Neurologic examination: From clinical evaluation to radiology

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Learning objectives

To briefly describe the fundamentals of the basic neurologic examination.

To summarize the functionality of the main cerebral areas.

To illustrate with different pathological cases the way an accurate neurological information can facilitate the neuroradiologist interpretation of images.

To emphazise the importance of the cooperation between neurologists and neurorradiologist to improve the accuracy of diagnosis.
Background

The neurologic examination is an essential step in the evaluation of the neurological patient. Information on the findings of the neurologic exam is required to give the neuroradiologist a global understanding of the patient and can influence the interpretation of the images. Neurorradiologist should be familiar with the main neurological syndromes and should be able to infer the potential anatomic location of the lesion after receiving an accurate clinical information.

The basic neurological examination

For patients presenting with symptoms suggestive of a neurological problem, the neurological examination should:

- Determine, on the basis of an organized and thorough examination, whether in fact neurological dysfunction exists.
- Identify which component(s) of the neurological system are affected (e.g. motor, sensory, cranial nerves, several systems simultaneously).
- If possible, determine the precise location of the problem (peripheral vs central nervous system; region and side of the brain affected etc.).
- On the basis of these findings, generate a list of possible etiologies. Unlikely diagnoses can be excluded and appropriate testing (e.g. brain and spinal cord imaging) then applied in an orderly and logical fashion.

For the purpose of simplicity, the neurologic examination is divided into several steps. The basic steps include the following:

- Higher functions
- Cranial nerves
- Motor system
- Somatosensory system
- Reflexes
- Coordination and gait
Findings and procedure details

We retrospectively collected several patients assessed at our institution with different neurologic syndromes, including visual deficits, motor impairment, sensory symptoms and gait disturbances. Following the aforementioned basic steps of the neurological examination, we focussed on the correlation between the focal neurologic sign and the exact anatomic location of the lesion, demonstrating the way an accurate neurologic information can help to predict the lesional topography.

Components of Higher Functions

Higher functions include mental status and speech.

Main components of **mental status** are level of consciousness, attention, orientation, mood and memory.

Main **speech** abnormalities include dysarthria and aphasia.

- **Dysarthria**: is the inability to articulate spoken words. The quality of oration is impaired, but the content remains intact (slurred speech). The patient’s ability to understand and synthesize speech remains intact. It can result from multiple causes, including paralysis of pharyngeal, palatal, lingual, or facial musculature. It also is observed with cerebellar or pyramidal lesions. Thus, dysartria is not a very useful sign to figure out the precise location of a neurological lesion.

- **Aphasia**: is a disorder of linguistic processing, resulting in an inability to understand (sensory or Wernicke aphasia), properly execute speech (motor or Broca aphasia) or transfer signals from the Wernicke to the Broca area (conduction aphasia). In Wernicke aphasia there is an impairment in comprehension of sentences spoken by others and the content of speech is fluent but unintelligible because of frequent errors in word choice. Contrary, a person with Broca aphasia will have preservation of understanding, but will not be able to produce language. The language-eloquent area is most often located in the **left hemisphere** (in most right-handed persons and in more than two-thirds of left-handers, the left hemisphere is dominant for language). **Wernicke’s area** is located in the posterior section of the **superior temporal gyrus** of the dominant cerebral hemisphere, while **Broca’s area** is located in the **inferior frontal gyrus** of the dominant hemisphere Fig. 1 on page 11. This means that in a patient with Wernicke aphasia we will probably find the lesion in the temporal lobe of the dominant hemisphere and in a patient with Broca aphasia the lesion
will probably be located in the frontal lobe of the dominant hemisphere Fig. 2 on page 11 Fig. 3 on page 12.

Cranial nerves

There are twelve CNs numbered in order as they emerge from cranial to caudal in the brain. These are: olfactory (I), optic (II), oculomotor (III), trochlear (IV), trigeminal (V), abducens (VI), facial (VII), vestibulocochlear (VIII), glosopharyngeal (IX), vagus (X), accessory (XI) and hypoglossal (XII) nerves.

The I and II CNs are not true nerves but extensions of the brain. Therefore they are myelinated by oligodendrocytes and not by Schwann cells like the rest of the CNs, and are covered by meninges.

We will review the most common CNs assessed in a basic neurological examination:

• **Optic nerve (II) and visual pathway**

The optic nerve is formed by the convergence of axons from the retinal ganglion cells. After its formation, the nerve leaves the bony orbit via the optic canal. It enters the cranial cavity, running along the surface of the middle cranial fossa. Here the optic nerves from each eye unite to form the optic chiasm. At the chiasm, fibres from the nasal (medial) half of each retina (which are the fibers from the temporal field of vision) cross over, forming the optic tracts:

• Left optic tract - contains fibres from the left temporal (lateral) retina (corresponding to the left nasal visual field quadrants), and the right nasal (medial) retina (corresponding to the right temporal visual field quadrants).
• Right optic tract - contains fibres from the right temporal retina (corresponding to the right nasal visual field quadrants), and the left nasal retina (corresponding to the left temporal visual field quadrants).

Each optic tract travels to its corresponding cerebral hemisphere to reach the lateral geniculate nucleus, a relay system located in the thalamus; the fibres synapse here. Axons from the LGN then carry visual information via a pathway known as the optic radiation to reach the visual cortex in the occipital lobe Fig. 4 on page 13.

Although visual deficits can be quite complicated, in a schematic way, provided the globe is normal, the field defects can be defined from anterior to posterior Fig. 5 on page 14:

• Unilateral visual deficit (monocular visual impairment): optic nerve pathology Fig. 6 on page 15.
• Bitemporal hemianopsia: optic chiasm lesions Fig. 7 on page 16.
• Homonymous hemianopsia/cuadrantanopsia: lesions in temporal and parietal lobes (optic radiations) or occipital lobe (primary visual cortex) Fig. 8 on page 17.

• Oculomotor nerve (III), trochlear nerve (IV) and abducens nerve (VI):

These nerves coordinate eye movements and can be divided in four segments: intraaxial, cisternal, cavernous and extracranial.

Oculomotor nuclear complex (III) contains motor and parasympathetic fibers. It emerges from the dorsal aspect of the midbrain and exit at the medial aspect of the cerebral peduncles. The cisternal segments of the nerves are located in the interpeduncular/prepontine cisterns, between the posterior cerebral arteries (PCA) and superior cerebellar arteries (SCA). The parasympathetic fibers are located in the periphery of the nerves; therefore, external compression (eg, posterior communicating aneurysms or uncal herniation) often cause pupil-involving. The nerves Pierce the dura to become the interdural segments in the cavernous sinuses. Then the nerve enters the orbit through the superior orbital fissure to inervate some of the extra-ocular muscles (superior rectus, inferior rectus, medial rectus, inferior oblique, and levator palpebrae).

Trochlear nerve (IV) nuclei are located in the dorsal midbrain. The long cisternal segments of this nerve course within the quadrigeminal and ambiens cisterns. Similar to oculomotor nerve it also has a cavernous segment and an extracranial segment in the orbit, where it inervates the superior oblique muscle.

Abducens nerve (VI) nuclei are located in the dorsomedial aspect of the pons. This nerve also have a cisternal and cavernous segments. Once in the orbit, it inervates the lateral rectus muscle.

Nerves III, IV and VI are tested by observing how the eye follows an object in different directions. Damage to these nerves affect the movement of the globe. Both or one eye may be affected; in either case binocular diplopia (double vision when both eyes are open, can be corrected by covering either eye) will likely occur because the movements of the eyes are no longer synchronized. Therefore, in a patient with binocular diplopia an imaging evaluation is indicated to exclude a specific disease of the ocular motor pathways.

• Trigeminal nerve (V)

This nerve is formed by four nuclei, three sensory and one motor, which are located in the brainstem and the upper cervical cord. The nerves emerge from both sides of the
pons and courses anterosuperiorly through the preoptic cistern. Afterwards, it enter the Meckel cave. The sensory root makes synapses with the trigeminal ganglion, also known as Gasserian or semilunar ganglion, which is located in the inferior aspect of the Meckel cave. The postganglionic segment is composed by three distinct branches: The Ophthalmic (V1), the Maxillary (V2), and the Mandibular (V3) nerves. V1 and V2 are sensory roots. V3 is formed by motor and sensory roots. Combined, these nerves provide sensation to the skin of the face and also controls the muscles of mastication.

- **Facial nerve (VII)**

The facial nerve is a mixed CN with motor, parasympathetic, and sensory branches. It emerges from the lateral brainstem in the pontomedullary junction to enter cerebellopontine angle cistern. The facial nerve in the temporal bone can be divided into four segments: intracanalicular segment (in the internal auditory canal), labyrinthine segment, tympanic segment and mastoid segment. Then, the nerve emerges from the stylomastoid foramen and becomes the extracranial segment, which runs through the parotid gland and gives rise to the superior temporozygomatic, inferior cervicofacial, temporal, zygomatic, buccal, mandibular, and cervical branches Fig. 9 on page 18.

Symptoms of facial nerve palsy depend on the location of injury. The motor nucleus of CN VII has dorsal and ventral divisions that supply the upper and lower face, respectively. There is bilateral innervation of the dorsal division but only contralateral innervation of the ventral division. **Supranuclear lesions** affect upper motor neurons proximal to the motor nucleus in such locations as the cerebral cortex, internal capsule, and cerebral peduncle. This results in central facial palsy that affects the contralateral lower face but spares the forehead and brow muscles Fig. 10 on page 19. **Infranuclear lesions** occur distal to the facial nerve nucleus and produce peripheral facial palsy affecting the ipsilateral upper and lower face. Therefore, clinical patterns of facial palsy help to localize lesions as supranuclear, nuclear, or infranuclear and to focus our attention to the segment of interest in imaging assessment.

- **Vestibulocochlear nerve (VIII)**

The vestibulocochlear nerve is a sensory nerve. It is made up of two nerves, the cochlear, which transmits sound and the vestibular which controls balance. It is an intracranial nerve which runs from the sensory receptors in the internal ear to the brainstem nuclei located in the lower pons and finally to the auditory areas: the post-central gyrus and superior temporal auditory cortex.

- **Lower cranial nerves**
The lower cranial nerves (IX, X, XI, XII) have their nuclei and emerge from the medulla. They innervate the pharynx and larynx by the glossopharyngeal (CN IX) and vagus (CN X) (mixed) nerves, and provide motor innervation of the muscles of the neck by the accessory nerve (CN XI) and the tongue by the hypoglossal nerve (CN XII). As with all CNs, the context and clinical examinations, in case of suspicion of impairment of the lower cranial nerves, are determinant in guiding the imaging. In fact, the impairment may be located in the brainstem, in the peribulbar cisterns, in the foramina or even in the deep spaces of the face. The clinical localization of the probable seat of the lesion helps in choosing the adapted protocol. In the medulla, the intraaxial pathology is dominated by brain ischemia and multiple sclerosis. Cisternal and foramen magnum pathology is predominantly tumoral (schwannoma, menigioma).

In general, for all CNs whose nuclei and fascicles reside in the brainstem (all al them except I and II), because of the close proximity with other brainstem structures, patients typically present with associated brainstem dysfunctions that may produce highly localizing clinical syndromes.

**Motor system**

To understand how damage to the descending pathways affects motor function, it is important to know the normal anatomy of the motor system. The primary motor pathway, also called the corticospinal tract, arises in pyramidal neurons of the precentral gyrus or "primary motor cortex" (frontal lobe). There is a motor homonculus in this gyrus (Penfield's homunculus), with the feet represented near the superomedial part of the motor cortex and the leg, trunk, arm, hand and head represented progressively further inferior on the lateral side of the brain Fig. 11 on page 20. Axons arising from neurons in the precentral gyrus exit through the white matter and pass through the internal capsule where they are topographically arranged in the posterior limb. Corticospinal fibers traverse the middle portion of the cerebral peduncle of the midbrain and then the basal pons. They enter the pyramids of the medulla (from whence they get their name). Over 90% of the axons in the pyramids decussate just before reaching the upper cervical spinal cord (the pyramidal decussation), forming the lateral corticospinal tract. The remaining 10% fibers remain ipsilateral and form the anterior corticospinal tract. These fibers enter the lateral and ventral funiculus of the spinal cord to send their axons out via the spinal roots to directly control the muscle activity Fig. 12 on page 21.

Damage to neurons of the corticospinal tracts affects motor control in different ways, depending on the level of damage. If a stroke causes a lesion in the primary motor cortex or in the internal capsule, motor function on the opposite side of the body will be affected - this is called contralateral hemiplegia (half-paralysis) or hemiparesis (half-weakness) Fig. 10 on page 19 Fig. 13 on page 22 Fig. 14 on page 23. On the other hand,
if there is damage to the lateral spinal cord (where the lateral corticospinal tract axons are located), this will cause a motor deficit in the limbs on the same side of the body.

Somatosensory system

The somatosensory system includes multiple types of body sensations. However, these modalities are lumped into two different pathways in the spinal cord and have different targets in the brain. The first modality is called discriminative touch, which includes touch, pressure, proprioception (joint and muscle position sense) and vibration perception. The second group is pain and temperature.

The cell bodies of the first-order somatosensory afferent neurons are located in the dorsal root ganglion of the spinal nerve. The cell body gives rise to a single process that divides to form a peripheral axon and a central axon. The peripheral axon travels to and ends in the skin, muscle, tendon or joint to receive the sensory stimuli. The central processes travels to the spinal cord in bundles, where they synapse with the second-order neuron. Here the fibers split into 2 different anatomical pathways depending on the information carried. The posterior column-medial lemniscal pathway carries discriminative touch and proprioceptive information from the body. Whereas, the spinothalamic pathways carry pain and temperature information from the body. As we previously mentioned, facial sensation is carried via the 3 branches of the trigeminal nerve. This neuron’s ascending axons decussate to the opposite side at different levels, the spinothalamic pathway in the spinal cord and the medial lemniscal in the medulla.

Both the medial lemniscus and the spinothalamic tract send fibers cephalad; these fibers travel in the basal part of the tegmentum in the pons and midbrain on their way to the thalamus. Once in the thalamus, they synapse on third-order neurons. The third-order neurons then project, via the posterior limb of the internal capsule, to the primary somatosensory cortex, which is located in the postcentral gyrus of the parietal lobe Fig. 15 on page 24. Somesthetic cortex is organized in a sensory homunculus, which is analogous to the motor homunculus. Genital and leg fibers are located medially, whereas arm, hand, face, and tongue fibers are on the lateral surface of the somatosensory area Fig. 16 on page 25.

Considering that both somatosensory systems cross over to the contralateral side on their way to the cortex, any lesion involving the sensory pathways in the brainstem, thalamus, posterior limb of the internal capsule or somatosensory cortex in the parietal lobe will produce a sensory impairment in the opposite side of the body Fig. 17 on page 26 Fig. 18 on page 27.
Coordination and gait

Coordination and gait are usually described under a separate section because cerebellar disorders can disrupt coordination or gait while leaving other motor functions relatively intact. The assessment of cerebellar function will depend on the normal functioning of other systems addressed in previous sections of the neurological exam. Motor control from the brain, as well as sensory input from somatic, visual, and vestibular senses, are important to cerebellar function.

Cerebellar lesions can cause different kinds of coordination problems depending on their location. The term ataxia is often used to describe the lack of voluntary coordination of muscle movements that includes gait abnormality (gait ataxia). Specific tests to look for impaired coordination in the limbs include finger-to-nose (patient alternately touches the outstretched finger and nose of the examiner), heel-knee-shin (patient runs the heel of one foot down the shin of the other) and rapid alternating movements (patient alternately taps the dorsal and plantar surface of one hand onto the other hand). Loss of the ability to judge and control distance, speed, and power of a motor act is termed dysmetria. Abnormal alternating movements is called dysdiadochokinesia. The use of these terms provided by the neurologist should suggest a cerebellar disorder Fig. 19 on page 28 Fig. 20 on page 29
**Fig. 1:** Broca’s area is located in the inferior frontal gyrus of the dominant hemisphere (left hemisphere in most people), while Wernicke’s area is located in the superior temporal gyrus of the dominant cerebral hemisphere.

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Fig. 2: 80 y/o man with acute motor/Broca aphasia. Non-enhanced emergency brain CT shows a focal ischaemic area in the left frontal lobe, involving the left inferior frontal gyrus and Broca’s area.

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**Fig. 3:** 72 y/o woman with acute sensory/Wernicke aphasia. Non-enhanced axial FLAIR image shows an acute focal ischaemic lesion in the left temporal lobe with partial effacement of the adjacent ventricle, involving the left superior temporal gyrus and Wernicke’s area.

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**Fig. 4:** Visual pathway anatomy.

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**Fig. 5:** Basic visual field defects and its correlation with the lesions of the visual pathway.

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**Fig. 6:** A 10 y/o child with known NF-1 who develops a monocular visual field loss. Non-enhanced orbit CT shows an optic nerve glioma.

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Fig. 7: 54 y/o man with bitemporal hemianopsia. Axial T1 image post gadolinium administration shows a sellar lesion with suprasellar extension (grey arrow) that produces an anterior displacement and compression of the optic chiasm (white arrow). The lesion was proven to be a metastasis and the patient was posteriorly diagnosed with lung cancer.

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**Fig. 8:** 65 y/o man with left homonymous hemianopsia. Diffusion-weighted MR imaging shows an acute right occipital ischaemic lesion, involving the optic radiations and the primary visual cortex.

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Fig. 9: Representation of facial nerve anatomy and its lesions.

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Fig. 10: 73 y/o man with right central facial palsy and right hemiparesis. Diffusion-weighted MR imaging shows an acute left ischaemic lesion in the corona radiata and the posterior lenticulocapsular region, involving the corticospinal tract (motor pathway). In these locations motor fibers are bundled so closely in their descending path that small strokes can in fact paralyze the entire contralateral body and face.

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Fig. 11: Topographic map of primary motor cortex, located in the precentral gyrus (coronal view).

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Fig. 12: Representation of motor pathway anatomy.

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Fig. 13: 82 y/o male with acute left hemiplegia and left central palsy. Non-enhanced emergency brain CT shows a right nontraumatic frontal lobe hemorrhage with vasogenic oedema that compromise the right precentral gyrus (primary motor cortex). In the coronal reconstruction an impairment of the entire cortical motor somatotopic map (motor Penfield's homunculus) can be appreciated, which explains the entire contralateral body and facial motor function loss.

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**Fig. 14:** 65 y/o woman with acute paresis of the left lower limb. Diffusion-weighted MR imaging shows an acute left ischaemic lesion in the right frontal lobe that compromise the superomedial part of the right motor Penfield’s homunculus, which controls the motor function of the contralateral lower limb.

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Fig. 15: Representation of somatosensory pathways.

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Fig. 16: Primary somatosensory cortex topography in postcentral gyrus.

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**Fig. 17:** 74 y/o woman with acute right somatosensory deficit. Diffusion-weighted MR imaging shows an acute left ischaemic lesion in the right thalamus, involving the sensory tracts (spinothalamic tract and medial lemniscal pathway) in their ascending path.

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**Fig. 18:** 80 y/o male with acute right upper limb somatosensory impairment. Non-enhanced emergency brain CT shows a small left cortico-subcortical parietal ischaemic lesion that compromise the left postcentral gyrus (primary somatosensory cortex). In the coronal reconstruction an impairment of the middle part of the sensory homunculus can be appreciated, which corresponds to the contralateral upper limb somatosensory control.

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Fig. 19: 75 y/o woman with acute nystagmus, right dysmetria and gait ataxia. Diffusion-weighted MR imaging shows an acute ischaemic lesion in the right cerebellar hemisphere and vermis, with compression of the 4th ventricle.

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Fig. 20: 24 y/o male with one year history of headache, dizziness and progressive gait ataxia. Axial gadolinium-enhanced T1-weighted MR image shows a well-defined right cerebellar mass, predominantly cystic with a mural nodule which enhances vividly. The cyst wall does not enhance. The lesions was a cerebellar hemangioblastoma.

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Conclusion

Both neurologic exam and neuroimaging play an important role in the diagnosis of patients with CNS lesions. The neurologist should give an accurate clinical information and the neuroradiologist should be familiar with the basic neurological syndromes. The cooperation between neurologist and neuroradiologist has the potential to improve neuroradiological diagnostic accuracy.
References