Handout for
Integrating the Neurologic Examination
for the Neuropsychologist:
Neuroanatomic Localization of Common
Pathologies, Interventions and Higher
Cognitive Functions

Lola Morgan, MD

- History and Examination are key to diagnosing a neurological complaint.
- Adequate knowledge of anatomy, physiology, biochemistry and clinical features of the nervous system.
- An experienced neurologist can hypothesize based on a history which neurologic findings will be present or absent.
 Neurologic examination is used to confirm or refute the hypothesis.

- Chief Complaint
- History of present illness
- Past Medical History (including developmental milestones, as appropriate)
- Medications
- Family History
- Social History
- Review of Systems

All elements are influenced by memory, intelligence, language function, social, economic, and psychiatric features.

- After completion of history, the goal is to have an anatomic localization.
- "Principle of parsimony"
- · Occam's razor

• Localization

Cortex

Subcortex

Brainstem and cerebellum

Spinal Cord

Radicular

Peripheral nerve

Neuromuscular junction

Cortex

Frontal

Motor, fluency, prosody, attention, processing, planning, behavior/personality

Temporal

Parietal

Memory, language, auditory function

Somatosensory, sensory

Occipital

Visual, visual association cortices

mages courtesy of Gray's anatomy and Hardin MD/images

Subcortical

basal ganglia

caudate, putamen, globus pallidus, substantia nigra, subthalamic nucleus

thalamus

sensory relay to cortex

hypothalamus

homeostasis, endocrine, autonomic, limbic

Brainstem

Cranial nerves, descending motor, sensory, and cerebellar pathways, cardiac and respiratory function reticular activating system, midline raphe

Cerebellum

indirectly influences motor pathways to coordinate and plan movement, regulate eye movements and balance

Spinal cord

• Radicular

31 pairs of nerves

8 cervical

12 thoracic

5 lumbar

5 sacral

1 coccygeal

· Peripheral nerves

motor

sensory

autonomic

• Neuromuscular junction

impairments result in motor dysfunction without sensory involvement

• Components of the neurologic examination⁷

General behavior and appearance

Stream of talk Mood and affective responses

Content of thought

Intellectual capacity Sensorium

Speech

Dysphonia

Dysarthria Dysprosody Aphasia

• Components of the neurologic examination

Head and Face

Facial gestalt

Head abnormalities in symmetry or shape, measure circumference Look at the eyes for ptosis, pupillary size, interorbital distance Auscultate for bruits over neck vessels, eyes, temples

 Components of the neurologic examination

Cranial nerves

I—Olfactory
use an aromatic (coffee e.g.) non

irritating substance

II--Optic

Acuity and testing of peripheral fields by confrontation

III, IV, and VI Pupillary light reflexes Ocular motility

• Components of the neurologic examination Cranial Nerves

V-inspect the masseter and temporalis muscle bulk test all three divisions of facial sensation corneal reflex (V-VII reflex arc)

VII—muscles of facial expression

VIII--hearing

• IX and X:

listen for phonation and articulation (labial, lingual, and palatal sounds)

XI-spinal accessory

test head movement and shoulder shrug

XII—hypoglossal

tongue protrusion and inspect for atrophy

Motor

Muscle tone—make passive movements of joints to test for spasticity, rigidity or hypotonia. Determine if findings are upper motor neuron or lower motor neuron in origin Strength testing—graded on scale of 0 to 5

0 no movement

- 1 twitch
- 2 some movement
- 3 movement against gravity
- 4 less than full power
- 5 full power against resistance

Upper motor neuron

- Weakness
- No atrophy
- No fasiculationIncreased tone
- Hyperreflexia

Lower motor neuron

- Weakness
- Atrophy
- Fasiculation
- Decreased tone

• Deep tendon reflexes graded on a 0 to 4+ scale

jaw jerk (V afferent; V efferent) biceps reflex (C5-C6) brachioradialis (C5-C6) triceps reflex (C7-C8) finger flexion reflex (C7-T1) knee jerk (L2-L4) ankle jerk (L5-S1)



Sensation

superficial sensory (light touch, temperature, pain) deep sensory (joint position sense, vibration sensation higher sensory function (graphesthesia, astereognosis, statognosis)

• Cerebellar

Eye movements
Finger to nose
Rapid alternating hand
movements (dysdiadochokinesia)
heel to shin

• Gait

initiation balance fluidity

tempo turns

adventitious movements

Formulation

- Formulation or assessment
- Several sentences synthesizing chief complaint, history, neurologic findings which localize the anatomic origin and provide a potential list of diagnoses

- Unusual presentations of common diseases are more frequent than common presentations of rare diseases
- Generation of a differential diagnosis--I VINDICATE AIDS

latrogenic Allergic
Vascular Idiopathic
Infectious Drugs
Neoplastic Social

Degenerative Inflammatory Congenital Autoimmune Trauma Endocrine Disease and the Neurologic Examination

Neurodegenerative

Vascular

Traumatic brain injury

Epilepsy

Psychosomatic

Post surgical

• Neurodegenerative

Alzheimer's disease

Amyotrophic lateral sclerosis Fronto-temporal Dementia

Parkinson's disease Lewy Body Disease Multiple System Atrophy

• Alzheimer's disease8

memory disturbance

subsequent language and visuospatial dysfunction

neuropsychiatric dysfunction

depression

delusions

behavioral disturbances

wide variation

most prominent abnormalities on examination are higher cognitive function

• Neurologic examination

frontal release signs or primitive reflexes (snout, suck, grasp)

gegenhalten

variable resistance to passive movement

myoclonus

seizures

• Amyotrophic Lateral Sclerosis

Degeneration of motor neurons resulting in progressive weakness

Neurologic examination classically with mixture of upper and lower motor neuron findings

- Hyperreflexia
- Increased tone
- Atrophy
- Fasciculations
- Weakness
- Overlap syndrome⁹
- 30% of ALS patients may have frontotemporal dementia
- 15% of FTD patients may have ALS

• Fronto-temporal dementia8

Otherwise, nonfocal exam

Primitive reflexes
Clinical features reflect regional atrophy
Frontal: Orofacial apraxia
Perseveration
Echolalia or palilalia
Apathy
Temporal: Aphasia
Oral exploratory behavior

• Parkinson's disease

Rigidity

Rest tremor

Bradykinesia

Asymmetry

Masked fascies Decreased blink

Micrographia

Hypophonia

Seborrhea

Bradyphrenia

Approximately 2/3rds of patients with PD develop cognitive deficits

 Up to 40% of patients with PD develop dementia attributed to the presence of Lewy bodies¹⁰ • Lewy Body Dementia

Neurologic exam with findings of Parkinson's Disease

Autonomic disturbances

Hallucinations

40-65% of people with RBD develop LBD¹²

• Multiple System Atrophy

Autonomic failure associated with variable manifestations of Parkinsonism, cerebellar and pyramidal dysfunction

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Vascular disease
 Stroke syndromes

Arterial Territory

Multi-infarct dementia

 Neurologic exam of middle cerebral artery strokes contralateral hemianopsia conjugate eye deviation to side of stroke contralateral hemiplegia hemianesthesia

Dominant hemisphere involvement with aphasia Nondominant hemisphere involvement with hemineglect Neurologic examination of anterior cerebral artery strokes rare--<3% of strokes lower extremity weakness Deviation of head and eyes toward side of lesion Abulia Neurologic examination of posterior cerebral artery strokes quadrantanopsia

involvement of splenium of corpus callosum results in alexia without agraphia $\,$

visual agnosia

amnesia

bilateral infarctions of occipital lobes result in Anton's syndrome cortical blindness and denial of blindness

bilateral infarctions of occipito-parietal lobes result in Balint's syndrome optic ataxia, oculomotor apraxia, and simultanagnosia

• Neurologic examination of multi-infarct dementia 12

accumulation of defects through multiple cortical strokes
type of cognitive decline related to regional involvement
Distinction between subcortical and cortical involvement
Neurologically, cortical involvement will demonstrate aphasia, amnesia, visuospatial disturbances

Subcortical involvment accompanied by motor signs of gait disturbance, stooped posture and lack of facial features

Neurologic findings reveal minor findings

increased tone, asymmetric reflexes, extensor plantar response, visual field abnormalities, subtle hemiparesis or clumsiness

• Traumatic Brain Injury¹³

mild TBI/concussion

defined as a blow to head with loss of consciousness less than 30 minutes, amnesia peri-event and up to a one day after the injury, normal structural imaging, GCS 13-15

present with physical, cognitive, emotional symptoms which are self limited to 4-6 weeks

10% of patients continue to complain of symptoms

Neurologic exam is nonlocalizing

Epilepsy

Geschwind Syndrome¹⁴—Intense emotional life, hypergraphia, circumstantiality, hyposexuality, hyper religiosity

Neurologic examination is usually nonfocal Subtle elements based on localization of seizure onset • Psychosomatic

Symptom complex presentation without identifiable medical cause

Neurologic examination with multiple soft findings which are inconsistent

Hoover's sign

Astasia/abasia

Waddell's signs

- Tenderness tests: superficial and diffuse tenderness and/or nonanatomic tenderness
- Simulation tests: these are based on movements which produce pain, without actually causing that movement, such as axial loading and pain on simulated rotation
- Distraction tests: positive tests are rechecked when the patient's attention is distracted
- Regional disturbances: regional weakness or sensory changes which do not localize to neuroanatomy
- Overreaction: subjective signs regarding the patient's demeanor and reaction to testing

Neurosurgical intervention **Epilepsy**

Epilepsy

temporal lobectomy

Visual field cut

Dominant lobe—word finding difficulty, memory

Laser ablation of hippocampus

Neurosurgical intervention **Epilepsy**

- Frontal lobectomy
- · Corpus callosotomy

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