The peripheral vitreoretinal interface is the final “ring of tissue” damaged in blunt trauma. This is the one area most difficult to examine in the immediate post-traumatic period. Examination of this area is particularly important in that undetected pathologic changes can have the most devastating effects on vision in a relatively shorter period of time. The impact of blunt injury affects the sclera, choroid, vitreous, retina and the optic nerve, the manifestations of which is dealt with in detail in this article.

**Mechanism of Blunt Injury**

The four phases of blunt injury are compression, decompression, overshooting and oscillations. Anterior – posterior compression of the globe at the cornea results in equatorial expansion and shortening of the globe along the visual axis. The anterior- posterior diameter of the globe decreases by as much as 41% and the equatorial diameter increases up to 128% of normal. The driving mechanism for pathological changes in this area is the equatorial elongation, creating shearing forces between the extensible ocular wall and the much less extensible vitreous. (Figure 1)

**Scleral rupture**

Direct rupture of sclera is uncommon and occurs at the site of impact. Indirect rupture occurs remote from site of impact in an area of scleral weakness. The sclera is thinnest at the insertion of the rectus muscles (Figure 2), where it measures 0.3mm. It is thickest at the posterior pole (1mm) and about 0.4 to 0.5mm at the equator. It is at the muscle insertions and the equator that a rupture is most likely to occur. At the clinical limbus, the thickness is reduced to approximately 50% and can infrequently result in rupture especially in operated eyes. Likewise, although thick in the area of insertion of the optic nerve, tissue density is least at the margin of lamina cribrosa and can result in tears or avulsions. Pathological processes and iatrogenically induced weakness can predispose the sclera to rupture.

Scleral rupture (Figure 3) is often occult mainly because ophthalmic examination may be limited by ocular media opacities, including hyphema, cataract and vitreous hemorrhage. Signs of occult rupture are visual acuity of perception of light or no PL, reduced ductions, ocular hypotony, hyphema, severe chemosis, abnormally deep or shallow anterior chamber, afferent pupillary defect, vitreous hemorrhage and a large bullous subconjunctival hemorrhage. The only definitive way to rule out scleral rupture is careful controlled exploration of the globe. Eyes with scleral rupture have a high incidence of microbial contamination and are often severely injured internally. Early primary repair becomes the most important step in management.
**Choroidal Rupture**

This can occur directly at the site of impact or indirectly in a contre-coup fashion. Choroidal ruptures occurring anterior to the equator are usually direct injuries while posterior choroidal ruptures are indirect injuries.

The classic posterior choroidal rupture (Figure 4) is a tear in the choriocapillaries and overlying Bruch’s membrane that typically forms concentric to the optic nerve. These breaks are caused by sudden horizontal expansion occurring as a result of anteroposterior compression. These may be associated with intra or subretinal hemorrhage or in some cases may be subtle identified only on angiogram. These ruptures generally heal with scarring and retinal pigment epithelial hyperplasia. Formation of choroidal neovascular membrane is a known late complication.

Direct ruptures form at the site of contusion impact from compression necrosis and tend to be anterior, oriented parallel to ora serrata, most commonly in the inferotemporal quadrant.

When choroidal rupture is accompanied by retinal rupture it is termed ‘retinitis sclopeteria’. This classically results from high velocity missile accidents. Though not strictly a result of blunt injury, this is caused by concussive forces transferred to the globe. Shock waves are transmitted from the object to the adjacent globe with resultant hemorrhagic rupture of the choroid and the retina leading to necrosis and fibrous proliferation.

**Commotio Retinae**

Commotio retinae or Berlin’s edema is a post traumatic retinal condition that may manifest even after mild blunt trauma. It may be observed in the retinal periphery and in the posterior pole. Sipperley and coworkers showed histologically that the clinical retinal changes were a result of structural changes, with disruption of photoreceptor outer segments. (Figure 5 a &b)

Commotio retinæ is characterized by a whitish gray retinal appearance observed several hours after blunt trauma. It may be accompanied by intraretinal hemorrhage or even choroidal rupture. Visual acuity may be normal or profoundly decreased to 20/400. As the white retinal changes resolve visual acuity improves unless severe retinal pigment epithelial damage, choroidal rupture or severe intraretinal hemorrhage has occurred. No effective treatment for commotion retinæ is known.

**Hypotonic Maculopathy**

Manifests as disc edema, choroidal folds and in severe cases choroidal detachment. (Figure 6)

**Post traumatic retinal breaks and dialysis**

Post traumatic retinal holes result from contusion necrosis due to the direct result of coup or contrecoup forces especially if they are situated posteriorly. Post traumatic macular holes also are known to occur due to contusion necrosis resulting in macular cyst formation. Subsequent rupture of the cyst leads to macular hole formation.

Traumatic retinal breaks occur in the periphery when the vitreous is violently shifted away from the retina. These occur in areas of strong vitreoretinal adhesion. The vitreous base, lattice degeneration, old chorioretinal scars and retinal blood vessels are the most common sites of retinal tearing. Superonasal is the most common site.

Retinal dialysis (Figure 7) followed by late retinal detachment can be one of the most devastating visual consequences of blunt injury to the posterior segment. This refers to a break occurring at the ora serrata, whose anterior edge is at the ora serrata and posterior edge is attached to the vitreous base. Trauma is the cause of about 20% of dialysis. This is commonest in the inferior temporal quadrant but is also known to occur in the superior quadrants and a superonasal dialysis is classically secondary to trauma. Retinal dialysis occurs when the vitreous base is avulsed. The retina and vitreous are tightly adherent at the vitreous base, and as the vitreous base is avulsed into the vitreous cavity the retina follows creating a tearing at or near vitreous base. Management of retinal dialysis depends on the presence or absence of retinal detachment.
Figure 7

**Traumatic retinal detachment**

Traumatic retinal detachments account for 10-19% of all phakic retinal detachments. This is associated with inferior temporal dialysis in 31%, superior nasal dialysis in 22%, giant retinal tears in 11% and lattice associated tears in 8%

**Vitreous changes in blunt trauma**

The vitreous can be injured in blunt trauma by disinsertion or hemorrhage. Disinsertion occurs at the vitreous base, optic nerve, retinal vessels, lattice degeneration or scars. Commonest changes are avulsion of the vitreous base (Figure 8) with associated retinal dialysis, posterior vitreous detachment with retinal tear and vitreous hemorrhage. The avulsed vitreous base has the appearance of a hammock or ribbon suspended loosely through the vitreous cavity. Vitreous haemorrhage commonly occurs due to torn retinal blood vessels. Pigment in the vitreous is an indication for careful search for retinal tear or dialysis.

Figure 8

**Optic nerve injury**

The optic nerve is rather resilient to injury from blunt trauma. Injury generally occurs following severe concussive force with concurrent multisystem trauma and brain stem injury. The common causes include motor vehicle or bicycle accidents, falls or physical assault. This has been rarely reported following minor injury. Blunt trauma results in indirect injury to the optic nerve. These kinds of injuries occur when the force of impact is transmitted to the nerve through the bones or by motion of the globe.

Optic nerve injuries can be divided into anterior or posterior types. Anterior indirect injuries involve the intraocular portion. Posterior indirect injuries damage the intracanalicular portion. The intraorbital portion is involved less frequently because this portion of the nerve is not tethered to the globe or the orbit, allowing movement when forces are applied. Anterior injuries are rare and result when the globe suddenly is rotated or displaced anteriorly, such as when the eyes are accidently poked. The most accepted theory suggests that blunt force to the globe causes extreme rotation and sudden strain on the nerve. These forces result in tears or avulsion of the anterior optic nerve at the margin of lamina cribrosa. Ophthalmoscopically this is seen as a defect in the optic disc with or without massive hypotony. Visual loss is usually profound and permanent. A permanent pit like defect is seen in the centre of the optic nerve later with fibrogial proliferation. (Figure 9) Prelaminar injuries are less severe usually and presents with disc swelling and peripapillary hemorrhages. Later this results in varying severity of optic atrophy.

Figure 9

Posterior optic nerve involvement occurs as a result of tethered intracanalicular portion where a 10mm segment of the nerve travels through a confined area. It is here that the nerve gets susceptible to primary involvement with contusion necrosis and in some cases ischemia as the pathogenic mechanism. Sphenoid fractures may be found in as much as 50% of cases with this injury. In these cases impingement on the optic nerve by bony fragments may contribute to optic neuropathy. In addition to the primary involvement, the optic nerve may suffer secondary pathological changes in the form of further necrosis either as a result of pressure effect or vascular compromise. Clinically, this presents as a relative or total afferent pupillary defect with minimal ophthalmoscopic findings.

Thus, blunt trauma can result in varied manifestations in the posterior segment many of which are vision threatening. They require careful evaluation, early recognition, timely, appropriate intervention and continued follow up to ensure a favourable outcome.