The Glaucomas

S. Yazdani, MD
Glaucoma Service
Labbafinejad Medical Center

Definition

- A chronic optic neuropathy
- Characteristic optic nerve head changes
- Associated visual field defects
- The most important risk factor is IOP

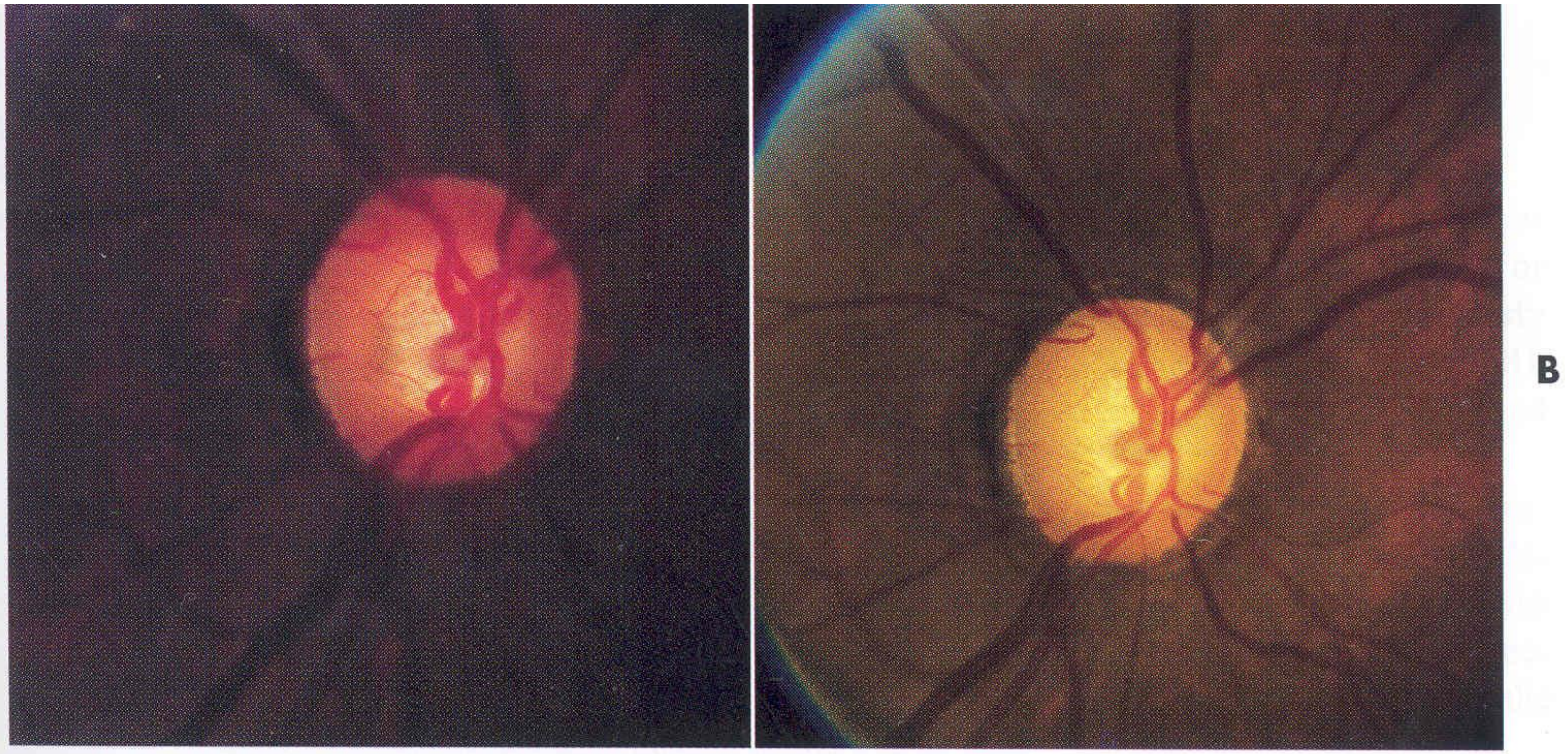


Figure 14-3 **A,** The initial photograph of the right optic disc in a patient with primary open-angle glaucoma. **B,** The patient after a 12-year interval. During this interval, there has been concentric enlargement of the cup. An area of focal thinning of the neural rim can be observed near a cilioretinal vessel in the superotemporal region. The bent portion of this vessel could not be visualized well before the loss of the overlying neural rim tissue. (From Campbell DG, Netland PA: *Stereo atlas of glaucoma,* St Louis, 1998, Mosby.)

OS +2.00 x 160 2013

SINGLE FIELD ANALYSIS EYE: LEFT

NAME: DOROSTIYAN-PARVANEDOKHT DOB: 06-06-1935

ID:

CENTRAL 24-2 THRESHOLD TEST

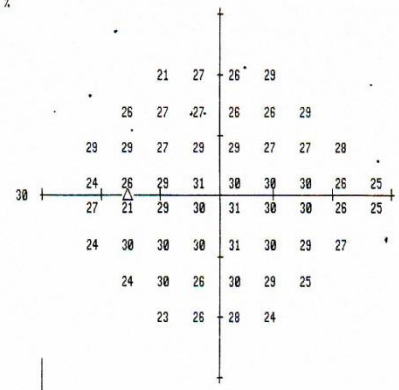
FIXATION MONITOR: GAZE/BLIND SPOT
FIXATION TARGET: CENTRAL
FIXATION LOSSES: 2/11
FALSE POS ERRORS: 0 %
FALSE NEG ERRORS: 2 %
TEST DURATION: 03:34

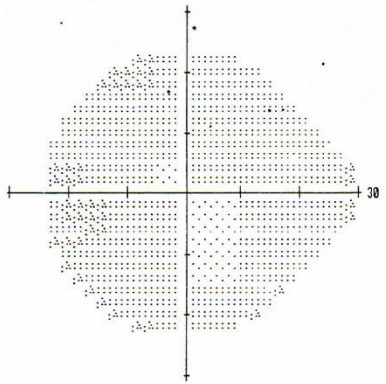
STIMULUS: III, WHITE
BACKGROUND: 31.5 ASB
STRATEGY: SITA-FAST

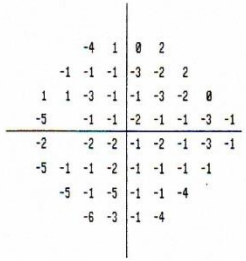
PUPIL DIAMETER: 4.4 MM
VISUAL ACUITY: 0.9
RX: +5.25 DS -2.00 DC X 165

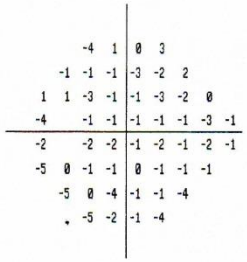
DATE: 25-08-2009
TIME: 11:12
AGE: 74

FOVEA: 28 DB







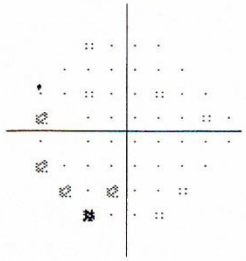


GHT
WITHIN NORMAL LIMITS

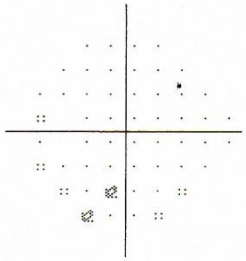
VFI 99%

MD -1.73 DB P < 10%
PSD 1.63 DB

TOTAL DEVIATION



PATTERN DEVIATION



:: < 5%
▒ < 2%
▓ < 1%
■ < 0.5%

SHAHID LABBAFI NEJAD HOSPITAL
EYE CLINIC
OPTOMETERY DEPARTMENT

HFA II 750-12279-4.2/4.2

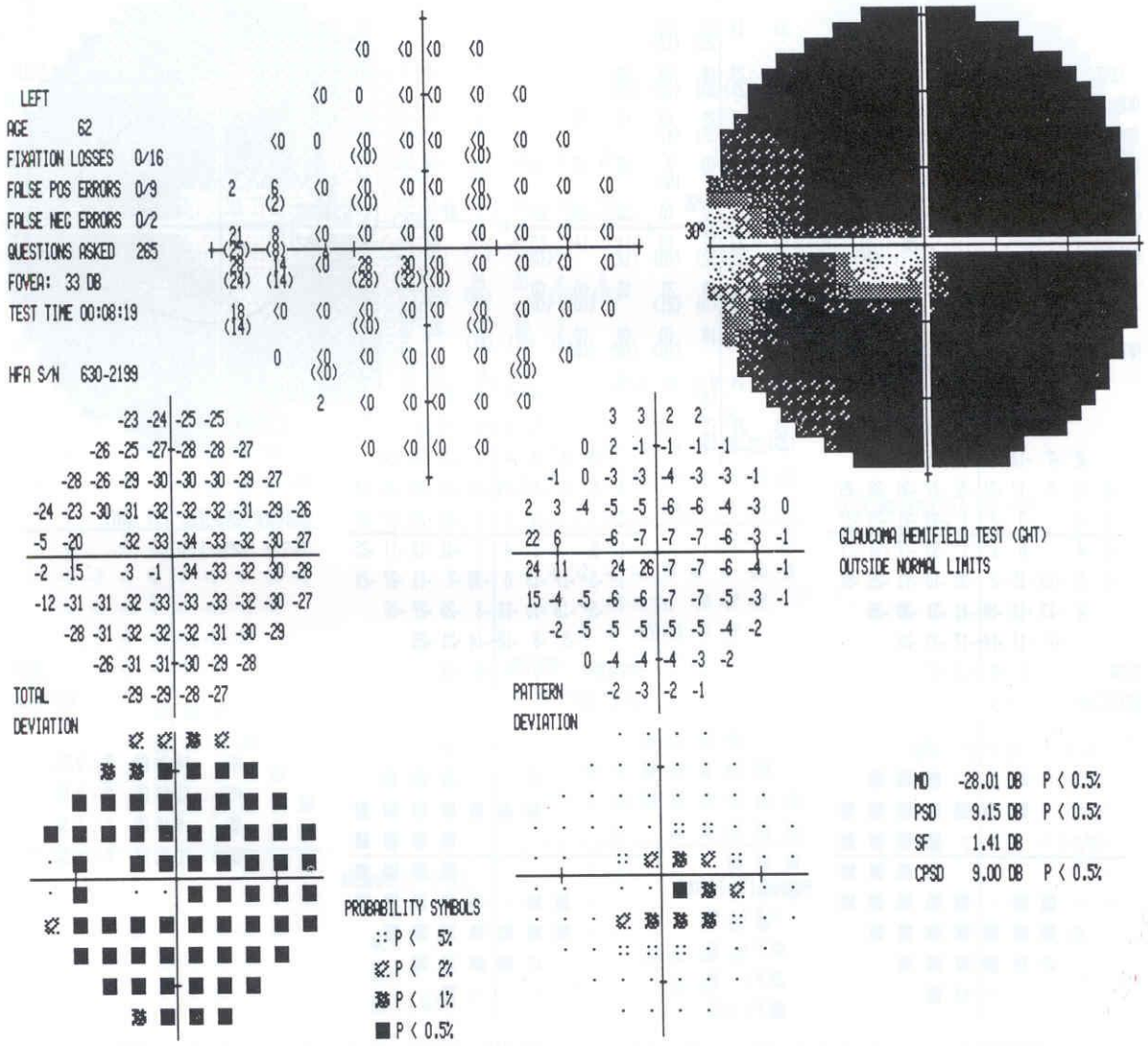


FIG. 4-20. More Advanced Glaucoma. Only a small central island and a temporal island remain, with broad, dense abnormality in both the upper and lower arcuate regions extending nasally from the blind spot. The foveal threshold is good (33 dB, within normal limits for age), and visual acuity is 20/20 (left eye).

Epidemiology and Global Importance

- The second most common cause of global blindness
- A lot of this blindness occurs in 3rd world countries
- The visual handicap and blindness are IRREVERSIBLE

Definite glaucoma

From a clinical point , 2 of the 3 following criteria:

- High IOP
- “Glaucomatous” ONH changes
- Visual field defects

Glaucoma Suspects

- IOP suspects
- ONH suspects
- VF suspects
- Angle suspects

Classification

- Etiologic: primary vs secondary
- Age dependent: Infantile (congenital), juvenile, adult onset
- Anatomical: open vs closed angle

Children with Glaucoma

Theoretical considerations

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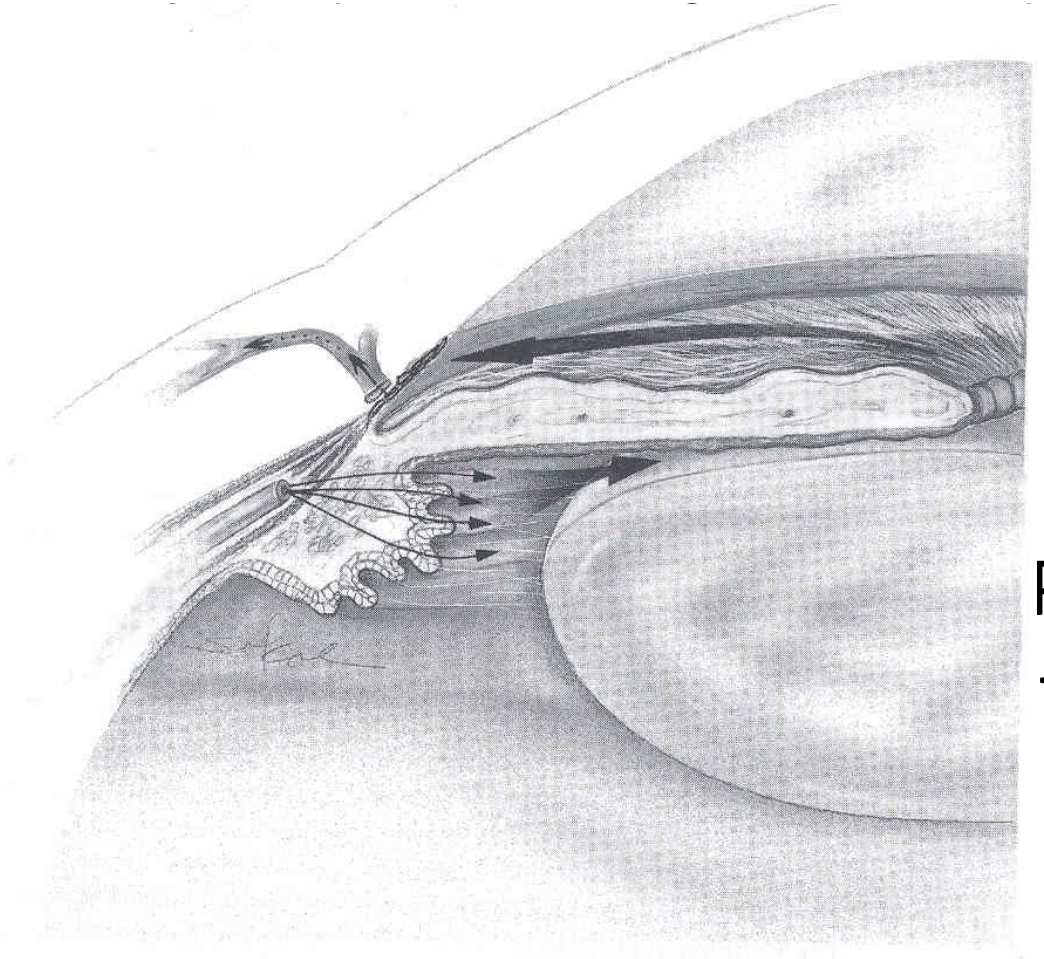


P exist:
tflow

Normal Outflow Pathways

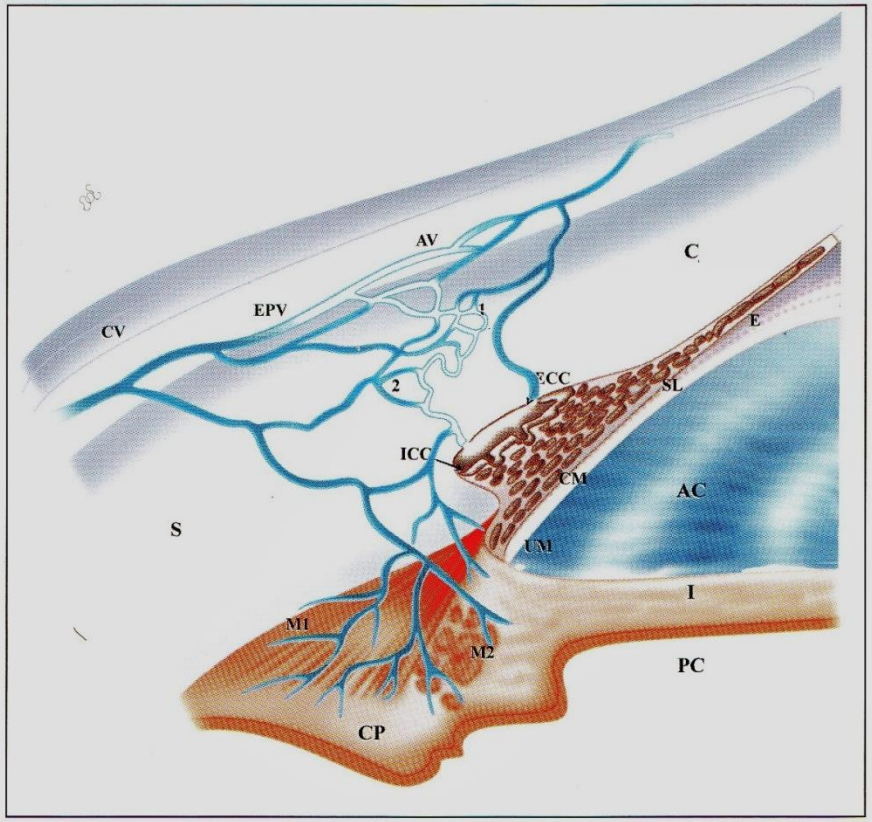
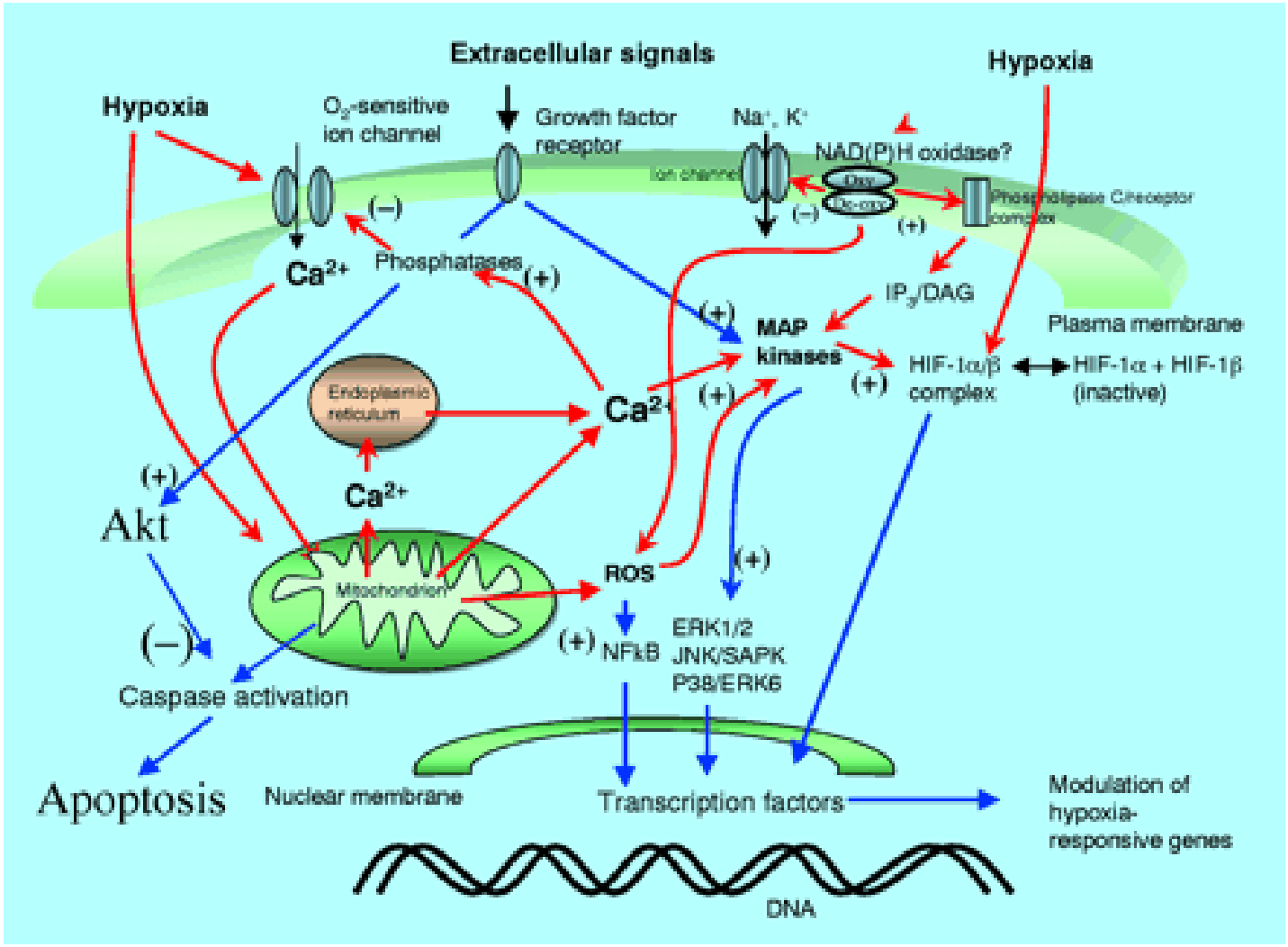


Figure 3.7
Collector channels.
AC = anterior chamber; PC = posterior chamber; C = cornea; I = iris; S = sclera; CI = ciliary processes; SP = scler spur; E = endothelium; L = Schwalbe's line; CM = corneoscleral meshwork; UM = uveal meshwork; SC = Schlemm's canal; ICC = interior collector channels; ECC = external collector channels; 1 = intrascleral venous plexus; 2 = deep scler plexus; 3 = ciliary venous plexus; AV = aqueous vein; EPV = episcleral vein; CV = conjunctival vein; M1 = longitudinal fibers of ciliary muscle; M2 = circular fiber c ciliary muscle (modified from Tripathi RC. *Experimental Eye Research* 1977;25:65–116).

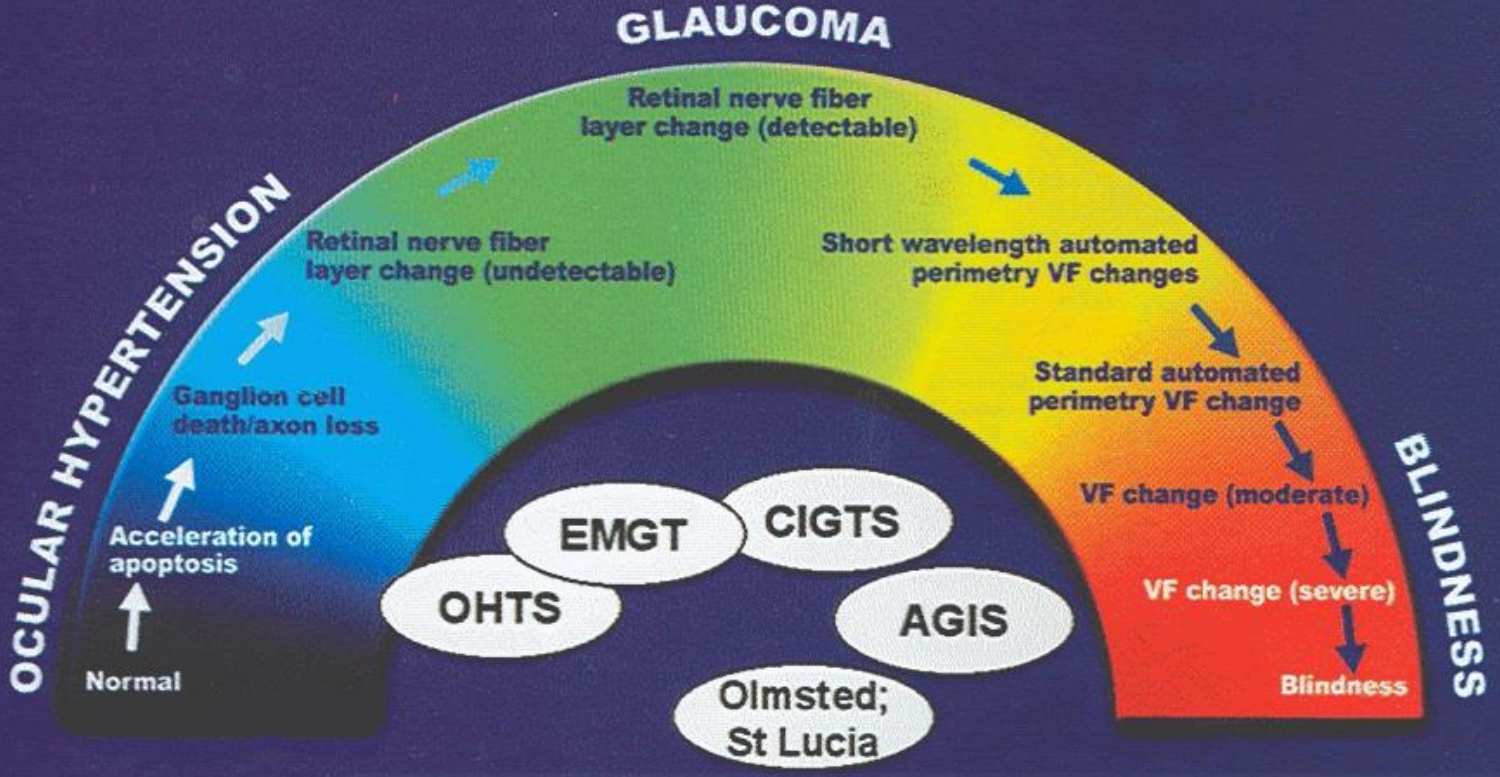
Why does glaucoma develop?

Glaucoma is a neurodegenerative disorder

- Mechanical theory
- Vascular theory
- Combined theory
- Unclear cellular events (oxidative stress, apoptosis, glutamate excitotoxicity, etc.)


Extracellular signals
Hypoxia
Hypoxia
O_2-sensitive ion channel
Growth factor receptor
Na^+, K^+
NAD(P)H oxidase?
Ion channel
Phosphatidyl C/receptor complex
(–)
(+)
Ca^{2+}
Phosphatases
(+)
IP_3/DAG
(+)
MAP kinases
Plasma membrane
HIF-1α/β complex
HIF-1α + HIF-1β (inactive)
(+)
Endoplasmic reticulum
Ca^{2+}
(+)
Ca^{2+}
Akt
Mitochondrion
ROS
(+)
(+)
ERK1/2
JNK/SAPK
P38/ERK6
(–)
(+) NFkB
Caspase activation
Apoptosis
Nuclear membrane
Transcription factors
Modulation of hypoxia-responsive genes
DNA

How Does Risk Change Over the Glaucoma Continuum?

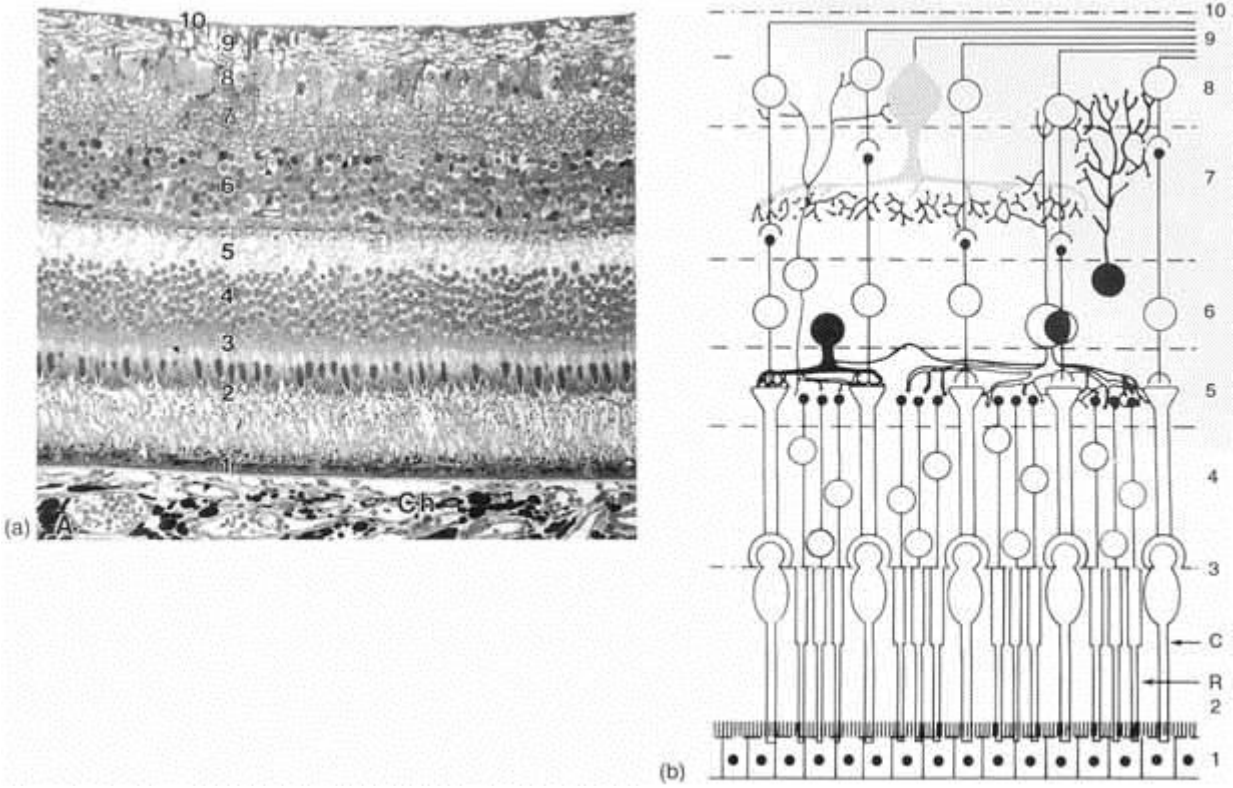


Adapted from Weinreb RN et al. *Am J Ophthalmol* 2004;138:458-467.

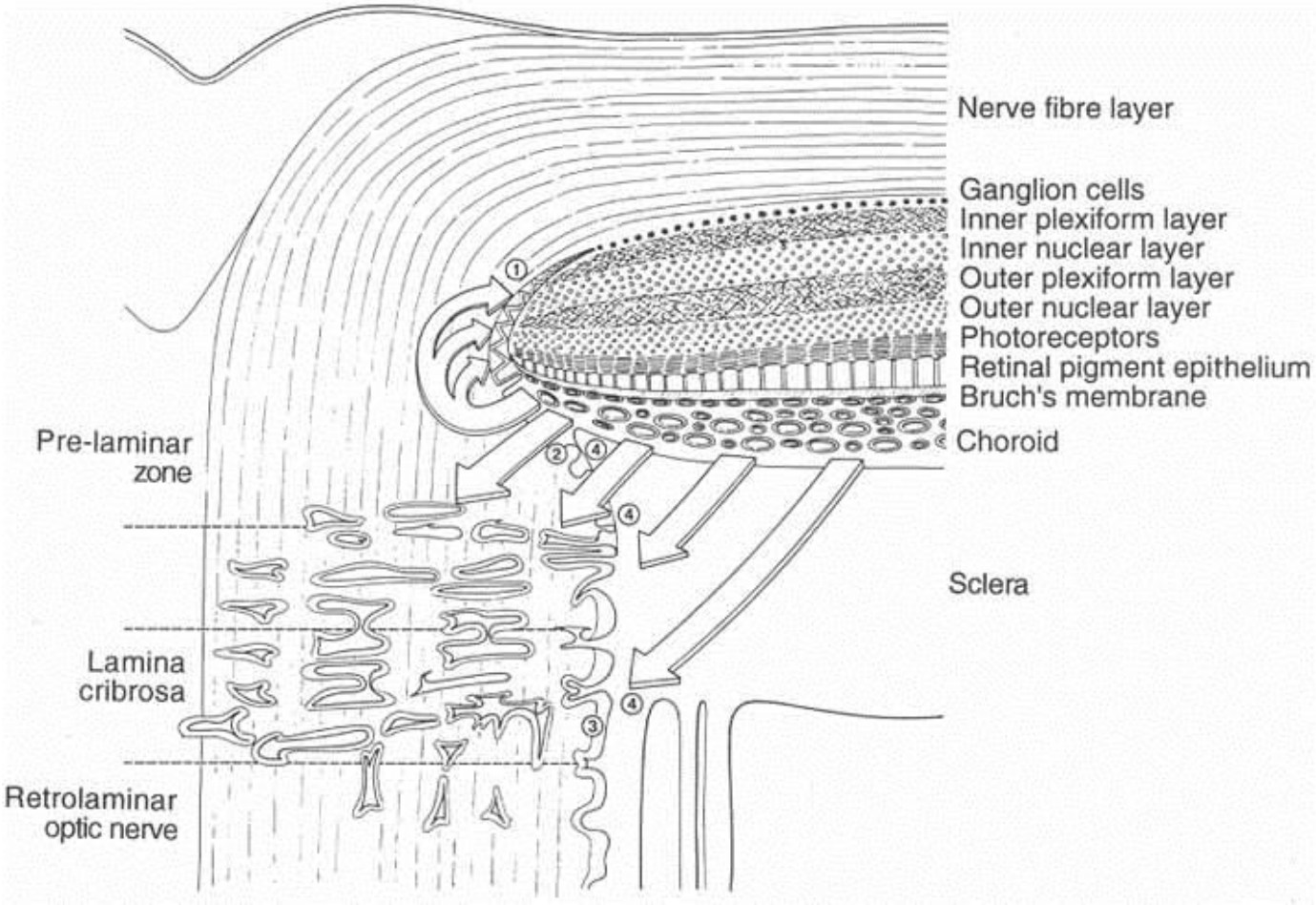
Which structure sustains damage?

- Retinal nerve fiber layer
- The axon of ganglion cells located in the retina
- It is a long axon starting from the retina and extending all the way to the LGB in the brain (going through the optic nerve, chiasm and optic tract without any synapse)
- This damage is most evident at the ONH

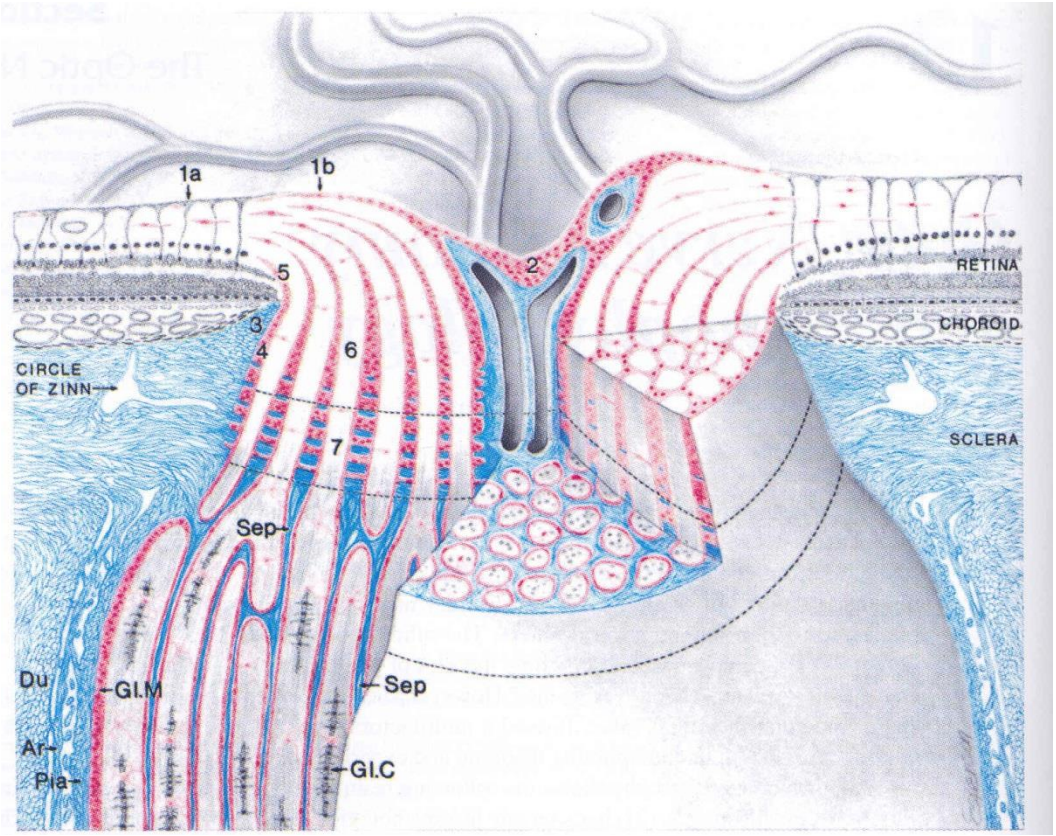
Retinal Layers



Retinal and optic nerve structure



ONH structure



Risk factors

- Age
- Sex?
- Family history
- Refractive error
- Diabetes mellitus?
- IOP

Intraocular Pressure

Determination of the intraocular pressure (IOP) is a central feature in the diagnosis and management of the glaucomas.

A true measurement of IOP requires a direct fluid connection to the anterior chamber.

IOP

- Why is IOP important?
- Where does it impose its effect?
- Are all tissues susceptible to the same degree?

IOP

No safe level for IOP

Relative risk for development of glaucoma:

IOP<16	RR=1
IOP 16-19	RR=1.7
IOP 20-23	RR=4
IOP >23	RR=10.5

Initial IOP may be misleading

Patients with initial diagnosis of glaucoma:

- 1st visit : only 50% have IOP>21
- 2nd visit : 75% have IOP>21
- Multiple visits: 85% IOP >21

The Shiotz Tonometer

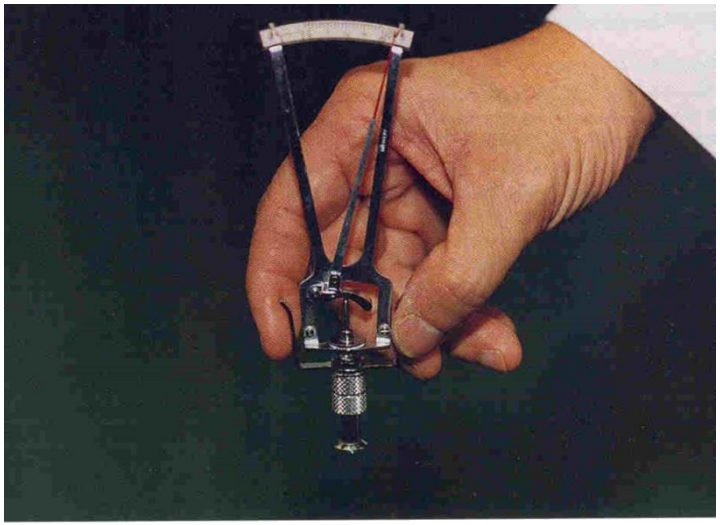
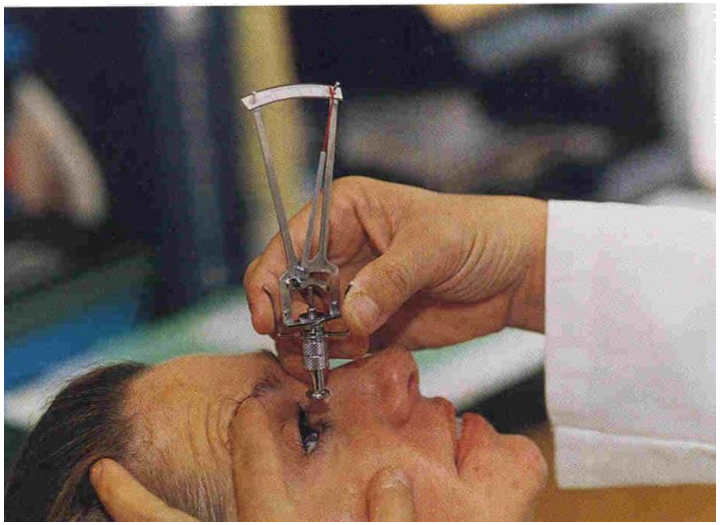


Figure 5-5 Shiøtz tonometer.



Airpuff Tonometer

The Tonopen

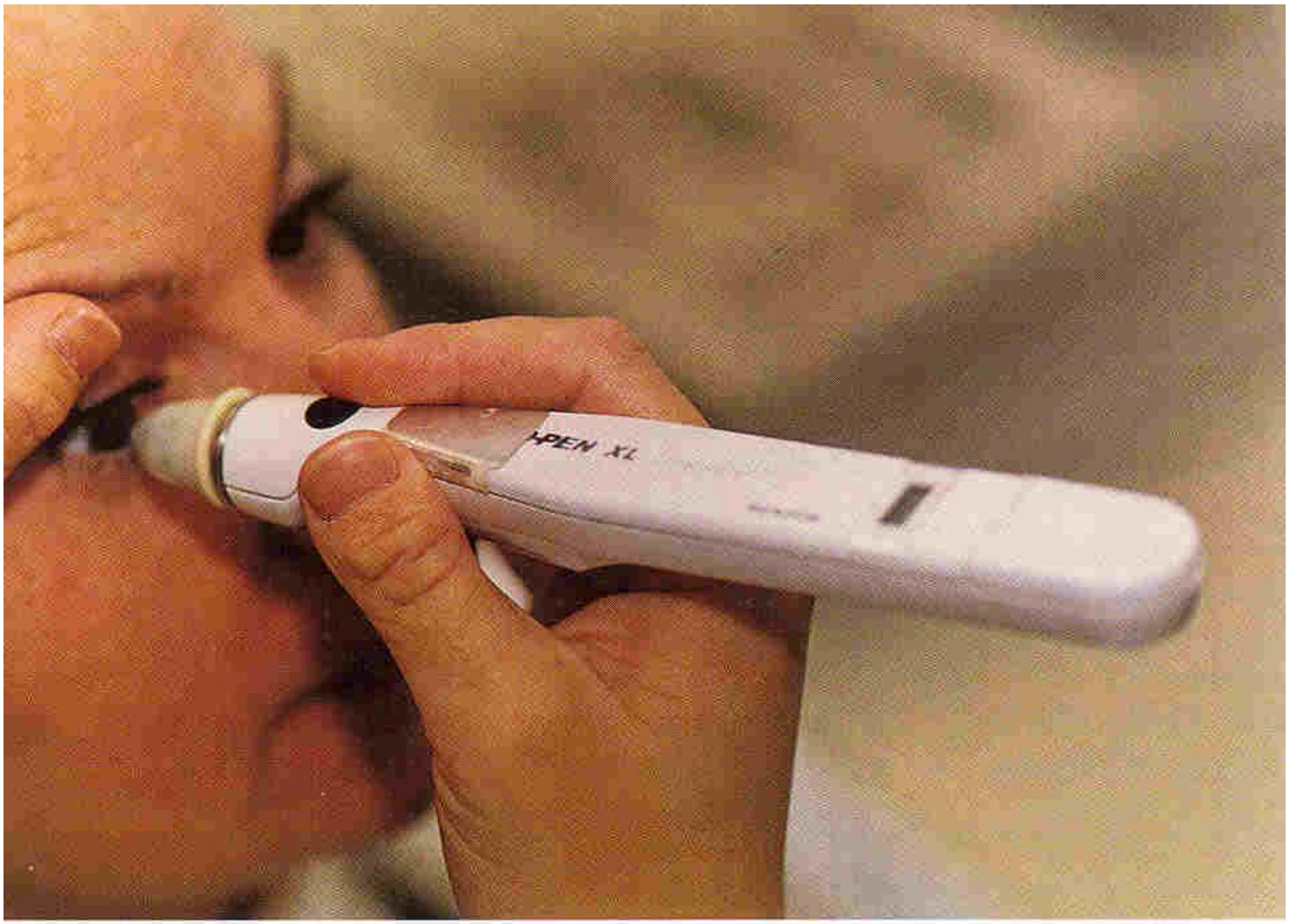


Figure 5-4 Tono-Pen.

Dynamic Contour Tonometry (Pascal Tonometer)

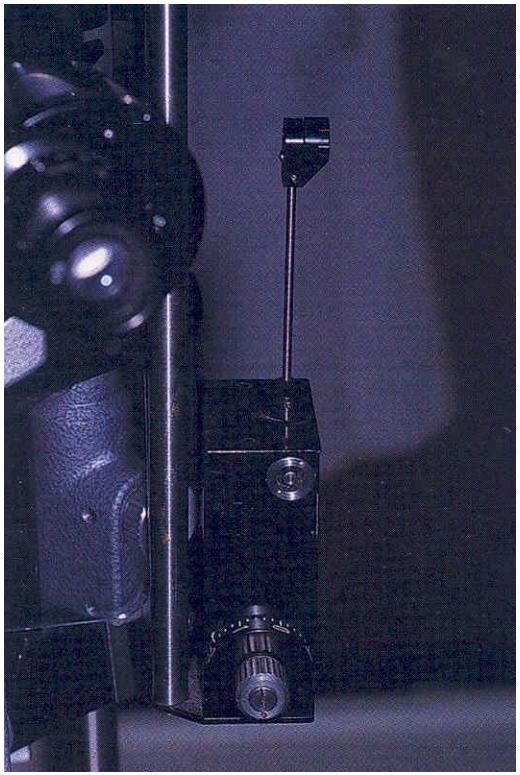
Ocular Response Analyzer

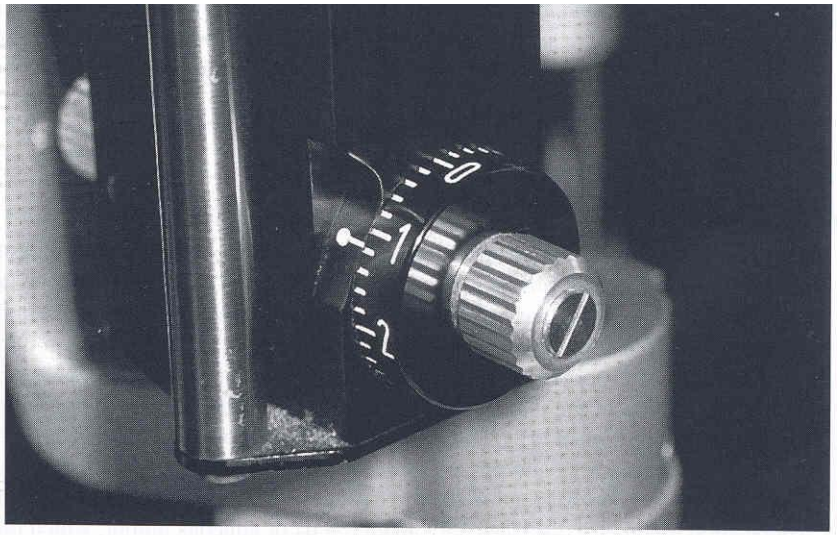
Rapid air pulse and an electro-optical system

The cornea resists the air puff causing delays in inward and outward applanation

Records two applanation pressure measurements

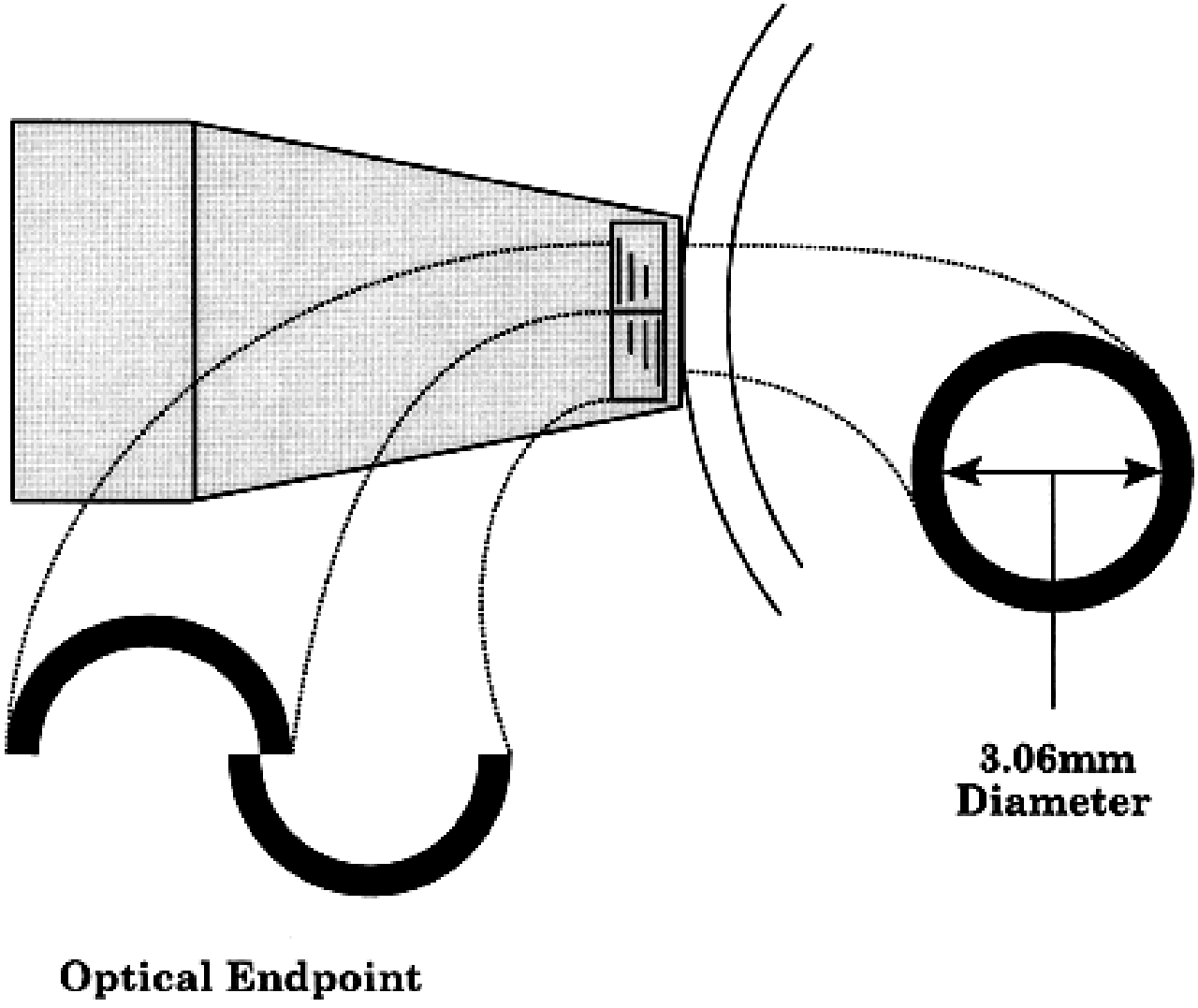
Goldmann Applanation Tonometer





gure 5-2 Goldmann applanation tonometer. Dial indicates force applied to applanate cornea; this number ultiplied by 10 equals intraocular pressure in millimeters of mercury.

Goldmann Applanation Tonometry



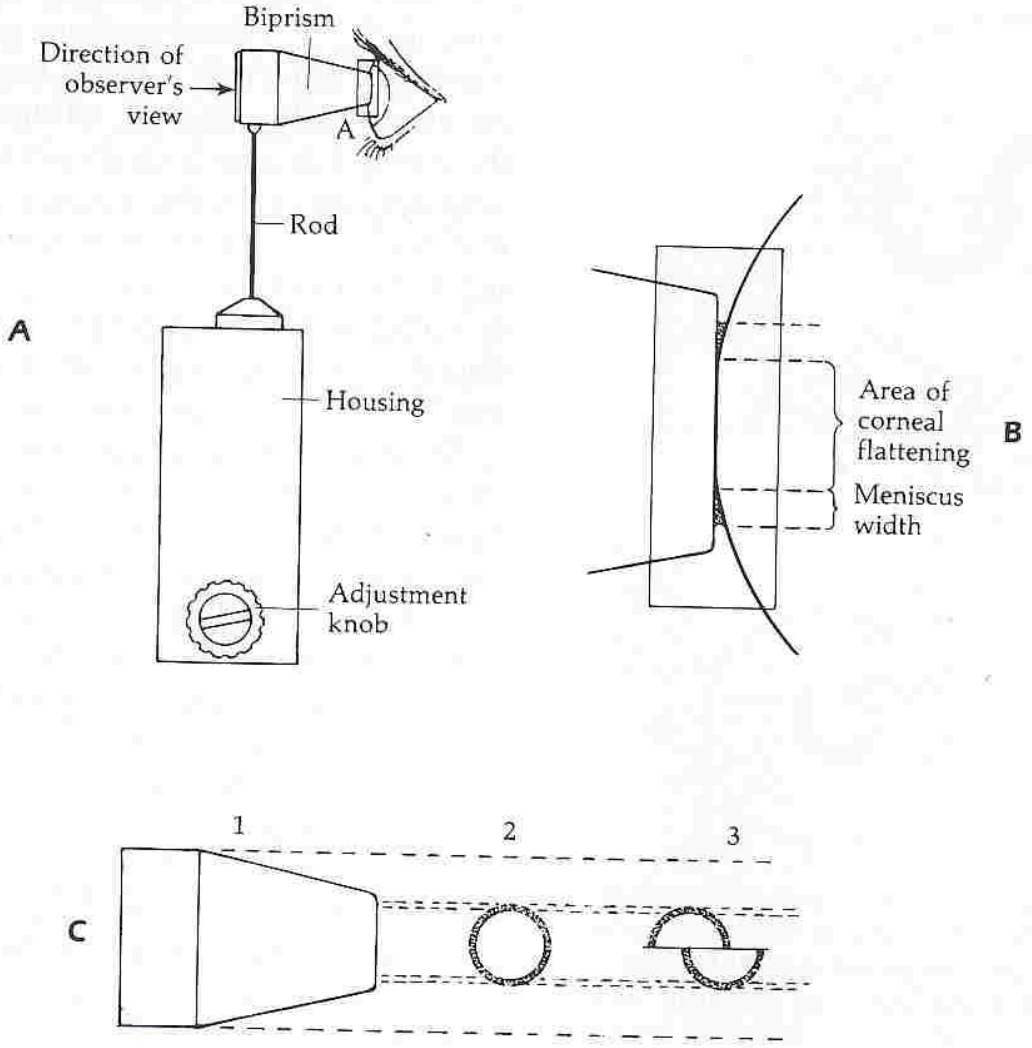


Fig. 20-4 Goldmann-type applanation tonometry. **A,** Basic features of the tonometer, shown in contact with patient's cornea. **B,** Enlargement shows the tear film meniscus created by contact of the biprism and cornea. **C,** View through the biprism *(1)* reveals a circular meniscus *(2)*, which is converted into semicircles *(3)* by the biprism. (From Shields MB: *Textbook of glaucoma*, ed 3, Baltimore, 1992, Williams & Wilkins.)

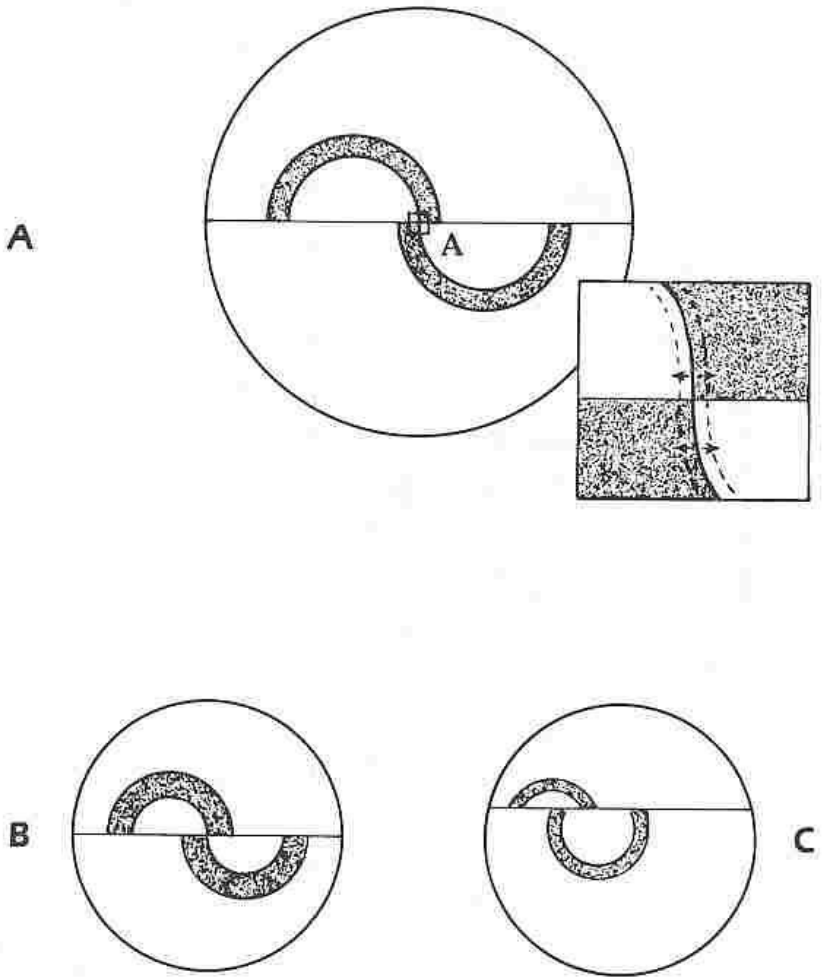


Fig. 20-5 Semicircles of Goldmann-type applanation tonometry. **A,** Proper width and position. Enlargement *(A)* depicts excursions of the semicircle caused by ocular pulsation. **B,** Semicircles are too wide. **C,** Improper vertical and horizontal alignment. (From Shields MB: *Textbook of glaucoma,* ed 3, Baltimore, 1992, Williams & Wilkins.)

What is the normal range for IOP?

- By statistical convention, the mean±2SDs covers 95% of subjects and represents the normal distribution
- Based on several studies the mean IOP for the general population is 16mmHg and the SD is 3
- Normal IOP: 10-22mmHg

Ocular hypertension (OHT)

IOP elevated above the statistically normal range.

Normal open angles.

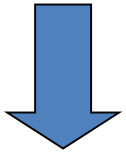
No evidence of glaucomatous optic nerve damage.

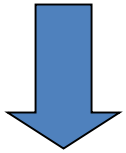
Absence of visual field abnormalities.

Absence of ocular or systemic conditions leading to high IOP.

OHT Study Results: Progression to Glaucoma

Progression to Glaucoma





Observation group	Treatment group
9.5%	4.4%

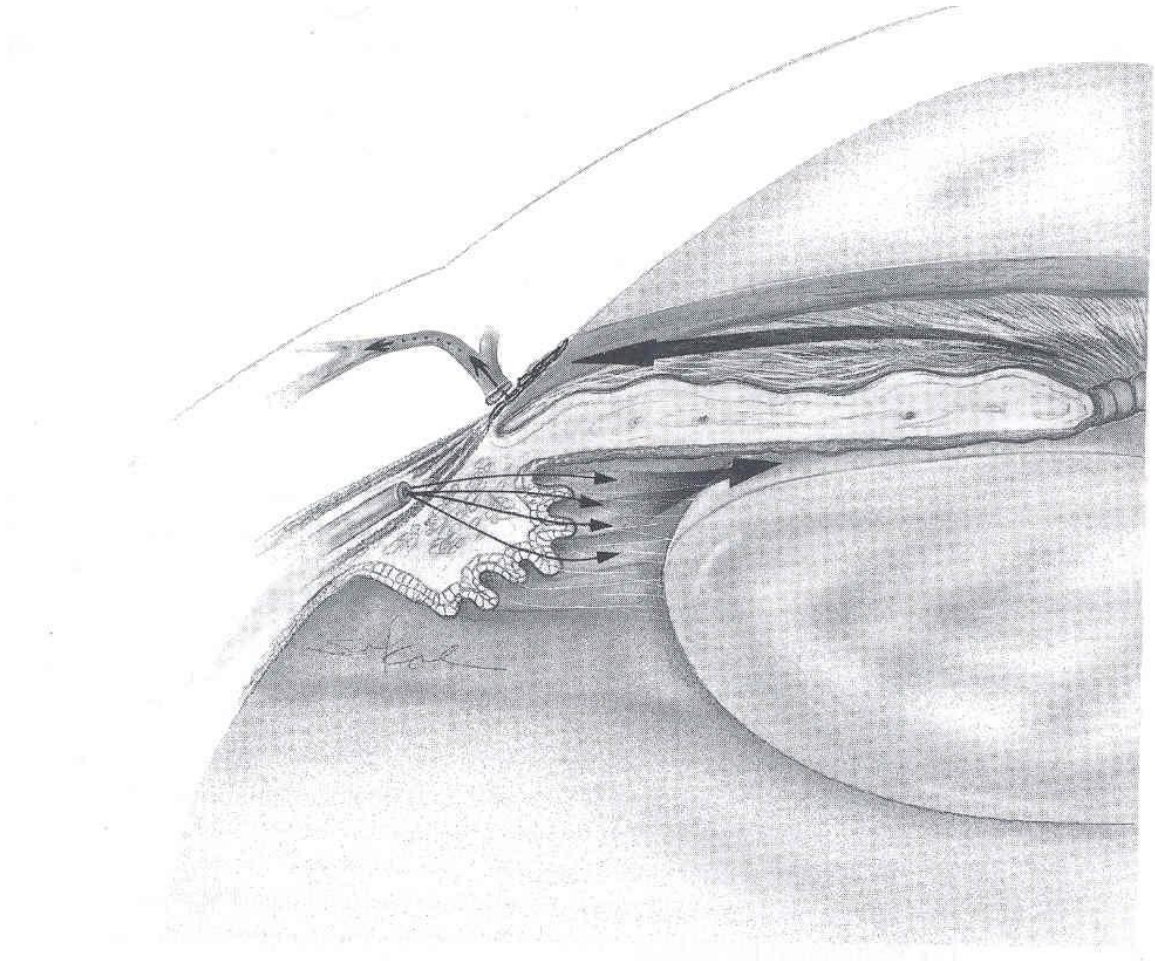
Treatment cuts risk of glaucoma cut by half

The great majority of OHT cases remain stable


Café Sidi Chebaane

Gonioscopy

Normal aqueous production and outflow



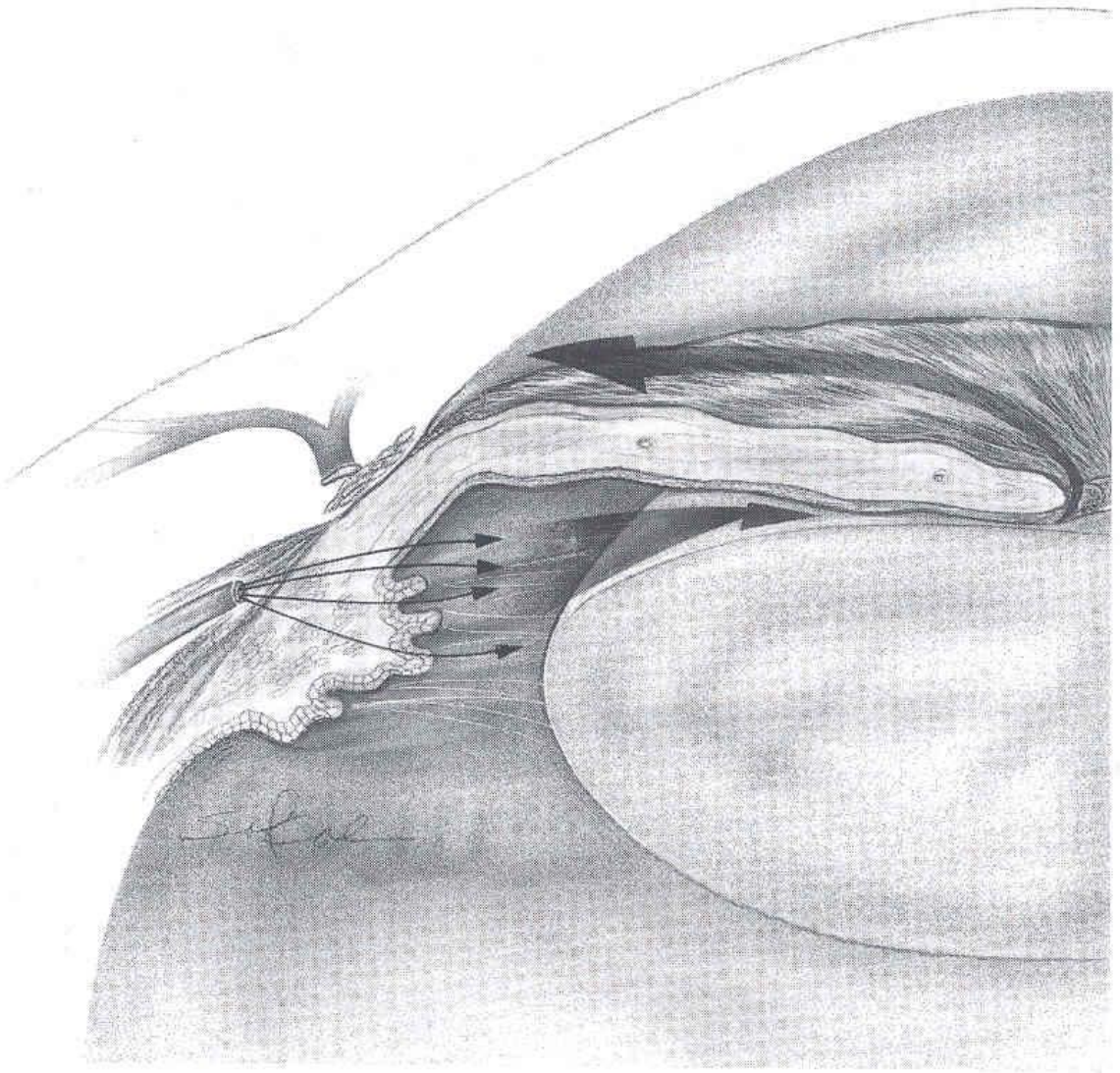
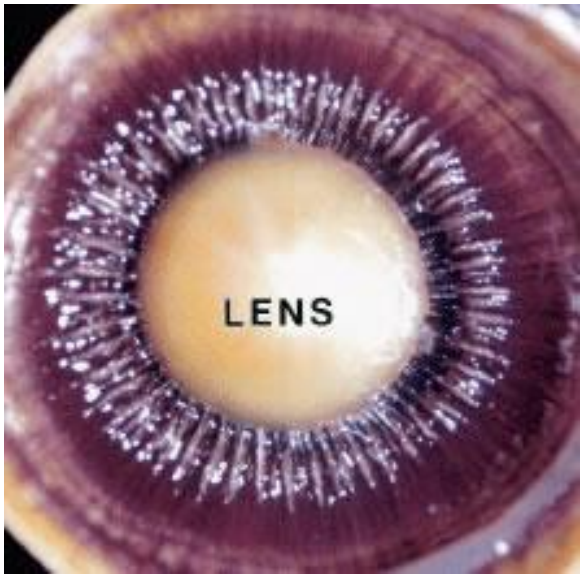
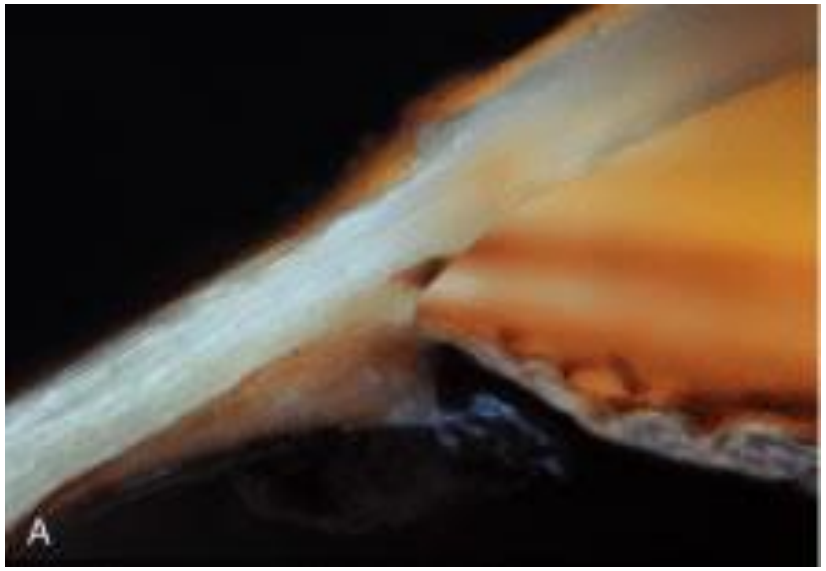
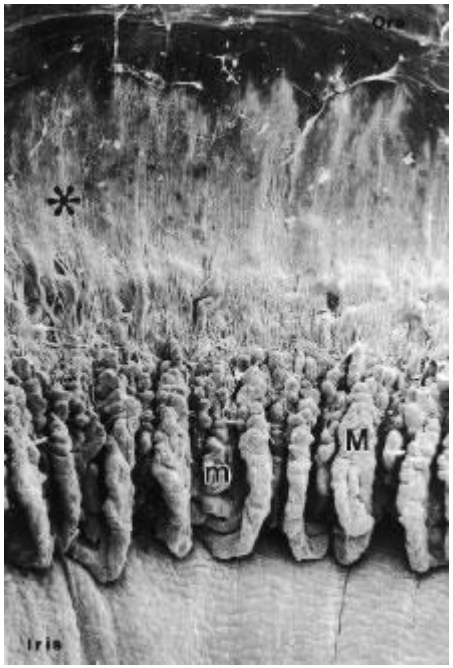


Figure 1-2 In angle-closure glaucoma, the peripheral iris covers the trabecular meshwork, obstructing aqueous humor outflow.


LENS


A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Iris

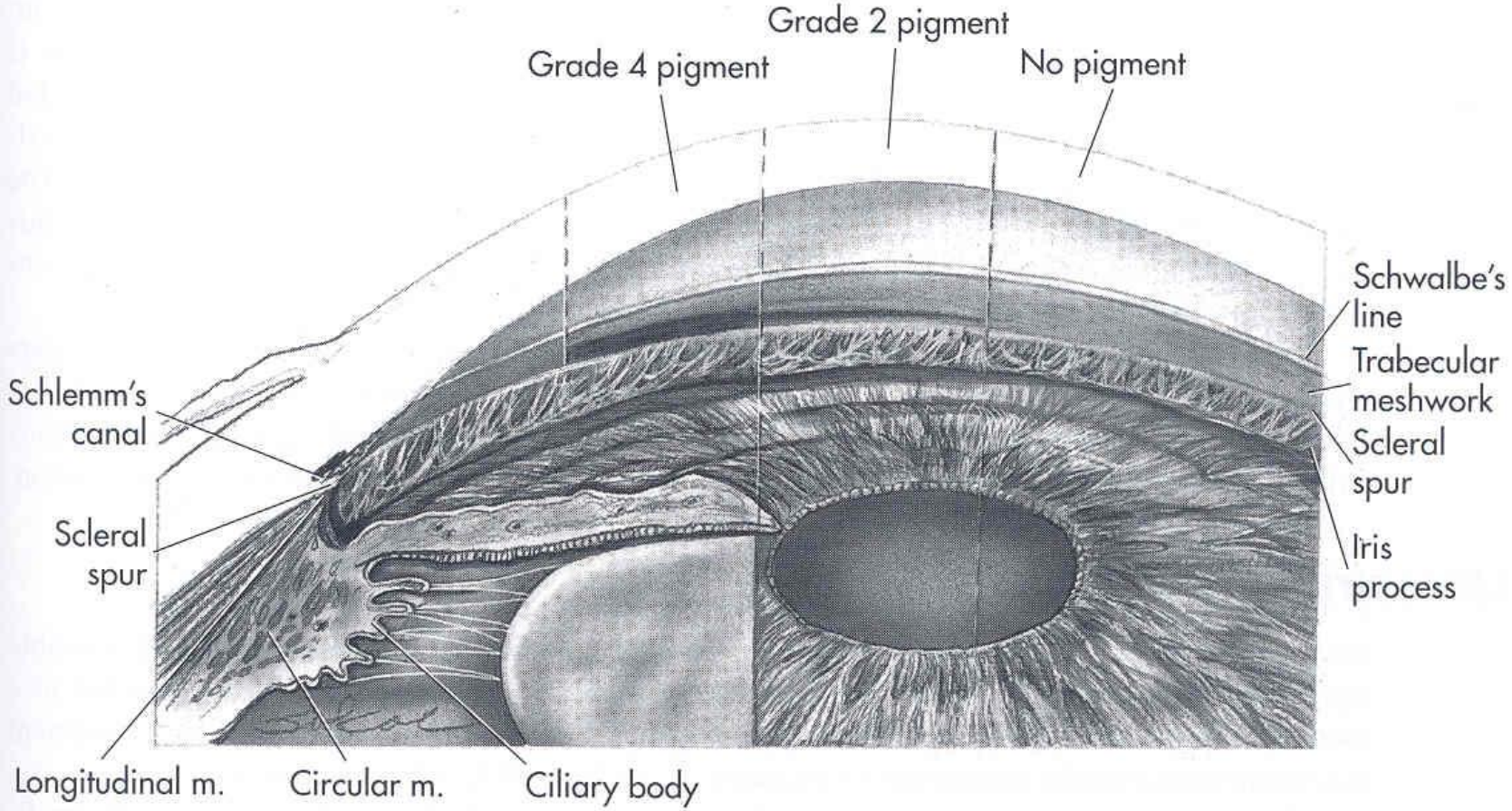


Figure 3-2 Schematic view of the zonules separating the vitreous cavity from the posterior chamber and the iris separating the anterior and posterior chambers.

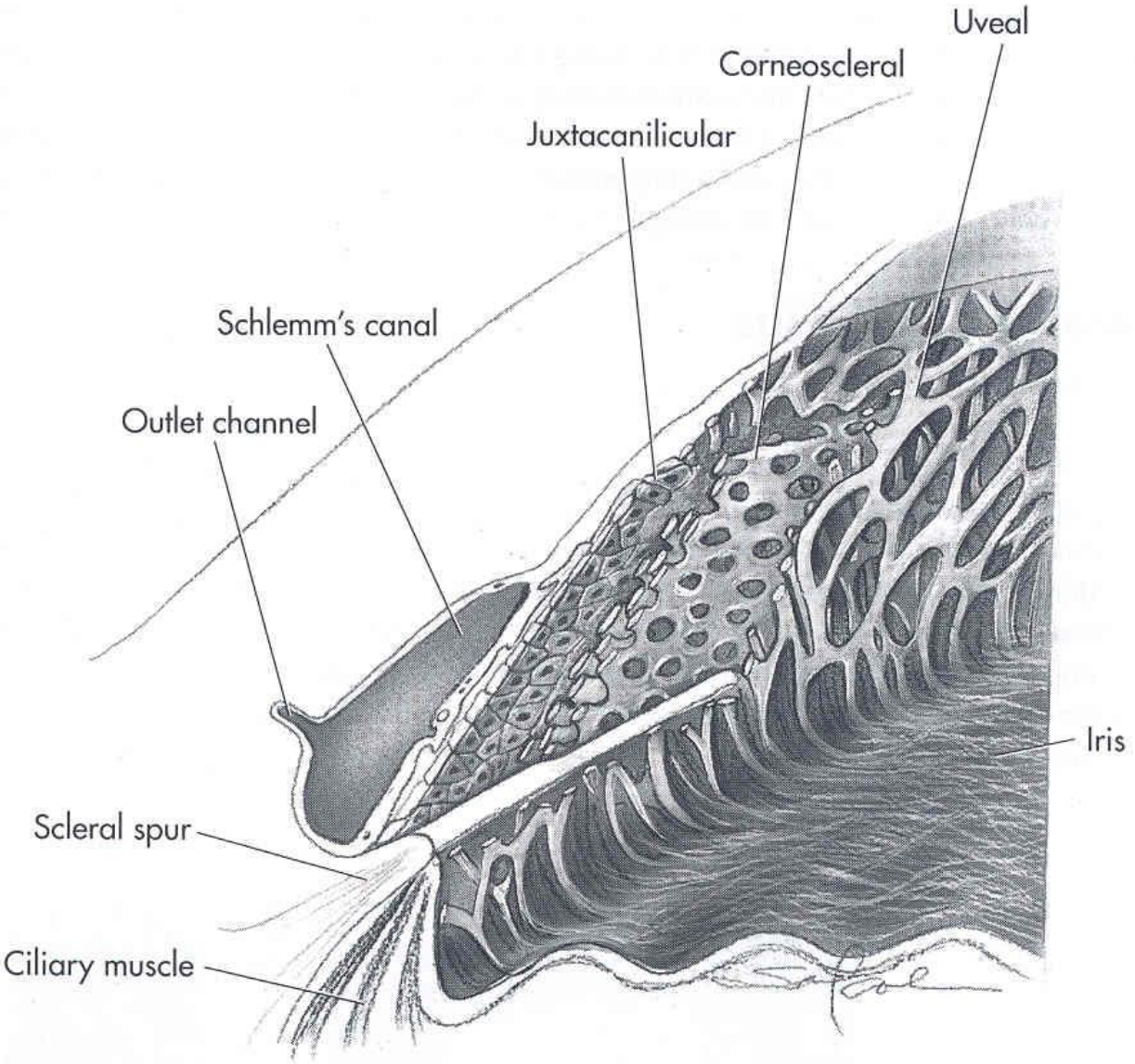


Figure 4-2 Schematic view of different layers of the outflow system. (Modified from Shields MB: *Textbook of glaucoma,* Baltimore, 1987, Williams & Wilkins.)

Why is the angle not visible ?

- The major reason for the angle being invisible is total internal reflection.
- Light rays emerging from the angle cannot reach the examiner’s eyes.
- The cornea/tear film interface with air has to be altered to allow gonioscopy.

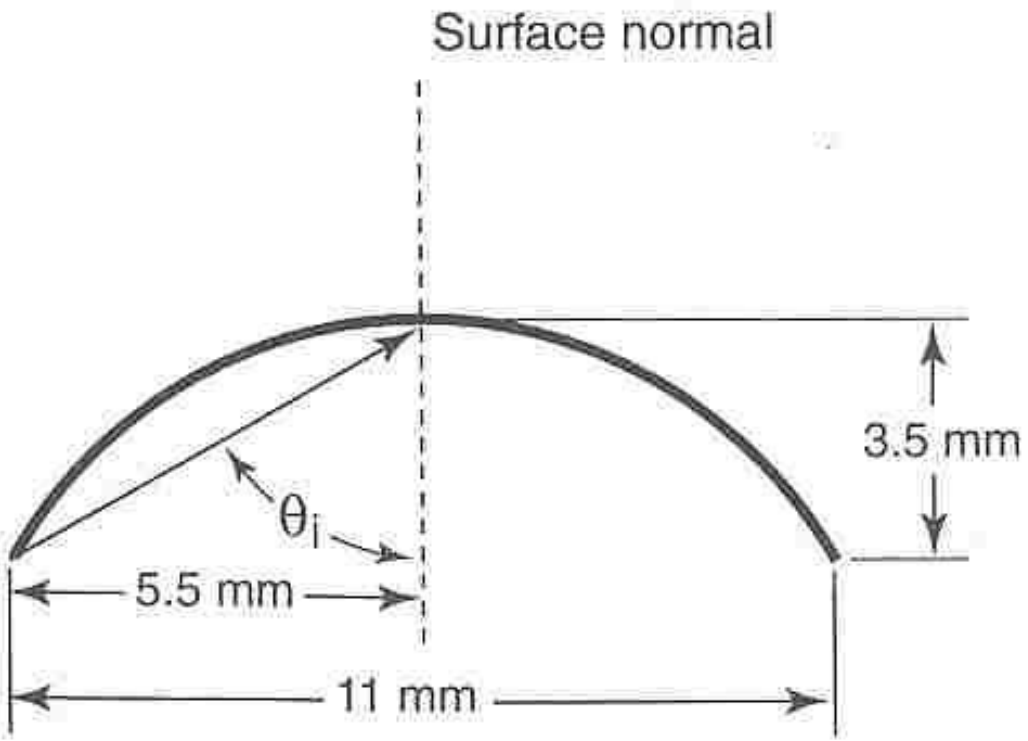


FIG II-24—Average anatomic dimensions of the anterior segment.

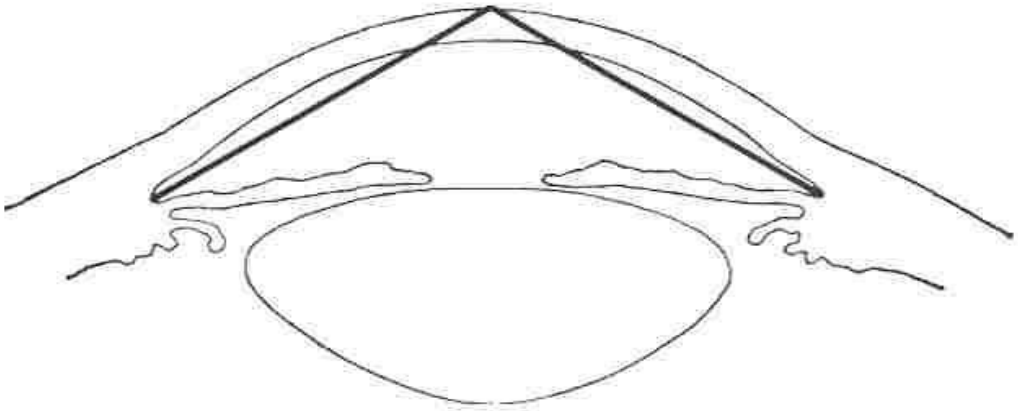


Figure 6-1 Rays of light originating at the anterior chamber angle. These rays undergo total internal reflection by the cornea.

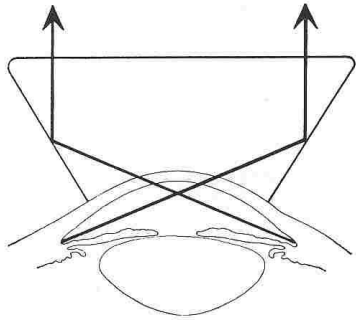


Figure 6-3 Rays of light emerging through a Zeiss indirect gonioscopic lens.

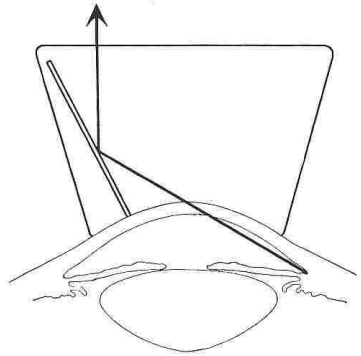


Figure 6-4 Rays of light emerging through a Goldmann lens.

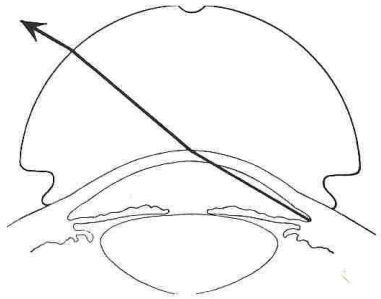


Figure 6-5 Rays of light from the angle, emerging through a Koeppe lens.

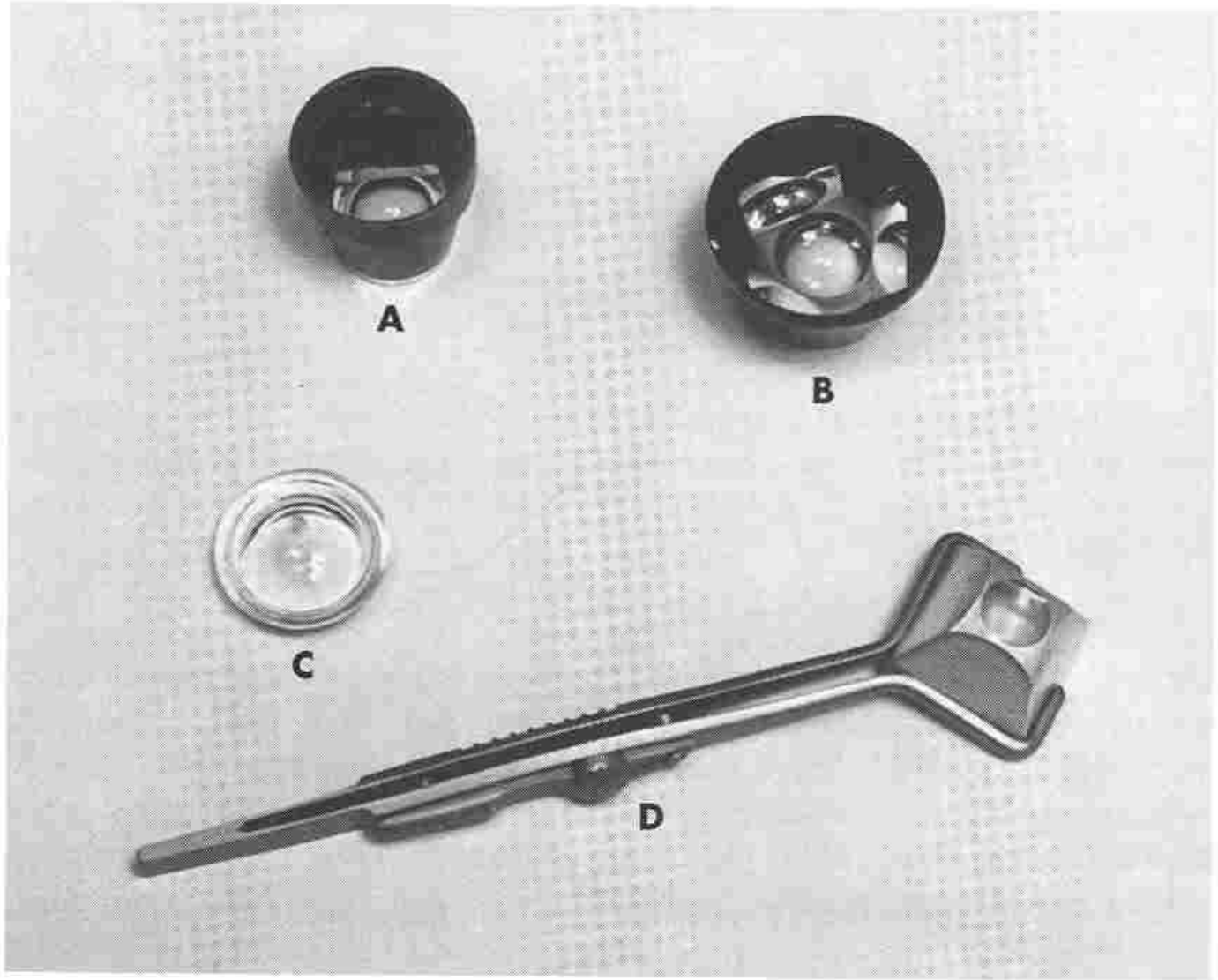


Figure 6-2 Gonioscopic contact lenses. **A,** One-mirror Goldmann; **B,** three-mirror Goldmann; **C,** Koeppe; **D,** hand-held Zeiss.

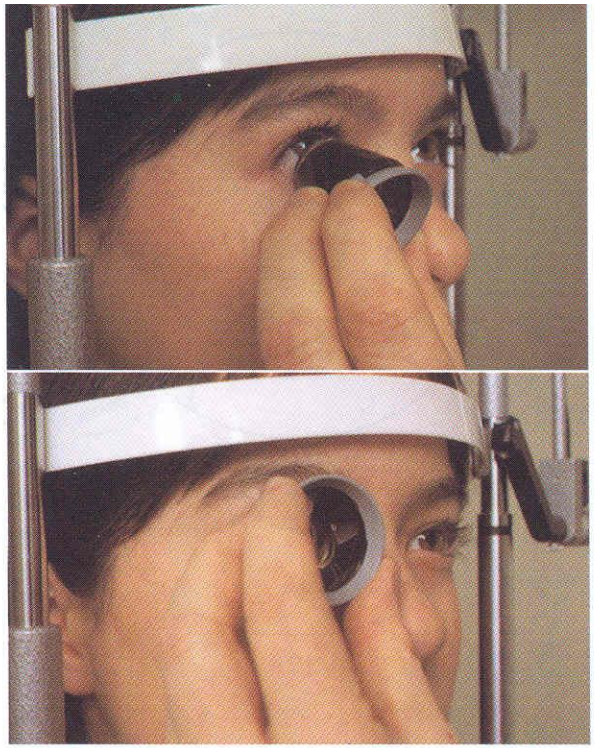


Figure 6-6 **A,** The Goldmann lens is brought into contact with the inferior sclera. **B,** The Goldmann lens tipped up into position. (From Alward WLM: *Color atlas of gonioscopy,* St Louis, 1994, Mosby.)

Figure 6-7 Zeiss four-mirror lens held in a diamond configuration. This position is more natural for some examiners, but the corners of the lens against the patient's eyelids can feel uncomfortable. (From Alward WLM: *Color atlas of gonioscopy,* St Louis, 1994, Mosby.)

A

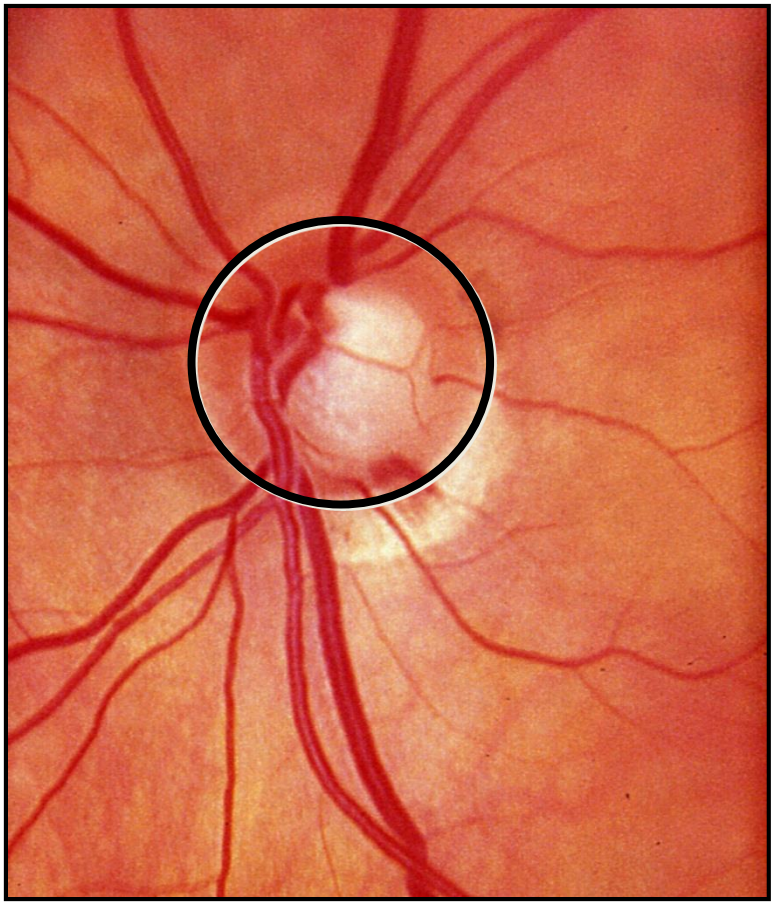
B

C

FIGURE 5–1 (A) Gonioscopic appearance of the normal anterior chamber angle. Schwalbe's line (SL), trabecular meshwork (TM), scleral spur (SS), and ciliary body band (CBB) all are visible in this open angle. (B) Drawing of the microscopic cross-section of the anterior chamber angle combined with the gonioscopic view to illustrate the anatomic basis for the corneal parallelepiped and the corneal light wedge. The parallelepiped (arrows) is formed by light reflecting from the anterior and posterior corneal surfaces. At the peripheral cornea, the outer margin of the parallelepiped curves posteriorly as the slit beam illuminates the junction of the clear cornea with the opaque sclera. This curved line forms the base of the corneal light wedge (asterisk). The point at which this base meets the inner, endothelial margin of the parallelepiped corresponds to Schwalbe's line (arrowhead). (C) Goniophotograph using a thin slit beam illustrating the base of the corneal light wedge (arrow) and Schwalbe's line (arrowhead). (Drawing by Lee Allen, copyright the University of Iowa. From Alward WLM. *Color Atlas of Gonioscopy*. Barcelona: Wolfe; 1994. Courtesy of W.L.M. Alward, M.D.)

Five Rules for Assessment of the Optic Disc in Glaucoma

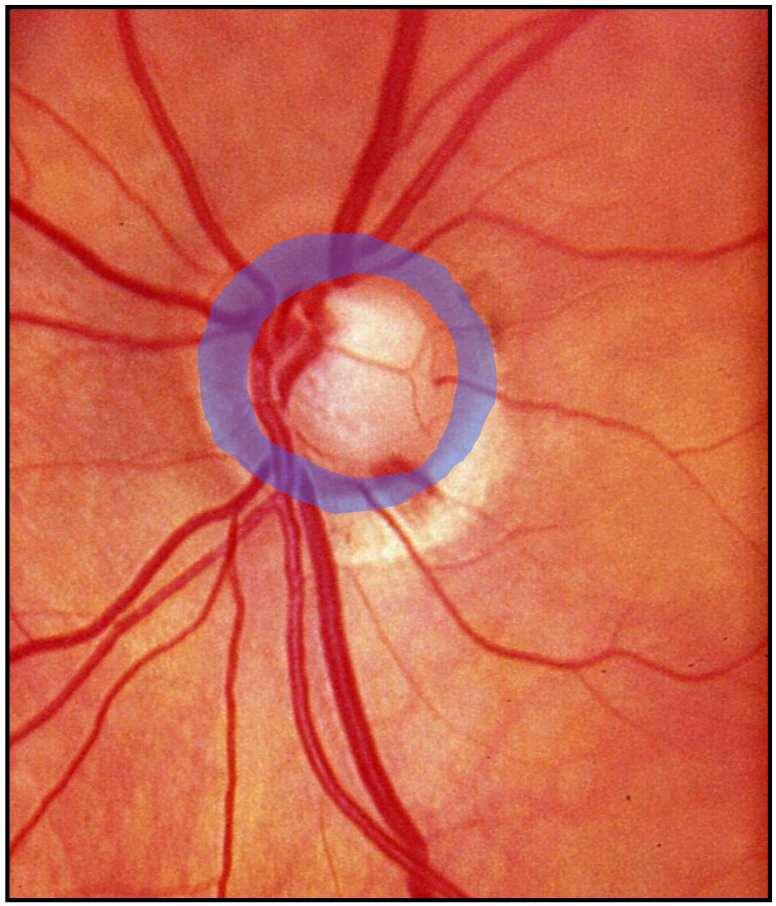
1 Observe the scleral Ring to identify the limits of the optic disc and its size



This section was developed by Robert N. Weinreb, MD, Felipe Medeiros, MD, and Remo Susanna Jr, MD.

1 Observe the scleral Ring to identify the limits of the optic disc and its size

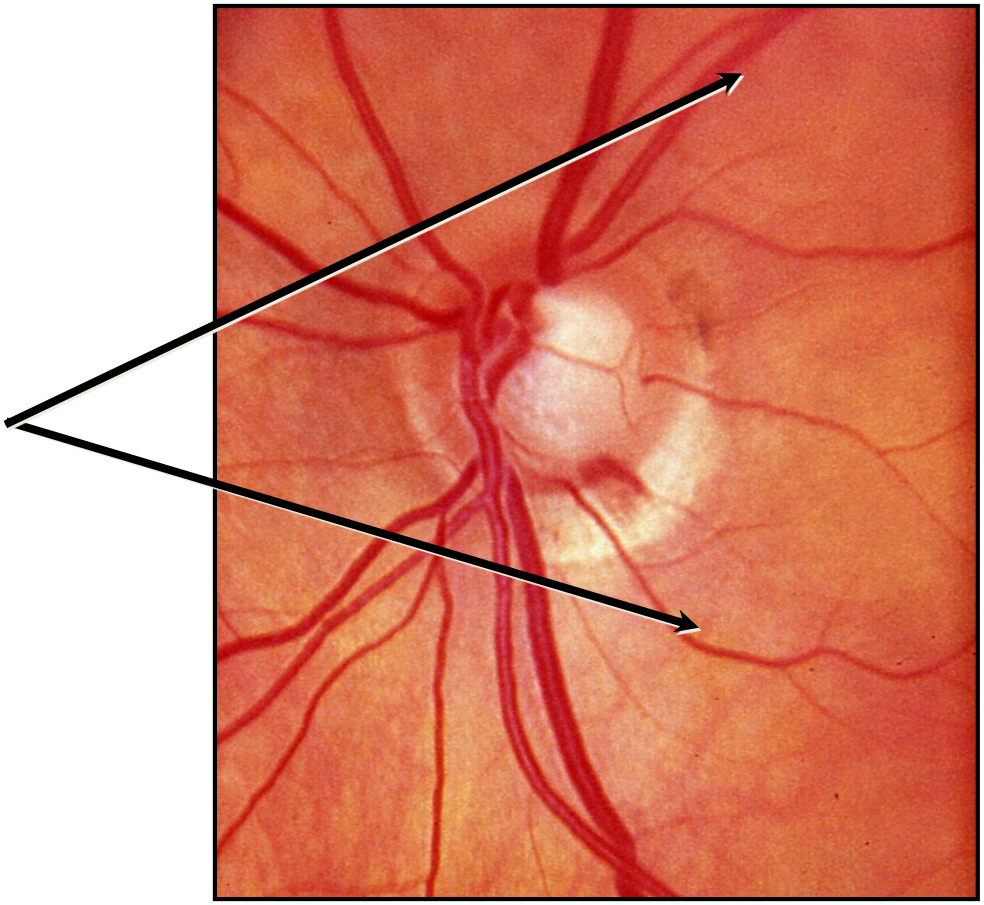
2 Identify the size of the Rim



1 Observe the scleral Ring to identify the limits of the optic disc and its size

2 Identify the size of the Rim

3 Examine the Retinal nerve fiber layer

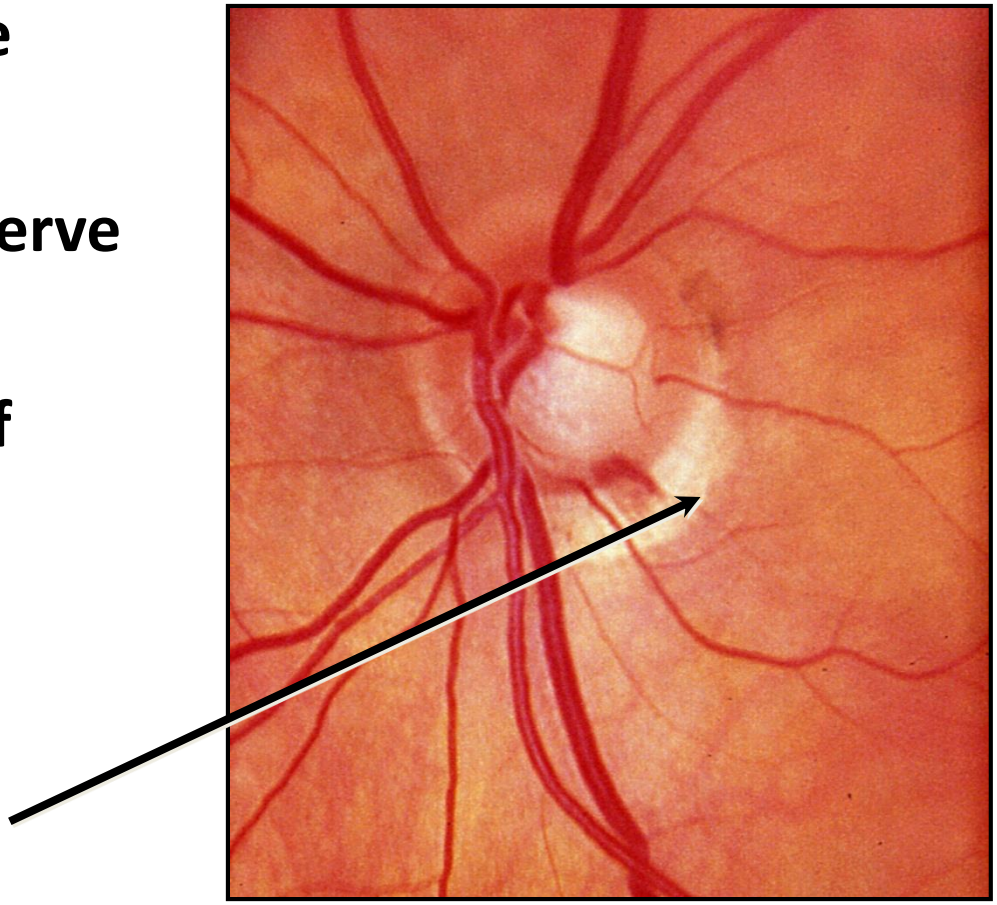


1 Observe the scleral Ring to identify the limits of the optic disc and its size

2 Identify the size of the Rim

3 Examine the Retinal nerve fiber layer

4 Examine the Region of parapapillary atrophy



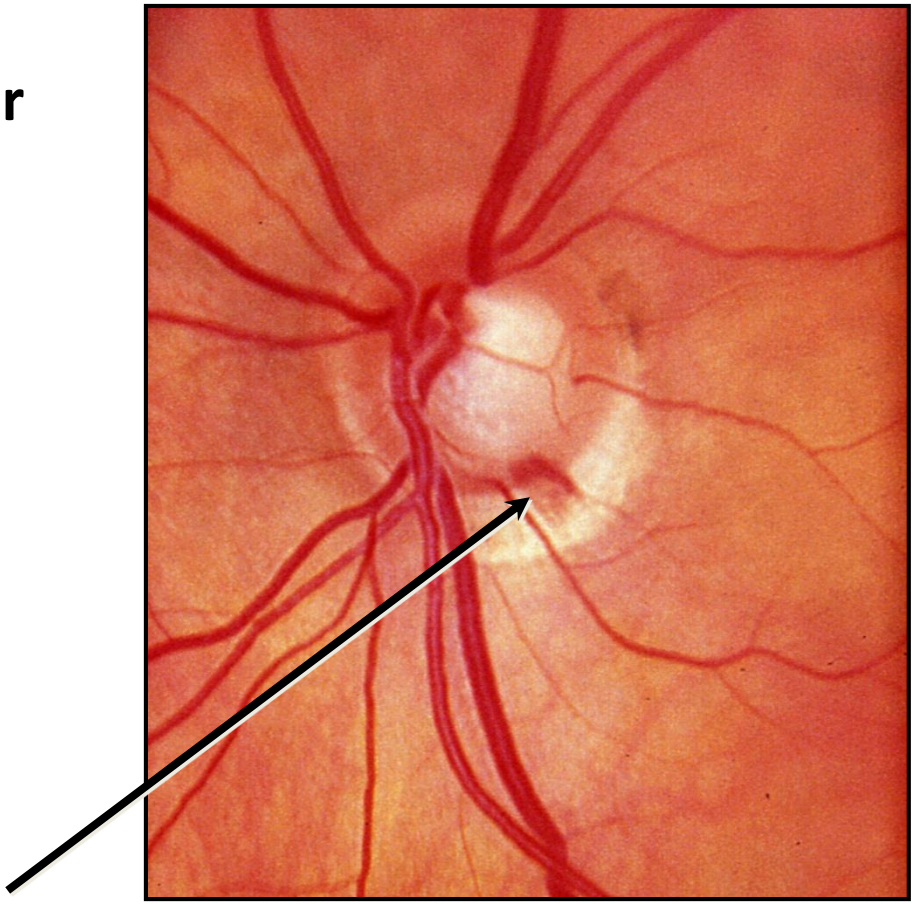
1 Observe the scleral Ring to identify the limits of the optic disc and its size

2 Identify the size of the Rim

3 Examine the Retinal nerve fiber layer

4 Examine the Region of parapapillary atrophy

5 Look for Retinal and optic disc hemorrhages

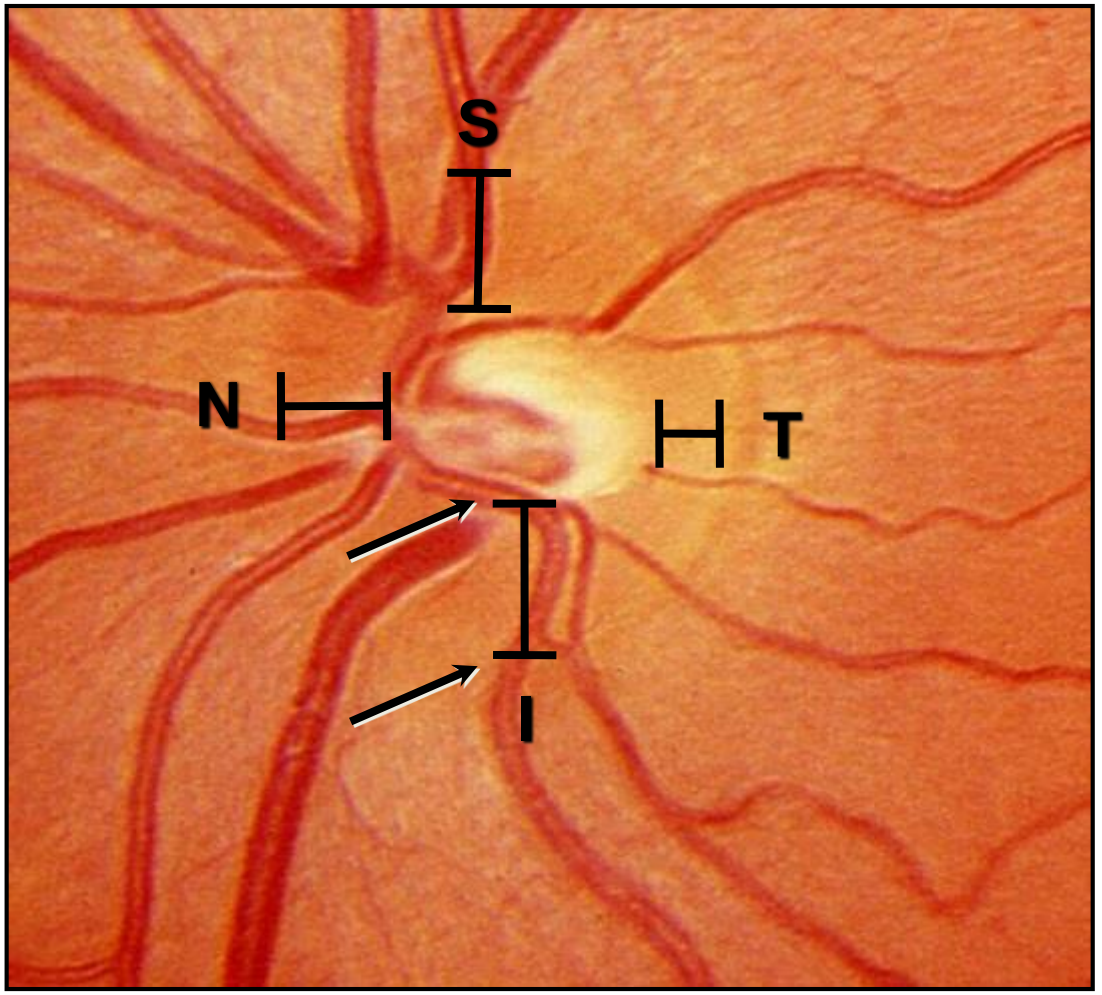


ISNT RULE

Rim width

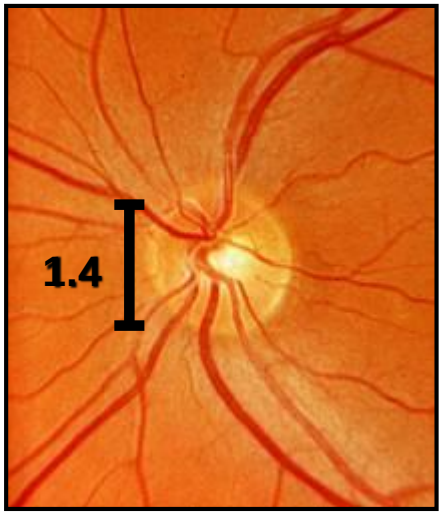
Distance between border of disc and position of blood vessel bending

ISNT rule
Inferior >
Superior >
Nasal >
Temporal



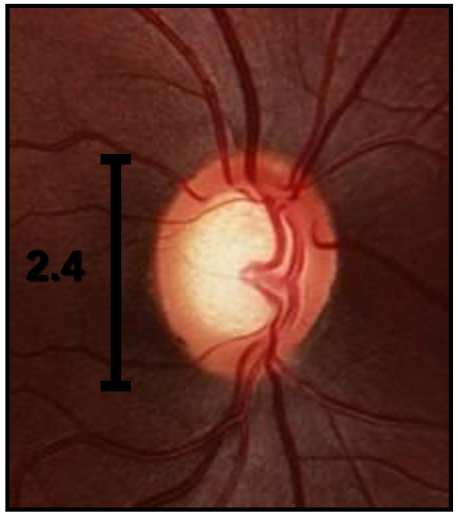
Optic Disc Size

Size of cup varies with size of disc
Large discs have large cups in healthy eyes



Small

Average



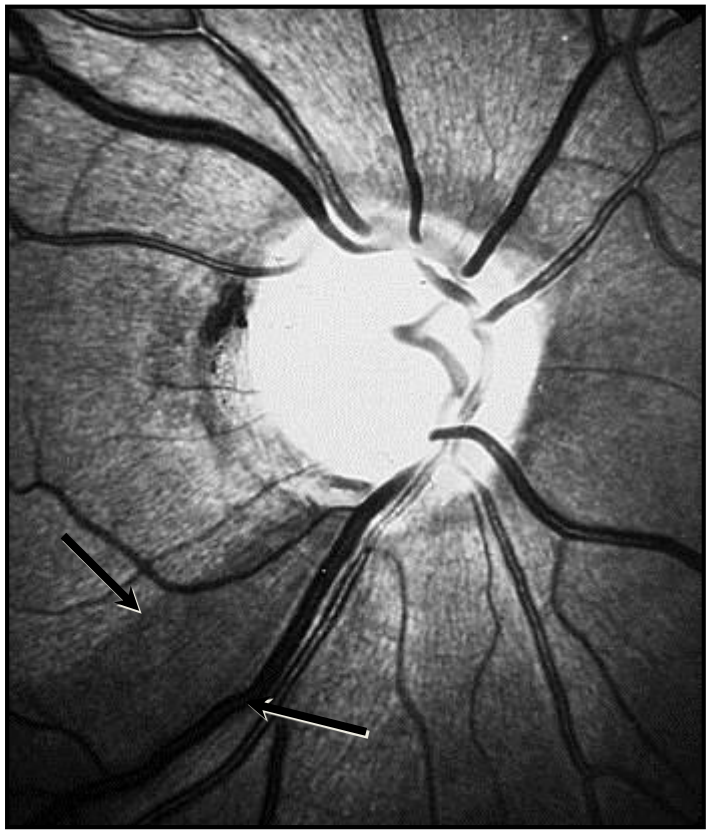
Large

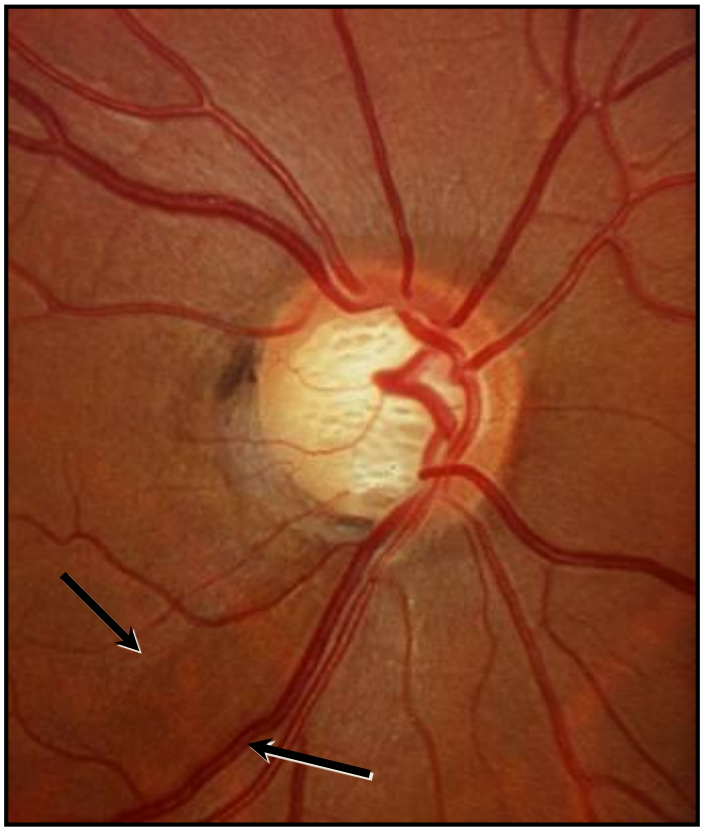
Identify small and large optic discs

Small discs: avg vertical diameter <1.5 mm

Large discs: avg vertical diameter >2.2 mm

Localized RNFL Loss





Localized RNFL defect

Wedge-shaped dark area

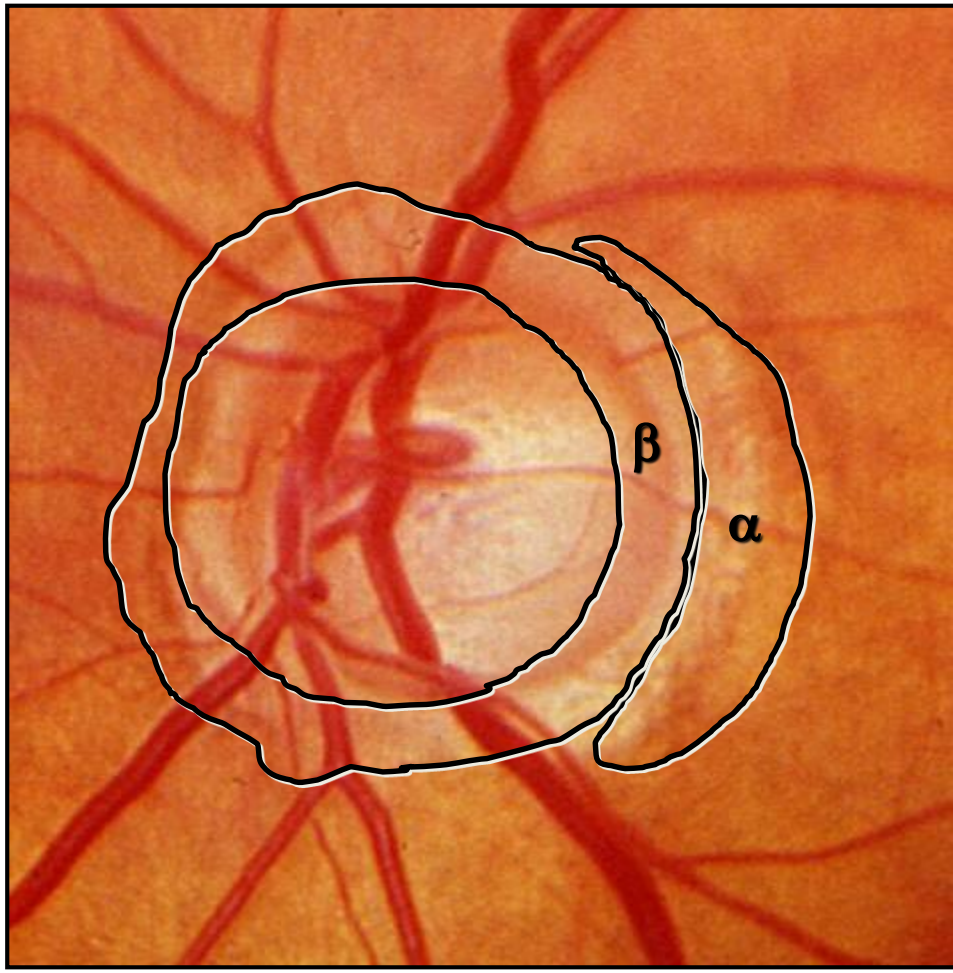
Parapapillary Atrophy

Alpha zone

- Hypo- and hyper-pigmented areas
- Present in normal as well as in glaucomatous eyes

Beta zone

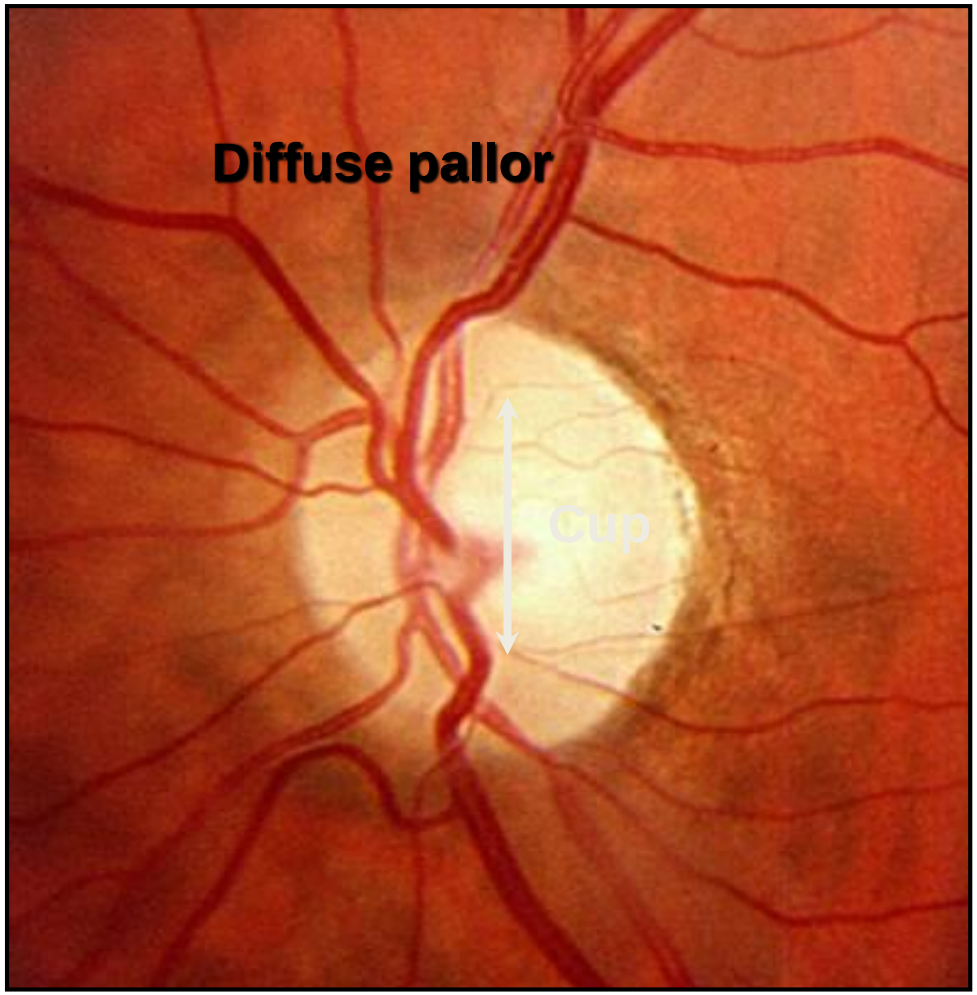
- Atrophy of the retinal pigment epithelium (RPE) and choriocapillaris
  - Large choroidal vessels become visible
- More common in glaucomatous eyes



Observe the color of the rim to identify pallor

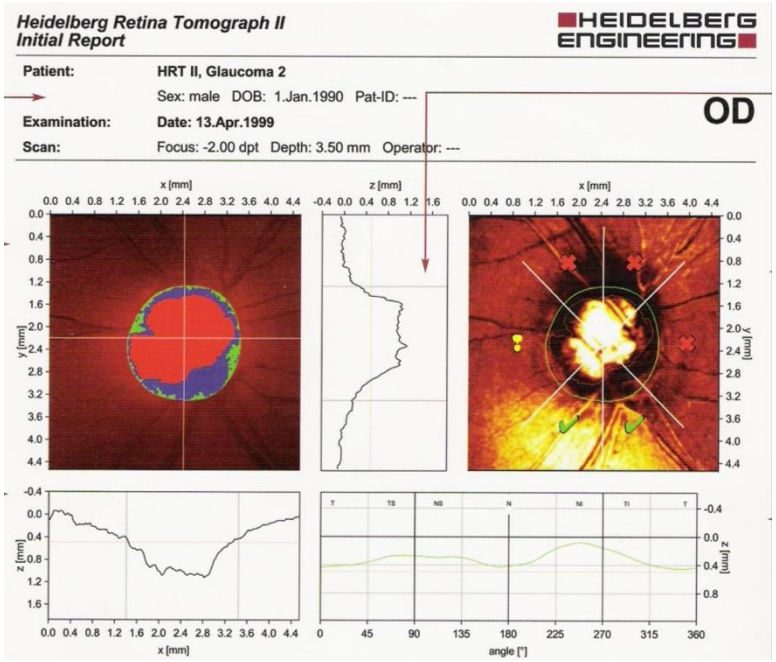
A pale rim increases the likelihood for a non-glaucomatous optic neuropathy

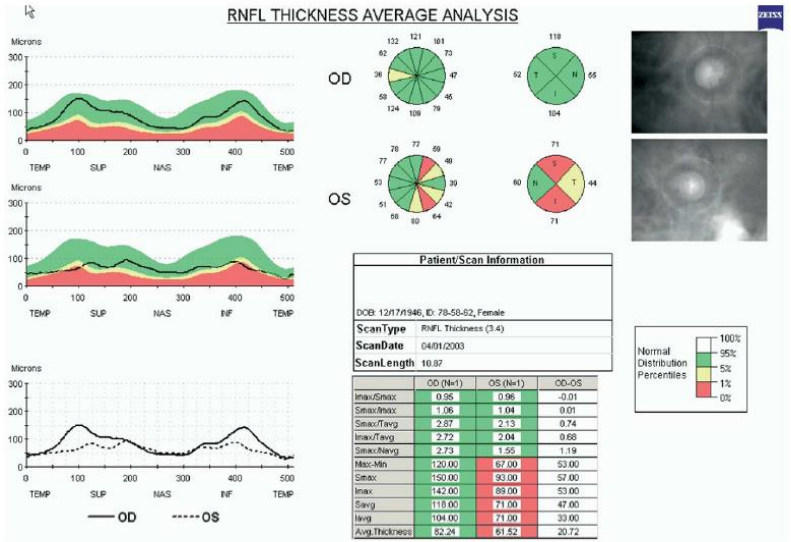
Pallor



Pallor > cup

Non-glaucomatous neuropathy





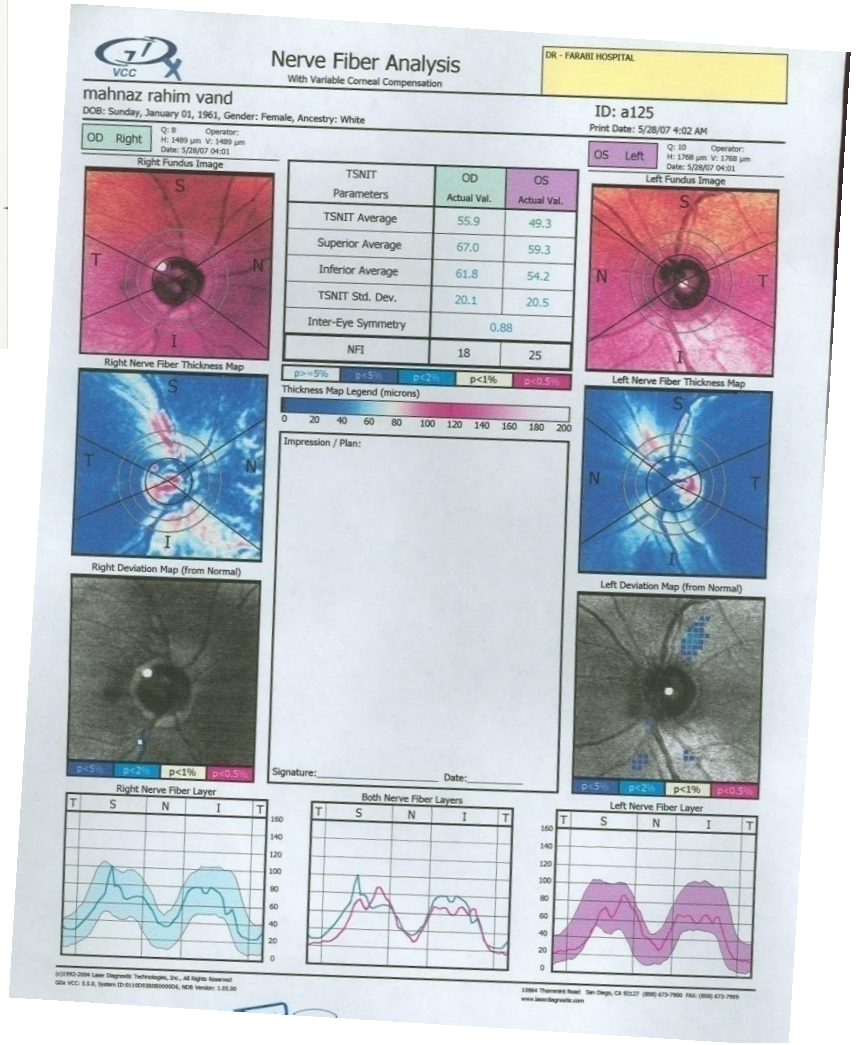
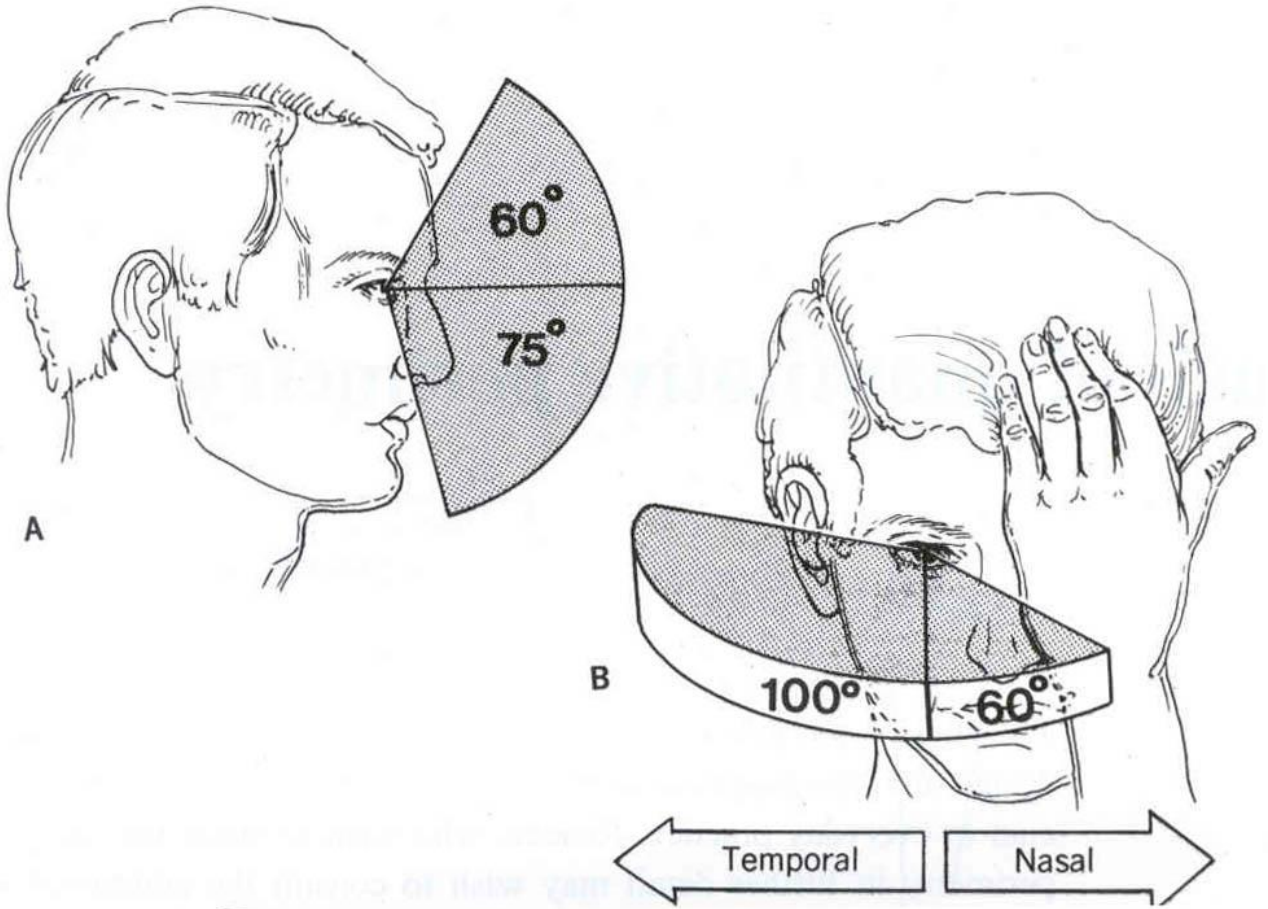


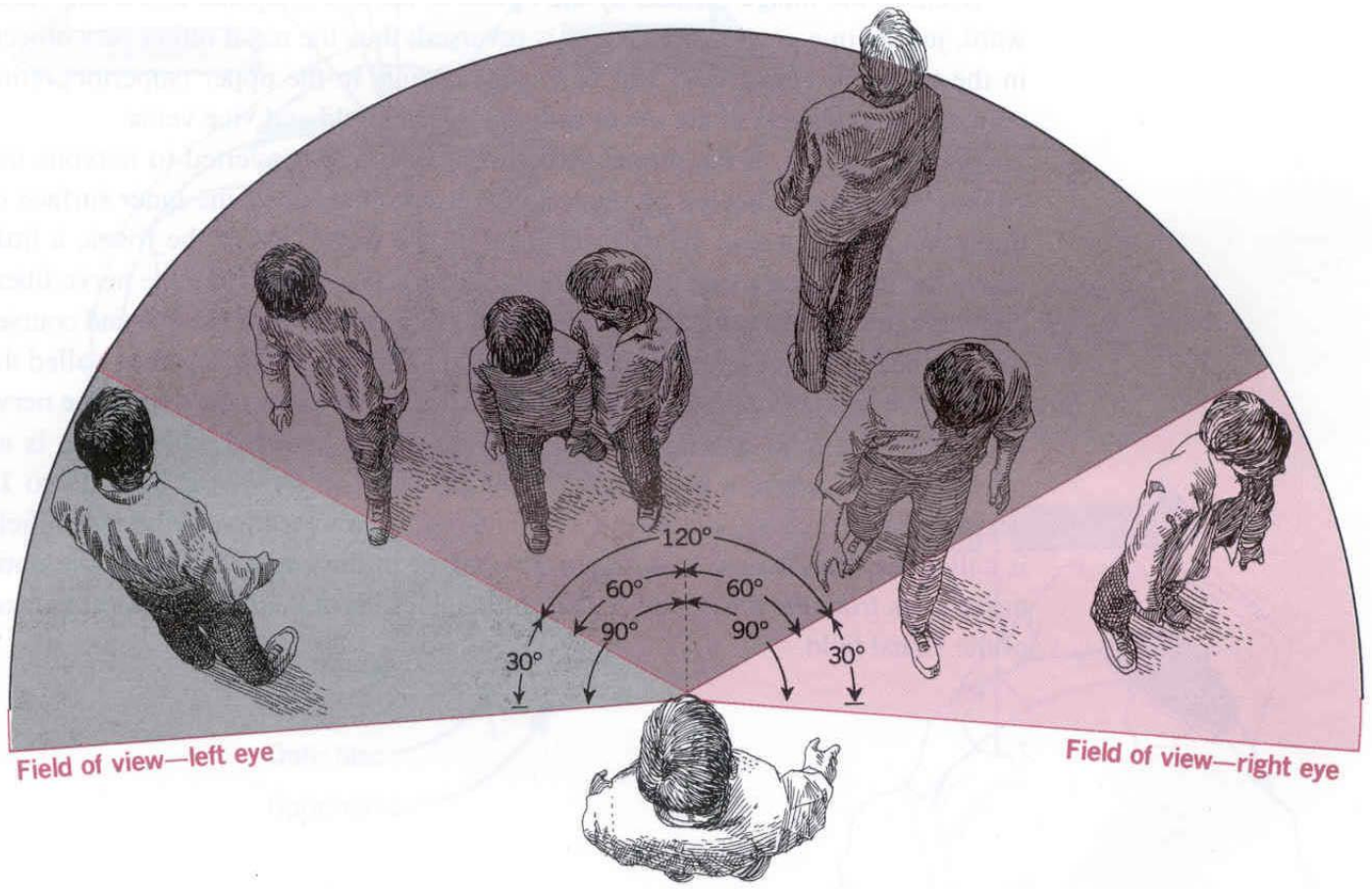
Figure 1. StratusOCT Fast retinal nerve fiber layer (RNFL) scan from a patient with glaucoma in the left eye only. Areas that are colored in green indicate normal RNFL thickness. Areas with RNFL thicknesses expected in <5% of age-matched controls are colored in yellow, and areas with RNFL thicknesses expected in <1% of age-matched controls are colored in orange. INF = inferior; NAS = nasal; OD = right eye; OS = left eye; SUP = superior; TEMP = temporal.

Perimetry

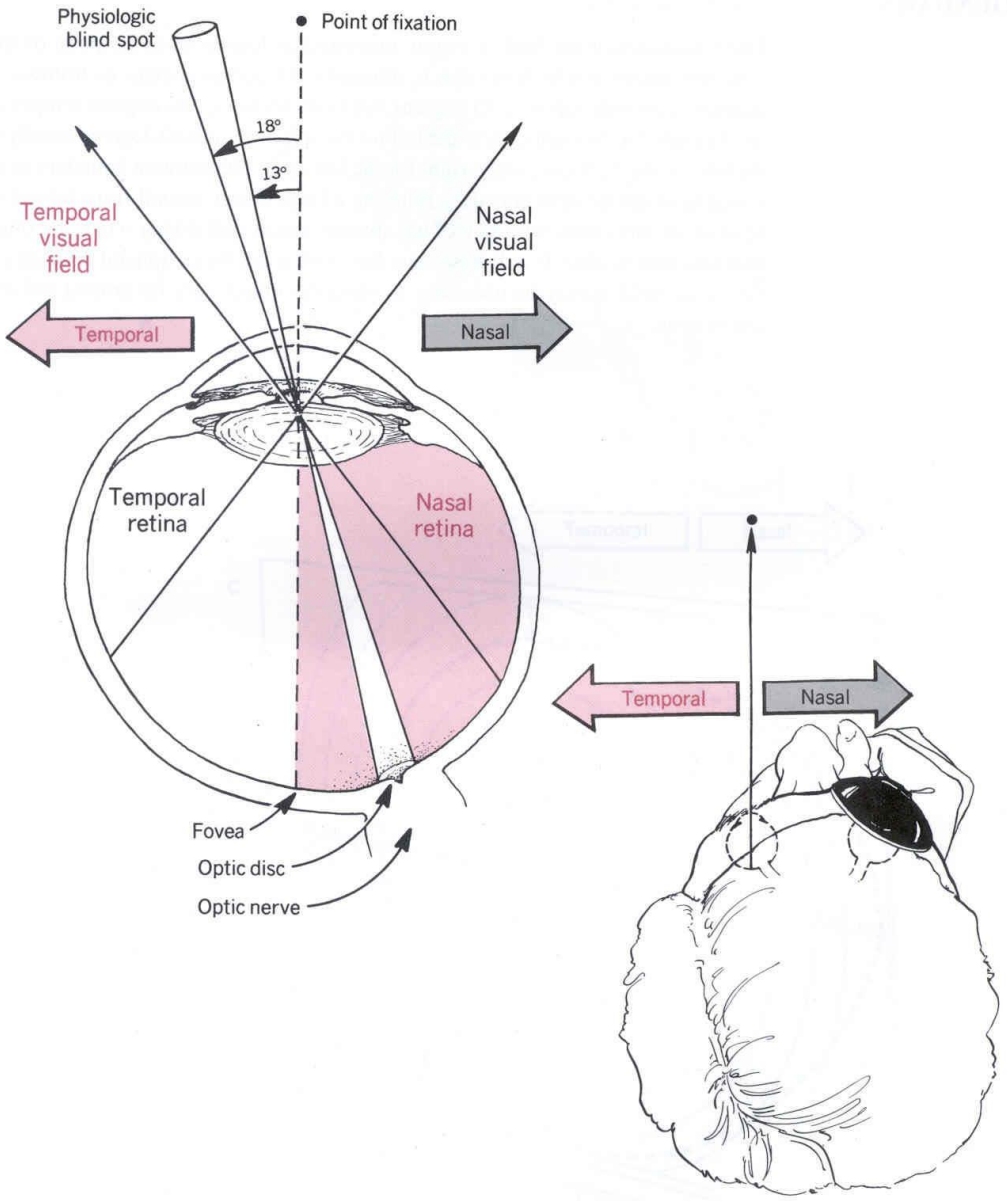
Boundaries of the normal human visual field



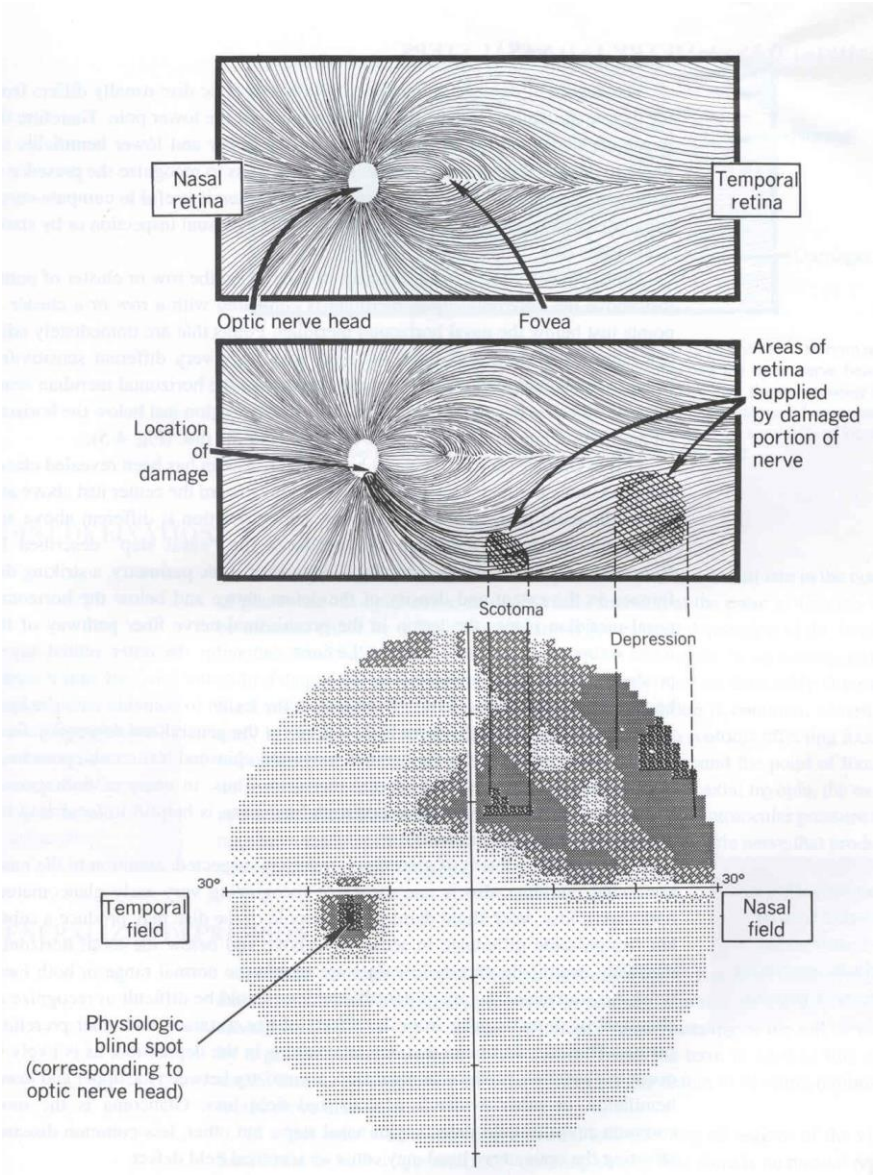
Binocular and monocular visual fields



Visual space orientation on the retina and projection of the physiologic blind spot



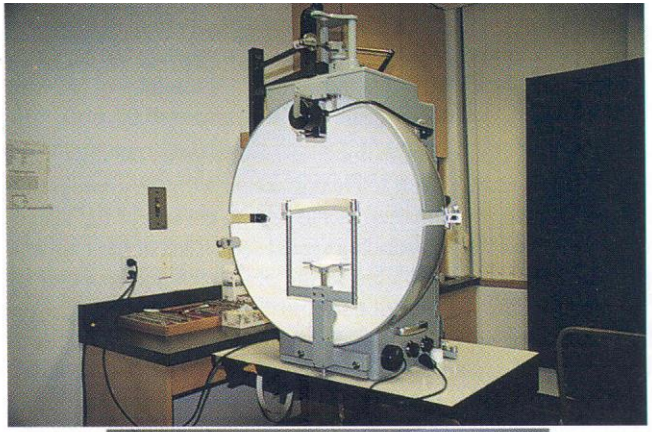
Schematic view of ONH damage and subsequent VF defect



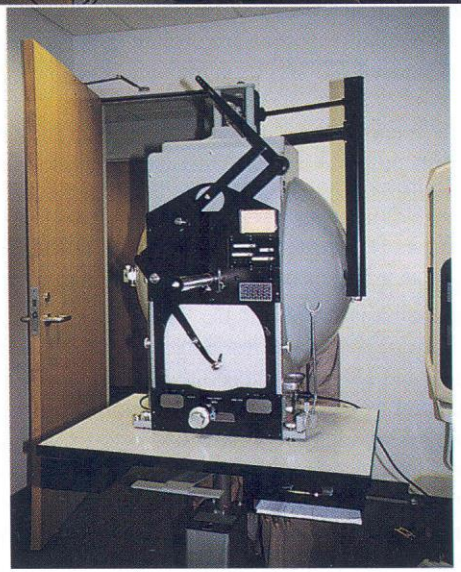
Glaucomatous Visual Field Defects

- Up to 50% of ganglion cells (or NFLs) may be lost before a definite VF defect develops
- The superior and inferior poles of the ONH are most susceptible to glaucomatous damage
- Temporal fibers (nasal field) do not cross the horizontal meridian
- Glaucomatous VF defects are not specific to or diagnostic of glaucoma
- R/O causes other than glaucoma when VF findings are inconsistent with clinical findings
- Always consider learning effect (obtain 2-3 VFs to reach a baseline field)

Perimeters



A



B

Figure 10-5 Goldmann perimeter for visual field examination as viewed from the patient's side **(A)** and the examiner's side **(B)**.

Figure 9-4 Humphrey 700 series perimeter.

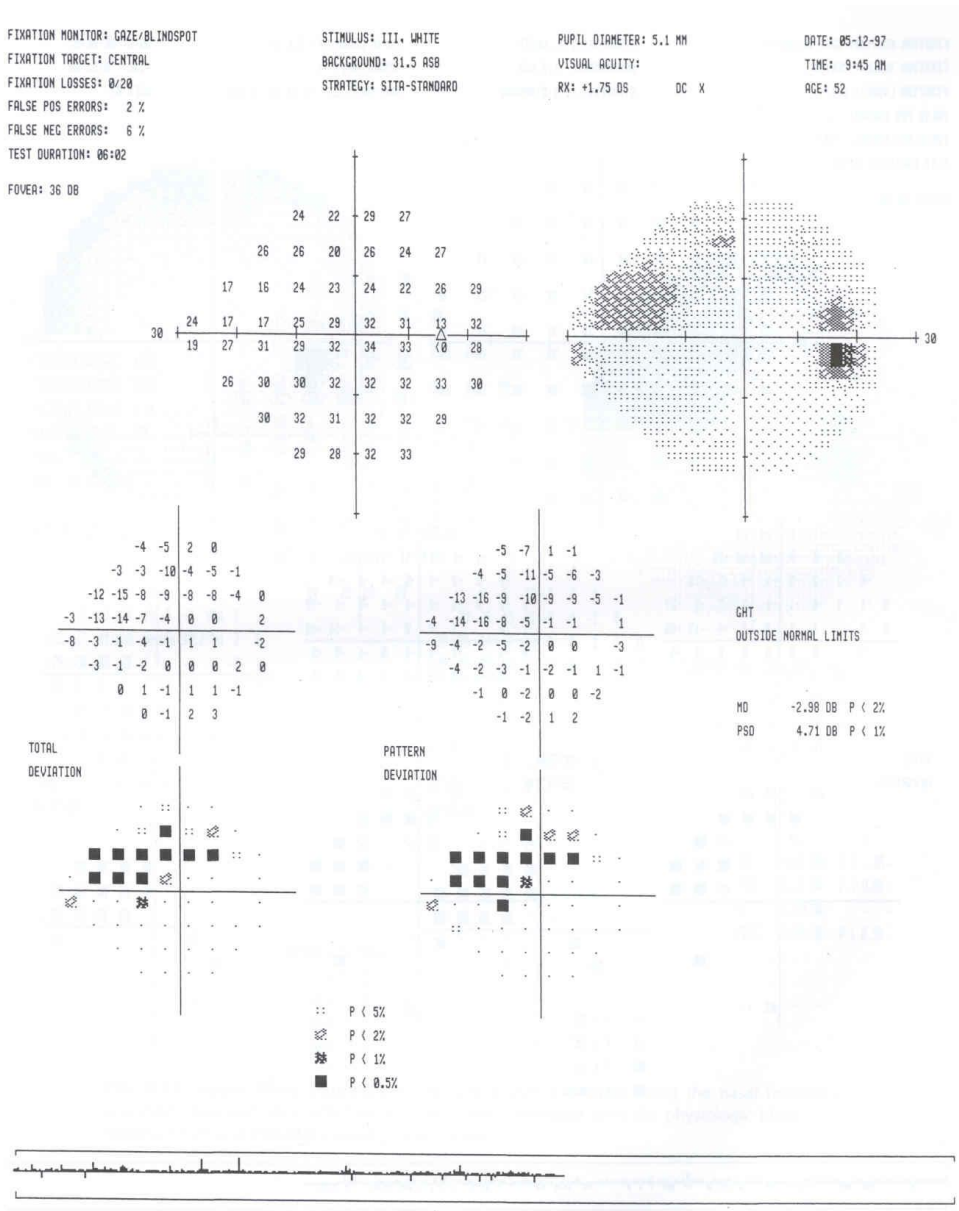


FIG. 4-9. Broad, Relative Upper Arcute Scotoma. Unlike the preceding examples, the field has no locations of absolute loss at which the 0-dB stimulus was not seen. In fact, all locations have threshold values of 15 dB or greater. Although the greyscale shows the mildness of the defect, the probability plots show that the abnormality is statistically strong at more locations than might be suspected from the greyscale alone. The Glaucoma Hemifield Test (GHT) and the PSD index also reflect the localized abnormality of the upper hemifield (SITA-Standard 24-2; right eye).

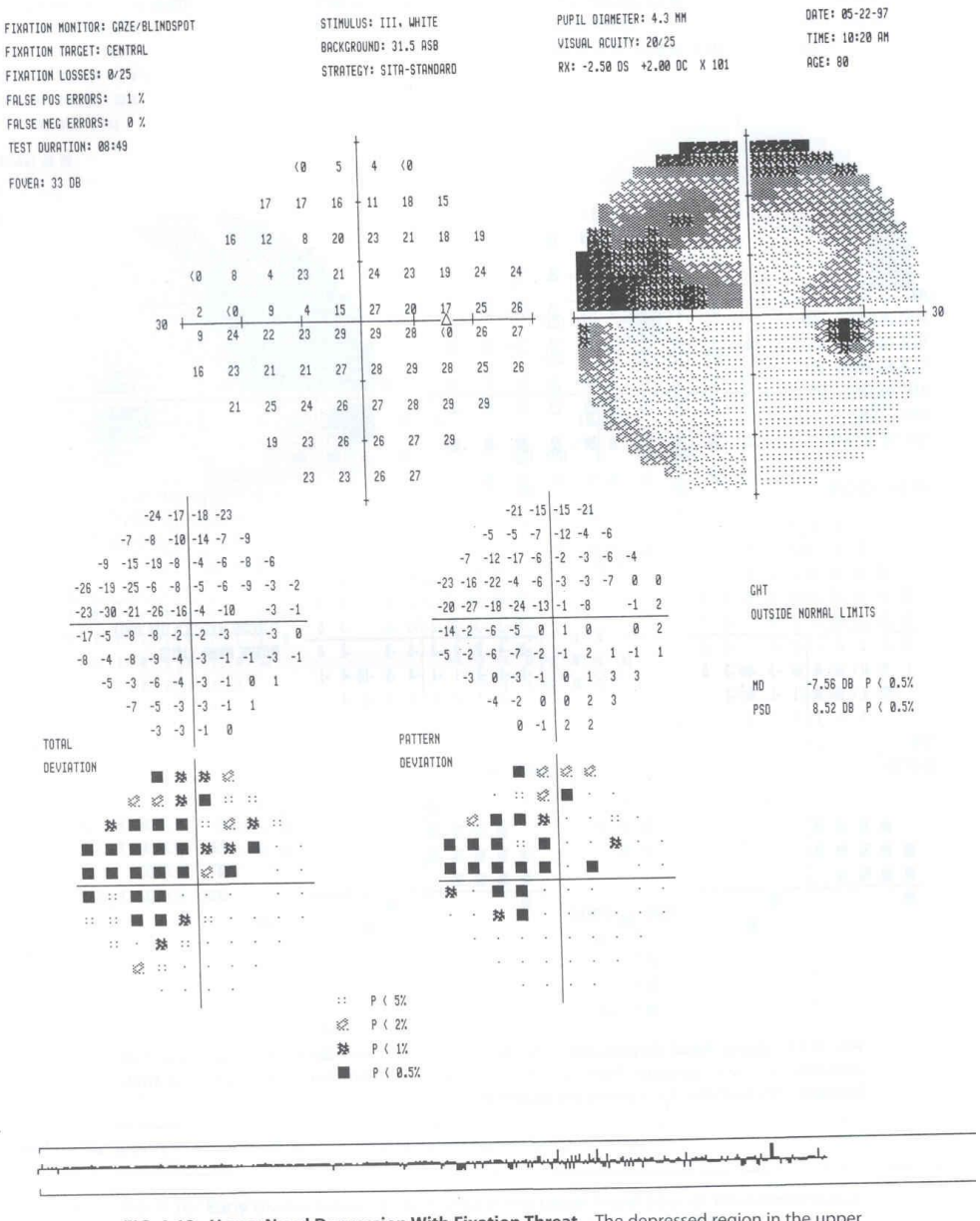


FIG. 4-12. Upper Nasal Depression With Fixation Threat. The depressed region in the upper hemifield is more extensive and approaches the point of fixation. The foveal threshold itself is normal (33 dB), and visual acuity is 20/20. The lower nasal quadrant is also abnormal, the extent of which is more evident on the deviation probability plots than on the greyscale (right eye).

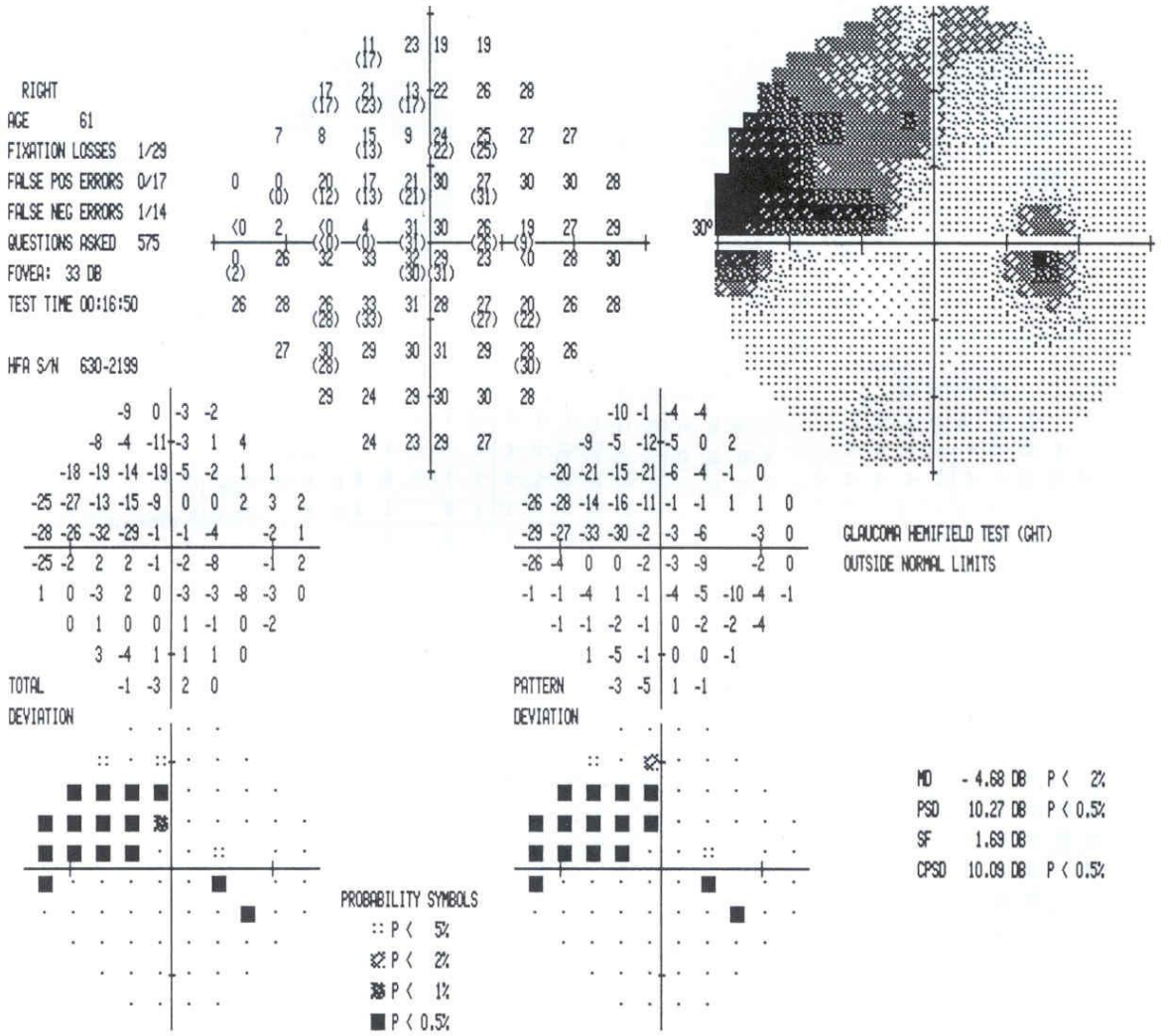


FIG. 4-11. Upper Nasal Depression. The depression is densest along the nasal horizontal meridian, does not approach fixation, and is not connected with the physiologic blind spot. Between 15% and 50% of the points are abnormal.

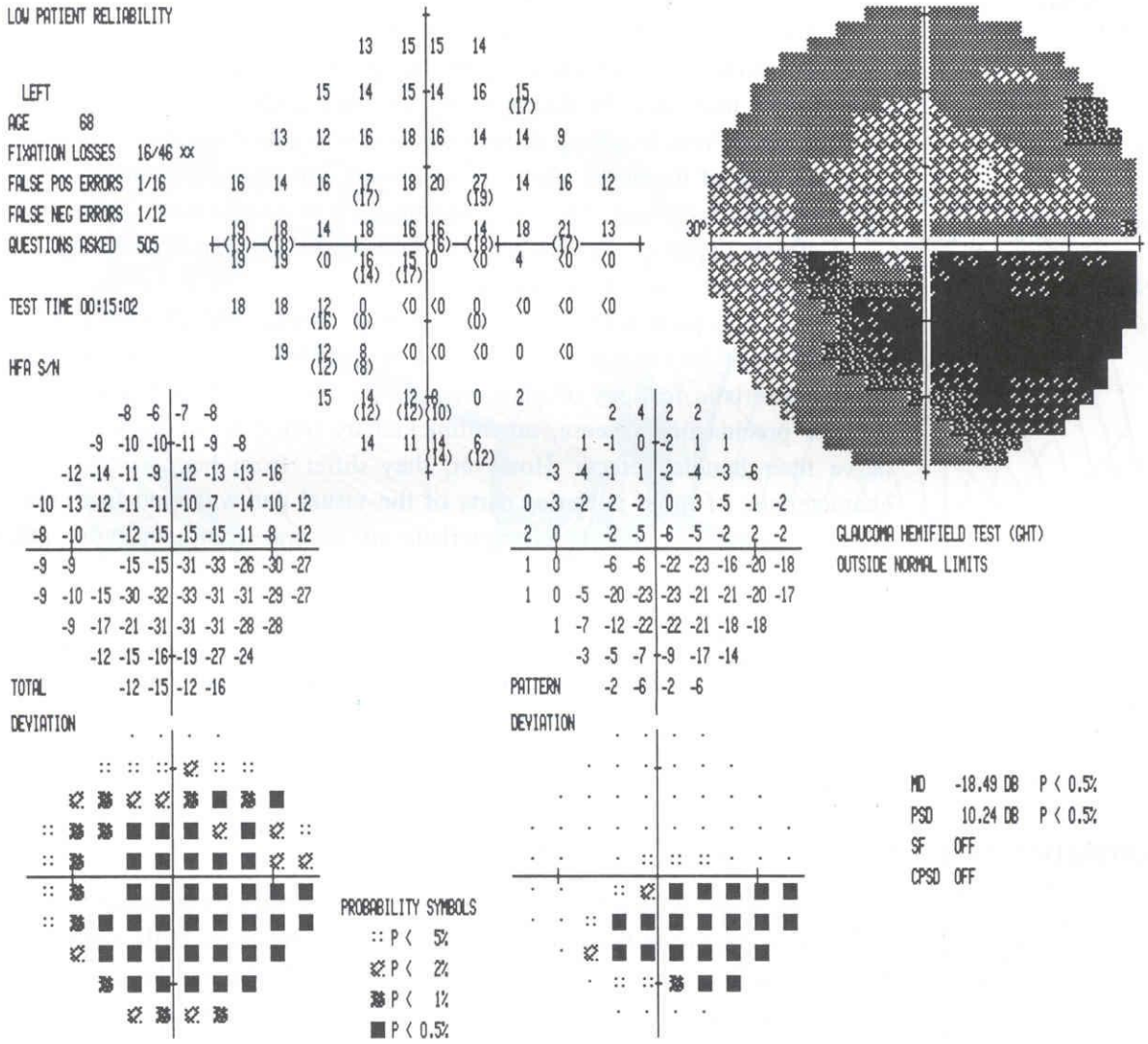


FIG. 4-2. Diffuse, Widespread Loss of Visual Field Caused by Glaucoma. There are no normal locations, but the lower nasal and inferior arcuate regions are much more densely abnormal (left eye).

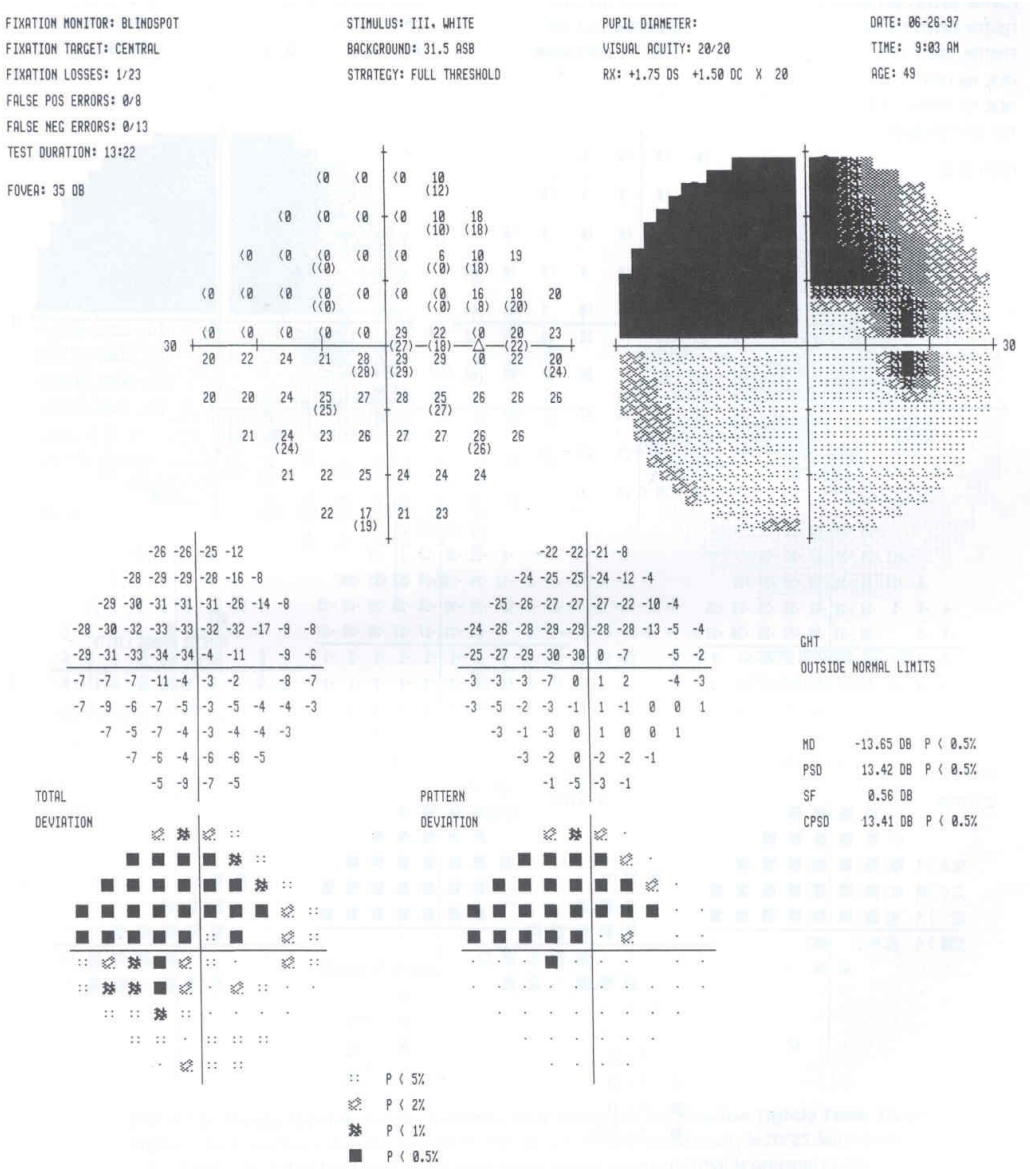


FIG. 4-13. Dense Upper Arcute Defect. The arcuate nature of the defect is evident. Although it comes close to fixation from the nasal side, the foveal threshold is normal (35 dB), and visual acuity is 20/20. Note the characteristic features: the defect clearly emanates from the physiologic blind spot, it becomes broader as it arcs over the point of fixation into the nasal field, and it comes closer to the foveal point from the nasal side than it does from the temporal side. The lower nasal quadrant also is depressed, and more than half the points are abnormal (Full Threshold 30-2; right eye).

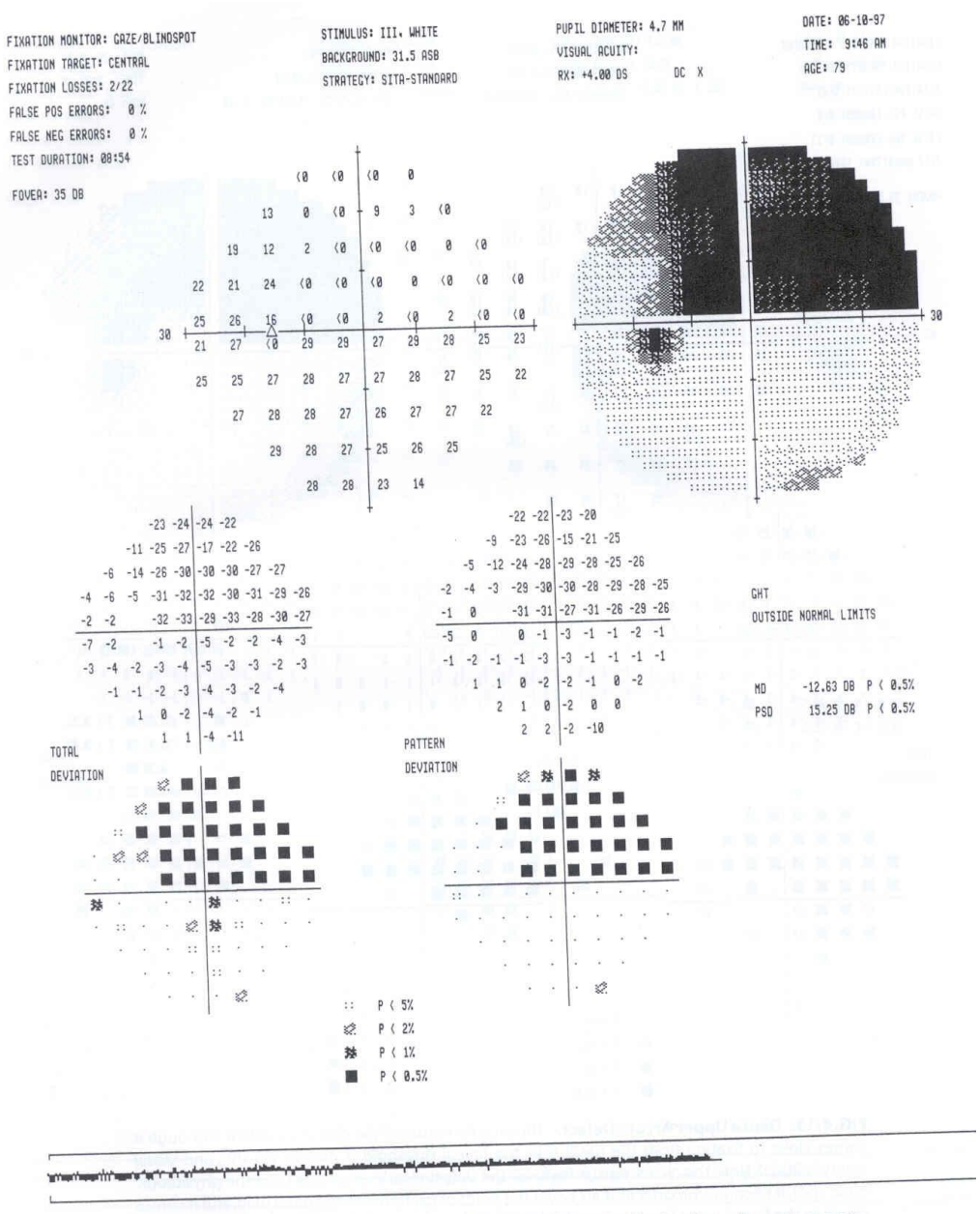


FIG. 4-14. Absolute Altitudinal Defect. The dense defect splits fixation from above. Its arcuate nature still is evident from the peninsula of retained vision extending upward on the temporal side of the physiologic blind spot. Of note is the striking contrast between the absolute visual loss in the upper half of the field and the completely normal lower half of the field. The foveal threshold is normal at 35 dB (SITA-Standard 30-2; left eye).

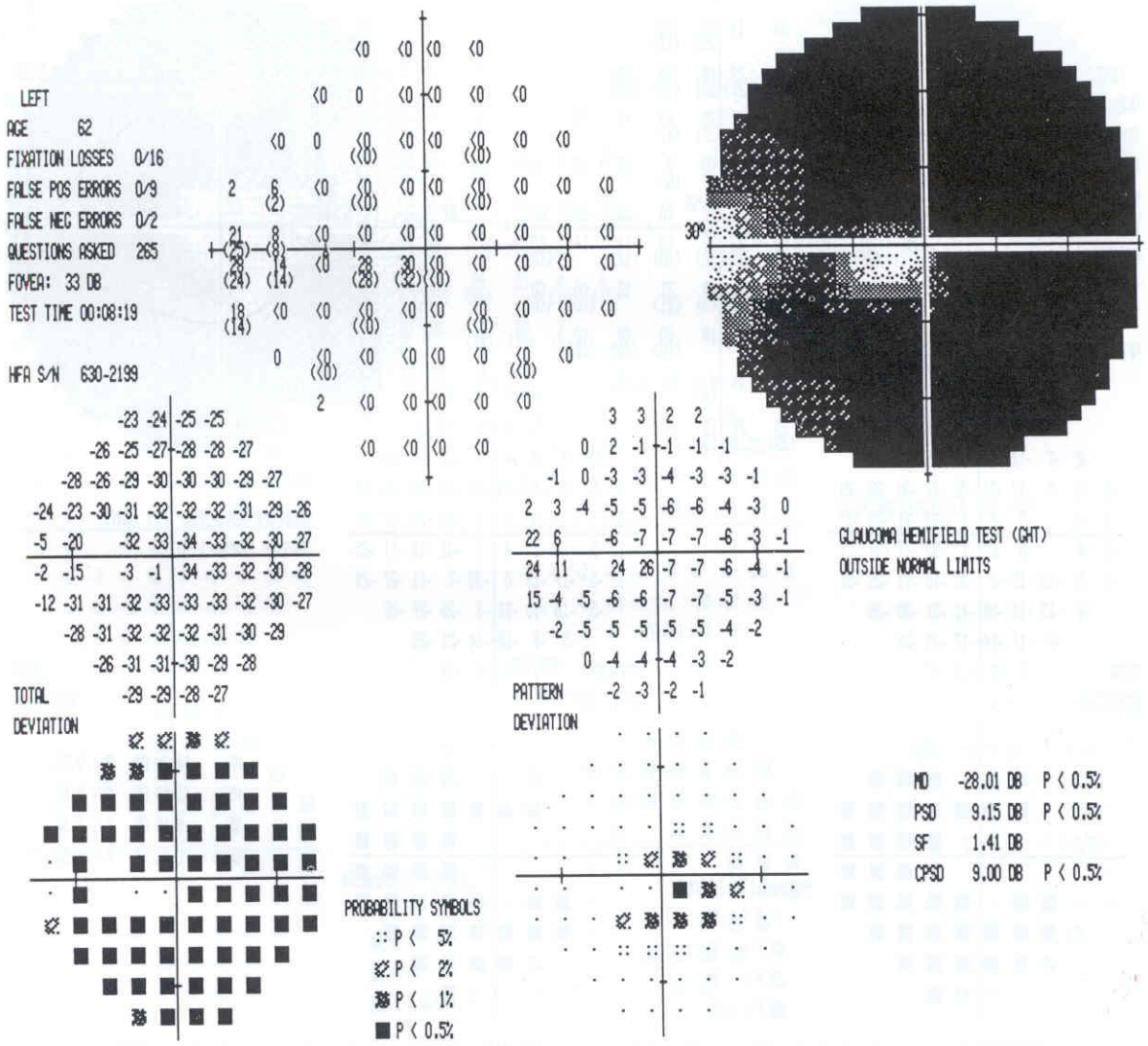


FIG. 4-20. More Advanced Glaucoma. Only a small central island and a temporal island remain, with broad, dense abnormality in both the upper and lower arcuate regions extending nasally from the blind spot. The foveal threshold is good (33 dB, within normal limits for age), and visual acuity is 20/20 (left eye).

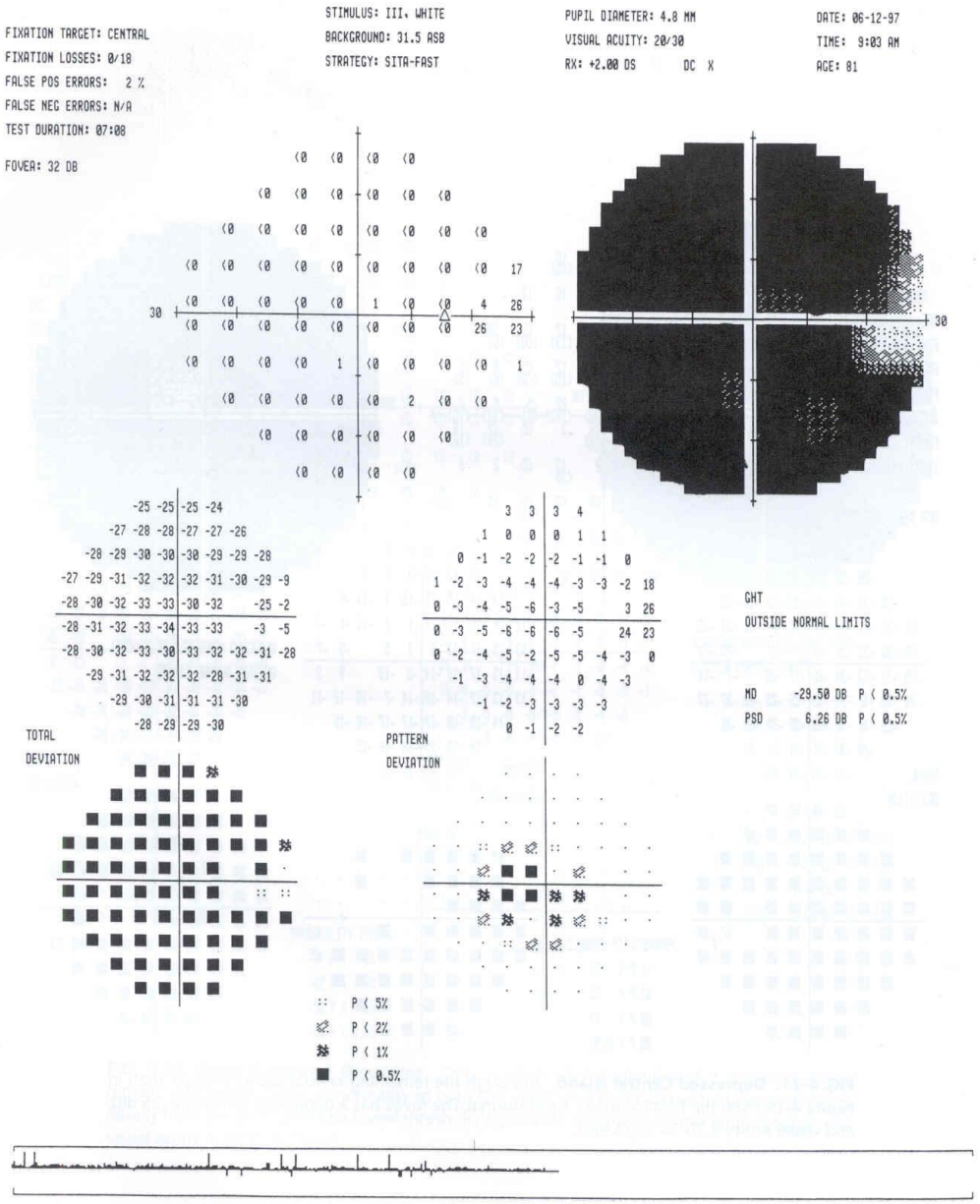


FIG. 4-22. Advanced Glaucoma. A few points with reduced sensitivity remain temporally. This remaining temporal island of vision would not have been detected with a 24-2 pattern. Remarkably, there also is residual vision in a tiny central island smaller than 3 degrees in radius, with a foveal threshold of 32 dB and visual acuity of 20/30; very steady fixation is recorded by the gaze tracker (SITA-Fast 30-2; right eye).

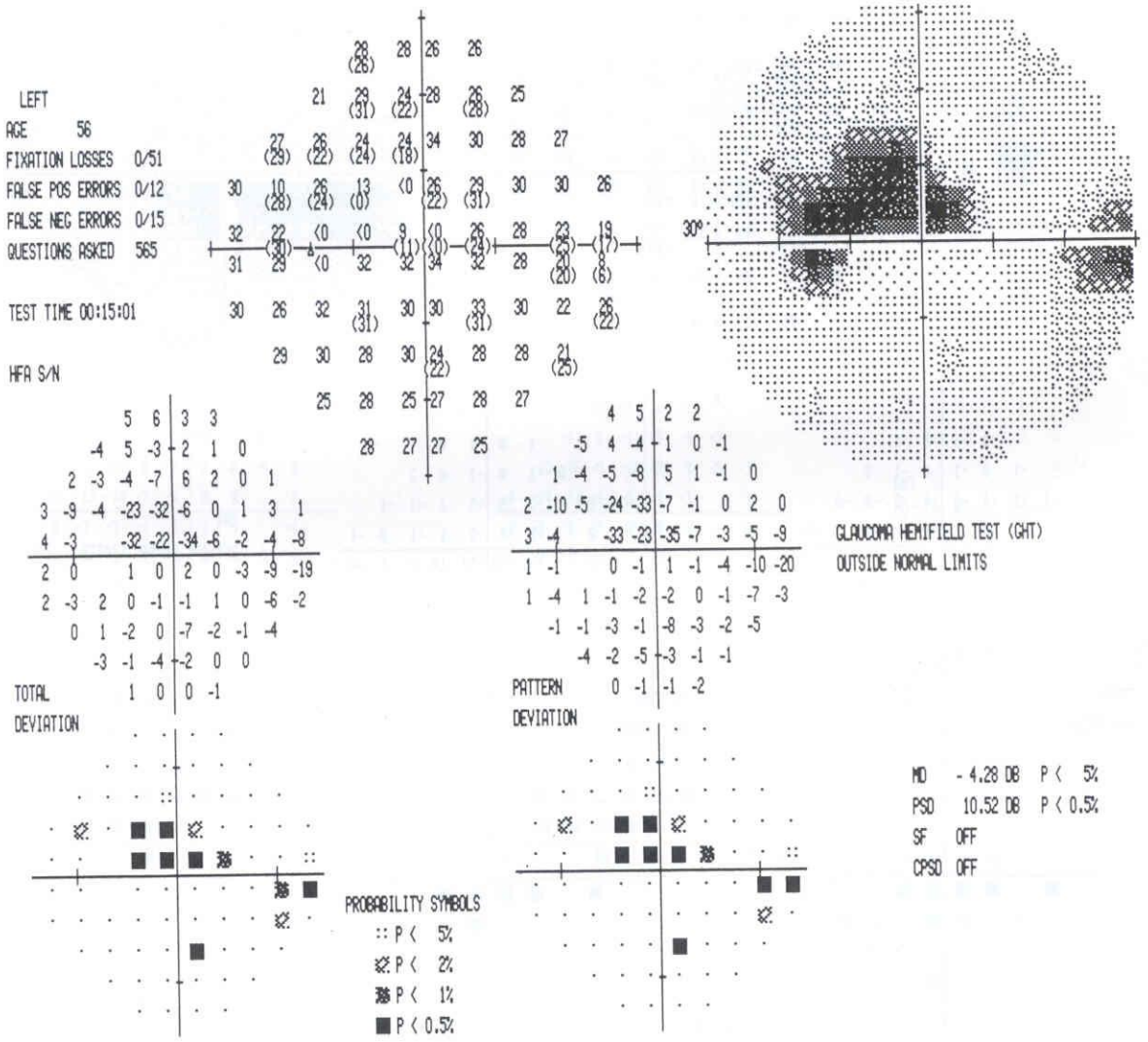


FIG. 4-7. Dense Localized Defect that Splits Fixation. A sharply localized, absolute defect impinges on fixation, but a small number of points is involved. Except for this sharply localized defect and the relative depression at the nasal edge, most of the points are unaffected. Fewer than 15% of the points are abnormal. Visual acuity is 20/15 (left eye).

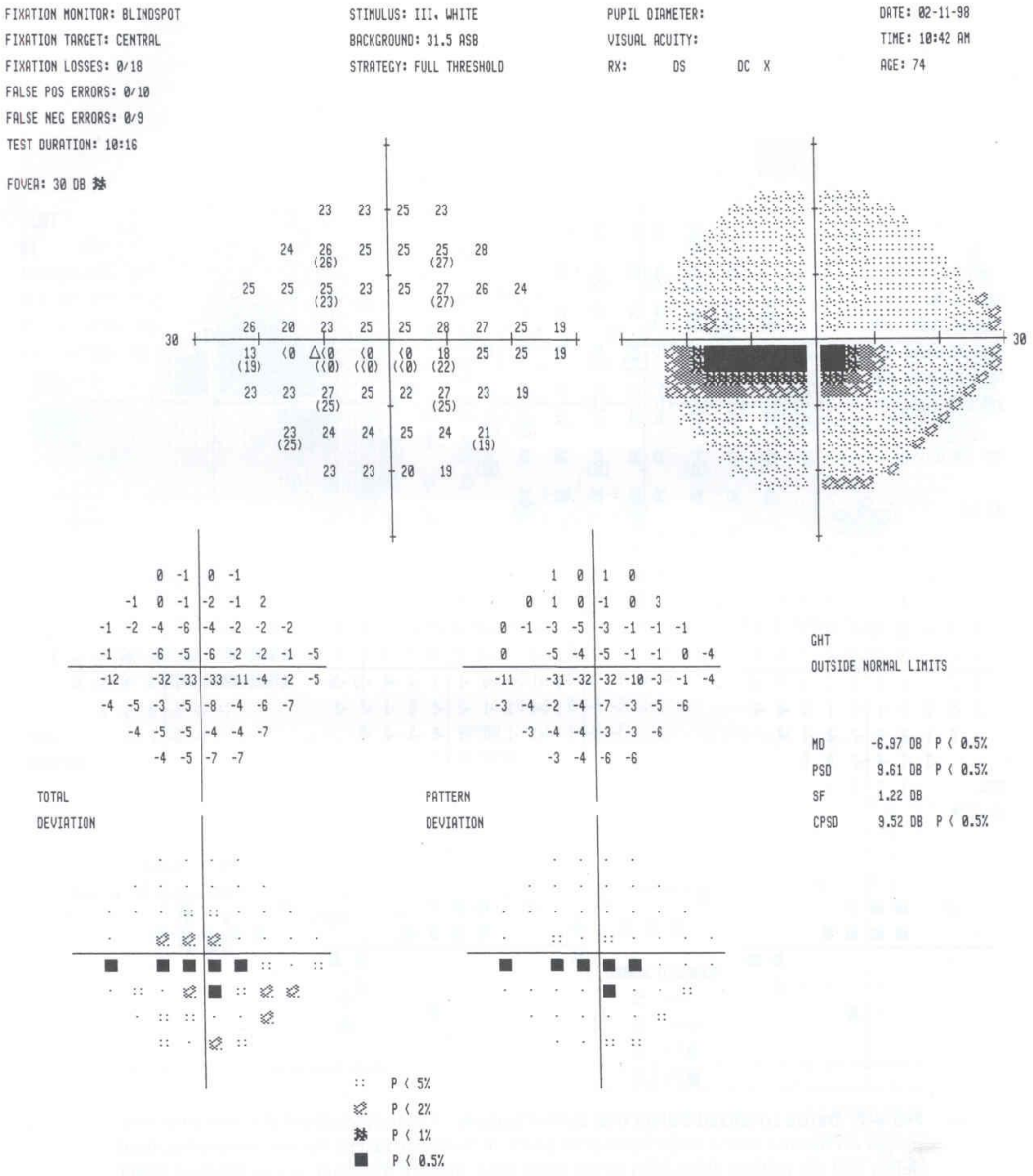


FIG. 4-8. Dense Localized Loss that Splits Fixation From Below. The patient described in Figure 4-7, and even the one described in Figure 4-1, may be asymptomatic. Defects in the inferior field, especially those that come close to the point of fixation, such as the one shown here, will cause considerable annoyance during reading. Broader involvement of the inferior field loss will cause trouble during walking (left eye).

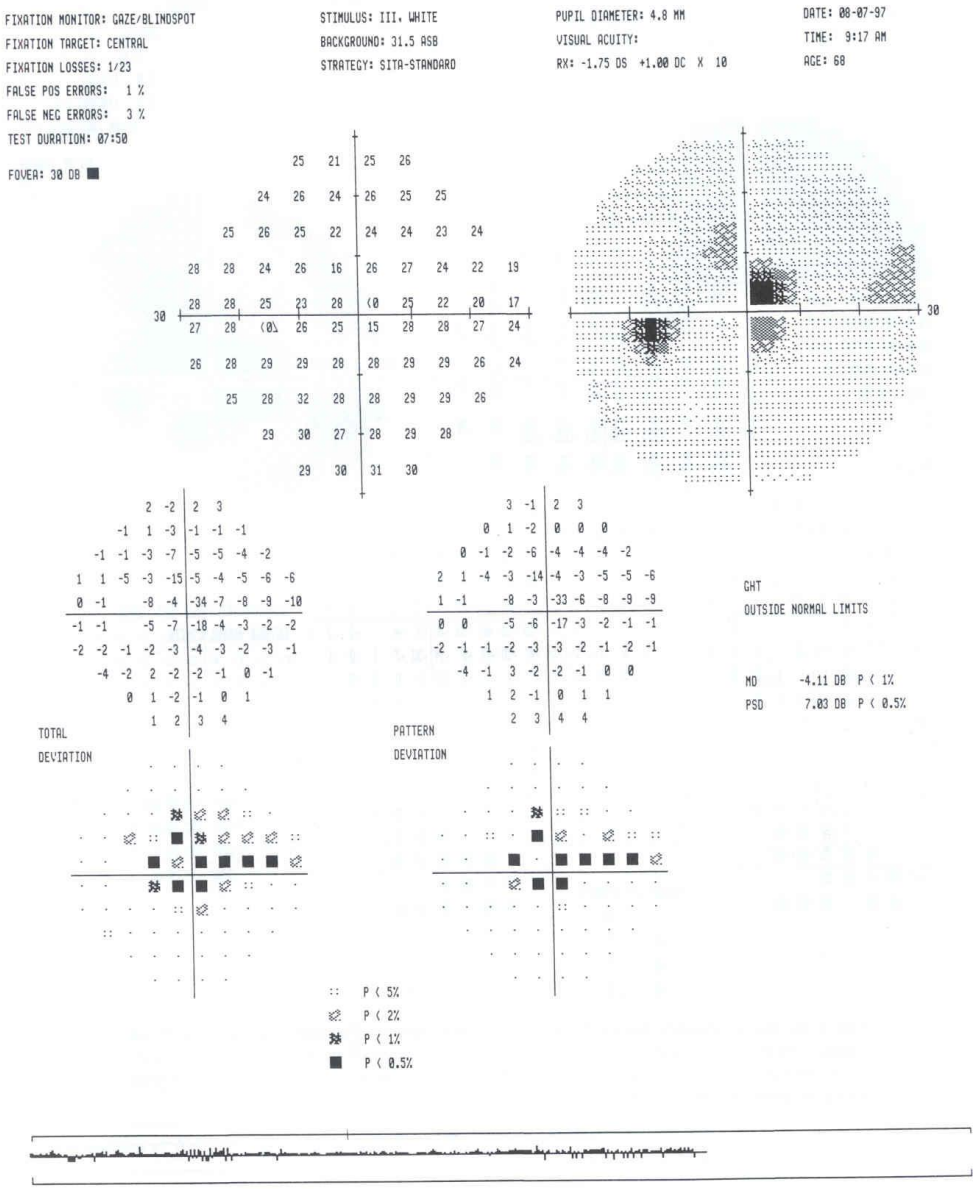
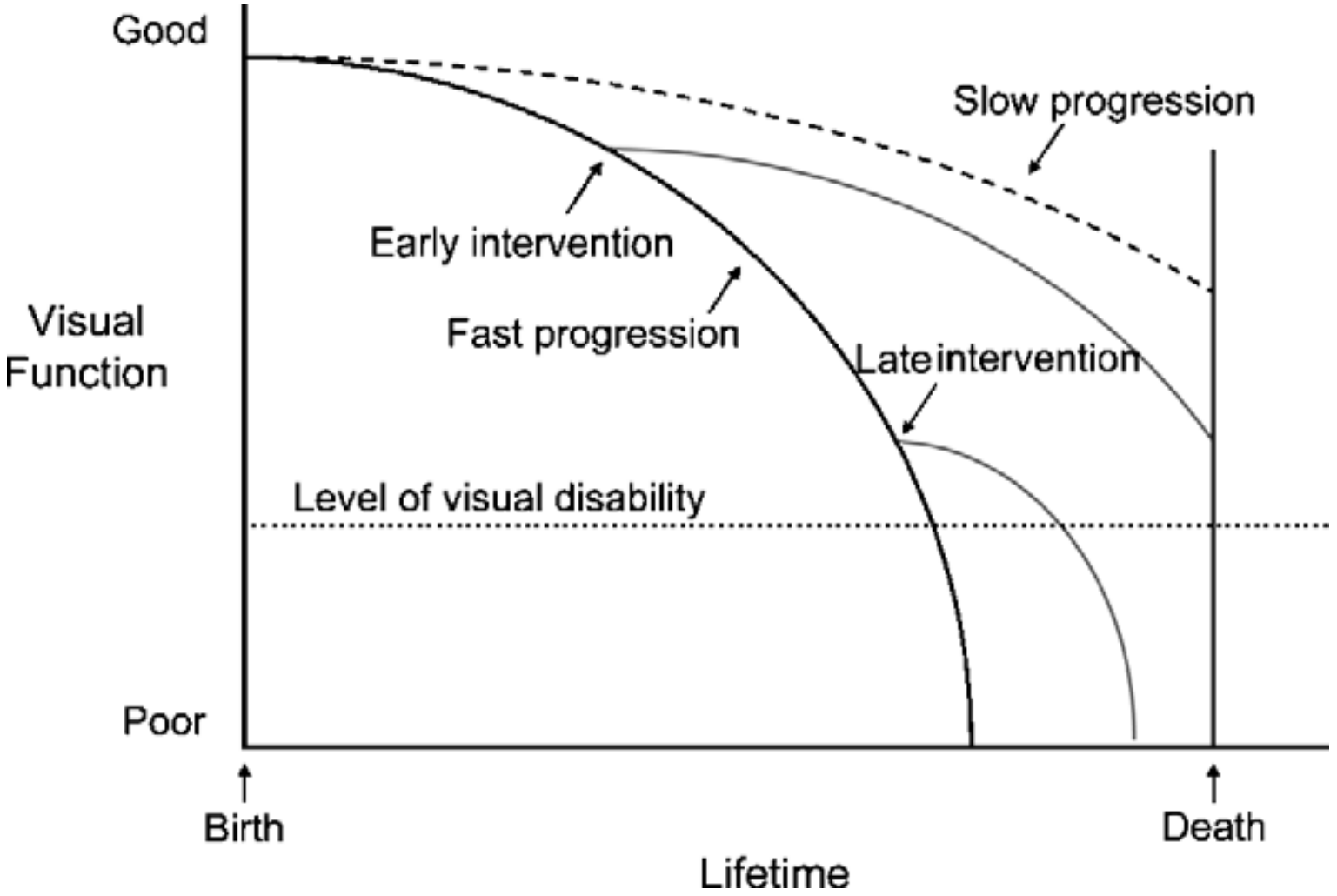


FIG. 4-16. Fixation Threat From Above and Below. The grey scale highlights the dense scotoma above and nasal to fixation. The probability plots show the parafoveal points in the inferior hemifield to be statistically abnormal, and the foveal threshold itself is reduced to 30 dB (SITA-Standard 30-2; left eye).

Treatment


Good
Slow progression
Early intervention
Visual
Function
Fast progression
Late intervention
Level of visual disability
Poor
Birth
Death
Lifetime

Basis of Treatment

- The only proven and established treatment for glaucoma is IOP reduction by medical or surgical means
- Alternative therapies in future:

- Neuroprotection
- Vasoprotection
- Anti-apoptosis
- Gene therapy

Medical Therapy

- General Pharmacologic categories of antiglaucoma medications:

- Beta blockers
- Sympathomimetics
- Parasympathomimetics
- Carbonic anhydrase inhibitors
- Prostaglandin analogues
- Hyperosmotic agents

Non-pressure mechanisms in glaucoma

- Glaucomatous optic neuropathy progresses in 15-20% of “controlled” glaucoma patients.
- We still lack good models to mimic the slow chronic neurodegenerative changes in glaucoma
- Most knowledge is based on experimental studies which only share similarities to some aspects of glaucomatous damage
- These new findings certainly do not diminish the importance of IOP control as the most significant and effective treatment in glaucoma

Neuroprotection

- To prevent or delay retinal ganglion cell (RGC) death
- To rescue and promote regeneration of compromised RGCs
- To protect optic nerve axons from further damage

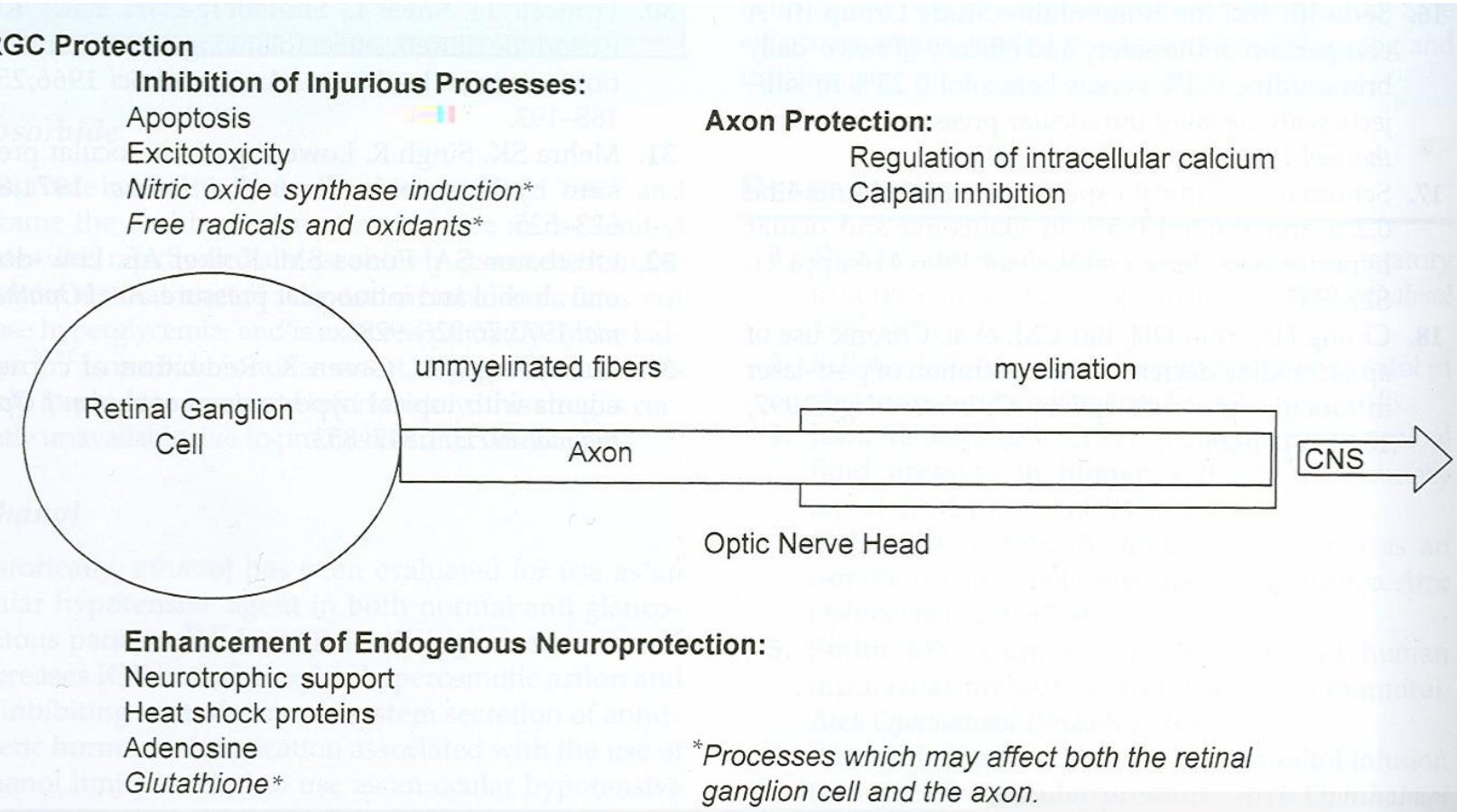
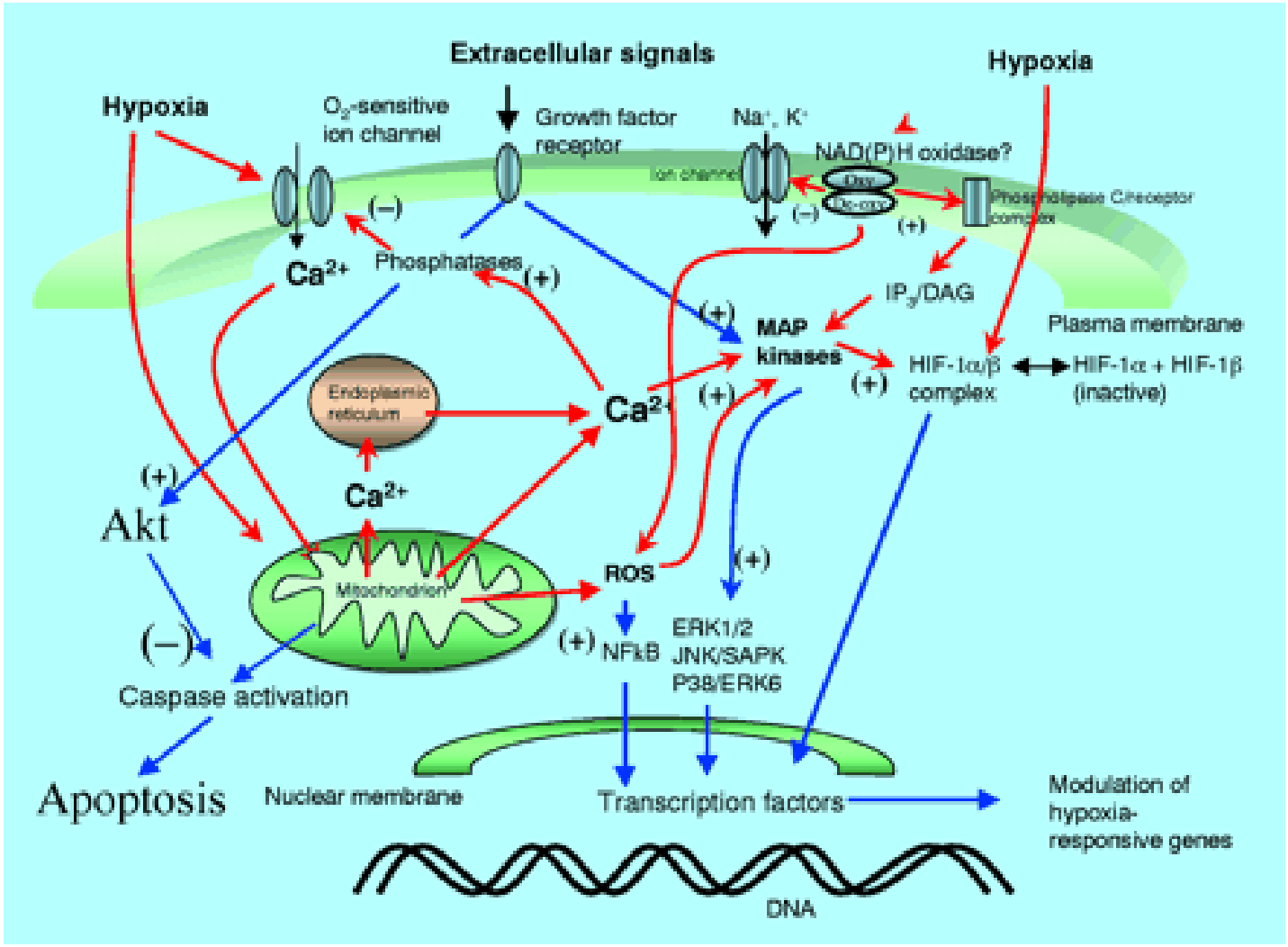


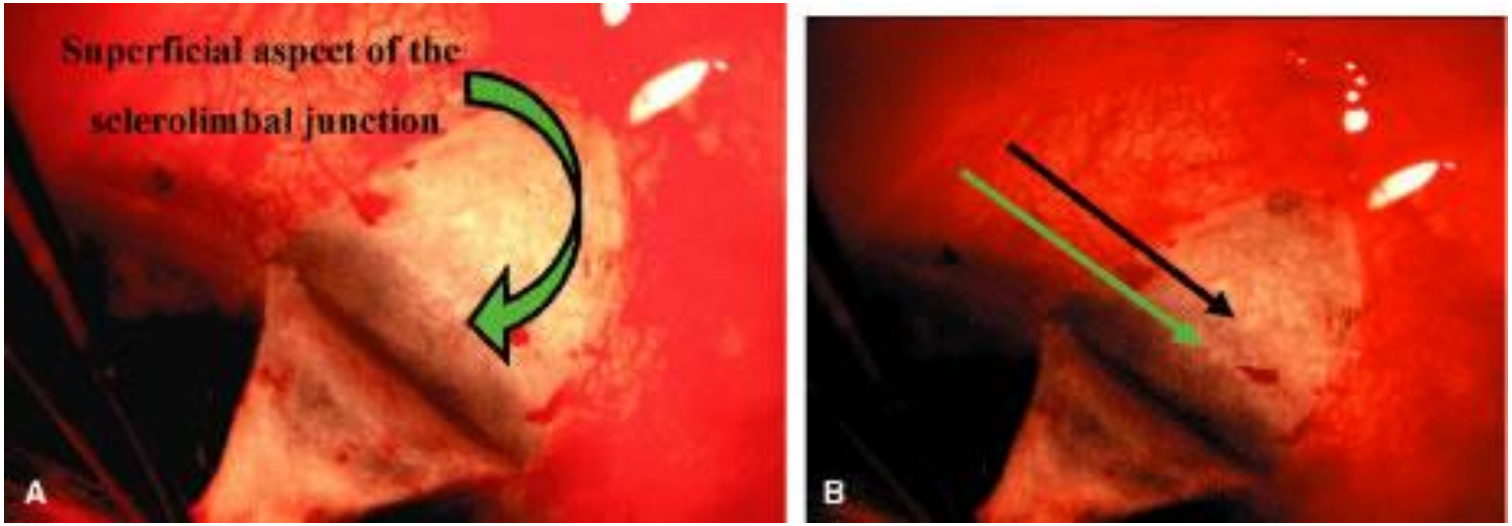
FIGURE 38–1 Potential strategies for neuroprotection in glaucoma include (1) protect the RGC by inhibiting processes that might injure it, (2) enhance endogenous RGC survival mechanisms, and (3) protect the optic nerve axons.

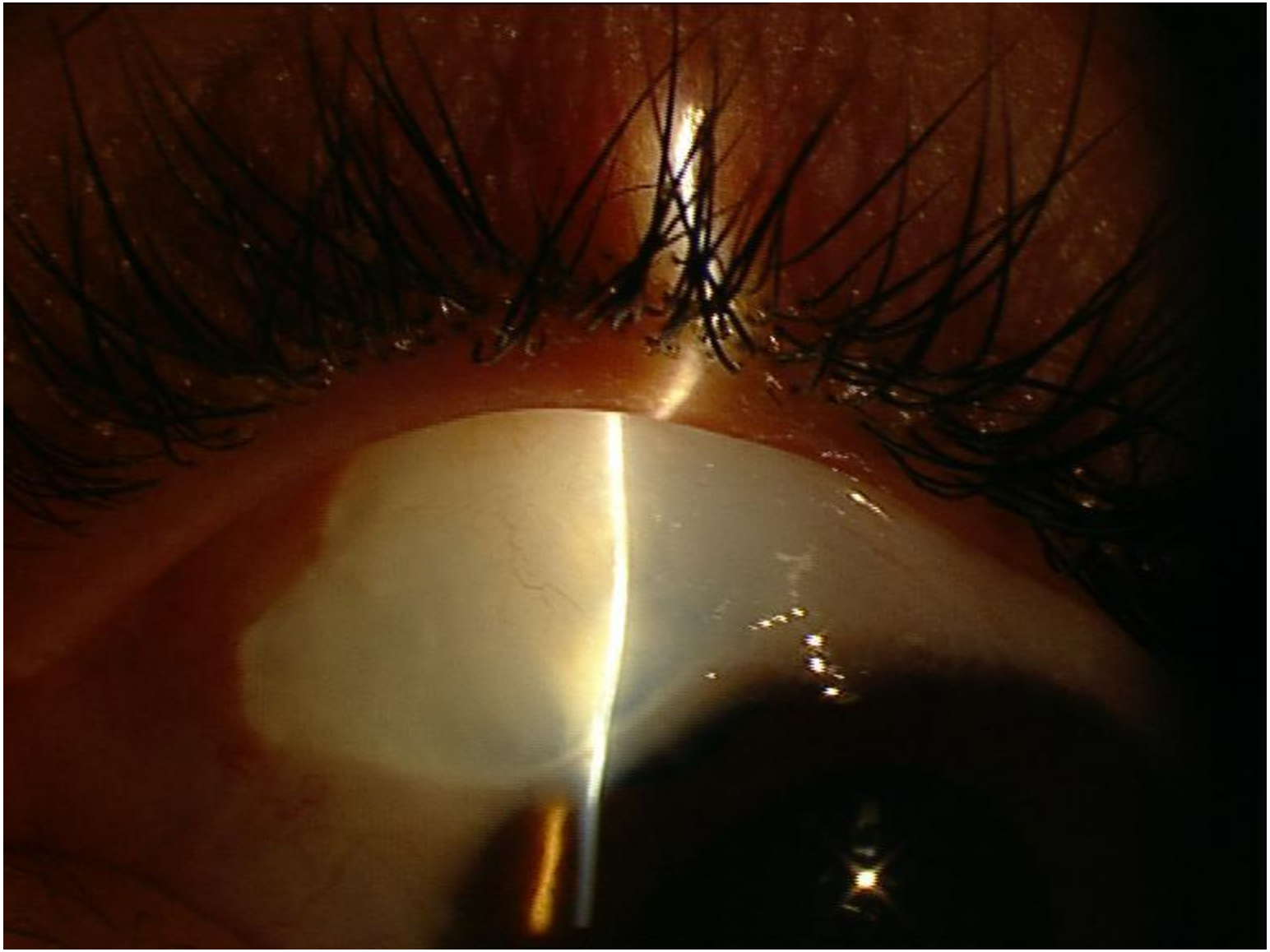

Extracellular signals
Hypoxia
Hypoxia
O_2-sensitive ion channel
Growth factor receptor
Na^+, K^+
NAD(P)H oxidase?
Ion channel
Phosphatidyl C/receptor complex
(–)
(+)
Ca^{2+}
Phosphatases
(+)
IP_3/DAG
(+)
MAP kinases
Plasma membrane
HIF-1α/β complex
HIF-1α + HIF-1β (inactive)
(+)
Endoplasmic reticulum
Ca^{2+}
(+)
Ca^{2+}
Akt
Mitochondrion
ROS
(+)
(+)
ERK1/2
JNK/SAPK
P38/ERK6
(–)
(+) NFkB
Caspase activation
Apoptosis
Nuclear membrane
Transcription factors
Modulation of hypoxia-responsive genes
DNA

Surgical Treatment

- Basically similar concept: to improve aqueous outflow, either through normal physiologic pathways or newly created artificial pathways.

Trabeculectomy

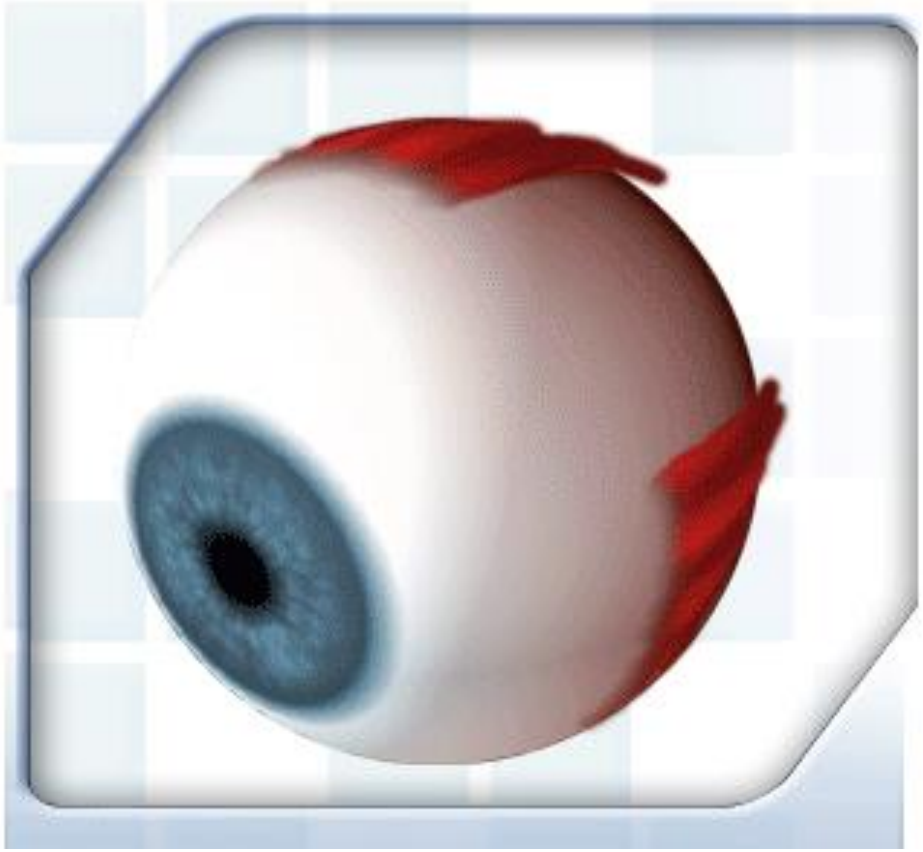




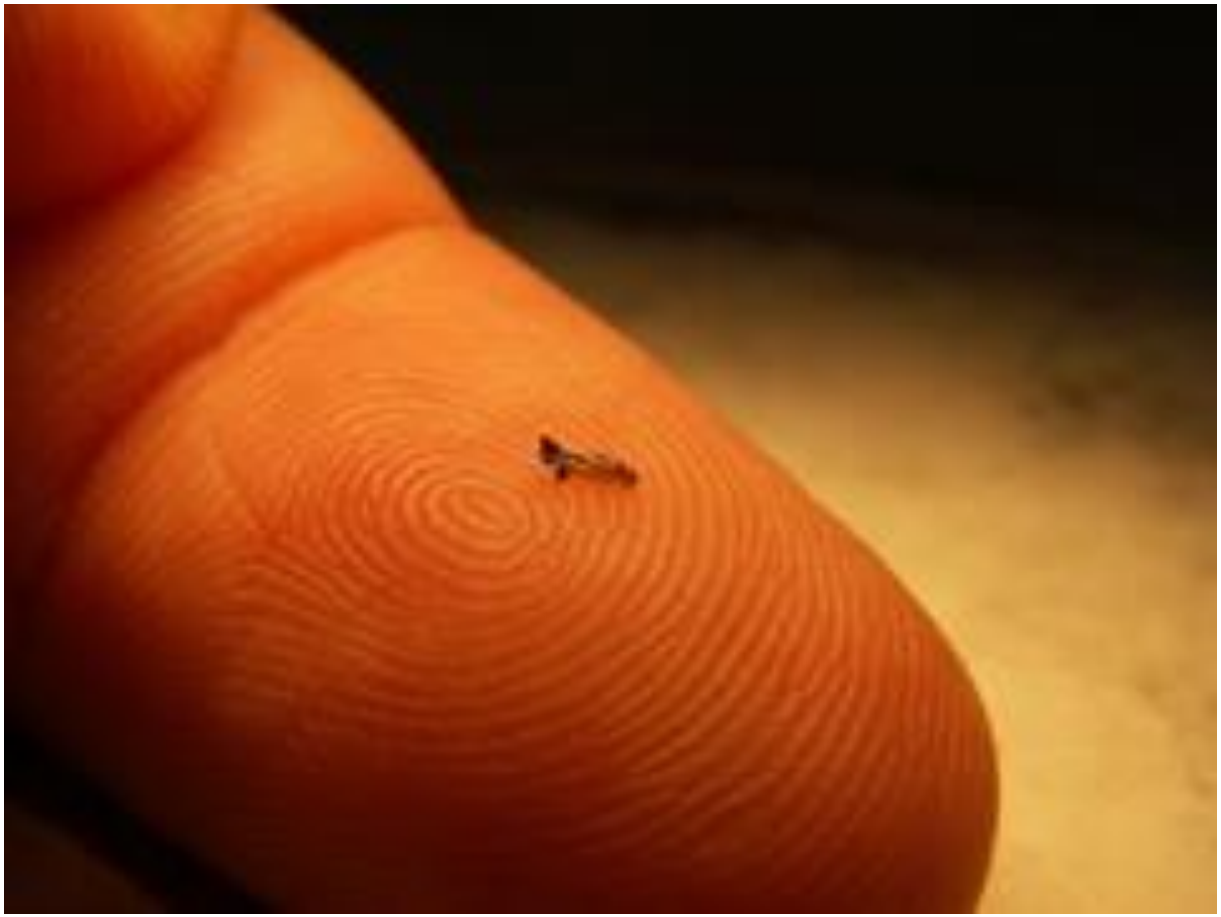
Molteno Implant

Baerveldt implant

Ahmed Glaucoma Valve, S2 and FP7



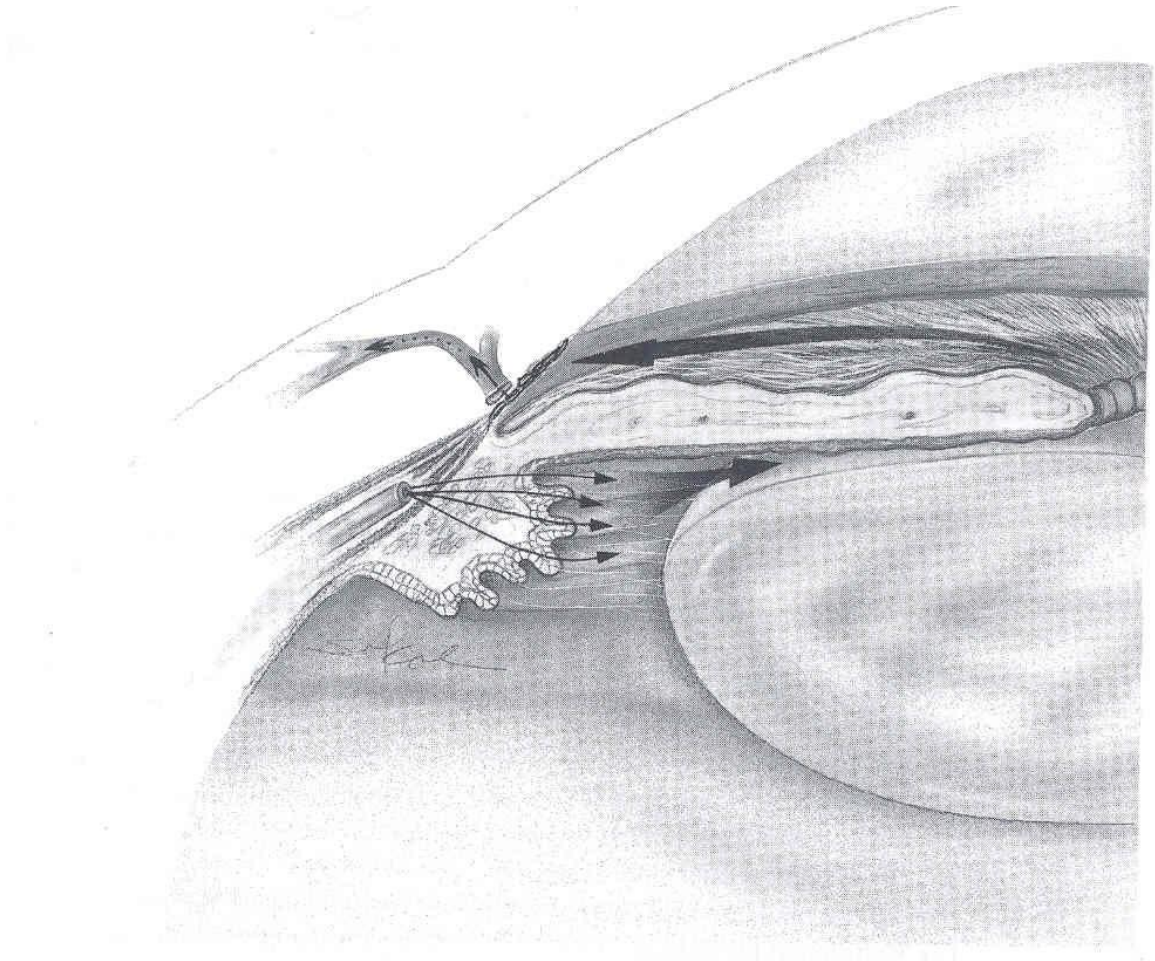
The Express miniature device



The Express miniature device

Acute Glaucoma

Normal aqueous production and outflow



Pupillary Block

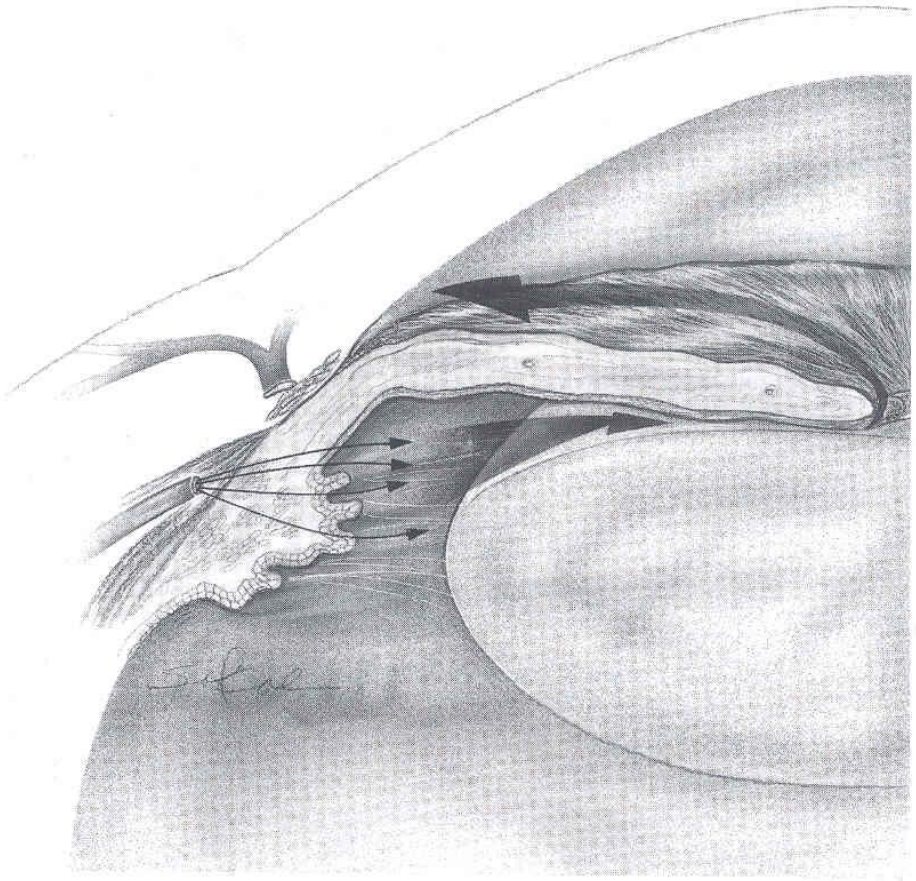
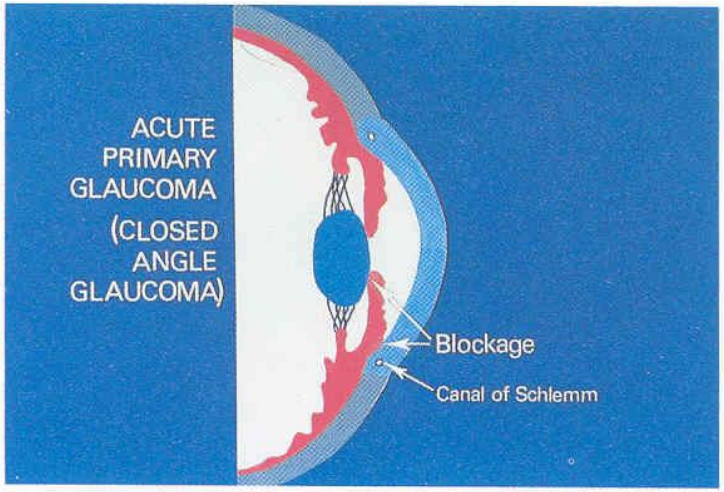


Figure 1-2 In angle-closure glaucoma, the peripheral iris covers the trabecular meshwork, obstructing aqueous humor outflow.

Acute angle closure glaucoma

- Angle-closure glaucoma must be given a high priority among ocular diseases.
- Effects can be devastating.
- Bilateral blindness can result in 2 to 3 days from onset.

Acute angle closure glaucoma



AACG, histopathology

FIGURE 16–1 Pupillary-block glaucoma results from blockage of aqueous humor behind the pupil. The pressure builds up in the posterior chamber and pushes the peripheral iris forward to cause iris bombé and angle closure.

FIGURE 16–2 Light micrograph of the anterior chamber angle from an eye with a history of acute angle closure. The peripheral iris is adherent to the trabecular meshwork.

Acute Angle Closure Glaucoma

- Severe attack
- Occurs rapidly, onset: 30 to 60 minutes
- Very high intraocular pressure
- Corneal epithelial edema
- Pain and congestion of the eye
- Markedly blurred vision
- Halos

Clinical Signs of AACG

- High intraocular pressure
- Mid-dilated, unreactive pupil
- Ciliary injection
- Corneal edema
- Engorged iris vessels
- Cells in aqueous (but no keratic precipitates)
- Closed angle gonioscopically
- Fellow eye-narrow angle judged “closable”
- Iris atrophy
- Posterior synechiae
- Glaukomflecken
- Optic atrophy

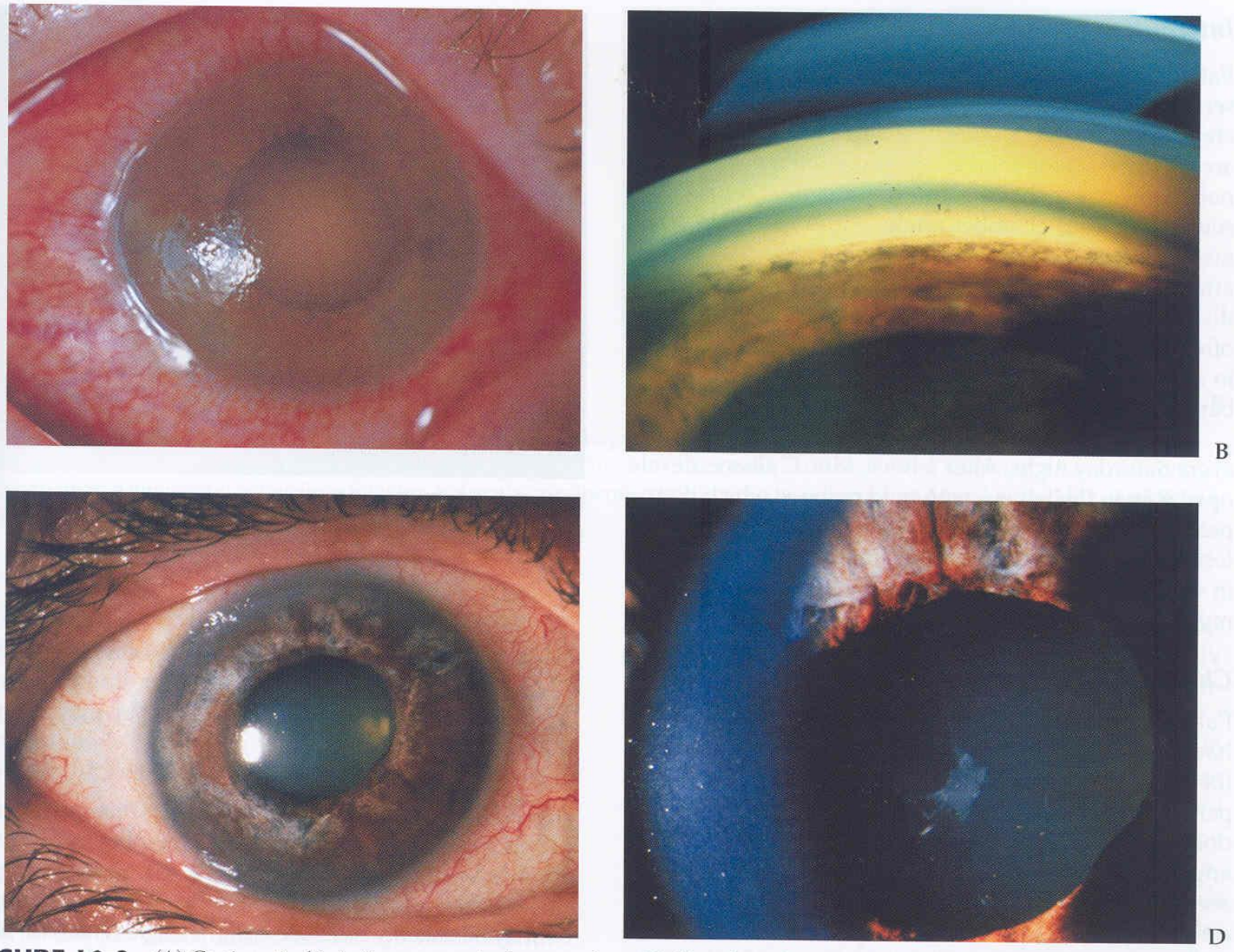


FIGURE 16–3 (A) Conjunctival injection, corneal edema, and a mid-dilated pupil are common signs of an acute angle-closure glaucoma attack. An eye with a history of previous attacks can demonstrate (B) focal regions of tentlike PAS; (C) iris atrophy with a fixed, mid-dilated pupil; and (D) glaukomflecken, coalescing here into a larger, central opcacity. [(A) Courtesy of Kenneth C. Swan, M.D.]

Gross technique for angle evaluation

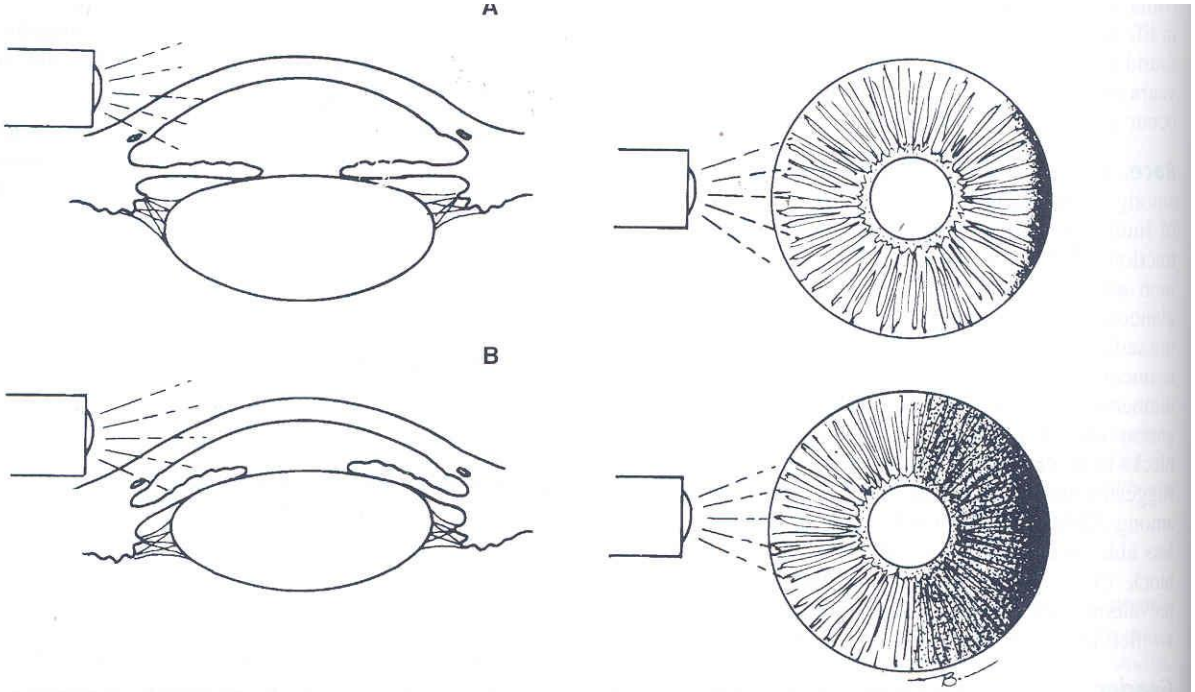


Figure 12.3. Oblique flashlight illumination as a screening measure for estimating the anterior chamber depth. **A:** With a deep chamber, nearly the entire iris is illuminated. **B:** When the iris is bowed forward, only the proximal portion is illuminated, and a shadow is seen in the distal half.

DDx

Any painful congested eye may mimic acute angle-closure glaucoma

Common disorders as keratoconjunctivitis, corneal abrasion or foreign body, and trauma are readily distinguished

APAC Treatment

Emergency Treatment

- IV mannitol, 20% solution 5 cc/kg over 30 minutes.
- Acetazolamide, initial dose of 2 × 250 mg oral.
- A drop of timolol twice at 30-minute intervals as part of initial treatment.
- Apraclonidine an alpha2 agonist ocular hypotensive agent.

Miotics

- 1or 2% pilocarpine every 15 minutes (×4).
- Try to break an early angle-closure attack.
- May be ineffective in attacks longer than 1 or 2 hours.
- May be ineffective if IOP is over 30
- The pupillary sphincter muscle becomes ischemic and unresponsive.

Preparation for definitive treatment

Once IOP is lowered or miosis is achieved, topical therapy with pilocarpine, beta-blockers, carbonic anhydrase inhibitors and apraclonidine should be continued until definitive surgical treatment is performed and reopening of the angle is assured.

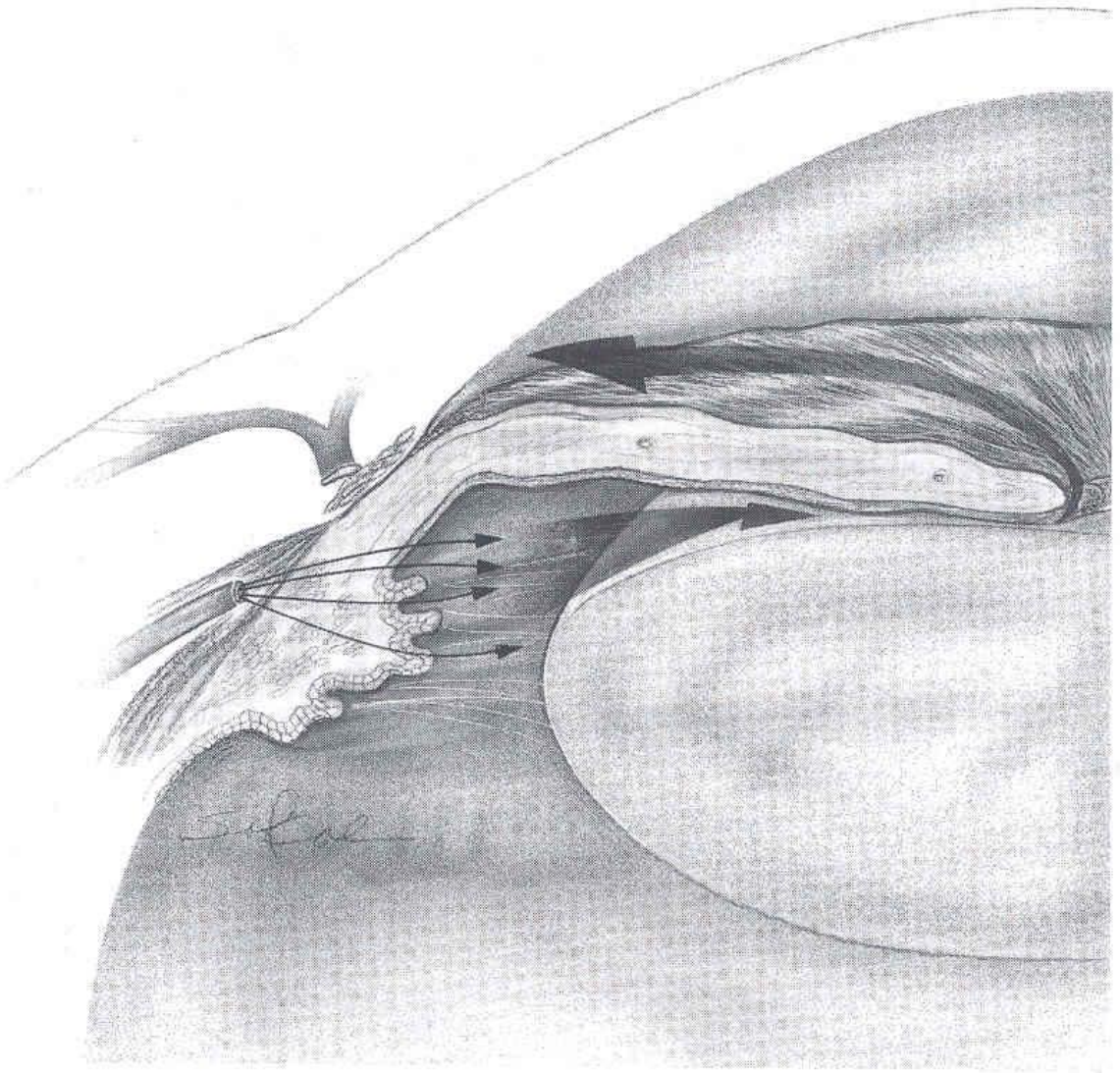


Figure 1-2 In angle-closure glaucoma, the peripheral iris covers the trabecular meshwork, obstructing aqueous humor outflow.

Laser Peripheral Iridotomy

- All eyes that have suffered a primary acute angle-closure attack should have a peripheral iridotomy.
- To reestablish aqueous flow between the posterior and anterior chambers.
- Allows an equilibrium between anterior and posterior chamber pressures, and if done before peripheral anterior synechiae develop, may be curative.
- The fellow eye must be also treated prophylactically.

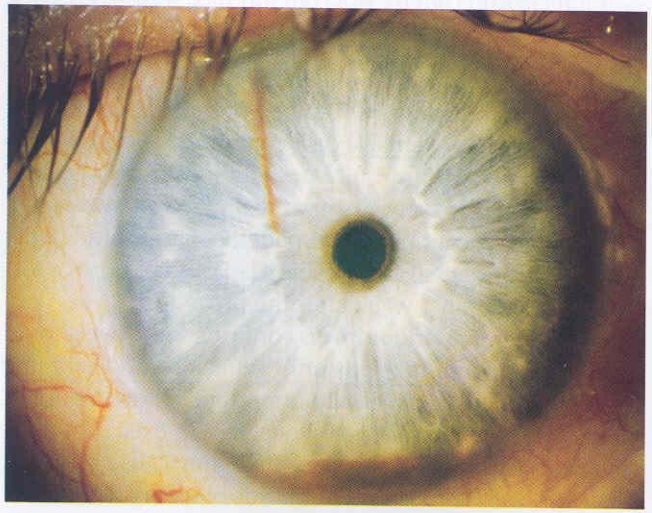


FIGURE 41–3 Iris bleeding and hyphema following Nd:YAG laser iridotomy.

FIGURE 41–4 Focal lens opacity beneath argon laser iridotomy.

After PI

- Lifelong monitoring and follow up for chronic glaucoma.