High resolution magnetic resonance imaging anatomy of the orbit

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Addendum

ANATOMY OF THE ORBITAL APEX AND CAVERNOUS SINUS ON HIGH RESOLUTION MAGNETIC RESONANCE IMAGES

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1. INTRODUCTION

The orbital and retroorbital regions are involved in various injuries and diseases: The intracanicular or intracranial optic nerve may be damaged by sphenoid fractures. Visual and neurological deficits may arise from inflammatory processes (e.g. infectious lesions, orbital pseudotumor, Tolosa-Hunt syndrome), vascular lesions (e.g. aneurysms, carotid-cavernous shunts, thrombosis of orbital veins or cavernous sinus) and neoplastic lesions (e.g. pituitary adenoma, meningioma, craniopharyngioma etc.). Lesions at the cranio-orbital junction present with different neuroophthalmic symptoms and signs, depending upon their size and location.

Due to the crowding of critical anatomical structures at the orbital apex, patients often suffer from dysfunction of more than one cranial nerve. Previously, various syndromes such as the „orbital apex syndrome“ (involvement of cranial nerves II, III, IV, V, VI), the „superior orbital fissure syndrome“ (nerves III, IV, VI) and the „cavernous sinus syndrome“ (nerves III, IV, VI, V2, VI and periarterial sympathetic plexus) have been described. In the past, physicians had to rely solely on clinical differences in order to estimate size and location of a lesion. Modern imaging techniques have proven these subtle differences in presentation to be unreliable indicators of the location and size of lesions. Therefore, Miller more practically described the clinical picture of mass lesions at the cranio-orbital junction, as „sphenocavernous syndrome“.

Imaging of the cranio-orbital junction necessitates sophisticated techniques because of its anatomical complexity. Additionally, good contrast resolution is required because lesions, such as cranial nerve tumors, have only little contrast to surrounding tissues.

Standardized echography and color Doppler ultrasonography are useful diagnostic techniques for the anterior and middle thirds of the orbit, but lead to image distortion and loss of resolution when applied to the orbital apex.

Computed tomography (CT) not only depicts orbital soft tissue details but also the complex bony anatomy of the orbital apex but CT scans of the orbital apex are distorted by beam-hardening and dental filling artifacts. CT cannot identify individual cranial nerves within the superior orbital fissure although contrast enhanced CT may demonstrate the cranial nerves III, V, and V2 within the cavernous sinus. In contrast to magnetic resonance imaging (MRI), plain CT scans do not allow a differentiation of the intracavernous carotid artery from the surrounding sinus tissue. The enthusiasm for high resolution MR imaging is based on the progress that has been demonstrated in both structural and vascular imaging. Precise maps of anatomic information allow comprehensive evaluation at high spatial, temporal and contrast resolution. MRI is now the diagnostic modality of choice for imaging the orbital apex and retroorbital region. Many of the important anatomic details of the anterior and posterior orbit are visualized by MRI. The interpretation of clinical MR images of the cranio-orbital junction requires a profound knowledge of anatomy, especially sectional anatomy. A schematic overview of the anatomic structures is provided in Fig. 1-3. Detailed knowledge of the anatomic relationships in the orbital apex region...
is not only crucial for diagnostic purposes, but also for successful surgical intervention in a region where critical structures are only a few millimeters apart. The purpose of this paper is to update the physician on current possibilities of imaging deep structures of the orbit and retroorbital region, focusing on standardized and widely utilized MR-techniques.

We describe the normal anatomy of the cranio-orbital junction on coronal (Fig. 4-12), axial (Fig. 13-23) and oblique-sagittal (Fig. 24-29) magnetic resonance images and relate our data to a review of the current literature.

2. EXAMINATION PROTOCOLS

MRI examinations were performed in 8 normal volunteers on a 1.5 T MR unit (Gyrosan ACS-NT, Philips Medical Systems, Best, The Netherlands). A circular polarized head coil was used for the cavernous sinus and brain stem, and a surface coil with a diameter of 17 cm was used for the orbital apex. 1.2 to 3 mm thick scans with -0.6 to 0.3 mm interslice gap were obtained in axial, oblique-axial (along the neuro-opthalmic plane), coronal, oblique-coronal (along the long axis of the petrous portion of the temporal bone) and oblique-sagittal (along cranial nerves III, V, VI) planes using T1-weighted (T1w) [TR=550-620 ms, TE=14-18 ms] spin-echo (SE) sequences with and without intravenous injection of Gadolinium-DTPA and T2-weighted (T2w) [TR=2800 ms, TE=120 ms] turbo-spin echo (TSE) sequences. Also, T2-weighted 3D-TSE acquisitions (TR=4000 ms, TE=250 ms, TF=48) were used for detailed demonstration of the cranial nerves in the subarachnoid cisterns. The fields of view (FOV) ranged between 120 and 140 and the images were usually obtained in a 256 x 256 matrix. This technique required a total examination time of 2.5-9 minutes.

3. IMAGING

3.1. Bony anatomy

Cortical bone does not produce a perceptible signal and is only indirectly seen by contrast demarcation to adjacent signal-generating tissue (e.g. brain, CSF, muscle, fat, sinus mucosa etc.). Cancellous bone is visualized indirectly by its fatty tissue content.

In the following, the orbital apex is defined as the region between the posterior ethmoidal foramen and the openings of the optic canal and the superior orbital fissure. The posterior ethmoidal foramen is visible on axial and coronal MR- scans. It transmits the corresponding neurovascular bundle at an average distance of 5 mm from the optic canal and represents an important landmark during orbital decompression surgery. The bony walls of the orbital apex can be demonstrated relatively well due to the contrast between orbital fat and tissue surrounding the orbit. The orbital roof of the apex consists of the lesser wing of the sphenoid bone (Fig. 9,10), its medial wall is the lateral wall of the ethmoidal sinus (Fig. 10-12), its lateral wall is the greater wing of the sphenoid (Fig. 10-12) and its floor is the orbital plate of the palatine bone. The lesser wings of the sphenoid bone terminate at the anterior clinoid processes (Fig. 7,8), symmetric bony spines between the optic canal and superior orbital fissure, onto which the tentorium cerebelli (Fig. 17,24) is attached.

The optic canal is 5-6 mm wide and 8-12 mm long, and forms an angle of approximately 35° with the sagittal plane. It transmits the optic nerve and the ophthalmic artery (within a dural slit inferior to the nerve) and is bounded by the sphenoid bone medially, its lesser wing superiorly, the anterior clinoid process laterally and the optic strut inferiorly (Fig. 8,9,15,16). The bony medial wall of the optic canal may be absent in about 4% of the population, which should be considered during transsphenoidal optic nerve decompression procedures. With extensive pneumatization, the optic canal may become entirely surrounded by a (posterior) ethmoidal „Onodi“ air cell or the sphenoid sinus proper or an aerated anterior clinoid process. On MR this would result in signal loss around the optic nerve. The various relationships between the parasellar sinuses and the optic canal and their clinical implications have been described in a comprehensive review on optic nerve trauma. Each optic canal opens into the chiasmatic groove which terminates posteriorly at the tuberculum sellae. Further posteriorly located is the sella turcica, which contains the pituitary gland (Fig. 4-6). The dorsum
Three superolateral intermuscular spaces (Fig. 8, 11) are formed in the orbital apex, the system of connective tissue septa between thesuperior and inferior rectus muscles (Fig. 9, 10, 13, 23). It lies between the orbital floor and the lateral orbital wall and communicates with the pterygopalatine (Fig. 9-11) and infratemporal fossae. It is bridged by the orbital muscle of Müller (Fig. 10-12) and transmits the infraorbital artery, venous collaterals between the inferior ophthalmic vein and the pterygoid plexus, and the maxillary nerve which exits the middle cranial fossa via the foraamen rotundum (Fig. 8, 23).

### 3.2. Extraocular muscle and connective tissue system anatomy

The extraocular muscles demonstrate intermediate signal on both T1w and T2w MR images. The four rectus muscles originate from the common tendinous annulus of Zinn which spans across the superior orbital fissure and encloses the optic foramen (containing optic nerve and ophthalmic artery) and the oculomotor foramen (Fig. 10). Zinn's annulus consists of two half-circles. Superiorly, the tendon of Lockwood serves as origin for the superior rectus muscle (Fig. 10), and inferiorly the tendon of Zinn for the medial, inferior and lateral rectus muscles (Fig. 9, 10).

The annulus divides the superior orbital fissure into three spaces, namely, the superolateral, central ("oculomotor foramen") and inferior space. The superolateral portion of the superior orbital fissure contains the frontal, lacrimal and trochlear nerves; the central portion contains the nasociliary nerve, superior and inferior branch of the oculomotor nerve, abducens nerve and the superior ophthalmic vein; the inferior portion of the superior orbital fissure contains the inferior ophthalmic vein (Fig. 1).

For the first 5 mm of their length, the rectus muscles do not appear as individual structures but are firmly embedded within Zinn's annulus. This anatomic relationship explains that enlargement of the rectus muscle origins associated with thyroid orbitopathy, may cause compression of the optic nerve. Histologically, the origins of the rectus muscles are separated from each other by thin connective tissue septa. On MRI, the entire inferior annulus appears as a single unit of muscle masses (Fig. 10).

Approximately 8 mm anterior to the optic strut, the rectus muscles separate and appear as individual structures (Fig. 11, 12). The superior oblique muscle originates superiorly and medially to Zinn's annulus from the lesser sphenoid wing with a short tendon (Fig. 11, 12).

The levator palpebrae superioris muscle originates from the lesser sphenoid wing and the annulus of Zinn, where it blends with the origin of the superior rectus muscle (Fig. 10-12). In the orbital apex, the system of connective tissue septa is less well developed. The superolateral intermuscular septum between the superior and the lateral rectus muscles starts at a distance of about 5 mm from the posterior end of the orbit and thin radial septa course from the rectus muscles to the orbital walls.
Fig. 4. T1-weighted coronal enhanced MR scan of the posterior cavernous sinus at the level of Meckel’s cave. ACA = anterior cerebral artery, CC = chiasmatic cistern, DS = diaphragm sellae, H = hypophysis, ICA = internal carotid artery, III = infundibulum of hypophysis, III = oculomotor nerve, IV = trochlear nerve, MCA = middle cerebral artery, OCH = optic chiasm, SS = sphenoidal sinus, TG = trigeminal ganglion, TLB = temporal lobe of brain, VI = abducens nerve.

Fig. 5. T1-weighted enhanced coronal MR scan of the posterior cavernous sinus at the level of the foramen ovale. DS = diaphragm sellae, H = hypophysis, ICA = internal carotid artery, III = infundibulum of hypophysis, III = oculomotor nerve, OCH = optic chiasm, SS = sphenoidal sinus, TLB = temporal lobe of brain, IV = trochlear nerve, VI = abducens nerve.

Fig. 6. T1-weighted coronal contrast-enhanced MR scan of the anterior cavernous sinus. ACA = anterior cerebral artery, H = hypophysis, ICA = internal carotid artery, II = optic nerve, III = oculomotor nerve, IV = trochlear nerve, SS = sphenoidal sinus, TLB = temporal lobe of brain, V1 = ophthalmic nerve, V2 = maxillary nerve, VI = abducens nerve.

Fig. 7. T1-weighted coronal MR scan of the anterior end of the cavernous sinus (CS) at the level of the foramen rotundum (FR). Cranial nerves III, IV, VI are not visible on this non-enhanced image. ACP = anterior clinoid process, FLB = frontal lobe of brain, ICA = internal carotid artery, II = optic nerve, LPP = lateral plate of pterygoid process, MPP = medial plate of pterygoid process, NPC = nerve of pterygoid canal (Vidian’s nerve), SS = sphenoidal sinus, TLB = temporal lobe of brain, V2 = maxillary nerve.
Fig. 8. T1-weighted coronal MR scan of the orbital apex at the level of the cranial openings of the superior orbital fissures (SOF). Cranial nerves VI (abducens nerve) and V.1 (ophthalmic nerve) cannot be separated from each other. ACP = anterior clinoid process, FLB = frontal lobe of brain, II = optic nerve, III = oculomotor nerve, IV = presumed trochlear nerve, OA = ophthalmic artery, SOF = superior orbital fissure, SS = sphenoidal sinus, TLB = temporal lobe of brain, V.1 = presumed ophthalmic nerve, V.2 = maxillary nerve, VI = abducens nerve.

Fig. 9. T1-weighted coronal MR scan of the orbital apex at the orbital opening of the superior orbital fissure. The superior orbital fissure is continuous with the inferior orbital fissure (IOF). The cranial nerves (CN) are not visualized as individual structures. ES = ethmoidal sinus, GWS = greater wing of sphenoid bone, IOF = inferior orbital fissure, LWS = lesser wing of sphenoid bone, MA = maxillary artery, MOM = Müller's orbital muscle, OA = ophthalmic artery, PPF = pterygopalatine fossa, PPG = pterygopalatine ganglion, of brain, TZ = tendon of Zinn (origin of medial, inferior and lateral rectus muscles). For explanation of other abbreviations, see Fig. 8.

Fig. 10. T1-weighted coronal MR scan of the orbital apex at the level of Zinn's tendinous annulus. ES = ethmoidal sinus, FLB = frontal lobe of brain, GWS = greater wing of sphenoid bone, II = optic nerve, III.s = presumed superior division of oculomotor nerve, IV, FN, LN = presumed trochlear, frontal and lacrimal nerves, LRM = lateral rectus muscle, LWS = lesser wing of sphenoid bone, MOM (IOF) = Müller's orbital muscle (inferior orbital fissure), MS = maxillary sinus, NCN = nasociliary nerve, OA = main trunk of ophthalmic artery (inferior to optic nerve), PPF = pterygopalatine fossa, SOF = superior orbital fissure, SOV = presumed superior ophthalmic vein, TL = tendon of Lockwood (origin of superior rectus muscle), TLB = temporal lobe of brain, TZ (IRM) = tendon of Zinn (origin of inferior rectus muscle), TZ (LRM) = tendon of Zinn (origin of lateral rectus muscle), V.2 = maxillary nerve, VI = abducens nerve.

Fig. 11. T1-weighted coronal MR scan of the orbital apex (scan level between Fig. 10 and 12). The cranial nerves III and VI appear in close contact to the corresponding extraocular muscles. The trochlear nerve lies on top of the superior rectus muscle from which it cannot be differentiated. CRA = central retinal artery, FN, LN = presumed frontal and lacrimal nerves, III.i = inferior division of oculomotor nerve, III.s = superior division of oculomotor nerve, IOV = inferior ophthalmic vein, IRM = inferior rectus muscle, MRM = medial rectus muscle, OA = bend of ophthalmic artery fossa, SRM = superior rectus muscle. For explanation of other abbreviations, see Fig. 10.
Fig. 12. T1-weighted coronal MR scan of the orbital apex just posterior to the posterior ethmoidal foramen. The cranial nerves III, IV and VI appear in close contact with the corresponding extraocular muscles. CG = presumed ciliary ganglion, CRA = central retinal artery, ES = ethmoidal sinus, FLB = frontal lobe of brain, FN = frontal nerve, GWS = greater wing of sphenoid bone, II = optic nerve, III.s = inferior division of oculomotor nerve, III.i = presumed superior division of oculomotor nerve, ION = infraorbital nerve, IOV = inferior ophthalmic vein, IRM = inferior rectus muscle, IV = presumed trochlear nerve, LN = presumed lacrimal nerve, LPS = levator palpebrae superioris muscle, LRM = lateral rectus muscle, MOM = Müller’s orbital muscle, MRM = medial rectus muscle, MS = maxillary sinus, NCN = nasociliary nerve, OA = ophthalmic artery, OPF = orbital plate of frontal bone, SOM = superior oblique muscle, SOV = superior ophthalmic vein, SRM = superior rectus muscle.

Fig. 13. T1-weighted axial MR scan at the level of the superior orbital fissure (SOF) showing parts of Müller’s orbital muscle (MOM). The bony borders of the SOF are indicated by white dots. GWS = greater wing of sphenoid bone, IOM = inferior oblique muscle, IOV = inferior ophthalmic vein, IRM = inferior rectus muscle, MS = maxillary sinus, SS = sphenoidal sinus.

Fig. 14. T1-weighted axial MR scan at the level of the superior orbital fissure (SOF). The bony borders of the SOF are indicated by white dots. ES = ethmoidal sinus, G = globe, GWS = greater wing of sphenoid bone, ICA = internal carotid artery, LRM = lateral rectus muscle, MRM = medial rectus muscle, OA = ophthalmic artery, SS = sphenoidal sinus.

Fig. 15. T1-weighted axial MR scan in the neuroophthalmic plane at the level of the optic canal (OCA) showing the intraorbital and intracanicular portion of the optic nerve (II). The bony borders of the OCA and SOF are indicated by white dots. ACP = anterior clinoid process, ES = ethmoidal sinus, G = globe, GWS = greater wing of sphenoid bone, LPCA = lateral posterior ciliary artery, LRM = lateral rectus muscle, MPCA = medial posterior ciliary artery, MRM = medial rectus muscle, OA = ophthalmic artery (before crossing over the optic nerve), OCA. II = optic canal, SOF = superior orbital fissure, SS = sphenoidal sinus.
3.3. Cranial nerve anatomy

3.3.1. Optic nerve and chiasm

The entire optic nerve (II) can be visualized on appropriate MRI scans. The optic nerve measures 45-50 mm in length and 3-5 mm in diameter including its the nerve sheath. It consists of a 3-16 mm long intracranial portion, a 6-10 mm long intracanicular portion, a 21-34 mm long intraorbital portion and an about 1 mm long intraocular portion i.e. the papilla. The optic nerve consists of myelinated nerve fibers and exhibits MR-signal characteristics similar to those of white matter of the brain. The intracanicular and intraorbital portions of the optic nerve are surrounded by pia, arachnoidea and dura. At the orbital opening of the optic canal, the dura of the intracanicular optic nerve splits into peri-orbita and periorbita dura. At the intracanal opening of the canal, it is continuous with the intracanal dura. The subarachnoid space of the intracoatal optic nerve appears hypointense on T1w and hyperintense on T2w (Fig. 21). The intracanal optic nerve is covered only by pia and is surrounded by the cerebrospinal fluid of the suprasellar cistern (Fig. 6-8).

In the primary gaze position, the intraorbital portion of the optic nerve describes an S-shaped path from the globe inferio-medially and then superiorly, to the optic foramen. The redundant length of the optic allows the globe to move freely and protects the nerve in case of proptosis. The intracanicular portion passes above the ophthalmic artery through the optic canal (Fig. 8,9,15,16). On axial scans, the intracanicular optic nerve can be identified medial to the anterior clinoid process (signal characteristics: see above) and lateral to the sphenoid sinus (signal void of air) (Fig. 15,16). Coronal scans show the optic nerve in the chiasmatic cistern (Fig. 6), in its canal (Fig. 8,9), in the orbital apex inside the annulus tendineus (Fig. 10), and in the orbit surrounded by the rectus muscles (Fig. 11,12).

The optic chiasm lies within the floor of the third ventricle and superiorly to the diaphragm sellae (Fig. 4,5,16). Here, nasal retinal ganglion cell axons cross from the optic nerve to the contralateral optic tract. The chiasm normally measures 10-20 mm in transverse,4-13 mm in antero-posterior, and 3-5mm in crano-caudal diameter.

3.3.2. Motor nerves

The motor nerves (with the exception of the inferior division branches of the oculomotor nerve to the inferior oblique muscle and the trochlear nerve) ramify far posteriorly in the orbital apex into numerous fascicles which travel anteriorly embedded between muscle fibers to innervate the extracocular muscles in their posterior third and from their intracanal surface. The tiny ramifications and the fascicles of the motor nerves cannot be visualized on MRI. Only the nerve trunks in the orbital apex and also the oculomotor nerve branch to the inferior oblique muscle is visualized on high-resolution MRI.

The small parasympathetic twig of the inferior division of the oculomotor nerve (usually the branch to the inferior oblique muscle) that joins the ciliary ganglion, cannot be seen on MRI. The ciliary ganglion that transmits afferent sensory fibers and efferent parasympathetic fibers for pupillary constriction and accommodation, is situated in the orbital apex very close to the lateral aspect of the optic nerve. It may be seen on high-resolution MRI just anterior to the knee of the ophthalmic artery between the optic nerve and the lateral rectus muscle (Fig. 12).

3.3.2.1. Oculomotor nerve

The neurons of the third cranial nerve (III\(^3\) nerve) arise in the oculomotor nuclei which lie in the ventral periaqueductal grey matter of the midbrain. The III\(^3\) nerve exits the brain medially to the cerebral peduncles (Fig. 18) and passes between the superior cerebellar and posterior cerebral arteries (Fig. 29) through the interpeduncular cistern (Fig. 17,27) where it lies adjacent to the posterior communicating artery (Fig. 19). Then, the III\(^3\) nerve pierces the dura at the top of the clivus (Fig. 27). Embedded within the dural border of the cavernous sinus, the III\(^3\) nerve courses forwards just superior to the trochlear nerve (Fig. 4-6). Just posterior to the orbital opening of the superior orbital fissure, the III\(^3\) nerve crosses under the trochlear nerve and divides into a superior and inferior division which both pass the central portion (oculomotor foramen) of the superior orbital fissure to enter the orbit (Fig. 9). The superior division of the III\(^3\) nerve courses superiorly to innervate the superior rectus and the levator palpebrae muscles (Fig. 10-12). The inferior division of the III\(^3\) nerve courses medially and inferiorly to innervate the medial and inferior rectus muscles (Fig. 10-12). A long nerve branch courses anteriorly to the inferior oblique muscle.

3.3.2.2. Trochlear nerve

The neurons of the fourth cranial nerve (IV\(^3\) nerve) originate from the trochlear nucleus in the ventral periaqueductal grey matter of the mid brain (rostral to the oculomotor nuclei), decussate and emerge on the dorsal surface of the mid-brain, below the level of the inferior colliculus. Running along the free border of the tentorium cerebelli, the IV\(^3\) nerve passes between the superior cerebellar and posterior cerebral arteries around the cerebral peduncles (Fig. 20) to enter the lateral border of the cavernous sinus. Due to its long intracranial course, the IV\(^3\) nerve is predisposed to injury from blunt head trauma. Inside the lateral dural border of the cavernous sinus, the IV\(^3\) nerve courses forwards, first inferior to the III\(^3\) nerve (Fig. 4) and finally superior to the III\(^3\) nerve. Then, the IV\(^3\) nerve exits the cavernous sinus and traverses the superolateral portion of the superior orbital fissure. In the orbital apex, the IV\(^3\) nerve crosses over the origin of the superior rectus and levator palpebrae muscles (Fig. 12) to innervate the superior oblique muscle from its lateral surface about 10 mm from the orbital apex.
Fig. 16. T2-weighted oblique-axial MR scan at the level of the optic chiasm (OCH). ACP = anterior clinoid process, CC = chiasmatic cistern, GWS = greater wing of sphenoid bone, ICA = internal carotid artery, II = optic nerve, LRM = lateral rectus muscle, MCA = middle cerebral artery, MRM = medial rectus muscle, OCA = optic canal, OT = optic tract, RBF = retrobulbar fat.

Fig. 17. T2-weighted axial MR scan at the level of the midbrain (MB) showing the oculomotor nerve (III) in the interpeduncular cistern. MEA = mesencephalic aqueduct, PCA = posterior cerebral artery, PCP = posterior clinoid process, SOF = superior orbital fissure, TC = tentorium cerebelli.

Fig. 18. T2-weighted axial MR scan at the level of the midbrain (MB) showing the origin of the oculomotor nerve. III = oculomotor nerve with partial volume effect (see paragraph 4.5 of text) BA = basilar artery, CLT = cisterna laminae tecti, COS = colliculus superior laminae tecti, CP = cerebral peduncles, CSF = cerebrospinal fluid, ICA = internal carotid artery, II = optic nerve, IPC = interpeduncular cistern, MEA = mesencephalic aqueduct, PCP = posterior clinoid process, PD = perioptic dura, SCA superior cerebellar artery.

Fig. 19. T2-weighted axial MR scan showing the origin of the ophthalmic artery (OA) from the internal carotid artery (ICA). IRM = inferior rectus muscle, LRM = lateral rectus muscle, MRM = medial rectus muscle, PCOA = posterior communicating artery.
Fig. 20. T2-weighted oblique-axial MR scan at the level of the dorsal caudal midbrain (MB) and ventral rostral pons showing the presumed trochlear nerve (IV). BA = basilar artery, MEA = mesencephalic aqueduct, SCA = superior cerebellar artery.

Fig. 21. T2-weighted axial MR scan at the level of the pons (P) showing the trigeminal nerve (V) in the prepontine cistern. V = trigeminal nerve with partial volume effect (see paragraph 4.5 of text), BA = basilar artery, FV = fourth ventricle, TG = trigeminal ganglion inside Meckel’s cave.

Fig. 22. T2-weighted oblique-axial MR scan at the level of the caudal pons (P) showing the abducens nerve (VI) in the subarachnoid space. AICA = anterior inferior cerebellar artery, BA = basilar artery, C = clivus, FV = fourth ventricle.

Fig. 23. T1-weighted oblique-axial MR scan at the level of the inferior orbital fissure (IOF) and foramen rotundum (FR). C = clivus, GWS = greater wing of sphenoid bone, ICA = internal carotid artery, MS = maxillary sinus, P = pons, SS = sphenoidal sinus, V.2, FR = maxillary nerve inside foramen rotundum.
Fig. 24. T2-weighted oblique-sagittal, reconstructed MR image in a plane along the trigeminal nerve (V). BA = basilar artery, C = clivus, P = pons, RBF = retrobulbar fat, TC = tentorium cerebelli, TLB = temporal lobe of brain.

Fig. 25. T2-weighted oblique-sagittal reconstructed MR image in a plane along the origin of the abducens nerve (VI) from the medulloponine sulcus. BA = basilar artery, COI = colliculus inferior laminae tecti, COS = colliculus superior laminae tecti, FV = fourth ventricle, MB = midbrain, MO = medulla oblongata, P = pons, TV = third ventricle.

Fig. 26. T2-weighted oblique-sagittal reconstructed MR image in a plane along the abducens nerve (VI) inside the prepontine cistern and underneath the petroclinoidal ligament (PCL). ACA = anterior cerebral artery, BA = basilar artery, COI = colliculus inferior laminae tecti, COS = colliculus superior laminae tecti, FV = fourth ventricle, III = oculomotor nerve, MB = midbrain, MCA = middle cerebral artery, MO = medulla oblongata, OT = optic tract, P = pons, PCA = posterior cerebral artery, SCA = superior cerebellar artery.

Fig. 27. T2-weighted oblique-sagittal reconstructed MR image in a plane along the intracisternal portion of the oculomotor nerve (III). BA = basilar artery, COI = colliculus inferior laminae tecti, COS = colliculus superior laminae tecti, FLB = frontal lobe of brain, FV = fourth ventricle, G = globe, IPC = interpeduncular cistern, MB = midbrain, MCA = middle cerebral artery, MO = medulla oblongata, MS = maxillary sinus, OT = optic tract, P = pons, PCA = posterior cerebral artery, RBF = retrobulbar fat, SCA = superior cerebellar artery.
3.3.2.3 Abducens nerve

The neurons of the VIth nerve arise in the abducens nucleus of the pons just inferior to the rostral part of the floor of the fourth ventricle. The nerve emerges in the ventral sulcus between pons and medulla oblongata (Fig. 25). It ascends along the ventral surface of the pons, pierces the dura, and passes over the petrous apex through Dorello's canal, an osteofibrous canal underneath Gruber's petroclinoidal ligament (Fig. 26,28). Then, the VIth nerve enters the cavernous sinus and proceeds inside this venous plexus, first laterally and then inferolaterally to the internal carotid artery (Fig. 4-6). The VIth nerve enters the orbit via the central portion of the superior orbital fissure (Fig. 8,9) to innervate the lateral rectus muscle from its intracranial surface (Fig. 10-12).

3.3.3 Sensory nerves

The sensory neurons of the trigeminal nerve (Vth) terminate in the main sensory nucleus in the pons and the spinal tract of the Vth nerve. The sensory root (together with the motor root) emerges from the pons just above the middle cerebellar peduncle (Fig. 21,24). The neurons synapse in the trigeminal ganglion Gasser which is situated in Meckel's cave, a dural split over the apex of the petrous portion of the temporal bone (Fig. 4,21). Three main divisions of the Vth nerve arise from the trigeminal ganglion: the ophthalmic (V.1), maxillary (V.2) and mandibular (V.3) division.

3.3.3.1 Ophthalmic nerve (V.1)

The ophthalmic nerve courses forwards inside the lateral dural border of the cavernous sinus just inferior to the IVth nerve (Fig. 6,8). In the anterior cavernous sinus, the ophthalmic nerve divides into the lacrimal, frontal and nasociliary nerves which exit the cavernous sinus as individual nerve branches.

The small lacrimal nerve enters the orbit through the superolateral portion of the superior orbital fissure. It courses anteriorly along the superior border of the lateral rectus muscle towards the lacrimal gland. It may be visualized on MR images of the anterior orbit25,27, but cannot be differentiated from the frontal nerve in the orbital apex (Fig. 10-12).

The frontal nerve also enters the orbit via the superolateral portion of the superior orbital fissure (Fig. 9). It passes forwards between levator palpebrae superioris muscle and orbital roof (Fig. 10-12) and divides into the supratrochlear nerve, the medial branch of the supraorbital nerve and the lateral branch of the supraorbital nerve.25,27

The nasociliary nerve (Fig. 10-12) enters the orbit through the central portion of the superior orbital fissure, crosses over the optic nerve about 10 mm from the orbital apex and courses anteriorly at the lateral side of the medial rectus muscle.27
3.3.3.2. Maxillary nerve (V.2)

The origin of the maxillary nerve lies inside the infero-lateral dural border of the cavernous sinus (Fig. 5,6). It exits the middle cranial fossa via the foramen rotundum\(^4\) (Fig. 7,8) to enter the inferior orbital fissure (Fig. 9,10). Inside the inferior orbital fissure, it divides into the zygomatic nerve and the infraorbital nerve which travels anteriorly inside the infraorbital canal\(^25\) (Fig. 12).

3.4. Vascular anatomy

3.4.1. Arterial system

3.4.1.1. Internal carotid artery

The arterial supply to the orbit mainly derives from the internal carotid artery (ICA) that courses through the petrous portion of the temporal bone in the carotid canal and enters the middle cranial fossa by passing over the foramen lacerum. The ICA courses superiorly to the posterior clinoid process (Fig. 23) and then turns anteriorly to enter the venous plexus of the cavernous sinus (CS). Within the cavernous sinus, the ICA proceeds anteriorly with the abducens nerve along its lateral side (Fig. 4-6) giving off several small branches to supply the surrounding cranial nerves and the hypophysis.

Then, the ICA makes an upward S-shaped turn to form the carotid siphon (Fig. 7,18,19). In most individuals, the ICA gives off the ophthalmic artery (OA) outside the cavernous sinus at the medial side of the anterior clinoid process (Fig. 19). Occasionally, the ophthalmic artery may arise from the ICA within the cavernous sinus.\(^35\) While coursing upwards, the ICA gives off the posterior communicating artery (Fig. 19) to the posterior cerebral artery (Fig. 17) and then divides into its two terminal branches, the middle cerebral artery (Fig. 4) and the anterior cerebral artery (Fig. 4,6) thus forming the circle of Willis.

3.4.1.2. Ophthalmic artery

The about 2-3 mm long intracranial portion of the ophthalmic artery (OA) originates from the ICA below the intracranial optic (Fig. 19) nerve and enters the optic canal within a split of the periorbita dura. Inside the canal, the vessel courses inferiorly to the optic nerve (Fig. 8,9). The ophthalmic artery usually emerges in the inferolateral portion of the optic foramen and then proceeds nasally (Fig. 10). The ophthalmic artery crosses over the optic nerve (Fig. 11,12,15) in 72%-95% of individuals or crosses under it in 5-28%.\(^24\) The first branch of the ophthalmic artery is usually the 0.3-0.4 mm thick central retinal artery (CRA) which courses inferiorly to the optic nerve (Fig. 12) and enters its dural sheath about 10 mm (range: 5-16 mm) behind the globe.\(^36\) Occasionally, the CRA branches off the ophthalmic artery together with or following the lateral posterior ciliary artery.\(^31\) The order of branching along the OA varies considerably. Other major branches that may be visualized on MRA\(^27\) include the posterior ciliary arteries (Fig. 15), the lacrimal artery, the posterior and anterior ethmoidal arteries, and the supraorbital and supratrochlear artery.

3.4.2. Venous system

3.4.2.1. Veins

The central retinal vein (not visualized in the present study) drains the blood from the retina. It traverses the optic nerve, courses in its subarachnoid space and exits the periorbita dura to either join the superior ophthalmic vein or drain into the cavernous sinus.

Drainage from the choroid of the eye is provided by the vortex veins which subsequently drain into the superior and inferior ophthalmic veins.\(^27\) The superior ophthalmic vein crosses under the superior rectus muscle (Fig. 12), passes between the origins of the superior and lateral rectus muscles (Fig. 10) and leaves the orbit through the superolateral portion of the superior orbital fissure. In axial scans below the level of the superior rectus muscle, it is usually possible to visualize the entire superior ophthalmic vein.\(^27\) The inferior ophthalmic vein (IOV) communicates with the pterygoid plexus via the inferior orbital fissure, passes between the origins of the inferior and lateral rectus muscles (Fig. 11,13) and leaves the orbit via the inferior portion of the superior orbital fissure. Occasionally, the inferior ophthalmic vein may pass superiorly to join the superior ophthalmic vein as it drains into the cavernous sinus. A medial ophthalmic vein that courses in the nasal extrascleral orbit\(^27\) may occur in about 40% of individuals. When present, it joins the superior ophthalmic vein near the superior orbital fissure.\(^29\)

3.4.2.2. Parasellar venous plexus (cavernous sinus)

The cavernous sinuses are situated on either side of the body of the sphenoid bone and sphenoid air sinuses, respectively (Fig. 4-7). They extend from the apex of the petrous portion of the temporal bone posteriorly to the superior orbital fissure anteriorly. The cavernous sinuses are not trabeculated sinuses, as originally described by Winslow in 1732\(^32\) but rather represent extradural venous plexuses surrounded by a dural fold\(^64\). The concept of a venous plexus, as opposed to a true venous sinus, has been verified using contrast-enhanced, dynamic CT scanning. Contrast-enhanced MRI studies have demonstrated that venous flow in the cavernous sinuses can be divided into rapid-flow, characterized by marked enhancement, and low-flow channels with less enhancement.\(^44\)
Orbital tributaries to the cavernous sinus are the superior ophthalmic vein, the inferior ophthalmic vein and the central retinal vein. The two cavernous sinuses communicate with each other via the anterior and posterior intercavernous sinuses which are located anterior and posterior to the pituitary stalk in the diaphragm sellae. The cavernous sinus mainly drain into the internal jugular vein via the petrosal sinuses.

The intracavernous internal carotid artery (ICA), with its periarterial sympathetic plexus, has originally been described to pass the sinus in direct contact with the venous blood. However, in reality, it runs between the venules of the parasellar venous plexus. The abducens nerve may run very closely to the ICA (Fig. 4-6), either separated from it only by its nerve sheath, or embedded inside the lateral dural border of the sinus. Within the lateral dural border of the cavernous sinus ("lateral wall"), from superior to inferior, run the oculomotor and trochlear nerves, and ophthalmic and maxillary divisions of the trigeminal nerve (Fig. 4-6). Occasionally, these nerves are only separated from the parasellar venous flexus by a thin connective tissue layer.

The trigeminal ganglion is situated in a dural fold (Meckel’s cave) in continuation to the lateral wall of the cavernous sinus (Fig. 4.21).

4. TECHNICAL COMMENTS

4.1. Sequences and coils

MRI demonstrates the anatomy of the orbit and retroorbital region with superb detail. The best resolution of orbital structures is presently obtained using standard T1-weighted spin echo (SE) or T2-weighted fast (turbo) spin echo (TSE) pulse sequences. Conventional T2-weighted and proton density images need too long an acquisition time leading to motion artifacts. For the orbits, local surface coils and, whenever possible, phased array coils should be utilized. However, since the signal decay in the orbital apex depends on the diameter of the coil, imaging of this region requires a larger surface coil (Fig. 30). When additional imaging of the middle cranial fossa is required, the use of a head coil is recommended. The cranial nerves within the subarachnoid cisterns are best visualized on T2-weighted images where they appear hypointense against the bright background of the cerebrospinal fluid.

The high fat content of the orbit is responsible for the excellent contrast of orbital MR images enhancing the detection of tiny anatomical structures. Fat appears hyperintense (bright) on T1-weighted and T2-weighted images and other structures such as vessels, nerves and muscles appear darker (hypointense) than orbital fat. Even parts of the orbital connective tissue system can be visualized.

Thin slices, such as in the present study (0.5 - 2 mm) enable visualization of relatively long segments of delicate blood vessels and nerves. Thicker sections may catch even longer segments of neurovascular structures but partial volume averaging affects the image quality. Those parts of anatomical structures that are partially out of the imaging plane, are not represented in focus and show an altered signal intensity (partial volume effect).

4.2. Signal void of flowing blood

Fast and turbulent flow in blood vessels results in loss of intravascular signal intensity (signal void) due to dephasing inside the measured volume element (voxel). Intravoxel dephasing can be minimized by using a short echo time and small voxels. Slow flow or vortex flow results in reduced intravascular signal intensity due to saturation effects. Saturation effects can be minimized by reducing the flip angle or lengthening the TR. Therefore, the lumen of blood vessels appears dark or hypointense depending on blood flow velocity and imaging parameters.

The internal carotid artery inside the cavernous sinus appears as signal void in both T1w (Fig. 7.20) and T2w images. On T2w images, this flowing blood is clearly differentiated from hyperintense CSF (Fig. 18). The hypointense lateral border of the cavernous sinus is also sharply demarcated from adjacent CSF on T2w images (Fig. 17).

4.3. Contrast enhancement

The paramagnetic contrast medium gadolinium-diethylenetriaminepentaacetic acid (Gd-DTPA) enhances vascular structures, such as cavernous sinus or the venous plexus surrounding Meckel’s cave and the hypophysis (Fig. 4-6).
The cranial nerves passing the sinus can be visualized as individual structures only in contrast enhanced coronal MR images (Fig. 4-6). Inside the orbit, the enhancing effect of Gd-DTPA may result in decreased contrast because of the intense signal of orbital fat on T1w. Various fat suppression techniques are available to permit the evaluation of gadolinium-enhanced tissue within the retrobulbar fat.\(^{20}\)

Fat suppression techniques with or without contrast enhancement are especially useful for the diagnosis of retrobulbar optic neuritis\(^{21}\) and intraorbital meningeomas\(^{50,51}\). Limitations of fat suppression techniques include magnetic field inhomogeneities causing alterations of signal intensities. Contrast enhancement is not necessary for depicting the cranial nerves inside the subarachnoid cisterns. They are also delineated on non-enhanced T2w images due to the excellent contrast between hypointense nerves and hyperintense CSF (Fig. 16-18, 20-22, 24-29).

4.4. Advantages and disadvantages of MRI

The advantages of MRI are not only related to its ability to obtain a high degree spatial resolution but also to the possibility to characterize tissues because of their different water content. Some experience in MR imaging technique and familiarity with both data acquisition and contrast to noise relations are necessary to achieve this goal. There are pitfalls in the interpretation of fast acquisition MR images and certain caveats have to be heeded. However, this recognition becomes less problematic as the radiologist gains experience.

Another major advantage of MRI is the possibility of multi-planar imaging without the need of repositioning the patient. The selection of imaging planes along specific anatomic structures of interest enables visualization of long sections of these structures which is of importance for depicting the relatively thin cranial nerves. MRI allows visualization of the intracanalicular optic nerve and details of the cavernous sinus and avoids the beam-hardening artifacts known from CT at the orbital apex.\(^{52}\)

For these reasons, MRI is generally superior to CT in imaging soft tissue lesions of the cranioorbital junction. However, in comparison with MRI, CT allows much better delineation of bones. Especially the complicated bony anatomy of the optic canal and superior orbital fissure is better visualized on CT scans\(^{19}\) than on MRI scans. Therefore, CT scans with bone window algorithms are the first choice imaging modality in patients with orbital trauma\(^{52}\), bone lesions or craniofacial deformities\(^{54}\). In soft tissue lesions, CT scans are useful to detect secondary bony abnormalities (e.g. hyperostosis in meningeomas or bone erosion by malignancies) or calcifications within tumors (e.g. meningeomas or gliomas). We recommend to order CT scans in addition to MRI scans in all soft tissue lesions of the cranioorbital junction when a surgical procedure is planned in order to detect bone involvement and show the topographical relationship of the lesion to bony structures. CT scans must also be ordered in uncooperative or claustrophobic patients and patients with contraindications to MRI (see paragraph 4.6.).

Although MRI and MR-angiography\(^{22}\) may be helpful in diagnosing intracranial aneurysms or shunts at the cavernous sinus, the „gold standard“ for intracranial vascular disease is catheter angiography and superselective vessel exploration. In this presentation, we review neither MR-angiography nor invasive carotid angiography. Instead, emphasis is placed on the discussion of vascular pathways as depicted by standard and widely accessible MR equipment, without the use of contrast material. We point out that anatomical knowledge remains the basic denominator of our diagnostic capabilities in face of emerging advanced-level spatial encoding techniques.

4.5. Imaging artifacts

It is important, to recognize imaging artifacts in order to avoid misinterpretation. The following artifacts may present considerable problems in high-resolution MRI of the orbit:

4.5.1. Motion artifacts : Eye motion and eyelid blinking result in creation of „ghost images“. This can be minimized by having patients keep their lids open and their view fixed on a point inside the MR gantry.\(^{53}\) Reflex blinking may be reduced by instillation of local anaesthetic eye drops and artificial tear drops. However, if a longer acquisition time is needed, good results may also be obtained by having patients close their lids. Because of differences in voxel size and signal-to-noise ratio, surface coils are more sensitive to motion artifacts than volume coils.\(^{55}\) Therefore, high-resolution MRI of the orbit should be restricted to cooperative subjects who are able to lie still in the scanner for at least 2 - 3 minutes. Motion artifacts can occur with all pulse sequences but increase with scanning time depending on repetition time, number of excitations and matrix size.\(^{50}\)

**Fig. 31.** T1-weighted axial MR scan demonstrating chemical shift artifacts. Black bands are noted at the interface of orbital fat and adjacent tissue. The arrow indicates the chemical shift artifact between lateral rectus muscle and orbital fat.
4.5.2. Partial volume artifacts: The signal intensity value measured is an average value for the signal intensities of the different tissue structures in the measured volume element (voxel). Different tissue structures inside one voxel contribute to the total signal intensity of that voxel. This partial volume effect can be reduced, for example, by using thinner slices and imaging matrices with higher resolution. When segments of the examined structure are partially out of the imaging slice, they appear hypointense or thinner (Fig. 18,21). Thus, partial volume averaging is a potential source of error during the identification of anatomical structures in MR-images. In order to circumvent this problem, it is always necessary to analyse the whole series of adjacent imaging slices (e.g. stacked on a workstation) and corresponding perpendicular orientations.

4.5.3. Chemical shift artifact: This artifact occurs because of a difference in the resonant frequency of protons within tissues containing water and fat. In the orbit, a black and white band may be seen at the interface of orbital fat and adjacent tissues (Fig. 31). In T1w images, the chemical shift artifact may cause hypointense borders the optic nerve which may be confused with the T1-hypointense subarachnoid space of the nerve. With alteration of signal encoding directions, utilization of the smallest possible pixel size, keeping the echo time in phase and application of suppression techniques, this artifact can be reduced or eliminated. Chemical shift artifacts in T2w MR images can especially be minimized by use of turbo spin echo (TSE) sequences instead of conventional spin echo (SE) sequences.

4.5.4. Metal artifacts: Ferromagnetic materials, such as steel wire, mascara, eyelining tattoos and palpebral springs (for facial nerve palsy) cause artifacts in all imaging sequences, whereas titanium orbital implants, miniplates and gold eyelid weights (for facial nerve palsy) are seen as signal voids.

4.6 Contraindication of MRI: Ferromagnetic particles can move in a strong magnetic field and electrical devices may suffer dysfunction due to the potential induction of currents or heat. Therefore, MRI is contraindicated in patients with cardiac pacemakers, ferromagnetic implants, especially those located near vital structures (e.g. aneurysm clips) and ferromagnetic foreign-bodies, especially if they are located close to important structures (e.g. intraorbital or intraocular foreign-bodies).

5. ANATOMICAL COMMENTS

All major anatomic structures including the origin of the extraocular muscles and the apical orbital connective tissue system can be demonstrated on MRI. In the selected pulse sequences, blood vessels usually appear dark ("signal void") as discussed earlier. All important arterial and venous vessels of the orbit can be identified without contrast enhancement. It is also possible to delineate the intraorbital and intracranial course of sensory and motor cranial nerves of the orbit on MRI. In the present study, nerves were traced from the brain stem via their passage through the cavernous sinus to the orbital apex. Most of the cranial nerves within the cavernous sinus and orbital apex which were delineated in the present investigation, have already been visualized in previous studies, although earlier MR technology has not provided the resolution necessary for discriminating individual nerves (except for the IIIrd nerve and maxillary nerve). With present MR technology, it is feasible to visualize individual nerves within the cavernous sinus and the orbit. Only the thin trochlear nerve is difficult to visualize. On appropriate sections, it may be depicted as it courses around the midbrain (Fig. 20), inside the dural border of the cavernous sinus (Fig. 4-6) and within the orbit (Fig. 12).

The imaging appearance of the optic nerve is influenced by its sinuous course as well as the plane and thickness of sectioning: Thin axial slices at the level of the optic canal show the intracanalicular portion of the optic nerve (Fig. 16), but not the infraorbital part and vice versa (Fig. 18). However, thicker (about 3 mm), either oblique-axial slices along the neuro-ophtalmic plane or oblique sagittal sections (parallel to the course of the nerve) can depict the entire optic nerve. If the entire optic nerve is to be visualized in one single image, it may be helpful to obtain scans in upgaze to stretch the nerve, as first proposed for CT-scanning. The cross-sectional diameter of the optic nerve can be measured in fat-suppressed, T2w MR images in an oblique-coronal plane, perpendicular to its course. The mean (±SD) pial diameter of the intraorbital portion of a normal optic nerve ranges between 3.2±0.4 mm anteriorly and 2.6±0.4 mm posteriorly, whereas the mean (±SD) dural diameter measures between 5.2±0.9 mm (anteriorly) and 3.9±0.4 mm (posteriorly).

6. CLINICAL IMPLICATIONS

High-resolution MRI enables exact delineation of space occupying orbital processes in relation to surrounding anatomical structures thus facilitating planning of surgical procedures. This feature will be essential for computer-assisted surgery using neuronavigation. MRI reveals informations on blood flow and may differentiate between flowing and stagnant blood in orbital vascular lesions. This is extremely important for treatment planning. MR-angiography may provide further non-invasive diagnostic insights, depicting the entire course of vessels, including the circle of Willis. The closeness of vessels to oculo-motor nerves and the chiasm explains the occurrence of eye muscle palsies, and occasionally visual field defects caused by aneurysms. For instance, the abducens nerve courses in close contact to the internal carotid artery through the cavernous sinus (Fig. 4-6). For this reason, the VIth nerve is usually the first nerve affected.
by intracavernous carotid aneurysms or arterio-venous shunts. It must be noted that small aneurysms may only be detected using catheter selective angiography techniques. Increased pressure within the cavernous sinus as in high flow carotid-cavernous or low flow dural-cavernous fistulae, results in enlargement of the ophthalmic veins. Low-flow fistulae may be supplied by small branches of the ICA to the oculo-motor nerves (see above) or dural arterioles, both of which are not visible on MRI. Enlarged ophthalmic veins may also be encountered in other disorders, such as arteriovenous malformation, cavernous sinus thrombosis and Graves’ ophthalmopathy. Septic cavernous sinus thrombosis is a rare but life-threatening disease. It always occurs bilaterally, because the intercavernous sinuses carry no valves. Enlargement of superior or inferior ophthalmic veins is best recognized on axial T1w scans. Attention should be paid not to misinterpret enlarged veins at the orbital apex as a space occupying lesion.

Tumors of the oculomotor cranial nerves, causing progressive eye muscle palsies, usually cannot be reliably differentiated from other tumors because of their unspecific imaging characteristics. High-resolution MRI might help to demonstrate an anatomical relation between tumor and nerve, thus suggesting the diagnosis of a neurogenic tumor. Similarly, a space-occupying lesion with extension to the orbital venous system (and enlargement during Valsalva maneuver), is suggestive of an orbital varix.

Using contrast enhanced MRI and fat-suppression, it is possible to localize inflammatory lesions of cranial nerves along their course. It has been demonstrated that high-resolution MRI may disclose compressive nerve lesions in patients with eye muscle palsies in whom routine brain MRI studies were unremarkable. In recent studies, spoiled gradient recalled steady state acquisitions revealed vascular or neoplastic compression of the subarachnoid portions of the III\(^{\circ}\) and VI\(^{\circ}\) nerve in patients with corresponding palsies.

The intracranial portion of the abducens nerve passes through Dorello’s canal underneath the petroclinoideal ligament (Fig. 26,28). Here, the V\(^{\text{th}}\) nerve is predisposed to injury from compressive lesions or head trauma. In some individuals, the petroclinoideal ligaments may be ossified causing compression of the VI\(^{\circ}\) cranial nerve. For these reasons, it is advisable to individually check this structure when interpreting MR images of patients with VI\(^{\circ}\) nerve palsies. The trigeminal ganglion is located close to the posterolateral wall of the cavernous sinus (Fig. 4,11). This topographic relation explains the potential involvement of the entire V\(^{\circ}\) nerve (including the mandibular nerve clinically apparent as loss of masticatory function) in lesions of the posterior cavernous sinus.

A new neuro-ophthalmologic application of high-resolution MRI is its use to demonstrate anatomic abnormalities in congenital motility disorders. Recently, absence of the abducens nerve in Duane’s syndrome, previously only described in post-mortem studies, has been verified in-vivo.

The dural optic nerve diameter can be measured on oblique-coronal MR images. An increased dural diameter with synchronous flattening of the posterior sclera, excessive tortuosity of the optic nerve, intravitreous protrusion, enhancement of the papillae, and an empty sella are suspicious of pseudotumor cerebri. The subarachnoid space of the optic nerve is 0.5-1 mm wide and contains cerebrospinal fluid that is freely exchanged with the subarachnoid space of the brain. Clinically, this communication provides a route of spread for infection, neoplastic cells or hemorrhage. The subarachnoid periopic space may be enlarged in optic nerve glioma where it is filled with water-containing T2-hyperintense gliomatous tissue. Echographic A-scan measurements of the optic nerve ("30°-test") imply a redistribution of cerebrospinal fluid following abduction and have been claimed to enable a differentiation between optic nerve thickening caused by fluid distension and optic nerve tumors. However, MRI studies could not verify a significant displacement of cerebrospinal fluid following gaze changes. Further comparative studies on the validity of MRI and echography for the differential diagnosis of optic nerve thickening are warranted.

The optic chiasm is separated from the pituitary gland by the diaphragm sellae and the up to 10 mm wide suprasellar cistern. Pituitary adenomas must therefore be quite large to produce visual field defects. The chiasm is normally situated on top of the diaphragm sellae. In some individuals it may extend onto the dorum sellae (postfixed chiasm) or close to the planum sphenoidale (prefixed chiasm) which accounts for variations in visual field defects associated with tumors in this region (e.g. pituitary adenoma). The superior orbital fissure represents a communication channel which transmits important neurovascular structures between the intracranial space and the orbit. This explains the spread of inflammatory or neoplastic lesions between the two compartments. Small lesions at the superior orbital fissure and retroorbital region (e.g. Tolosa-Hunt syndrome) may easily be overlooked. Therefore, one should always pay careful attention to this region when interpreting cranio-orbital MR images.

These clinical examples show that MRI may contribute to a specific diagnosis in space-occupying lesions at the cranio-orbital junction. The present article has provided the basic morphological knowledge which is essential for a successful application of this non-invasive diagnostic technique.