European Glaucoma Society Terminology and Guidelines for Glaucoma 2nd Edition 2003

OHTS
Ocular Hypertensive Treatment Study
5 Year Study

9% Untreated converted to POAG (10%)
4.4% Treated converted to POAG (5%)

90% of untreated did NOT convert

Hence it would be handy if we had predictive factors to see who will convert and who won't.
Predictive Factors

1. Baseline Age
2. Horizontal and Vertical CD ratio
3. PSD
4. IOP
5. CCT – thinner corneas, higher risk (this is strictly not true in my opinion, it simply means the IOP reading is artefactual. Thicker corneas will read higher (but will not be actually higher), whereas thinner corneas will read low but may in actuality be higher.

The CCT, IOP and Age link in with the NICE guidelines. OHT and Suspect COAG we treat prophylactically :

1. Everyone with IOP over 32 regardless of age or CCT
2. 25 to 32 up to age 80 with CCT less than 555 (with PTG)
3. 21 to 25 up to age 65 with CCT less than 555 (with PTG)
4. 25 to 32 up to age 60 with CCT 555 to 590 (with BB)
5. Do not treat if
    a. CCT greater than 590 and IOP up to 32
    b. CCT 555 to 590 but IOP up to 25.

Treatment is effective

Conversion could be reduced from 10% to 5% (50% reduction in conversion) with treatment.

Disc monitoring is important
IOP MUST be adjusted for CCT
Not all OHT need to be treated

Treat moderate risk Patients only (This also correlates with the NICE guidelines)

1. Age
    a. The first NICE treatment category is 555-590 up to 32mmHg and up to 60 years of age.
    b. Suggesting that younger people with OHT are obviously far more likely to convert.
    c. Once 60 (and if Px never converted) the option NOT to treat can be considered but of course we wouldn't know if the Px would not have converted if NOT treated so we would need to empower the Px and monitor very carefully to ensure conversion does not occur
2. Medical status
3. Life Expectancy
    a. NICE even suggests that IOP up to 32mmHg may not be considered necessary to treat in an over 80 year old since life expectancy is not long.
4. Treatment benefit

Conversion to POAG does not necessarily equate to a reduced QoV
Treatment should be based on probability of reducing QoV, which is based on Risk or progression
Risk evaluation guided by Conversion. Low Risk profile – NO treatment as long as there is meticulous follow-up.

CIGTS
Collaborative Initial Glaucoma Treatment Study

4 Years Medical versus Surgical

VF progression did NOT differ between cohorts
VF equal and MD equal between groups

Even though the IOP reduction was higher in Surgical group

VA was also equal at end of study
QoV also equal between groups at the end
Cataracts higher in Surgical Group (17%) versus Medical Group (6%)

CNTGS
Collaborative Normal Tension Glaucoma Study

Treated arm 20% progressed
This cohort either 1. Not at Target IOP or
2.IOP independent disease process
80% survived

Untreated arm 60% progressed
40% survived

Over 50% of the untreated group showed no HARMFUL VF progression over 5 to 7 years

Reducing IOP is beneficial <u>(For patients at Risk)</u>
So what would constitute an 'at risk' Px? (From EGS – see later - and IP Caledonia NTG Case Record)
FOR NTG (EGS)(See later)

1. Disc Haemorrhages FOR NTG (EGS)
2. Migraine, Raynauds, Vasospastic Phenomenon JUST
3. Systemic Hypotension/Nocturnal Dips THINK
4. Silent Myocardial Infarct EVERYTHING
5. Cerebral Infarcts VASCULAR
6. Hypotensive shock / Severe Blood Loss

GENERAL RISK FACTORS FOR GLAUCOMA (EGS) (See later)

1. IOP (OBVIOUSLY NOT APPLICABLE)
2. Age
3. Race (Blacks)

4. Vascular Risk Factors (SAME AS NTG ABOVE)

5. Low Diastolic Perfusion Pressure

While not necessarily inclusive, the risks of disease progression identified Drance et al 2001 and Anderson et al 2003, were

1. Female gender
2. Presence of disk haemorrhages
3. Migraine (and probably all vasospasm or vascular dysregulation)
4. Race

as the only clearly identifiable predictive markers for progression.

A 30% reduction in IOP reduced the rate of VF progression
BUT many untreated patients did NOT show progression
IOP does play a role in progression for SOME NTG patients

AGIS
Advanced Glaucoma Intervention Study

Average IOP >17.5 showed greater progression than if Average IOP was <14
Level of Progression increased with Time
IOP fluctuations seemed to correlate with increased progression

If IOP was <18 100% of time checked - No progression
If IOP was < 18 at 75% or less checks - Progression

LOW IOP + LOW FLUCTUATIONS = REDUCED VF PROGRESSION

Reduce IOP – Reduce VF progression

EMGTS
Early Manifest Glaucoma Treatment Study

25% reduction in IOP = 50% reduction in progression
Conversely – progressed in 50% of patients even with 25% reduction in IOP
Treatment helped ALL groups (age etc)

But

Progression varied significantly with individuals

Individuals should be monitored more closely in the first few years than is commonly done

Progression was reduced with Greater initial IOP reduction (greater the reduction, greater the help)

Risk of progression was reduced by 10% per very 1mmHg reduction in IOP

Some patients did not progress anyway

Patients at lower risk could be left untreated and monitored (must be meticulous)

Results do NOT imply that all Glaucoma patients should receive MAXIMUM treatment

Some patients do not progress even without treatment

Hence the NICE guidelines of treatment of COAG

Target IOP	Progression	Management	IOP Check	Review
Reached	No	No Change	IOP N/A	6-12mths
Reached	Yes	Change treatment	1-4 mths	2-6mths
Reached	No(uncertain)	No change	IOP N/A	2-6mths
No	No	Change treatment	1-4 mths	6-12mths

I must say I do not agree with this. It may be that the original target chosen was too low. Monitor more regularly, I would suggest 2-6 months rather than the 6-12, yes, but change management(?) why? If there is NO progression). They are basing this on the note that perhaps only tensions were checked – but if not at target then for sure this would elicit another field!! Then if truly no progression review in 2-6 months.

No	Yes	Change treatment	1-4mths	2-6mths

Far more frequent checks that previously done.

OVERALL

1. Reducing IOP is beneficial for NTG and POAG
2. The lower the IOP achieved the better VF protection
3. Reducing IOP will NOT be of benefit to ALL
4. Majority of OHT (90%) did not convert
5. A 20% reduction in IOP may not be enough to prevent conversion
6. CCT is essential for OHT
7. CCT of limited value with POAG – more reliant on ON, RNFL, VF

8. Large variation between individuals as regards level of IOP reduction and VF progression
9. It may therefore be OK to monitor some (low risk) to establish rate or progression
10. A large decrease in IOP necessary in Advanced Disease (40-50%!!!!)
11. Disease progression increases with time
12. Large initial reductions in IOP has a favourable influence on later progression
13. Progression does not necessarily equate to reduced QoV
    a. The aim of treatment is therefore not necessarily 'No Progression' but progression at a rate that will not endanger QoV.

RISK FACTORS

OHT

Baseline Age (Life Expectancy)
Medical Status
Vertical & Horizontal CD *(PF – in other words disc appearance)*
PSD
IOP
CCT

Evidence of Progression
Likely to reduce QoL

NTG

Disc Haemorrhages
Migraine, Raynauds, Vasospastic Phenomenon
Systemic Hypotension/Nocturnal Dips
Silent Myocardial Infarct
Cerebral Infarcts
Hypotensive shock / Severe Blood Loss

GENERAL RISK FACTOR CONSIDERATIONS

MAJOR RISK FACTORS - EGS

1. IOP
2. Age
3. Race (Blacks)
4. Vascular Risk Factors
5. Low Diastolic Perfusion Pressure
    a. Low Diastolic Perfusion Pressure however is a significant risk
    b. Diastolic Perfusion Pressure is the pressure gradient between IOP and Diastolic BP.
        i. Diastolic Perfusion Pressure = Diastolic BP – IOP
        ii. Example DPP (64) = 120/80 – 16mmHg
        iii. If it goes below 55mmHg high risk of poor vascular perfusion and hence glaucoma

There needs to be a balance between BP and IOP. Optic Nerve will be compromised if blood perfusion is reduced. TWO Scenarios :

1. Aggressive lowering of BP. If mean BP falls, but IOP remains high, the blood supply to ON may fall below a critical level.
2. If BP is good BUT you take glaucoma drops that also reduce BP – prime example B Blockers

The EYE DIGEST (Illinois University) also include

6. Suspicious discs
7. Corneal Thickness <555µm risky
    a. *(PF – this really is wrong! It is surely not a risk factor in itself but leads to artefact in our measurement of IOP. What we really need is a method of bypassing Scleral rigidity when recording IOP).*
8. Positive Family History
    a. Tends to run in families but a hereditary basis has not been identified but two studies have shown a higher incidence (particularly siblings and lesser parents and children).

High BP does cause a very slight rise in IOP but of such a small order as to be of no clinical significance

ASSOCIATED CONDITIONS

1. Ocular
2. Extraocular

ADDITIONAL USEFUL INFORMATION

1. Blood Pressure
2. Heart Rate
3. Blood Sugar
4. Blood lipid
5. Migraine
6. Raynauds
7. Neurological disease
8. Thyroid Disease
9. History of Blood Loss
10. History of kidney Disease
11. Smoking habits
12. Drinking habits
13. Family History of Vision Loss
14. Family History of Glaucoma

RISK FACTORS

Not of developing glaucoma but of picking target pressured

(FROM TARGET PRESSURE CALCULATIONS)

CCT

Family history

Gonioscopic Findings

IOP Range *(PF – Fluctuations and Peaks)*

Life Expectancy *(PF – Initial Age, General Health)*

Pigmentary Dispersion / PEX

Stage of ON damage

Stage of VF loss

Systemic Diseases

OPTIC NERVE NOTES

Disc Haemorrhages

0-0.21% of normals

Up to 4% of Glaucoma patients

But very much more common in NTG – up to 40%

PeriPapillary Atrophy

Temporal Crescent very common – up to 80% of normals
Nasal Crescent least common in normals (Tilted discs?)

Frequency and area increase with glaucoma
Position of PPA corresponds to neural rim loss
PPA extent may be greater in NTG
Extra clue – not pathognomonic

Barring

Circumlinear vessel present in 50% of normals

RNFL

Use green light and 90D

NTG

1. Localised Rim loss – notching early in disease
2. Flat disc Excavation
3. Disc Haemorrhages
4. PPA
5. Narrowing of Retinal Arteries
6. RNFL – localised loss (corresponds to localised rim loss)
   It must be noted that these are trends suggested by some, but not all observers. It is unlikely that ONH changes alone could be pathognonomic for a specific type of glaucoma.

BLOOD PERFUSION AND GLAUCOMA
(particularly NTG)

Evidence that this is the case :

1. The existence of NTG itself would suggest a non-pressure component
2. Disc haemorrhages
3. Higher prevalence of Retinal Vein Occlusion in glaucoma
4. Association of NTG & Migraine, Raynaud's, Vaso spasm
5. Association of NTG & Systemic hypotension and nocturnal dips
6. Association of glaucoma & Abnormal blood coagulation profile
7. Association of NTG & Silent myocardial ischaemia

8. Association of NTG & Cerebral infarcts
9. Association of NTG & hypotensive shock and severe blood loss

EXTRA FIELD NOTES

No standardised, objective method for determining field progression has been agreed.

Pragmatic (recent) approach
Glaucoma field loss is usually slow
Hence
Will rarely be detected within one year, even with strict test/retest regimes
Hence
An individual approach
With stricter follow up for advanced disease or VF defects close to fixation
Otherwise
2-3 tests to 'train' patient and provide mean values for baseline
Then repeat testing twice a year

Reduced Sensitivity

1. In a cluster of points on the same hemifield (non-edge points) by ≥5dB
2. Single test point by ≥10dB

MUST be confirmed – with 2 extra tests (3 altogether)

For example Humphries 'Glaucoma Change Probability Map'

1. Confirmed 3 consecutive times – CONFIRMED Glaucoma field progression
2. 2 out of 3 – TENTATIVE Glaucoma field progression

TARGET PRESSURE

'An estimate of the mean IOP obtained with treatment expected to prevent further glaucomatous damage'

(PF says this may not be the total case. Target Pressure may be a level that allows some progression but not at a rate that will cause loss of QoL)

There is no single IOP level safe for everyone

RULE OF THUMB

20% reduction from initial pressure at which damage occurred
Or
Below 18mmHg at ALL check-ups for advanced glaucoma

Unfortunately it is only hindsight that will tell us that the target pressure is achieved – that is fields must deteriorate to know we haven't reached target
(PF says ; the definition of Target Pressure uses the term 'pressure at which damage occurred'. This is not necessarily the threshold pressure at which damage could occur. This could well explain why the rule of thumb may not achieve the target pressure goal).

Target IOP can vary according to:

1. IOP before treatment (see PFs note)
2. Overall risk of IOP related ON damage
    a. Average IOP
    b. Maximum IOP
    c. IOP fluctuations
3. Stage of glaucoma
4. Rate of progression
5. Age and life expectancy of patient
6. Other risk factors

If disease continues to deteriorate
Lower target pressure again
But possibly also question compliance
IOP spikes (phase?)

DRUGS

<u>BETA-BLOCKERS</u>

Betaxolol	B1 Selective 1	Betoptic	0.5%
Carteolol	ISA	Teoptic	1%, 2%
Levobunolol		Betagan	0.5%
Metipronolol	Non-preserved		0.1%

Timolol Non-preserved/preserved/EA 0.25%, 0.5%

ACTION
Decreases AqH production

BID – more does not give enhanced effect. Peak effect 2 Hrs
If IOP target not reached increase concentration (ex 0.25% timolol to 0.5% timolol)
Can be mixed with many other drugs BUT minimal extra effect with Dipivefrin and NO extra effect with Adrenalin (same thing really), and yet Combigan is (Brimonidine + Timolol) and that works!

INDICATION
To reduce IOP when pressure could be deleterious to VF.
In a variety of types of glaucoma including

1. POAG
2. OHT
3. OK in aphakes
4. Some secondary glaucomas

MAJOR CONTRAIDICATIONS

1. Asthma, COAD. Not so bad with Betaxolol as this is β1 Selective (note : selective not specific)
2. Heart block, Cardiac failure

MAJOR SIDE EFFECTS

1. Following on from (1) above – Bronchospasm, Airway obstruction
2. Following on from (2) above – Bradycardia, Heart failure, Arrythmia
    a. Leading to Hypotension, Distal Oedema
    b. Especially on guard with nocturnal dips which are associated with progressive disc damage

DRUG INTERACTIONS
May have additive effects with
Systemic β blockers
Calcium blockers - verapimil
ACE inhibitors

PROSTAGLANDIN ANALOGUES

Bimatoprost Lumigan 0.03% Combo Ganfort
both once daily
Latanoprost Xalatan (refrigerate) 0.005% Combo Xalacom
both once daily
Travaprost Travatan 0.004% Combo DuoTrav both once daily

ACTION
Increases uveoscleral outflow

Once daily – more can actually reduce effect. Effect after 2-4Hrs
maximum after 8-12

Can be mixed with many other drugs and is mixed with timolol

INDICATION
To reduce IOP when pressure could be deleterious to VF.
In a variety of types of glaucoma including

1. POAG
2. OHT

MAJOR CONTRAIDICATIONS
Hypersensitivity to ingredients or BAK

MAJOR SIDE EFFECTS

1. Cystoid macula oedema
    a. Primarily in Aphakes, torn lens capsules and as well in px of high risk
2. Conjunctival Hyperaemia
3. Anterior Uveitis

The first three directly relate to pro-inflammatory nature of prostaglandins

4. Iris Pigmentation – more of an issue if unilateral treatment
5. Eyelash growth
6. Reactivation of HSK (?)

DRUG INTERACTIONS
Virtually none (Thimerosol)

ALHPA AGONISTS

Brimonidine	Alphagan	α2 Selective	0.2%	Combo	Combigan	both BID 12hrs apart
Dipivefrin	Propine		0.1%		BID	12 hrs apart

ACTION
Reduces aqueous production and Increases uveoscleral outflow

Twice daily. Maximum effect after 12Hrs, hence BID but also spaced 12 hours apart
Can be mixed with many other drugs and Brimonidine is mixed with timolol

INDICATION
To reduce IOP when pressure could be deleterious to VF.
In a variety of types of glaucoma including

1. POAG
2. OHT

Either as Monotherapy when B Blockers are contra-indicated or
As Adjunctive therapy when target pressure is not reached.

MAJOR CONTRAIDICATIONS

1. Occludable angles (not Brimonidine as it is α2 selective). I would suggest we would need to be careful anyway. If in doubt do iridotomies first.
2. MAO inhibitors (antidepressants)

MAJOR SIDE EFFECTS

1. Follicular conjunctivitis
2. Lid elevation
3. Pupil dilation

DRUG INTERACTIONS
MAO and Tricyclic antidepressants
CARBONIC ANHYDRASE INHIBITORS

Asetazolamide	Diamox		
Binzolamide	Azopt (also non-preserved)	1%	BID to TID
Dorzolamide	Trusopt BID	2%	Combo Cosopt (also non-preserved)BID

ACTION
Reduce Aqueous production but by a different mechanism than B blockers so can have a additive effect with B blockers

Can be mixed with other drugs and Trusopt is mixed with timolol

INDICATION
To reduce IOP when pressure could be deleterious to VF.
As monotherapy in Px unresponsive or contraindicated to b blockers
As adjunctive therapy to B blockers or Prostaglandins

MAJOR CONTRAIDICATIONS
Before we do this : Carbonic Anhydrase is an enzyme found in many tissues, including the eye, but also RBCs. CA catalyses hydration of CO_2 and the dehydration of Carbonic Acid. It is also a suphonimide.

1. Hypersensitivity to any ingredient
2. Hypersensitivity to sulphonimides
3. Severe renal impairment
4. Hyperchloraemic acidosis
5. Pregnancy and Breast feeding

Basically where Sodium or potassium blood levels are depressed.

MAJOR SIDE EFFECTS
1. Sulphonide effects – anaphylaxis, rash, S-J Syndrome
2. Corneal Oedema (in Px with low endothelial count)
3. Corneal erosions
4. Metabolic acidosis and electrolyte imbalance.

DRUG INTERACTIONS
Caution with Steroids

PARASYMPATHOMIMETICS (DIRECT) PILOCARPINE

Pilocarpine Nitrate	Pilocarpine 2%	QID
Pilocarpine Hydrochloride	Pilogel 4%	nocte

ACTION
Increases conventional outflow facility. Open up inefficient drainage channels in TM resulting from contraction or spasm of the ciliary muscle
It can be used in combination with all Hypotensives (Miotics, B Blockers, sympathomimetics, CAIs, Hyperosmotics) The hyperosmotics makes sense in AACG.
Prostaglandins are NOT listed, because constriction of ciliary muscle is assumed to reduce uveoscleral outflow.
Emc says you should NOT combine different miotics

INDICATION
Pilogel :

1. To reduce IOP when pressure could be deleterious to VF – POAG, OHT.

Pilocarpine :

1. to reverse action of weaker mydriatics (PF – should be antimuscarinics as these act on the same muscle – sympathomimetics are not only stronger but act on dilator rather than sphincter)
2. emergency treatment of AACG.

MAJOR CONTRAIDICATIONS

Uveitis
Neovascular glaucoma
Possible worsening of pupil block glaucoma

MAJOR SIDE EFFECTS

1. Bronchospams
2. Intestinal cramps (parasympathetic effects – (+) gut wall (-) sphincters hence we are increasing gut constriction)
3. Bradycardia (and Hypotension as a result of bradycardia) – VAGAS Nerve
4. Pupil constriction
5. Accomodative spasm, induced myopia, brow ache

6. Increased pupil block
7. Retinal Detachment, tears

DRUG INTERACTIONS
A competitive interaction with Prostaglandins is assumed since constriction of ciliary muscle is assumed to reduce uveoscleral outflow.

<u>COMBINATION DRUG THERAPIES</u>

1. Do not combine drugs of the same group
2. When available combined drug preparations are preferable
    a. Improves compliance
    b. Improves QoL
3. To use more than 2 drugs in combination is not recommended
    a. PF says – think surgical interventions
4. Additional drugs should be considered if target pressure is not attained
    a. Mike Birch Says
        i. If xalatan non responsive (<20% iop drop)- switch: Choices different prosta analogue or B-blocker or Cosopt. Choice will partly depend if B-blockers contraindicated or not.
        ii. If Xalatan partially responsive - Add. Same as above except you wouldn't add second prosta analogue
5. Effect of drug combination is measured purely by Hypotensive effects
6. Assuming equal Hypotensive effects no drug is preferable in terms of ONH and VF preservation
7. If initial drug has no effect or tachyphalaxis occurs – change the initial therapy rather than add to it.
8. Increasing recommended dose will not increase hypotensive effect but will increase side effects.

Drugs that DON'T Combine
1. Any of the drugs with the same type
2. Prostaglandins and parasympathomimetics (Pilocarpine)

<u>COMPLIANCE</u>

Px co-operation is essential

Chronic long term problem
Progressive disease
Requires frequent and regular medication

Poor compliance can be due to:

1. Failure to instil drops (includes ineffective technique of self administration – arthritis)
2. Excessive use of eye drops (may cause systemic side effects)
3. Self administration of non-prescribed eye drops (remember in the SPCs for all drugs it mentions the need to space out drop instillation to ensure optimal effects of both, so the likelihood of diluting effects is high with more than one drug, whether self prescribed or prescribed)
4. Improper timing of eye drops and eye drop administration for the wrong reasons. (a more frequent problem if numerous drops are to be instilled after a change in medication regime) or (during short term change as after cataract extraction when suddenly they need t put extra drops in qid).

Improving Compliance:

1. Make the patient an active and informed participant. (EMPOWER)
    a. Glaucoma itself and mechanisms of medications have to be explained and understood
    b. Must also be advised on drug side effects so they don't discontinue as a result
    c. Written
2. Number, concentration and frequency should be kept to a minimum
    a. Ocular irritation can be reduced by reducing the number of preserved drops
    b. Combo drops are obviously better than numerous individual drops
3. Inconvenience of instillation kept to a minimum
    a. Timed to daily landmarks and daily activities
    b. Minimum number of drops
4. Patient taught how to instil drops correctly
    a. Technique
        i. closure, Punctal occlusion
        ii. use on instillation frames
        iii. time intervals for administration of different drugs
        iv. PF – AND WHAT TO DO IF ONE IS MISSED!
    b. Must be checked at reviews
    c. Ancillary staff to help

i. PF – BUT ALSO FAMILY AND CARERS!

TREATMENT GUIDELINES

POAG
Identify Target pressure
Involve patient as informed partner in management and *decisions (PF this would be particularly important if SP is involved, some patients may not like this while others would see the convenience as mush better)*

1. Medical Treatment (from flow chart)
    a. Monotherapy
    b. Combination therapy
2. Laser Trabeculoplasty (LTP)
3. Filtration Surgery
    a. Adjunctive medical therapy
4. Insertion of aqueous drainage tubes/stents
5. Cyclo-destructive techniques

POAG SUSPECT
Risks and benefits of treatment must be weighed against the risks of glaucomatous disc damage
The risk of developing glaucoma increases with the number of risk factors
Involve the patient as partner (*PF they may make the choice of Treatment/No Treatment*).

1. Medical therapy
    a. Any mono/combo, as long as it is tolerated and effective
    b. Avoid adjunctive therapies
2. Laser Traculoplasty – not usually indicated
3. Filtering Surgery – No indicated
4. Follow-up 6/12. To be increased if parameters remain normal ie ON, IOP, VF, ONH/RNFL Photos

NTG
Very few papers indicting that treatment even works.
Target IOP

1. Peak IOP between 8 and 15mmHg on diurnal curve
   or
2. 30% reduction from baseline

1. Medical Therapy
    a. Any drug effective and tolerated that reaches target
    b. Avoid medications with vaso-constrictive effects or with systemic hypotension
        i. Example EGS gives Beta Blockers (Non-selective ones)
        ii. *PF – This all makes sense as NTG is more susceptible to perfusion issues and is more prevalent with Hypotensive dips and vascospastic phenomenon. THEREFORE NON-SELECTIVE α-SYMPATHOMIMETICS WOULD ALSO BE CONTRA-INDICATED.*
2. Laser Trabeculoplasty
3. Surgery
    a. In case of progressive damage in spite of maximal medication or laser trabeculoplasty and failure to reach target pressure
    b. Intensive post-operative care with bleb manipulation may be needed to maintain low IOP
4. Follow-up
    a. 3/12 to 12/12 with examination of ON, NF, IOP
    b. ONH/RNFL photos every 2 to 3 years

NTG SUSPECT

Observe these patients very carefully *(PF – and may need to investigate the possibility of alternative pathologies)*

Treatment is NOT indicated unless there is the suggestion of disease progression.

If progression is due to glaucoma the treat as NTG ie

1. Medical Therapy
    a. Any drug effective and tolerated that reaches target
    b. Avoid medications with vaso-constrictive effects or with systemic hypotension
        i. Example EGS gives Beta Blockers (Non-selective ones)
        ii. *PF – This all makes sense as NTG is more susceptible to perfusion issues and is more prevalent with Hypotensive dips and vascospastic phenomenon. THEREFORE NON-SELECTIVE α-SYMPATHOMIMETICS WOULD ALSO BE CONTRA-INDICATED.*
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    a. 3/12 to 12/12 with examination of ON, NF, IOP
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OHT
Although in the past this was used for diagnosis, the term OHT should only be used to indicate that IOP is consistently above 2SD of the mean
Consider CCT
A modest increase in IOP is NOT sufficient reason to treat. BUT consider it in patients with repeated tensions in the high twenties, even without risk factors.

If left untreated:
1. 10% will convert over 5 years
2. Risk increases with increasing IOP
3. Prophylactic therapy should be discussed with individual patients considering the presence of risk factors

Follow-up intervals of 12/12, examine
ON
IOP
VF ONH/RNFL photography ever 2-3 years

PIGMENTARY GLAUCOMA
1. Topical Medications
    a. Beware medications that induce dilation (PF – Sympathomimetics, particularly non-selectives such as Dipivefrin) as they may cause additional pigment shedding
    b. Check peripheral retina for tears before using pilocarpine
2. Argon Laser Trabeculoplasty
    a. IOP response is highly variable
3. Filtering Procedures
4. Peripheral Nd:YAG laser Iridotomy
    a. To eliminate reverse pupil block
    b. Potential long term role is to
        i. Reduce iris rubbing and consequent pigment shedding
        ii. Prophylactic importance to prevent irreversible TM damage