Neuroanatomical Localization

Cerebral Disease

-Altered mentation

-Behavioral Abnormalities

-Abnormal movement and posture

-Pacing, compulsive walking, circling, head pressing

-NOT paralysis, could have minimal paresis but the primary motor pathways are NOT from the cerebrum (they originate in the brainstem rubrospinal and vestibulospinal tract).

-Proprioceptive deficits

-Central blindness

-Seizures *Hallmark of cerebral disease*

Postural reaction deficits will be <u>CONTRALATERAL</u> with cerebral disease think about the dorsal column-medial lemniscus pathway

Blindness

PLR: A = CN II, E = CN III

Menace: A = CN II, E = CN VII

Loss of vision and PLR indicates the lesion is in the eye, optic nerve, optic chiasm, or optic tract

Central Blindness

If there is only vision loss (normal PLRs) the lesion is in the thalamus or cerebrum

-<u>Contralateral</u> loss of vision

-<u>Contralateral</u> menace deficits

Vestibular Disease

-Nystagmus

-Head tilt (leaning, rolling) *towards the side of the lesion

-Tight circling

-Strabismus *usually ipsilateral to the lesion

-Ataxia ipsilateral to the lesion without weakness

-Signs of nausea (vomiting/salivation)

Vestibular signs are almost always on the same side of the lesion!

CN deficits are always on the side of the lesion

When trying to differentiate between central and peripheral, keep in mind that the signs of central disease can mimic those of peripheral disease, so you need to look specifically for signs of central disease! Nothing specific to peripheral vestibular disease, just the absence of central signs

Peripheral vestibular disease will have "boring" nystagmus (horizontal or rotary, non-positional, conjugate) with the fast phase AWAY from the lesion

Associated problems for Peripheral Vestibular Disease

- Facial nerve paralysis/paresis
- Horner's Syndrome
- Otitis externa, otitis media-interna
- Pain on palpation of bulla +/- TMJ
- Pharyngeal pain

Central vestibular disease

-Nystagmus vertical, positional, deconjugate

-Can have horizontal, rotary, non-positional, conjugate nystagmus that mimics peripheral vestibular disease

-Altered level of consciousness

-Other CN deficits (V, IX, X, XI, XII) not so much VII since that can be seen with peripheral vestibular disease

-Long tract signs

Ascending (sensory) -> postural reaction deficits

Descending (motor) -> paresis or paralysis

*Ipsilateral thoracic and pelvic limbs affected

Ipsilateral cerebellar signs

-Hypermetria

-Intention tremor

-Absent menace

Neuroanatomical Localization

Paradoxical Vestibular Disease: Vestibular signs are **opposite** to the lesion

*Disease can be in the caudal cerebellar peduncle, flocculonodular lobe of the cerebellum, and some of the vestibular nuclei

-CN deficits, CP deficits, and hemiparesis will be ipsilateral to the lesion

-Ipsilateral cerebellar signs

-Head tilt/vestibular signs contralateral to the lesion

Peripheral Vestibular Diseases

-Congenital vestibular disease: Birth to three months of age (puppies and kittens)

-Hypothyroidism

-Neoplasia involving CN VIII: Schwannoma, lymphoma

-Osteosarcoma, fibrosarcoma, squamous cell carcinoma, adenocarcinoma

-May progress to cause central vestibular disease

-Otitis media-interna (OMI) common and usually caused by an extension of otitis externa

-Usually bacterial but *may* be fungal -Long-term treatment minimum of 6 weeks abx therapy based on culture and sensitivity

-Inflammatory polyp of the middle ear in cats

-Canine idiopathic vestibular disease "Old Dog Vestibular Disease"

-Diagnosis of exclusion, no CN VII or sympathetic signs

-Tx = supportive, good prognosis!

-Feline idiopathic vestibular disease

-Any age, sex, breed, unknown cause, dx, prognosis, and tx is the same as for dogs

-Trauma to petrous temporal bone

-Toxicity: Hearing loss/vestibular signs could be due to topical, oral, or parenteral toxins/drugs

Central Vestibular Disease

Neuroanatomical Localization

-Neoplasia: Progressive clinical signs, diagnosed via MRI +/- biopsy after a minimum database

-Primary: Glioma, choroid plexus tumor, ependymoma, meningioma

-Secondary: Lymphoma, schwannoma, other metastatic

-Nutritional: Thiamine deficiency – vestibular clinical signs are early in the disease progression

-Bacterial: Uncommon but seen in Frenchies, could be secondary to migrating foxtails, extension of otitis media-interna

-Viral: Canine Distemper virus, Feline infectious peritonitis

-Fungal: Cryptococcus

-Inflammatory: Meningoencephalitis of Unknown Etiology

GME, NE, NME

*Central vestibular disease is common in dogs with GME

-Metronidazole Toxicity: Dosages over 67 mg/kg/day but can be seen at any dosage and duration

-Injury to Purkinje cells in the cerebellum

Clinical signs of auditory disease

Peripheral vs Central

Peripheral Deafness

Conductive: Failure of sound transmission to the inner ear

-Problem is in the external or middle ear cavities

Sensorineural: Failure of sound transduction by the organ of Corti or failure of propagation of nerve impulse by the cochlear nerve

Anomalous-Degeneration of the organ of Corti (white and merle coat colors, white cats with blue eyes, puppies)

Diagnosed with Brainstem Auditory Evoked Potential (BAER)

-Helps differentiate peripheral from central disease

Neuroanatomical Localization

-No treatment, can test puppies at 6 weeks of age

Central Deafness Testing

-CSF analysis after imaging

-Serology, PCR, CSF culture

-Histopathology/cytology on biopsied material

Traumatic Brain Injury Lecture Key Points

Monro-Kellie Doctrine: The sum of the volumes of the brain, CSF, and blood within the fixed incompressible volume of the cranium is constant

Increased ICP > ischemia, necrosis, death

Goal is to maintain cerebral perfusion pressure (CPP)

Cushing Reflex: Global response of the brain to dangerously high ICP
Vasoconstriction leading to arterial hypertension
Baroreceptors leading to reflex bradycardia

Administering CORTICOSTEROIDS is NOT what we do in a TBI patient!

Goals of ICH therapy

- Maintain CPP by watching / not making these worse!
  - $PaCo_2$
  - Mean arterial blood pressure
  - PaO_2
  - Cerebral metabolic activity
  - Drugs

What is the most potent factor in controlling ICP/CBF? **$PaCO_2$**

-Chemical autoregulation, elevated **$PaCO_2$** leads to vasodilation

-Vasodilation leads to increased ICP!

TBI checklist

- Normotensive *not hypotensive
- PaCO2 (25-45 mmHg)
- PaO2/Pulse oxim (>60 mmHg/ >94%)
- Normoglycemic

Neuroanatomical Localization

- Jugular occlusion
- Head position 30 degrees
- Monitor repeatedly!

Clinical signs of elevated ICP

- Declining mental status
- Changes in pupillary size and reactivity
- Cranial nerve dysfunction
    - Gag reflex, physiologic nystagmus, declining motor signs
- Abnormal respiratory patterns
- Abnormal posturing

Cranial Nerve Review

Neuro Exam	Afferent CN	Efferent CN
Palpebral Reflex	V Medial: Ophthalmic Branch Lateral: Maxillary Branch	VII blink
Menace Response	II	VII blink
Pupillary Light Reflex	II	III pupillary constriction
Physiological nystagmus	VIII	III, IV, VI
Corneal Reflex	V Ophthalmic Branch	VI retract globe VII blink
Visual Tracking	II	
Trigeminofacial Reflexes -Corneal and palpebra -Vibrissae reflex -Lip pinch -Nasal stimulation	V	VII
Facial Symmetry	VII	V Mandibular *Masticatory muscle mass
Gag Reflex	IX, X	IX, X, XII

Neuro Localization

Cerebrothalamic	Cerebellum	Brainstem
+/- Altered mentation Behavioral Abnormalities Abnormal movement and posture	Ataxia Dysmetria Intention Tremor	Altered mentation (obtunded-coma)

Neuroanatomical Localization

Proprioceptive deficits Central Blindness (No Menace, Normal PLR) Contralateral Menace Deficit Seizures	Vestibular signs *can be paradoxical Absent menace ipsilateral Delayed postural reactions *ipsilateral Increased muscle tone and normal to hyperreflexive reflexes Anisocoria (rare)	Cranial Nerve Deficits (III-XII) Ipsilateral! Central vestibular signs can also be present Ipsilateral weakness and ataxia Postural reaction deficits ipsilateral

Head pressing, wide circling (towards lesion), compulsive walking/pacing = Cerebral

Postural reaction deficits are NOT specific! Can be cerebrum, brainstem, spinal cord, peripheral nerve

Proprioceptive placing deficits: All sensory information decussates so the lesion will be on the opposite side of the deficit

Minimal motor effect so if there are motor issues, it is likely brainstem or vestibular

Central blindness: Only vision loss, PLRs are normal