The trochlear nucleus lies ventral to the cerebral aqueduct at levels #8 (rostral pons or isthmus) and #9 (caudal midbrain; inferior collicular level). The nucleus lies on top of our old friend the MLF. Axons from this nucleus pass **dorsally** around the aqueduct and **decussate** immediately caudal to the inferior colliculi. The trochlear nerve (which is quite thin) then winds around the cerebral peduncle and eventually innervates the **superior oblique** (SO4). This is the only cranial nerve to emerge from the **dorsal** aspect of the brain stem.
While at first glance it appears that contraction of the superior oblique turns the eye down and out, the rest of the story (Paul Harvey would love this) is slightly more complicated. If you are interested, read on! The vector diagram resolves the arrow R-B into effective components. Vector R-A depresses the eye around the lateral axis. Vector R-C abducts the eye around the vertical axis and intorts (medial rotation) the eye around the anteroposterior axis. Therefore, vector R-B acts to depress, abduct and intort the eye.

When the eye is in the primary position, the superior oblique lies medial to the A-P axis of the globe. However, when the eye is adducted, the line of pull of the tendon of the superior oblique is parallel to the A-P axis of the globe. In this position, none of the actions of the muscle are dissipated in the other actions (abduction and intorsion). Hence the clinical test for the strongest action of the superior oblique is to ask the patient to look in (medially) and then down.

The three rotational axes of the eye.
V = vertical. L = lateral.
A-P = anteroposterior.
A 4th nerve lesion causes atrophy of the superior oblique muscle. When looking down and in (medially) with the bad eye there will be DIPLOPIA. The false image will lie below the true image (vertical diplopia) and will be somewhat oblique (torsional diplopia). The weakness of downward movement of the affected eye, most markedly when the eye is turned inward, results in the patient complaining of difficulty in especially reading or going downstairs.

The weakness of the superior oblique in the primary position (looking straight ahead) results in the “bad” eye being slightly extorted and elevated due to the unopposed action of the inferior oblique. This will result in torsional and vertical diplopia. For instance, if the LEFT superior oblique is paralyzed, the LEFT eye is extorted and elevated. In order to get rid of the torsional part of the double vision, the patient will tilt their head to the side OPPOSITE the paralyzed muscle, that is to the RIGHT. This causes reflex (from the otoliths) intorsion of the normal RIGHT eye (on side of head tilt) so that the vertical axis of the two eyes become parallel (the eye associated with the paralyzed superior oblique is already extorted by the unopposed inferior oblique). To alleviate the vertical diplopia, the patient will also FLEX his/her chin when tilted to the RIGHT. In this position the patient will have to elevate the normal RIGHT eye in order to look straight ahead. The “bad” (LEFT) eye is already elevated and when the two eyes are located at the same vertical (up-down) position in the socket, the vertical diplopia is ameliorated.

REMEMBER, LESION OF TROCHLEAR NERVE (after it has crossed the midline) = HEAD TILTED AWAY FROM PARALYZED MUSCLE; HEAD ALSO FLEXED IN THIS POSITION. HOWEVER, IF LESION IS IN THE TROCHLEAR NUCLEUS, HEAD TILT = TOWARDS THE LESION
Brain stem
Trochlear nucleus
Shade in the location of a single, continuous, unilateral lesion in the drawing above that will account for the following neurological problems: ALL OF THE LESION IS IN THE BRAIN STEM—MAKE THE LESION AS SMALL AS POSSIBLE

subtle auditory deficits, head tilted to the left to ameliorate double vision
PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column.

1. left corticobulbar tract
2. left motor nucleus V
3. left superior olive
4. left superior cerebellar peduncle
5. left trochlear nerve

A. cells of origin lie in the deep cerebellar nuclei on the left
B. lesion results in incoordination of the right side of the body
C. lesion results in deviation of the tongue to the right upon protrusion
D. lesion results in jaw deviation to the left upon opening
E. lesion results in the head tilted and flexed to the left to ameliorate diplopia
F. lesion results in atrophy of the left inferior oblique muscle
G. lesion results in subtle auditory deficits
H. lesion results in head tilted and flexed to the right to ameliorate diplopia
I. lesion results in weakness in the muscles of the lower face on the left
J. axons terminate in the right VPM
The substantia nigra lies in the midbrain immediately dorsal to the cerebral peduncles. This nucleus is an important motor center that will be discussed at greater length later in the course. Right now you need to know that some of the cells project to the caudate and putamen, two nuclei of the basal ganglia that together comprise what is called the STRIATUM. These NIGROSTRIATAL cells utilize the neurotransmitter DOPAMINE.

The substantia nigra is thought to be the lesion site in PARKINSON’S disease or paralysis agitans. In this disease there is muscular rigidity, a fine tremor at rest (resting tremor), akinesia or bradykinesia and a slow and shuffling gait and postural instability. You do not have to worry about the laterality (right or left) of these deficits at this time. The most consistent pathological finding in Parkinson’s disease is degeneration of the melanin-containing cells in the pars compacta (another part is called the pars reticulata) of the substantia nigra (melanin is an inert by-product of the synthesis of dopamine). As mentioned above, cells within the nigra produce dopamine normally. This substance passes—via axoplasmic flow—to the nerve terminals in the striatum (caudate nucleus and putamen), where it is released as a transmitter. It is the absence of this transmitter that produces the crippling disorder called Parkinson’s disease.
Brain stem
Substantia nigra

LEVEL 9. MESENCEPHALON AT LEVEL OF INFERIOR COLLICULUS

LEVEL 16

LEVEL 19

LEVEL 10

NIGROSTRIATAL PROJECTION
PROBLEM SOLVING

Shade in the location of a single, continuous lesion in the drawing above that will account for the following neurological problems:

inability to turn the left eye to the right upon attempted horizontal gaze to the right, inability to turn the right eye to the left upon attempted horizontal gaze to the left.
PROBLEM SOLVING ANSWER

RIGHT       LEFT
PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column

___1. right motor nucleus VII
   A. lesion results in loss of salivation from the left parotid gland

___2. left nucleus ambiguus
   B. lesion results in tremor, bradykinesia and rigidity

___3. left anterolateral system (ALS)
   C. cells of origin lie in the left dorsal horn

___4. right trochlear nucleus
   D. receives input from the right trigeminothalamic tract (TTT)

___5. substantia nigra
   E. lesion results in hyperacusis in the right ear

F. lesion results in an increased dopamine production in the right nigrostriatal pathway

G. lesion results in atrophy of the left superior oblique muscle

H. lesion results in deafness in the right ear

I. axons terminate in the left VPL

J. lesion results in a loss of vibrational sense from the right side of the body
ANSWERS TO PROBLEM SOLVING QUESTIONS RELATED TO POINTS 16-20.

NOTE: The answers to ALL shade-in questions are illustrated on the back side of the question.

Point #16 Pontine Nuclei - Middle Cerebellar Peduncle
Matching H,J,F,B,G

Point #17 Motor V, Chief Sensory V, Mesen. Tract and Nuc. V
Matching D,C,E,I,H

Point #18 Superior Cerebellar Peduncle
Matching J,F,G,C,E

Point #19 Trochlear
Matching C,D,G,A,H

Point #20 Substantia Nigra
Matching E,D,I,G,B
ACROSS
1. results from a lesion of dorsal motor X
3. ganglia of IX and X associated with pain from the "EAR"
5. arises from cells in rostral nucleus solitarius
9. disease associated with lesions of substantia nigra
10. movement of head following lesion of the trochlear nucleus
11. type of fiber associated with pontocerebellar projection
13. target of medial lemniscus and ALS
15. follows a lesion of the corticospinal tract
16. conveyed via ALS and TTT
18. thalamic target of STT and TTT
19. trochlea (L________)  
20. ______ cuneatus  
22. a little eminence - think!!
25. sensory supply involves three cranial nerves  
27. projection is bilateral to motor V and nucleus ambiguous
29. medial and lateral ________
31. "Much ______ About Nothing"  
Shakespeare
32. results from a lesion of nucleus ambiguous
34. cells of origin lie in contra abducens nucleus
35. is defined as "right" following a lesion of the left vestibular nerve
36. ________ pons or artery
38. ganglion associated with taste from ant. two-thirds of tongue
40. associated with chief sensory V
41. head movement following lesion of abducens nerve
42. largest deep cerebellar nucleus
43. supplied by 4 cranial nerves

DOWN
1. dorsal part of pons
2. corrected by specific head positions
4. results from lesions of ALS and associated descending tract
6. lesion results in contra loss of pain and temp. from the face
7. deviation (direction) of left eye following lesion of left abducens nerve
8. result of stimulation of ALS (and associated descending pathways) on pupil
12. cerebellar peduncle that "joins together"  
13. thalamic target of the superior cerebellar peduncle
14. inferior, middle and superior
17. peduncle that contains cuneocerebellar fibers
20. associated with lower motor neuron disease
21. associated with lesions of the corticospinal tract
23. associated with a decrease in dopamine production by the substantia nigra
24. arm (L_______)
26. wasting
28. job of motor V neurons
30. associated with lesions of the vestibular apparatus, nerve or nuclei
32. one of the two cochlear nuclei
33. deviates to the strong side following lesions of nucleus ambiguous
37. direction of eyes following lesion of left frontal eye field
39. primary input to the pontine grey
The oculomotor nucleus proper is comprised of cells that innervate all extraocular eye muscles except the lateral rectus (LR6) and superior oblique (SO4). Remember that it also innervates the levator palpebrae. The EDINGER-WESTPHAL nucleus, which lies dorsal to the oculomotor nucleus proper, contains preganglionic parasympathetic (visceromotor) neurons whose axons end in the ciliary ganglion. Short postganglionic parasympathetic axons then pass from the ciliary ganglion to the sphincter pupillae of the iris and the ciliary muscles of the eye (for changing shape of lens in accommodation). Input to the Edinger-Westphal nucleus arises from a cell group called the pretectum, a cell complex that receives retinal input and is part of the pathway involved in reducing the size of the pupil upon light stimulation of the retina. Don’t worry about the pretectum now!

A lesion involving C.N. III involves the axons headed for the eye muscles and the levator, as well as the visceromotor preganglionic parasympathetics destined for the ciliary ganglion. Following a unilateral lesion of C.N. III there will be outward and slightly downward deviation of the ipsilateral eye (due to unopposed action of the lateral rectus and superior oblique) and the inability to rotate the eye upward, downward or inward. You have to raise the eyelid to see the position of the eyeball, because the levator is not working! Because the eyelid is closed, the DIPOPLIA that would result from the lack of alignment of the visual axes of the two eyes is masked. There also is drooping of the eyelid or PTOSIS (levator palpebrae is not working), and DILATION of the pupil (unopposed action of sympathetics due to loss of parasympathetics). Other deficits that you DO NOT have to deal with are loss of the pupillary light reflex and convergence, and loss of accommodation of the lens. I want you to remember the PTOSIS and DILATED PUPIL in the eye ipsilateral to the lesion of C.N. III. DON’T WORRY ABOUT CONVERGENCE AND ACCCOMMODATION AT THIS TIME!
Both a third nerve palsy and Horner’s syndrome can result in ptosis and pupillary asymmetry. But with a third nerve palsy the ptosis is on the side of the large pupil while with a Horner’s the ptosis is on the side of the small pupil.
PROBLEM SOLVING

Shade in the location of a single, continuous lesion in the drawing above that will account for the following neurological problems:

ptosis of the left eyelid, dilated pupil in the left eye, fine tremor at rest, slow and shuffling gait, muscle rigidity
PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column.

1. right motor nucleus VII
   - A. lesion results in atrophy of the left masseter muscle

2. right spinal nucleus V
   - B. lesion results in a dilated pupil in the right eye
   - C. contains cells that project to the right oculomotor nucleus via the right MLF

3. right trochlear nucleus
   - D. pathway terminates in the left VPL

4. left abducens nerve
   - E. lesion results in atrophy of the medial pterygoid muscles on the right

5. right oculomotor nerve
   - F. lesion results in the inability to turn the left eye past the midline to the right
   - G. axons terminate in the left lateral rectus muscle
   - H. lesion results in the left eyeball being extorted and elevated
   - I. receives input from cells in the right trigeminal ganglion
   - J. cells project to the muscles of facial expression on the right
The red nucleus is a prominent structure within the rostral midbrain and lies just dorsal to the substantia nigra. It appears to have a high iron content and is more vascular than the surrounding tissue, and in some brains is pinkish. Very little is known about the function(s) of the red nucleus in humans. Inputs to the ruber arise from motor areas of the brain and in particular the deep cerebellar nuclei (via superior cerebellar peduncle; crossed projection) and the motor cortex (corticorubral; ipsilateral projection).
The most important efferent projection of the red nucleus is to the contralateral spinal cord i.e., the **RUBROSPINAL** projection. Thus red nucleus neurons possess axons that cross just ventral to the nucleus and descend in the midbrain, pons and medulla (we cannot identify this pathway in our brain stem series of cross sections) to reach the spinal cord. In the spinal cord the rubrospinal tract courses within the LATERAL FUNICULUS JUST VENTRAL TO THE LATERAL CORTICOSPINAL TRACT.

The rubrospinal tract is thought to be involved in the control of both the flexor and extensor muscles, but even this is debated. This tract courses adjacent to the **lateral corticospinal tract** and terminates in roughly the same region (laminae) of the spinal cord gray. These two pathways are therefore thought to act somewhat in concert. This close association (rubrospinal/corticospinal) is further exemplified by the fact that the motor cortex also projects to the red nucleus. This means that the corticospinal tract is paralleled by an “indirect corticospinal tract” with a relay in the red nucleus, i.e., the corticorubrospinal tract. The rubrospinal projection is also, of course, influenced by the motor information coming out of the cerebellum, as well as from the motor cortex.
As far as I know there are no clinical case studies involving a lesion limited to the red nucleus. You already know that many of the fibers of the **brachium conjunctivum** run through and around the ruber on their way to VA and VL of the thalamus (have they crossed yet?). Therefore a lesion in the ruber will not only destroy rubrospinal neurons but also cerebellothalamic axons destined for VA and VL. Since the fibers in the brachium conjunctivum have already crossed, their interruption will result in a contralateral motor deficit (remember VA, VL to ipsi motor cortex and then the crossed corticospinal system). A contralateral motor deficit would also result from a lesion of the rubrospinal neurons, whose axons innervate the contralateral spinal cord gray. Thus it is difficult to know just what particular motor deficit(s) are associated with damage to the ruber versus damage to the cerebellothalamic fibers. It has been reported that large lesions of the midbrain tegmentum involving the red nucleus (called the tegmental syndrome) result in **hemichorea**, which is a tremor or involuntary movement of the **contralateral limbs**. Since we don’t know that this problem is due to involvement of only the rubrospinal system, I feel it is best to consider a lesion of the ruber to result in **MOTOR PROBLEMS OF THE CONTRALATERAL LIMBS** for our problem solving exercises.
PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column:

___1. left chief sensory V  A. cells project directly to the secretory cells in the right lacrimal gland
___2. right lacrimal nucleus  B. receives input from axons in the right superior cerebellar peduncle
___3. right pontine grey nuclei  C. contains preganglionic parasympathetic neurons that project to the left ciliary ganglion
___4. left Edinger-Westphal complex  D. lesion results in a loss of vibratory sense from the left side of the face
___5. left red nucleus  E. cells project to motor nucleus V
                   F. cells project to the right side of the cerebellum
                   G. receives input from cells in the right trigeminal ganglion
                   H. preganglionic parasympathetic neurons that project to the right pterygopalatine ganglion
                   I. cells project to the left side of the cerebellum via the left middle cerebellar peduncle
                   J. cells project to the left medial rectus muscle
PROBLEM SOLVING

RIGHT       LEFT

Shade in the location of a single, continuous lesion in the drawing above that will account for the following neurological problems:

subtle auditory deficits, constricted pupil in left eye, loss of pain and temperature from the right side of the tongue, loss of taste from the left side of the tongue
PROBLEM SOLVING ANSWER

RIGHT       LEFT
The superior colliculi form the rostral two bumps (one on each side) on the dorsal aspect of the midbrain. The caudal two bumps are the inferior colliculi and together they (inferior and superior colliculi) comprise the **TECTUM** or roof of the midbrain. In contrast to the inferior colliculus, which is an **AUDITORY** structure, the superior colliculus is usually described as a **VISUAL** reflex center. It is a highly laminated (layered) structure. The top or dorsal-most three layers receive visual information primarily from two sources, i.e., the retina (retinocollicular) and the visual cortex (area 17; corticotectal). In contrast to the exclusively visual nature of the superficial layers, the intermediate and deep layers receive projections from many functionally different areas of the brain. These inputs are both “motor” and “sensory”. Since the latter category includes visual, auditory and somatosensory inputs, you can see that the superior colliculus is not exclusively related to visual function. Instead, it plays a role in helping orient the head and eyes to all types of sensory stimuli.
Brain stem
Superior colliculus

TECOTOSPINAL TRACT

Neck muscles that turn the head to the LEFT
One of the major efferent projections of the superior colliculus is to the **CERVICAL SPINAL CORD**. This **TECTOSPINAL TRACT** arises from cells within the intermediate and deep layers, crosses at midbrain levels and courses caudally through the midbrain, pons and medulla close to the MLF (we do not identify it in our sections). Upon reaching the spinal cord tectospinal axons course within the **VENTRAL funiculus** and terminate upon medially placed neurons within the cervical cord. This tract is important in reflex turning of the head in response to visual, auditory and somatosensory stimuli. For instance, a flash of light to your **LEFT** causes you to turn your head to the **LEFT**. This reflex would involve a projection from the retinæ to the superficial layers of the **RIGHT** superior colliculus (retinocollicular), a short pathway from cells in the superficial layers to cells in the intermediate and deep layers and then the long **CROSSED** tectospinal axons to the **LEFT** side of the cervical spinal cord. Spinal cord neurons on the **LEFT** side then innervate muscles such as the splenius capitus and semispinalis capitus, which rotate your head to the **LEFT**.

Cells within the intermediate and deep layers also are involved in the control of eye movements. We will not go into how the collicular neurons participate in such control, but you are already familiar with the **PPRF**, which is an area of the pons involved in the control of horizontal eye movements. For example, cells in the intermediate and deep layers of the **LEFT** superior colliculus project to the **RIGHT** PPRF (this pathway is not illustrated in the coursebook). You know the circuitry from here that moves both eyes to the **RIGHT**. If not, see that **INFAMOUS POINT 13** for a review!

A syndrome that you might see in the clinical literature involving the superior colliculus is called the dorsal midbrain or **PARINAUD** syndrome. This is usually caused by a tumor of the pineal gland that compresses the superior colliculi and results in a **paralysis of UPWARD gaze**. It is not clear if this deficit is due to involvement of **ONLY** the superior colliculi. A center for vertical eye movements lies just rostral to the superior colliculi and could be involved. For now, **REMEMBER-PARINAUD SYNDROME==PARALYSIS OF UPWARD GAZE**.
PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column

____1. right oculomotor nucleus
A. axons originate in the right motor cortex

____2. right nucleus ambiguus
B. receives input from cells in the left abducens nucleus

____3. right lateral corticospinal tract at C1
C. axons terminate in the left red nucleus

____4. right rubrospinal tract
D. axons terminate in the right side of the cerebellum

____5. superior colliculus
E. lesion results in a shuffling gait and resting tremor

F. cells receive direct input from the retina

G. lesion results in right hemiplegia

H. lesion results in motor incoordination (not paresis or plegia) of the right arm and leg

I. lesion results in deviation of the uvula to the left when saying “ahhh”

J. lesion results in atrophy of the right lateral rectus muscle
Shade in the location of a single, continuous lesion in the drawing above that will account for the following neurological problems:

- dilated pupil in the left eye, right Babinski, deviation of the tongue to the right upon protrusion, drooling of food from the right side of the mouth, when left eyelid is elevated manually, left eyeball can be seen to be rotated laterally and ventrally
Brain stem
Superior colliculus
Problem solving

PROBLEM SOLVING ANSWER

RIGHT       LEFT
As you have been studying brain stem levels #9 and #10, I am certain that you have been wondering just what that area surrounding the cerebral aqueduct is. Well, surprisingly it is called the periaqueductal grey (or gray)! This is an interesting area that is involved the regulation of pain. It has been demonstrated that stimulation of this area in rats eliminates their perception of pain. This is called stimulation produced analgesia or SPA (or stimulation induced analgesia; SIA).
The pathway over which this pain reduction takes place is a projection from cells in the PAG to a serotonergic nucleus in the medulla called the nucleus raphe magnus (NRM). The NRM, which is not seen on your fiber-stained sections, lies right down the middle of the medulla and is thus called a raphe (Gr., zipper) nucleus. Cells in the NRM project to the dorsal horn along the entire length of the spinal cord and these axons inhibit the cell of origin of the anterolateral system (spinothalamic tract). Thus, when the PAG fires the NRM fires and the end result is a decrease in pain impulses traveling up the ALS to reach the VPL and somatosensory cortex, i.e., consciousness.

I know that you are wondering “what turns on this pain-reducing system.” Think back about the last time you were really mad at me for lecturing too long or calling on you (or waking you up!!) in front of your classmates. You were so upset that your “emotional brain” took over and caused you ignore your recently sprained ankle or newly acquired blister on your big toe (what dermatome is that??!). What was happening is that pathways from your emotional brain (to be defined later) were exciting some cells in the PAG. The cells in the PAG that are turned on have receptors for opiate peptides called endorphins (enkephalins are one of the three groups of endorphins).

Since the cells in the PAG contain receptors for opiate peptides, systemic injections of morphine-like substances can reduce pain by turning on the smaller inhibitory PAG neurons in the midbrain.

Some of the pathways just discussed may underlie the results of classical acupuncture. Rotation of thin needles could stimulate the C and D fibers in body tissue and in turn the ALS. While you are thinking this should hurt, branches of ALS fibers (collaterals of those heading to the VPL) could end in the PAG (or NRM) and somehow turn on the descending pain modulation circuit. We do not understand the details of this circuit, but it is known that acupuncture is “opiate dependent.” That is, intravenous injection of naloxone (an opiate inhibitor) is thought to block the effects of acupuncture (and SPA and SIA).
PROBLEM SOLVING #1

Which of the following statements is/are **TRUE**?

A. there are endogenous opiate receptors in the brain
B. the PAG contains cells with opiate receptors
C. your emotional brain turns on the PAG
D. cells in the nucleus raphe magnus are serotonergic, lie on the midline(zipper) and are excited by PAG cells
E. all of the above responses are true

PROBLEM SOLVING #2

Which of the following statements is/are **TRUE**?

A. stimulation of the PAG excites the NRM
B. morphine injected systemically can inhibit pain by turning on the PAG
C. acupuncture is opiate dependent
D. dorsal horn cells that project into the ALS are excited by c and d fibers
E. all of the above responses are true
Brain stem
ANSWERS TO PROBLEM SOLVING QUESTIONS-POINTS 21-24.

Point #21 Oculomotor Nucleus
Matching  J, I, H, G, B

Point #22 Red Nucleus
Matching  D, H, I, C, B

Point #23 Superior Colliculus
Matching  B, I, G, H, F

Point #24 Periaqueductal Grey
1. E
2. E
Brain stem regions
Problem solving

RIGHT        LEFT

RIGHT        LEFT

RIGHT        LEFT

RIGHT        LEFT

Medulla Levels 1-4
PROBLEM SOLVING

Which of the following neurological deficits could result from a lesion of **ANY FIBER TRACT OR NUCLEUS ON THE LEFT SIDE OF THE MEDULLA (LEVELS #1-#4)**. Do not worry about the little bit of the pons on level #4.

1. nausea, vomiting, stumbling
2. loss of vibratory sense from the left side of the face
3. atrophy of the inferior rectus muscle
4. atrophy of the superior oblique muscle
5. inability to elevate both eyes (Parinaud’s syndrome)
6. deficit associated with lesion of C.N.VII
7. pronounced atrophy of the left temporalis muscle
8. subtle auditory deficits
9. loss of pain and temperature from the arms and legs on the right
10. loss of the blink reflex in at least one eye
11. internuclear ophthalmoplegia
12. atrophy of the orbicularis oculi muscle
13. head positioning to ameliorate double vision
14. nystagmus
15. deficits associated with Parkinson’s disease (rigidity, resting tremor, akinesia)
16. inability to move both eyes past the midline of the orbits to the left
17. hyperacusis
18. deviation of the jaw to the left upon opening
19. constricted pupil in the left eye
20. incoordination of the left arm
21. dysphagia
22. atrophy of the left lateral rectus muscle
23. atrophy of the left medial rectus muscle
24. loss of pain and temperature from the left side of the face
25. diplopia (double vision)
26. loss of taste from the left side of the tongue
27. drooling from the left side of the mouth
28. right Babinski sign
29. deafness in the left ear
30. hiccups
31. atrophy of the left side of the tongue
32. ptosis of the left eyelid
33. loss of vibratory sense from the left arm and leg
34. right hemiplegia
35. dilated pupil in the left eye
36. increase in heart rate
37. dysphonia
38. loss of gag reflex (on either side)
Brain stem
Brain stem regions
Problem solving

RIGHT LEFT
RIGHT LEFT
RIGHT LEFT
RIGHT LEFT

Pons Levels 5-8
PROBLEM SOLVING

Which of the following neurological deficits could result from a lesion of **ANY FIBER TRACT OR NUCLEUS ON THE LEFT SIDE OF THE PONS (LEVELS #5-#8)**.

1. nausea, vomiting, stumbling
2. loss of vibratory sense from the *left* side of the face
3. atrophy of the inferior rectus muscle
4. atrophy of the superior oblique muscle
5. inability to elevate both eyes (Parinaud’s syndrome)
6. deficit associated with lesion of C.N.VII
7. pronounced atrophy of the *left* temporalis muscle
8. subtle auditory deficits
9. loss of pain and temperature from the arms and legs on the *right*
10. loss of the blink reflex in at least one eye
11. internuclear ophthalmoplegia
12. atrophy of the orbicularis oculi muscle
13. head positioning to ameliorate double vision
14. nystagmus
15. deficits associated with Parkinson’s disease
   (rigidity, resting tremor, akinesia)
16. inability to move both eyes past the midline of the orbits to the *left*
17. hyperacusis
18. deviation of the jaw to the *left* upon opening
19. constricted pupil in the *left* eye
20. incoordination of the *left* arm
21. dysphagia
22. atrophy of the *left* lateral rectus muscle
23. atrophy of the *left* medial rectus muscle
24. loss of pain and temperature from the *left* side of the face
25. diplopia (double vision)
26. loss of taste from the *left* side of the tongue
27. drooling from the *left* side of the mouth
28. *right* Babinski sign
29. deafness in the *left* ear
30. hiccup
31. atrophy of the *left* side of the tongue
32. ptosis of the *left* eyelid
33. loss of vibratory sense from the *left* arm and leg
34. *right* hemiplegia
35. dilated pupil in the *left* eye
36. increase in heart rate
37. dysphonia
38. loss of gag reflex (on either side)
Midbrain Levels 9, 10
PROBLEM SOLVING

Which of the following neurological deficits could result from a lesion of ANY FIBER TRACT OR NUCLEUS ON THE LEFT SIDE OF THE MIDBRAIN (LEVELS #9 and #10).

1. nausea, vomiting, stumbling
2. loss of vibratory sense from the left side of the face
3. atrophy of the inferior rectus muscle
4. atrophy of the superior oblique muscle
5. inability to elevate both eyes (Parinaud’s syndrome)
6. deficit associated with lesion of C.N.VII
7. pronounced atrophy of the left temporalis muscle
8. subtle auditory deficits
9. loss of pain and temperature from the arms and legs on the right
10. loss of the blink reflex in at least one eye
11. internuclear ophthalmoplegia
12. atrophy of the orbicularis oculi muscle
13. head positioning to ameliorate double vision
14. nystagmus
15. deficits associated with Parkinson’s disease (rigidity, resting tremor, akinesia)
16. inability to move both eyes past the midline of the orbits to the left
17. hyperacusis
18. deviation of the jaw to the left upon opening
19. constricted pupil in the left eye
20. incoordination of the left arm
21. dysphagia
22. atrophy of the left lateral rectus muscle
23. atrophy of the left medial rectus muscle
24. loss of pain and temperature from the left side of the face
25. diplopia (double vision)
26. loss of taste from the left side of the tongue
27. drooling from the left side of the mouth
28. right Babinski sign
29. deafness in the left ear
30. hiccups
31. atrophy of the left side of the tongue
32. ptosis of the left eyelid
33. loss of vibratory sense from the left arm and leg
34. right hemiplegia
35. dilated pupil in the left eye
36. increase in heart rate
37. dysphonia
38. loss of gag reflex (on either side)
PROBLEM SOLVING ANSWER

The following neurological deficits could result from a lesion of ANY FIBER TRACT OR NUCLEUS ON THE LEFT SIDE OF THE MEDULLA (LEVELS #1-#4).

1. nausea, vomiting, stumbling
2. loss of vibratory sense from the left side of the face
3. atrophy of the inferior rectus muscle
4. atrophy of the superior oblique muscle
5. inability to elevate both eyes (Parinaud’s syndrome)
6. deficit associated with lesion of C.N.VII
7. pronounced atrophy of the left temporalis muscle
8. subtle auditory deficits
9. loss of pain and temperature from the arms and legs on the right
10. loss of the blink reflex in at least one eye
11. internuclear ophthalmoplegia
12. atrophy of the orbicularis oculi muscle
13. head positioning to ameliorate double vision
14. nystagmus
15. deficits associated with Parkinson’s disease (rigidity, resting tremor, akinesia)
16. inability to move both eyes past the midline of the orbits to the left
17. hyperacusis
18. deviation of the jaw to the left upon opening
19. constricted pupil in the left eye
20. incoordination of the left arm
21. dysphagia
22. atrophy of the left lateral rectus muscle
23. atrophy of the left medial rectus muscle
24. loss of pain and temperature from the left side of the face
25. diplopia (double vision)
26. loss of taste from the left side of the tongue
27. drooling from the left side of the mouth
28. right Babinski sign
29. deafness in the left ear
30. hiccup
31. atrophy of the left side of the tongue
32. ptosis of the left eyelid
33. loss of vibratory sense from the left arm and leg (lesion left dorsal column nuclei)
34. right hemiplegia
35. dilated pupil in the left eye
36. increase in heart rate
37. dysphonia
38. loss of gag reflex (on either side)
PROBLEM SOLVING ANSWER

The following neurological deficits could result from a lesion of ANY FIBER TRACT OR NUCLEUS ON THE LEFT SIDE OF THE PONS (LEVELS #5-#8).

*1. nausea, vomiting, stumbling

*2. loss of vibratory sense from the left side of the face

3. atrophy of the inferior rectus muscle

*4. atrophy of the superior oblique muscle

5. inability to elevate both eyes (Parinaud’s syndrome)

*6. deficit associated with lesion of C.N.VII

*7. pronounced atrophy of the left temporalis muscle

*8. subtle auditory deficits

*9. loss of pain and temperature from the arms and legs on the right

*10. loss of the blink reflex in at least one eye

NOTE: touch receptor

*11. internuclear ophthalmoplegia NOTE: INO

*12. atrophy of the orbicularis oculi muscle

*13. head positioning to ameliorate double vision

*14. nystagmus NOTE: INO

15. deficits associated with Parkinson’s disease (rigidity, resting tremor, akinesia)

*16. inability to move both eyes past the midline of the orbits to the left

*17. hyperacusis

*18. deviation of the jaw to the left upon opening

*19. constricted pupil in the left eye

*20. incoordination of the left arm

21. dysphagia

*22. atrophy of the left lateral rectus muscle

23. atrophy of the left medial rectus muscle

*24. loss of pain and temperature from the left side of the face

*25. diplopia (double vision)

*26. loss of taste from the left side of the tongue

*27. drooling from the left side of the mouth

*28. right Babinski sign

29. deafness in the left ear

30. hiccup

31. atrophy of the left side of the tongue

*32. ptosis of the left eyelid

33. loss of vibratory sense from the left arm and leg

*34. right hemiplegia

35. dilated pupil in the left eye

36. increase in heart rate

*37. dysphonia

NOTE: possibly from lesion of motor VII

38. loss of gag reflex (on either side)
The following neurological deficits could result from a lesion of any fiber tract or nucleus on the left side of the midbrain (levels #9 and #10).

1. Nausea, vomiting, stumbling
2. Loss of vibratory sense from the left side of the face
3. Atrophy of the inferior rectus muscle
4. Atrophy of the superior oblique muscle
5. Parinaud's syndrome NOTE: lesion is not bilateral
6. Deficit associated with lesion of C.N.VII (p&t from the ear + solitary + drooling corticobulb. to CN VII)
7. Pronounced atrophy of the left temporalis muscle
8. Subtle auditory deficits
9. Loss of pain and temperature from the arms and legs on the right
10. Loss of the blink reflex in at least one eye
11. Internuclear ophthalmoplegia NOTE: INO
12. Atrophy of the orbicularis oculi muscle
13. Head positioning to ameliorate double vision
14. Nystagmus NOTE: INO
15. Deficits associated with Parkinson's disease (rigidity, resting tremor, akinesia)
16. Inability to move both eyes past the midline of the orbits to the left
17. Hyperacusis
18. Deviation of the jaw to the left upon opening
19. Constricted pupil in the left eye
20. Incoordination of the left arm
   Note: (interruption of the decussating fibers of SCP)
21. Dysphagia
22. Atrophy of the left lateral rectus muscle
23. Atrophy of the left medial rectus muscle
24. Loss of pain and temperature from the left side of the face
25. Diplopia (double vision)
26. Loss of taste from the left side of the tongue
27. Drooling from the left side of the mouth
28. Right Babinski sign
29. Deafness in the left ear
30. Hiccup
31. Atrophy of the left side of the tongue
32. Ptosis of the left eyelid
33. Loss of vibratory sense from the left arm and leg
34. Right hemiplegia
35. Dilated pupil in the left eye
   NOTE: (the descending fibers to the lateral cell column and CN III are damaged; your guess is as good as mine!!)
36. Increase in heart rate
37. Dysphonia
38. Loss of gag reflex (on either side)