Preface

Let me start with a joke from my medical school days. We had a tough professor of anatomy who once asked a student: "What is the normal weight of a salivary gland?" The student had a sense of humor and instantly retorted: "Sir, with the capsule or without?" That well illustrates the dilemma of anatomy most of us have to face in our medical school days! But my personal opinion is that a sound knowledge of correlative anatomy of an organ can be an asset for a busy clinician who can thus easily anticipate its clinical presentation in disease. This atlas is a humble effort to present such knowledge for the eye.

We start with some solid examples. We can easily divide the eye anatomy into some regions, the starting point being the bony socket, the orbits on either sides of the nose, in which the eyes are safely lodged. Pyramidal in shape, the orbits have to have an apex on the back and an open base in the front. They are a bony socket closed by the orbital septum. They have a limited physical space suited for their normal contents—the eyeball along with its nerves and vessels, which come from middle cranial fossa through its apertures called superior orbital fissure and optic foramen.

So any growing lesion inside the orbit will displace its normal contents in a direction opposite to the growth. This may result in an oblique or forward proptosis of the eye, creating a loss of parallelism of the visual axis. It may also cause double vision (diplopia) (a good example is mucocele of the frontal or ethmoid sinus). A very severe proptosis, such as occurs in thyroid orbitopathy, may cause exposure keratitis because of incomplete closure of lids. A glioma of the optic nerve will cause a forward proptosis of the eyes. The structures inside the orbit are prone to trauma (contusion), which can cause fractures of weak spots in the orbit. Since the medial wall of the orbit is the thinnest, it can be fractured in severe contusions, resulting in air entry into the orbit and the periorbital tissues called crepitus; this, in turn, may cause bleeding from the nose if the patient blows his or her nose. Another weak spot in the orbit is that part of orbital floor near the infraorbital groove that changes into the infraorbital canal lodging the infraorbital vessels and nerves. In very severe contusions of the orbital margin, a sudden gross rise of intraorbital pressure can cause fracture of the floor (called a blow-out fracture) with herniation (displacement) of the neighboring inferior oblique or inferior rectus muscle into the underlying maxillary sinus. Because of the involvement of the infraorbital vessels and nerves, this may result in

anesthesia of the upper lip. Thus a knowledge of anatomy of the orbit is sure to make things easier. Severe unilateral headache may be brought about by frontal sinusitis because this sinus exists in the superomedial angle of the roof of the orbit and may even trigger migraines in vulnerable female patients. Similarly, a complicated case of ethmoiditis in children may cause orbital cellulitis because a papery thin bone separates the eye from the ethmoid sinus. A painful red swelling just below the inner canthus is likely to be brought about by acute dacryocystitis because the lacrimal sac is situated there. Similarly, because the palpebral part of the main lacrimal gland is situated in the lateral part of the roof of the orbit, its inflammation, called acute dacryoadenitis, may present as a painful red swelling in the lateral part of the upper lid. We know that the sclera is very thin at the insertions of the rectus muscles on its front and therefore is a common site of rupture of the globe in severe injuries. The retina is thinnest at the ora serrate and the fovea; therefore retinal holes occur there when a traumatic detachment of the retina takes place. The visual pathway starts from the orbital, canalicular, and intracranial parts of the optic nerve, the optic chiasma, and the optic tracts, which end in the nucleus of the lateral geniculate body; there a synapse occurs and a new neuron of the nucleus of the lateral geniculate body takes over. The axons of these new neurons are further continued as the optic radiation, which finally ends in the visual center in the occipital lobe. If the arrangement of nerve fibers in various part of the visual pathway is known, the type of field of vision defects can be easily anticipated. These typical defects can help in localizing the site of a lesion in the visual pathway.

An ischemic microvascular lesion of the capillaries supplying a cranial nerve (as occurs in longstanding hypertension or uncontrolled diabetes) may cause an isolated palsy of the third, fourth, or sixth cranial nerves. The blood supply of the canalicular and cranial parts of the optic nerve is more vulnerable to trauma (especially the macular fibers) because the capillary meshes are wider and fewer vessels supply more nerve fibres via the septa of the pial network. The chiasma, in its position mostly above the pituitary fossa, is likely to suffer in tumors of the pituitary gland causing bitemporal hemianopia, especially with colored objects. Because the pupillary fibers separate themselves from the distal part of the optic tract, it is easy to remember that any patient with a lesion above the level of the lateral geniculate body will have normal pupils although he or she may be totally blind (cerebral blindness).

If one keeps in view the arrangement of nerve fibers in various parts of the visual pathway, it is not difficult to anticipate the type of field defects that may occur. For example, lesions anterior to the chiasma will cause unilateral field defects, while those posterior to the chiasma will cause contralateral homonymous hemianopia because the nasal fibers in the chiasma cross to its opposite side before entering the optic tract. A lesion in the occipital region tends to cause identical defects in each field, whereas optic tract lesions tend to cause dissimilar homonymous field defects. Because of its dual vascular supply, lesions of the occipital cortex may not affect macular fibers (macular sparing).

Optic nerve swelling (papilloedema) occurs mostly in lesions of the proximal part of the optic nerve but can also be seen in cases of raised

intracranial pressure and compression of the orbital part of the optic nerve. This is because its cranial subarachnoid space freely communicates with the subarachnoid spaces of the orbital and canalicular parts. The most common causes are cerebral tumors, abscesses, subdural hematomas, subarachnoid hemorrhage, meningitis, and encephalitis.

Optic Neuritis

The most common cause of optic neuritis is demyelinating disease; it may be retrobulbar with a normal disc, but a painful sudden unilateral loss of vision with lowering of color vision and contrast sensitivity may occur, mostly in females in the fourth decade. An afferent pupillary defect is always present, and movements of eye maybe painful because of a blending of the sheath of the optic nerve with the origin of some rectus muscles.

Optic Nerve Compression

In optic neuropathy, which is not explained by intraocular lesions, compression of the optic nerve should be suspected. Early imaging of the orbit by MRI or CT scan is a great help. Cerebrovascular disease and tumors are responsible for most optic radiation lesions, although any intraocular disease can be involved.

Cranial Palsies

Ischemia (as in diabetes or hypertension), intracranial aneurysm, head injury, and intracranial tumors are causes of third cranial nerve palsy, which produces ipsilateral dysfunction. Aneurysms are more common at the junction of the internal carotid and posterior communicating arteries. Pupillary signs are common in these compression cases because pupillary fibers are superficial, whereas in ischemic lesions the pupils are normal.

Palsy of the sixth cranial nerve does not have much localizing value because it has a 90° bend at the apex of the petrous part of the temporal bone. Trochlear nerve palsies may be congenital or acquired.

Shape of the Eyeball

For proper refraction to occur at the cornea, it is essential that the walls of the eyeball are tight enough so that atmospheric pressure cannot indent them. This is achieved by circulation of a fluid inside the eye (the aqueous humor) that is chiefly secreted in the posterior chamber, from where it travels to the anterior chamber via the pupil. Then it goes to the periphery of the anterior chamber, called the angle of the anterior chamber; this structure has microscopic outlet channels of the aqueous humor (i.e., the trabecular meshwork,

the canal of Schlemm, and the aqueous veins and collecting trunks). These drain the aqueous to the episcleral veinous plexus. This circulation of the aqueous maintains a positive pressure inside the eye called the intraocular pressure, which is responsible for maintenance of the shape of the eyeball and is essential for refraction at the cornea. Because of this intraocular pressure, atmospheric pressure cannot indent the eyeball. For proper maintenance of intraocular pressure within normal limits (12–20 mm Hg), the anterior chamber should have an optimum width so that the aqueous has adequate access to the microscopic outlet channels situated there. A narrow angle and a shallow anterior chamber can cause a sudden gross rise of intraocular pressure up to 60 mm Hg, creating a sudden stretching of the corneal nerves and severe pain and vomiting, a condition called acute congestive glaucoma, which is a serious ophthalmic emergency. Therefore before dilating the pupils for examination of the fundus, one must make sure that the anterior chamber is not shallow and that its angle is not too narrow. This can be clinically tested by the Iris Shadow Test.

No book can claim to be perfect including this one but since our medical school days we have wanted to see an anatomy book that could be simple, self-explanatory, and at the same time enjoyable. This demanded a correlation of structure with function. This also demanded highly schematic diagrams instead of photographs of dissected specimens that could aid the easy grasp of the subject. This atlas is our humble effort to achieve this.

In the end, I must thank the team of Springer editors whose cooperation was an asset for me in preparation of this atlas. Special thanks are due to Dr. Asefa Ansari and Dr. Farozan Islam who helped me whenever I got stuck up with my computer.

Suggestions and comments are welcome.

Illinois, USA Mohammad Wakeel Ansari
Illinois, USA Ahmed Nadeem

http://www.springer.com/978-3-319-42780-5

Atlas of Ocular Anatomy

Ansari, M.W.; Nadeem, A.

2016, XI, 106 p. 89 illus., 88 illus. in color., Hardcover
ISBN: 978-3-319-42780-5