Kanski's Synopsis of Clinical Ophthalmology

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John F. Salmon

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Content Strategist: Kayla Wolfe
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PREFACE

This fourth edition of *Kanski's Synopsis of Clinical Ophthalmology* is intended to be used principally as a companion to the ninth edition of *Kanski's Clinical Ophthalmology*. It provides a summary of the larger book and is filled with beautiful illustrations. *Synopsis* can be used as a portable, rapidly searchable reference source that is suitable for use in a busy clinic. Alternatively, the contents and images are available in an online electronic format. The book is ideal as a basis for revision and is presented as a series of easily absorbed topic summaries. General practitioners, medical students, optometrists and specialist nurses requiring a shorter, but comprehensive review of ophthalmology, may find *Synopsis* a more appropriate text than the lengthier consideration of *Kanski's Clinical Ophthalmology*.

Jack Kanski's unique approach of presenting core knowledge in a systematic and succinct form has been maintained. Brad Bowling had a significant influence on the previous edition and his accuracy and meticulous attention to detail has been extremely helpful. I have started this edition with a new chapter on examination techniques and a short overview of the most important ophthalmic special investigations. Each chapter has been updated and the latest evidence-based diagnostic and therapeutic advances have been included.

All the illustrations have been reformatted and many new images have been added. I am grateful to colleagues past and present, whose images are included in this edition. I have once again had the good fortune of working with Jon Brett, a world-class photographer and artist, whose expertise has been invaluable.

I am indebted to Elsevier for entrusting me with this work and, in particular, I wish to thank Julie Taylor, Kayla Wolfe and Deborah Poulson for their editorial contribution. I hope you, the reader, obtain as much enjoyment from the book as I have obtained from preparing it!

J.F. Salmon
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ABBREVIATIONS

AAION: arteritic anterior ischaemic optic neuropathy
AAU: acute anterior uveitis
AC/A ratio: accommodative convergence/accommodation ratio
AD: autosomal dominant
AHP: abnormal head posture
AI: accommodative insufficiency
AIDS: acquired immune deficiency syndrome
AION: anterior ischaemic optic neuropathy
AKC: atopic keratoconjunctivitis
ALT: argon laser trabeculoplasty
AMD: age-related macular degeneration
ANA: antinuclear antibody
APD: afferent pupillary defect
APMPPE: acute posterior multifocal placoid pigment epitheliopathy
AR: autosomal recessive
AREDS: Age-Related Eye Disease Study
ARN: acute retinal necrosis
BCC: basal cell carcinoma
BP: blood pressure
BRAO: branch retinal artery occlusion
BRVO: branch retinal vein occlusion
BSV: binocular single vision
BUT: breakup time
CAI: carbonic anhydrase inhibitor
CAU: chronic anterior uveitis
CCT: central corneal thickness
CDCR: canaliculodacryocystorhinostomy
CHED: congenital hereditary endothelial dystrophy
CHRPE: congenital hypertrophy of the retinal pigment epithelium
CI: convergence insufficiency
CMO: cystoid macular oedema
CNS: central nervous system
CNV: choroidal neovascularization
CPEO: chronic progressive external ophthalmoplegia
CRAO: central retinal artery occlusion
CRP: C-reactive protein
CRVO: central retinal vein occlusion
CSMO: clinically significant macular oedema
CSS: central suppression scotoma
CT: computed tomography
DALK: deep anterior lamellar keratoplasty
CDR: dacryocystorhinostomy
DR: diabetic retinopathy
DSEK: Descemet stripping endothelial keratoplasty
DVD: dissociated vertical deviation
ECG: electrocardiogram

EDTA: ethylenediaminetetraacetic acid
EKC: epidemic keratoconjunctivitis
EOG: electro-oculogram
ERG: electroretinogram
ESR: erythrocyte sedimentation rate
FA: fluorescein angiography
FAP: familial adenomatous polyposis
FAZ: foveal avascular zone
FBC: full blood count
FFM: fundus flavimaculatus
GCA: giant cell arteritis
GPC: giant papillary conjunctivitis
HAART: highly active antiretroviral therapy
HIV: human immunodeficiency virus
HRT: Heidelberg retinal tomograph
HSV-1: herpes simplex virus type 1
HSV-2: herpes simplex virus type 2
HZO: herpes zoster ophthalmicus
ICGA: indocyanine green angiography
Ig: immunoglobulin
IK: interstitial keratitis
ILM: internal limiting membrane
INO: internuclear ophthalmoplegia
IOFB: intraocular foreign body
IOID: idiopathic orbital inflammatory disease
IOL: intraocular lens
IOP: intraocular pressure
IRMA: intraretinal microvascular abnormality
ITC: iridotrabecular contact
IU: intermediate uveitis
JIA: juvenile idiopathic arthritis
KCS: keratoconjunctivitis sicca
KP: keratic precipitate
LA: local anaesthesia
LASEK: laser epithelial keratomileusis
LASIK: laser *in situ* keratomileusis
LN: latent nystagmus
MLF: medial longitudinal fasciculus
MR: magnetic resonance imaging
MS: multiple sclerosis
MU: mega units
NF1: neurofibromatosis 1
NF2: neurofibromatosis 2
NRR: neuroretinal rim
NSAID: nonsteroidal anti-inflammatory drug
NSR: neurosensory retina
NVD: new vessels at the disc
NVE: new vessels elsewhere
OCT: optical coherence tomography
OHT: ocular hypertension
OKN: optokinetic nystagmus

PAC: primary angle-closure
PACG: primary angle-closure glaucoma
PACS: primary angle-closure suspect
PAM: primary acquired melanosis
PAS: peripheral anterior synechiae
PCF: pharyngoconjunctival fever
PCO: posterior capsular opacification
PCR: polymerase chain reaction
PCV: polypoidal choroidal vasculopathy
PDR: proliferative diabetic retinopathy
PDS: pigment dispersion syndrome
PDT: photodynamic therapy
PED: pigment epithelial detachment
PIOL: primary intraocular lymphoma
PION: posterior ischaemic optic neuropathy
PKP: penetrating keratoplasty
POAG: primary open-angle glaucoma
POHS: presumed ocular histoplasmosis syndrome
PPCD: posterior polymorphous corneal dystrophy
PPRF: paramedian pontine reticular formation
PPV: pars plana vitrectomy
PRK: photorefractive keratectomy
PRP: panretinal photocoagulation
PVD: posterior vitreous detachment
PVR: proliferative vitreoretinopathy
PXF: pseudoexfoliation
RAPD: relative afferent pupillary defect
RD: retinal detachment
ROP: retinopathy of prematurity
RP: retinitis pigmentosa
RPE: retinal pigment epithelium
RRD: rhegmatogenous retinal detachment
SCC: squamous cell carcinoma
SF: short-term fluctuation
SJS: Stevens–Johnson syndrome
SLK: superior limbic keratoconjunctivitis
SLT: selective laser trabeculoplasty
SRF: subretinal fluid
TAL: total axial length
TB: tuberculosis
TEN: toxic epidermal necrolysis
TGF: transforming growth factor
TIA: transient ischaemic attack
TTT: transpupillary thermotherapy
UBM: ultrasonic biomicroscopy
US: ultrasonography
VA: visual acuity
VEGF: vascular endothelial growth factor
VHL: von Hippel–Lindau syndrome
VKC: vernal keratoconjunctivitis
VKH: Vogt–Koyanagi–Harada syndrome
VZV: varicella zoster virus
X-L: X-linked

To Susie

CHAPTER 1

Examination of the Eye

CHAPTER OUTLINE

Ophthalmic history

Before examining the eye, a thorough ophthalmic history should be taken. The history can be divided into the following basic categories of questioning:

- ***Main complaint:*** (a) rapidity of onset, (b) circumstances surrounding the onset, (c) severity, (d) duration of symptoms, (e) frequency of symptoms.
- ***Past ocular history:*** e.g. previous surgery, inflammation, trauma.
- ***Past medical history:*** e.g. diabetes and hypertension.
- ***Systemic medication:*** e.g. corticosteroids, tamsulosin.
- ***Allergies:*** e.g. antibiotics, topical glaucoma medications.
- ***Family history:*** e.g. glaucoma, macular degeneration, inherited retinal disease.
- ***Occupation and hobbies***

Common ocular symptoms

- ***Abnormality in vision:*** (a) visual loss and blurring (central or peripheral), (b) change in colour vision, (c) visual aberration (scotoma, distortion, flashing lights, floaters), (d) diplopia (monocular, binocular, neurological symptoms).
- ***Pain and discomfort:*** (a) ocular, (b) periocular (lids, sinus, temporal artery), (c) retrobulbar (orbital inflammation), (d) nonspecific (eyestrain, dryness, scratching).
- ***Change in appearance:*** (a) redness, (b) swelling of the eyelids, (c) displacement of the eyeball, (d) changes to the lids and periocular tissues, (e) discharge and watering.

Visual acuity

Visual acuity is directly related to the minimum angle of separation between two objects that allows them to be seen distinctly. Visual acuity should always be determined first, regardless of whether the patient complains of visual disturbance or not. Each eye is tested separately, with and without spectacles. A pinhole disc is a simple method of focusing light and temporarily removes the effect of refractive error.

SNELLEN VISUAL ACUITY

A Snellen chart is used, with the subject reading the chart from a standard distance (Fig. 1.1A). Normal visual acuity equates to 6/6 (20/20 in non-metric notation). If the patient is unable to see the chart using either spectacles or a pinhole disc the vision can be determined by counting fingers (CF), seeing hand movements (HM), or by assessing the ability to see light (PL).

LOGMAR VISUAL ACUITY

LogMAR is an acronym for the base-10 logarithm of the minimum angle of resolution. A Bailey–Lovie chart is used, which has an equal number of letters on each line and the lines are balanced for consistency of readability (Fig. 1.1B). LogMAR 0.00 is equivalent to 6/6 and logMAR 1.00 is equivalent to 6/60. Because logMAR acuity addresses many of the deficiencies of the Snellen chart it is commonly used when research is undertaken.

Contrast sensitivity

Contrast sensitivity is a measure of the ability of the visual system to distinguish an object against its background. The Pelli–Robson contrast sensitivity letter chart is viewed at 1 m and consists of rows of letters of equal size, but with decreasing contrast of 0.15 log units for groups of three letters (Fig. 1.2A).

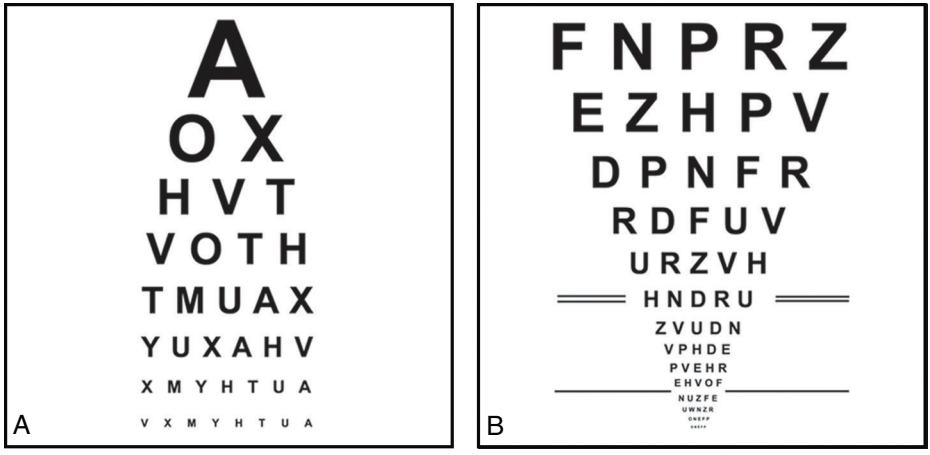

Fig. 1.1 Visual acuity chart: (A) Snellen, (B) Bailey–Lovie logMAR chart. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

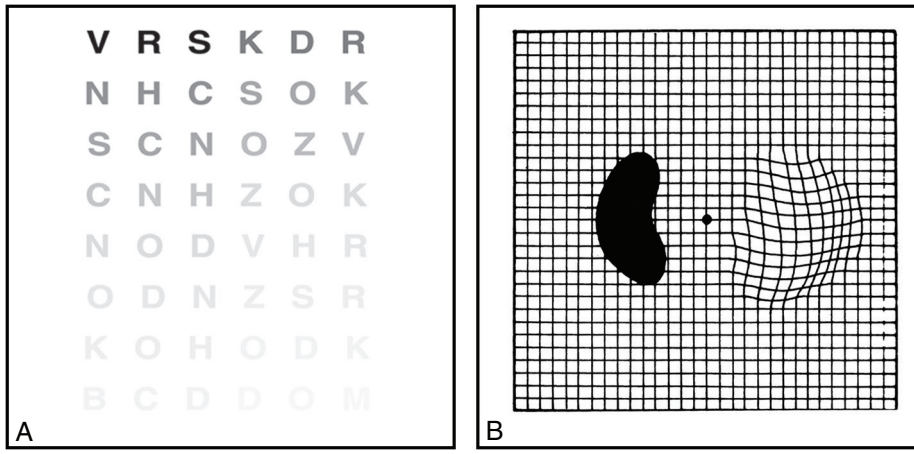

Fig. 1.2 (A) Pelli–Robson contrast sensitivity letter chart, (B) Amsler grid showing wavy lines indicating metamorphopsia and a dense scotoma. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

Amsler grid

The Amsler grid evaluates the central 20° of the visual field centred on fixation (Fig. 1.2B). It is an easy method of monitoring central visual field and is commonly abnormal in patients with macular disease.

Colour vision

ISHIHARA

This test is simple to undertake, is widely available, and is frequently used to screen for red-green colour anomalies. Inherited colour vision deficiency affects 8% of men and 0.5% of women. The test can also be used to assess optic nerve disease (Fig. 1.3A).

FARNSWORTH–MUNSELL 100-HUE TEST

This test is sensitive but takes longer than the Ishihara to perform. It is used for congenital and acquired colour defects (Fig. 1.3B).

Visual field

Visual field results should always be used in conjunction with the clinical findings. The test is particularly important in glaucoma and neurological disease.

- ***The visual field:*** can be represented as a three-dimensional structure akin to a hill of increasing sensitivity. The outer aspect extends approximately 50° superiorly, 60° nasally, 70° inferiorly and 90° temporally.
- ***Static perimetry:*** is a method of assessing fields in which the stimulus remains fixed, with intensity increasing until it is seen by the subject or decreasing until it is no longer detected. Standard automated perimetry (SAP) uses this method (Fig. 1.4).

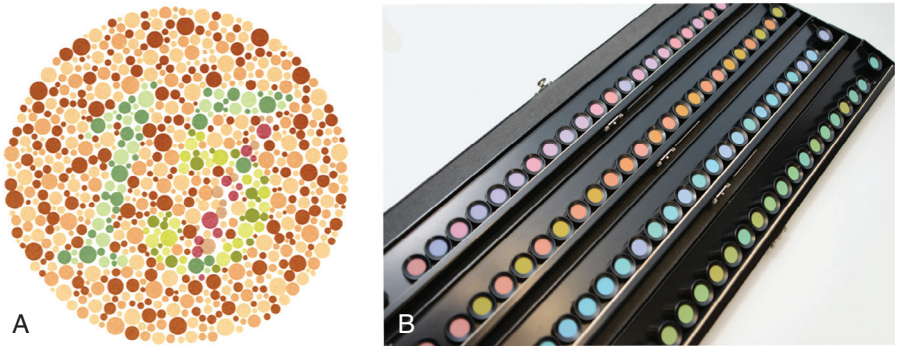

Fig. 1.3 Colour vision tests: (A) Ishihara, (B) Farnsworth–Munsell 100-hue test. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

- ***Kinetic perimetry:*** is undertaken by moving a stimulus of constant intensity from a non-seeing area to a seeing area at a constant speed until it is perceived.

ANALYSIS OF VISUAL FIELDS

- ***Reliability indices (Fig. 1.4A):*** with SITA strategies false positive or false negative responses over 15% should be considered significant. If the test is found to be unreliable, further evaluation of the printout is pointless.
- ***A numerical display (Fig. 1.4B):*** gives the threshold in dB at each point tested in the field. A grey scale ***(Fig. 1.4C)*** represents the numerical display in graphical form; decreasing sensitivity is represented in darker tones.
- ***Total deviation (Fig. 1.4D):*** shows the difference between a test-derived threshold at a given point and the normal sensitivity at that point for the general population.
- ***Pattern deviation (Fig. 1.4E):*** is the total deviation adjusted for a generalized decrease in sensitivity in the whole field (for example, the presence of cataract).
- ***Summary values (Fig. 1.4F):*** represent distilled statistical information: (a) visual field index (VFI) is a measure of overall visual field function expressed as a percentage, (b) mean deviation (MD) provides an indication of the overall sensitivity of the field, (c) pattern standard deviation (PSD) is a measure of focal loss (an increased PSD is an indicator of glaucoma), (d) the glaucoma hemifield test (GHT) compares corresponding areas in the superior and inferior hemifields.

Microperimetry

Microperimetry is a subjective visual field test that measures retinal sensitivity and fixation behaviour in patients with macular disease and glaucoma involving the central 9° of visual field (Fig. 1.5).

EXTERNAL EXAMINATION

External examination of the eye, periorbital tissues and orbit should be undertaken before magnification is used. Fluorescein dye allows pathology on the surface of the cornea to be visualized (see Fig. 22.8B) and is used when Goldmann applanation tonometry is undertaken.

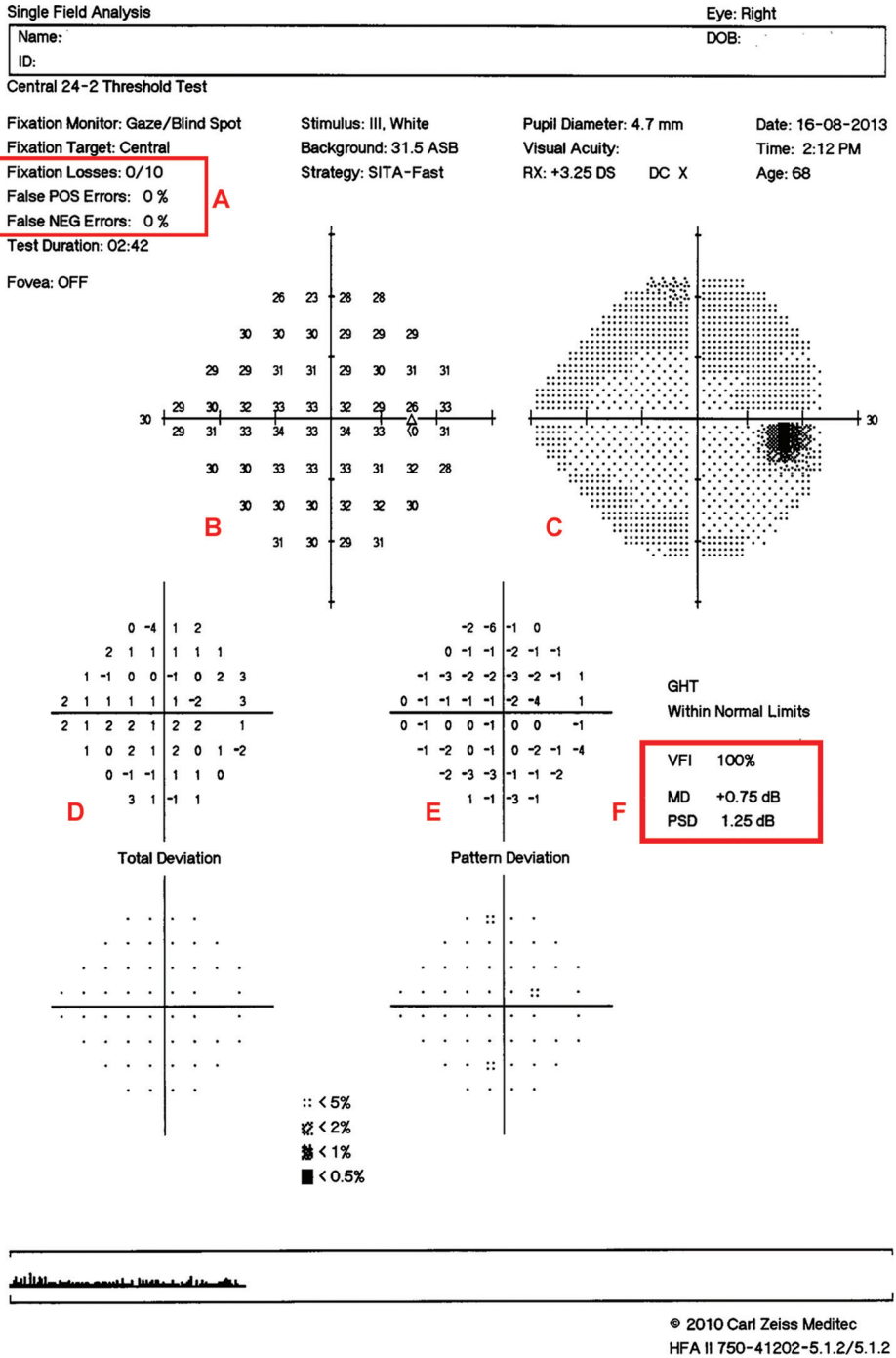

Fig. 1.4 Humphrey SITA-Fast printout (A–F; see text). (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

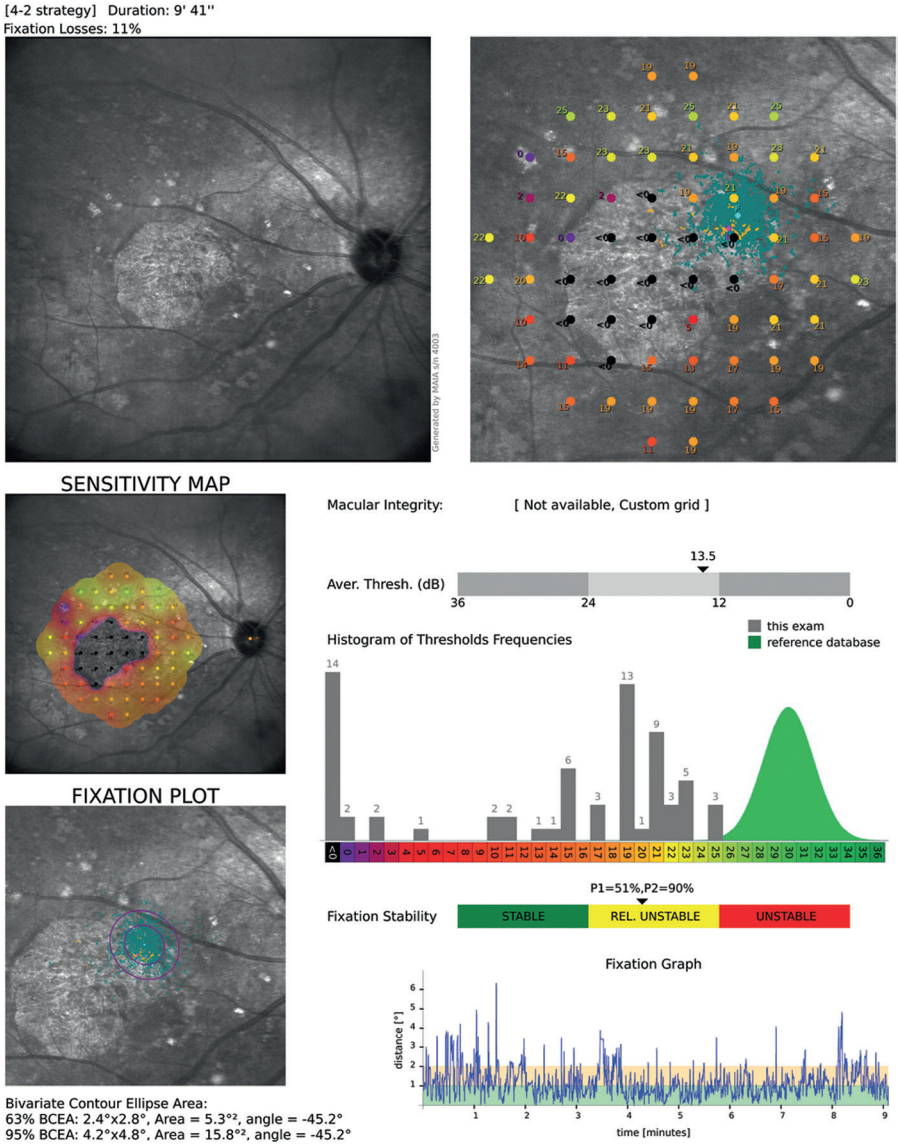

Fig. 1.5 Microperimetry in a patient with geographic atrophy of the macula. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

Slit lamp biomicroscopy of the anterior segment

The purpose of slit lamp examination is to determine the position, depth and size of any abnormality of the cornea and anterior segment. It provides good lighting and a stereoscopic view: (a) direct illumination with a diffuse light is used to detect gross abnormalities, (b) scleral scatter involves decentring the slit beam laterally so that the light is incident on the limbus with the microscope focused centrally; light is transmitted within the cornea by total internal reflectivity which allows subtle

Fig. 1.6 (A) Direct ophthalmoscopy, (B) indirect slit lamp biomicroscopy. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

stromal haze to be detected, (c) retroillumination uses reflected light from the iris to illuminate the cornea, permitting the detection of fine epithelial and endothelial changes, (d) specular reflection shows abnormalities of the endothelium such as reduced cell density and guttata.

DIRECT OPHTHALMOSCOPY

- ***Ophthalmoscope*** *(Fig. 1.6A)*. Direct examination of the structures of the fundus using an ophthalmoscope can reveal disease of the eye itself or may reveal an abnormality indicative of disease elsewhere in the body (for example: diabetes, systemic hypertension, raised intracranial pressure). The image obtained is magnified (×15), but the disadvantages are that there is no stereopsis and that the field of view is small.

INDIRECT OPHTHALMOSCOPY

This can be undertaken using a slit lamp or a head-mounted ophthalmoscope. A condensing lens is held at the focal point of the eye and provides an inverted and laterally reversed image.

- ***Slit lamp biomicroscopy*** *(Fig. 1.6B)*: (a) non-contact lenses: 60D (high magnification); when estimating optic disc size use a correction factor of ×1.0; for the 90D (wide-field) lens use a correction factor of 1.3 and for the 78D lens use ×1.1, (b) contact lenses: the Goldman three mirror lens has a central lens and three mirrors set at different angles. A viscous coupling solution is required.
- ***Head-mounted binocular indirect ophthalmoscopy*** *(Fig. 1.7A and B)*: allows retinal visualization through a greater degree of media opacification than slit lamp ophthalmoscopy. A 20D lens magnifies ×3 and a 28D lens (shorter working distance; used in a smaller pupil) magnifies ×2.27.

Tonometry

Tonometry is the method of measuring the intraocular pressure (IOP) using calibrated instruments. The normal range for individuals over 40 years of age is 11–21 mm Hg, but 4–7% of normal individuals have an IOP of more than 21 mm Hg (see Chapter 11).

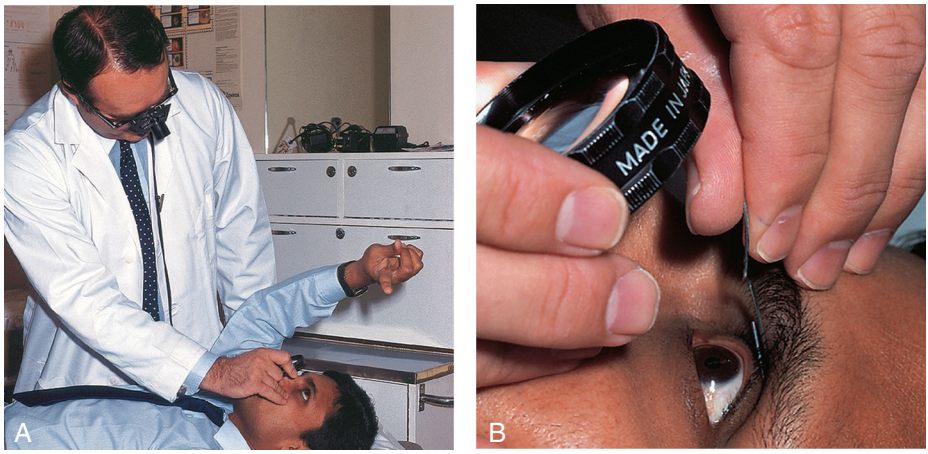

Fig 1.7 (A) Head-mounted binocular indirect ophthalmoscopy, (B) showing technique of indentation. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

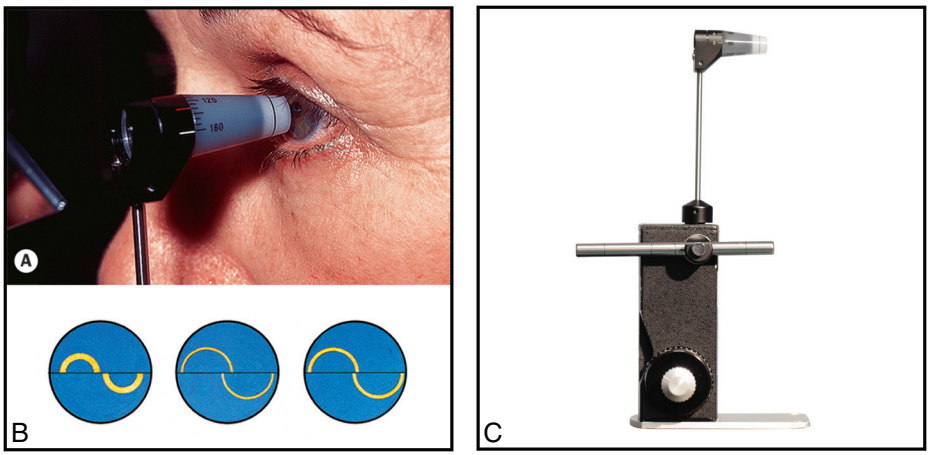

Fig. 1.8 Goldmann applanation tonometry: (A) contact between the tonometer prism and the cornea, (B) correct end-point using mires of appropriate thickness, (C) tonometer calibration bar in position. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

- ***Goldmann tonometry***: This is an accurate variable-force tonometer consisting of a double prism which applanates the central cornea (Fig. 1.8A). Fluorescein stain is used to create semi-circular mires (Fig. 1.8B). Accuracy is lost with constant use and the tonometer should be checked on a regular basis for calibration error (Fig. 1.8C). Calculations are based on a central corneal thickness of 520 microns (if the cornea is thinner, an underestimation of IOP is likely to result and if thicker, an overestimation). Corneal oedema may result in an artificial reduction in the IOP value. Other sources of error include inappropriate fluorescein pattern and pressure on the globe.
- ***Perkins applanation tonometry***: as above, but hand-held with portable light source.

- ***Other methods:*** (a) pneumotonometry ('air-puff') is based on the principle of applanation using a jet of air rather than a prism, (b) electronic applanation tonometry (tonopen); the probe tip contains a transducer that measures applied force, (c) dynamic contour tonometry; a solid-state sensor and a corneal contour-matching surface is used. It has the advantage of measuring the IOP independent of corneal mechanical factors.
- ***Ocular response analyser and corneal hysteresis***

This instrument uses air-puff technology to record two applanation measurements: one while the cornea is moving inward and one when the cornea returns to its normal position. The average of these two measurements provides a Goldmann-related IOP measurement. The difference between the IOP measurements is called corneal hysteresis. The value obtained is accurate in individuals who have undergone laser refractive surgery. Patients with a low hysteresis value are at greater risk of glaucoma progression and it may serve as a biomarker to aid glaucoma case detection.

Central corneal thickness

This can be measured using pachymetry or by imaging with an Orbscan. The average value is 540 microns (range: 510–570 microns). It is an important measurement when determining the risk of conversion to glaucoma in individuals with raised intraocular pressure.

Gonioscopy

Gonioscopy is a method of evaluating the anterior chamber angle. A contact lens is used, e.g. Goldmann one-mirror lens (Fig. 1.9A), Zeiss four-mirror lens (Fig. 1.9B). The examination should take place in a darkened room. Abnormalities that can be detected using gonioscopy include: (a) angle closure, (b) neovascularization, (c) hyper-pigmentation, (d) angle recession (see Chapter 11).

- ***Indirect gonioscopy*** provides an inverted view of the portion of the angle opposite the mirror. Non-indentation requires a coupling fluid. Indentation gonioscopy does not require a coupling fluid and allows a view of the angle when there is apposition between the peripheral iris and cornea. It allows the degree of synechiae to be determined.

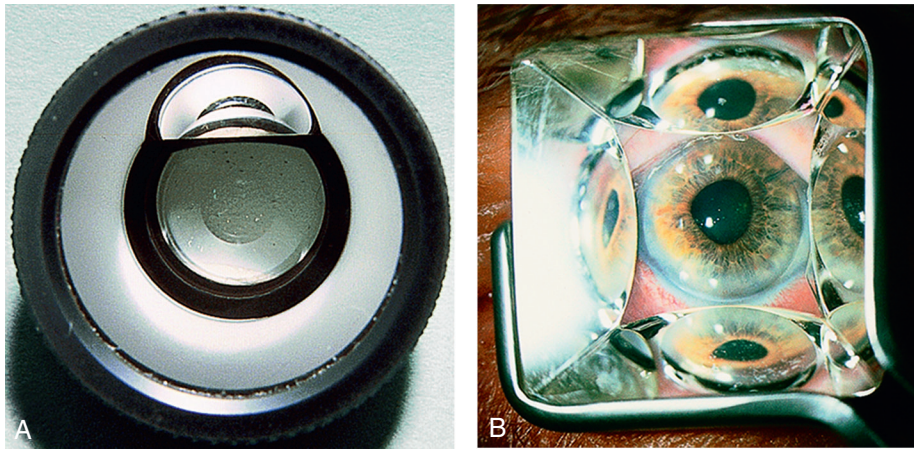

Fig 1.9 (A) Goldmann one-mirror goniolens, (B) Zeiss lens in position. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

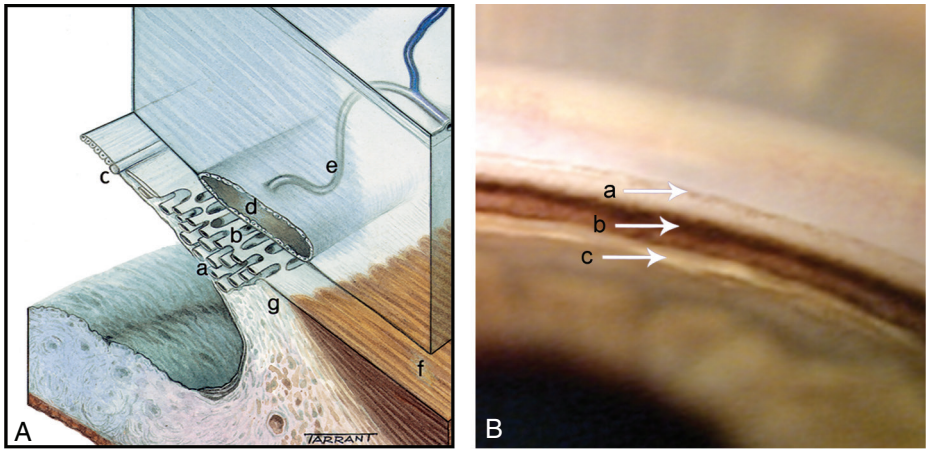

Fig. 1.10 (A) Anatomy of outflow channels: (a) uveal meshwork, (b) corneoscleral meshwork, (c) Schwalbe line, (d) Schlemm canal, (e) collector channels, (f) longitudinal muscle of the ciliary body, (g) scleral spur. (B) Normal structures on gonioscopy: (a) Schwalbe line, (b) pigmented meshwork, (c) ciliary body band. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

- ***Direct gonioscopy***: light rays from the angle are viewed directly. The examination is undertaken with the patient in a supine position, usually under general anaesthesia in the evaluation and surgical treatment of congenital glaucoma or under local anaesthesia when undertaking MIGS.
- ***Identification of angle structures:*** (a) Schwalbe line is the most anterior structure and demarcates the peripheral termination of Descemet membrane and the anterior limit of the trabeculum, (b) the corneal wedge is useful in identifying an inconspicuous Schwalbe line (see Fig. 11.5), (c) the trabeculum extends from the Schwalbe line to the scleral spur; the non-functioning part has a whitish colour, while the functioning part is pigmented in adults, (d) the Schlemm canal is a slightly darker line deep to the posterior trabeculum, (e) the ciliary body stands out just behind the scleral spur as a pink, dull brown or slate-grey band (Fig. 1.10A and B).

Optical coherence tomography (OCT)

OCT is a non-invasive, non-contact imaging system that provides high resolution images of the anterior and posterior segments (Fig. 1.11). The diagnosis and monitoring of macular pathology has been revolutionized by this technology (see Chapters 13–15). OCT is commonly used in the imaging of the retinal nerve fibre layer in individuals with ocular hypertension and glaucoma (see Chapter 11).

OCT angiography

This is a new, non-invasive diagnostic technique that allows the blood flow in the retina and choroid to be visualized without the need for an injection of contrast medium. The disadvantage of the technique is that the classic abnormalities of traditional angiography (leakage, staining, pooling) are not shown.

- ***Indications:*** (a) diagnosis and monitoring of choroidal neovascular membranes (Fig. 1.12A), (b) diabetic retinopathy (Fig. 1.12B), (c) visualization of abnormal choroidal vessels and polyps, (d) chronic central serous retinopathy, (e) some intraocular tumours.

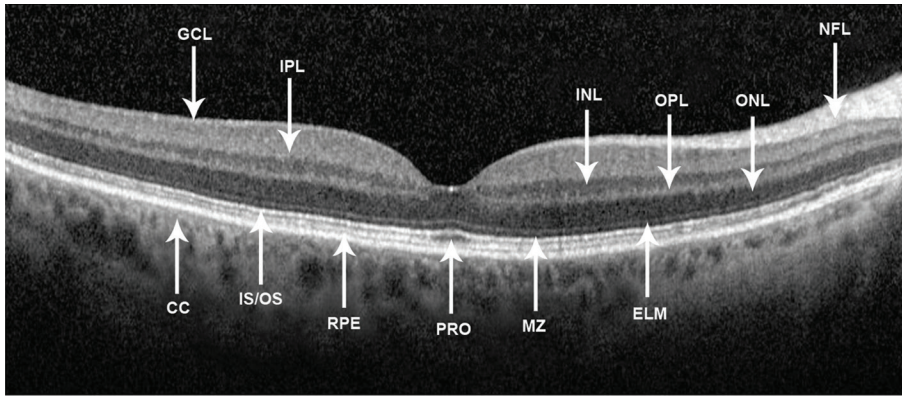

Fig. 1.11 High resolution image provided by spectral-domain OCT: cc = choriocapillaris, ELM = external limiting membrane, GLC = ganglion cell layer, INL = inner nuclear layer, IPL = inner plexiform layer, IS/OS = photoreceptor inner-segment/outer segment junction, MZ = myoid zone, NFL = nerve fibre layer, ONL = outer nuclear layer, OPL = outer plexiform layer, PRO = photoreceptor outer segments, RPE = retinal pigment epithelium. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

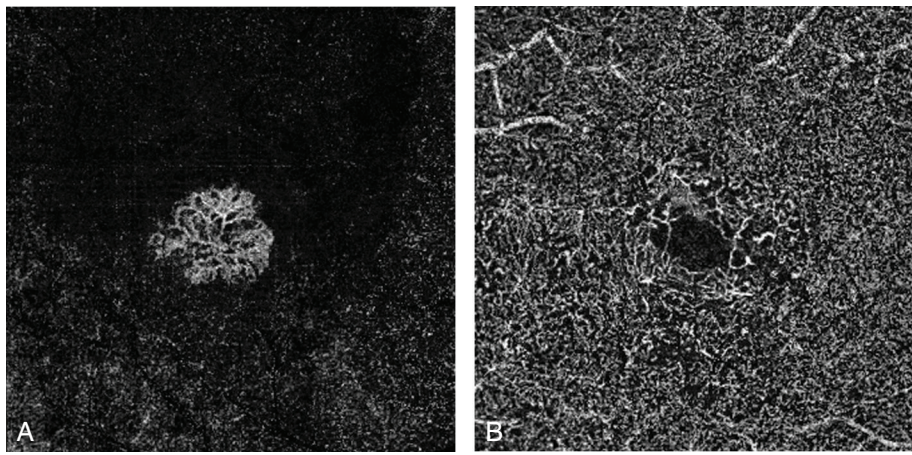

Fig. 1.12 OCT angiography: (A) choroidal neovascular membrane, (B) loss of perifoveal network in diabetic maculopathy. (Courtesy of A Ambresin.)

Fundus angiography

FLUORESCEIN ANGIOGRAPHY (FA)

This involves photographic surveillance of the passage of fluorescein through the retinal and choroidal circulations following intravenous injection.

- ***Fluorescein binding:*** 70–85% of fluorescein molecules are bound to serum proteins and the rest is unbound (free).
- ***The outer blood–retinal barrier:*** while the major choroidal vessels are impermeable to fluorescein, free molecules can pass through the choriocapillaris into the extravascular space. The fluorescein passes across Bruch membrane, but cannot cross the tight junctions of the retinal pigment epithelium (RPE).

- ***The inner blood–retinal barrier:*** tight junctions between the retinal capillary endothelial cells confine fluorescein to the lumen of the capillaries. Disruption of this barrier permits leakage into the extravascular space.
- ***Adverse effects:*** (a) discolouration of skin and urine, (b) nausea and vomiting, (c) itching and sneezing, (d) rarely: syncope, laryngeal oedema, bronchospasm and anaphylactic shock.
- ***Phases of the angiogram:*** (a) choroidal, (b) arterial, (c) arteriovenous, (d) venous, (e) late (elimination) (Fig. 1.13A-D).
- ***Hyperfluorescence:*** (a) transmission (window) defect caused by atrophy or absence of the RPE, e.g. atrophic AMD, (b) pooling of dye because of break-down of the outer blood–retinal barrier, e.g. central serous retinopathy, pigment epithelial detachment, (c) leakage of dye from abnormal vessels, e.g. choroidal neovascular membrane (CNV), breakdown of the inner blood–retinal barrier, e.g. cystoid macular oedema or retinal neovascularization, e.g. proliferative diabetic retinopathy, (d) staining of tissue, e.g. drusen.

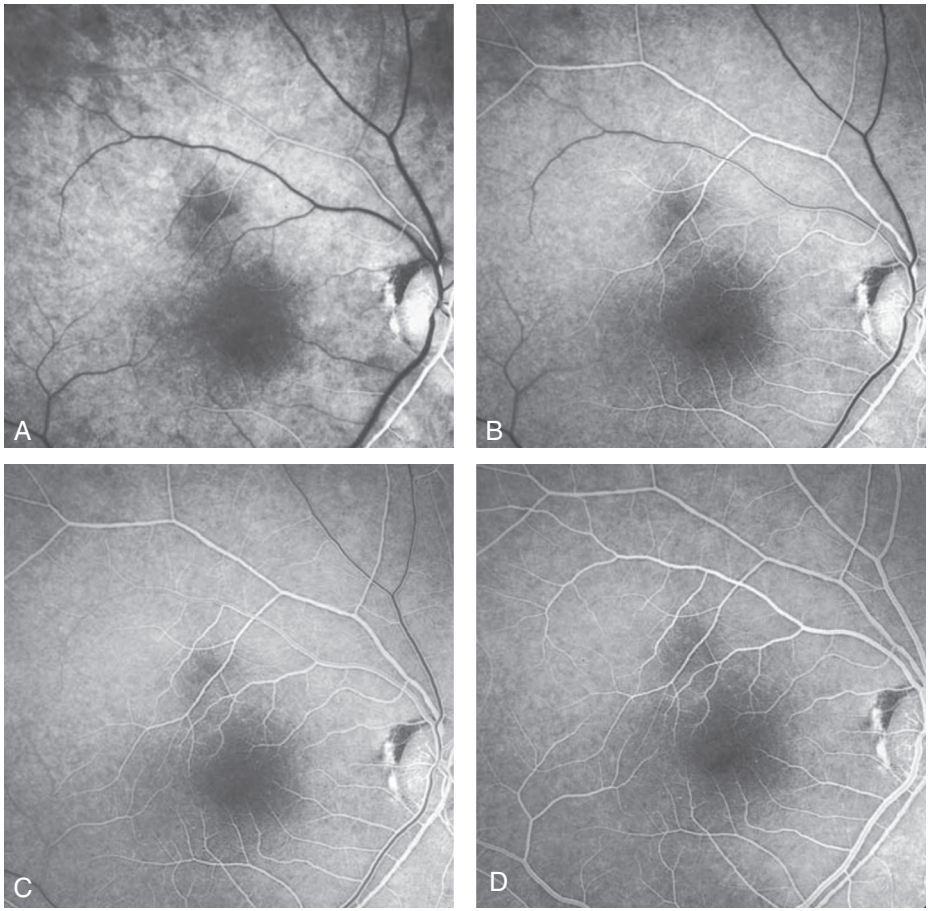

Fig. 1.13 Fluorescein angiography: (A) arterial phase showing filling of the choroid and retinal arteries, (B) arteriovenous phase showing complete arterial filling and early laminar venous flow, (C) early venous phase showing marked laminar venous flow, (D) mid-venous phase showing almost complete venous filling. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

- ***Hypofluorescence:*** (a) blockage of retinal fluorescence, e.g. retinal haemorrhage, (b) blockage of background choroidal fluorescence, e.g. subretinal or sub-RPE blood, increased RPE density, choroidal lesions, (c) filling defects, e.g. vascular occlusion, loss of capillary bed.

INDOCYANINE GREEN ANGIOGRAPHY (ICG)

This is of value when studying the choroidal circulation and is a useful adjunct to FA.

Particular indications include: (a) occult CNV, (b) CNV associated with pigment epithelial detachment, (c) recurrent CNV adjacent to a laser scar, (d) identification of feeder vessels, (e) polypoidal choroidal vasculopathy, (f) chronic central serous retinopathy, (g) breaks in Bruch membrane.

- ***ICG binding:*** about 98% of ICG molecules bind to serum proteins on entering the circulation. This phenomenon reduces the passage of ICG through the fenestrations of the choriocapillaris.
- ***Adverse effects:*** (a) should not be used in patients who are allergic to iodine or who are pregnant, (b) staining of stools, (c) nausea and vomiting, (d) sneezing, (e) pruritis, (f) rarely: skin eruptions, pyrexia, backache, skin necrosis at the injection site.
- ***Phases of the angiogram:*** (a) early phase, (b) early mid-phase, (c) late mid-phase, (d) late phase (Fig. 1.14A and B).
- ***Hyperfluorescence:*** (a) RPE window defect, (b) leakage from the retinal or choroidal circulations or optic nerve head, (c) abnormal blood vessels and polyps.
- ***Hypofluorescence:*** (a) blockage by blood, pigment or exudate, (b) obstruction of the circulation, (c) loss of vascular tissue, (d) pigment epithelial detachment.

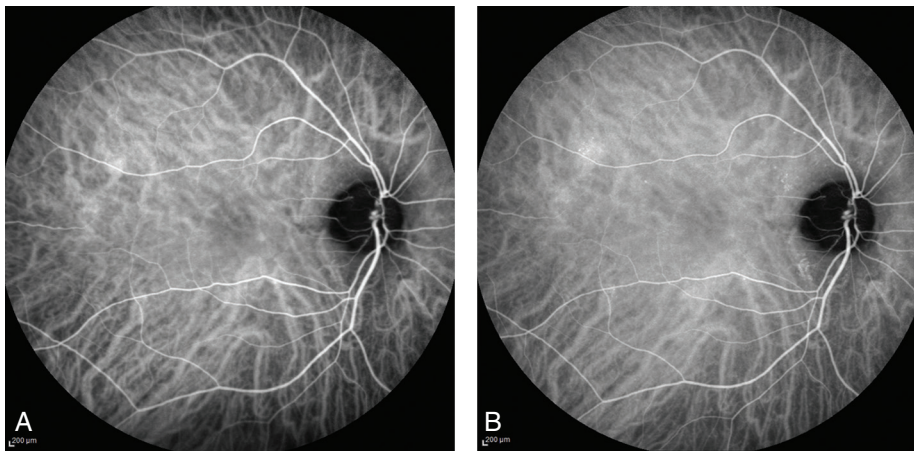

Fig. 1.14 Indocyanine green angiography: (A) early mid-phase (1–3 minutes) showing prominence of choroidal and retinal vessels, (B) late mid-phase (3–15 minutes) showing fading of choroidal vessels and diffuse tissue staining. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

CHAPTER 2

Eyelids

CHAPTER OUTLINE

Benign Nodules and Cysts

CHALAZION (MEIBOMIAN CYST)

Definition:
very common chronic sterile inflammation of a meibomian gland that may resolve spontaneously.

Diagnosis

- ***Signs:*** (a) gradually enlarging tarsal nodule (Fig. 2.1A), (b) conjunctival granulomatous extension is common, (c) secondary infection (internal hordeolum) may occur (Fig. 2.1B).
- ***Associations:*** (a) meibomian gland dysfunction, (b) rosacea, (c) seborrhoeic dermatitis, (d) use of bortezomib, used in the treatment of myeloma.

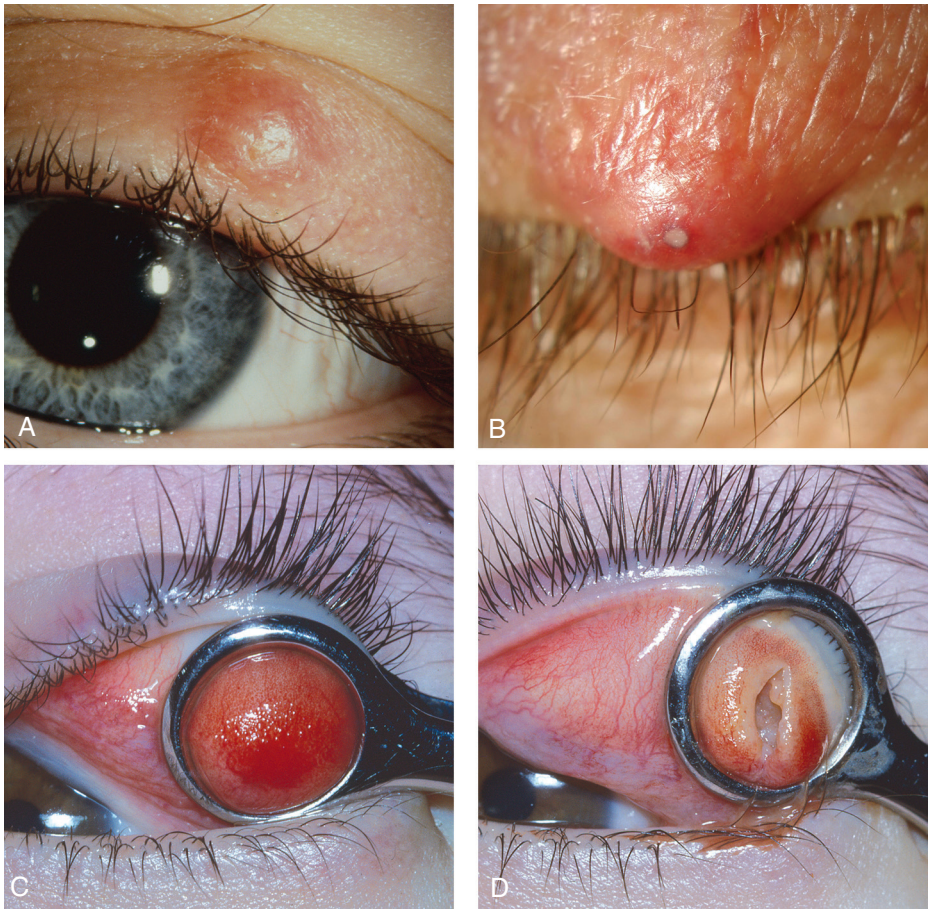

Fig. 2.1 Meibomian cyst: (A) tarsal nodule, (B) superimposed bacterial infection, (C) conjunctival view of cyst with clamp in position, (D) after curettage. (From Salmon *JF, Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

Treatment:
(a) incision and curettage (Fig. 2.1C and D), (b) local steroid injection (0.2–1 ml of 5 mg/ml triamcinolone diacetate), (c) prophylactic systemic tetracycline in severe recurrent disease.

MISCELLANEOUS

- ***Cyst of Zeis:*** nontranslucent cyst on the anterior lid margin arising from an obstructed sebaceous gland associated with a lash follicle (Fig. 2.2A).
- ***Cyst of Moll:*** translucent, fluid-filled retention cyst on the anterior lid margin (Fig. 2.2B) arising from an apocrine gland.
- ***Epidermal inclusion cyst:*** slow-growing, firm, round lesion containing keratin; located away from the lid margin. Caused by implantation of epidermis into dermis following trauma or surgery.
- ***Sebaceous (pilar) cyst:*** may occasionally occur at the medial canthus (Fig. 2.2C).

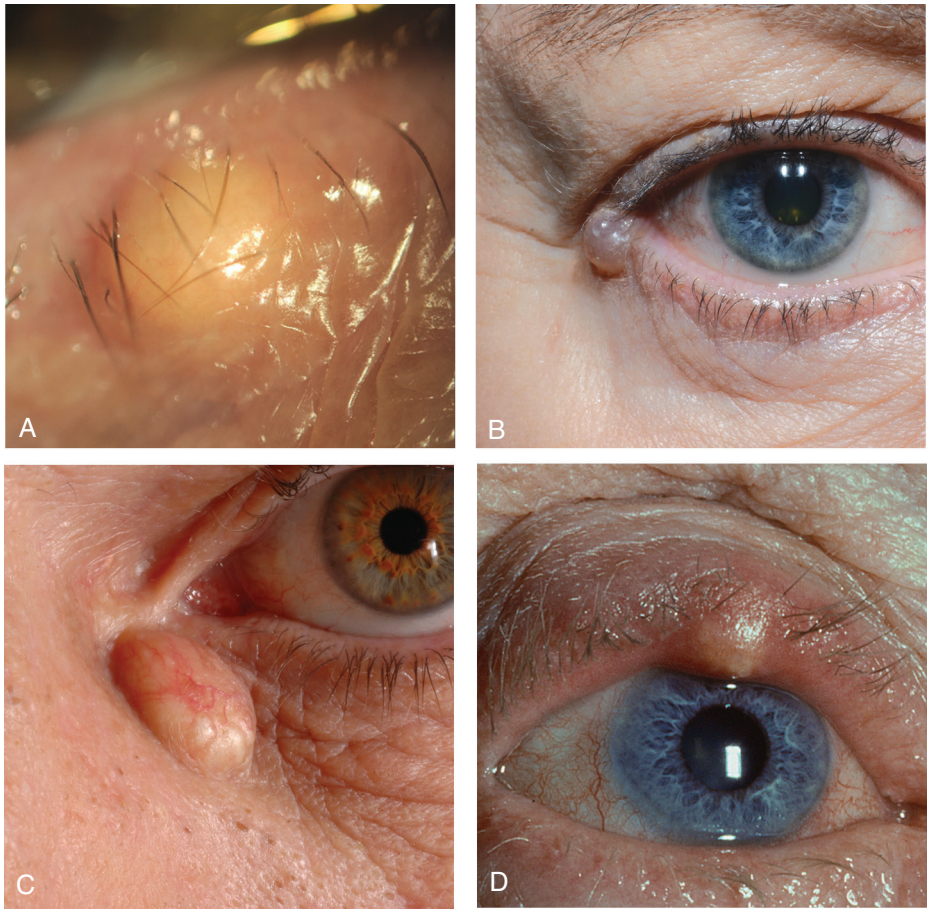

Fig. 2.2 (A) Cyst of Zeis, (B) cyst of Moll, (C) sebaceous cyst, (D) external hordeolum. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

- ***External hordeolum (stye):*** tender, pointing swelling in the lid margin, usually with a lash at its apex (Fig. 2.2D). Caused by an acute staphylococcal infection of a lash follicle.

Benign Tumours

SQUAMOUS CELL PAPILLOMA

Pathogenesis:
human papilloma virus.

Diagnosis:
narrow-based pedunculated (skin tag; Fig. 2.3A) or broad-based sessile lesion (Fig. 2.3B).

Treatment:
simple excision.

Fig. 2.3 (A) Pedunculated squamous papilloma, (B) sessile squamous papilloma, (C) basal cell papilloma, (D) actinic keratosis. (Figure 2.3C courtesy of A. Pearson.)

BASAL CELL PAPILLOMA (SEBORRHOEIC KERATOSIS)

Diagnosis:
discrete brown pedunculated or sessile lesion, often with a 'stuck on' appearance (Fig. 2.3C), in an elderly individual.

Treatment:
curettage or excision.

ACTINIC (SOLAR, SENILE) KERATOSIS

Predisposition:
elderly fair-skinned individuals with a history of chronic sun exposure; carries low malignant potential (squamous cell carcinoma).

Diagnosis:
hyperkeratotic plaque with a scaly surface and well-defined borders (Fig. 2.3D).

Treatment:
cryotherapy, or excision biopsy if there is suspicion of malignancy.

CONGENITAL MELANOCYTIC NAEVUS

Diagnosis:
small and of uniform colour. A rare variant is a 'kissing' or split naevus, that involves the upper and lower eyelid and may contain hair (Fig. 2.4A)

Treatment:
large lesions may need surgical excision, as there is a 15% risk of late malignant transformation.

ACQUIRED MELANOCYTIC NAEVUS

Diagnosis:

- ***Intradermal naevus:*** nonpigmented papilloma that may show protruding lashes (Fig. 2.4B), in an elderly individual. The cells are confined to the dermis and have no malignant potential.
- ***Junctional naevus:*** flat brown lesion (Fig. 2.4C) in a young individual. The cells are located at the junction of the dermis and epidermis and they carry very low malignant potential.
- ***Compound naevus:*** raised papule with variable pigmentation (Fig. 2.4D), in a middle-aged individual. The cells extend from the epidermis into the dermis and have low malignant potential.

Treatment:
excision for cosmesis or suspicion of malignancy.

CAPILLARY HAEMANGIOMA (STRAWBERRY NAEVUS)

Definition:
common tumour of childhood with a female-to-male ratio of 3:1. Visceral haemangiomas may be present in patients with multiple cutaneous lesions. Most present soon after birth with a rapid growth phase during infancy, followed by gradual involution.

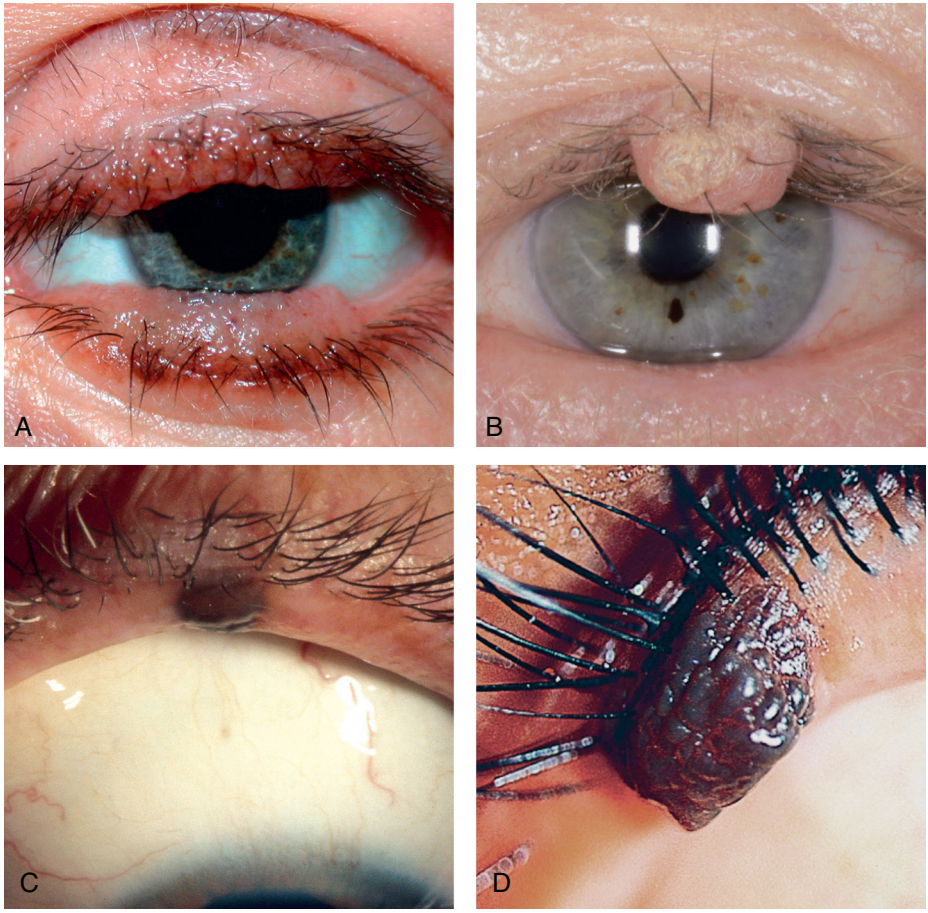

Fig. 2.4 Melanocytic naevus: (A) congenital, (B) intradermal, (C) junctional, (D) compound. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

Diagnosis:
raised, bright red lesion (Fig. 2.5A) that blanches on pressure and may swell on crying; orbital extension may be present (see Chapter 4).

Treatment
- ***Indications:*** (a) cosmesis, (b) severe ptosis, (c) corneal distortion that may give rise to amblyopia.
- ***Treatment:*** topical timolol or systemic propranolol (2 mg/kg/day in two divided doses).

PORT-WINE STAIN (NAEVUS FLAMMEUS)

Definition:
congenital lesion that is usually unilateral and occasionally bilateral. In some cases, it forms a component of Sturge–Weber syndrome.

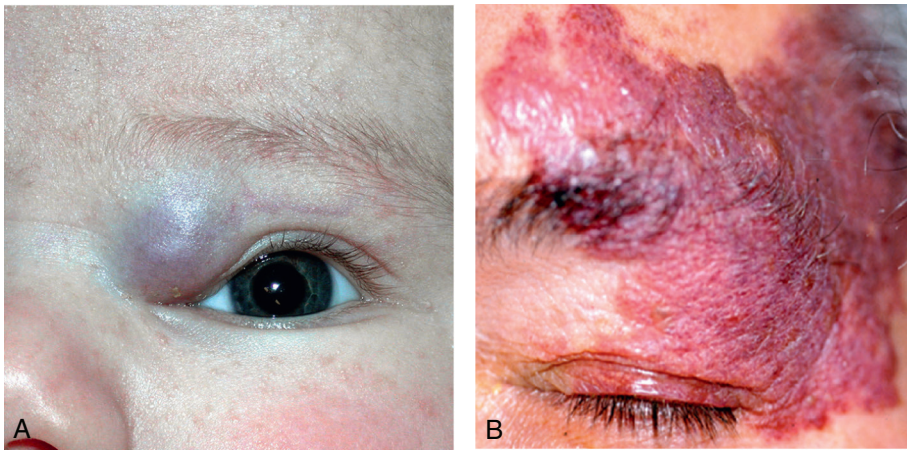

Fig. 2.5 (A) Capillary haemangioma, (B) port-wine stain. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

Diagnosis:
(a) sharply demarcated, soft pink patch that does not blanch with pressure (Fig. 2.5B), (b) darkens with age, but does not enlarge, (c) overlying skin may become hypertrophied, coarse and nodular.

Treatment:
erbium laser may decrease skin discoloration if undertaken early; photodynamic therapy. Topical imiquimod and rapamycin alone or with adjunctive laser may be considered.

Diagnosis of Sturge–Weber syndrome (encephalotrigeminal angiomatosis)

- ***Skin:*** unilateral naevus flammeus in the distribution of one or more branches of the trigeminal nerve.
- ***Brain:*** ipsilateral parietal or occipital leptomeningeal haemangioma.
- ***Ipsilateral ocular features:*** (a) glaucoma, (b) episcleral haemangioma, (c) diffuse choroidal haemangioma (see Chapter 20), (d) heterochromia iridis is uncommon.
- ***Classification:*** (a) trisystem involves the face, leptomeninges, and eyes, (b) bisystem disease involves the face and eyes or the face and leptomeninges.

XANTHELASMA

Definition:
common, typically bilateral lesion occurring in middle-aged and elderly individuals. It is associated with increased risk of coronary heart disease. In younger patients xanthelasma may indicate hypercholesterolaemia.

Diagnosis:
white-yellow subcutaneous plaques usually located medially (Fig. 2.6A).

Treatment:
(a) excision, (b) laser ablation, (c) cryotherapy, (d) systemic cholesterol abnormalities should be addressed to reduce risk of recurrence.

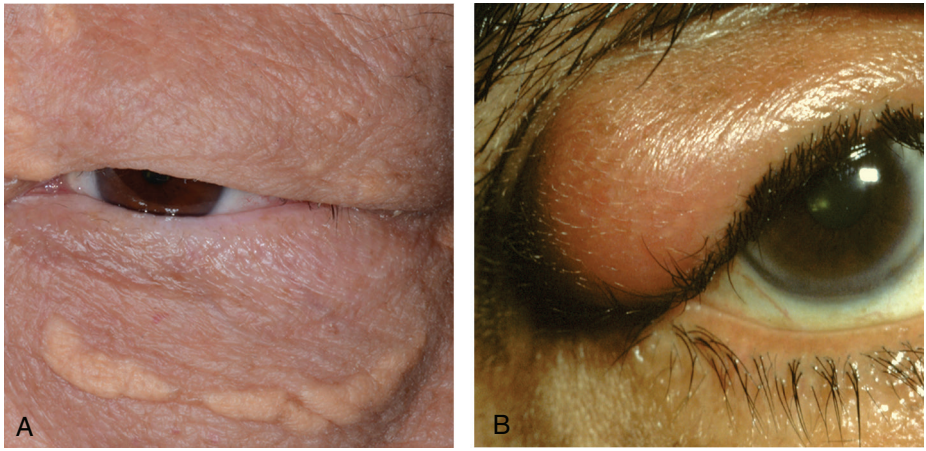

Fig. 2.6 (A) Xanthelasma, (B) neurofibroma. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

NEUROFIBROMA

- ***Plexiform:*** affects children with neurofibromatosis type 1 (NF1).
- ***Solitary:*** occurs in adults, 25% of whom have NF1.

Diagnosis:
upper lid involvement by a plexiform lesion gives rise to a characteristic S-shaped deformity (Fig. 2.6B).

Treatment:
solitary lesions can be excised, but removal of diffuse plexiform lesions may be difficult.

Malignant Tumours

RARE PREDISPOSING CONDITIONS

- ***Xeroderma pigmentosa:*** AR inheritance. Skin damage on exposure to sunlight. Predisposes to BCC, squamous carcinoma, melanoma. 90% have ocular or periocular involvement (Fig. 2.7A)
- ***Gorlin–Goltz syndrome:*** AD inheritance. Extensive congenital deformities of eye, face, CNS, developing BCC in the second decade of life.
- ***Immunosuppression***
- ***Other:*** albinism, Muir–Torre syndrome, Bazex syndrome, dysplastic naevus syndrome.

BASAL CELL CARCINOMA (BCC)

Definition:
common, slow-growing, and locally invasive but non-metastasizing tumour. 90% occur on the head and neck and 10% of these involve the eyelids; most commonly the lower.

Diagnosis
- ***Nodular:*** shiny, pearly nodule with overlying fine irregular blood vessels (Fig. 2.7B).

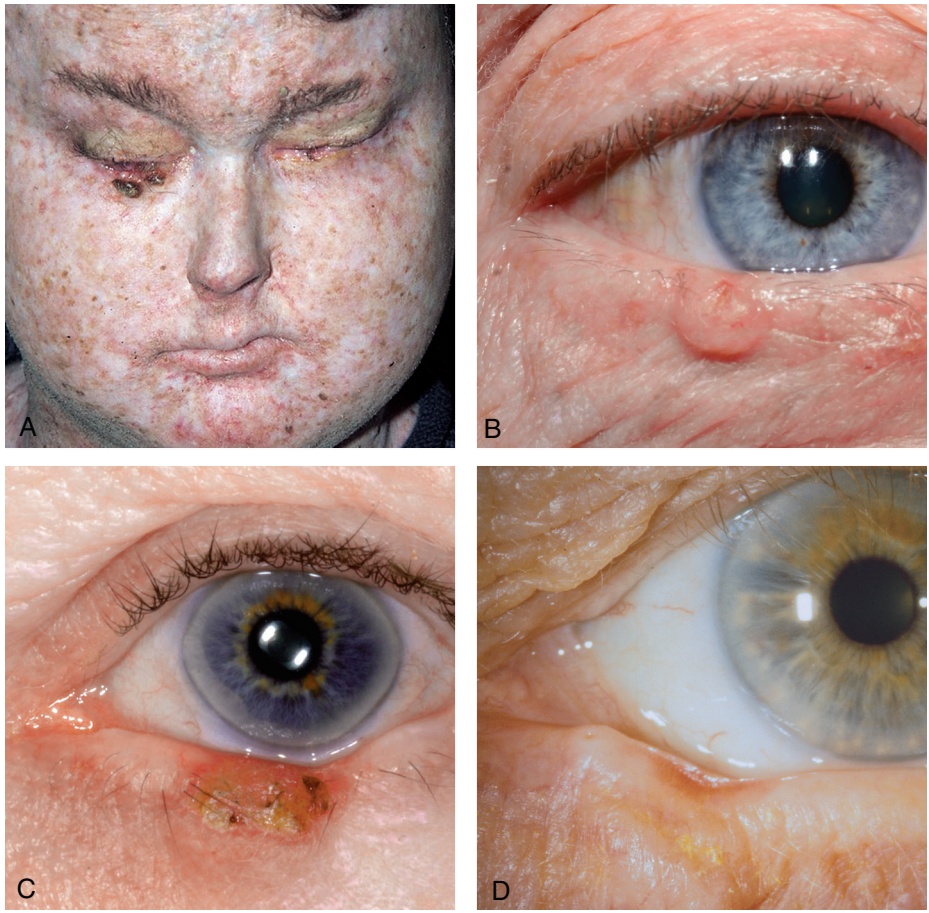

Fig. 2.7 Basal cell carcinoma: (A) xeroderma pigmentosa, (B) nodular, (c) nodulo-ulcerative, (D) sclerosing. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

- ***Nodulo-ulcerative (rodent ulcer)*:** nodule with central ulceration and rolled telangiectatic edges (Fig. 2.7C).
- ***Sclerosing (morphoeic):*** indurated plaque whose margins may be impossible to delineate clinically; often associated with loss of overlying lashes. This can mimic a localized area of chronic blepharitis (Fig. 2.7D).

Treatment
(see below).

SQUAMOUS CELL CARCINOMA (SCC)

Introduction:
SCC is much less common than BCC but is more aggressive, with metastasis to lymph nodes in about 20%. Perineural spread into the orbit may occur.

- ***Origin:*** (a) *de novo*, (b) in pre-existing actinic keratosis or (c) from carcinoma *in situ* (Bowen disease). (Fig. 2.8A)
- ***Risk factors:*** (a) increasing age, (b) fair skin, (c) chronic sun exposure, (d) immunosuppression (e.g. HIV, post-transplantation).

Diagnosis

- ***Signs:*** (a) nodular (Fig. 2.8B), (b) nodulo-ulcerative (Fig. 2.8C), (c) associated with a cutaneous horn (Fig. 2.8D). It has a predilection for the lower eyelid and the lid margin.
- ***Differentiation from BCC:*** hyperkeratosis is frequent; telangiectasis is less common and growth is usually more rapid.

Treatment

(see below).

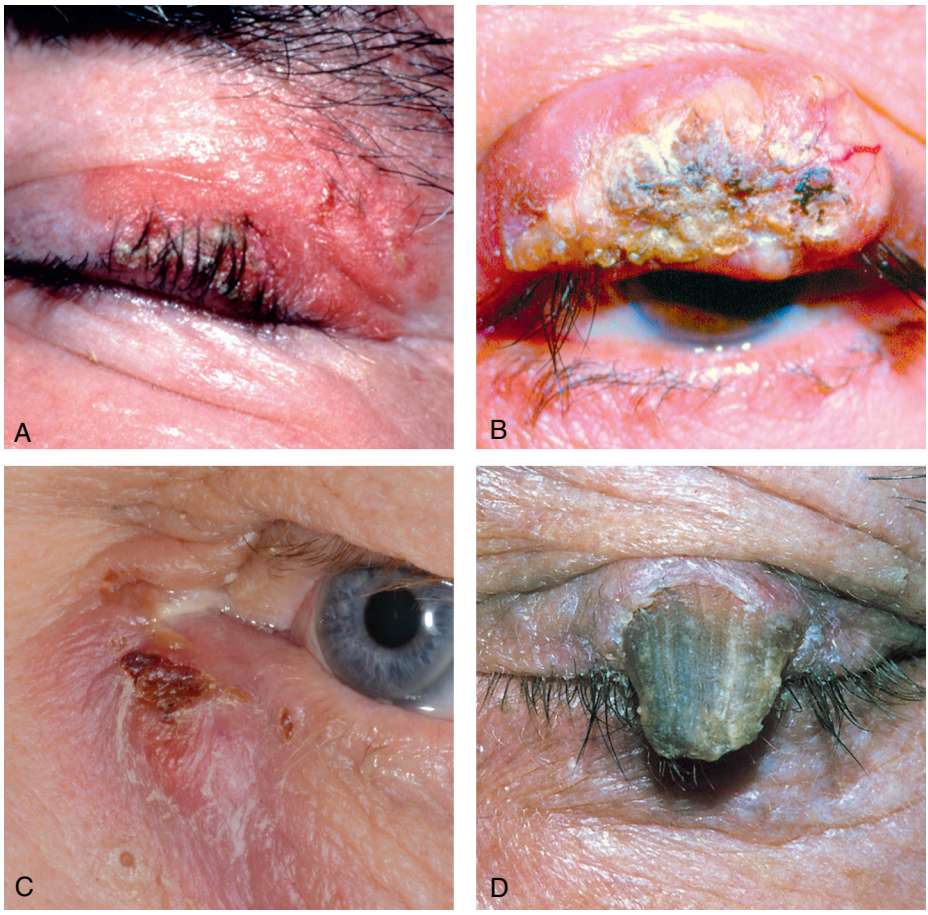

Fig. 2.8 Squamous cell carcinoma: (A) carcinoma *in situ*, (B) nodular, (C) nodulo-ulcerative, (D) associated with a cutaneous horn. (Figure 2.8B from Singh AD, Damato BE, Pe'er J, Murphree AL, Perry JD. *Clinical Ophthalmic Oncology*, Saunders 2007.)

KERATOACANTHOMA

Definition:
often regarded as a well-differentiated form of SCC. Risk factors include chronic sun exposure and immunosuppression.

Diagnosis

- ***Presentation:*** fast-growing, pink, dome-shaped hyperkeratotic lesion (Fig. 2.9A).
- ***Course:*** (a) development of a keratin-filled crater (Fig. 2.9B), (b) no change in size for 2 or 3 months, then (c) slow involution.

Treatment:
excision biopsy, radiotherapy or cryotherapy if less than 0.5 cm.

SEBACEOUS GLAND CARCINOMA

Definition:
rare, slow-growing but aggressive tumour that usually arises from the meibomian glands. It most commonly affects elderly females and has a mortality of 5–10%. In contrast to BCC and SCC, it occurs more commonly on the upper eyelid.

Diagnosis

- ***Nodular:*** beware mistaken diagnosis of chalazion. Biopsy should be performed on any atypical chalazion or suspicious persistent eyelid thickening, particularly in an older individual.
- ***Spreading:*** diffuse thickening of the lid margin (Fig. 2.10A), which can be mistaken for chronic blepharitis.
- ***Pagetoid spread:*** extension of the tumour within the epithelium including the conjunctiva, which may be mistaken for chronic inflammation.

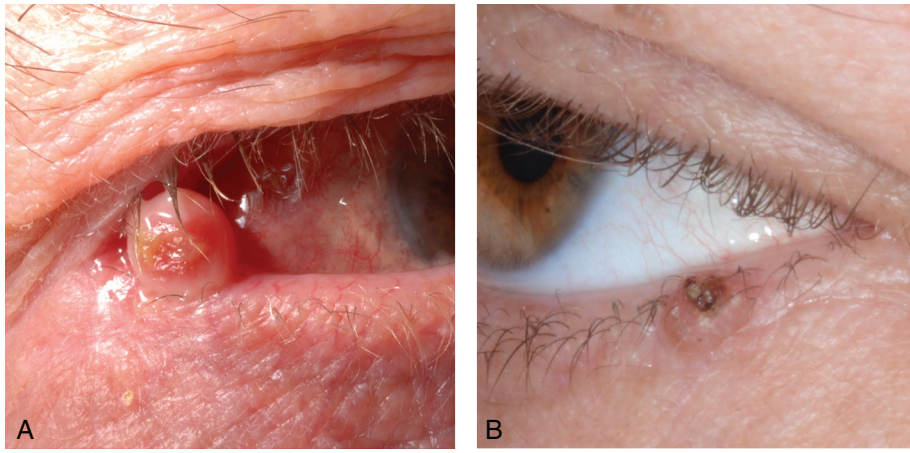

Fig. 2.9 Keratoacanthoma: (A) dome-shaped, hyperkeratotic lesion, (B) keratin-filled crater. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

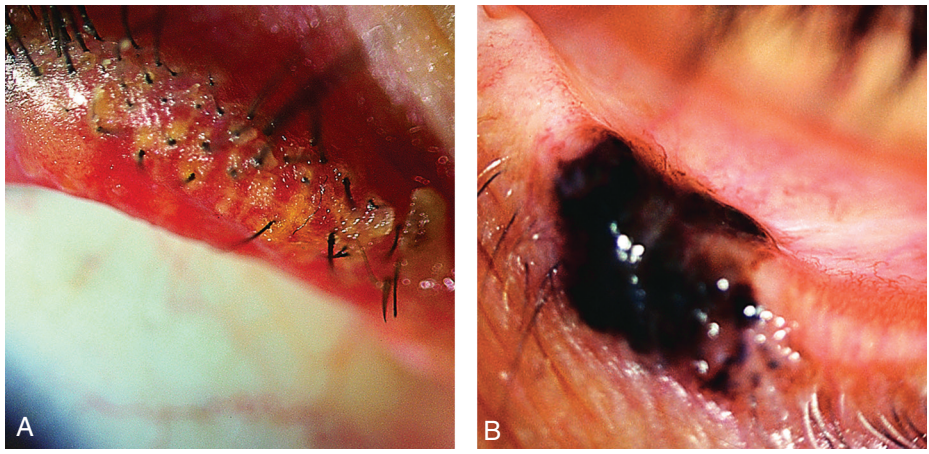

Fig. 2.10 (A) Sebaceous gland carcinoma, (B) malignant melanoma. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

MALIGNANT MELANOMA

Diagnosis

- ***Nodular:*** blue-black nodule (Fig. 2.10B)
- ***Superficial spreading:*** plaque with irregular outline and variable pigmentation.

Treatment

(see below).

PRINCIPLES OF SURGICAL TREATMENT

Biopsy

- ***Incisional:*** only part of the lesion is removed to allow histological diagnosis.
- ***Excisional:*** entire lesion is removed.

Excision

- ***Shave excision:*** for shallow epithelial tumours, such as papilloma and seborrhoeic keratosis.
- ***Full-thickness skin excision:*** most small BCCs can be excised with a 2 to 4 mm clearance margin.
- ***Radical surgical excision:*** for large BCCs and aggressive malignant tumours.
- ***Mohs micrographic surgery:*** allows maximal tumour detection and is particularly useful for lesions in which extension may not be clinically detectable such as sclerosing BCC and in difficult anatomical sites such as the medial canthus.

Reconstruction

- ***Skin defects:*** closed directly or with a local flap or skin graft.
- ***Small defects:*** (less than one-third of lid) can be closed directly, with a lateral cantholysis if necessary (Fig. 2.11A and B)
- ***Moderate defects:*** (up to half of lid) require a flap (e.g. Tenzel semicircular; Fig. 2.12A and B)
- ***Large defects:*** (over half of lid) may require: (a) posterior lamellar reconstruction using hard palate graft, buccal mucous membrane graft or a Hughes flap, (b) anterior lamellar reconstruction may involve skin advancement, a local skin flap or a free skin graft.
- ***Laissez-faire:*** approximation of wound edges with residual defect left to heal spontaneously.

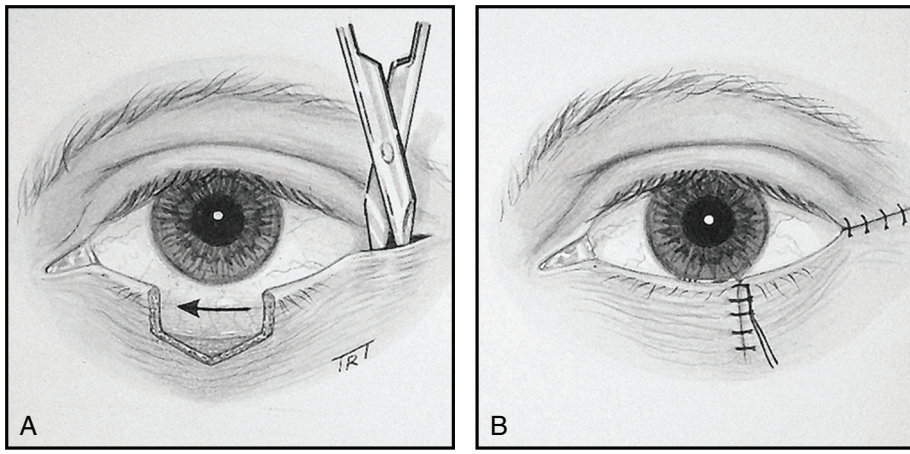

Fig. 2.11 (A and B) Reconstruction, showing incisions, direct closure and lateral cantholysis. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

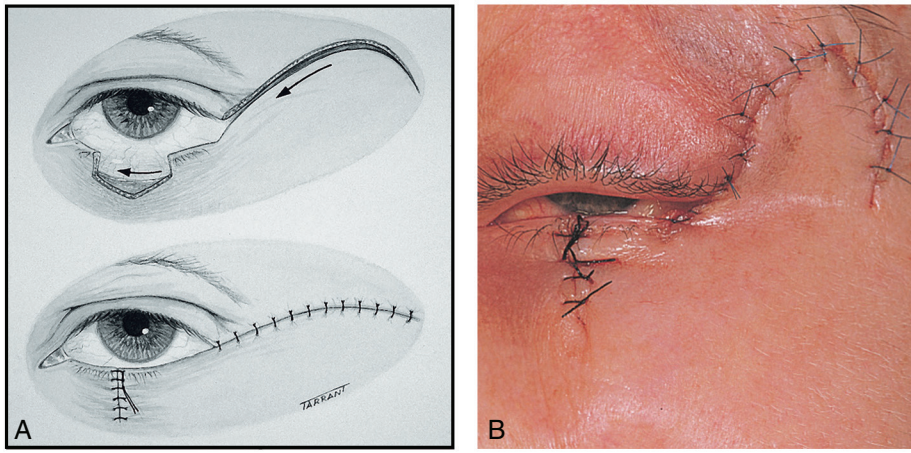

Fig. 2.12 (A and B) Tenzel semicircular flap. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

Disorders of Eyelashes

TRICHIASIS

Definition:
common acquired condition, which may occur in isolation or secondary to scarring of the lid margin.

Diagnosis

- ***Presentation:*** foreign body sensation worse on blinking; sometimes asymptomatic, particularly in long-standing cases.

- ***Signs:*** lashes are posteriorly misdirected but arise from normal sites. Corresponding punctate corneal epithelial erosions are common.
- ***Complications:*** corneal ulceration and pannus, in severe cases.

Treatment

- ***Epilation:*** with forceps for temporary control.
- ***Ablation:*** (a) argon laser for sparse lashes, (b) electrolysis (may cause scarring) or (c) cryotherapy for profuse lashes.
- ***Surgery:*** full-thickness wedge resection or anterior lamellar rotation in resistant cases.

CONGENITAL DISTICHIASIS

Definition:
very rare disorder which may be autosomal dominant (AD), and is frequently associated with lymphoedema of the legs (lymphoedema–distichiasis syndrome).

Diagnosis:
partial or complete second row of lashes emerge at or behind the meibomian gland orifices; usually well tolerated during infancy.

Treatment:
cryotherapy for lower lid distichiasis, or lamellar lid splitting with cryotherapy to the posterior lamella for upper lid involvement.

ACQUIRED DISTICHIASIS (METAPLASTIC LASHES)

Pathogenesis:
metaplasia and dedifferentiation of the meibomian glands to become hair follicles; typically associated with cicatrizing conjunctivitis (e.g. chemical injury, Stevens–Johnson syndrome, ocular cicatricial pemphigoid; see Chapter 6).

Diagnosis:
nonpigmented, often stunted, lashes originating from meibomian gland orifices (Fig. 2.13A).

Treatment:
mild cases as for trichiasis; severe cases require lamellar lid splitting and cryotherapy to the posterior lamella.

EYELASH PTOSIS

- ***Definition:*** downward sagging of upper lashes.
- ***Causes:*** (a) involutional changes, (b) long-standing facial palsy, (c) floppy eyelid syndrome (see below).

TRICHOMEGALY

- ***Definition:*** excessive eyelash growth (Fig. 2.13B).
- ***Acquired causes:*** (a) drug-induced (topical prostaglandin analogues, phenytoin, ciclosporin), (b) malnutrition, (c) AIDS, (d) porphyria, (e) hypothyroidism, (f) familial.

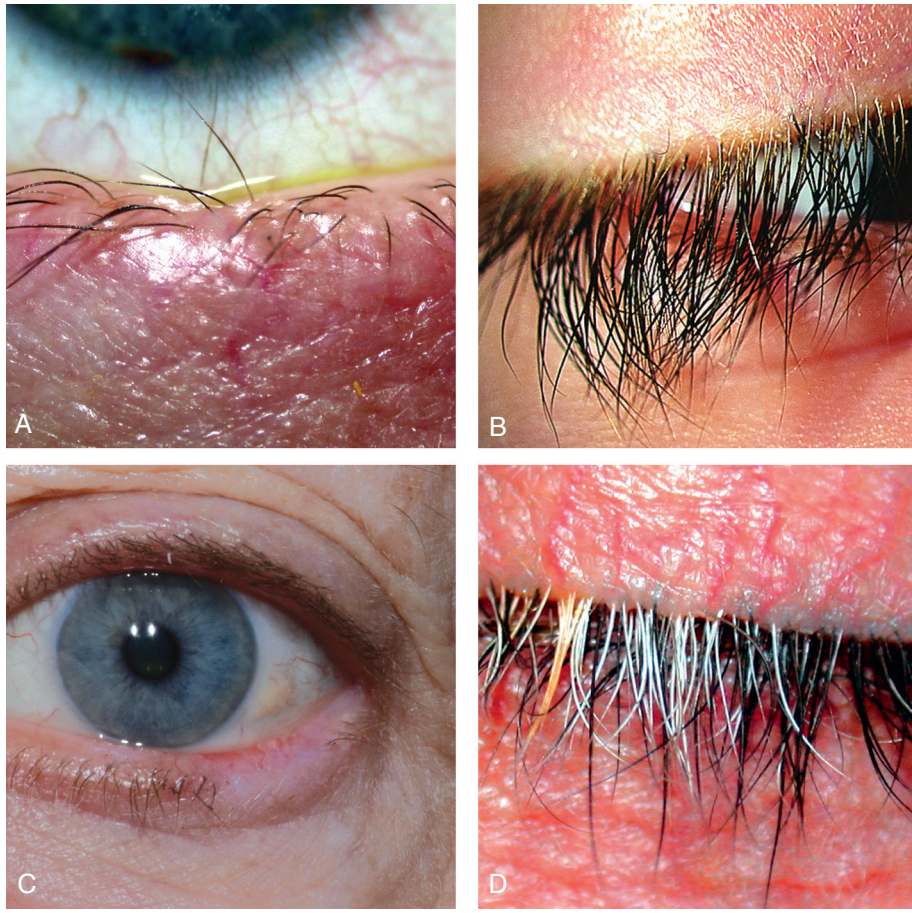

Fig. 2.13 (A) Acquired distichiasis, (B) trichomegaly, (C) madarosis, (D) poliosis. (From Salmon JF, *Kanski's Clinical Ophthalmology: A Systematic Approach*, 9th edition. Oxford, UK: Elsevier; 2020.)

- ***Associated congenital syndromes:*** (a) Oliver–McFarlane (pigmentary retinopathy, dwarfism, mental handicap), (b) Cornelia de Lange (mental and physical developmental abnormalities), (c) Goldstein–Hutt (cataract, hereditary spherocytosis), (d) Hermansky–Pudlak (albinism, bleeding diathesis).

MADAROSIS

- ***Definition:*** absence or decreased number of lashes (Fig. 2.13C).
- ***Local causes:*** (a) infiltrating lid tumours, (b) burns, (c) iatrogenic following radiotherapy or cryotherapy to the lids.
- ***Associated skin disorders:*** (a) generalized alopecia, (b) psoriasis, (c) atopic dermatitis.
- ***Associated systemic diseases:*** (a) myxoedema, (b) systemic lupus erythematosus, (c) acquired syphilis, (d) lepromatous leprosy.
- ***Following lash removal:*** (a) iatrogenic for trichiasis, (b) trichotillomania (psychiatric disorder of hair removal).

POLIOSIS

- ***Definition:*** premature localized whitening of hair, which may involve the lashes and eyebrows (Fig. 2.13D).
- ***Ocular causes:*** (a) chronic anterior blepharitis, (b) sympathetic ophthalmitis, (c) idiopathic uveitis.
- ***Systemic associations:*** (a) Vogt–Koyanagi–Harada syndrome, (b) Waardenburg syndrome, (c) vitiligo, (d) Marfan syndrome, (e) tuberous sclerosis.

Allergic Disorders

ACUTE ALLERGIC OEDEMA

Pathogenesis:
pollens that typically affect children during the spring/summer months.

Diagnosis:
sudden onset of profuse bilateral periorbital oedema (Fig. 2.14A), often accompanied by prominent jelly-like conjunctival swelling (chemosis).

Treatment:
usually unnecessary as spontaneous resolution occurs within a few hours, once exposure to the allergen is discontinued.

Differential diagnosis
- ***Dermatomyositis:*** rare chronic inflammatory disorder of muscles. In children, about half develop eyelid swelling and a heliotrope rash (Fig. 2.14B)

CONTACT DERMATITIS

Pathogenesis:
inflammatory response following exposure to a causative substance, usually a medication or contained preservative, a cosmetic preparation, or a metal. This is a type IV delayed hypersensitivity response with initial sensitizing exposure and reaction to subsequent exposure.

Diagnosis
- ***Presentation:*** itching and tearing.
- ***Signs:*** (a) eyelids show oedema, scaling, angular fissuring and tightness (Fig. 2.14C), (b) chemosis and papillary conjunctivitis, (c) mild punctate corneal epithelial erosions.

Treatment
- Avoidance of exposure to antigen, if identified.
- Change to preservative-free drops, if sensitivity to preservative is suspected.
- Topical steroids are rarely required.
- Oral antihistamines for severe cases.

ATOPIC DERMATITIS (ECZEMA)

Definition:
common idiopathic condition, typically associated with asthma and hay fever; eyelid involvement is infrequent.

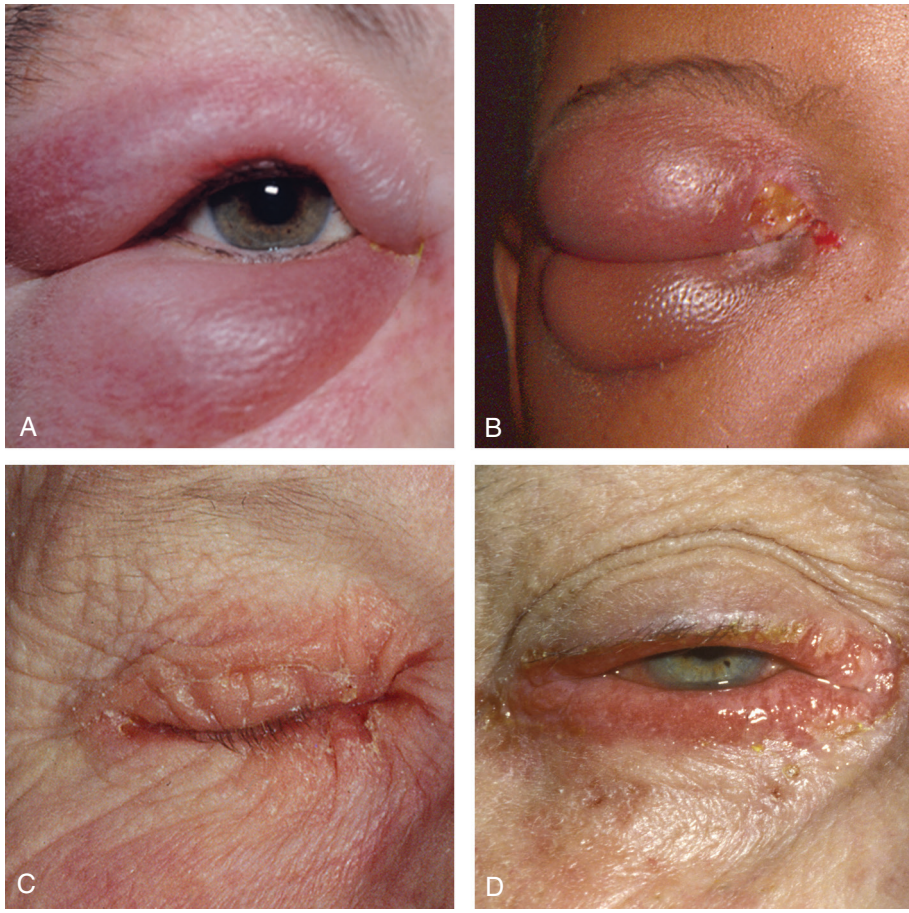

Fig. 2.14 (A) Acute allergic oedema, (B) dermatomyositis, (C) contact dermatitis, (D) atopic dermatitis. (Figure. 2.14D courtesy of S. Tuft.)

Diagnosis

- ***Presentation:*** itching and irritability of eyelid skin.
- ***Signs:*** (a) eyelids show erythema, thickening, crusting, and fissuring (Fig. 2.14D), (b) staphylococcal blepharitis, (c) madarosis, (d) keratinization of the lid margin, (e) tightening of facial skin and lower lid ectropion.

Ocular associations

- ***Common:*** vernal disease in children and chronic atopic keratoconjunctivitis in adults.
- ***Uncommon:*** (a) keratoconus, (b) presenile cataract, (c) retinal detachment.

Treatment:

emollients and mild topical steroids (e.g. hydrocortisone 1% skin cream). Beware of excessive use of periocular steroids as this can cause glaucoma.

Viral Infections

MOLLUSCUM CONTAGIOSUM

Pathogenesis:
skin infection typically affecting healthy children (peak 2–4 years) or immunocompromised individuals; transmission is by contact and subsequent autoinoculation.

Diagnosis

- Single or multiple pale, waxy, umbilicated nodules (Fig. 2.15A), (b) cheesy material can be expressed from the lesions, (c) ipsilateral chronic follicular conjunctivitis may be present.
- The lid margins should be examined carefully in any patient with chronic conjunctivitis because a causative molluscum lesion may be overlooked (Fig. 2.15B).

Treatment

- Spontaneous resolution is the rule in the immunocompetent, although autoinoculation may cause recurrences.
- Lid margin lesions with secondary conjunctivitis may be treated with: (a) shave excision, (b) cauterization, (c) cryotherapy, (d) curettage, or (e) laser.

HERPES ZOSTER OPHTHALMICUS

Pathogenesis:
shingles affecting the first division of the trigeminal nerve. It is caused by varicella zoster virus (VZV) and typically affects the elderly; tends to be more severe in immunocompromised individuals.

Diagnosis

- ***Presentation:*** 3- to 5-day prodromal phase of tiredness, fever, malaise and headache precedes the appearance of the rash. There may be pain in the affected dermatome.

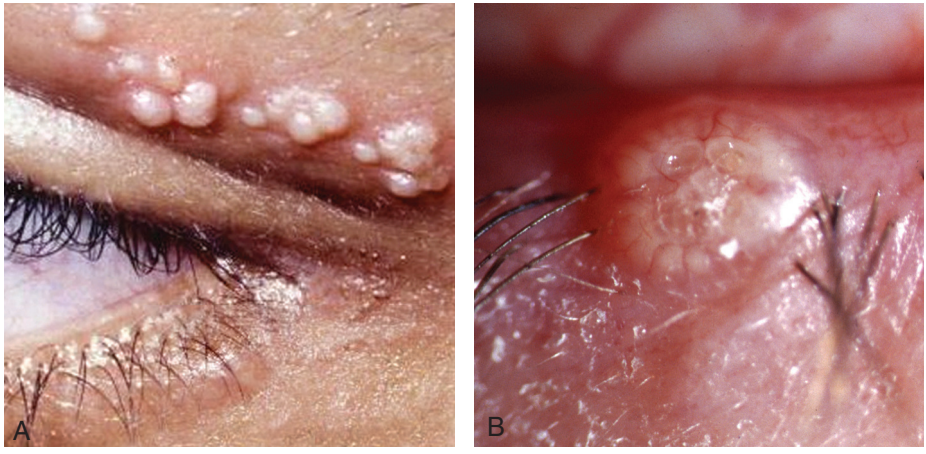

Fig 2.15 Molluscum contagiosum: (A) multiple nodules, (B) single nodule involving the lid margin, causing follicular conjunctivitis.