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Cranial Nerves

How to Incorporate Cranial Nerves in Your Assessment

Terminology

- CNS (Central Nervous System): Brain and spinal cord.
- PNS (Peripheral Nervous System): Made up of Somatic Function (cranial nerves and spinal nerves) and Autonomic Function (involuntary smooth muscle, cardiac muscle and glands, both sympathetic and parasympathetic).

Terminology

- Afferent (sensory): Impulses from peripheral tissues toward CNS.
- Efferent (motor): Impulses from CNS to muscles and/or glands.

Terminology

- LMN (Lower Motor Neurons): extremities
- UMN (Upper Motor Neurons): corticospinal tract (internal capsule, brainstem, spinal cord)
- UMN Paralysis=spasticity
- LMN Paralysis=flaccidity
- When LMN fires without UMN modulation=spasticity.

Terminology

- Reticular Formation=midbrain, pons, medulla
- Nucleus Tractus Solitarius - houses VII, IX, X
- Nucleus Ambiguus - houses IX, X, XI

Cranial Nerves

- Pons - houses nuclei of V, VI, VII
- Medulla Oblongata - houses nuclei of VIII, IX, XI, XII
- Medulla - axons form here from CN V, VII, IX, X, XI, XII
- All cranial nerves except XII receive innervation from both right and left tracts.

Neurological Disorders

- Wallenberg's Syndrome (Lateral Medullary Syndrome) - can affect V, IX, X, XI
- CVA
- TBI
- Multiple Sclerosis - myelin breakdown in CNS
- Huntington's - loss of neurons
- Parkinson's - decreased dopamine reaching the basal ganglia

Neurological Disorders

- ALS - affects UMN and LMN
- Guillain Barre Syndrome - peripheral nerves
- Myasthenia Gravis - affects nerve impulses
- Lambert-Eaton Myasthenia Syndrome
- Muscular Dystrophy

Neurological Disorders

- Polymyositis or Dermatomyositis
- Scleroderma - atrophy of esophageal smooth muscle
- Sjogren's Syndrome - severe dryness of mucosa
- Cancer
- Intubation

CN V-Trigeminal

- Sensory (tactile facial sensation)
  - Position bolus in the mouth
  - Pocketing
  - Facial sensation
- Motor (muscles of mastication)
  - Mastication
  - Hyoid Elevation
  - Velar Elevation

CN V-Trigeminal

- Bilateral innervation
- Unilateral UMN Lesion: Typically no deficits; maybe some mild and transient deficits.

CN V-Trigeminal

- Bilateral UMN Lesion: Difficulty with mastication; hypertonia in muscles of mastication; sensory deficits; reduced hyolaryngeal elevation with submandibular muscle involvement; significant oral phase deficits with impact on pharyngeal phase.

CN V-Trigeminal

- Unilateral LMN Lesion: Mandible deviates toward side of paralysis/paresis upon lowering; muscle hypotonia and atrophy apparent; impaired hyolaryngeal elevation; ipsilateral sensory dysfunction; likely mild to moderate oral phase deficits.

CN V-Trigeminal

- Bilateral LMN Lesion: Significantly impaired chewing abilities; muscle hypotonia bilaterally; bilateral sensory deficits; significantly impaired hyolaryngeal elevation; significant oral phase deficits with negative impact on pharyngeal phase.

Assessment of CN V

- Have the patient clench his teeth while you palpate the masseter and temporalis, feeling for symmetry and mass.
- Have the patient swallow while you palpate the hyoid.
- Have the patient close his eyes while you touch areas of his face, anterior 2/3 of tongue, teeth, inner cheek and hard/soft palate.
- Have patient open mouth with and without resistance and move jaw laterally.

CN VII-Facial

- Parasympathetic (salivation)
  - Submandibular
  - Sublingual
- Sensory (taste anterior 2/3 tongue)
  - Sensation tongue
  - Taste
- Motor (movement of facial muscles)
  - Tone/movement cheeks
  - Lip closure
  - Hyoid Elevation

CN VII-Facial

- Lower part of the face=contralateral innervation
- Upper part of the face=bilateral innervation

CN VII-Facial

- Unilateral UMN Lesion: Spastic paralysis; weakness of contralateral lower face and neck; weakness apparent during voluntary but not emotional movements; reduced salivary secretion contralaterally; reduced taste sensation from contralateral anterior ⅔ of tongue.

CN VII-Facial

- Bilateral UMN Lesion: Spastic paralysis of the entire face; severe loss of salivary secretion; loss of sense of taste from anterior ⅔ of the tongue; significant oral phase deficits.

CN VII-Facial

- Unilateral LMN Lesion: Flaccid paralysis of entire ipsilateral face; no or substantially impaired movement of all facial structures for both voluntary and emotional movements; eye tearing; drooling from corner of mouth; loss of salivation ipsilaterally; loss of taste from the ipsilateral anterior ⅔ of the tongue; significant oral phase deficits.

CN VII-Facial

- Bilateral LMN Lesion: Flaccid paralysis of the entire face; hypotonia and atroph;, severe loss of salivary secretion and sense of taste from the anterior ⅔ of the tongue; severe oral phase deficits.

Assessment of CN VII

- Have the patient wrinkle her forehead, close both eyes, close mouth, smile, pucker, frown, puff cheeks with air and say /pa/. Assess symmetry and range of movements.
- Test a variety of flavors (sweet, salty, sour, bitter.)

CN IX-Glossopharyngeal

- Parasympathetic
  - Parotid
- Sensory
  - Senses arrival of the bolus at the palate
  - Taste (posterior 1/3 tongue and oral pharynx)
  - Gag Reflex
- Motor
  - Pharyngeal constriction and shortening (stylopharyngeus)
  - Elevation of palate

CN IX-Glossopharyngeal

- Unilateral UMN Lesion: Little evidence of contralateral weakness or sensory loss.

CN IX-Glossopharyngeal

- Bilateral UMN Lesion: Complete loss of sensation and taste from the posterior ⅓ of the tongue; complete loss of sensation from the faucial pillars and posterior pharyngeal wall; impaired salivation from the parotid gland; impaired or absent gag; significant pharyngeal phase deficits, particularly with pharyngeal phase initiation.

CN IX-Glossopharyngeal

- Unilateral LMN Lesion: Loss of touch, pain, thermal and taste sensation in the ipsilateral posterior ⅓ of tongue; ipsilateral loss of sensation to faucial pillars and posterior pharyngeal wall; loss of salivary secretion from ipsilateral parotid gland.

CN IX-Glossopharyngeal

- Bilateral LMN Lesion: Complete loss of sensation and taste from the posterior ⅓ of the tongue; complete loss of sensation from the faucial pillars and posterior pharyngeal wall; difficulty in initiation of pharyngeal phase

Assessment of CN IX

- Apply hot and cold to posterior 1/3 of tongue, faucial pillar, palatine tonsils, posterior pharyngeal wall.
- Assess with CN X, by assessing symmetry and movement of the velum while patient says "ah".

CN X-Vagus

- Sensory (90%)
  - Taste in oropharynx (epiglottis/pharynx)
  - Sensation residue in pharynx, larynx, esophagus

CN X-Vagus

- Motor
  - Velopharyngeal Closure
  - Vocal Fold Approximation
  - Middle/inferior pharyngeal constriction
  - Pharyngoesophageal Segment Relaxation
  - Esophageal Peristalsis
  - Gag Reflex

CN X-Vagus

- Unilateral UMN Lesion: Mild contralateral vocal fold weakness possible, paralysis is unlikely; contralateral laryngopharyngeal sensory deficit probable.

CN X-Vagus

- Bilateral UMN Lesion (pseudobulbar palsy): strain/struggle characteristics; monopitch due to hypertonicity; hypertonic cricopharyngeal muscle; pyriform pooling; bilateral laryngopharyngeal sensory deficits; increased jaw and gag reflexes and emotional lability; significant pharyngeal phase deficits.

CN X-Vagus

- Unilateral LMN Lesion: Deficits vary by lesion location: possible ipsilateral deficit in velar elevation; possible ipsilateral defect in pitch modulation; possible ipsilateral loss of sensation from the laryngopharynx, valleculae and epiglottis; possible ipsilateral vocal fold paralysis in paramedian position; possible ipsilateral vocal fold paralysis in the intermediate position; decreased opening of the UES.

CN X-Vagus

- Bilateral LMN Lesion: Deficit pattern depends on level of lesions: possible velar immobility, vocal fold impairment or immobility due to bilateral cricothyroid paralysis or paralysis of all other intrinsic laryngeal muscles bilaterally; possible loss of sensation from the pharynx, laryngopharynx, valleculae and epiglottis; decreased opening of the UES; pyriform pooling; severe pharyngeal phase dysphagia with risk of aspiration and choking.

Assessment of CN X

- Have patient phonate; you listen to vocal quality.
- Assess pharyngeal movement through VFSS.

CN XI-Spinal Accessory Nerve

- Motor Function
  - Assists with velopharyngeal closure
  - Innervates the sternocleidomastoid muscle for head turn

CN XII-Hypoglossus

- Motor ONLY
  - Power source for the tongue muscles
  - Hyoid-Thyroid Approximation
  - Hyoid Anterior Movement

CN XII-Hypoglossus

- Only nerve with contralateral innervation only

CN XII-Hypoglossus

- Unilateral UMN Lesion: Spastic paralysis of contralateral genioglossus muscle; deviation of tongue toward weak side on protrusion (side opposite of the lesion)

CN XII-Hypoglossus

- Bilateral UMN Lesion: Weakness on both sides; unable to protrude the tongue beyond the lips; increased tone or spasticity; consonant imprecision; difficulty manipulating the bolus.

CN XII-Hypoglossus

- Unilateral LMN Lesion: The entire ipsilateral side of the tongue will appear shrunken or atrophied; may see fasciculations or fibrillations, seen as tiny ripplings under the surface of the tongue; the tongue will deviate toward the weak side (same side of the lesion); reduced range of tongue movement; consonant imprecision.

CN XII-Hypoglossus

- Bilateral LMN Lesion: Paralysis of both sides of the tongue, characterized by atrophy and fasciculations; movements of the tongue for speech and swallowing will be significantly impaired.

Assessment of CN XII

- Have patient stick out his tongue, move tongue and repeat /ka/ and /ta/. Assess movement, symmetry, fasciculations and atrophy.

Trigger Swallow Response - Pharyngeal

- Bolus stimulates IX, X, XI in Medullary Reticular Formation or Nucleus Tractus Solitarius (NTS).
- Incorporates NTS input from V, VII, XII.
- NTS signals motor nuclei in Nucleus Ambiguus to help fire IX, X, XI.
- Nucleus Ambiguus innervates muscles of velum, pharynx, larynx and upper esophagus (IX, X, XI), producing the pharyngeal swallow response.

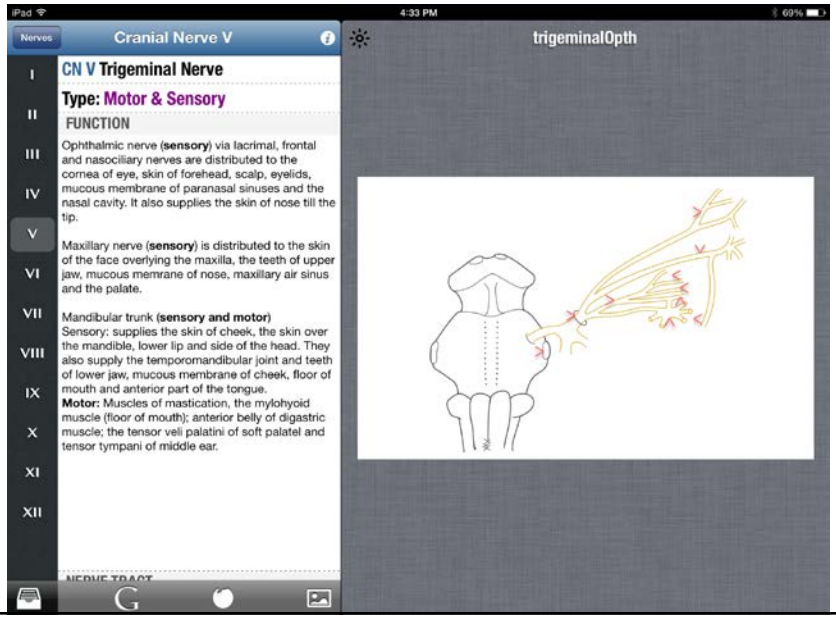
Stimulation

- Strongest ties to the NTS are anterior faucial arches, posterior tongue at the lower edge of the mandible, valleculae, pyriform sinuses and laryngeal aditus.
- Anterior faucial arches - strong connection between sensory receptors and NTS via afferent fibers of IX.
- Sensory stim: when NTS receives appropriate intensity of sensory input ,efferent response is triggered at the Nucleus Ambiguus; probably overall pattern of stim that triggers a swallow.

There's an App for That!

Cranial Nerves by Med Gears

https://itunes.apple.com/us/app/cranial-nerves-pocket-clinical/id469519587?mt=8



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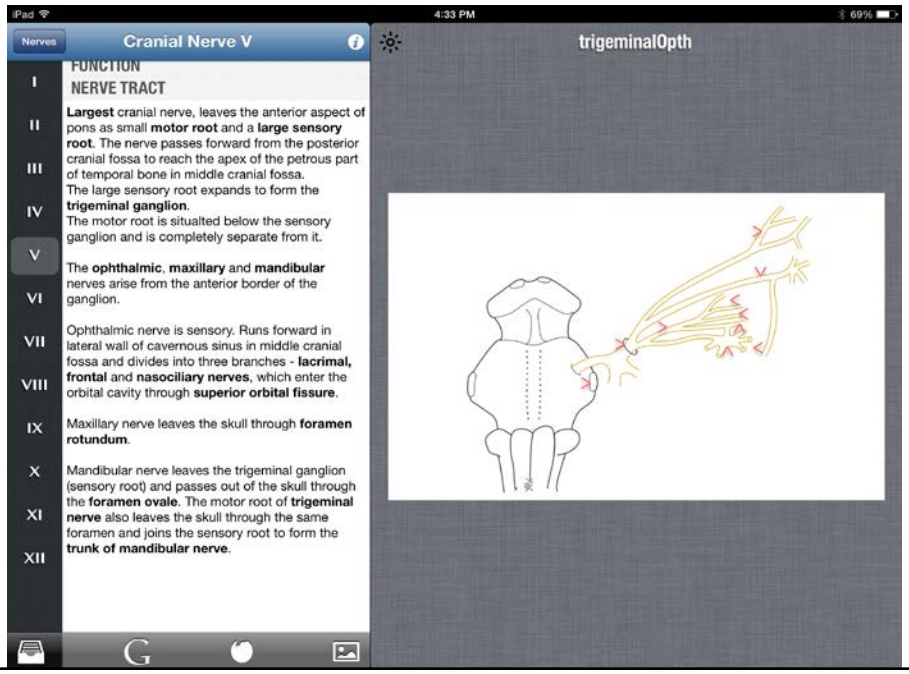

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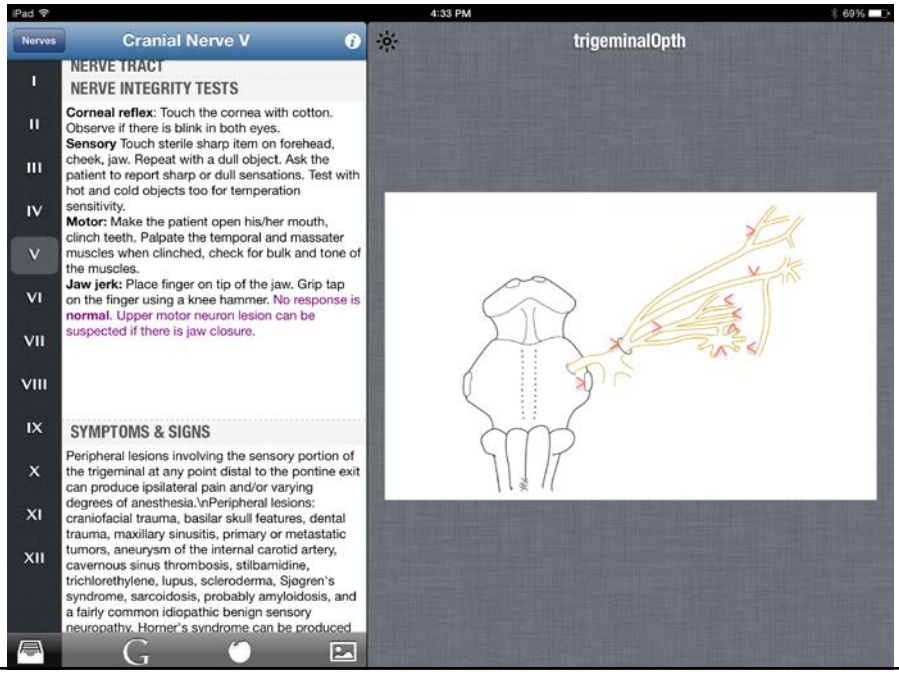

References

- K. Susie Jennings, Denise Siroky, C. Gary Jackson. Swallowing Problems with Excision of Tumors of the Skull Base: Diagnosis and Management in 12 Patients. Dysphagia 7:40-44 (1992)
- D Buchholz. Neurologic Causes of Dysphagia. Dysphagia 1:152-156 (1987)
- A. J. Miller Neurophysiological Basis of Swallowing. Dysphagia 1:91-100 (1986)
- W. J. Dodds. The Physiology of Swallowing. Dysphagia 3: 171-178 (1989)
- M Kronenberger and A Meyers. Dysphagia Following Head and Neck Cancer Surgery. Dysphagia 9: 236-244 (1994)
- S Jaradeh Neurophysiology of Swallowing in the Aged. Dysphagia 9: 218-220 (1994)

References

- Clinical Anatomy and Physiology of the Swallow Mechanism, Kim Corbin-Lewis, Julie M. Liss, Kellie L. Sciortino; Delmar Cengage Learning 2004.
- Evaluation and Treatment of Swallowing Disorders, Jeri Logemann; Pro-ed 1998.
- www.yale.edu/cnerves/