MANAGEMENT OF GLAUCOMA EMERGENCIES

We are grateful to our colleagues at Moorfields Eye Hospital for allowing us to adapt their comprehensive guidelines for Sussex Eye Hospital.

MANAGEMENT OF ACUTE PRIMARY ANGLE CLOSURE

'Acute' angle closure is an ophthalmic emergency and the potential for inadequately treated symptomatic primary angle closure to cause blindness should not be underestimated.

The treatment regime depends on mechanism of angle closure. Ninety per cent of patients with primary angle closure have PB as the underlying mechanism, the other 10% have other mechanisms other than or in addition to PB. The importance of determining the mechanism is that secondary angle closure may be made worse by the routine treatment for primary angle closure. To determine the mechanism of angle closure, the most important question to answer is whether PB (iris bombe) is present or absent. Assess peripheral AC depth first. If PB is present, indentation gonioscopy may lead to the flattening of the peripheral iris but not in the case of secondary angle closure where little change occurs to the iris contour e.g. phacomorphic or closure secondary to choroidal effusions where the iris contour may be flat or convex giving the impression of iris bombe. Then look at central AC depth. If PB is present and the central depth is shallow but asymmetrical then consider secondary causes e.g. lens related such as lens subluxation, phacomorphic glaucoma or posterior segment pathology. If the AC depth is very asymmetrical between the two eyes, then discuss the possible use of atropine with a glaucoma consultant BEFORE using pilocarpine. Inability to open the angle with indentation gonioscopy during an acute attack does not imply that the angle will remain closed after an iridotomy, nor the presence or extent of PAS.

Principles of management

1. Break acute attack
   a) break pupil block (pilocarpine, aqueous suppressants and indentation)
   b) control IOP medically with aqueous suppressants
   c) treat ocular inflammation
2. Prevent further attacks with PIs
3. Detect & control ongoing optic disc and visual field damage

Remember that many of these pts are elderly, small ladies often with other medical problems. Electrolyte disturbances and hypotension can be exacerbated by vomiting, diamox, beta blockers and mannitol. Topical aqueous suppressants (ß blockers) are additive with diamox but take longer to act as their absorption through the cornea is slowed by corneal oedema. Aqueous suppressants can reverse pupil block by opening the angle. A rapid decrease in inflow of aqueous reduces PC IOP so AC
IOP becomes transiently higher which forces the iris back and angle opens. Miotics may be ineffective when the IOP > 50mmHg due to iris sphincter ischaemia and paralysis, but are still able to induce ciliary muscle contraction and cause forward shifting of lens iris diaphragm ‘paradoxical AC shallowing’ compounding pupil block. So intensive pilocarpine should not be used, furthermore it can produce cholinergic toxicity which mimic the features of persistently raised IOP.

**Treatment**

**Immediate**

1. **Diamox** 500mg IV *(if present acutely with symptoms, otherwise it should be given orally, max effect in 2 hrs, can reverse PB by opening angle)*

2. **Pilocarpine 2% stat** *(4% in dark irides)*

3. **Timolol 0.25% stat** *(if no medical contraindications)*

4. **Iopidine 0.5% stat**

5. **Pred Forte 1% stat**

6. **Lie patient supine** permits lens to fall back with vitreous dehydration

7. **Analgesics and antiemetics as indicated**

**Reassess after 60mins**

→ **if IOP elevated**

- consider indenting central cornea gently with 4 mirror gonioprism or a moistened cotton bud, 2-3 cycles of 30 secs on / 30 secs off. This allows the displacement of aqueous from the posterior to the anterior chamber producing a widening of the angle.

→ **if IOP reduced**

Refer to Glaucoma Service for PIs to both eyes.

→ **If IOP remains elevated**

- Perform emergency argon laser peripheral iridoplasty (ALPI) following consultation with Miss Lewis or on call consultant.

**Reassess after 60mins**

→ **if IOP elevated**

- Admit and notify on call consultant or Miss Lewis

**Aftercare**

Following laser iridotomies to both eyes, if the IOP is controlled then patients should be discharged on:
1. **Pilocarpine 2% (4% in dark irides) qid**  
2. **Apraclonidine 0.5% tid**  
3. **Pred Forte 1%** 2 hourly affected eye and qid fellow eye  
4. **Acetazolamide** 250mg qid omit if IOP < 10mmHg

**Clinic**
Patient should be reviewed in outpatients *within 7 days* in Miss Lewis’s clinic (GC) and managed as per same section in laser peripheral iridotomy.

**MANAGEMENT OF ACUTE PRIMARY ANGLE CLOSURE**

*Under no circumstances should a patient be discharged without patent PIs in both eyes unless approved by a consultant.*

Consider/exclude secondary glaucoma: specifically secondary pupil block and neovascular or uveitic processes which may present with very high pressure and corneal oedema.

If anterior chamber depth is very asymmetrical between eyes, discuss the possible use of atropine with a glaucoma consultant BEFORE using pilocarpine.

If the patient presents
- *during working hours* → START Rx & let Miss Lewis or on call consultant know
- *out of working hours, weekends and bank holidays* → START Rx, ADMIT & NOTIFY on call Consultant
**IMMEDIATE TREATMENT**

1. **Diamox 500mg IV** (if present acutely with symptoms otherwise give orally)
2. **Pilocarpine 2% (4% in dark irides)** stat
3. **Timolol 0.25%** stat (if no medical contraindications)
4. **Iopidine 0.5%** stat
5. **Pred Forte 1%** stat
6. **Lie patient supine**
7. Analgesics and antiemetics as indicated

**Reassess after 60 mins**

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**IOP elevated 25mmHg**

Consider indenting central cornea or a moistened cotton bud

2-3 cycles of 30 secs on / 30 secs off

**IOP reduced ≤**

1. **REFER to Glaucoma Service for Laser PIs BE gently** with a 4 mirror gonioscope
2. **Pilocarpine 2% qid**
3. **Apraclonidine 0.5% tid**
4. **Pred Forte 1% 2 hourly**
5. **Acetazolamide 250mg qid** (omit if IOP ≤ 10mmHg)

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**IOP remains elevated**

**Pils**

Argon laser peripheral iridoplasty following consultation with on call glaucoma consultant

**Home following laser**

Review within 7 days in Miss Lewis’s Glaucoma Clinic (GC) for assessment of PI patency, gonioscopy and dilated disc examination

**Reassess IOP after 60 mins**
IOP remains elevated

ADMIT & NOTIFY Miss Lewis or on call consultant
References


BLEB RELATED INFECTION
Miss Maria Papadopoulos, Nov 2008

‘Seriousness is the characteristic of this complication.’ ARRUGA

INTRODUCTION
Bleb related ocular infection (BRI) is a potentially blinding, late complication of glaucoma filtering surgery reported in both intentional and inadvertent filtering blebs. A high degree of vigilance and aggressive treatment are essential in minimising the blinding nature of this complication.

DEFINITION
Bleb related infection refers to a spectrum of disease severity ranging from infection limited to the bleb to that of fulminant endophthalmitis. Blebitis is generally regarded as an isolated bleb infection without clinically apparent vitreous involvement and bleb related endophthalmitis (BRE) as fulminant endophthalmitis in which a vitreous biopsy and intraocular antibiotics are appropriate.

INCIDENCE
The cumulative incidence of bleb related infection has been reported to range from 0.2-9.6% depending on the type of glaucoma operation, with the incidence generally lower for trabeculectomy than other forms of filtering surgery. The incidence of infection after trabeculectomy without antiproliferatives ranges from 0.2-1.8%, it may be higher with antiproliferatives.

ONSET
Onset of infection from time of filtration surgery ranges from months to years.

PATHOGENESIS
The pathogenesis of bleb related infection is believed to involve either direct spread through a ruptured, leaking bleb or transconjunctival migration of bacteria from ocular surface into the eye through a very thin walled filtering bleb possibly aided by the production of exotoxins and endotoxins. Pathogenesis is thought to relate to the virulence of the bacterial flora and their capacity to penetrate the bleb wall. The presence of a bleb epithelial defect is believed to allow less virulent bacteria to easily enter the bleb and anterior chamber. These proposed mechanisms are different to the pathogenesis of acute post surgical endophthalmitis where the organism is thought to be introduced at the time of surgery.
Pathogens

1. Streptococcal species - pneumoniae, faecalis, viridans \( \text{commonest in BRE} \)
2. Haemophilus influenzae
3. Staphylococcus aureus \( \text{*Staphylococci commonest organism} \)
4. Staphylococcus epidermidis \( \text{cultured from infected bleb surface} \)
5. Pseudomonas aeruginosa
6. Moraxella
7. Fusarium species

PRACTICE POINT

*The spectrum of organisms differs considerably from that of early post cataract endophthalmitis.*

PRACTICE POINT

*The same organism may not necessarily be isolated from both the external and intraocular specimens.*

RISK FACTORS

1. Thin-walled, avascular, cystic blebs

Thin walled blebs are thought to be the most common contributing factor to the development of infection possibly because of a reduced physical and immunological barrier to bacteria. The role of the *filtering operation* in the development of these types of blebs has been known for a long time as they were found to be more common after full thickness filtration surgery (e.g. corneoscleral trephine) than after trabeculectomy. The use of *antimetabolites* in glaucoma filtering surgery is associated both clinically and histologically with cystic thin walled blebs. In particular, Mitomycin C (MMC) is a potent inhibitor of fibroblastic proliferation and vascular endothelial cells. As a result of these effects, there is an increased incidence of thin, cystic, avascular blebs following the use of MMC.

2. Inferior bleb location

There is a *four-fold increase in relative risk with inferior blebs*. The incidence for inferiorly located antimetabolite blebs has been reported as high as 13.2%. This may be related to the bleb being bathed in bacteria-rich tear film and less protected by the lower lid leading to bleb exposure and subsequent epithelial drying. In light of this increased risk, if a superior trabeculectomy with antimetabolite is not an option then use alternate methods of treatment including drainage implants or cyclodestructive procedures.
3. **Chronic bleb leak**
Thin, avascular blebs are fragile, easily ruptured and vulnerable to bacterial invasion. A chronic bleb leak has been suggested as a risk factor in the development of delayed infection but a clear causative relationship between them has not been established. A recent case control study found eyes with BRI were 26 times more likely to have bleb leak detected at the time of infection than eyes without BRI. Furthermore, whether leakage leads to subsequent infection or results from localised necrosis after the development of infection cannot be determined. At presentation, bleb leaks are more common in cases of blebitis. The association is less strong with BRE. Antimetabolite blebs are associated with a higher risk of chronic bleb leak but this may be more a feature of small area of treatment and surgical technique that encourages the formation of a focal bleb, delimited by a ‘ring of steel’ rather than just the use of the antimetabolite *per se*.

4. **Bacterial conjunctivitis**
The occurrence of bacterial conjunctivitis in patients with filtering blebs requires conjunctival cultures and treatment with topical bactericidal antibiotics. But even this may not prevent endophthalmitis, so they should be closely observed during and after treatment for the development of intraocular inflammation.

5. **Blepharitis, trichiasis, dry eyes**

6. **Nasolacrimal duct obstruction**

7. **Contact lens use**
8. **Ocular trauma** - accidental/iatrogenic bleb manipulation (needling, auto blood injection)
9. **URTI**
10. **Myopia**
11. **Non-ocular**
capsule

Advanced age - prone to thin walled blebs b/c of thinner Tenon’s capsule

Immunosuppression - DM, malignancy, systemic steroids

Malnutrition

**CLINICAL FEATURES**

**Symptoms**
- redness
- photophobia
- purulent discharge
- blurred VA
- pain

* sudden onset and rapid progression is characteristic of endophthalmitis
* prodrome of a few days is characteristic of blebitis
PRACTICE POINT

Patients who present with a prodrome of less than 48 hours should be closely observed.

**Signs**
- mucopurulent infiltrate ‘white on red’ or opalescent fluid within bleb, +/- leak
- surrounded by intense conjunctival inflammation
- purulent discharge
- variable AC activity
- +/- AC shallowing & hypotony
- +/- vitritis (if vitreous can’t be visualised, then the eye must be treated as having fulminant endophthalmitis and also perform US)

- **If 1+ AC cells consider this to be endophthalmitis and treat it as such irrespective of absence of vitritis.**

**ACUTE MANAGEMENT**

**(i) BLEBITIS**

1. Admit
2. Conjunctival swab - micro / culture / sensitivities
3. Instil povidone iodine 5% into conjunctival sac
4. *Levofloxacin* hourly day and night  
   *Cefuroxime* hourly day and night
5. *Moxifloxacin*** 400mg mane 10 days (warn pt of risk & symptoms of hepatitis - document in notes)  
   *Augmentin* 625mg tds orally 7 days (Azithromycin 500mg 3/7 if penicillin allergy)
6. Topical steroids  
   *Pred Forte* 1% qid
   Shouldn’t be initiated until course of infection clear, usually 24-48 hrs after therapy started
7. All patients MUST BE REVIEWED within 4-6 hours following admission to evaluate progression.

***MOXIFLOXACIN IS CONTRAINDICATED IN CHILDREN AND PATIENTS WITH LIVER DISEASE. USE CIPROFLAXACIN INSTEAD.***

**(i) BLEB RELATED ENDOPTHALMITIS**

1. Admit
2. Conjunctival swab / AC tap / Vitreous biopsy
3. Instil povidone iodine 5% into conjunctival sac
4. Intravit antibiotics  
   *vancomycin* 2mg + *amikacin* 0.4mg or *ceftazidime* 2mg
5. Antibiotics  
   *topical and systemic as above*
6. Topical steroids  
   *Pred Forte* 1% qid
7. Systemic steroids  
   **Consider** *Prednisolone* 1mg / kg 12 hours after admission if no med CIs (liaise with consultant)
Consider formal vitrectomy if significant vitreous involvement.

(ii) BLEB LEAK

Most late onset leaks that are associated with ocular infection do not produce hypotony or AC shallowing, so they may be addressed after the resolution of infection as a separate issue. In general, intervention to correct AC depth in the setting of infection should be discouraged.

**LATE MANAGEMENT**

(i) BLEB FAILURE - medical / surgical management

(ii) BLEB LEAK

The bleb leak may seal following the infection. But if it persists following an episode of bleb related infection then repairing the leak is strongly advised. The optimal management of persistent bleb leaks remains unclear.

**PROGNOSIS**

Long term visual outcome and bleb function vary depending on:

1. **Extent of infection**
   Although some eyes recover useful vision, patients with culture proven endophthalmitis generally have a very poor prognosis. In contrast to eyes with blebitis, most of which achieve vision back to or within one line of pre-infection visual acuity. Correlating final visual acuity outcome with the infecting organism is difficult in some patients as their advanced glaucoma disease limits visual potential. Generally, visual acuity outcomes in bleb related infection are worse than in acute onset endophthalmitis after cataract surgery.

2. **Virulence of organism**
   Bleb related endophthalmitis with Streptococci and gram negative organisms such as Pseudomonas is usually associated with a poor prognosis. Exotoxins released by Streptococci are toxic to the retina and possibly stimulate increased intraocular inflammation. This is contrary to the good prognosis seen in patients with blebitis where most cases are associated with Staphylococci. The reason for the differing prognosis is that blebitis is possibly caused by a less virulent spectrum of organisms than fulminant bleb related endophthalmitis where the relative thinness and leakiness of blebs allows penetration of the bleb wall.

3. **Timing of therapy**
   Blebitis being a precursor of endophthalmitis is more effectively treated at an earlier stage resulting in a better prognosis. While visual acuity outcomes are variable depending on extent of infection, in general, blebs maintain filtration capacity after infection.

**PRACTICE POINT**
Bleb related infection should be treated early and aggressively to maximize visual prognosis.

PREVENTION

Risk factor reduction

1. Surgical technique

   Inferiorly placed blebs should be avoided as should blebs with nasal and temporal extensions within the interpalpebral fissure. Avoid focal, thin, avascular blebs by changing your surgical technique. A diffuse posterior bleb can be achieved by using a fornix based conjunctival flap, treating a large area with antimetabolite, making a large scleral flap whose radial edges do not reach the limbus and placing a combination of releasable, adjustable and fixed sutures as required to achieve posterior flow through the flap. Releasables should always be buried and antibiotics used when sutures are exposed. Furthermore, the antimetabolite should be cautiously and appropriately selected according to the patient’s risk factors for failure.

2. Patient education

   Symptoms, signs and significance of bleb related infection must be made clear to the patient at high risk. Patient should be instructed to seek immediate attention.

3. Prophylactic antibiotics

   The role of prophylactic antibiotics is controversial. They have not been proven to decrease the incidence of infection, instead a recent study suggested that chronic use may be associated with an increased risk of infection. Possibly long term antibiotics may select for more virulent strains however one study found that long term antibiotic prophylaxis did not alter resident conjunctival flora and so it can not be depended on as a fail-safe method to prevent BRI.

   • It is recommended that only in the situation where a patient is unable to reach an ophthalmologist for advice and treatment should they be given a broad-spectrum antibiotic to use until examined promptly by an ophthalmologist.

4. Treat chronic blepharitis, ocular surface and lid disorders

   Advise patient on lid hygiene (+/- topical or systemic antibiotics).

5. Management of chronic bleb leak

   Chronic bleb leak presents a challenging management dilemma. The risks and benefits of proposed intervention should be evaluated based on the specific characteristics of the patient. First, decide whether intervention is necessary depending on previous history of bleb related infection and whether vision is compromised. Intervention is indicated if there has been a previous episode of blebitis or bleb related endophthalmitis. Similarly if vision is substantially compromised from hypotonous maculopathy or optic


**disc swelling then the decision to intervene is straightforward. When vision is not compromised then the decision is more difficult.**

**Secondly, decide between conservative Vs surgical intervention.**

Observation and conservative, non-interventional options preserve bleb function but success is often short lived because of continued upper lid rubbing during blinking against the compromised conjunctival barrier, still leaving the eye at risk of infection. Incisional surgical interventions are usually successful at addressing the problem of leakage but the price may be compromised bleb function and reduced glaucoma control.

In deciding conservative Vs surgical options consider the following:

1. **Perceived risk of endophthalmitis and consequences**
   - **Patient risk factors which make definitive surgical intervention more appropriate are** (i) young age  (ii) exposed bleb  (iii) poor contralateral VA  (iv) poor facial hygiene, bad blepharitis  (v) occupational/hobby contact with dirt or other potential contaminants (vi) inability or lack of motivation to follow advice and act on the symptoms or signs of infection  (vii) lives or frequently travels to remote areas without ophthalmic care.

2. **Bleb leak**
   - **The magnitude of the bleb leak will determine intervention, as a large leak will definitely require surgical intervention. The character of the leak, for example, a ‘sweating’ bleb responds more favourably to conservative measures such as autologous blood.**

3. **Extent of glaucomatous damage and risk of progressive glaucoma damage should the IOP rise following repair.**

**Approaches include:**

1. **Autologous blood** in and around the bleb is appropriate for very small leaks or ‘sweating blebs’. In theory it forms a fibrin scaffold that becomes repopulated with cells which heal the leak. Often it needs to be repeated and so not often performed. (Beware of intracameral leakage and hyphaema).

2. **Compression sutures** to isolate the leak from the rest of the bleb ‘Palmberg’ sutures (8/0 vicryl or 9/0 nylon). This is thought to be a more effective technique for reducing bleb dysaesthesia rather than reducing bleb leakage. These can be combined with autologous blood. It often needs to be repeated.

3. **Bleb revision** involving bleb excision and conjunctival advancement (undermine conjunctiva and separate it from Tenons to minimise ptosis) / rotation or autologous free flap from the inferior fornix. This is the most definitive treatment. Often during the revision, scleral thinning or a full thickness sclerostomy is evident and must also be addressed. Otherwise you will again be faced with the same problem of a leaking bleb in the near future. In these cases a scleral (full thickness defect) or Tenon’s patch (partial thickness defect) graft is necessary and must be thought of pre operatively.
Glaucoma Guidelines  Sussex Eye Hospital
MEDICAL TREATMENT OF OHT & GLAUCOMA

In general, when patients are seen in casualty or in primary care with an intraocular pressure <35mmHg they are not started on any medication until they have been seen in the glaucoma phenotyping and research department. They should have a referral form filled out with the patients contact details and intraocular pressure and disc cupping ratio written on the referral form. They will then be seen within 2 weeks in the glaucoma phenotyping unit. This is not the case if the patient is at risk of central retinal vein occlusion, and if there is any dilemma whether to start the patient on medication this should be discussed with the consultant on call.

Drugs used in treating glaucoma are all licensed as intraocular pressure lowering agents. There is no evidence of other beneficial effects such as neuro-protection or improvement in ocular blood flow sufficient for licensing purposes for any compound.

CLASSES OF DRUGS USED TO TREAT GLAUCOMA

1) Prostaglandin agonists
2) Drugs acting on sympathetic and parasympathetic nervous system
3) Carbonic anhydrase inhibitors
4) Osmotic agents

There is good evidence that lowering IOP prevents progression in ocular hypertension (OHTS) and reduces the rate of visual field loss in glaucoma (EMGT studies).

DRUGS ACTING ON THE SYMPATHETIC & PARASYMPATHETIC NERVOUS SYSTEM

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class of Drug</th>
<th>Preparations available</th>
<th>Ocular actions</th>
<th>Ocular side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pilocarpine</td>
<td>Cholinergic agonist</td>
<td>0.5,1,2,3,4,6%</td>
<td>constriction of ciliary muscle increasing aqueous outflow</td>
<td>miosis &amp; pain on instillation, visual blurring and accommodation</td>
</tr>
<tr>
<td>Eserine</td>
<td>Cholinergic agonist</td>
<td>Withdrawn</td>
<td></td>
<td>as above</td>
</tr>
<tr>
<td>Carbachol</td>
<td>Cholinergic agonist</td>
<td>Withdrawn</td>
<td></td>
<td>as above</td>
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<tr>
<td>Phospholine iodide</td>
<td>Acetylcholine-esterase inhibitor</td>
<td>Withdrawn</td>
<td>increased aqueous outflow</td>
<td>red eye</td>
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<tr>
<td>Adrenaline</td>
<td>α &amp; β receptor agonist</td>
<td>Withdrawn</td>
<td>reduced aqueous production &amp; increased outflow</td>
<td>red eye, pupil dilation, cystoid macular edema in aphakic eyes</td>
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<tr>
<td>Guanethidine</td>
<td>Sympathomimetic</td>
<td>Withdrawn</td>
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<td>symblepharon formation **</td>
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<tr>
<td>Brimonidine</td>
<td>α2 receptor agonist</td>
<td>Alphagan 0.2%</td>
<td>reduced aqueous production &amp;</td>
<td>ocular allergy 10%</td>
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Combigan– with
### Glaucoma Guidelines

**Sussex Eye Hospital**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mode of action</th>
<th>Preparations available</th>
<th>Ocular actions</th>
<th>Ocular effects</th>
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<tbody>
<tr>
<td><strong>Acetazolamide</strong></td>
<td>carbonic anhydrase inhibitor</td>
<td>oral Diamox 250mg Diamox SR</td>
<td>reduced aqueous secretion</td>
<td>none</td>
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<tr>
<td><strong>Dorzolamide</strong></td>
<td>carbonic anhydrase inhibitor</td>
<td>Trusopt 2% Cosopt (Timolol+Dorzolamide) Azarga (Timolol+Brinzolamide)</td>
<td>reduced aqueous secretion</td>
<td>corneal erosions, corneal thickening, red eye</td>
</tr>
<tr>
<td><strong>Latanoprost</strong></td>
<td>Prostaglandin PGF2α agonist</td>
<td>Xalatan 0.005% Travatan 0.004% Lumigan 0.03%, 0.01% Xalatan+Timolol 0.5% Bimatoprost+Timolol 0.5% Travoprost+Timolol 0.5%</td>
<td>increased uveoscleral flow*</td>
<td>increase in iris and eyelash, periorbital melanogenesis, cystoid macula oedema in aphakic eyes</td>
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<tr>
<td><strong>Travoprost</strong></td>
<td>? prostamide activity for bimatoprost</td>
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<td><strong>Bimatoprost</strong></td>
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<tr>
<td><strong>Glycerol</strong></td>
<td>osmotic agent</td>
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<tr>
<td><strong>Mannitol</strong></td>
<td>osmotic agent</td>
<td>IV use only</td>
<td>reduced aqueous secretion</td>
<td>None</td>
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</tbody>
</table>

- About 15% of the aqueous outflow is through the longitudinal fibres of the ciliary muscle into the supraciliary / suprachoroidal space and out through the sclera.
PROSTAGLANDIN AGONISTS

Latanoprost is one of the greatest success stories of medical treatment of glaucoma. Developed in the early 1990’s Latanoprost quickly became the best selling eye drop for glaucoma. Its use led to a sharp decline in surgical treatment for glaucoma in the developed world. Latanoprost replaced Timolol as the drug of choice for glaucoma treatment in the United States of America within three years.

Travoprost and Bimatoprost have similar chemical structures to Latanoprost. Both have similar efficacy and side effects. Bimatoprost is claimed to have slightly different receptor activity and is marketed as a prostamide rather than a prostaglandin agonist and in one study non-responders to Latanoprost did show IOP lowering when switched to Bimatoprost.

Apart from hyperaemia and increased periorbital, iris and lash pigmentation, the commonest ocular side effect is punctate keratitis.

Tafluprost was marketed in the UK in 2009. The product does not contain a preservative.

TOPICAL β ANTAGONISTS

Topical beta antagonists were introduced in 1978 and swept the market because they were very effective at lowering IOP with few local side effects.

Timolol - Timoptol 0.25% and 0.5% bd, Timolol LA od, Nyogel 0.1% (β₁ and β₂)
Levobunolol - Betagan 0.5% od or bd (β₁ and β₂)
Carteolol - Teoptic 1%, 2% (β₁ and β₂) & partial agonist with intrinsic sympathomimetic act (ISA)
Betaxolol - Betoptic Suspension 0.25% (β₁ only)

Lower IOP by blocking β receptors on the ciliary processes and reducing aqueous secretion. Betaxolol lowers IOP less effectively. There are no β₁ receptors on the ciliary processes and it is thought to act non selectively in high concentration. Beta receptors are presumed to be present in outflow channels but appear to have little effect.

Well tolerated topically. Mild local anaesthesia is inherent property of beta antagonists.

Levobunolol is most likely to cause sensitivity - due to sodium metabisulphate in preparation therefore not alleviated by preservative free version. All commercial preparations (except Timolol LA) contain benzalkonium chloride and preservative toxicity is more common than sensitivity to the drug. Timolol LA is preserved with benzododecinium bromide.
ALPHA ADRENERGIC PREPARATIONS

Alpha adrenergic agonist preparations include $\alpha_1$ and $\alpha_2$ agonists such as adrenaline and dipivefrine which have been withdrawn, only $\alpha_1$ and $\alpha_2$ agonist Apraclonidine (Iopidine) and the selective $\alpha_2$ agonist Brimonidine (Alphagan) are available.

This class of drugs is most likely to cause a late follicular conjunctivitis especially dipivefrine (Propine), Brimonidine, Apraclonidine. If the eye becomes red, even after months of treatment, it is reasonable to assume that ocular allergy has developed and the eye drop should be stopped or substituted.

PRACTICE POINT

An allergic conjunctivitis may occur even after months of treatment. The drug should be stopped and an alternative substituted.

CARBONIC ANHYDRASE INHIBITORS

Topical preparations are Dorzolamide (Trusopt) and Brinzolamide (Azopt). Both cause metallic taste and may have sulphonamide-like systemic side effects.

Acetazolamide is poorly tolerated causing depression, poor appetite, hypokalaemia in acute dosage. Serum potassium and a full blood count should be checked after one week of treatment. With chronic treatment potassium supplements are necessary.

ANTICHOLINERGIC DRUGS

Pilocarpine is the only drug in this class now available for treating glaucoma. Pilocarpine causes mild redness, pain on instillation for 20 minutes and increased accommodation with visual blurring. With chronic treatment the iris becomes fibrosed and the pupil small, making subsequent cataract surgery difficult as well as exacerbating the symptoms of cataract.

<table>
<thead>
<tr>
<th>Preparation</th>
<th>Systemic side effects</th>
<th>Drug interactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pilocarpine</td>
<td>vomiting and confusion in overdose</td>
<td>may reduce effectiveness of anticholinergics</td>
</tr>
<tr>
<td>Timolol (Timoptol)</td>
<td>exacerbation of asthma and COAD, impotence, nightmares, hypotension and bradycardia</td>
<td>verapamil - prolongs A-V conduction additive to other hypotensive agents</td>
</tr>
<tr>
<td>Levobunolol (Betagan)</td>
<td>as above but fewer CNS effects</td>
<td>as above</td>
</tr>
<tr>
<td>Carteolol (Teoptic)</td>
<td>as for timolol but fewer respiratory side effects, same cardiac effects</td>
<td>as above</td>
</tr>
<tr>
<td>Brimonidine</td>
<td>dry mouth lethargy, respiratory</td>
<td>monoamine oxidase</td>
</tr>
</tbody>
</table>
Glaucoma Guidelines  
Sussex Eye Hospital

<table>
<thead>
<tr>
<th>(Alphagan)</th>
<th>failure in infants</th>
<th>inhibitors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latanoprost (Xalatan)</td>
<td>theoretical risk of inducing abortion</td>
<td>none</td>
</tr>
<tr>
<td>Bimatoprost (Lumigan)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Travoprost (Travatan)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tafluprost (Taflutan)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dorzolamide (Trusopt)</td>
<td>metallic taste, blood dyscrasias, poor appetite, impotence, depression</td>
<td>as for sulphonamides</td>
</tr>
<tr>
<td>Brinzolamide (Azopt)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

IOP lowering of commonly used glaucoma drops  
(Van der Valk et al Ophthalmology 2005 Jul; 112(7):1177-85)

<table>
<thead>
<tr>
<th></th>
<th>Peak mmHg</th>
<th>Trough mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>5 (9-1)</td>
<td>5(10-0)</td>
</tr>
<tr>
<td>Brinzolamide</td>
<td>17 (19-15)</td>
<td>17(19-15)</td>
</tr>
<tr>
<td>Dorzolamide</td>
<td>22(24-20)</td>
<td>17(19-15)</td>
</tr>
<tr>
<td>Betaxolol</td>
<td>23(25-22)</td>
<td>20(23-17)</td>
</tr>
<tr>
<td>Brimonidine</td>
<td>25(28-22)</td>
<td>18(21-24)</td>
</tr>
<tr>
<td>Timolol</td>
<td>27(29-25)</td>
<td>26(28-25)</td>
</tr>
<tr>
<td>Latanoprost</td>
<td>31(33-29)</td>
<td>29(32-25)</td>
</tr>
<tr>
<td>Travoprost</td>
<td>31(32-29)</td>
<td>29(32-25)</td>
</tr>
<tr>
<td>Bimatoprost</td>
<td>33(35-31)</td>
<td>28(29-27)</td>
</tr>
<tr>
<td>Tafluprost</td>
<td>no data in literature as of Feb 2010</td>
<td></td>
</tr>
</tbody>
</table>

SIDE EFFECTS OF EYE DROPS

Systemic
Systemic side effects with topical therapy are common because eye drops are absorbed directly into the circulation, by-passing first pass metabolism in the liver. Two drops of timolol may equate to a 10mg oral dose, the type of dose that produces significant blood pressure lowering in the elderly. Drops may pass down the nasolacrimal duct, to the back of the nose and into the lungs, which probably explains why respiratory side effects are so common with non-selective topical β antagonists. Brimonidine and latanoprost have only been used widely in the last three years and familiarity with side effects is less than with topical beta antagonists.

PRACTICE POINT
Systemic side effects of topical treatment are common. Any unusual symptoms on commencing eye drops such as lethargy, poor appetite or depression should be considered as possible side effects and treatment stopped or substituted to assess the response.

Local
All eye drops will cause stinging and burning depending on the pH of the preparation and the state of the tear film prior to instilling the eye drops.

EFFICACY OF EYE DROPS TO TREAT GLAUCOMA
Although the introduction of Timolol was considered a major therapeutic advance it was soon realised that the drops were dangerous in people with asthma and there were 32 deaths reported to the Federal Drug Administration in the USA between 1978 and 1985. In 1995 a paper published showed unrecognised respiratory impairment in 30% of patients over the age of 60 taking Timolol. Surveys showed that 20% of people taking topical beta antagonists attending eye clinics were also prescribed inhalers for chronic obstructive airways disease so that in total about half of elderly people taking topical beta antagonists were having respiratory problems related to eye drops. These findings coincided with the development of several new preparations for treating glaucoma; Dorzolamide, Brimonidine and Latanoprost.

**COMBINATION THERAPIES**

Combining two IOP lowering drugs and delivering them in one bottle has the attraction of convenience but pharmacological difficulties can be impossible to overcome. Combining a prostaglandin agonist with a topical carbonic anhydrase inhibitor in one bottle of eye drops has not proved pharmacologically possible.

*Fixed combination therapy with latanoprost and timolol was introduced in Europe in the 2000 with hopes that convenience and better compliance would improve clinical outcomes but results of treatment have been disappointing with less intraocular pressure reduction than using the two drops separately and confusion as to whether it should be administered as a morning or evening dose.* Several preparations of Prostaglandin agonist/Timolol fixed combinations have been marketed and the fixed combination Latanoprost/Timolol (Xalacom\textsuperscript{®} Pfizer Laboratories) has been the most extensively studied. Fixed combination Prosta/Timolol combinations are more expensive than the cost of the two drugs separately. Dispensing costs may however be reduced by using the fixed combination product. *Fixed combination Timolol/Dorzolamide (Cosopt) has proved as effective as using the two drugs separately.*

**Additional IOP lowering – with concomitant or fixed combination therapies**

**DRUG INTERACTIONS**

<table>
<thead>
<tr>
<th>Eye drops</th>
<th>Potential drug interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latanoprost</td>
<td>None known</td>
</tr>
<tr>
<td>Beta antagonist</td>
<td>Systemic β blockers</td>
</tr>
<tr>
<td></td>
<td>Quinidine</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
</tr>
<tr>
<td>Dorzolamide or brinzolamide</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Brimonidine</td>
<td>Monoamine oxidase inhibitor eg furazolidone, isocarboxazid, phenelzine, selegiline and tranylcypromine</td>
</tr>
<tr>
<td>Iopidine</td>
<td>None known</td>
</tr>
<tr>
<td>Pilocarpine</td>
<td>None known</td>
</tr>
</tbody>
</table>

**COMPLIANCE**

Compliance may be defined as the correct following of (medical) instructions, in this case the correct timing and application of topical medication. Poor compliance is common among glaucoma patients. One study using especially constructed dropper bottles that timed the application of medication showed how irregular patients were in instilling drops 3-4 times a day, and how this was improved with a bd medicine. It has been found that compliance improves with better communication with the patient, and with single rather than multiple bottles. In addition there have been several recent studies demonstrating that drugs administered once rather than twice a day are continued with for a longer period (persistency). These results suggest that medical treatment be restricted to as few bottles as possible and administered once or twice a day.

Many patients find it very difficult to instill eye drops, and if dependent on someone else to do so, may find a regime of even once daily treatment difficult to maintain. Aids are available to assist with the instilling of drops and can be obtain from Sr Smith.

The treatment regime needs to be as simple as possible and safe with minimal local and systemic side effects, a difficult thing to achieve.

**PRACTICE POINT**

Compliance depends on:
- Simplicity of treatment regime
- Memory
- Manual dexterity
- Lack of topical side effects
- Lack of systemic side effects
A single drug should be started and its effectiveness at lowering eye pressure and any side effects should be assessed within 4 weeks. If there is no IOP response the drug should be stopped and another tried. If there is a satisfactory IOP drop but insufficient to meet the target pressure then a second drug may be added. It is rarely necessary or sensible to use more than two types of eye drops, as compliance will be poor and the effect on quality of life unacceptable.

**GLAUCOMA AND SYSTEMIC DISEASES**

**PRACTICE POINT**

**GLAUCOMA AND SYSTEMIC HYPERTENSION**

It is best to avoid topical \( \beta \) antagonists for elderly patients taking other blood pressure lowering drugs as they may precipitate hypotension. The effectiveness of lowering IOP by a topical \( \beta \) blocker is reduced in patients taking the same class of drug orally.

If patients are hypertensive and have glaucoma, then it is possible to consider treating both conditions with a single medication: a cardioselective \( \beta \) blocker such as atenolol, in which case avoid a topical \( \beta \) blocker.

**GLAUCOMA AND CHRONIC OBSTRUCTIVE AIRWAYS DISEASE**

If the patient is known to have COAD including asthma, topical \( \beta \) antagonists should not be prescribed for glaucoma. If there is no such history, then peak flow, blood pressure and pulse should be measured. If there is more than a 12% fall in peak flow after one month of treatment topical \( \beta \) antagonists should be stopped. If in the course of time breathlessness develops, topical \( \beta \) antagonists should be discontinued for three weeks before starting chronic inhaler therapy as this may become unnecessary when the effect of the eye drops has worn off.

**GLAUCOMA AND DEPRESSIVE ILLNESS**

Most of the commonly used glaucoma therapies can exacerbate depression: In one large study no association was found between prescription of \( \beta \) antagonist and clinically diagnosed depressive illness. Eye drops which do not pass the blood brain barrier and therefore have fewer CNS effects are Carteolol (Teoptic) and Apraclonidine (Iopidine). Apraclonidine has a high rate of ocular allergy and may possibly lose its effectiveness at lowering IOP over time. Brimonidine may interact with several antidepressant drugs as well as causing depression itself and should be avoided. Acetazolamide causes malaise associated with poor appetite and depression.
GLAUCOMA AND IMPOTENCE

Topical β blockers may cause impotence. If patients are taking sildenafil (Viagra) or related drugs, stop beta antagonists and use an alternative agent and inform the patient and their GP.

APPENDIX

NICE Guidance

Economic evidence for OHT treatment in adults:

- Beta-blockers are more cost-effective than prostaglandin analogues in patients with IOP >21 – 25 mmHg and CCT 555 – 590 μm in patients under the age of 58.

- Prostaglandin analogues are more cost-effective than beta-blockers in patients with IOP >21-25 mmHg and CCT < 555μm until the age of 58, and in patients with IOP >25 – 32 mmHg and CCT < 555μm until the age of 77.

NICE Guidance for OHT and glaucoma treatment

- Offer people with OHT or suspected COAG with high IOP treatment based on estimated risk of conversion to COAG using IOP, CCT and age.

- Do not treat people with suspected COAG and normal IOP.

- Check that there are no relevant comorbidities or potential drug interactions before offering medication.

- Offer alternative pharmacological treatment (a prostaglandin analogue, beta-blocker, carbonic anhydrase inhibitor or sympathomimetic) to people with OHT or suspected COAG and high IOP who are intolerant of the current medication.

- Offer alternative pharmacological treatment (a prostaglandin analogue, beta-blocker, carbonic anhydrase inhibitor or sympathomimetic) to treated patients with OHT or suspected COAG whose IOP cannot be reduced sufficiently to prevent the risk of progression to sight loss. More than one agent may be needed concurrently to achieve target IOP.

- Offer a preservative-free preparation to people with OHT or suspected COAG and an allergy to preservatives only if they are at high risk of conversion to COAG (IOP more than 25 and up to 32 mmHg and CCT less than 555 micrometres; or IOP more than 32 mmHg).
GUIDELINES FOR PRESCRIBING FOR GLAUCOMA, OCULAR HYPERTENSION, IN PREGNANCY AND CHILDREN

Guideline development group, Feb 2010

REASON FOR THE GUIDELINE

Glaucoma is one of the commonest diseases of old age and one of the major causes of blindness worldwide. In the developed world the mainstay of treatment is eye drops. In the past five years a number of new medications have been introduced and there is a need for guidance as to their appropriateness, effectiveness and relative safety. One third of all outpatient visits to ophthalmology clinics are for glaucoma assessment. Important studies have been published improving the evidence base for treating glaucoma and ocular hypertension. The Ocular Hypertension Treatment Study (OHTS)\(^1\) and the Early Manifest Glaucoma Trial (EMGT)\(^2\) have provided good evidence for the effectiveness and treatment and improved our understanding of who should be treated. This has prompted the need to produce guidelines for treatment of ocular hypertension and glaucoma.

The National Institute for Health and Clinical Excellence (NICE) produced evidence based guidance for glaucoma management in April 2009. (see page 37)

OBJECTIVES OF THE GUIDELINE

The objective of the guideline is to improve glaucoma prescribing practice and offer safer treatment whilst reducing the complexity of treatment regimens. An essential part of the guideline is that patients are given more information about their treatment and written advice about to use their eye drops, how to gain further supplies and who to contact if problems arise. (See patient information leaflet)

CLINICAL CONDITIONS TO WHICH THIS GUIDELINE APPLIES:

Chronic open angle glaucoma and ocular hypertension

Several classes of drug can be used to treat chronic open angle glaucoma and ocular hypertension. If such treatments fail laser or surgery can be offered with a high chance of success in controlling the disease. If patients do not consent to alternative treatments such as medicine or laser then additional eye drops should be prescribed, in excess of those recommended by the guideline sufficient to control intraocular pressure.

The recommended management in these guidelines should improve patient safety and compliance with treatment and ultimately help preserve their eyesight.

The guidelines do not apply to cases of acute or secondary glaucoma.
LEVELS OF EVIDENCE
The highest level of evidence is based on systematic reviews of the effectiveness of interventions for glaucoma. Published systematic reviews, Rossetti et al\textsuperscript{3} and Maier et al\textsuperscript{4} are referred to in the guideline.

Well designed and conducted randomised controlled trials provide the next level of evidence and these are sited where relevant. Of particular relevance are the OHTS and EMGTS.

Observational studies are sited for the side effects of medical treatment (Diggory et al\textsuperscript{5}, Kirwan et al\textsuperscript{6})

Recommendations on the "maximum" medical therapy were formulated by consensus among the group.
GUIDELINES FOR PRESCRIBING FOR GLAUCOMA, OCULAR HYPERTENSION, IN PREGNANCY AND CHILDREN

This guideline applies to prescribing for ocular hypertension and chronic open angle glaucoma. Prescribing for low tension glaucoma, acute angle closure and secondary glaucoma are not included in this guideline.

OCULAR HYPERTENSION (OHT)

Treatment will usually be started if
- IOP > 29mmHg (taking into account corneal thickness - with an unusually thick cornea treatment may not be necessary)  
- IOP > 21mmHg and two or more risk factors for progression to glaucoma

Other risk factors to be taken into consideration are increasing age, family history and decreased perfusion pressure.

The decision to treat ocular hypertension should be made only by the Glaucoma Service.

Eye drops are the only treatment and should achieve a 20% reduction of intraocular pressure. No more than two medicines should be prescribed.

PRIMARY OPEN ANGLE GLAUCOMA (POAG)

- Eyes with typical glaucomatous field loss and concordant signs of optic disc cupping and raised intraocular pressure.
- Eyes with progression of optic disc cupping and raised intraocular pressure.
- Raised intraocular pressure along with a nerve fibre layer defect or optic disc/cup ratio asymmetry of > 0.2 suggestive of neuronal loss in the absence of disc size asymmetry.
- An IOP reduction of 20% or ≤ 18mmHg should be the target. This may be modified in respect of disease severity and life expectancy.
- Initially a single medication, usually a prostaglandin agonist, should be prescribed whatever the presenting IOP.
- A post treatment visit should be made within 4 weeks to assess response to treatment.

The Glaucoma Service should normally start treatment. If in the unusual circumstance that urgent treatment is indicated the doctor making the diagnosis will prescribe and an appointment made for the service within 4 weeks.
Principles for choosing a drug for treating for glaucoma and OHT in adults

1) Systemic safety and drug interactions
2) Efficacy
3) Compliance
4) Topical tolerability
5) Cost

Maximum medical therapy

Maximum medical therapy is three topical agents – one separate and one combination. More than this should trigger offering either laser or surgery. (Consensus)

Choice of medicines

First line therapy

Prostaglandin agonists

There is no evidence of increased effectiveness of prostaglandin agonists versus beta antagonists in terms of preventing visual field loss. However prostaglandin agonists are undoubtedly safer than β blockers and are probably more effective at lowering IOP. Therefore they should be used as first line. There is no consistent evidence of any difference between the three commercially available preparations and no good evidence for not using the cheapest option.

Second line therapy

β antagonists

Are the best topically tolerated eye drops but should only be prescribed where there is no history of bronchospasm. Peak flow should be recorded prior to prescribing and at the next outpatient visit. If peak flow falls by 12% or more the drops should be stopped. Patients should be told to stop the drops immediately if they develop wheezing or other beta blocker related complications such as heart block. Once daily preparations help compliance and are currently the preferred option eg Nyogel (Timolol 0.1%) or Timolol LA 0.25%. Beta antagonists should be instilled in the morning. If a twice-daily dose is given the second dose should be 12 hours after the early morning dose.

Alternatives to β blockers

Carbonic anhydrase inhibitors are the safest third line option and as effective as betaxolol. There is no evidence of different effectiveness between the products (Brinzolamide and Dorzolamide) and the cheapest option should be prescribed.

Brimonidine is less effective than β blockers and has a 14% rate of follicular conjunctivitis. Patients should be specifically warned about this. For short-term use lopidine is safer systemically but its effectiveness in lowering IOP may reduce over time.

Combination therapies

Combination therapies available are:
Prostaglandin agonist plus once daily β antagonist (eg. Xalacom)
β blocker plus carbonic anhydrase inhibitor (eg Cosopt)
β blocker plus adrenergic agonist (Combigan)

All combination preparations contain timolol 0.5%.

A lower dose of Timolol – either Nyogel 0.1% or Timolol LA 0.25% can be used once daily in the morning with a Prostaglandin agonist at night with equal or better effect in lowering IOP over 24 hours.

Beta blocker and carbonic anhydrase inhibitor or a β blocker plus alpha adrenergic agonist should be used as third line treatment when use of a prostaglandin agonist with a β blocker is insufficient to achieve the desired target IOP.

The combination preparation should be used particularly when there are compliance difficulties - eg district nurses or carers need to come to the patient’s home to apply eye drops.

PRACTICE POINT
Prostaglandin / β blocker combination drops are only dispensed when prescribed by a consultant.

PRESCRIBING IN PREGNANCY AND IN NURSING MOTHERS
Physiological IOP falls in 3rd trimester by about 15%. Therapeutic dilemmas arise due to risks of:
- Impaired fertility
- Maternal toxicity
- Foetal toxicity
- Teratogenicity
- Harmful effects on the nursing infant

This is an unusual clinical problem but likely to increase as older women have children. *It must be made clear that nothing is proven to be safe.* The patient may choose to stop treatment particularly in the first trimester. *Patients should be advised about punctual occlusion as it can significantly reduce systemic absorption.*

Effects in pregnancy and lactation of common glaucoma drugs

<table>
<thead>
<tr>
<th>Agent</th>
<th>Effect on Placenta</th>
<th>Crosses placenta</th>
<th>Secreted in breast milk</th>
<th>FDA risk category</th>
</tr>
</thead>
<tbody>
<tr>
<td>Timolol</td>
<td>N/K</td>
<td>yes</td>
<td>Yes</td>
<td>C*</td>
</tr>
<tr>
<td>Levobunolol</td>
<td>N/K</td>
<td>yes</td>
<td>unknown</td>
<td>C</td>
</tr>
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<td>Carteolol</td>
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<td>Yes</td>
<td>C</td>
</tr>
<tr>
<td>Medicine</td>
<td>N/K</td>
<td>N/K</td>
<td>N/K</td>
<td>C</td>
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<tr>
<td>---------------</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
<td>---</td>
</tr>
<tr>
<td>Metipranolol</td>
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<td>N/K</td>
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<td>C</td>
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<td>Epinephrine</td>
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<td>D?</td>
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<td>N/K</td>
<td>D**</td>
</tr>
<tr>
<td>Mitomycin</td>
<td>N/K</td>
<td>N/K</td>
<td>N/K</td>
<td>D**</td>
</tr>
</tbody>
</table>

* Classified as maternal medication usually compatible with pregnancy by American Academy of Paediatrics Committee on Drugs.

** Teratogenic effect in animals well established – **contraindicated in pregnancy**

**Suggested pathway for management**

1st line: consider ALT

2nd line: β blocker (timolol & betaxolol are concentrated in breast milk up to 5x serum levels, consider weakest concentration eg Nyogel)

3rd line: Pilocarpine (long history of its use)

4th line: Topical CAI

5th line: Filtration surgery without antimetabolites
**PREScribing IN CHIlDHOOD**

Many limitations of studies assessing the safety and efficacy of medication in paediatric glaucoma exist. All eye drops and systemic treatments may cause side effects in children who are at higher risk than adults. Infants and children often cannot communicate the side effects which may differ from those in adult eg a β antagonist may induce asthma presenting as a nocturnal cough rather than wheezing.

When prescribing for children:
- individualise treatment for each child taking into account their age, general health, type of glaucoma and known efficacy and safety profile of each drug
- use the lowest dose possible
- use the minimum number of medications
- minimise systemic uptake by punctual occlusion and blotting excess drops
- warn parents of potential systemic side effects

**First line therapy**

Beta antagonists are the first line treatment if there are no contraindications. Nyogel (Timolol 0.1%) is the drug of choice or Timolol LA 0.25% mane.

**Second line therapy**

In children Dorzolamide has similar efficacy to Betaxolol but greater safety. Acetazolamide should be used cautiously in a dosage of 5-10mg/kg very six hours in the form of crushed tablets or elixir on a very short term basis prior to surgery.

Prostaglandin agonists role in paediatric glaucoma is unclear but appear to be well tolerated. Some papers suggest that its efficacy is reduced in children as compared to adults. Heterochromia in children has been reported. They are less effective in Sturge Weber patients. Patients with JOAG may respond particularly well and should be considered first line in these patients.

Apraclonidine is theoretically safer in children than Brimonidine and its use should be considered when beta antagonists are contraindicated. **Brimonidine should not be used at all in children under 6 years of age.**

Pilocarpine 0.5-2% every 6-8 hours is used in primary congenital glaucoma and may lead to an improvement in symptoms prior to surgery. Postoperatively it may enhance aqueous outflow and prevent anterior synaechiae formation. It may also be indicated for aphakic glaucoma. Pilogel may cause less side effects and be better tolerated.
SUMMARY

FOR ADULTS WITH OHT OR POAG

1st line
Prostaglandin agonist*

2nd line
PG agonist + once daily β blocker**

3rd line
PG agonist + β blocker / CAI or β blocker + Alpha 2 agonist

FOR PREGNANT OR NURSING MOTHERS

1st line
β blocker

2nd line
Pilocarpine

3rd line
Topical CAI

FOR CHILDREN

1st line
Once daily β blocker (preferably Nyogel or Timolol LA)

2nd line
β blocker + CAI

3rd line
β blocker + CAI + PG agonists

* Avoid PG agonists for patients with uniocular disease because of cosmetic problems (heterochromia)
** Avoid β blockers in patients with chronic obstructive airways disease. Always ask about inhaler use.

All patients should be given verbal and written information about their treatment, including instruction on how to instil eye drops, punctal occlusion, where to obtain supplies of eye drops and what to do if side effects occur.

PRACTICE POINT

- New prescription for eye drops are dispensed by the Moorfields NHS Trust pharmacy
- Repeat prescriptions should be supplied through the patients GP
- The prescriber has an obligation to ensure that the patient and their GP have been informed of any new medications or changes to medications
References


PRIMARY ANGLE CLOSURE  
Mr Paul Foster, Feb 2010

Primary angle-closure (PAC) is an anterior segment disease caused by anatomical disproportion. The defining characteristic is contact between iris and trabecular meshwork sufficient to cause a significant reduction of aqueous outflow (whether permanent or transient). PAC is a risk factor for glaucomatous optic neuropathy, which is rapidly progressive in comparison with primary open-angle glaucoma. Population surveys suggest that untreated angle-closure glaucoma blinds about half those affected. Most cases are NOT symptomatic.

CLASSIFICATION AND NONMENCLATURE

A clear, logical framework for describing the mechanism of a disease, and the functional implications of damage to tissues, is essential in managing the condition effectively. The old classification scheme based on symptoms (acute, intermittent, subacute, chronic,) is no longer adequate. A more appropriate classification is that based on signs to help identify the stage of disease and mechanism of closure.

STAGING

Primary angle-closure suspect (PACS) or “occludable angle” (OA)  
= gonioscopically visible contact between iris and trabecular meshwork (TM)

Primary angle-closure (PAC)  
= PACS/OA + either  
  • pigment on trabecular meshwork (with no other explanation)  
  • peripheral anterior synechiae (PAS) (with no other explanation)  
  • raised IOP (> 21 mm Hg)

Primary angle-closure glaucoma (PACG)  
= PAC + glaucomatous optic neuropathy (disc +/- field changes)

MECHANISMS OF ANGLE CLOSURE

1. Pupil-block (main mechanism)  
2. Peripheral iris crowding (includes plateau iris syndrome, and prominent last iris roll)  
3. Lens induced  
4. Causes behind the lens
Categories 3 and 4 are “secondary” forms resulting from other pathological processes. As the position of the anterior lens surface determines the depth of the anterior chamber, and lens thickness increases with age (as does lens opacity), all cases of primary angle-closure are to a certain extent “lens induced”. The term, ‘phacomorphic glaucoma’ should only be used when there has been a sudden event precipitating angle-closure such as dislocation or phacomorphic change (i.e rapid swelling of a mature, white lens). Causes behind the lens are many and varied. They include those which are presumed to act axially such as aqueous misdirection (malignant glaucoma) and intraocular haemorrhage. Rotation of the ciliary body has a more peripheral site of action and results from uveitic processes (eg. VKH) or other causes of supra-ciliary effusion.

**ASSESSMENT OF THE DRAINAGE ANGLE**

*van Herick limbal chamber depth (LCD) estimation*

This is a rapid, non-contact method of predicting the appearance of an angle on gonioscopy. A very fine, very bright beam of light is shone perpendicularly onto the temporal limbus. There is no benefit in examining the nasal LCD as well. Aim to position the beam so that it creates an optical cross-section of the most peripheral part of the cornea through which a clear view is possible, and also the underlying anterior chamber. Estimate the depth of the LCD as a % of peripheral corneal thickness (PCT). The performance of the test in identifying people with narrow angles and established angle-closure in a high-risk population is given in a BJO paper that includes standard photos of the grades.¹

When performing the test, finish by swinging the beam to and fro across the peripheral 3 mm of the cornea to examine the divergence between iris and cornea.

<table>
<thead>
<tr>
<th>Categories</th>
<th>Theoretical range</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0%</td>
<td>0</td>
<td>Contact</td>
</tr>
<tr>
<td>5%</td>
<td>1-10%</td>
<td>Narrowest imaginable gap</td>
</tr>
<tr>
<td>15%</td>
<td>11-20%</td>
<td>&lt; ¼ PCT</td>
</tr>
<tr>
<td>25%</td>
<td>21-30%</td>
<td>= ¼ PCT</td>
</tr>
<tr>
<td>40%</td>
<td>31-50%</td>
<td>&gt; ¼ and &lt;¼ ½ PCT</td>
</tr>
<tr>
<td>75%</td>
<td>51-99%</td>
<td>&gt;½ and &lt; 1 PCT</td>
</tr>
<tr>
<td>100% +</td>
<td>100%+</td>
<td>&gt;¼ 1 PCT</td>
</tr>
</tbody>
</table>

**Central anterior chamber depth measurement**

Measurement of central AC depth is being considered as a method of screening populations for angle-closure.² Axial biometry (ie. IOLMaster) is useful when planning the management of cases, especially when considering lens extraction. If an eye falls into the nanophthalmic range (axial length < 20 mm) request a B-mode ultrasound specifying measurement of scleral thickness.
Gonioscopy

This is currently the gold-standard for assessment of the angle. You should use a Goldmann one or two mirror gonioscope as standard. The patient should be well anaesthetised with benoxinate, or amethocaine if they are very sensitive. Make sure the ambient illumination is as low as practically possible. Use a short (1mm), narrow, bright beam of light that is kept well away from the pupil. Use a vertical beam, horizontally offset to examine superior and inferior angles. Use a horizontal beam that is vertically offset (tilt the illumination column) for the nasal and temporal angles. When recording your findings, it aids clarity if a written description of what was observed is given. When using a derivative system, it is important to understand the different characteristics that may be examined, and the conventions for recording them. The two most widely used are the Scheie and Shaffer schemes, compared in Table 1. The Shaffer scheme is preferable to the Scheie (structures seen) in characterising the angle in most cases of angle-closure. The most complete scheme, as it records most characteristics, is the Spaeth scheme. This records: geometric angle width between TM and peripheral third of iris (i.e. Shaffer); iris profile (steep, regular, concave or plateau) and apparent and true level of iris insertion (i.e. Scheie).

Table 1. A comparison of the Scheie & Shaffer gonioscopic grading schemes

<table>
<thead>
<tr>
<th>Scheie Grade</th>
<th>Description</th>
<th>Risk of Closure</th>
<th>Shaffer Grade</th>
<th>Angle width</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td>Wide open</td>
<td>Impossible</td>
<td>4</td>
<td>35-45°</td>
<td>Wide open</td>
</tr>
<tr>
<td>I</td>
<td>Sl. Narrowed</td>
<td>Impossible</td>
<td>3</td>
<td>20-35°</td>
<td>Wide open</td>
</tr>
<tr>
<td>II</td>
<td>Angle apex not visible</td>
<td>Possible</td>
<td>2</td>
<td>20°</td>
<td>Narrow</td>
</tr>
<tr>
<td>III</td>
<td>Posterior ½ of trabeculum not visible</td>
<td>Probable</td>
<td>1</td>
<td>10°</td>
<td>Extremely Narrow</td>
</tr>
<tr>
<td>IV</td>
<td>No structures seen</td>
<td>Closed</td>
<td>0</td>
<td>0°</td>
<td>Closed</td>
</tr>
</tbody>
</table>

* The two schemes are presented as approximately equivalent. This is a generalisation, but is valid in most circumstances.

The modified Spaeth scheme should be used to describe:
- geometric angle width = degrees between surface of TM and adjacent peripheral iris
- iris profile- steep, regular, concave or angulated (plateau)
- level of true iris insertion
- presence, height and circumferential extent of PAS
- presence of pigment on the TM. Differentiate iris stromal pigment blotches from granular or diffuse pigment originating from the iris pigment epithelium.
Geometric angle width should be assessed using the Shaffer system and written in Arabic (not Roman) numerals to indicate degrees (i.e. 0, 10, 20, 30, 40).

**PRACTICE POINT**
Gonioscopy is mandatory at:
1. First assessment
2. First clinic visit following laser procedures aimed at altering the angle configuration
3. Any clinic visit after starting or stopping pilocarpine
4. Annually in phakic PAC patients

**LASER PERIPHERAL IRIDOTOMY**

*The aim of laser peripheral iridotomy (PI) is to alleviate pupil block. One adequately sized iridotomy is sufficient.*

Consent
Informed consent should cover the following issues. The procedure is the essential first step in effectively managing angle-closure. Generally it is a very low risk procedure. *The commonest adverse event is a transient pressure rise.* This may be prolonged in cases with trabecular meshwork damage (best estimated from pre-treatment IOP and extent of PAS). A small number of patients notice a change in their vision. This is often difficult for them to describe, but is probably related to glare. Anecdotally, glare symptoms are most common and pronounced if the iridotomy is positioned at the level of the lid margin, because of the prismatic effect of the marginal tear strip. People with PI's in the interpalpebral fissure may notice glare as well. *It is our policy that PI's are positioned at 12 o'clock underneath the upper lid, and not at the level of the lid margin.* About 1% of patients with an iridotomy that is well-covered by the upper lid notice deterioration in the quality of their vision. Bleeding from the iridotomy & blurring of vision are fairly common, but are typically transient side-effects, lasting less than 24 hours. Patients taking Warfarin should have had a recent blood test (within 1 week) confirming INR < 3.0.

Around a third of all patients need additional treatment. This may take the form of topical or surgical management, or laser iridoplasty for persistent appositional closure after a PI. In all patients with thick brown irises (where the radial fibres of the peripheral iris cannot be see because of a thick iris stroma), continuous wave laser (i.e. the “argon” class of lasers) should be used to pre-treat at the site of the iridotomy. Retinal burns have been reported following argon iridotomy, and therefore macular burns are possible. People having CWL pre-treatment should be warned of this, although they can be reassured that the combined argon-YAG approach we use minimizes the possibility of this occurrence.
**Preparation**

Record past medical and ophthalmic history. All patients should be examined and their best-corrected visual acuity recorded, their IOP measured in both eyes, limbal chamber depth (van Herick) and gonioscopic characteristics recorded if this has not already been done in clinic.

**Premedication**

Apraclonidine (0.5%) and Pilocarpine (2% in blue eyes, 4% in brown eyes). These should be used at least 30 minutes before, with a second dose immediately before starting treatment. Omit Apraclonidine if there is history of ischaemic heart disease.

**Procedure**

1. Check consent and follow guidelines as set out in Laser Safety document.
2. Ensure the patient understands the procedure and explain what they can expect to hear and feel.
3. Anaesthetize the eye with tetracaine, apply a Wise or Abraham’s iridotomy contact lens.
4. Check the defocus is set to zero.
5. Look for iris crypts or thin areas and treat an area as peripheral as possible, but still having an adequate view through clear cornea between 11-1 o’clock.
6. Use the lowest power possible to achieve a patent iridotomy. Start with lower power shots and increase as required. Careful focussing of the laser enhances the effect of the treatment considerably. Most eyes should require single 0.8 to 1.5 mJ shots, expecting a maximum total power of around 50 mJ. For thick brown irises that have had argon laser pre-treatment, settings and power consumption should be similar. If you encounter any bleeding, gentle pressure will help this to stop. If you have problems seeing because of pigment clouds, stop and wait for 10 minutes.
7. Enlarge the iridotomy circumferentially up to 200 microns diameter.
8. Verify by direct inspection that you have completed the iridotomy through the iris pigment epithelium.
9. **Record: total power, number of shots, location treated.**
10. All patients should receive Prednisolone 1% (Pred Forte) hourly for 24 hours (taking a break through the night), and then 4 times a day until seen in clinic 1 week later.

Leave patient on all topical treatment.

**Argon pretreatment for thick brown irises**

*This is particularly helpful for Chinese and African people.* It is very useful in other Asian people and Caucasians with a thick iris, and those with end-stage angle-closure glaucomatous optic neuropathy. You can use either the argon, or frequency doubled YAG lasers for this.

1. Activate the laser as per the YAG
2. Use a Wise or Abrahams contact lens.
3. The treatment is given in two phases;
i. start with low power shots (80-130 mW, 0.05s, 50 microns) to treat a confluent area of iris stoma in a rosette pattern, to produce a soft pitting or a tiny adherent bubble. You should need about 15-20 shots to do this. The aim is to prevent large, adherent bubbles forming in the second phase.

ii. increase the power (700-750 mW, 0.1s, 50 microns), and apply another 10 -20 shots to produce a punched out crater down to the radial muscle fibres and vasculature. If there is any charring or popping, reduce the power.

4. Complete the iridotomy with a few shots of YAG.

5. In difficult cases (typically African patients, and those who need more than 50 shots) consider aborting the procedure and listing for a surgical iridectomy.

Aftercare
Intraocular pressure should be measured at least one hour after treatment.

If IOP > 30 mm Hg
- 250mg diamox orally stat
- 125mg diamox tds for 2 days

If IOP > 40 mm Hg
- diamox and topical treatment as appropriate

A response to therapy must be demonstrated before discharge.

All patients should receive Prednisolone 1% (Pred Forte) hourly for 24 hours (taking a break through the night), and then 4 times a day until seen in clinic 1 week later. In addition, write up all additional glaucoma medication for both eyes. In the acute setting, leave patient on all topical treatment and diamox until seen in clinic.

Clinic
All patients are seen 1 week later in clinic for assessment of PI patency, gonioscopy and dilated disc examination. Retroillumination through the PI should not be obscured by stromal strands. If one side is definitely patent but the fellow eye is in doubt, compare gonioscopic findings. Often what you find is that the eye with the questionable PI is associated with a narrower angle in which case it is worth enlarging the PI. Stop steroid unless there is evidence of continued inflammation. If the IOP is raised and there is anterior segment inflammation, swap to a topical NSAID. In the acute setting, Pilocarpine should be discontinued at 1 week and the gonioscopy repeated 3-4 weeks later to assess angle configuration. Furthermore, in the acute setting maintain steroids until the inflammation has resolved and slowly
Glaucoma Guidelines  

Sussex Eye Hospital

wean antiglaucoma drops as clinically indicated, *ceasing each a few days prior to next review.*
PERIPHERAL LASER IRIDOPLASTY

The aim of iridoplasty is to induce contraction and compaction of the peripheral iris, drawing the iris away from the trabecular meshwork, and creating more space in the peripheral anterior chamber. Iridoplasty can be used in the management of symptomatic and asymptomatic cases (i.e. “acute” and “chronic”) angle-closure. A recent audit suggests that patients with established PAS are at risk of an extension of PAS after iridoplasty. **DO NOT list patients with PAS for iridoplasty without discussing this with a consultant.**

Consent

Informed consent should cover the following issues. Generally iridoplasty is a very low-risk procedure, with a lower side-effect profile than PI. **The commonest adverse event is a transient pressure rise, or a dull ache which may persist for up to one week.** A small number of patients notice a change in their vision. This is often difficult for them to describe, but is probably related to altered accommodative or pupil function. The most significant adverse reaction encountered to date has been a dilated pupil, unresponsive to light, causing photophobia and glare. This appears specifically related to burns, which are contiguous, and hence should not occur provided the correct technique is used. The procedure is successful (opens an appositionally closed angle) in about 50% cases in our patients. The other 50% may need additional treatment. This may take the form of topical pilocarpine or surgical treatment (lens extraction). Corneal burns are possible, and more often occur when treating symptomatic “acute” cases. They are very rare in elective treatment.

Preparation and premedication

This is performed in exactly the same manner as for iridotomy. Ensure that patients do not have PAS. The inflammation induced by iridoplasty will, in some cases cause an extension of PAS. **Under consultant guidance,** some people with low, early (sawtooth) PAS can be treated, provided they are given pilocarpine qid for 2 weeks post laser, to splint the angle open. These people need more intensive steroids following laser.

This procedure is difficult, and should only be attempted by experienced laser users, and after discussion with a consultant.

Procedure

1. Check consent and lock the door.
2. Ensure the patient understands the procedure, and explain what they can expect to hear and feel.
3. Anaesthetize the eye with tetracaine, apply a Wise or Abraham’s iridotomy contact lens (contact lenses are available to borrow from Sister’s office in casualty).
4. Any continuous wave laser used in pan-retinal photocoagulation may be used to perform iridoplasty. Different classes of lasers vary somewhat in their efficacy relative to power, and there is substantial variation in power uptake between eyes. As a general rule, you should start with a low power and increase until the desired effect is achieved. Starting from 100 mW, the
desired response is usually achieved at between 180 mW and 300 mW. Pulse duration 0.5 to 0.7s, and spot size 500 microns.

5. A “full” treatment requires about **15-20 shots around the entire circumference**, each burn being placed about 2 to 3 aiming beam widths from the area of discoloration marking the previous shot. The ideal end-point is a brisk contraction of the iris stroma, without charring or a pop (if these occur, turn the power down). In acute cases even 90-180 degree circumference can be sufficient. It is usually easiest to treat the inferior half of the iris.

6. The aiming beam should be crisply focused in a regular circle. Varying the direction of gaze of the subject often helps clear visualization of the area to be treated. Most often, directing gaze away from the quadrant being treated improves the view.

7. All patients should receive Prednisolone 1% (Pred Forte) hourly for 24 hours (taking a break through the night), and then at least 4 times a day (as clinically indicated) until seen in clinic 1 week later.

**Leave patient on all topical treatment.**

The procedure is the identical for treatment of “acute” cases. The view may be improved a little using topical glycerine to clear a steamy cornea. The AC should be examined carefully to determine where the cornea and iris are in contact. **These areas should not be treated.** If there is 360° peripheral iridocorneal apposition, start the treatment more centrally, and as the burns pull open the angle, you can rapidly spiral the treatment into the extreme periphery.

**Aftercare**

This is exactly the same as for cases having laser PI. In addition, any cases with PAS should be given Pilocarpine (2% in blue eyes, 4% in brown eyes) to use qid for 1 week.

**Clinic**

All patients are seen 1 week later in clinic and re-gonioscoped. Stop steroid unless there is evidence of continued inflammation. If the IOP is raised and there is anterior segment inflammation, swap to a topical NSAID. If the patient is on Pilocarpine, it should be discontinued at 1 week, and the gonioscopy repeated 3-4 weeks later to assess the effect of iridoplasty on angle configuration.

**PRACTICE POINT**

In the acute setting, a PI must be performed at the earliest opportunity after laser iridoplasty, that is, within hours of IOP normalization.
MEDICAL MANAGEMENT OF ANGLE-CLOSURE

The literature informing the strategy of medical management in angle-closure is extremely sparse. If a patient presents with symptomatic pressure elevation and a closed angle (acute angle closure), the first priority is to control the IOP and symptoms medically. This is best achieved with aqueous suppressant including systemic acetazolamide, topical beta-blockers, and topical alpha-agonists. When the IOP has been reduced slightly, topical pilocarpine is given to try to open the angle. (Refer to Management of acute angle closure.)

The definitive method of opening the angle remains laser (iridotomy +/- iridoplasty) and occasionally surgery (e.g. lens extraction). If a patient has appositional angle-closure following a laser iridotomy and iridoplasty, it is appropriate to evaluate the benefit of topical pilocarpine. In blue eyes, try pilocarpine 1% tid or bd, and 2% in brown eyes. While pilocarpine drops often cause brow ache, and a variable induced myopia with dimming of vision, long-acting pilocarpine gel is better tolerated, and has the advantage of a single, night-time dosage regime. Pilocarpine use should be carefully considered in patients with pseudo-exfoliation. Chronic miotic therapy is indicated only if it favourably alters the angle configuration after laser iridotomy so gonioscopy following commencement of Pilocarpine is essential. Phakic patients with non-occludable angles after PIs will still need to be followed up because the angle may narrow again over time with lens enlargement. Annual review for assessment including gonioscopy is adequate.

In patients with raised intraocular pressure in chronic angle-closure following a laser iridotomy, prostaglandin analogues have been shown to outperform beta-blockers in IOP control and should be consider first line treatment followed by β blockers if not contraindicated. The two factors that determine successful treatment are the presence of > 6 clock hours of PAS extending across the TM and the presence of glaucomatous optic neuropathy. If either of these are present, then medical and laser treatment are unlikely to control the disease satisfactorily and so a trabeculectomy with antimetabolites should be considered.

SURGICAL MANAGEMENT OF ANGLE-CLOSURE

A randomised clinical trial of surgical iridectomy versus laser iridotomy identified no difference in IOP control and visual acuity at 3 years post treatment. Surgical iridectomy should be performed in African and Afro-Caribbean patients in whom a laser iridotomy has been difficult. In the acute setting, if a surgical iridectomy is required and extensive synechial closure is present, consider primary filtering surgery. Providing trabecular function is reasonably intact any patient with angle-
closure and a visually significant cataract can be very effectively managed by cataract extraction and IOL implantation.\textsuperscript{15-17} 

\textit{Intractable acute angle closure} poses a serious therapeutic challenge. There is no consensus on how to manage these patients after medical treatment, corneal indentation, argon laser peripheral iridoplasty or pupilloplasty (which may be better as minimises risk to endothelium) have failed to break the block. Surgical options, which have high complication rates, include surgical PI (if laser PI not possible), cataract extraction with goniosynechialysis\textsuperscript{18} and trabeculectomy whose success rate is poor\textsuperscript{19} even with antiproliferatives. More recently, cyclodiode has proved an effective temporizing measure before lensectomy in these cases.

When filtering surgery is necessary, it is important to avoid post op AC shallowing because of the risk of precipitating \textit{malignant glaucoma} (occurs in 2-4\% of eyes undergoing surgery for angle closure glaucoma). Consider using an AC maintainer intraoperatively and atropine post-operatively. When it occurs, the full-blown syndrome (axial AC shallowing, high IOP and patent PI) is readily apparent, but a disease spectrum exists. In the early stages of the disease, \textit{partial or incomplete malignant glaucoma} may be overlooked and is characterized by the presence of an IOP that is inconsistent with other clinical findings e.g. high IOP with a formed bleb. Not missing this early stage is important because that’s your best chance of relieving the attack with medical treatment alone.
PROTOCOL FOR LASER THERAPY
Mr Ian Murdoch / Miss Wendy Franks, 2000

TRABECULOPLASTY

Preparation
1) Iopidine 0.5% or 1% drops should be instilled up to half an hour prior to treatment.
2) Benoxinate or amethocaine is instilled into the conjunctival sac.
3) A single mirror non-indentation gonioscopy mirror is applied using 2% hypromellose.

Settings
40-50 burns
50 microns
300-850 mWatts

Procedure
1) Laser is applied to the pigment of the anterior trabecular meshwork over 180 degree at the junction of pigmented and non pigmented TM.
2) A round aiming beam and burn should be produced as shown in the diagram on the left. An oval beam and burn as on the right is less desirable.
3) Enough energy should be used to blanche the pigment in the angle (diagram on the left below). If a gas bubble forms, the energy is too high (central diagrams), if the burn is too posteriorly placed, peripheral anterior synechiae form (diagram on right below).
4) The **area treated, number of burns and power** should be recorded in the notes together with any unusual findings / occurrences.

5) The **IOP should be checked one hour after treatment** and appropriate action taken if found to be raised (see below).

6) The patient should use a minimum of **predsol 0.3% qds** for 5 days post treatment.

7) A follow-up appointment should be made for an absolute minimum of **6 weeks** after laser treatment or sooner if an IOP spike or other complication was encountered during treatment.

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**CAPSULOTOMY**

**Preparation**

1) **Iopidine 0.5 or 1%** drops should be instilled.

2) **Tropicamide is optional.**

3) After instillation of **benoxinate or amethocaine** the capsulotomy lens is applied using **2% hypromellose.**

4) A **capsulotomy contact lens** must always be used.

**Settings**

- single burst
de-focussed by 1 unit on the machine (0.2mm)
**power 0.5-1.2mJ** should be applied starting away from the centre of the lens.

**Procedure**

1) The **IOP should be checked one hour after treatment** and appropriate action taken if found to be raised.

2) The patient should use a minimum of **predsol 0.3% qds** for 5 days post treatment.
3) A follow-up appointment for **2 weeks** should be made, with refraction as appropriate.

4) **DIODE LASER OF THE CILIARY BODY**

Cyclodiode laser treatment may only be administered in a laser safe area. All doors and window shutters must be closed. Laser hazard warning signs should be posted on all doors. Staff within the laser safety area should wear appropriate laser safety glasses. **It is the responsibility of the operating surgeon to ensure the safety of those working in the area.**

**Anaesthesia**

Diode laser ablation is painful, topical anaesthetic is not acceptable, either general anaesthesia or local anaesthesia (retrobulbar, peribulbar or sub-Tenon's) should be employed with possible anaesthetic assistance.

- **Full anaesthesia should be demonstrated before commencing therapy.**

**Procedure**

1) **Avoid** areas of: *subconjunctival haemorrhage* as it will interfere with laser penetration and conjunctival burns will occur, *pigmentation and scleral thinning* due to scleromalacia or previous surgery as perforation can occur.

2) The **ciliary body should be transilluminated** by applying a transilluminator during treatment.

3) The probe should be positioned over the ciliary body, usually by resting the heel at the anterior margin of the ciliary body. The probe applies the laser burn 1.2 mm from the limbus but the instrument may need to be positioned further back if the ciliary body is more posterior.

4) The sclera should be indented as this improves energy penetration.
5) An initial test burn of **1000mW x 1500msecs** should be applied. If pain occurs, further anaesthetic should be given before proceeding.

6) The appropriate number of burns as prescribed by the consultant should be applied **avoiding the 3 and 9 o'clock positions** to prevent ciliary nerve damage.

7) A default of **# 40 1500mW x 1500msecs** should be used if not specified.

8) If popping occurs the power should be reduced.

9) Post op **Pred Forte 6-8x day** for 1/12 should be prescribed.

10) **Pre op medication should be continued until OP review** and then weaned as appropriate.