OCULAR MOTOR ANATOMY AND PHYSIOLOGY

1. **Extra-ocular muscles (EOMs)**
    - Need to overcome *viscous drag* and *elastic forces* in the orbit
    - Must have the ability to contract rapidly and resist fatigue
    - Compared to skeletal limb muscles:
        i. EOMs have <u>structural differences</u> including small motor units and both singly and multiply innervated fibers
        ii. EOMs have <u>biochemical differences</u> including high metabolic rate and different gene expression
        iii. EOMs are *less susceptible* to classic muscular dystrophies e.g. Duchenne's
        iv. EOMs are *more susceptible* to mitochondrial disorders and myasthenia gravis
    - 6 EOMs move the eye in different directions
        i. Medial rectus: adduction
        ii. Lateral rectus: abduction
        iii. Superior rectus: elevation
        iv. Inferior rectus: depression
        v. Superior oblique: intorsion and depression
        vi. Inferior oblique: extorsion and elevation
    - Clinical pearl
        i. Weakness of the EOMs affects *all classes* of eye movements i.e. smooth pursuit, saccades, etc.

2. **Innervation of the EOMs: 3rd, 4th and 6th cranial nerves**
    - <u>6th nerve (abducens nerve)</u>
        i. Innervates: lateral rectus
        ii. Nucleus is in the pons
        iii. Nerve exits the brainstem ventrally
        iv. Horizontal gaze requires coordinated contraction of lateral rectus in one eye, and medial rectus in the other eye
            a) To facilitate this, nerve fibers from the 6th nucleus cross the midline, enter the medial longitudinal fasciculus (MLF) and ascend to the 3rd nucleus in the midbrain
            b) So, each 6th nucleus is responsible for triggering horizontal gaze of *both* eyes in an ipsilateral direction
        v. Clinical pearl
            a) Lesions of the 6th nerve/fascicle cause ipsilateral weakness of lateral rectus (i.e. *one* eye cannot look that direction)
            b) Lesions of the 6th nucleus cause an ipsilateral horizontal gaze palsy (i.e. *both* eyes cannot look that direction)
    - <u>4th nerve (trochlear nerve)</u>
        i. Innervates: superior oblique
        ii. Nucleus is in the midbrain
        iii. Nerve exits the brainstem dorsally
        iv. 4th nerve palsy
            a) Manifests with diplopia and hypertropia that is most evident when looking *down and medially* with the affected eye

b) *Excycloduction* of the affected eye may be evident on fundus examination
c) Head tilt affects the degree of hypertropia and diplopia
    a. *Tilt away* from the affected side=better
    b. *Tilt towards* the affected side=worse

- 3rd nerve (oculomotor nerve)
    i. Innervates: superior rectus, inferior rectus, medial rectus, inferior oblique
    ii. ALSO innervates: levator palpebrae superioris (responsible for eyelid elevation), parasympathetic pupillomotor fibers (responsible for pupillary constriction)
    iii. Nucleus is in the midbrain
    iv. Complete 3rd nerve palsy manifests with:
        a) Ptosis
        b) Mydriasis
        c) Eye pointing down and out
    v. Clinical pearl: pupillomotor fibers travel in the outermost portion of the nerve
        a) Microvascular 3rd nerve palsy is usually *pupil-sparing*
        b) Compressive/infiltrative 3rd nerve palsy is usually *pupil-involving*

3. Classes of eye movements and their central control

- Vestibular-ocular reflex
    i. Allows us to fixate while we are moving e.g. running
    ii. Align the eyes during angular and linear acceleration of the head
    iii. Semicircular canal pathways
        a) Respond to *angular acceleration*
        b) Anterior canal, posterior canal, horizontal canal
    iv. Otolith pathways
        a) Respond to *linear acceleration*
        b) Saccule (movement in the vertical plane), utricle (movement in the horizontal plane, and with head tilts)
- Smooth pursuit
    i. Keeps a moving object on the fovea
    ii. Pathway involves: occipital cortex, cortical networks, pons, vestibular nuclei and cerebellum, culminating in firing of the appropriate ocular motor neurons
    iii. Choppy pursuit can be seen with lesions along this pathway
- Fixation
    i. Holds the eyes steady when focusing
    ii. Small fixational eye movements to prevent visual fading are normal e.g. physiological square wave jerks
    iii. Abnormalities of fixation:
        a) Nystagmus=slow drift away from the target
            a. Pendular nystagmus: slow phase initiates the movement, back-to-back-to-back slow phases gives the pendular appearance
            b. Jerk nystagmus: slow phase initiates the movement, each slow phases is followed by a (fast phase) corrective movement
        b) Saccadic intrusions=intrusive saccade away from the target
            a. Square wave jerks (excessive frequency seen in some basal ganglia and cerebellar disorders)
            b. Macrosaccadic oscillations
            c. Opsoclonus and flutter

iv. Eccentric gaze-holding: counteracts elastic forces of the orbit, with control from the neural integrator
    a) Key structures of the neural integrator include:
        a. Cerebellum=flocculus/paraflocculus
        b. Brainstem
            - For horizontal gaze=nucleus prepositus hypoglossi (NPH) & medial vestibular nucleus (MVN)
            - For vertical/torsional gaze=interstitial nucleus of Cajal (INC)
    b) Clinical relevance: *gaze-evoked nystagmus* occurs when the neural integrator is *leaky* (impaired)

- <u>Saccades</u>
    i. Rapid conjugate movements
    ii. Pathway includes cortex, basal ganglia, superior colliculus, cerebellum and brainstem
    iii. Brainstem structures that contain the excitatory burst neurons for *triggering saccades*:
        a) For horizontal saccades: paramedian pontine reticular formation (PPRF)
        b) For vertical/torsional saccades: rostral interstitial nucleus of the MLF (riMLF)
        c) Clinical relevance: a lesion of the PPRF will affect *horizontal saccades*, while a lesion of the riMLF will affect *vertical saccades*
    iv. Omnipause neurons
        a) Provide a constant brake to prevent excessive saccades (brake is lifted when a saccade is required)
        b) Contained in the nucleus raphe interpositus
        c) Clinical relevance: loss of omnipause neurons results in *opsoclonus* or *ocular flutter* (back to back saccades in all directions or only horizontally, respectively)
- <u>Optokinetic nystagmus</u>
    i. Response to a moving visual field (e.g. looking out a car window)
    ii. Input from visual and vestibular pathways
    iii. Made up of a slow phase (smooth pursuit) and fast phase (saccade)
    iv. Clinical relevance
        a) Saccadic palsies will result in impairment of fast phases
        b) Differences between horizontal and vertical OKN can help localization e.g. progressive supranuclear palsy causes impairment of fast phases in the vertical (down>upward)>>horizontal plane
- <u>Vergence</u>
    i. Aligns the eyes to achieve binocular single vision when viewing near and distant targets
    ii. Relies on sensory fusion
    iii. Clinical relevance
        a) Convergence insufficiency: blurring/diplopia and exotropia worse with near vision
            - Common with parkinsonism, aging, concussion/head trauma
        b) Divergence insufficiency: binocular blurring or diplopia with esodeviation greater at distance
            - Common with cerebellopathy