Cranial Nerves 1, 5, 7-12

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Cranial Nerve I
Olfactory Nerve
- Nerve fiber modality: Special sensory afferent
- Function: Olfaction
- Remarkable features:
  - Peripheral processes act as sensory receptors (the other special sensory nerves have separate receptors)
  - Primary afferent neurons undergo continuous replacement throughout life
  - Primary afferent neurons synapse with secondary neurons in the olfactory bulb without synapsing first in the thalamus (as do all other sensory neurons)
  - Pathways to cortical areas are entirely ipsilateral

Cranial Nerve I
Clinical Testing
- Frequently overlooked in neurologic examination
- Aromatic stimulus placed under each nostril with the other nostril occluded, eg coffee, cloves, or soap
- Note that noxious stimuli such as ammonia are not used due to concomitant stimulation of CN V
- Quantitative clinical tests are available: eg, University of Pennsylvania Smell Identification Test (UPSIT)

Cranial Nerve I
Pathology
- Anosmia, hyposmia: loss of or impaired olfaction
  - 1% of population, 50% of population >60 years
  - Note: patients with bilateral anosmia often report impaired taste (ageusia, hypogeusia), though taste is normal when tested
- Dysosmia: disordered olfaction
  - Parosmia: distorted olfaction
  - Olfactory hallucination: presence of perceived odor in the absence of odor
    - Aura preceding complex partial seizures of temporal lobe origin
    - Schizophrenia
- Nasal passages, Olfactory epithelium, Central Pathways
  - Nasal: stimuli do not reach olfactory receptors
    - Smoking
    - Nasal and paranasal sinus disease: infection (influenza, herpes simplex, hepatitis), allergy, antihistamine overdose, cocaine
  - Olfactory epithelium: impairment of receptors or axons
    - Trauma: tearing of epithelium at cribiform plate
      - 5-10% of head trauma patients have impaired olfaction
    - Note: head trauma causing dural tear may result in CSF rhinorrhea
  - Intranasal tumor: papilloma
  - Toxic: certain antibiotics (aminoglycosides, tetracycline), corticosteroids, opiates, organic solvents
- Central pathways: olfactory bulb, olfactory tract, central projections
  - Intracranial tumor: olfactory groove meningioma, frontal lobe glioma
  - Aneurysm: anterior cerebral artery
  - Degenerative disease: Alzheimer’s, Parkinson’s, Huntington’s
    - Note: olfaction diminishes with age
Cranial Nerve V
Trigeminal Nerve
Nerve Fiber Modalities

- **General sensory afferent**
  - **Function:** All general sensory modalities from the:
    - Face and anterior scalp
    - Conjunctiva and eye
    - Paranasal sinuses and nasal cavities
    - Oral cavities, anterior 2/3 of the tongue, and teeth
    - Part of the external tympanic membrane
    - Meninges of the anterior and middle cranial fossae

- **Branchial motor efferent**
  - **Function:** Innervation of the muscles of mastication

Cranial Nerve V
Three Divisions

- **Ophthalmic branch (V1):** superior orbital foramen
  - Forehead, nose, and anterior scalp to the lateral canthus of the eye
  - Concha, eye, orbit
  - Nasal and frontal sinus mucosa
  - Dura mater of the dura

- **Maxillary branch (V2):** foramen rotundum
  - Cheek to the corner of the mouth, sparing the angle of the jaw
  - Upper lip, teeth, and gums, and palate
  - Floor of middle cranial fossa

- **Mandibular branch (V3):** foramen ovale
  - Sensory
    - Chin
    - Lower lip, teeth, and gums
    - Anterior 2/3 of tongue
    - Dura mater above the tentorium
  - Motor

  **Note:** brainstem lesions may produce an ‘onion skin’ pattern if sensory loss centering on the nose and lips

Cranial Nerve V
Sensory Clinical Testing

- **Pain (pinprick), touch (cotton swab, light finger touch), and temperature (cool tuning fork)** are tested in each of the three divisions

- **Note that vibration (tuning fork) is transmitted through the skull and is normally appreciated approximately equally on each side of the forehead when unilaterally stimulated**
  - Significant asymmetry of vibration is usually interpreted as a nonphysiologic response, so called ‘splitting’ of vibratory sensation at the forehead

Cranial Nerve V
Motor Clinical Testing

- **Palpation of masseter and temporalis muscles as the patient clenches the teeth**

- **Forced jaw opening, closure, and lateral movement**
  - **Note:** unilateral CN V motor weakness results in jaw deviation to the ipsilateral side with mouth open

- **Jaw (mandibular) jerk:** tapping the examiner’s finger placed on the patient’s chin normally elicits a brisk but slight contraction
  - An exaggerated response is a sign of upper motor neuron pathology above the foramen magnum
  - An unelicitable response has little clinical significance

Cranial Nerve V (and VII)
Clinical Testing

- **Corneal Reflex**
  - **CN V (V1):** afferent
  - **CN VII:** efferent
  - Cotton wisp touched to the cornea unilaterally produces a bilateral blink response
    - A unilateral CN V lesion results in absent blink bilaterally with stimulation ipsilateral to the lesion
    - A unilateral CN VII lesion results in absent blink ipsilateral to the lesion with stimulation of either cornea

Cranial Nerve V
Pathology

- **Supranuclear, Brainstem, Preganglionic/Ganglion, Peripheral branches**
  - Supranuclear: ischemia, hemorrhage, neoplasia, demyelinating disease, trauma
    - Contralateral facial and body sensory loss
  - Brainstem: as above, synapsis
    - Ipsilateral facial sensory loss
  - Preganglionic and ganglion ganglion:
    - Tumor (meningioma, schwannoma)
    - Inflammation (sarcoïd)
    - Infection (bacterial, fungal, mycobacterial)
Cranial Nerve V
Pathology

- Peripheral branches
  - Infection:
    - Herpes zoster
      - Usually V1: acute herpetic eruption and pain
      - May result in post-herpetic neuralgia: burning, aching, or lancinating; mild sensory loss; aggravated by touch
      - May result in corneal damage and visual loss
  - Other: bacterial, fungal
  - Inflammation: sarcoid, Sjogren’s syndrome, SLE
  - Tumor: sinus tumor, lymphoma, skin cancer, metastasis
  - Trauma

Cranial Nerve V
Pathology

- Trigeminal Neuralgia (Tic deloureux)
  - The most common neuralgia
    - Approximately 4 per 100,000; women to men 3:2
    - Symptoms:
      - Paroxysms of extremely severe pain in one or more of the divisions of the trigeminal nerve, usually V2 or V3
      - Pain is typically lancinating, lasting a few seconds
      - Pain may occur spontaneously or be triggered by touch, eating, or talking
      - Sensory testing of CN V is usually normal
  - Pathology
    - Usually of unknown etiology
    - May be due to compression by a tortuous blood vessel
    - May be a symptom of multiple sclerosis
  - Treatment
    - Medication: anticonvulsants (e.g., carbamazepine, gabapentin)
      - Less commonly, surgery

Cranial Nerve VII
Facial Nerve
Nerve Fiber Modalities

- Branchial motor efferent
  - Function: Innervate the muscles of facial expression, plus stapedius, stylohyoid, and posterior belly of digastric
- Visceral motor (parasympathetic) efferents
  - Function: Stimulation of the lacrimal, submandibular, and sublingual glands, and mucous membranes of the nose and hard and soft palates
- General sensory afferent
  - Function: General sensation from the concha of the auricle, possibly the external auditory meatus, and part of the external tympanic membrane
- Special sensory afferent
  - Function: Taste sensation from the anterior 2/3 of the tongue

Cranial Nerve VII
Clinical Testing
Muscles of Facial Expression

- Observation of the patient’s facial expression when relaxed and when speaking
- Observation when the patient is asked to wrinkle the forehead, smile, forcibly close the eyes, puff the cheeks, and contract the platysma
- Weakness is demonstrated by facial asymmetry, diminished nasolabial fold, widened palpebral fissure, reduced forehead wrinkling, impaired smile, and inability to puff out the cheeks
- Corneal reflex (see CN V)

Cranial Nerve VII
Upper and Lower Motor Neuron Lesions

- Upper motor neuron lesions: contralateral deficit
  - Frontalis and obicularis oculi muscles are spared due to bilateral motor cortex input to the part of the facial nucleus supplying these muscles
  - Loss of lower facial motor voluntary expression contralateral to the CNS lesion due to unilateral input to the part of the facial nucleus supplying these muscles
  - Note: Facial motor emotional expression is sometimes preserved as the corresponding neuronal input follows a different pathway
- Lower motor neuron lesions: ipsilateral deficit
  - Both upper and lower muscles of facial expression are affected ipsilaterally to the lesion
  - Note: Taste is affected if the lesion is proximal to the chorda tympani and spared if the lesion is distal to this nerve

Cranial Nerve VII
Clinical Testing
Taste

- Tested using a moist cotton swab dipped into sweet, sour, or salty preparations
- The patient protrudes the tongue, the cotton swab is applied unilaterally, and the patient is asked to identify the taste without retracting the tongue
Cranial Nerve VII
Pathology

- Upper Motor Neuron, Brainstem, Peripheral Nerve
  - Upper motor neuron/central facial palsy: ischemia, hemorrhage, tumor, trauma, infection, demyelination
    - Contralateral lower facial and extremity weakness
  - Brainstem: ischemia, hemorrhage, tumor, trauma, infection, demyelination
    - CN VI often involved

- Peripheral Nerve
  - Facial canal: facial weakness, loss of taste (if lesion proximal to chorda tympani), variably hyperacusis (if lesion proximal to nerve to stapedius), and decreased lacrimation and salivation (if lesion proximal to greater superficial petrosal nerve)
    - Bell’s palsy
    - Tumor: cholesteatoma, hemangioma
    - Trauma
    - Syringolostoid foramen: facial weakness in isolation
    - Trauma
    - Tumor: parotid gland tumor
  - Note: paradoxically tearing may increase ipsilaterally due to tears spilling over a weakened lower lid and to increased stimulation by corneal exposure

- Upper motor neuron/central facial palsy:
  - Ischemia, hemorrhage, tumor, trauma, infection, demyelination

- Brainstem: ischemia, hemorrhage, tumor, trauma, infection, demyelination
  - CN VI often involved

Bell’s Palsy
- Facial palsy of historically idiopathic etiology
  - Herpes simplex infection now most commonly accepted etiology
  - The most common CN VII disorder
  - Incidence: 23 per 100,000
  - Symptoms
    - Acute unilateral facial paralysis over hours to days
      - Often a viral prodrome
    - Variable loss of taste, lacrimation, and salivation, and hyperacusis
    - Numbness or pain of the ear, face, and tongue
  - Treatment
    - Antiviral: acyclovir, famcyclovir
    - Corticosteroid: prednisone
  - Management of ocular complications, eg corneal exposure
  - Prognosis
    - Majority recover fully over several weeks to months

Hemifacial spasm
- Unilateral involuntary variable clonic contraction of facial muscles, painless
- Etiology
  - Idiopathic
  - Compression of CN VII by branch of basilar artery
  - May develop following Bell’s palsy
- Treatment
  - Medication: baclofen, gabapentin, carbamazepine
  - Botulinum toxin
  - Surgical decompression

- Anomalous regeneration following CN VII lesion
  - Marcus-Gunn ‘jaw wink’ phenomenon: jaw movement causes ipsilateral partial ptosis
  - Crocodile tears: unilateral tearing with salivation

Cranial Nerve VIII
Vestibulocochlear Nerve
Nerve Fiber Modality

- Special sensory afferent
  - Function
    - Vestibular nerve: Balance
    - Cochlear nerve: Hearing

Cranial Nerve VIII
Clinical Testing
Cochlear Nerve

- Gently rub fingers near the patient’s ear and ask the patient to identify the sound and symmetry between the two ears
- Rinne test: 256 Hz tuning fork held to the mastoid and then near the patient’s ear for comparison
  - Normal response: the patient hears the vibrating sound better with air conduction than with bone conduction
  - Abnormal response: bone conduction is greater than air conduction, ie there is defect of the conduction of sound in the external auditory canal or middle ear or bone conduction hearing loss
- Weber test: 256 Hz tuning fork held to the middle of the forehead
  - Normal response: perception of the vibrating sound in the midline
  - Abnormal response: sound lateralizes to one ear
    - Abnormal response indicating a conduction defect: the sound lateralizes to the unaffected side
    - Abnormal response indicating a sensorineural hearing defect: the sound lateralizes to the opposite ear
Cranial Nerve VIII
Clinical Testing
Vestibular Nerve

- Observation of nystagmus
- Nystagmus of peripheral vestibular disorders is best observed with reduced ocular fixation
  - Examination in dim illumination
  - Observation of nystagmus on optic funduscopic exam with the opposite eye fixating and then covered (impairing fixation)
- Nystagmus of peripheral and central origins
  - Nystagmus of peripheral origin is:
    - Suppressed by fixation
    - Of mixed character, with both horizontal and rotary features
  - Nystagmus of central origin is:
    - Not suppressed by fixation
    - May be purely horizontal, vertical, or rotary, or of mixed type
  - Multidirectional, the fast component changing with the direction of gaze

- Dix-Hallpike positional test: patient positioned rapidly from a seated to supine position, the head hanging centrally, then to the right, then to the left
  - Nystagmus of peripheral vestibular origin: latency of 2-5 seconds, mixed horizontal-rotary, fast component beating away from the diseased side, lasting 5-60 seconds, fatigues with repetition
  - Nystagmus of central origin: no latency, horizontal, vertical, rotary, or mixed, often multidirectional, often lasting >60 seconds, does not fatigue

Cranial Nerve VIII
Pathology Primarily Affecting Hearing

- Conductive hearing loss
  - Sound is not transmitted to the inner ear
  - Causes: excess cerumen or foreign body in external canal, otitis externa, external or middle ear infections, allergy with serous otitis, tympanic membrane perforation
- Sensorineural hearing loss
  - Most common type; 23% population >60 years
  - Disorder of CN VIII or cochlea
  - Causes: older age (sometimes called presbycusis), Meniere’s disease, toxins (eg high dose aspirin), noise
- Central hearing loss
  - Rare
  - Causes: pure word deafness (disturbed auditory comprehension with intact visual comprehension), auditory agnosia (inability to recognize nonverbal sounds, eg ringing telephone, with otherwise intact hearing)

Cranial Nerve VIII
Pathology Primarily Affecting Vestibular Function

- Benign paroxysmal positional vertigo (BPPV)
  - Most common, 50% of peripheral vertigo
  - Symptom: vertigo precipitated by movement or change in position of head or body, eg getting out of bed or turning over in bed
  - Etiology: dislodged otoconia in the semicircular canals due to trauma, infection, or degeneration
  - Treatment:
    - Medication
      - Vestibular suppressants: meclizine, dimenhydrinate (Dramamine), lorazepam, diazepam, scopolamine
      - Antiemetics: meclizine, metoclopropamide, promethazine, prochlorperazine
    - Vestibular exercises

- Vestibular neuritis (vestibular neuronitis)
  - Symptoms: acute vertigo, nausea, and ataxia, with intact hearing, usually monophasic, lasting 2-3 days to 1-2 weeks
  - When hearing affected termed labyrinthitis
  - Etiology: viral infection of vestibular ganglion
  - Treatment: Vestibular suppressants and antiemetics and vestibular exercises similar to BPPV
    - Corticosteroids and antiviral agents
  - Meniere’s disease (endolymphatic hydrops)
    - Symptoms: episodic recurrent vertigo, fluctuating hearing, monaural fullness, and tinnitus
    - Etiology: dilation and rupture of endolymphatic compartment of inner ear due to trauma, autoimmune process, or genetic predisposition
    - Treatment: Medications similar to BPPV
      - Reduction of dietary salt plus salt-wasting diuretic

Cranial Nerve VIII
Pathology Cerebellopontine Angle

- Clinical syndrome
  - Progressive loss of hearing and vertigo
  - Sometimes associated with symptoms of involvement by CN VII (most common), V, VI, IX, X
- Etiology
  - Acoustic/vestibular neurroma (schwanomma): derived from Schwann cells of the vestibular portion of CN VIII
  - Meningioma
- Diagnosis
  - Neuroimaging: MRI, CT
  - Brainstem auditory evoked potentials
- Treatment
  - Surgery
  - Radiotherapy
Cranial Nerve IX
Glossopharyngeal Nerve
Nerve Fiber Modalities

- General sensory afferent
  - Function: General sensation to:
    - Posterior 1/3 of tongue, tonsils, and pharynx
    - External ear
    - Internal surface of tympanic membrane
- Visceral sensory afferent
  - Function:
    - Carotid body chemoreceptors
    - Carotid body baroreceptors
- Special sensory afferent
  - Function: Taste from the posterior 1/3 of the tongue

Note: CN IX provides both general sensation and taste to the posterior 1/3 of the tongue

Cranial Nerve IX (and X)
Clinical Testing

- Gag Reflex
  - CN IX: afferent (some contribution by CN X)
  - CN X: efferent
  - Procedure: Cotton swab or tongue blade gently touched to each side of the soft palate or posterior pharynx
  - Normal response: Bilateral palatal elevation and gag
  - Abnormal response:
    - Unilateral CN IX lesion: no response of the palate bilaterally when stimulating the side contralateral to the lesion and to command to say 'aah'
    - Bilateral CN IX lesion: no response of the palate bilaterally when stimulating either side, palatal elevation remains intact to command to say 'aah'
    - Unilateral CN X lesion: no response of the palate ipsilateral to the lesion with stimulation of either side; contralateral palatal elevation causes deviation of the palate and uvula away from the side of the lesion
    - Bilateral CN X lesion: no response of the palate when stimulating either side and no response of the palate to command to say 'aah'

Cranial Nerve IX
Pathology

- Cerebellopontine angle (see CN VIII)
- Jugular foramen
  - CN IX and X, sometimes associated with CN XI and XII
  - Etiology
    - Neoplasm
    - Infection
    - Trauma

Cranial Nerve IX
Pathology

- Glossopharyngeal Neuralgia
  - Rare: 0.7 per 100,000
  - Severe, sharp, lancinating pain in the region of the tonsil, radiating to the ear
  - Similar to trigeminal neuralgia in timing and nature of pain
  - May occur spontaneously or be triggered by swallowing, chewing, or speaking
  - May be associated with syncope
  - Pathology
    - Usually of unknown etiology
    - Occasionally due to compression of CN IX by carotid aneurysm, peritonsillar infection, or tumors of the oropharynx or skull base
  - Treatment
    - Medication: anticonvulsants (eg, gabapentin, carbamazepine)
    - Surgery

Cranial Nerve IX
Glossopharyngeal Nerve
Nerve Fiber Modalities

- Branchial motor efferent
  - Function: Innervates stylopharyngeus
- Visceral motor (parasympathetic) efferent
  - Function
    - Parotid gland
    - Carotid body
Cranial Nerve X
Vagus Nerve
Nerve Fiber Modalities

- General sensory afferent
  - Function: General sensation from:
    - Posterior meninges
    - Concha, posterior ear, and external acoustic meatus
    - Part of the external tympanic membrane
    - Pharynx and larynx

- Visceral sensory efferent
  - Function:
    - Larynx, trachea, esophagus, and thoracic and abdominal viscera
    - Aortic arch stretch receptors
    - Aortic body chemoreceptors

Branchial motor efferent
- Function: Innervates muscles of the oropharynx
  - Superior, middle, and inferior constrictors
  - Levator palati, salpingopharyngeus, palatopharyngeus, palatoglossus
  - Cricothyroid
  - Intrinsic muscles of the larynx

Visceral motor (parasympathetic) efferent
- Function:
  - Smooth muscle and glands of the pharynx, larynx
  - Thoracic and abdominal viscera
  - Cardiac muscle

Clinical Testing
- Observation of the palate on phonation, the patient instructed to say ‘aah’
  - A lesion affecting the palate may produce nasal speech
- Gag reflex (see Cranial Nerve IX)
- Vocal cord paralysis
  - May produce hoarse speech
  - Note: speech may be normal due to compensation by the intact contralateral vocal cord

Pathology
- Supranuclear lesions
  - Pseudobulbar palsy: disruption of corticobulbar tracts supplying CN motor nuclei from the precentral motor cortex produces a disturbance of speech, swallowing, and chewing, often associated with emotional lability and dementia
  - Unilateral lesion may produce no symptom or mild symptoms
  - Note: not solely a supranuclear disorder of CN X
  - Etiologies: ischemia, hemorrhage, infection, demyelination, trauma, degenerative
- Brainstem
  - Usually associated with CN IX

- Cerebellopontine angle (see CN VIII)
- Jugular foramen (see CN IX)
- Recurrent laryngeal nerve
  - Clinical presentation: ipsilateral vocal cord paralysis and hoarseness
  - Left recurrent laryngeal nerve has longer course, with looped recurrence in the chest around the aorta, the right is located in the neck
  - Etiologies:
    - Left recurrent laryngeal nerve: aortic arch aneurysm, pulmonary neoplasm, mediastinal adenopathy
    - Right or left recurrent laryngeal nerve: neck surgery

- Glomus Jugulare Tumor
  - Pathology: Vascular tumor arising from the glomus bodies (paraganglia chemoreceptor cells) of the jugular bulb
  - Erodes the jugular foramen
  - Causes compression of CN IX, X, and XI (sometimes extending to involve CN VIII and XII)
  - Peak incidence middle adult life
  - Symptoms and signs: ipsilateral pulsatile tinnitus and occasional bruit, impaired gag reflex, hoarse voice, dysphagia, ipsilateral sternomastoid weakness (CN XI), and occasionally ipsilateral hearing loss and vertigo (CN VIII), tongue paralysis (CN XII), and Horner’s syndrome
  - Diagnosis: neuroimaging (MRI preferred)
  - Treatment: mastoidectomy, resection, radiation
Cranial Nerve XI
Spinal Accessory Nerve
Nerve Fiber Modality

- Branchial motor efferent
  - Function: innervates
    - Sternocleidomastoid (sternomastoid) muscle
    - Trapezius muscle

Cranial Nerve XI
Clinical Testing

- Sternocleidomastoid muscle
  - Tilt the head up and towards the opposite side
  - Assess muscle bulk with the face turned to the opposite side
  - Patient tilts the head against resistance

- Trapezius muscle
  - Retracts the head and elevates, retracts, and rotates the scapula; enables abduction of the shoulder beyond 90 degrees
  - Assess muscle bulk with the shoulder and upper back fully exposed
  - Impaired trapezius motor function causes downward and lateral rotation of the scapula (winged scapula) and downward displacement of the shoulder
  - Test shoulder shrug against resistance

- A unilateral CN XI lesion causes:
  - Ipsilateral shoulder shrug weakness and winged scapula
  - Impaired head turn to the contralateral side

Cranial Nerve XI
Pathology

- Supranuclear lesion
  - Effect is incompletely understood

- Brainstem lesion
  - Usually associated with CN XII
  - Etiologies: ischemia, hemorrhage, infection, demyelination, syrinx, trauma

- Region of foramen magnum
  - Usually associated with CN XII, also CN IX and X
  - Etiologies: platybasia, Paget’s disease, tumors (meningioma, metastasis)

- Jugular foramen (see CN IX)

Cranial Nerve XI
Pathology

- Distal peripheral CN XI lesions
  - Axons to the trapezius muscle are potentially more vulnerable in the posterior triangle of the neck than are those to the sternocleidomastoid
  - Etiologies
    - Compression by carrying heavy loads on the shoulder
    - Excessive shoulder rotation or compression during surgery
    - Neck bites
    - Attempted suicide by hanging

Cranial Nerve XII
Hypoglossal Nerve
Nerve Fiber Modality

- Somatic motor efferent
  - Function: Innervates
    - 3 of the 4 extrinsic muscles of the tongue
      - Genioglossus, styloglossus, and hypoglossus
    - Palatoglossus supplied by CN X
    - All intrinsic muscles of the tongue

Cranial Nerve XII
Clinical Testing

- Examination of the tongue at rest for position, bulk, atrophy, and fasciculations
- The patient protrudes the tongue, moves it side to side, and resistance against the cheek is assessed
Cranial Nerve XII Pathology

- **Upper motor neuron lesion**
  - Unilateral lesion results in paralysis of the contralateral genioglossus muscle (supplied only by contralateral CNS, unlike the other tongue muscles which are bilaterally innervated by the CNS), causing the tongue to deviate away from the side of the CNS lesion.
  - Bilateral upper motor lesions cause a decrease in rapidity of tongue movements.

- **Lower motor neuron lesion**
  - Unilateral lesion results in ipsilateral paralysis of the tongue, causing the tongue to deviate to the side ipsilateral to the lesion.
  - Bilateral lower motor lesions result in bilateral tongue paralysis, impaired tongue protrusion, and poor lingual sounds (‘la, la, la’).

- **Note:** With a unilateral lesion the tongue deviates to the side of its weak muscle or muscles, whether the lesion is upper motor or lower motor neuron in origin.

Cranial Nerve XII Pathology

- **Supranuclear lesions**
  - Etiologies: ischemia, hemorrhage, tumor, trauma.

- **Brainstem lesions**
  - Often associated with CN XI.
  - Etiologies: ischemia, hemorrhage, demyelination, syrinx, tumor, trauma.

- **Region of foramen magnum (see CN XI)**
- **Jugular foramen (see CN IX)**

Cranial Nerve XII Pathology

- **Carotid Artery Dissection**
  - Pathology: Dissection of the intima of the carotid artery, allowing blood to enter the arterial wall, forming an intramural hematoma, and causing carotid artery stenosis, aneurysm, and/or mass effect.
  - Usually due to trauma: direct blow, lifting weights.
  - May occur spontaneously.
  - Symptoms and signs:
    - Pain involving the anterior neck, frontotemporal area, face, or eye.
    - Horner’s syndrome.
    - Cerebral or retinal stroke or TIA.
    - CN IX, X, XI, XII palsy due to compression.
  - Diagnosis: neuroimaging.
    - MRA, CTA, cerebral angiogram.
  - Treatment:
    - Anticoagulation.
    - Surgical or neuroradiologic intervention.