Optic nerve injuries are classically divided into direct and indirect injuries. Direct injuries are open injuries where an external object penetrates the tissues to impact the optic nerve. Indirect optic nerve injuries occur when the force of collision is imparted into the skull and this energy is absorbed by the optic nerve. The prognostic value in knowing that an injury was direct or indirect is unclear. Historically, direct optic nerve injury is associated with a poor visual outcome. It may be safe to classify an optic nerve injury as direct if orbital imaging reveals a bullet at the orbital apex, but this provides no insight into the cellular injury mechanism. The classification does not illuminate whether the nerve is severed with no hope of recovery or only mildly injured with significant recovery potential. This becomes important as strategies for treating nerve injuries evolve.

Optic nerve injuries are also classified anatomically. Injuries that occur anterior to where the central retinal artery enters the optic nerve produce abnormalities in the retinal circulation in association with visual loss (1,2) (Fig. 9.1). Disturbances in retinal circulation can be associated with orbital hemorrhages that compromise the optic nerve. Avulsions of the optic nerve from the globe produce a distinct picture with a partial ring of hemorrhage at the optic nerve head (3) (Fig. 9.2). In contrast, posterior optic nerve injuries occur posterior to the site where the central retinal artery enters the optic nerve. The most common site of indirect optic nerve injury is the bony optic canal, referred to as an intracanalicular injury (4,5). The intracranial optic nerve is the next most common site of injury (6). Chiasmal injuries can be diagnosed on the basis of visual field changes when this test can be obtained (7). Chiasmal injuries are associated with a high incidence of diabetes insipidus, reported to be 37% in one series (8). Neuroimaging is usually able to separate intracanalicular injuries from orbital injuries occurring posterior to where the central retinal artery enters the optic nerve. Orbital optic nerve injuries can be associated with intrasheath hemorrhage, which anecdotally have benefited from clot drainage (9).

EPIDEMIOLOGY

Traumatic optic neuropathy occurs in association with high momentum deceleration injuries. Optic nerve injury is often associated with midface trauma. Table 9.1 lists the most common causes of traumatic optic neuropathy. Motor vehicle and bicycle accidents are the most frequent cause, accounting for 17–63% of cases, depending on the series. Motorcycle accident victims may be particularly vulnerable to traumatic optic neuropathy. A consecutive series of 101...
Figure 9.1. Central retinal vein occlusion in a case of anterior (proximal) optic neuropathy. The patient was a 24-year-old man who was struck in the right eye while playing basketball and who immediately experienced loss of vision in the eye. Visual acuity was light perception OD and 20/15 OS. A, Ophthalmoscopic appearance of right ocular fundus reveals moderate swelling of the optic disc. The disc is surrounded by hemorrhage and soft exudates. The retinal veins are dilated and tortuous. B, Computed tomographic (CT) scan, axial view, shows moderate enlargement of the orbital portion of the right optic nerve. C, CT scan, coronal view, shows enlargement of right optic nerve compared with left nerve. Note small areas of increased density, consistent with hemorrhage, with the enlarged nerve. (Courtesy of Dr. Neil R. Miller.)

Figure 9.2. Traumatic avulsion of the optic disc. Note ring of hemorrhage around the optic disc. The site of avulsion is clearly visible as a crescentic dark area at the temporal portion of the disc (arrowhead). (Courtesy of Dr. Neil R. Miller.)
patients with head trauma after a motorcycle accident found 18 cases of traumatic optic neuropathy (18%) (10). Falls are the next most common cause, producing 14–50% of cases. Traumatic optic neuropathy has also resulted from frontal impact by falling debris (11,12), assault (11), stab wounds (13), gun shot wounds (13,14), skateboarding (11), bottle-cork injuries (15), following seemingly trivial injuries (3,16), and following endoscopic sinus surgery (17–20). Iatrogenic optic nerve trauma with visual loss is rare following elective LeFort I osteotomy. When it does occur, it may be related to uncontrolled and unpredictable pterygomaxillary disjunction with propagation of the fractures into the optic canal (22).

Optic nerve injury is usually associated with concomitant multi-system trauma or serious brain injury. Loss of consciousness is associated with traumatic optic neuropathy in 40–72% of cases (11,21–23). The incidence of indirect traumatic optic neuropathy has been defined in the past as a subpopulation of individuals with head trauma (4,24). In a large study of maxillofacial trauma patients, traumatic optic neuropathy occurred in 2.5% of patients with midface fracture (25). The percentage of patients with light perception and no light perception vision following traumatic optic neuropathy varies from 43% (13,26) to 78% (27) (Table 9.2). However, recent studies have higher percentages of patients with more subtle forms of traumatic optic neuropathy (11,13,26). Patients with mild forms of traumatic optic neuropathy were probably not identified in the older series.

Optic nerve injury following orbital hemorrhage defines an important subset that does not fit well into the classic delineation of direct and indirect optic nerve injuries (28,29). The mechanism of neuropathy is an orbital compartment syndrome resulting in elevated orbital pressure comprom-

<table>
<thead>
<tr>
<th>Table 9.1</th>
<th>Etiology of Traumatic Optic Neuropathy</th>
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<tbody>
<tr>
<td>First Author</td>
<td>Cases</td>
</tr>
<tr>
<td>Nau (22)</td>
<td>18</td>
</tr>
<tr>
<td>Bodin (188)</td>
<td>6</td>
</tr>
<tr>
<td>Millies (189)</td>
<td>29</td>
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<tr>
<td>Seiff (11)</td>
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<tr>
<td>Anderson (178)</td>
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<td>Matsuzaki (174)</td>
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<td>21</td>
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<tr>
<td>Kountakis (190)</td>
<td>34</td>
</tr>
<tr>
<td>Lubben (194)</td>
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</tr>
<tr>
<td>Total</td>
<td>263</td>
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</table>

<table>
<thead>
<tr>
<th>Table 9.2</th>
<th>Frequency of Improvement Over Initial Visual Acuity by Series in Chronological Order</th>
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</thead>
<tbody>
<tr>
<td>Initial Visual Acuity</td>
<td>No Light Perception</td>
</tr>
<tr>
<td></td>
<td>Total</td>
</tr>
<tr>
<td>Hooper (191)</td>
<td>58</td>
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<tr>
<td>Hughes (5)</td>
<td>90</td>
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<tr>
<td>Edmund (192)</td>
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<td>Anderson (178)</td>
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<td>Lubben (194)</td>
<td>65</td>
</tr>
<tr>
<td>Wang (27)</td>
<td>61</td>
</tr>
<tr>
<td>Totals</td>
<td>636</td>
</tr>
</tbody>
</table>

Articles listed represent those series where sufficient visual acuity data was published to permit this analysis.

³ Visual improvement was defined as any improvement in visual function according to the authors of each listed article.
ing the circulation to the optic nerve. Orbital hemorrhage may occur spontaneously following thrombolytic therapy, in association with surgery, childbirth, and in the presence of sickle cell disease as well as known coagulopathies (30–36). In one report, a subperiosteal orbital hematoma with visual compromise occurred due to a subgaleal hemorrhage in a child following hair pulling (37). The best-studied group of patients with orbital hemorrhage are those undergoing retrobulbar injection of anesthetic for ophthalmic surgery. The prevalence of an orbital hemorrhage following a retrobulbar block is 0.44–3% (38–41). When orbital hemorrhage occurs following a retrobulbar block, it is generally recognized and readily managed with little impact on visual outcome.

Although rare, direct optic nerve injury from a retrobulbar needle has been reported (42,43). When retrobulbar hemorrhage occurs in association with trauma, the risk of visual loss is much greater. Hemorrhage following blepharoplasty is also a recognized cause of potential visual loss (44). Orbital hemorrhage is associated with alloplastic repair of orbital fractures (45,46). Blood can be dispersed in the orbit, in the subperiosteal space, and in the optic nerve sheath, or result in a hematic cyst (47,48). Imaging studies can help localize the hemorrhage (49–51).

Orbital emphysema is generally a benign condition. However, air can become trapped in the orbit due to a ball-valve mechanism typically following orbital fracture. Compulsive optic neuropathy and visual loss can result (52). Vomiting and nose blowing in the setting of orbital fracture may be associated with optic nerve compromise when air is forced into the orbit (53). Optic neuropathy in conjunction with simultaneous tension orbital emphysema and pneumocephalus following blunt trauma has been reported (54). Orbital emphysema with visual loss has also been reported in one patient following the use of a high-speed air-cooled drill in the dental setting (55).

**CLINICAL ASSESSMENT**

Traumatic optic neuropathy remains a clinical diagnosis. By definition, there must be a history of trauma. Associated loss of consciousness is common. A diagnosis of optic nerve trauma should not be made in the presence of normal vision and normal pupillary function. Following trauma, respiratory and cardiovascular resuscitation are the first priority. Every case of optic nerve injury, however mild, represents head trauma. Therefore care of these patients usually occurs in a team setting involving emergency physicians, trauma surgeons, head and neck surgeons, neurosurgeons, and ophthalmologists. Under these circumstances, the initial ophthalmic examination may be limited by other clinical considerations. The ophthalmic assessment should be completed at the earliest opportunity.

**HISTORY**

Clinical evaluation of a patient with visual loss following trauma should begin with as complete a history as possible. If the patient is unconscious, the history should be obtained from significant others when present. Witnesses at the scene, including responding emergency personnel, may provide an understanding of the mechanism of injury. The possibility of exposure to hazardous materials should be considered. An ocular history must be explored to rule out visual loss prior to the injury. A detailed medical, drug, and drug allergy history is also necessary. Open injury creates a risk for tetanus, and the patient’s tetanus immunization status should be investigated.

**EXAMINATION**

**Visual Acuity**

Visual acuity following indirect optic nerve trauma is often significantly reduced. Each patient in Hughes’ series of 56 cases presented with no light perception (5). All 46 patients described by Turner also presented with no light perception on the side with optic nerve injury (4). In Hoop-er’s series, 14 of 21 patients presented with no light perception (56). In the series reported by Edmund, 17 of 22 patients presented with no light perception (21). While vision better than 20/400 is not inconsistent with traumatic optic neuropathy, it is unusual in older reported cases.

As can be seen from Table 9.2, recent reports contain larger numbers of patients with visual acuity of better than 20/200 in the affected eye. In the past, patients with minor decreases in visual function may have gone undetected. Commonly, patients with traumatic optic neuropathy will have vision that is 20/400 or less in the affected eye. However, the clinician must maintain a high index of suspicion to avoid missing more subtle cases of traumatic optic neuropathy. Delayed visual loss is reported to be as high as 10% in some series (23,57).

**Pupillary Reflexes**

In cases of unilateral traumatic optic neuropathy, the presence of an afferent pupillary deficit is a necessary condition for the diagnosis of traumatic optic neuropathy. Afferent pupillary defects can be quantitated using photographic neutral density filters (58). These filters are calibrated in log units that measure the reduction in transmitted light. The lenses can be stacked to produce an additive effect in front of the normal eye until the reduction in light reaching the normal eye matches the reduction caused by the relative afferent pupillary defect. When the input in both sides is equal, the afferent pupillary defect will be neutralized. The log unit reduction in light reaching the good eye is a measure of the afferent pupillary defect. Afferent pupillary deficits of greater than 2.1 log units are predictive of poor visual prognosis (59).

**Biomicroscopy and Fundoscopy**

A complete and thorough examination of the eye and ocular adnexa is essential following trauma. Palpation of the orbital rim can identify step-off fractures. Periorbital swell-
ing may mask the presence of proptosis. Resistance to retro-
pulsion of the globe followed by tonometry can rapidly iden-
tify an orbit that is tense from a retro-orbital hemorrhage. Lid
swelling can increase the difficulty of the ocular examination
necessitating an assistant to retract the eyelids. Evidence of a
penetrating ocular injury should be sought. Blunt injury
to the iris may result in a hyphema or angle recession. The
force of trauma may dislocate the lens.

Posteriorly, blood in the vitreous may obscure the fundus.
If the patient is neurologically unstable, the treating neuro-
surgeon or trauma surgeon should be consulted prior to dilat-
ing the eyes. If a dilated examination is performed, to avoid
confusion the time of dilation and the type of agents used
must be documented both in the nursing and physician notes,
and posted prominently at the bedside. Only short-acting
agents should be used. When intracranial pressure is moni-
tored invasively, pupillary response may not be critical.
However, a dilated fundus exam to rule out retinal disinsert-
tion and tears may need to be deferred until the patient is
neurologically stable.

An adequate fundus examination will include assessment
of abnormalities of the retinal circulation. Partial and com-
plete avulsion of the optic nerve head produces a ring of
hemorrhage at the site of injury or the appearance of a deep
round pit (3,60). Anterior injuries between the globe and
where the central retinal vessels enter the optic nerve pro-
duce disturbances in the retinal circulation including venous
obstruction and traumatic anterior ischemic optic neuropathy
(1,61). Hemorrhages in the optic nerve sheath posterior to
the origin of the central retinal vessels may leave the circula-
tion of the retina intact, but produce optic nerve head swell-
ing (62). Frank papilledema may be seen in the setting of
raised intracranial pressure despite the presence of traumatic
optic neuropathy (63). The presence of choroidal rupture or
commotio retinae may explain visual loss. Clinical judgment
must be exercised to decide if these conditions are consistent
with an afferent pupillary defect. The presence of decreased
visual acuity and an afferent pupil defect in the absence of
intraocular pathology should suggest a posterior orbital,
intracanalicular or intracranial optic nerve injury.

VISUAL EVOKE POTENTIAL

The visual evoked potential (VEP) may be of use in the
unresponsive patient suspected of having traumatic optic
neuropathy (22,64,65). This is especially true in possible
bilateral cases where an afferent pupillary deficit may not
be evident. An unresponsive patient with midface fractures
and other than normal pupillary responses warrants a VEP
investigation. Generally, the actual clinical utility of VEP is
limited logistically. A VEP may not be available at the bed-
side, when the information is most critical, and moving the
patient to the evoked potential laboratory may not be possi-
ble. In addition, the test requires a highly qualified technican
who may not be available after hours. The VEP is useful
only when it is not recordable—in which case the chance
of visual recovery is very unlikely (22,66,67). The VEP can
frequently be abnormal but recordable in an amaurotic eye
immediately following optic nerve trauma with subsequent
extinction (22). It is thought that the false-positive VEP may
be due to a limited number of still intact axons that are able
to conduct.

VISUAL FIELD

Visual fields can be obtained only when sufficient vision
is present following optic nerve trauma. The visual field,
by virtue of the retinotopic organization of the optic nerve,
provides an understanding of the localization of optic nerve
damage. Within the canal, the pial penetrating vessels that
provide blood to the optic nerve are subject to shearing
forces at the moment of injury. Since the superior portion
of the optic nerve is most tightly bound within the canal,
these pial vessels are thought to be the most susceptible to
shearing forces. Partial avulsion of the optic nerve at the
globe produces visual field deficits that correspond to the
site of avulsion. However, there is no pathognomonic visual
field loss diagnostic of optic nerve trauma. Visual field test-
ing is useful in documenting the return of visual function
following injury (68). As a practical matter, confrontational
visual fields may be the only type of visual field available
at the bedside. Recording the ability to count fingers, detect
hand motion, or light in each quadrant may crudely quanti-
tate this. Ambulatory patients should be considered for for-
mal visual field testing.

IMAGING

The superior imaging afforded by computed tomography
and magnetic resonance imaging has made plain films and
hypoclinoidal tomograms obsolete. Manfredi and coworkers
reviewed the medical records of 379 patients with facial
fractures (69). Twenty-one patients lost all vision in one eye
(6%) and 3 of these 21 lost vision in both eyes; 12 of the
21 patients with visual loss had CT scans of the head as part
of the initial assessment and of these 12, visual loss was
attributed to traumatic globe injuries in 7 cases. CT scans
in the remaining 5 patients demonstrated a fracture through
the optic canal. The fracture may injure the optic nerve di-
rectly or it may serve as a marker of the severity of force
transferred into the optic nerve (Fig. 9.3). Seiff et al. reported
CT results of nine patients with traumatic optic neuropathy
(70). Six of the nine patients demonstrated fractures of the
optic canal. Fractures were present in adjacent structures but
not extending into the optic canal in two additional patients.
In a follow-up study, canal fractures were found in 16 of 36
patients and fractures of the bones adjacent to the optic canal,
but not involving the optic canal, in an additional 10 of
36 patients (11). However, 63% of the patients with canal
fractures presented with no light perception compared with a
40% incidence of no light perception when there was no
fracture or the fracture did not extend to the optic canal.

CT scanning in the setting of traumatic optic neuropathy
has demonstrated specific pathology implicated in the com-
promise of optic nerve function including optic nerve sheath
hematoma and presumed arachnoid cyst (9,71,72). While CT
scanning is clearly superior to magnetic resonance imaging
(MRI) in delineating fractures of bone, MRI is superior to
CT scanning in its ability to image soft tissue. Often both CT
and MRI are required to fully evaluate a particular clinical
situation (62,73) (Fig. 9.4). As a note of caution, MRI should
be performed only after a metallic orbital or intraocular foreign body has been ruled out by CT scan or conventional x-ray. MR imaging is useful in assessing chiasmal trauma (74).

DIFFERENTIAL DIAGNOSIS

Causes of visual loss following trauma, other than traumatic optic neuropathy, must be considered. With a thorough history and complete ocular examination, the differential diagnosis of visual loss with disturbed pupillary function can be rapidly narrowed. An optic nerve injury can accompany other ocular injuries. If the obvious ocular injuries do not account for the loss of vision and altered pupillary function, traumatic optic neuropathy should be suspected. Traumatic optic neuropathy can follow trivial head injuries (16,75). One must also be wary of visual loss that is merely coincidental to trauma. Monocular visual loss may not be noticed until the individual has occasion to close the uninvolved eye. In this setting the patient may attribute the visual loss to a recent traumatic event. Deciding if the visual loss is coincidental or caused by recent trauma may be difficult. Any process that can result in visual loss may be coincidentally associated with a traumatic incident such as decompression of a vascular aneurysm, orbital or optic nerve inflammation, anterior ischemic optic neuropathy, or acute sinus disease with orbital involvement. A traumatic cavernous sinus fistula is usually accompanied by numerous orbital findings, permitting easy differentiation from traumatic optic neuropathy (76).

PATHOLOGY

Pringle conducted 174 autopsies on patients who were left unconscious from the time of head injury until death (77). In 16 cases, blood was found in the optic nerve sheath, leading him to hypothesize that indirect injury to the optic nerve was caused by hemorrhage compressing the optic nerve. These observations are also supported by the work of Hughes who explored six nerves in five patients (5).

Crompton reported the visual lesions from a series of 84 autopsies performed soon after closed head trauma (6). Optic nerve dural sheath hemorrhages were present in 69 of 84 cases (83%). Intersitial optic nerve hemorrhages were present in 30 of 84 patients (36%). The interstitial hemorrhage was present in the optic canal in 20 of these 30 cases. Shearing lesions and ischemic necrosis were present in 37 of 84 cases (44%) and the intracanalicular optic nerve was involved in 30 of 37 cases. Significantly, the intracranial optic nerve also demonstrated shearing lesions and ischemic necrosis in 20 of these 37 cases. In Turner’s series, skull fractures were present in 27 of 46 cases of optic nerve injury. Only four patients demonstrated an abnormality of the optic canal suggesting, at least on the basis of plain film radiographs, the rarity of canal fractures (56). More recently, studies utilizing computerized tomography suggest a 50% incidence of sphenoid fractures in cases of traumatic optic neuropathy (11). However, these studies also demonstrate the important point that traumatic optic neuropathy can occur in the absence of an optic canal fracture.

Studies using laser interferometry suggest that forces applied to the frontal bone are transferred and concentrated in the area of the optic canal (78) (Figs. 9.5 and 9.6). The entire