Figure 1-3  Parasellar bony anatomy demonstrates the relationship of the pituitary fossa to the cavernous sinus, including the foramina of the skull base. The foramen lacerum is filled with cartilage and contains the artery of the pterygoid canal, the nerve of the pterygoid canal, and the venous drainage structures. The carotid artery enters the skull base through the carotid canal. (Courtesy of Albert L. Rhoton Jr, MD.)

Figure 1-4  Anatomy of the orbit. Bony anatomy of the right orbital apex. The optic canal transmits the optic nerve, ophthalmic artery, and some oculosympathetic fibers. The superior orbital fissure, between the greater and lesser wings of the sphenoid bone, transmits CNs III, IV, and VI; the ophthalmic division of CN V (CN V₁); the oculosympathetics; and the superior ophthalmic vein. (Illustration by Dave Peace.)

Table 1-1  Bones of the Orbit

<table>
<thead>
<tr>
<th>Orbital Roof</th>
<th>Lateral Wall</th>
<th>Orbital Floor</th>
<th>Medial Wall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal</td>
<td>Zygomatic</td>
<td>Zygomatic</td>
<td>Maxillary</td>
</tr>
<tr>
<td>Lesser wing of sphenoid</td>
<td>Greater wing of sphenoid</td>
<td>Maxillary</td>
<td>Lacrimal</td>
</tr>
<tr>
<td></td>
<td>Palatine</td>
<td>Ethmoidal bone</td>
<td>Ethmoid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lacrimal sac fossa</td>
<td>Lesser wing of sphenoid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Maxillary bone</td>
<td></td>
</tr>
</tbody>
</table>
The superior orbital rim is made up of the *frontal bone*, which connects to the *zygomatic bone* laterally at the *frontozygomatic suture*. The inferior orbital rim is made up of the zygomatic bone inferolaterally and the maxillary bone inferonasally, which meet at the *zygomaticomaxillary suture*. Medially, the orbital rim consists of the *maxillary* and *lacrimal bones*, which join the frontal bone superiorly. Three additional bones contribute to the orbit: the *ethmoid bone* medially, the *palatine bone* inferiorly in the posterior orbit, and the *sphenoid bone* laterally and superiorly in the orbital apex (Activity 1-1).

**ACTIVITY 1-1** Bony anatomy of the orbit.
*Developed by Zoë R. Williams, MD. Illustrations by Dave Peace.*

At the orbital apex, the annulus of Zinn gives rise to the 4 rectus muscles. CNs II and III, the nasociliary nerve of CN V, and CN VI pass through the annulus of Zinn. In contrast, CN IV and the frontal and lacrimal nerves of CN V, as well as the superior ophthalmic vein, pass through the superior orbital fissure outside the annulus of Zinn (Fig 1-5).

**CLINICAL PEARL**
Because the superior oblique muscle is innervated by CN IV, which bypasses the annulus of Zinn, it is often not paralyzed—or is the last extraocular muscle to be paralyzed—by a retrobulbar block.

The orbit is surrounded by several important structures, including 4 *paranasal sinuses* (Fig 1-6):

- the *maxillary sinus*, which is adjacent to the orbital floor
- the *ethmoid sinus*, which is adjacent to the orbital medial wall
- the *sphenoid sinus*, also adjacent to the orbital medial wall
- the *frontal sinus*, with variable relationship to the anterior orbital roof

The *sphenoid sinus* forms the medial wall of the optic canal (Fig 1-7). In approximately 4% of patients, the bone may be incomplete, leaving only mucosa separating the sinus from the optic nerve. Surgery within the sphenoid sinus can potentially damage the optic nerve. In patients with pituitary or suprasellar lesions, use of the sphenoid sinus for an endoscopic surgical approach facilitates decompression of the optic chiasm. However, decompression can also be approached via craniotomy depending on the extent and site of pathology.

Other major structures around the orbit are the *anterior cranial fossa* superiorly (containing the frontal lobe) and the *temporal fossa* laterally (containing the temporalis muscle). The roof of the *ethmoidal complex*, delineated by the *frontoethmoidal suture* (top of the *ethmoid bone*, or *lamina papyracea*), marks the inferior boundary of the anterior
CDVA using a standard 20/200 E optotype with documentation of the distance at which the patient can identify the letter orientation using standard Snellen notation (eg, “5/200”). This test provides a more accurate and reproducible measurement than does the finger-counting method.

Distance and near corrected visual acuity should be similar; a disparity may suggest a specific pathology. The clinician should document the presence of eccentric fixation (possible central scotoma), tendency to read half of the eye chart (possible hemianopic field defect), or improvement in CDVA when single optotypes are read (which may suggest amblyopia).

**Color Vision Testing**

Color vision testing complements visual acuity assessment. Optic nerve disease, particularly demyelinating optic neuritis, may disproportionately affect color vision compared with CDVA. In macular disease, visual acuity and color vision tend to decline correspondingly; the exception is cone dystrophy, in which color vision is more significantly decreased than visual acuity. Thus, an optic neuropathy rather than a maculopathy is the more likely etiology in the differential diagnosis for an eye with 20/30 visual acuity but severe loss of color vision. In optic neuropathy, persistent dyschromatopsia can occur even after recovery of visual acuity.

Color vision should be tested separately in each eye to detect unilateral disease. The Ishihara test (Fig 3-1A) uses a series of pseudoisochromatic color plates to test color discrimination along the protan (red) and deutan (green) axes. The plates were designed to detect congenital red-green color deficiencies and may fail to detect mild cases of acquired dyschromatopsia. Bilateral, symmetric color vision loss in men may signify congenital color deficiency rather than bilateral optic neuropathies. The Hardy-Rand-Rittler (HRR) plates can

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**Figure 3-1** Pseudoisochromatic plates for color vision testing. **A,** Ishihara plate. **B,** Hardy-Rand-Rittler (HRR) plate. (Courtesy of Zoë R. Williams, MD. Photography by Brittany Figenschcher, CRA.)
be used to screen for tritan (blue-yellow) axis defects as well as red-green defects (Fig 3-1B). Blue-yellow color defects often accompany acquired optic neuropathy but also can occur in a maculopathy.

More detailed color testing may comprehensively characterize a color vision defect. In the Farnsworth panel D-15 test, the patient is asked to arrange 15 colored discs in order of hue and intensity. The Farnsworth–Munsell 100-hue test is the most comprehensive test and provides the best discrimination; however, it requires a substantial amount of time to take (patients arrange 4 sets of 25 colored discs) and score, thus limiting its use in routine clinical testing. Color vision testing is discussed further in BCSC Section 12, Retina and Vitreous.

**Pupillary Testing**

Normally, light directed at either pupil causes equal constriction of both pupils (see Chapters 1 and 11). When light is shined into an eye with impaired conduction of the afferent pupillomotor signal along its optic nerve, pupillary constriction in both eyes is slower and smaller in amplitude compared with the response that occurs when light is shined into the eye with normal optic nerve conduction. In other words, both the direct and the consensual response are sluggish. This equal consensual response enables detection of a relative afferent pupillary defect (RAPD) as the light is moved back from the side with the normal conduction to the side with impaired conduction, causing dilation of the pupil on the impaired conduction side.

The most popular clinical method for detecting an RAPD is the swinging flashlight test (Video 3-1), which compares the pupillary response in the 2 eyes when they are tested with the same light source. The test involves swinging a bright light (which is shined directly into the pupil along the visual axis) between the 2 eyes. If the afferent input is significantly asymmetric, the pupils redilate immediately when the light is shined into the affected eye (Fig 3-2).

**VIDEO 3-1** Left relative afferent pupillary defect. 
*Courtesy of M. Tariq Bhatti, MD. Narration by Helen Danesh-Meyer, MD, PhD.*

**Figure 3-2** Assessing for a relative afferent pupillary defect (RAPD) in a patient with left traumatic optic neuropathy; the left pupil is pharmacologically dilated. **A,** The right pupil constricts in response to light directed at the right eye only. **B,** The right pupil dilates in response to light directed at the left eye only, indicating a left RAPD. * (Courtesy of Michael S. Lee, MD.)