I. Introduction

Hyphaema (blood in the anterior chamber) can occur after blunt or lacerating trauma, or after intraocular surgery. Hyphaema can occur spontaneously in conditions such as rubeosis iridis (e.g. Associated with diabetic retinopathy, central retinal vein occlusion, carotid occlusive disease or chronic retinal detachment), vascular tufts at the pupillary margin, juvenile xanthogranuloma, iris melanoma, myotonic dystrophy, keratouveitis (e.g. Herpes zoster), leukemia, hemophilia, thrombocytopenia or Von Willebrand disease. Hyphaema also occurs in association with the use of substances that alter platelet or thrombin function (e.g. Ethanol, aspirin, warfarin).

II. Mechanisms of Haemorrhage and Blood Resorption

Blunt injury is associated with anterior-posterior compression of the globe and simultaneous equatorial globe expansion. Equatorial expansion induces stress on anterior chamber angle structures, which may lead to rupture of iris stromal and/or ciliary body vessels with subsequent haemorrhage. Secondary haemorrhage also termed re-bleeding, may be due to clot lysis and retraction from traumatized vessels. Lacerating injury can also be associated with direct damage to blood vessels and hypotony both of which can precipitate hyphaema. Hyphaema after intraocular surgery can be due to granulation tissue in the wound margin or due to damaged uveal vessels (e.g. from surgical trauma or IOL induced uveal trauma).

In conditions such as rubeosis iridis, juvenile xanthogranuloma, iris melanoma, iris leiomyosarcoma, myotonic dystrophy, and iris vascular tufts, iris vessel fragility itself may predispose to hyphaema. Minor trauma can precipitate hyphaema in this setting. Duke-Elder proposed that hyphaema absorption might occur through the anterior part of the iris. Several groups have shown that erythrocytes leave the anterior chamber via the trabecular meshwork as relatively intact, undamaged cells. Uncomplicated hyphaema usually clear within approximately one week.

III Epidemiology

The mean annual incidence of hyphaema is approximately 17 per 100,000 population. The peak incidence is between ages 10-20 years. The average age of patient is less than 25 years. The majority (80%) of hyphaema patients are males probably because most cases develop after trauma.

IV Clinical Examination and grading of hyphaema

At the baseline examination, a detailed history should be taken about the specific circumstances under which trauma took place and also a general medical history about other diseases (anemia, blood disorders, medications used, and liver or kidney disease).

The eye examination included a detailed examination at the slit lamp, intraocular pressure (IOP) measurement, fundus examination when it was possible (clear ocular media) with a +78 Diopter lens or with the indirect ophthalmoscope. There is no use of the three-mirror Goldmann or any other type goniolens during the initial examination in order to avoid any pressure on the globe and a secondary haemorrhage. Gonioscopy is performed after absorption of hyphaema to check for the presence of a possible angle recession. In certain cases where the patient underwent CT scan, MRI of the orbit or a B-ultrasonograph special care should be taken not to exert pressure on the globe.
Grading of hyphaema is important for study purposes. There are different types of grading for hyphaema. According to one study hyphaema was graded as follows.  

- **Grade 1**: Hyphaema filling less than one third of the anterior chamber (AC)
- **Grade 2**: Hyphaema filling one third to one half of the AC
- **Grade 3**: Hyphaema filling more than half of the AC
- **Grade 4**: Total hyphaema with either red or black blood clots.

Another classification system is as follows:  

- **Grade 1**: those filling up to 25% of the anterior chamber
- **Grade 2**: up to 50%
- **Grade 3**: up to 75%
- **Grade 4**: 76% and over

In one another simple classification system patients were divided into three groups according to the height of hyphaema.  

- **Small hyphaema**: 3-4 mm height
- **Moderate hyphaema**: Reaching the pupillary border
- **Severe hyphaema**: Involving the total volume of anterior chamber

**V Complications:** In general visual prognosis and complications are substantially worse in the setting of total hyphaema as opposed to subtotal hyphaema

**D. Corneal blood staining**

The incidence of traumatic hyphaema- associated corneal blood staining varies from 2-11%. Among patients with total hyphaema, however, the incidence is substantially higher ranging from 33-100% in two studies. Corneal blood staining tends to occur in the setting of larger hyphaema, re-bleeding, prolonged clot duration, sustained increased intraocular pressure, and corneal endothelial cell dysfunction. Corneal blood staining can occur, however, with a less than total hyphaema in the setting of endothelial dysfunction, in the presence of low or normal intraocular pressure. Corneal endothelial damage associated with traumatic disruption of Descemet’s membrane or with mechanical damage induced during surgery can lead to corneal blood staining. Corneal blood staining can cause decreased visual acuity after hyphaema resolution and can cause deprivation amblyopia in infants and children.

Because Read and Goldberg found that corneal blood staining was more likely to occur in patients with a total hyphaema associated with intraocular pressure >25 mmHg and ≥6 days duration, these investigators recommend that one manage such eyes surgically by day 6 if the hyphaema does not resolve below 50%.

The earliest sign of corneal blood staining is a straw yellow discoloration of the deep stroma, which should be distinguished from the light reflected off the surface of the blood clot in the anterior chamber. One clue to the presence of blood staining Vs. reflected light is the presence of greater stromal discoloration centrally than peripherally. Early signs of corneal blood staining include the presence of tiny yellowish granules in the posterior third of the corneal stroma or blurring of the fibrillary appearance of the corneal stroma. Crouch and Crouch believed that these easy biomicroscopic signs precede gross blood staining by 24-36 hours, and they suggest that clot evacuation at this stage can prevent gross staining with corneal clearing in 4-6 months. As indicated above, even if the intraocular pressure is normal, it is important to perform daily slit lamp examination to detect corneal blood staining. The opacity usually clears from the periphery towards the center, and the process can require 2 or 3 years. The blood product protoporphyrin has been identified by Gottsch and coworkers as a phototoxic compound in the anterior chamber of patients with hyphaema and has been demonstrated to photosensitize the endothelium experimentally. Endothelial cell decompensation or degeneration is the earliest event in the pathogenesis of corneal blood staining. Mechanical disruption of the endothelium may play a role in the pathogenesis of endothelial decompensation, but photosensitization of the endothelium by hemoglobin-derived porphyrins in the presence of ambient light may also disrupt endothelial function. For this reason, patching the eyes with longstanding hyphaema may reduce the chance of corneal blood staining.

Pathologically endothelial degeneration and eosinophilic deposits distributed throughout the stroma characterize corneal blood staining. Ultra structural studies reveal that hemoglobin tends to be extra cellular between collagen fibrils and hemosiderin tends to be in the keratocyte cytoplasm. Messmer et al posited the following mechanism for corneal blood staining. First, hemoglobin is released from the erythrocytes in the anterior chamber, diffuses across Descemet’s membrane, and aggregate focally within the membrane as well as within the stromal lamellar. Second, the keratocyte phagocytose and metabolize hemoglobin, producing intracellular hemosiderin. Excess intracellular hemosiderin/hemoglobin induces keratocyte necrosis, with attendant decreased cellularity of the posterior stroma. Third, released hemosiderin is phagocytosed by keratocyte in the anterior stroma. Most clearing occurred from periphery towards the center, and the demarcation between cleared and stained corneal stroma was abrupt both clinically...
and histopathologically. Corneal blood staining clears by diffusion.

**B. Increased intraocular pressure**

Approximately one third of all hyphaema patients exhibits increased intraocular pressure. In the setting of traumatic hyphaema intraocular pressure may be elevated due to the following:

1. Occlusion of the trabecular meshwork by the clot, inflammatory cells, or erythrocyte debris or
2. Pupillary block secondary to a collar button shaped clot involving both the anterior and posterior chambers.

In general larger the hyphaema volume, the greater the likelihood of increased intraocular pressure secondary haemorrhage is often associated with increased intraocular pressure. In the setting of a total hyphaema, a normal or low intraocular pressure should alert one to the possibility of a ruptured globe. An initial period of elevated intraocular pressure can be followed, however, by a period of normal or low intraocular pressure even in the absence of a ruptured globe, provided that a secondary haemorrhage does not occur. This period of temporarily reduced pressure may be due to decreased aqueous humor production and may play a role in predisposing patients to secondary hyphaema, particularly as the normal process of clot lysis proceeds.

The incidence of late-onset glaucoma in eyes with a history of traumatic hyphaema ranges from 0-20%. Glaucoma developing days to years after the initiating injury can arise from damage to the trabecular meshwork (often associated with angle recession), descemetization and fibrosis of the trabecular meshwork, sclerosis of the trabecular endothelium or peripheral anterior synechiae formation leading to secondary angle closure glaucoma. The incidence of angle recession after eye trauma ranges from 20-94%. The possibility of developing glaucoma in an eye with angle recession appears to be related to the extent of angle recession. The greater the circumferential extent of angle recession, the greater the chance of subsequently developing glaucoma, particularly if more than 180° of the anterior chamber angle is involved. If extensive, posterior synechiae which can form as a result of inflammation also can cause secondary angle closure glaucoma. Ghost-cell glaucoma, causes by dehaemoglobinized erythrocyte diffusion from the vitreous cavity in to the anterior chamber weeks to months after a vitreous haemorrhage, can be associated with a khaki coloured hyphaema and is another cause of late onset intraocular pressure elevation after trauma.

Elevated intraocular pressure is routinely managed medically with topical beta adrenergic antagonists or alpha-2 adrenergic agonists. If these medications are inadequate, topical or systemic carbonic anhydrase inhibitors are added. If these measures are ineffective isosorbide, oral glycerin or intravenous mannitol is administered. Pilocarpine is not recommended for these reasons. First, Pilocarpine may increase vascular permeability and promote fibrin deposition in an already inflamed eye. Second, the possibility of iridolenticular adhesions and seclusio pupillae may be greater with a miotic pupil. Third, fundus examination is impaired. Prostaglandins usually are not employed in this setting because of a presumed increase in the inflammatory response.

**C. Peripheral Anterior Synechiae**

Persistence of the hyphaema for more than one week can result in the formation of peripheral anterior synechiae (PAS). The incidence of PAS increased with size and duration of visible hyphaema greater than 8 days. Posterior synechiae also can form presumably synechiae formation is the result of inflammation or clot organization.

**D. Optic Atrophy**

In the setting of traumatic hyphaema, optic atrophy tends to occur as a result of elevated intraocular pressure or due to optic nerve contusion. In a prospective study, Read and Goldberg found that 6% eyes had optic atrophy characterized by pallor without glaucomatous cupping. In 4% eyes transient IOP elevation was noted, and optic atrophy without cupping was attributed to this pressure elevation. In 2% eyes, no period of elevated intraocular pressure was detected. The latter cases may represent traumatic optic neuropathy secondary to short posterior ciliary artery damage caused by optic nerve contusion. The rest of optic atrophy related to elevated intraocular pressure appears to be greater if the pressure is allowed to remain at 50 mmHg or more for 5 days or 35 mmHg or more for 7 days, in otherwise healthy individuals. In these eyes optic nerve head cupping does not develop with optic nerve atrophy as it does in chronic glaucoma patients. Patients with sickle cell disease/trait can develop optic atrophy with smaller intraocular pressure elevations.

**E. Secondary haemorrhage**

Secondary haemorrhage is said to be present if the size of the hyphaema increases or if a layer of fresh blood is noted over the older darker clot in the anterior chamber, or if dispersed erythrocytes appear over the clot after the blood has settled. Total and near total hyphaema, which often appear dark red, may become bright red at the clot periphery as the clot dissolves. This change in colour is due to clot lysis and this should be distinguished from secondary haemorrhage. Rebleeding can cause a substantial increase in the size of hyphaema. For this reason rebleeding can be associated with complications such as increased intraocular pressure, corneal blood staining, optic atrophy, and peripheral anterior synechiae. Considering the relatively high incidence of surgical intervention for complications of rebleeding, the
risks of surgery (including general anaesthesia) may justify the use of a treatment that significantly reduces the incidence of rebleeding. Although some studies report a greater likelihood of secondary haemorrhage with larger hyphaema, others report no clear relationship between the initial size of the hyphaema and the incidence of secondary haemorrhage. Thus, one should consider the use of medications to reduce the likelihood of rebleeding regardless of hyphaema size.

F. Accommodative impairment

In one study 7% patients had reading disability requiring asymmetric spectacle correction of greater than 2.5 Diopter. Thus evaluation of accommodative amplitude may important when following these patients.

VI The Prognosis of traumatic hyphaema

The prognosis of the traumatic hyphaema depends on the hyphaema height its colour, the reoccurrence of the haemorrhage, the time that takes for the anterior chamber to clear the blood, and mostly on the IOP rise and the corneal blood staining. Patients with predisposing factors are associated with a higher risk of complications and should be more closely followed.

Regarding the blood colour as prognostic indicator, light red colour is indicative of a continuous circulation of the aqueous humor and of an efficient oxygen supply in the anterior chamber. On the contrary, dark red or black colour of the blood (due to the transformation of hemoglobin in methemoglobin) shows the discontinuation of the aqueous humor circulation and the lack of oxygen in the anterior chamber, which means the prognosis of hyphaema must be made very cautiously.

VII. Medical Management to prevent rebleeding

A. Pharmacologic Therapy:

1. Antifibrinolytic drugs: In most studies antifibrinolytic agents (i.e. tranexamic acid and E-aminocaproic acid) significantly lower the rate of rebleeding after traumatic hyphaema and also may delay clot resorption. E-aminocaproic acid is a water soluble antifibrinolytic agent that resemble amino acid lysine. Amicar competitively inhibits fibrin clot digestion by occupying plasmin lysine binding site. Also, E-aminocaproic acid competitively inhibits activating substances in plasma that convert plasminogen to plasmin, perhaps by binding to plasminogen and preventing its binding to fibrin, even after activation to plasmin. Tranexamic acid also resembles lysine and is similar to E-aminocaproic acid in mechanism of action. Dosage is 50 mg/kg up to max. 30 gm/daily for 5 days in 4 hourly divided doses. The dose of Amicar must be adjusted for patients with renal failure. Amicar can precipitate renal colic in patients with renal failure and even mild cases of hemophilia. Active intra vascular clotting and known allergy to E-aminocaproic acid are contraindicators to the use of Amicar. Relative contraindications include a history or predisposition to thrombosis, haematuria of upper renal tract origin renal failure and hemophilia. Adverse effects of Amicar are nausea, vomiting or diarrhea. Other side effects includes pruritis muscle cramps, rash nasal stuffiness, arrhythmia and confusional states.

2. Corticosteroids: Trauma induced breakdown of the blood ocular barrier might enhance the diffusion of some plasma proteins into the anterior chamber including plasminogen, thus increasing the risk of secondary haemorrhage. By stabilizing the blood ocular barrier and by directly inhibiting fibrinolysis corticosteroids might reduce the risk of secondary haemorrhage. Systemic prednisolone appears to be as effective as systemic amicar. Both antifibrinolytics and corticosteroids use are not associated with a statistically significant benefit on final usual outcome despite of the fact that both decrease the rate of rebleeding.

3. Conjugated estrogens: E.g. Premarin can increase the prothrombin concentration and decrease antithrombin activity. It can reduce likelihood of secondary haemorrhage.

4. Mydriatic and Miotic agents: Rakusin found no significant difference in the incidence of rebleeding, in the final visual acuity in the rate of clot absorption, or in the incidence of complications, regardless of whether the patient was using a mydriatic, miotic, neither or both. Because patients with traumatic hyphaema commonly have iridocyclitis, it is preferable to prescribe a cycloplegic agent (atropine 1% once per day) to relieve photophobia and to prevent formation of posterior synechiae. Atropine is preferred because once a day dosing reduces the amount of ocular manipulation needed which may reduce the chance of secondary haemorrhage.

5. Aspirin: It seems prudent to avoid aspirin and nonsteroidal anti-inflammatory analgesic medications in this setting.

In summary, there is strong evidence that systemic as well as topical medications including corticosteroids, E-aminocaproic acid and tranexamic acid decrease the risk of rebleeding among patients with traumatic hyphaema. Recommended treatment regime include Topical corticosteroid (e.g. Prednisolone acetate 1% OID) to reduce intraocular inflammation, long acting cycloplegic (e.g. Atropine 1%). In addition either systemic Prednisolone or a systemic amicas is used in most cases. Acetaminophen or codeine is used as analgesics and is better to avoid aspirin and NSAIDs.

B. Bed rest versus Ambulation Management

For most patients, it appears that there is no clear advantage to prescribe bed rest instead of quiet ambulation as long as the environment can be controlled. Children may represent
a subset of patients in whom bed rest may be preferable to ambulation in a hospital setting, if there is a question of control.

C. Effect of eye Patching:
It is recommended that patients with hyphaema wear a metal or hard plastic shield at all times (including sleep) to prevent further trauma to the eye. Gottsch et al suggested that patients with long standing hyphaema who may have prolonged light exposure might be at risk for developing endothelial dysfunction and corneal blood staining. Patching of these patients affected eyes may be prudent.

D. Outpatient hyphaema management:
One should consider outpatient management only if the parents and child were likely to comply with medical recommendations and keep follow up appointments. Similar considerations were to be given to patients with time delay before presentation, penetrating ocular injuries, markedly elevated intraocular pressure and monoclonal status.

VIII. Surgical Management
Rakusin found that the surgically treated cohort had a higher proportion with absorption in one week. The medically treated cohort had better final visual acuity and a lower incidence of complications).

Read and Goldberg and Deutsch et al developed the followed empirical criteria for surgical intervention, Hyphaema evacuation is recommended in the following cases:
1. A patient has sickle cell disease or trait and if the mean intraocular pressure is greater than 24 mmHg over the first 24 hours or if the intraocular pressure spikes repeatedly over 30 mmHg.
2. In non-sickling patients if the intraocular pressure is greater than 60 mmHg for 2 days (to prevent optic atrophy)
3. The intraocular pressure is greater than 25 mmHg with a total hyphaema for 5 days (to prevent corneal blood staining)
4. There is microscopic blood staining
5. The hyphaema fails to resolve to less than 50% of anterior chamber volume by 8 days (to prevent peripheral anterior synechiae formation)

The surgical approach used depends on the clinical setting and to some degree the training of the surgeon.
There are various surgical methods to evacuate hyphaema few are described below:

To lower intraocular pressure quietly, an anterior chamber paracentesis can be performed at the slit lamp under topical anaesthesia if the patient can co-operate. With a sterile lid speculum in place and after sterilizing the ocular surface with topical povidone iodine, a 0.5 inch 30 gauge needle attached to a tuberculin syringe is introduced at the limbus. While the surgeon holds the syringe in place the assistant aspirate the bloody aqueous humor slowly. This approach will not be effective if most of the anterior chamber is filled with clot.

Definitive clot evacuation is done in the operating room. A clear corneal incision is created near the limbus just superior to the horizontal meridian on the side of the surgeon’s non-dominant hand. A bent 23 gauge needle is introduced through this incision for balanced salt solution infusion. The infusion pressure is adjusted to 30-40 mmHg. The vitrectomy probe is introduced into the anterior chamber through a second clear corneal incision near the limbus just superior to the horizontal meridian on the opposite side. The vitrectomy probes cutting port is occluded with the clot. Working at low suction (~50 mmHg) to avoid anterior chamber collapse, the surgeon first attempts to aspirate the liquefied blood. Solid clot is engaged with the probe, drawn centrally and excised by activating the cutting action of the probe. The cutting port is not directed towards the crystalline lens, particularly, while the cutting suction mode is activated. The clot that adheres firmly to the iris is left behind. Clear corneal incisions parallel to the iris are preferred to avoid contact between the intraocular instruments and the iris and crystalline lens. At the end of the procedure, the limbal incisions are closed with 10-0 nylon suture. A peripheral iridectomy is performed if there is a concern regarding the development of pupillary block post-operatively or if the iris prolapses and cannot be repositioned.

Other methods include irrigation and if necessary, aspiration of the anterior chamber blood with simcoe irrigation aspiration probe leaving any firmly adherent clot in the eye. Compared to the scleral tunnel incisions, clear corneal incisions seem less likely to predispose to contact between the instruments and the crystalline lens, particularly if a collar button clot with Pupillary block is present, and the iris-lens diaphragm is displaced anteriorly.

If the intraocular pressure is elevated primarily because of dispersed red blood cells, one can irrigate the anterior chamber.

Another surgical technique used was an ab externo corneal section of 90 under a limbus based flap with preplaced sutures. The clot was removed by a combination of spontaneous extrusion and gently manual expression with a muscle hook. Residual clot was removed with week sponges and the corneoscleral section was irrigated with balanced salt solution.

Viscoelastic evacuation of traumatic hyphaema was described early. The viscoelastic properties of Healonid are used to separate the hyphaema from other ocular tissues and to extrude it through a small corneal incision. Healonid maintains a deep anterior chamber and a stable intraocular pressure. It also protects the lens, cornea, and iris and allows clear observation.

A 1 mm stab incision is made with a keratome shaped
diamond knife on the corneal side of the limbus. This opening is used for the injection of Healonid. The opening is better made on the side to suit the surgeon’s master hand. A similar sized incision is made on the opposite side of the cornea if the hyphaema is fluid. When the hyphaema is clotted, this incision is enlarged to 3 mm. Healonid is injected through a fine cannula. Begin close to the internal opening of the incision. The hyphaema is pushed towards the other side. Angle the cannula up and down to clear all of the hyphaema from the region around the stab opening. If the injection is made slowly, there is no mixing between the Healonid and blood. The intraocular pressure will begin to rise as Healonid is injected. The injection is therefore accompanied by opening of the incision on the opposite side- gently pressure with an iris spatula on the limbal side of the incision. Blood will begin to extrude, and the pressure will remain constant. The Healonid cannula is gradually advanced across the anterior chamber. Clots are separated from the iris or angle by directing flow towards the appropriate position. Perfect visibility through Healonid allows clear observation of the iris and lens and of the depth of the anterior chamber. The cannula can therefore be manipulated inside the eye with safety. The healonid is left in the anterior chamber once the hyphaema has been removed. The cornea is sutured if necessary. Post-operative control of the intraocular pressure is achieved by the use of oral acetazolomide or topical timolol drops. It offers advantages over other methods. The necessary incisions in to the cornea are small and may not require sutures in contrast to the large 180º incision often advocated. The hyphaema, clotted or fluid is readily separated from other intraocular structures and extends through the small exit wound. The sudden fall of the intraocular pressure to zero which must occur after any large incision, and which is probably a major factor in causing a second haemorrhage is thus avoided. The Healonid filled part of the anterior chamber allows perfect observation while protecting the cornea, iris and lens. All manipulations within the eye are therefore made under direct observation in a deep and stable chamber. The danger of instrumental injury to the lens in particular, and the iris and cornea is thus greatly reduced.

**Role of Trabeculectomy**

Trabeculectomy is done uncommonly. Trabeculectomy can be an effective intervention, particularly in patients with total hyphaema, very high intraocular pressure, and particular susceptibility to intraocular pressure-induced damage (e.g. Pre-existing glaucomatous optic atrophy, sickle cell disease/trait). Also if corneal blood staining severely compromises the view of the anterior chamber a trabeculectomy may offer the safest approach to management. A standard technique is used. It may be appropriate to make the ostium some what larger than normal if the retained clot is large.

**Special Situations**

**Sickle cell haemoglobinopathy**

Patients with sickle cell disease or trait have a higher incidence of increased intraocular pressure optic nerve atrophy and secondary haemorrhage is the settling of traumatic hyphaema compared to non-sickle all patients. Fibrinolysis may be enhanced in patients with sickle cell trait which could predispose to secondary haemorrhage. In the setting of sickle cell disease or trait the size of the hyphaema may not be a reliable indicator of the subsequent clinical course. For example, there is a poor correlation between hyphaema size and the ease with which intraocular pressure is controlled. Sickled erythrocytes are less able to pass through the outflow channels of the trabecular meshwork than are normal erythrocytes which are analogous to the inability of “ghost erythrocytes” to pass through the outflow channels. Increased intraocular pressure is poorly tolerated in patients with sickle cell disease as evidenced by the fact that central retinal artery occlusion has followed the formation of small hyphaema in young individuals with sickling haemoglobinopathy. Flow in the central retinal artery of sickle cell patients may be impaired significantly at intraocular pressures greater than 40 mmHg.

These observations have led Goldberg to suggest avoiding medical treatments that promote sickling when managing patients with sickle cell disease/trait and hyphaema. Repeated or excessive dosages of hyperosmotic diuretic agents (e.g. glycerin, isosorbide, and mannitol) should be avoided, as they may cause haemo concentration and increased blood viscosity in the ocular microvasculature. Systemic carbonic anhydrase inhibitors not only promote haemoconcentration but also induce systemic acidosis, which is known to exacerbate erythrocyte sickling. Besides lowering the aqueous humor PH, acetazolamide increases the concentration of ascorbic acid in the aqueous humor and ascorbate may exacerbate the sickling process itself possibly by acting as a reducing agent. Methazolamide creates less systemic acidosis than acetazolamide and hence it is recommended in patients with traumatic hyphaema and sickle cell haemoglobinopathy. The following protocol is recommended in patients with sickle cell disease/trait. Use timolol as the primary topical agent. Add topical brimonidine or apraclonidine if an additional agent is needed. If a further agent is necessary, use topical Dorzolamide before using methazolamide. A hyperosmotic should be used infrequently, for example, only once every 24 hours and as last resort to avoid surgery. Surgical evacuation of hyphaema should be considered at lower intraocular pressures than proposed in the management of hyphaema in the non-sickle patient. 1Deutsc et al have suggested that one consider surgical intervention if the intraocular pressure averages more than 24 mmHg over any consecutive 24 hours period despite
maximum tolerated medical therapy. In addition, if the intraocular pressure increases transiently and repeatedly above 30 mmHg surgery should be considered.

**Clotting disorders:**
It is recommended that in the presence of even minor signs of intraocular haemorrhage the patient should be admitted to the hospital and the deficient clotting factor (or cryo precipitate) should be infused regularly during the high risk period for secondary haemorrhage (i.e., first 5-7 days after the injury). If the patient must undergo surgery to evacuate hyphaema, it may worthwhile to provide replacement therapy sufficient to restore levels of clotting factor levels to 100% of normal during the procedure. Patients with even mild cases of hemophilia may be at increased risk for acute renal failure if treated with E-aminocaproic acid.

**Management of Hyphaema in Children**
Hospital admission for children are recommended if there are concurrent injuries mandating admission; if the hyphaema is large (e.g.: ≥ 50% of anterior chamber volume); if the intraocular pressure is elevated; if the patient has sickle cell disease/trait or clotting diathesis, if there is a time delay before presentation; or if there is concern regarding medication delivery, compliance with activity restrictions, ability to return for follow up, or safety of the home environment. One hyphaema complication unique to the paediatric population is the development of amblyopia, which can occur as a result of corneal blood staining.

The use of Amicar or prednisone is recommended routinely in children with hyphaema. Both the apparent efficacy of systemic and topical steroids in reducing the incidence of secondary haemorrhage and the commonly accepted value of steroids in attenuating the sequelae of intraocular inflammation (which often accompanies ocular trauma) lead to prefer the use of systemic steroids with or without topical steroids, rather than Amicas, in managing children with traumatic hyphaema, independent of whether they are managed as inpatients or outpatients.

**Recommendations**

**Step 1:** Obtain complete history. Ocular (Fuchs, glaucoma, amblyopia etc) systemic, medications and details of injury

**Step 2:** Identify and treat associated injuries and conditions along with complete eye examination

**Step 3:** Medical management. Topical corticosteroid, cycloplegic, IOP lowering agent, analgesic antiemetic agents, rigid shield and patch

**Step 4:** Systemic medication to reduce secondary haemorrhage (Amicar/prednisone./Clotting factors/platelets in case of haematomal abnormalities)

**Step 5:** Daily slit lamp examination. Visual acuity, hyphaema height, IOP, cornea

**Step 6:** Discharge the patient if IOP is satisfactory and hyphaema <1/2 of AC. Also depends on patient compliance

**Step 7:** Surgery if risk of corneal blood stain, risk of optic atrophy or synechiae formation (See above)

**References**