Lecture 10 – Orbit and control of eye movements

Overview of structures in the orbit (Moore pp 899, Netter Plate 1)
The orbit contains the eye, from which the optic nerve exits into the cranial cavity – optic canal. There are extraocular muscles to orientate the eye, with two separate muscles to move the eyelid. The cranial nerves that innervate these are III, IV & VI. The eye is responsible for lacrimation, so the lacrimal apparatus is situated medially in the orbit. The orbit also receives some supply from the ophthalmic and maxillary branch of CNV. The eye is also covered posteriorly by a fascial sheath (Fig 7.31A).

Relations / Bones of the orbit (Moore pp 899 Netter Plate 1)
Superiorly, the roof of the orbit is formed horizontally by the orbital part of the frontal bone. This separates the orbit from the anterior cranial fossa. Inferiorly, the floor is mainly formed by the maxilla and the orbital part of the zygomatic bone. In the lateral part of the floor, there is an inferior orbital fissure. Medially, the wall is primarily formed by the orbital plate of the ethmoid bone, also getting contributions from the frontal and lacrimal bones. The lacrimal bone contains a lacrimal fossa for the lacrimal sac, and the proximal part of the nasolacrimal duct. The posterior aspect is formed by the optic canal, lying in the lesser wing of the sphenoid just medial to the superior orbital fissure. Note that the orbit is actually a pyramidal shape, with an apex (posterior) and a base (anterior). The lateral wall is formed by the greater wing of the sphenoid and frontal process of the zygomatic bone.

Foramina in/around orbit (Notes Netter Plate 1)
The orbit is a crucial structure communicating with the middle cranial fossa posteriorly, and the outside world anteriorly. It consists of a number of foramina which will be highlighted here. Optic canal, superior and inferior orbital fissures, infraorbital foramen (just inferior to the orbital opening anteriorly), infraorbital groove (orbital surface of maxilla), supraorbital foramen/notch (frontal bone), lacrimal fossa (lacrimal bone), posterior + anterior ethmoidal foramen (orbital part of ethmoid).

Eyelids (Moore pp 901 Netter Plate 76)
The eyelids protect the cornea from external dust, too much light, and also help in the spreading of lacrimal fluid. The internal aspect is called palpebral conjunctiva. Note that there is a little bit of conjunctiva that reflects anterior surface of the eye (bulbar conjunctiva). The lines of reflection where the palpebral conjunctiva reflects to become the bulbar conjunctiva is called conjunctival fornix (inferior, superior). The superior and inferior eye lids are strengthened by bands of connective tissue called tarsal plates, which contain tarsal glands (these secrete lipids that protect lacrimal fluid from overflowing under normal conditions). The muscles associated with the eyelids are: levator palpebrae superioris, superior tarsal muscle, & palpebral part of orbicularis oculi. Note that superior tarsal is the deep part of levator palpebrae superioris.

The medial and lateral palpebral ligaments run in the medial and lateral angles of the eye. The medial palpebral ligament attaches to the medial wall of the orbit. The medial palpebral ligament provides origin & for attachment of the orbicularis oculi muscle. The eyelashes (cilia) extend from the eyelids. The orbital septum arise from the tarsal plates to the orbital margins (continuous with the periosteum of the orbit).

Lacrimal apparatus (Moore 901 Netter Plate 77)
The lacrimal apparatus is made up of: lacrimal glands (consisting of an orbital and palpebral part), lacrimal ducts, lacrimal canaliculi, lacrimal sac, and nasolacrimal duct. Lacrimal fluid flows from the glands, via the ducts (opening into the superior conjunctival fornix) to the lacrimal lake and then to the lacrimal sac via the lacrimal canaliculi (beginning called the lacrimal punctum). From here, they flow to the posterior part of the nasal cavity via the nasolacrimal duct, and then are swallowed. If lacrimation increases due to emotion, then it overcomes the lipid barrier provided by the tarsal glands. Parasympathetic fibres from CN VII innervate the lacrimal gland and increase production of lacrimal fluid.

The nerve supply to the lacrimal gland is as follows: presynaptic parasympathetic fibres from CN VII travel in the facial nerve, and then in the greater petrosal nerve to the pterygopalatine ganglion. Postsynaptic fibres from here innervate the lacrimal glands. Postsynaptic

sympathetic fibres arise from the superior cervical ganglion, traverse the pterygopalatine ganglion with the parasympathetic fibres to innervate the lacrimal glands.

Structure of eye (Moore pp 905 Netter Plate 82/83, Visual Systems Lect.)
The eye is made up of three layers namely: fibrous, vascular pigmented layer, retinal layer. The fibrous layer is formed by the sclera (posterior 5/6 of eye ball), and cornea (anterior 1/6 of eyeball). The sclera is continuous with the cornea. The middle vascular layer is formed by the choroid, ciliary body and iris. The choroid becomes the ciliary body anteriorly. The inner most layer is the retina, which contains light receptor cells (cones and rods) that are involved in phototransduction and neural processing.

Muscles of the orbit (Moore pp 909 Netter Plate 79)
The muscles of the orbit consist of the extrinsic eye muscles. These are: levator palpebrae superioris (elevates the superior eyelid), four recti muscles (sup, inf, lat, med), and oblique muscles (sup + inf). The recti muscles arise in the common tendinous ring, and splits into horizontal and vertical planes to attach to the anterior ½ of the sclera.

Arteries of the orbit: ophthalmic artery (Moore pp 912 Netter Plate 80)
The main arterial supply to the orbit is from the ophthalmic artery (branch of internal carotid artery), which enters the orbit via the optic canal, the infraorbital artery running the infraorbital foramen, and the central artery of the retina. The central artery of the retina arises inferiorly to the ophthalmic artery to enter the optic nerve. It emerges at the optic disk and then spreads laterally to run along the inner layer of the retina, supplying it. The retina is also supplied by the highly vascular choroid layer. The ophthalmic artery gives rise to many posterior ciliary arteries, which nourish the outer-non-vascular retinal layer by entering the choroid, and some also enter between the sclera and choroid to anastomose with the anterior ciliary arteries (continuations of muscular branches of ophthalmic artery)

Veins of the orbit: ophthalmic veins (Moore pp 913 Netter Plate 80)
Basically: superior and inferior ophthalmic veins drain the eye to the cavervous sinus, via the superior orbital fissure. The central vein of the retina usually enters cavernous sinus separately. The vorticose veins, from vascular layer of eye, join the inferior ophthalmic vein. Note that any anastomoses with facial veins may spread facial infection to orbit, then into cranial cavity (cavernous sinus).

Nerves of the orbit: from ophthalmic nerve - V_1 (Moore pp 911 Netter Plate 116)
The ophthalmic nerve branches off into smaller nerves. From superior to inferior these are: frontal, lacrimal, nasociliary nerves. The frontal nerve proceeds to branch into the supraorbital, and supratrochlear nerves → supplies the upper eyelid, forehead and scalp. The lacrimal nerve innervates the lacrimal gland, contains both sensory fibres, and parasympathetic secretomotor fibres that stimulate lacrimal production. The nasociliary nerve gives rise to: sensory root of ciliary ganglion, long ciliary nerve, posterior and anterior ethmoidal nerves (supply mucous membranes of ethmoidal and sphenoid sinuses), and infratrochlear nerves (supplies sensory innervation to eyelids, conjunctiva, skin of nose, and lacrimal sac).

Nerves of the orbit (Moore Table 7.8 pp 910, Netter Plate 115)
Now, considering the innervation of muscles of the orbit. Muscles include: superior, inferior rectus, medial, lateral rectus, levator palpebrae superioris, superior, inferior obliques. Note that the superior oblique muscle runs through the trochlea (pulley) before attaching to the sclera → hence supplied by the trochlear nerve (CN IV). The lateral rectus muscle is supplied by the abducens nerve (CN VI, i.e.: muscle which **abduc**ts the eye). The rest of the muscles is supplied by the oculomotor nerve (CN III).

The oculomotor nerve has two main divisions: superior and inferior divisions. The superior division supplies the levator palpebrae superioris + superior rectus muscles. The inferior division supplies the: inferior rectus, inferior oblique, and medial rectus muscles. It also forms a branch to the ciliary ganglion, which gives short ciliary nerves that provide parasympathetic fibres to the pupillary sphincter muscle and the ciliary muscle.

Nerves of the orbit – optic (CN II) and maxillary (CN V_2) (Netter Plate 114/116)
The optic nerve is central placed in the orbit and gains access to it via the optic canal. This is responsible for vision. The optic nerve has a meninges covering including: dura, arachnoid, and pia). It enters the eye at the optic disk – convergence of all the fibres from the ganglion cell layer. The maxillary branch of the trigeminal nerve (CN V_2) exits the cranial cavity via the pterygopalatine fossa. Gives off a zygomatic branch that runs along floor of orbit, and gives off parasympathetic fibres to innervate the lacrimal gland, via the lacrimal nerve. Other branch is the infraorbital nerve.

Sympathetic and parasympathetic innervation of eye (Moore pp 905, Nolte 5th Ed pp 444))
We now know that sympathetic innervation of the eye causes dilation of the pupil, whereas parasympathetic innervation (CN III) causes pupillary constriction, and lens thickening (accommodation). The pupillary sphincter muscle (circular smooth muscle) is responsible for constriction and the pupillary dilator muscle is responsible for the dilation of pupil. Note that the pupillary dilatory muscle is a radial muscle within the iris. The lens is regulated in thickness thereby allowing for focusing on near objects. The parasympathetics via CN III, cause thickening of the lens via shortening of the suspensory ligaments, therefore allowing for near vision. Both the pupillary light reflex, and accommodation reflex tests for the oculomotor nerve (CN III).

Course of parasympathetics to eye (Nolte 5th Ed pp 444, Fig 17-38)
Note that Fig 17-38 was in our formative practical exam and it is good chance it will also be in the summative exam – so learn it!

Note that parasympathetics constrict the pupil and thicken the lens. So if we want constriction of the pupil, lets follow the pupillary constriction reflex pathway (Fig 17-38). Light is shined into one eye, it is interpreted by the photoreceptors (cones and rods), the signal is passed onto the bipolar neurons and retinal ganglion cells. The axons of which converge to form the optic nerve. The fibres of the optic nerve merge at the optic chiasm. The optic tract axons now project to the lateral geniculate nucleus (some) and also to the brachium of the superior colliculus to terminate in the pretectal area. The pretectal neurons project bilaterally to the Edinger-Westphal nucleus of oculomotor nerve. The axons of neurons in the Edinger Westphal nucleus travel in CN III as preganglionic parasympathetic fibres, within the inferior division of the oculomotor nerve, to synapse at the ciliary ganglion. Post – synaptic parasympathetic fibres travel in the short ciliary nerves that innervate the pupillary sphincter muscle – causing constriction of the pupil.

Course of sympathetics to eye (Notes)
Sympathetic nerve fibres to the eye dilate the pupil. Cell bodies of preganglionic sympathetic fibres lie in the lateral horn of the spinal cord – T1 segment. Preganglionic axons exit via the ventral root, to enter the spinal nerve. Then they exit via the white ramus communicans to enter the sympathetic chain (paravertebral ganglia). They DO NOT synapse here, but ascend superiorly to the superior cervical ganglion – synapsing here. Post-synaptic sympathetic fibres travel with the internal carotid artery – the internal carotid nerve and plexus – and then with the ophthalmic artery to the ciliary ganglion. The fibres do not synapse here, but travel through to the pupillary dilator muscle and superior tarsal muscle of the upper eyelid via the short ciliary nerves.

Horner's Syndrome (Nolte 5th Ed pp 278, Moore pp 912)
Horner's syndrome is characteristic condition resulting in ptosis (drooping eyelid – paralysis of superior tarsal muscle), miosis (pupillary constriction due to pupillary dilator muscle paralysed), and anhydrosis (lack of sweating in ipsilateral face). The reason for Horner's syndrome is because there is an interruption in the descending sympathetic pathway.

Course of nerves and vessels from orbit and cranial cavity (Notes)
Optic canal: ophthalmic artery and optic nerve
In the superior orbital fissure, there is a common tendinous ring that encircles the origins of the recti muscles of the eye. Lateral to this ring runs: lacrimal n, frontal n, trochlear n (CN IV), ophthalmic veins (fusion of superior and inferior ophthalmic veins). Structures that run within

the tendinous ring include: sup + inf divisions of oculomotor nerve (CN III), abducens n (CN VI), nasociliary n (branch of V_1).

Elevators of upper eyelid (Moore pp 910 Table 7.8, Netter Plate 76/79)
The upper eyelid is elevated by two muscles: levator palpebrae superioris + superior tarsal muscle. Note that parasympathetic innervation of the former, and sympathetic innervation of the latter causes the elevation to occur. The parasympathetic fibres concerning the former are conveyed within CN III, whereas sympathetic fibres are conveyed from T1. CN III damage will cause dilated pupils (due to loss of pupillary sphincter action), and severe ptosis (due to paralysed levator palpebrae superioris muscle)). Sympathetic interruption → Horner's syndrome will cause mild ptosis and small pupil.

Movements of eye by extraocular muscles (Notes, Moore pp 910 Table 7.8)
The eye can move in an extraordinary number of positions. Note that the visual axis is medial to the orbital axis (23^0). This is because of the angle at which the optic nerve exits the orbit. The movements we refer to as only concerning a single eye (i.e.: when you abduct your right eye, your left eye seems to adduct). The can perform the following actions: elevation/depression, abduction/adduction, intorsion/extorsion (medial and lateral rotation of eyeball). Note that clinically, we test all the muscle actions, but anatomically we associate individual muscle with an action.

Extraocular muscles – lateral and medial recti (Notes, Moore pp 909, Netter Plate 78)
Basically the medial rectus → CN III (Oculomotor), lateral rectus → CN VI (Abducens). Medial rectus → adducts eye, lateral rectus → abducts eye. Check ligaments are expansions of the fascia associated with these muscles, and these are attached to the lacrimal and zygomatic bones, which in turn contribute to the walls of the orbit.

Extraocular muscles – superior and inferior recti (Notes, Moore pp 910 Table 7.8)
Note we mentioned how all the muscles work concurrently, and each muscle can have more than one function. Thus, the superior rectus muscle elevates, adducts and intorts the eyeball → CN III (Oculomotor). Inferior rectus muscle depresses, adducts, extorts the eyeball → CN III (Oculomotor).

Extraocular muscles – superior and inferior obliques (Notes, Moore pp 910 Table 7.8)
Innervation: superior oblique → CN IV (Trochlear), inferior oblique → CN III (Oculomotor). Actions: superior oblique → depress, abduct, intort eyeball (i.e.: 'SIN'), inferior oblique → elevate, abduct, extort eyeball.

Summary of actions and muscles responsible (Use this as a test to check yourself!)

Testing extraocular muscles (Talley & O Connor pp 367, Fig 10.9)
Note that medial and lateral movements are solely done by the medial and lateral rectus. The other eye movements are more complicated. When the eye is abducted: the superior movement is done by the superior rectus, inferior movement by the inferior rectus. When the eye is adducted: the superior movement is done by the inferior oblique, the inferior movement is done by the superior oblique. You can test by holding a pin and moving it in a H shape – starting from the middle. Do this and say the muscles you are testing!

Damage to CN III – Oculomotor (Notes, Moore pp 912, Nolte 5th Ed pp 297)
Note that CN III innervates all the extraocular muscles except: lateral rectus, and superior oblique muscles. Thus these functions will prevail. Lateral rectus → abduct the eye. Superior oblique: depress, abduct, intort eyeball. Thus a person with oculomotor palsy has a depressed, abducted, intorted eyeball. Note that CN III also supplies levator palpebrae superioris (parasympathetic), and also pupillary sphincter muscle (parasympathetic). Thus the upper lid becomes droopy and pupils are dilated due to CN III palsy. Is this all ipsilateral, contralateral, or unilateral? It turns out that these characteristics are seen ipsilaterally to the lesion, because the nerve has emerged from the oculomotor nucleus, therefore only supplies muscles ipsilaterally.

Damage to CN IV or VI (Notes)

Trochlear damage (CN IV) will cause paralysis of superior oblique muscle. The functions involved are: depress, abduct, intort eyeball. Note that other muscles may compensate for some of the actions, but there is a level of difficulty in performing these actions. Thus, the person will not be able to depress their eyeball in an adducted position (H sign test earlier). CN VI damage will cause paralysis of lateral rectus muscle. Actions affected: abduction of eye. Eye is deviated medially at rest – called medial strabismus. Some abduction may be possible due to superior and inferior oblique being intact but not much at all.

Strabismus refers to a deviated eye – which means the visual axis of one eye is not parallel to the other eye. Diplopia refers to double vision. This is because light is falling on different parts of the retina due to slight movement differences. This causes a paler image to be seen.

Location of oculomotor, trochlear, and abducens nuclei (Nolte 5th Ed pp 297, Fig 12-4)
The oculomotor nucleus is located in the rostral midbrain. Note that the nuclei are located depending on the supply of their fibres. That is: the nucleus supplying the levator palpebrae superioris is actually located along the midline – innervating the muscle bilaterally. The nuclei supplying the superior rectus muscle, supplies the contralateral eye. The nuclei supplying the medial rectus, inferior oblique and inferior rectus, supply the ipsilateral eye. What is the significance of all these crossing over? It turns out, that there is little significance because the oculomotor nuclei of both nerves lies so close to the midline, that lesion to one nuclei usually affects the other as well.

The trochlear nucleus us located in the caudal midbrain, at the level of the inferior colliculus. Cell bodies of fibres supplying one eye are actually located contralaterally. Thus damage to a particular nucleus will cause paralysis of the contralateral eye.

The abducens nucleus is located in the caudal pon, just beneath the floor of the 4th ventricle. Fibres from the abducens nucleus project ipsilaterally to supply the lateral rectus muscle. Thus, damage to one abducens nucleus will affect the ipsilateral lateral rectus muscle.

Types of eye movements (Dorland's Medical Dictionary)
Note that the eye is able to do a variety of movements, in addition to the usual functional movements. Saccades: this is when the eye is able to fixate on an object, abruptly moving the eye to follow the object from one point in space to another point. Fixation is when the eye is able to fixate on an object although the head is moving. For example: looking at the book while moving your head in all directions. The eye can also fixate on moving objects → smooth pursuit. For example: You are playing cricket, need 6 runs to win, bowler bowls – your eye is on the moving ball – you hit from the 'meat' of the bat – goes for 6 – you win the match for India! Each eye can match the exact movement of the other eye, called conjugate movements. The eyes can also converge and diverge.

Control of eye movements – Conjugate movements (Nolte 5th Ed pp 515-524, Fig 21-12)
Note that conjugate movements can be fast (saccades) or slow (smooth pursuit). Basically what the brain is trying to do is to keep the image in the fovea to achieve maximum acuity. How does this occur? One thing you must understand is that we are talking about one eye only, but if we start talking about both eyes: then when one eye abducts, the other adducts. You should also understand that you can have conjugate movements along the horizontal and vertical axis. (i.e.: dogging suddenly starts running / someone falling from the sky). These are called lateral gaze, vertical gaze.

How does lateral gaze work? (You need abduction of one eye and adduction of another eye, which are made possible by lateral and medial recti → which are supplied by two different cranial nerves namely: CN VI & CN III).

- When an object moves, if you want to follow it along the horizontal plane, then the muscles involved must be stimulated
- This stimulation is provided by a region referred to as: paramedian pontine reticular formation (PPRF)
- Fibres from here directly stimulate the abducens nucleus (lateral rectus) situated on the same side as PPRF, fibres also project to the contralateral oculomotor nucleus (medial rectus) via the medial longitudinal fasciculus (MLF)

How does vertical gaze work?

- The neural machinery required for rapid vertical movements is situated in the rostral interstitial nucleus of the MLF
- The two nerves that control elevation and depression are: CN III (IO, SR, IR), CN IV (SO)
- Thus the riMLF coordinates the CN III + CN IV nucleus.

Vergence eye movements (Nolte 5th Ed pp 521)

Vergence eye movements are extremely important for focussing on near and far objects. Near objects require convergence of the eyes, whereas far objects require divergence of the eyes. Accommodation causes pupillary constriction, lens thickening due to shortening of the suspensory ligaments. Thus, the firing of the oculomotor nerve (parasympathetics and somatic components) causes the medial recti to contract, pupils to constrict and ciliary bodies to contract → results in shortening of the suspensory ligaments. It is thought that the pathways involve the visual association cortex and the pretectal area.

Internuclear ophthalmoplegia (Notes)

This is when the medial longitudinal fasciculus is damaged. Suppose the left MLF is damaged. This means, the right abducens nucleus is able to be stimulated by the PPRF therefore contraction of the lateral rectus muscle is possible in the right eye. But oculomotor nucleus is not able to be stimulated; therefore medial rectus muscle of left eye is not contracting.

Thus damage to MLF allows contraction of lateral rectus muscle on the on the contralateral side (allows lateral gaze to contralateral side) but prevents medial rectus from contracting in the contralateral eye (does not allow lateral gaze on ipsilateral side). *This is just worded a little differently but still proves correct.* This does not mean the medial rectus muscle is paralysed! Remember the oculomotor nerve is still intact so accommodation can still occur, therefore activating the medial rectus. Only the **MLF** is damaged.

Vestibuloocular reflex (Nolte 5th Ed pp 518, Fig 14-30 pp 365)

The vestibuloocular reflex is responsible for focusing the object onto the retina even though the head is moving in different directions. This means, the object falls on the same point of the retina (i.e.: ideally the fovea). This involves the vestibular system, along with the visual system. Unlike saccade movements, these slow movements can have continuous feedback to regulate the movements accordingly. What happens in this reflex?

- Receptors of the vestibular system pick up signals of head movements. Afferent signals are transmitted to their cell bodies located in the vestibular ganglion.
- Post synaptic fibres travel to the vestibular nuclei where they synapse with interneurons.
- Interneurons (excitatory / inhibitory) then project to the various cranial nerve nuclei bilaterally (i.e.: CN III, IV, VI) and synapse with LMN's
- LMNs innervate their respective muscles.

Now what happens when you rotate your head to the left. Notice that your right eye abducts, whilst your left eye adducts. This is completely different to the pathways involved in lateral gazes. So, what happens is that your vestibular receptor cells will pick this up → then send signals to the vestibular ganglion → vestibular nuclei. From here, excitatory interneurons will project to the contralateral abducens nucleus which contracts your contralateral lateral rectus. From here, signals are sent to the contralateral (relative to abducens nucleus now) oculomotor nucleus via the MLF pathway, and the medial rectus muscle contracts. Meanwhile, inhibitory neurons inhibit the antagonistic muscles.

Nystagmus (Nolte 5th Ed pp 366 – 368 Fig 14-31)

In a normal vestibuloocular reflex, you have head movements and eyeball stays constant (contraction of appropriate extraocular muscle). In nystagmus, you have an interruption of the vestibuloocular reflex – so there is a slow phase – where eye ball fixates on an object while head moves, and a fast phase – where the eye ball quickly saccades in the opposite direction as if to start all over again. There are different types of nystagmus: rotational nystagmus

(caused by head rotations), caloric nystagmus (caused by endolymphatic convection currents), optokinetic nystagmus (induced by moving visual stimuli). These are all physiological nystagmus.

Saccades – scanning (Nolte 5th Ed pp 517, Fig 21-12)
Saccades have already been described previously under the heading conjugated eye movements. The term saccades refers to the rapid eye movements that occur when one refocuses from one point to another point in space. Unlike lateral gaze – this is much more rapid but uses a similar principle. A number of areas are involved including: PPRF (horizontal saccades), riMLF (vertical saccades).

The frontal eye field is important in initiating saccades. Stimulation here will send signals to the contralateral PPRF, which sends ipsilateral signals to the abducens nucleus and contralateral signals to oculomotor nucleus. This causes the saccade (the same as a lateral gaze).

Smooth pursuit eye movements – tracking (Nolte 5th Ed pp 521, Fig 21-15)
Smooth pursuit refers to when you follow an object through the visual field. Basically, what we are trying to do is the keep the moving object in the fovea. How does this occur? There are a lot of areas involved. Intentions for smooth pursuit movements originate in the extra-striate cortex, which in turn projects to the brain stem directly or via the frontal eye field. The pathway to the brain stem is: pontine nuclei, flocculus of cerebellum, juxtarestiform body, vestibular nuclei (ipsilateral), PPRF, MLF → oculomotor nucleus.