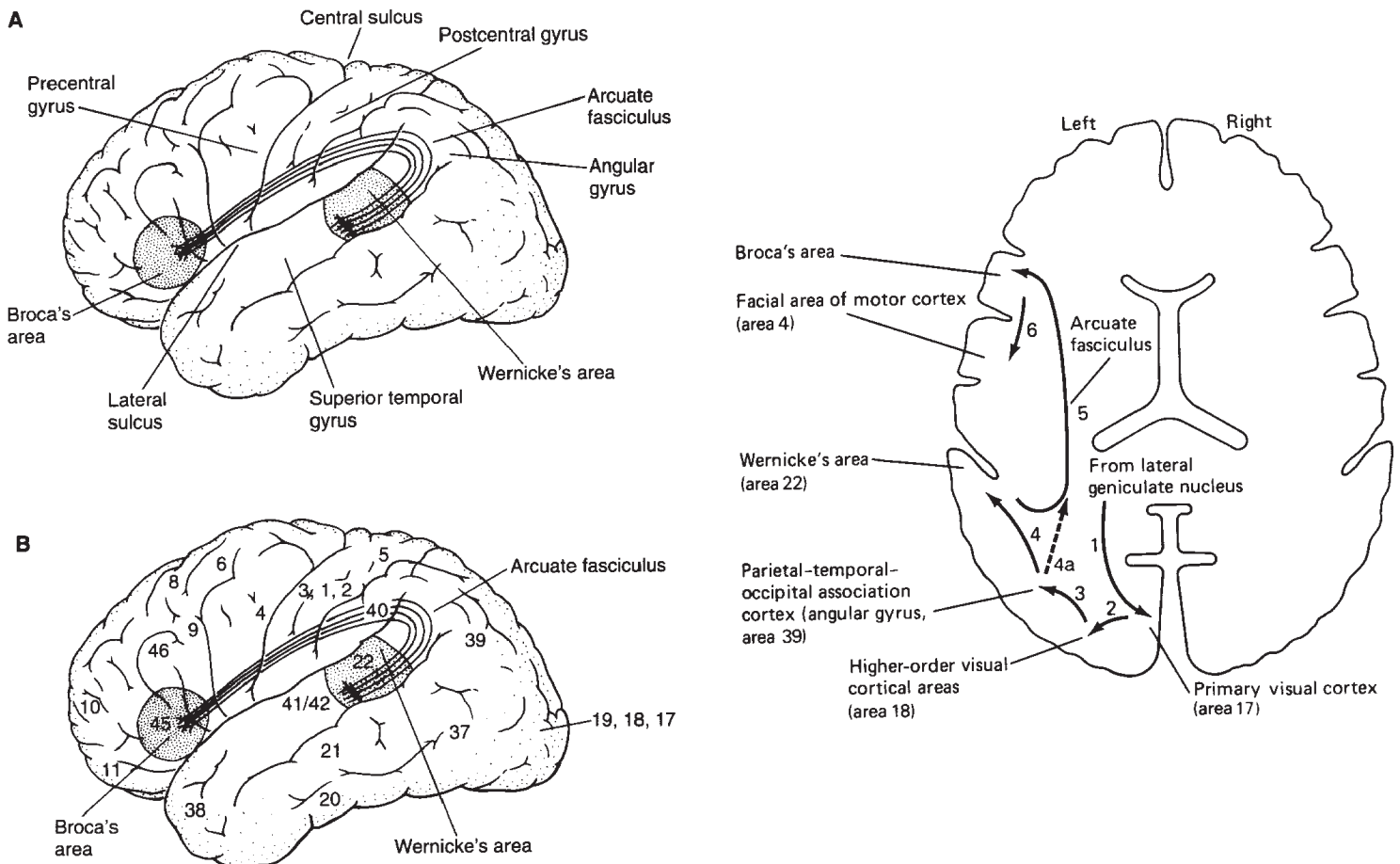
The narrative reveals the typical pattern of compound and complex sentence structures, which get nowhere because semantically meaningless sequences are juxtaposed. Paraphasia consists of totally irrelevant English words, neologisms, and repetitious overuse of phrases built around the words "time" and "work."



Answer to Case History XV

WERNICKE'S APHASIA - This condition is the most common form of fluent aphasia. The critical features of this syndrome include impaired auditory comprehension and fluent, easily articulated speech. The speech contains paraphasias. The impairment of auditory comprehension is evident even at the one word level. The individual may repeat the examiner's words uncomprehendingly, or with paraphasic distortions. Reading is poor as is writing, although a subgroup may write better than they speak. Paraphasia refers to the production of unintended syllables, words or phrases during efforts to speak. Literal or phonemic paraphasia arises when syllables are produced in the wrong order or when words are distorted by inclusion of unintended syllables. "Pipe" may become hike or pike. In the extreme the produced word may end up a non-word, or neologism (e.g. "Prike"). Verbal or semantic paraphasia arises when an unintended word is inadvertently used in place of another (e.g. my mother instead of my wife). The individual may have little awareness of the condition. Affected individuals usually appear content, happy, and sometimes even jocular. They typically appear to make light of their problem, and express little or no frustration.

Hemiparesis is unlikely, because the lesion is in the posterior portion of the superior temporal gyrus of the left hemisphere.



A.M. had a vascular accident diagnosed as a thrombosis in the distribution of the left middle cerebral artery, with effects extending posteriorly to the angular gyrus.

Clinical Characteristics of Cortical Aphasias

(these will be discussed by your instructor in more detail)

Type	Verbal Output	Comprehension	Repetition	Naming	Associated Signs	Lesion Site
Broca's	nonfluent	+	-	+/-	RHP	3rd frontal gyrus
Wernicke's	fluent	-	-	-	RHH	superior temporal gyrus
Global	nonfluent	-	-	-	RHH	
Anomic	fluent	+	+	-		
Conduction	fluent	+	-	-		arcuate fasciculus
Transcortical Motor	nonfluent	+	+	-		
Transcortical Sensory	fluent	-	+	-		
Mixed	nonfluent	-	+	-		

+ good/present

- poor/absent

RHP = right hemiparesis

RHH = right homonymous hemianopsia

Goodglass, H., & Kaplan, E. (1983). The assessment of aphasia and related disorders (2nd ed.). Philadelphia, PA: Lea & Febiger.

Answer to Case History XVI

A 34-year-old woman comes to see you because of problems with her balance and walking. She has noticed this for the past few days and her symptoms do not appear to be getting better. In addition, she feels numbness and tingling in her hands and feet. Sometimes when she bends her head forward she may feel an electrical shock going down the spine. When she is out in the summer heat, her symptoms seem to get worse and she may develop double vision. About 12 years ago she lost vision in her right eye. This recovered with treatment. She had some double vision in the past but not recently except when she was out in the heat. There is no family history of neurological problems.

On exam you find her mental status is normal. Cranial nerves exam was remarkable for a right afferent papillary defect (light in the left eye caused constriction but moving the light to the right eye resulted in both pupils to dilate) and visual acuity in the right eye was 20/30 even with correction. When looking to the left there was nystagmus in the left eye and the patient complained of double vision with looking to the left, but not to the right. A right eye adductor weakness was present on lateral gaze but not convergence. The rest of the cranial nerves were normal. Motor exam showed good strength in the upper extremities proximally but mild right grip weakness. Reflexes in the upper extremity were normal. In the lower extremities there was mild weakness bilaterally with some focal weakness in right foot dorsiflexion. Tone seemed increased in the legs, reflexes were brisk, and a definite right Babinski sign was present. Sensory exam was normal to pin prick but there was a decrease in vibration and proprioception in the toes and fingers. Vibration sensation changes on the cervical vertebral bodies around C4-5. Cerebellar function was okay on finger-nose-finger testing, although rapid alternating movements in her right hand were clumsy. Heel to shin testing was performed slowly but accurately. Her gait was unsteady and wide-based. She could stand with her feet together but tended to fall with her eyes closed.

1. Explain the deficits on neurological exam. Where and how many lesions can explain this woman's findings?

Answer:

Lesion 1: Cranial nerve II on the right, afferent papillary defect and change in vision in one eye points to a lesion anterior to the chiasm in the optic nerve. The afferent papillary defect points to a decrease in light recognition in the right eye. The efferent part of the papillary reflex mediated by the third nerve is intact as observed by the convergence. Visual acuity is also affected.

Lesion 2: Right medial longitudinal fasciculus lesions produce a right internuclear ophthalmoplegia. This resulted in lack of yoking of the left VI motor neurons and right III motor neurons to the innervate the right medial rectus muscle of the eye. Convergence is intact because this pathway is generated from midbrain neurons not from the left PPRF (para pontine reticular formation) that sends connections to the contralateral III n nucleus via the medial longitudinal fasciculus.

Lesion 3: There appears to be mild right-sided weakness that had characteristics of upper motor neuron lesions (increased reflex and Babinski sign). A lesion anywhere in the corticospinal tract could produce this. The increased reflexes in the legs point to the possibility there may also be a lesion in the corticospinal tract. Subcortical white matter lesions can cause hemiparesis.

Lesion 4: There is bilateral loss of vibration and position sense in the all four extremities with an apparent level at C4-5. Positive Lhermitte's sign is suggested by the history. Positive Romberg test (loses balance with eyes closed) points to a problem with either the vestibular system or proprioceptive system. These findings point to cervical dorsal column involvement.

What could cause this clinical picture and what tests would you perform?

This clinical picture is consistent with multiple lesions in the central nervous system that occurred at different times. This picture may be seen for a variety of reasons but most commonly occurs with multiple sclerosis when the optic nerve and medial longitudinal fasciculus are involved. Vasculitis may also produce multiple small lesions and would need to be considered.

We would image to define where the lesions are and in this case would consider a head and cervical spine MRI scan. In multiple sclerosis periventricular lesions of the white matter are observed. Some active change in the blood barrier may be observed if gadolinium contrast is used. Active involvement of the immune system may be demonstrated by abnormal CSF findings of increased protein level and the presence of a modest number of lymphocytes (10-100/cc). A relative increase in the IgG component of the protein content present and associated with oligoclonal bands of IgG.

2. Why does she have more problems in the heat?

Multiple sclerosis is characterized by a loss of central myelin with associated inflammatory changes. Demyelination can cause conduction block and loss of function, particularly at warmer temperatures when the sodium action potential shortens in duration and conduction failure of propagating action may be more likely to occur.

3. What treatment did she receive 12 years ago and what treatments should be considered?

The patient's original symptoms of loss of vision in the right eye probably relate an episode of optic neuritis. Optic neuritis may be treated acutely with steroid drugs that decrease inflammation. Flair-ups or exacerbation of multiple sclerosis symptoms may also be treated acutely with steroids. Some treatments are directed at preventing attacks by altering the immune system (beta-interferon or a injection with a polymer that has similarity to myelin basic protein). In some cases more robust immunosuppression is used including cancer chemotherapeutic agents.

Answer to Case History XVII

A 70-year-old man presents with diplopia and right eye and forehead pain. He first noted troubles about 6 months ago. At first his complaints were intermittent but now they are present nearly all the time. His past medical history is pertinent for hypertension and colon cancer, treated surgically 5 years ago. He was involved in a car accident about 2 years ago that was associated with loss of consciousness but not hospitalization. A CT scan in the ER was normal.

On exam: his mini-mental status was 27/30 (losing points for memory 1, county 1, figure 1). Cranial nerves: right pupil larger than the left by 2 mm and light reflex less brisk than the left pupil. Fundoscopic exam negative. Visual acuity and fields normal. Mild right ptosis was noted. Full extraocular movements were obtained but the patient complains of double vision when looking to the left either downward or upward with images vertically displaced, more horizontally displaced with direct left lateral gaze. Mild decrease sensation first division of V cranial nerve. Otherwise cranial nerves normal. Motor and sensory exam remarkable for a decrease in ankle jerks and vibration bilaterally. Gait normal for age.

1. Where is the lesion?

There is involvement of the right III n (ptosis and larger pupil with weakness of the medial rectus and inferior oblique). A right IV may also be present because the trouble of vision down and to the left. If the IV n is affected, there should also be loss of intorsion when the affected eye is tested to look downward. Also loss of sensation in the forehead suggests the first division of V is involved. These nerves all are close to the superior orbital fissure and travel in the lining of cavernous sinus.

The decrease in reflexes at the ankle and decreased vibration in the feet are common findings in the elderly and probably are unrelated to his current symptoms. They may represent a mild peripheral neuropathy and if macrocytosis was present on his blood smear, B12 and folate levels should be checked.

2. What could cause the lesion?

A small meningioma (tumor of the meninges) could cause these deficits at the superior orbital fissure or an aneurysm of the internal carotid artery. Invasive tumors from the sphenoid sinus or sella tursica can invade the walls of the cavernous sinus and affect II, III, IV, VI, and V 1 or 2 cranial nerves. In some cases trauma as occurred in this man could produce a fistula formation between the internal carotid artery and the cavernous sinus. In these cases proptosis of the eye may occur and a bruit may be present. The etiology can be defined by imaging studies (MRI, MRA, angiogram).

