Neurology Clerkship Learning Objectives

Clinical skills

Perform a neurological screening examination of the cranial nerves, motor system, reflexes, and sensory system under the observation and guidance of an attending neurologist.

Localization skills: focal weakness or numbness

1. For a patient with limb weakness, recognize any signs and symptoms of lower motor neuron (LMN) versus upper motor neuron (UMN) lesions in order to localize the problem to the brain, brainstem, spinal cord, or nerves.

2. For a patient with facial weakness, recognize the lower motor neuron (LMN) versus upper motor neuron (UMN) signs and symptoms in order to localize the problem to the facial nerve/nucleus or a more rostral level, respectively.

3. For a patient with weakness of speech or swallowing, recognize the lower motor neuron (LMN) versus upper motor neuron (UMN) signs and symptoms in order to localize the problem to lower brainstem motor nuclei (CNs 7, 9, 10, or 12: bulbar palsy) or a more rostral level (pseudobulbar palsy), respectively.

4. If there are no LMN or UMN signs in a patient with weakness, recognize any signs and symptoms suggesting that myopathy or a neuromuscular junction disorder is the cause.

5. Recognize the typical patterns of sensory deficit which localize, in conjunction with other signs and symptoms, the cause of numbness in a patient to the nerves, roots, spinal cord, brainstem or brain.

Localization skills: visual changes

6. Recognize that visual impairment from binocular diplopia (where covering either eye normalizes vision) is caused by dysfunction of the extraocular muscles (lesions of the muscles, their neuromuscular junctions, their cranial nerves or connecting pathways).

7. Recognize how certain patterns of visual loss in a patient localize the problem to the optic nerve, optic chiasm or visual pathways.

8. Recognize which abnormalities in the pupillary light reflex and appearance of the optic disc are caused by a problem with the optic nerve, retina or optic chiasm, in a patient with visual loss.

Localization skills: delirium/acute mental status change

9. Recognize the typical clinical features of a patient with the acute confusional state (delirium), and list common disorders (primarily or secondarily affecting the brain) which cause it.

Localization skills: dementia/memory/cognitive loss

10. Recognize which clinical features in a patient with memory and cognitive loss are typical of dementia, and list common disorders which cause it, emphasizing treatable or reversible causes.

11. Recognize and contrast the signs and symptoms of Broca's versus Wernicke's aphasia.

Localization skills: dizziness/ abnormal gait or balance

12. Recognize the clinical features that distinguish near-syncope or syncope from vertigo as the cause of dizziness in a patient, and list common causes of each.

13. Recognize which signs and symptoms in a patient with abnormal balance or gait relate the problem to the sensory, cerebellar, motor or extrapyramidal parkinsonism) systems, and list common causes of each.

Localization skills: headache or regional pain

14. Recognize and contrast the usual clinical features of migraine versus cluster versus tension headache.

15. Recognize which signs and symptoms are suggestive of headache from increased intracranial pressure and list common causes of it.

16. Recognize the typical signs and symptoms in patients with common regional pain syndromes (trigeminal neuralgia, zoster, painful neuropathy), and the usual causes of each.

Localization skills: impaired consciousness or sleep disorder

17. Recognize which signs and symptoms in a patient with impaired consciousness or coma localize the problem to the brain stem versus brain, and that asymmetrical findings suggest a structural lesion requiring emergent evaluation (especially a dilated, fixed pupil from uncal herniation).

18. Recognize and contrast the symptoms suggestive of sleep apnea versus narcolepsy.

Localization skills: seizure or abnormal movements

19. Recognize which signs and symptoms distinguish a seizure from syncope, and list the common causes of a seizure.

20. Recognize the clinical features of different types of seizures.

21. Recognize the clinical features of certain involuntary movements (tremor, dystonia, choreoathetosis, myoclonus, tic), and the disorders commonly associated with them, such as parkinsonism, Huntington's disease, and toximetabolic encephalopathy.

Knowledge and management of specific disorders or diseases

22. Describe the basic pathophysiology, common clinical manifestations, and appropriate work-up and treatment of transient ischemic attacks (TIAs) and ischemic infarction of brain or brainstem.

23. Describe the basic pathophysiology, common clinical manifestations, and appropriate work-up and treatment of intracranial hemorrhage.

24. Describe the evaluation and treatment of a seizure disorder, listing the anticonvulsants for partial or secondarily generalized seizures (phenytoin, carbamazepine, lamotrigine, gabapentin, valproate, oxcarbazepine and levetiracetam) and the anticonvulsants for absence or primarily generalized seizures (valproate, ethosuximide and lamotrigine).

25. Recognize that anticonvulsants may cause confusion, somnolence and ataxia at high serum levels and teratogenicity at minimal serum levels.

26. Describe this protocol for treating generalized status epilepticus: first, give lorazepam 0.1 mg/kg (4-8 mg) as an IV bolus, repeatable in 5-10 minutes if needed, followed by loading with either fosphenytoin 20 phenytoin equivalents (PE)/kg IV, no faster than 150 mg/min, or phenytoin 20 mg/kg IV, given in saline no faster than 50 mg/min.

27. Recognize that an emergent lumbar puncture (LP) is needed in patients suspected of meningitis, encephalitis or subarachnoid hemorrhage (if no blood is detected by CT scan in the latter), and describe the contraindications for an emergent LP.

28. Describe the cerebrospinal fluid (CSF) abnormalities which are typically found in meningitis, encephalitis, subarachnoid hemorrhage, or traumatic lumbar puncture.

29. Describe the emergent treatment of impaired consciousness from toximetabolic causes, particularly hypoglycemia, hypothermia, narcotic or benzodiazepine toxicity.

30. Describe how intravenous dexamethasone can reduce edema or herniation from certain cerebral lesions (tumor, abscess or encephalitis) or spinal cord lesions (metastatic cord

compression, myelitis), but primary treatment directed at the underlying lesion must soon follow.

31. Describe the typical signs and symptoms of *Herpes simplex* encephalitis, its diagnosis and initial treatment with an antiviral like acyclovir.

32. Describe the basic pathophysiology, common clinical manifestations, and appropriate work-up and treatment of multiple sclerosis.

33. Describe the treatments for migraine (abortive versus prophylactic therapy) and tension headache, and evaluation and treatment of headache from raised intracranial pressure.

34. Describe the evaluation of an acutely confused or demented patient, emphasizing reversible or treatable causes, with particular attention to Alzheimer's dementia.

35. Describe the basic pathophysiology, common clinical manifestations, and appropriate work-up and treatment of peripheral neuropathy.

36. List the pain treatment options for trigeminal neuralgia, postherpetic neuralgia, and radiculopathy.

37. Describe the diagnosis and treatment of more common neuromuscular disorders like polymyositis, myasthenia gravis, and amyotrophic lateral sclerosis.

38. For a patient with acute paralysis which may require mechanical ventilation, describe how myasthenic crisis, Guillain-Barre syndrome, or a spinal cord syndrome (spinal cord compression, acute myelitis) is diagnosed and initially treated.

39. Describe the basic pathophysiology, common clinical manifestations, and treatment of Parkinson's disease.

40. Describe the diagnosis and treatment of (familial) essential tremor.

41. Describe the usual indications and limitations of electroencephalography (EEG) and electromyography (EMG).

42. Describe the neurological criteria for brain death.

43. Describe various means of supportive care in incurable neurological conditions like anoxic encephalopathy (chronic vegetative state), amyotrophic lateral sclerosis and end-stage dementia.

Neuroradiology (CT, MRI) interpretation skills:

MRI is the superior imaging modality of the brain and spinal cord. CT is used if there are contraindications for MRI, or if the patient is unstable and quickly

deteriorating neurologically. In the latter case, a significant brain hemorrhage or midline shift should probably be detectable on CT scan. Knowledge of the patient allows for meaningful interpretation of CT or MRI scan findings. Recognize the following basic abnormalities:

44. Acute hemorrhage appears bright on CT scan, whether in the brain itself, or outside (subarachnoid, subdural) the brain parenchyma. On T2 weighted MRI, the center of an acute hemorrhage is brighter, with a darker periphery, which changes as the hematoma ages.

45. Acute infarction is seen sooner with MRI (DWI earlier than T2 weighted or other images) than CT. This appears to be a bright lesion on MRI (DWI, T2 weighted or FLAIR) or a darker lesion on CT that occurs within a vascular territory. Very early infarcts on CT may only appear as subtle effacement of the gray-white matter junction or sulci, or not appear until hours later.

46. Local mass effect or edema appears as a surrounding darkness (CT) or bright signal (T2 weighted or FLAIR series MRI) around the lesion itself. Contrast may help delineate the lesion within the surrounding edema. Greater mass effect may produce lateral shifts of cerebral hemispheres beneath the falx (across the midline) or down the foramen magnum.

47. Hydrocephalus, or ventricular enlargement, may involve some or all of the ventricles, depending on whether there is a specific site of obstruction to CSF flow. The ventricles appear enlarged (ex vacuo) also if there is significant loss of brain tissue.

48. CNS infection includes abscesses (mass lesions with surrounding edema), encephalitis or myelitis (inflammation, often viral, of the brain or spinal cord, appearing bright on T2 weighted or FLAIR series MRI) or meningitis (which may be noted as contrast enhancement of leptomeninges on MRI).

49. Primary brain tumors are typically solitary lesions, which may be hemorrhagic or heterogeneous, with surrounding edema. Metastatic tumors are often multiple, with surrounding edema, usually found at the gray-white matter junction of the brain. Epidural spinal cord metastases often arise from vertebral bone, and expand toward the spinal cord.

50. Multiple sclerosis plaques occur in the white matter of the cerebral hemispheres, brain stem and spinal cord, seen as bright lesions on T2 weighted or FLAIR series MRI. Acute lesions may enhance. The bright MRI lesions of MS may be impossible to distinguish from subcortical infarctions, so clinical knowledge of the patient is crucial.

51. Degenerative spine disease (spondylosis, disc herniations, and central canal stenosis) and its relation to the spinal cord and nerve roots are best seen with MRI. Intrathecal contrast may be necessary to better view these relationships with CT scanning (CT myelography).