



KANSKI'S Clinical Ophthalmology

A Systematic Approach

Ninth Edition



KANSKI'S Clinical Ophthalmology

A Systematic Approach

John F. Salmon MD, FRCS, FRCOphth

Consultant Ophthalmic Surgeon Oxford Eye Hospital Oxford United Kingdom

KANSKI'S Clinical Ophthalmology

A Systematic Approach

Ninth Edition



© 2020, Elsevier Limited. All rights reserved.

First edition 1984 Second edition 1989 Third edition 1994 Fourth edition 1999 Fifth edition 2003 Sixth edition 2007 Seventh edition 2011 Eighth edition 2016 Ninth edition 2020

The right of John F. Salmon to be identified as author of this work has been asserted by him in accordance with the Copyright, Designs and Patents Act 1988.

No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, recording, or any information storage and retrieval system, without permission in writing from the publisher. Details on how to seek permission, further information about the Publisher's permissions policies and our arrangements with organizations such as the Copyright Clearance Center and the Copyright Licensing Agency, can be found at our website: www.elsevier.com/permissions.

This book and the individual contributions contained in it are protected under copyright by the Publisher (other than as may be noted herein).

Notices

Practitioners and researchers must always rely on their own experience and knowledge in evaluating and using any information, methods, compounds or experiments described herein. Because of rapid advances in the medical sciences, in particular, independent verification of diagnoses and drug dosages should be made. To the fullest extent of the law, no responsibility is assumed by Elsevier, authors, editors or contributors for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions, or ideas contained in the material herein.

ISBN: 978-0-7020-7711-1 978-0-7020-7712-8

Content Strategist: Kayla Wolfe Content Development Specialist: Kim Benson/Sharon Nash **Project Manager: Julie Taylor** Design: Amy Buxton Illustration Manager: Paula Catalano Marketing Manager: Claire McKenzie

Printed in China



Working together to grow libraries in ELSEVIER Book Aid developing countries

www.elsevier.com • www.bookaid.org

Contents

	Dedication	vii
	In Memoriam	
	Preface to the Ninth Edition	>
	Abbreviations	x
1	Examination Techniques	1
1	Introduction	
	Psychophysical tests	
	Perimetry	
	-	
	Slit lamp biomicroscopy of the anterior segment Fundus examination	
	Tonometry	
	Gonioscopy	
	Central corneal thickness	
2	Eyelids	37
	Introduction	38
	Non-neoplastic lesions	39
	Benign epidermal tumours	43
	Benign pigmented lesions	44
	Benign adnexal tumours	47
	Miscellaneous benign tumours	47
	Malignant tumours	50
	Disorders of the eyelashes	60
	Allergic disorders	66
	Immune-related inflammation	66
	Bacterial infections	67
	Viral infections	68
	Blepharitis	70
	Ptosis	74
	Ectropion	80
	Entropion	85
	Miscellaneous acquired disorders	87
	Cosmetic eyelid and periocular surgery	91
	Congenital malformations	93
3	Lacrimal Drainage System	90
J	Introduction	
	Acquired obstruction	
	Congenital obstruction	
	Chronic canaliculitis	
	Dacryocystitis	
4	Orbit	
	Introduction	
	Thyroid eye disease	
	Infections	
	Non-infective inflammatory disease	
	Non-neoplastic vascular abnormalities	129

	Cystic lesions Vascular tumours Lacrimal gland tumours Neural tumours Lymphoma Rhabdomyosarcoma Metastatic tumours The anophthalmic socket Craniosynostoses	134 140 142 144 147 148 150
5	Dry Eye	155
	Introduction	156
	Sjögren syndrome	158
	Clinical features	158
	Investigation	159
	Treatment	162
6	Conjunctiva	167
•	Introduction	
	Bacterial conjunctivitis	
	Viral conjunctivitis	
	Allergic conjunctivitis	
	Conjunctivitis in blistering mucocutaneous disease.	
	Miscellaneous disorders of the conjunctiva	
	Degenerations	197
	Subconjunctival haemorrhage	200
7	Cornea	
7	Cornea	203
7	Cornea	203
7	Cornea Introduction Bacterial keratitis	203 204 209
7	Cornea Introduction Bacterial keratitis Fungal keratitis	203 204 209 216
7	Cornea Introduction Bacterial keratitis Fungal keratitis Herpes simplex keratitis	203 204 209 216 218
7	Cornea Introduction Bacterial keratitis Fungal keratitis Herpes simplex keratitis Herpes zoster ophthalmicus	203 204 209 216 218 224
7	Cornea Introduction Bacterial keratitis Fungal keratitis Herpes simplex keratitis	203 204 209 216 218 224 229
7	Cornea Introduction Bacterial keratitis Fungal keratitis Herpes simplex keratitis Herpes zoster ophthalmicus Interstitial keratitis Protozoan keratitis Helminthic keratitis	203 204 209 216 218 224 229 232
7	Cornea Introduction Bacterial keratitis Fungal keratitis Herpes simplex keratitis Herpes zoster ophthalmicus Interstitial keratitis Protozoan keratitis Helminthic keratitis Bacterial hypersensitivity-mediated	203 204 209 216 218 224 229 232 234
7	Cornea Introduction	203 204 209 216 218 224 229 232 234
7	Cornea	203 204 209 216 218 224 229 232 234 234 236
7	Cornea Introduction Bacterial keratitis Fungal keratitis Herpes simplex keratitis Herpes zoster ophthalmicus Interstitial keratitis Protozoan keratitis Helminthic keratitis Bacterial hypersensitivity-mediated corneal disease Rosacea Peripheral corneal ulceration/thinning.	203 204 209 216 218 224 229 232 234 234 236 238
7	Cornea Introduction Bacterial keratitis Fungal keratitis Herpes simplex keratitis Herpes zoster ophthalmicus Interstitial keratitis Protozoan keratitis Helminthic keratitis Bacterial hypersensitivity-mediated corneal disease Rosacea Peripheral corneal ulceration/thinning. Neurotrophic keratopathy	203 204 209 216 218 224 229 232 234 234 236 238 241
7	Cornea Introduction Bacterial keratitis Fungal keratitis Herpes simplex keratitis Herpes zoster ophthalmicus Interstitial keratitis Protozoan keratitis Helminthic keratitis Bacterial hypersensitivity-mediated corneal disease Rosacea Peripheral corneal ulceration/thinning. Neurotrophic keratopathy.	203 204 209 216 218 224 229 232 234 234 234 236 238 241 242
7	Cornea	203 204 209 216 218 224 229 234 234 234 236 238 241 242 243
7	Cornea	203 204 209 216 218 224 229 234 234 234 234 234 234 234 234 234 243 243 243 248
7	Cornea	203 204 209 216 218 224 229 232 234 234 236 238 241 242 243 243 248 252
7	Cornea	203 204 209 216 218 224 229 232 234 234 236 238 241 242 243 248 252 261
7	Cornea	203 204 209 216 218 224 229 232 234 234 234 234 236 238 241 242 243 243 243 243 241 242 261 266
7	Cornea	203 204 209 216 218 224 229 232 234 234 234 234 234 234 241 242 243 243 242 243 246 266 268

8	Corneal and Refractive Surgery	275
	Keratoplasty	276
	Keratoprostheses	
	Refractive procedures	283
9	Episclera and Sclera	291
	Anatomy	
	Episcleritis	
	Immune-mediated scleritis	293
	Porphyria	
	Infectious scleritis	300
	Scleral discolouration	301
	Blue sclera	
	Miscellaneous conditions	302
10	Lens	307
	Acquired cataract	
	Management of age-related cataract	
	Congenital cataract	
	Ectopia lentis	
	Abnormalities of lens shape	342
11	Glaucoma	345
	Introduction	
	Ocular hypertension	
	Overview of glaucoma	
	Primary open-angle glaucoma	349
	Normal-tension glaucoma	367
	Primary angle-closure glaucoma	370
	Classification of secondary glaucoma	378
	Pseudoexfoliation	379
	Pigment dispersion syndrome and	
	pigmentary glaucoma	
	Neovascular glaucoma	
	Inflammatory glaucoma	
	Steroid-induced glaucoma	
	Traumatic glaucoma	
	Ghost cell 'glaucoma'	
	Iridocorneal endothelial syndrome	
	Glaucoma associated with intraocular tumours	
	Glaucoma secondary to epithelial ingrowth	
	Iridoschisis	
	Primary congenital glaucoma	
	Iridocorneal dysgenesis	399
	Glaucoma in phacomatoses	403
	Medical treatment of glaucoma	404
	Laser treatment of glaucoma	
	Trabeculectomy	
	Non-penetrating glaucoma surgery	
	Minimally invasive glaucoma surgery (MIGS)	
	Drainage shunts	
12	Uveitis	
	Classification	
	Clinical features	424

	Investigation	429
	Treatment	
	Immunomodulatory therapy for	102
	non-infectious uveitis	433
	Uveitis in spondyloarthropathies	
	Fuchs uveitis syndrome	
	Uveitis in juvenile idiopathic arthritis (JIA)	
	Uveitis in bowel disease	
	Uveitis in renal disease	
	Intermediate uveitis	
	Vogt-Koyanagi-Harada (VKH) syndrome	
	Sympathetic ophthalmitis	
	Lens-induced uveitis	
	Sarcoidosis	
	Behçet disease	
	Parasitic uveitis	
	Viral uveitis	
	Fungal uveitis	
	Bacterial uveitis	
	Miscellaneous idiopathic chorioretinopathies	
13	Retinal Vascular Disease4	-95
	Retinal circulation	.496
	Diabetic retinopathy	
	Non-diabetic retinopathy	
	Retinal venous occlusive disease	514
	Retinal arterial occlusive disease	525
	Ocular ischaemic syndrome	
	Hypertensive eye disease	532
	Hypertensive eye disease Sickle-cell retinopathy	.532 .533
	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy	.532 .533 .536
	Hypertensive eye disease Sickle-cell retinopathy	.532 .533 .536
	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm	.532 .533 .536 .536 .540
	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia	.532 .533 .536 .536 .540 .543
	Hypertensive eye disease	.532 .533 .536 .536 .540 .543 .546
	Hypertensive eye disease	.532 .533 .536 .536 .540 .543 .546 .546
	Hypertensive eye disease	.532 .533 .536 .536 .540 .543 .546 .546
	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy	.532 .533 .536 .536 .540 .543 .546 .546 .546 .547 .549
	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy Lipaemia retinalis	.532 .533 .536 .536 .540 .543 .546 .546 .547 .549 .549
	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy	.532 .533 .536 .536 .540 .543 .546 .546 .547 .549 .549
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders	532 533 536 540 543 546 546 546 547 549 549 550
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders	532 533 536 540 543 546 546 547 549 549 550
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders Introduction	532 533 536 540 543 546 546 546 546 549 550 550 555
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders Clinical evaluation of macular disease	532 533 536 540 543 546 547 549 550 550 555 556 557
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders Introduction Clinical evaluation of macular disease	532 533 536 540 543 546 547 549 550 550 555 556 557 558
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders Introduction Clinical evaluation of macular disease Age-related macular degeneration	532 533 536 540 543 546 544 549 550 550 555 556 557 558 572
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders Introduction Clinical evaluation of macular disease Investigation of macular disease Age-related macular degeneration Retinal angiomatous proliferation	532 533 536 540 543 546 546 547 549 550 555 556 557 558 5572 558
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders Introduction Clinical evaluation of macular disease Age-related macular degeneration Retinal angiomatous proliferation Polypoidal choroidal vasculopathy	532 533 536 540 543 546 546 547 549 550 555 556 557 558 5572 558
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders Introduction Clinical evaluation of macular disease Age-related macular degeneration Retinal angiomatous proliferation Polypoidal choroidal vasculopathy Peripheral exudative haemorrhagic	532 533 536 540 543 546 547 549 549 550 555 556 557 558 572 589 589
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Valsalva retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders Introduction Clinical evaluation of macular disease Age-related macular degeneration Retinal angiomatous proliferation Polypoidal choroidal vasculopathy Peripheral exudative haemorrhagic chorioretinopathy	532 533 536 540 543 546 547 549 549 550 555 556 557 558 572 589 589 589
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders Introduction Clinical evaluation of macular disease Age-related macular degeneration Retinal angiomatous proliferation Polypoidal choroidal vasculopathy Peripheral exudative haemorrhagic	532 533 536 540 543 546 547 549 550 555 556 557 558 572 589 589 591 591
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders Introduction Clinical evaluation of macular disease Age-related macular degeneration Retinal angiomatous proliferation Polypoidal choroidal vasculopathy Peripheral exudative haemorrhagic chorioretinopathy	532 533 536 540 543 546 546 547 549 550 556 557 558 572 589 589 591 591 592
14	Hypertensive eye disease Sickle-cell retinopathy Thalassaemia retinopathy Retinopathy of prematurity Retinal artery macroaneurysm Primary retinal telangiectasia Eales disease Radiation retinopathy Purtscher retinopathy Valsalva retinopathy Valsalva retinopathy Lipaemia retinalis Retinopathy in blood disorders Acquired Macular Disorders Introduction Clinical evaluation of macular disease Investigation of macular disease Age-related macular degeneration Retinal angiomatous proliferation Polypoidal choroidal vasculopathy Peripheral exudative haemorrhagic chorioretinopathy Idiopathic choroidal neovascularization Vitreomacular interface disorders	532 533 536 540 543 546 547 549 550 555 556 557 558 572 589 589 591 591 592 598

Microcystic macular oedema	604
Degenerative myopia	604
Angioid streaks	607
Choroidal folds	609
Hypotony maculopathy	610
Solar retinopathy	610
Focal choroidal excavation	611
Dome-shaped macula	612
Low visual aids	613

15 Hereditary Fundus Dystrophies615

Introduction	616
Investigation	
Generalized photoreceptor dystrophies	
Macular dystrophies	
Generalized choroidal dystrophies	
Hereditary vitreoretinopathies	
Albinism	
Cherry-red spot at the macula	
Gilen y-leu spot at the macula	

16 Retinal Detachment653

Introduction	654
Peripheral lesions predisposing to	057
retinal detachment	657
Posterior vitreous detachment	663
Retinal breaks	666
Rhegmatogenous retinal detachment	668
Tractional retinal detachment	681
Exudative retinal detachment	681
Pars plana vitrectomy	683

17 Vitreous Opacities691

18	Strabismus	697
	Introduction	698
	Amblyopia	707
	Clinical evaluation	
	Pseudostrabismus	726
	Heterophoria	727
	Vergence abnormalities	727
	Esotropia	728
	Exotropia	
	Congenital cranial dysinnervation disorders	735
	Monocular elevation deficiency	737
	Brown syndrome	737
	Alphabet patterns	738
	Surgery	739
	Complications of strabismus surgery	
	Botulinum toxin chemodenervation	743

19	Neuro-ophthalmology	745
	Neuroimaging	746
	Optic nerve	751
	Pupils	779
	Chiasm	
	Retrochiasmal pathways	
	Ocular motor nerves	
	Supranuclear disorders of ocular motility	
	Nystagmus	
	Ocular myopathies	
	Miller Fisher syndrome	
	Neurofibromatosis	
	Migraine	
	Neuralgias	
	Facial spasm	
	Disorders of circadian rhythm	
	Neuro-ophthalmology of space flight	825
20	Ocular Tumours	
	Benign epibulbar tumours	
	Malignant and premalignant epibulbar tumours	
	Iris tumours	
	Iris cysts	
	Ciliary body tumours	
	Tumours of the choroid	
	Neural retinal tumours	
	Retinal vascular tumours	
	Primary intraocular lymphoma	
	Tumours of the retinal pigment epithelium	
	Paraneoplastic syndromes	8/8
21	Ophthalmic Side Effects of	
	Systemic Medication	
	Eyelids	882
	Cornea	882
	Ciliary effusion	882
	Lens	
	Uveitis	
	Retina	
	Optic nerve	
	Visual cortex	
22	Trauma	891
	Eyelid trauma	
	Orbital trauma	
	Trauma to the globe	
	Chemical injuries	
	Thermal burns	916

Dedication

This book is dedicated to my wife Susie, to my children Mark and Nicola, and to my sister, Margaret, who initially awakened my interest in ophthalmology

In Memoriam

Jack J. Kanski MD, MS, FRCS, FRCOphth, Consultant Ophthalmic Surgeon (1939–2019)



Jack Kanski wrote more than 30 books, but is best known for *Clinical Ophthalmology*, which was first published in 1984. The book has been studied by ophthalmology and optometry students throughout the world since that time. It has justifiably become a classic because of its highly organized format, succinct but comprehensive text and superb clinical photographs. His encyclopaedic knowledge of ophthalmology, meticulous attention to detail and unique ability to sort the wheat from the chaff will be sorely missed. His legacy will live on in the minds of those who benefitted from his teaching.

Preface to the Ninth Edition

In presenting this new edition of *Kanski's Clinical Ophthalmology*, I am reminded of a quotation from Lewis Carroll's *Alice's Adventures in Wonderland*: "What is the use of a book", thought Alice, "without pictures or conversations?". The ninth edition of this classic textbook is filled with beautiful illustrations and considerable information and is intended to be a useful and comprehensive basis for general ophthalmic practice. It has been a privilege to work on this remarkable book and I am grateful to Jack Kanski and the staff of Elsevier for entrusting me with the task.

The challenge has been to cover the entire field of ophthalmology for a worldwide audience without depending on subspecialists to prepare each chapter. In order to do this, I have maintained Jack Kanski's unique approach of presenting core clinical knowledge in a systemic and succinct form. Brad Bowling has had a significant influence on the two previous editions and his accuracy and meticulous attention to detail has been extremely helpful. I have reverted to the format that was used in the sixth edition by starting with an initial chapter on examination techniques. Special investigations remain in the chapters where they are most relevant.

Each chapter has been updated and the latest evidence-based diagnostic and therapeutic approaches have been covered, including genetics, immunotherapy and imaging techniques. Many new illustrations have been added and better examples of a range of conditions have been used. Jack Kanski's idea of including important 'tips' has been reintroduced. I have included sufficient practical information for trainees to manage common ophthalmic conditions in the clinic and enough detail on rare conditions to enable them to prepare for their examinations without resorting to the internet.

I have been extremely fortunate to have received help from colleagues past and present, to whom I wish to express my grateful thanks. The photographers and research staff at the Oxford Eye Hospital have been wonderfully supportive. Jack Kanski generously gave me his huge collection of images. My friends in

South Africa, Tony Murray (strabismus) and Trevor Carmichael (cornea), provided help with the text and images of pathology that are not easily found in developed countries. I received many pictures from Jonathan Norris and Elizabeth Insull (oculoplastics), Darius Hildebrand and Manoj Parulekar (paediatrics), Peter Issa and Christine Kiire (medical retina), Bertil Damato (ocular oncology), Martin Leyland (corneal surgery), C.K. Patel (vitreoretinal surgery), Patsy Terry (ultrasound) and Pieter Pretorius (neuroradiology). Mitch Ménage provided good pictures of common conditions. Aude Ambresin and Carl Herbort (Switzerland) supplied state-of-the-art retinal images. I have kept many of the outstanding pictures that Chris Barry and Simon Cheng (Australia) provided for the eighth edition. Single examples of rare conditions have been kindly provided by a number of colleagues throughout the United Kingdom and elsewhere and their contribution has been recognized next to the images provided. Other individuals have helped substantially with previous editions of Clinical Ophthalmology, including Terry Tarrant, the artist who produced the meticulous ocular paintings. I also wish to thank Kim Benson, Sharon Nash, Kayla Wolfe, Julie Taylor, Anne Collett and the production team at Elsevier.

The ninth edition of *Kanski's Clinical Ophthalmology* could not have been produced in the time available without the help of my assistant, Carolyn Bouter, whose resilience, diligence, intelligence and skill were evident throughout the 6 months that she worked with me. I have also had the good fortune to work with Jonathan Brett, a world-class photographer and artist, whose genius is present in hundreds of the images included in this edition. My wife, Susie, has been extremely supportive throughout this project and her happy and helpful nature has made the task a pleasant and enjoyable experience.

> John F. Salmon 2019

Abbreviations

AAION	arteritic anterior ischaemic optic neuropathy
AAU	acute anterior uveitis
AC	anterior chamber
AC/A ratio	accommodative convergence/accommodation
110/11 10010	ratio
ACE	angiotensin converting enzyme
AD	autosomal dominant
AF	autofluorescence
AGIS	Advanced Glaucoma Treatment Study
AHP	abnormal head posture
AI	accommodative insufficiency
AIBSE	acute idiopathic blind spot enlargement syndrome
AIDS	acquired immune deficiency syndrome
AIM	(unilateral) acute idiopathic maculopathy
AION	anterior ischaemic optic neuropathy
AIR	autoimmune retinopathies
AKC	atopic keratoconjunctivitis
ALT	argon laser trabeculoplasty
AMD	age-related macular degeneration
AMN	acute macular neuroretinopathy
ANA	antinuclear antibody
ANCA	antineutrophil cytoplasmic antibodies
APD	afferent pupillary defect
APMPPE	acute posterior multifocal placoid pigment epitheliopathy
AR	autosomal recessive
AREDS	Age-Related Eye Disease Study
ARN	acute retinal necrosis
ARPE	acute retinal pigment epitheliitis
AZOOR	acute zonal occult outer retinopathy
AZOR	acute zonal outer retinopathy
BADI	bilateral acute depigmentation of the iris
BCC	basal cell carcinoma
BCVA	best-corrected visual acuity
BIO	binocular indirect ophthalmoscopy
BP	blood pressure
BRAO	branch retinal artery occlusion
BRVO	branch retinal vein occlusion
BSV	binocular single vision
BUT	breakup time
CAI	carbonic anhydrase inhibitor
CCDD	congenital cranial dysinnervation disorders central corneal thickness
CCT	
C/D	cup/disc
CDCR	canaliculodacryocystorhinostomy
CF CHED	counts (or counting) fingers
CHED CHP	congenital hereditary endothelial dystrophy compensatory head posture
CHP	
	congenital hypertrophy of the retinal pigment epithelium
CI	convergence insufficiency

C-MIN	conjunctival melanocytic intraepithelial neoplasia
СМО	cystoid macular oedema (US = CME)
CNS	central nervous system
CNV	choroidal neovascularization
CNVM	choroidal neovascular membrane
COX-2	cyclo-oxygenase-2
CPEO	chronic progressive external ophthalmoplegia
CRAO	central retinal artery occlusion
CRP	C-reactive protein
CRVO	central retinal vein occlusion
CSC	central serous chorioretinopathy
CSC/CSCR	central serous chorioretinopathy
CSMO	clinically significant macular oedema (US = CSME)
CSR	central serous chorioretinopathy
CSS	central suppression scotoma
СТ	computed tomography
CXL	corneal collagen cross-linking
DALK	deep anterior lamellar keratoplasty
DCCT	Diabetes Control and Complication Trial
DCR	dacryocystorhinostomy
DCT	dynamic contour tonometry
DMEK	Descemet membrane endothelial keratoplasty
DMO	diabetic macular oedema ($US = DME$)
DR	diabetic retinopathy
DRCR.net	Diabetic Retinopathy Clinical Research Network
DRPPT	dark room prone provocative test
DRS	Diabetic Retinopathy Study
DSAEK	Descemet stripping automated endothelial keratoplasty
DVD	dissociated vertical deviation
ECG	electrocardiogram
EDTA	ethylenediaminetetraacetic acid
EGFR	epidermal growth factor inhibitors
EKC	epidemic keratoconjunctivitis
EMGT	Early Manifest Glaucoma Trial
EOG	electro-oculography/gram
ERG	electroretinography/gram
ERM	epiretinal membrane
ESR	erythrocyte sedimentation rate
ETDRS	Early Treatment Diabetic Retinopathy Study
ETROP	Early Treatment of Retinopathy of Prematurity
FA	fluorescein angiography (also FFA)
FAF	fundus autofluorescence
FAP	familial adenomatous polyposis
FAZ	foveal avascular zone
FBA	frosted branch angiitis
FBC	full blood count
FDT	frequency doubling test
FFM	fundus flavimaculatus
FTMH	full-thickness macular hole

5-FU	5-fluorouracil	MMC	mitomycin C
GA	geographic atrophy	MPS	mucopolysaccharidosis
GAT	Goldmann applanation tonometry	MRI	magnetic resonance imaging
GCA	giant cell arteritis	MS	multiple sclerosis
GDD	glaucoma drainage device	NF1	neurofibromatosis type I
GHT	glaucoma hemifield test	NF2	neurofibromatosis type I
GPA	guided progression analysis	NPDR	non-proliferative diabetic retinopathy
GPC	giant papillary conjunctivitis	NRR	neuroretinal rim
G-TOP	glaucoma tendency orientated perimetry	NSAID	
HAART	highly active antiretroviral therapy	NSR	non-steroidal anti-inflammatory drug neurosensory retina
HFA		NTG	
	Humphrey field analyser		normal-tension glaucoma
HIV	human immunodeficiency virus	NVD	new vessels on the disc
HM	hand movements	NVE	new vessels elsewhere
HRT	Heidelberg retinal tomography	NVG	neovascular glaucoma
HSV-1	herpes simplex virus type 1	NVI	new vessels iris
HSV-2	herpes simplex virus type 2	OCT	optical coherence tomography/gram
HZO	herpes zoster ophthalmicus	OHT	ocular hypertension
ICE	iridocorneal endothelial	OHTS	Ocular Hypertension Treatment Study
ICG	indocyanine green	OIS	ocular ischaemia syndrome
ICGA	indocyanine green angiography	OKN	optokinetic nystagmus
ICROP	International Classification of Retinopathy of	OVD	ophthalmic viscosurgical devices
IFIC	Prematurity	PAC	primary angle closure
IFIS	intraoperative floppy iris syndrome	PACG	primary angle-closure glaucoma
Ig	immunoglobulin	PACS	primary angle-closure suspect
IK	interstitial keratitis	PAM	primary acquired melanosis
ILM	internal limiting membrane	PAN	polyarteritis nodosa
IMT	idiopathic macular telangiectasia	PAS	peripheral anterior synechiae
INO	internuclear ophthalmoplegia	PC	posterior chamber
INR	international normalized ratio	PCO	posterior capsular opacification
IOFB	intraocular foreign body	PCR	polymerase chain reaction
IOID	idiopathic orbital inflammatory disease	PCV	polypoidal choroidal vasculopathy
IOL	intraocular lens	PDR	proliferative diabetic retinopathy
IOP	intraocular pressure	PDS	pigment dispersion syndrome
IRMA	intraretinal microvascular abnormality	PDT	photodynamic therapy
IRVAN	idiopathic retinal vasculitis, aneurysms and	PED	pigment epithelial detachment
M	neuroretinitis syndrome	PEE	punctate epithelial erosions
ITC	iridotrabecular contact	PEK	punctate epithelial keratitis
IU	intermediate uveitis	PEHCR	peripheral exudative haemorrhagic
IVTS	International Vitreomacular Traction Study		chorioretinopathy
JIA	juvenile idiopathic arthritis	PH	pinhole
KC	keratoconus	PHM	posterior hyaloid membrane
KCS	keratoconjunctivitis sicca	PIC	punctate inner choroidopathy
KP	keratic precipitate	PIOL	primary intraocular lymphoma
LA	local anaesthetic	PION	posterior ischaemic optic neuropathy
LASEK	laser (also laser-assisted) epithelial keratomileusis	РКР	penetrating keratoplasty
LASIK	laser-assisted in situ keratomileusis	PMMA	polymethyl methacrylate
LN	latent nystagmus	POAG	primary open-angle glaucoma
LSCD	limbal stem cell deficiency	POHS	presumed ocular histoplasmosis syndrome
LV	loss variance	РР	pars planitis
MALT	mucosa-associated lymphoid tissue	PPCD	posterior polymorphous corneal dystrophy
MCP	multifocal choroiditis and panuveitis	PPDR	preproliferative diabetic retinopathy
MEWDS	multiple evanescent white dot syndrome	PPM	persistent placoid maculopathy
MFC	multifocal choroiditis and panuveitis	PPRF	paramedian pontine reticular formation
MIGS	minimally invasive glaucoma surgery	PPV	pars plana vitrectomy
MLF	medial longitudinal fasciculus	PRK	photorefractive keratectomy
MLT	micropulse laser technology	PRP	panretinal photocoagulation
	_ 07	1 101	Pulletinai photocougulation

PS	posterior synechiae	SJS	Stevens–Johnson syndrome
PSD	pattern standard deviation	SLK	superior limbic keratoconjunctivitis
PSS	Posner-Schlossman syndrome	SLT	selective laser trabeculoplasty
PUK	peripheral ulcerative keratitis	SMILE	small incision lenticule extraction
PVD	posterior vitreous detachment	SPARCS	Spaeth Richman contrast sensitivity test
PVR	proliferative vitreoretinopathy	SPK	superficial punctate keratitis
PVRL	primary vitreoretinal lymphoma	SRF	subretinal fluid
PXE	pseudoxanthoma elasticum	SS	Sjögren syndrome
PXF	pseudoexfoliation	STIR	short T ₁ inversion recovery
RA	rheumatoid arthritis	SWAP	short-wave automated perimetry
RAO	retinal artery occlusion	TAL	total axial length
RAPD	relative afferent pupillary defect	ТВ	tuberculosis
RD	retinal detachment	TEN	toxic epidermal necrolysis
RNFL	retinal nerve fibre layer	TGF	transforming growth factor
ROCK	Rho-kinase	TIA	transient ischaemic attack
ROP	retinopathy of prematurity	TM	trabecular meshwork
RP	retinitis pigmentosa	TRD	tractional retinal detachment
RPC	relentless placoid chorioretinitis	TRP	targeted retinal photocoagulation
RPE	retinal pigment epithelium	TTT	transpupillary thermotherapy
RRD	rhegmatogenous retinal detachment	UBM	ultrasonic biomicroscopy
RS	retinoschisis	US	ultrasonography
RVO	retinal vein occlusion	VA	visual acuity
SANS	space flight-associated neuro-ocular syndrome	VEGF	vascular endothelial growth factor
SAP	standard automated perimetry	VEP	visual(ly) evoked potential(s)
SCC	squamous cell carcinoma	VFI	visual field index
SCN	suprachiasmic nucleus	VHL	von Hippel–Lindau syndrome
SD-OCT	spectral domain optical coherence tomography	VKC	vernal keratoconjunctivitis
SF	short-term fluctuation	VKH	Vogt–Koyanagi–Harada syndrome
SFU	progressive subretinal fibrosis and uveitis	VMA	vitreomacular adhesion
	syndrome	VMT	vitreomacular traction
SIC	solitary idiopathic choroiditis	VZV	varicella zoster virus
SITA	Swedish Interactive Thresholding Algorithm	XL	X-linked

Chapter

Examination Techniques

INTRODUCTION 2

PSYCHOPHYSICAL TESTS 2

Visual acuity 2 Near visual acuity 4 Contrast sensitivity 4 Amsler grid 5 Light brightness comparison test 6 Photostress test 6 Colour vision testing 7 Plus lens test 9

PERIMETRY 9

Definitions 9 Testing algorithms 11 Testing patterns 14 Analysis 14 High-sensitivity field modalities 17 Sources of error 18 Microperimetry 18

SLIT LAMP BIOMICROSCOPY OF THE ANTERIOR SEGMENT 20

Direct illumination 20 Scleral scatter 20 Retroillumination 20 Specular reflection 21

FUNDUS EXAMINATION 21

Direct ophthalmoscopy 21 Slit lamp biomicroscopy 22 Goldmann three-mirror examination 23 Head-mounted binocular indirect ophthalmoscopy 25 Fundus drawing 27

TONOMETRY 27

Goldmann tonometry 27 Other forms of tonometry 29 Ocular response analyser and corneal hysteresis 30

GONIOSCOPY 30

Introduction 30 Indirect gonioscopy 32 Direct gonioscopy 33 Identification of angle structures 34 Pathological findings 35

CENTRAL CORNEAL THICKNESS 36

INTRODUCTION

Patients with ophthalmic disease must have their vision accurately measured and their eyes need to be examined using specialized techniques. Special investigations should be used to supplement the findings of clinical examination. Electrophysical tests, fluorescein angiography and optical coherence tomography are discussed in later chapters.

PSYCHOPHYSICAL TESTS

Visual acuity

Snellen visual acuity

Distance visual acuity (VA) is directly related to the minimum angle of separation (subtended at the nodal point of the eye) between two objects that allow them to be perceived as distinct. In practice, it is most commonly carried out using a Snellen chart, which utilizes black letters or symbols (optotypes) of a range of sizes set on a white chart (Fig. 1.1), with the subject reading the chart from a standard distance. Distance VA is usually first measured using a patient's refractive correction, generally their own glasses or contact lenses. For completeness, an unaided acuity may also be recorded. The eye reported as having worse vision should be tested first, with the other eye occluded. It is important to push the patient to read every letter possible on the optotypes being tested.

- Normal monocular VA equates to 6/6 (metric notation; 20/20 in non-metric 'English' notation) on Snellen testing. Normal corrected VA in young adults is often superior to 6/6.
- **Best-corrected VA** (BCVA) denotes the level achieved with optimal refractive correction.

- Pinhole VA: a pinhole (PH) aperture compensates for the effect of refractive error, and consists of an opaque occluder perforated by one or more holes of about 1 mm diameter (Fig. 1.2). However, PH acuity in patients with macular disease and posterior lens opacities may be worse than with spectacle correction. If the VA is less than 6/6 Snellen equivalent, testing is repeated using a pinhole aperture.
- **Binocular VA** is usually superior to the better monocular VA of each eye, at least where both eyes have roughly equal vision.

Very poor visual acuity

- **Counting (or counts) fingers** (CF) denotes that the patient is able to tell how many fingers the examiner is holding up at a specified distance (Fig. 1.3), usually 1 metre.
- Hand movements (HM) is the ability to distinguish whether the examiner's hand is moving when held just in front of the patient.
- **Perception of light (PL)**: the patient can discern only light (e.g. pen torch), but no shapes or movement. Careful occlusion of the other eye is necessary. If poor vision is due solely to a dense media opacity such as cataract, the patient should



Fig. 1.2 Pinhole

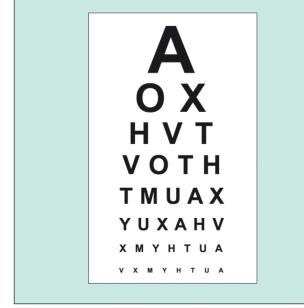


Fig. 1.1 Snellen visual acuity chart



Fig. 1.3 Testing of 'counts fingers' visual acuity

CHAPTER Examination Techniques

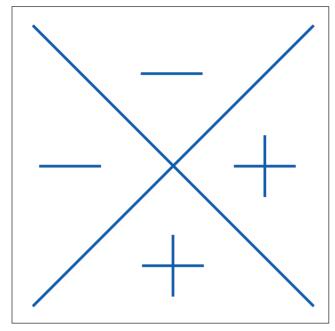


Fig. 1.4 Notation for the projection of light test (right eye); the patient cannot detect light directed from the superior and temporal quadrants



Fig. 1.5 Bailey-Lovie chart

Testing

readily be able to determine the direction from which the light is being projected (Fig. 1.4).

LogMAR acuity

LogMAR charts address many of the deficiencies of the Snellen chart (Table 1.1), and are the standard means of VA measurement in research and, increasingly, in clinical practice.

- LogMAR is an acronym for the base-10 logarithm of the minimum angle of resolution (MAR) and refers to the ability to resolve the elements of an optotype. Thus, if a letter on the 6/6 (20/20) equivalent line subtends 5' of arc, and each limb of the letter has an angular width of 1', a MAR of 1' is needed for resolution. For the 6/12 (20/40) line, the MAR is 2', and for the 6/60 (20/200) line it is 10'.
- The logMAR score is simply the base-10 log of the MAR. Because the log of the MAR value of 1' is zero, 6/6 is equivalent to logMAR 0.00. The log of the 6/60 MAR of 10' is 1, so 6/60 is equivalent to logMAR 1.00. The log of the 6/12 MAR of 2' is 0.301, giving a logMAR score of 0.30. Scores better than 6/6 have a negative value.
- As letter size changes by 0.1 logMAR units per row and there are five letters in each row, each letter can be assigned a score of 0.02. The final score can therefore take account of every letter that has been read correctly and the test should continue until half of the letters on a line are read incorrectly.

LogMAR charts

- The Bailey–Lovie chart (Fig. 1.5).
 - \circ $\;$ Used at 6 m testing distance.
 - Each line of the chart comprises five letters and the spacing between each letter and each row is related to the width

Table 1.1 Comparison of Snellen and logMAR Visual Acuity

Snellen	LogMAR
Shorter test time	Longer test time
More letters on the lower lines introduces an unbalanced 'crowding' effect	Equal numbers of letters on different lines controls for 'crowding' effect
Fewer larger letters reduces accuracy at lower levels of VA	Equal numbers of letters on low and higher acuity lines increases accuracy at lower VA
Variable readability between individual letters	Similar readability between letters
Lines not balanced with each other for consistency of readability	Lines balanced for consistency of readability
6 m testing distance: longer testing lane (or a mirror) required	4 m testing distance on many charts: smaller testing lane (or no mirror) required
Letter and row spacing not systematic	Letter and row spacing set to optimize contour interaction
Lower accuracy and consistency so relatively unsuitable for research	Higher accuracy and consistency so appropriate for research
Straightforward scoring system	More complex scoring
Easy to use	Less user-friendly

and the height of the letters. The letter signs are rectangular rather than square, as with the EDTRS chart. A 6/6 letter is 5' in height by 4' in width.

- The distance between two adjacent letters on the same row is equal to the width of a letter from the same row, and the distance between two adjacent rows is the same as the height of a letter from the lower of the two rows.
- Snellen VA values and logMAR VA are listed to the right and left of the rows respectively.
- The ETDRS (Early Treatment Diabetic Retinopathy Study) chart is calibrated for 4 m. This chart utilizes balanced rows comprising Sloan optotypes, developed to confer equivalent legibility between individual letters and rows. ETDRS letters are square, based on a 5 × 5 grid, i.e. 5' × 5' for the 6/6 equivalent letters at 6 m.
- Computer charts are available that present the various forms of test chart on display screens, including other means of assessment such as contrast sensitivity (see below).

TIP LogMAR charts are commonly used in clinical trials because they are the most accurate method of measuring VA.

Near visual acuity

Near vision testing can be a sensitive indicator of the presence of macular disease. A range of near vision charts (including logMAR and ETDRS versions) or a test-type book can be used. The book or chart is held at a comfortable reading distance and this is measured and noted. The patient wears any necessary distance correction together with a presbyopia correction if applicable (usually their own reading spectacles). The smallest type legible is recorded for each eye individually and then using both eyes together (Fig. 1.6).

Contrast sensitivity

 Principles. Contrast sensitivity is a measure of the ability of the visual system to distinguish an object against its background.



Fig. 1.6 Near visual acuity using a magnifying lens

A target must be sufficiently large to be seen, but must also be of high enough contrast with its background. A light grey letter will be less well seen against a white background than a black letter. Contrast sensitivity represents a different aspect of visual function to that tested by the spatial resolution tests described above, which all use high-contrast optotypes.

- Many conditions reduce both contrast sensitivity and VA, but under some circumstances (e.g. amblyopia, optic neuropathy, some cataracts, and higher-order aberrations), visual function measured by contrast sensitivity can be reduced whilst VA is preserved.
- Hence, if patients with good VA complain of visual symptoms (typically evident in low illumination), contrast sensitivity testing may be a useful way of objectively demonstrating a functional deficit. Despite its advantages, it has not been widely adopted in clinical practice.
- The Pelli–Robson contrast sensitivity letter chart is viewed at 1 metre and consists of rows of letters of equal size (spatial frequency of 1 cycle per degree) but with decreasing contrast of 0.15 log units for groups of three letters (Fig. 1.7). The patient reads down the rows of letters until the lowest-resolvable group of three is reached.
- Sinusoidal (sine wave) gratings require the test subject to view a sequence of increasingly lower contrast gratings.
- **Spaeth Richman contrast sensitivity test** (SPARCS) is performed on a personal computer with internet access. It can be accessed online. Each patient is supplied with an identification number and instructions on how to do the test. The test takes 5–10 minutes per eye and measures both central and peripheral contrast sensitivity. Since the test is based on gratings it can be used on illiterate patients.



Fig. 1.7 Pelli-Robson contrast sensitivity letter chart

The Amsler grid evaluates the 20° of the visual field centred on fixation (Fig. 1.8). It is principally useful in screening for and monitoring macular disease, but will also demonstrate central visual field defects originating elsewhere. Patients with a substantial risk of choroidal neovascularization (CNV) should be provided with an Amsler grid for regular use at home.

TIP An Amsler grid is a simple and easy method of monitoring central visual field and is commonly abnormal in patients with macular disease.

Charts

There are seven charts, each consisting of a 10-cm outer square (Figs 1.9 and 1.10).

- Chart 1 consists of a white grid on a black background, the outer grid enclosing 400 smaller 5-mm squares. When viewed at about one-third of a metre, each small square subtends an angle of 1°.
- Chart 2 is similar to chart 1 but has diagonal lines that aid fixation for patients with a central scotoma.
- Chart 3 is identical to chart 1 but has red squares. The red-onblack design aims to stimulate long wavelength foveal cones. It is used to detect subtle colour scotomas and desaturation in toxic maculopathy, optic neuropathy and chiasmal lesions.
- Chart 4 consists only of random dots and is used mainly to distinguish scotomas from metamorphopsia, as there is no form to be distorted.

- Chart 5 consists of horizontal lines and is designed to detect metamorphopsia along specific meridians. It is of particular use in the evaluation of patients describing difficulty reading.
- Chart 6 is similar to chart 5 but has a white background and the central lines are closer together, enabling more detailed evaluation.
- Chart 7 includes a fine central grid, each square subtending an angle of a half degree, and is more sensitive.

Technique

The pupils should not be dilated, and in order to avoid a photostress effect, the eyes should not yet have been examined on the slit lamp. A presbyopic refractive correction should be worn if appropriate. The chart should be well illuminated and held at a comfortable reading distance, optimally around 33 cm.

- One eye is covered.
- The patient is asked to look directly at the central dot with the uncovered eye, to keep looking at this, and to report any distortion or waviness of the lines on the grid.
- Reminding the patient to maintain fixation on the central dot, he or she is asked if there are blurred areas or blank spots anywhere on the grid. Patients with macular disease often report that the lines are wavy whereas those with optic neuropathy tend to remark that some of the lines are missing or faint but not distorted.
- The patient is asked if he or she can see all four corners and all four sides of the square – a missing corner or border should raise the possibility of causes other than macular disease, such as glaucomatous field defects or retinitis pigmentosa.

The patient may be provided with a recording sheet and pen and asked to draw any anomalies (Fig. 1.11).

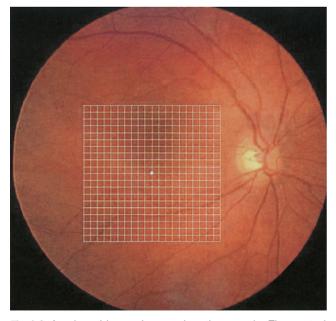


Fig. 1.8 Amsler grid superimposed on the macula. The central fixation dot of the grid does not coincide with the foveal anatomical centre in this image (*Courtesy of A Franklin*)

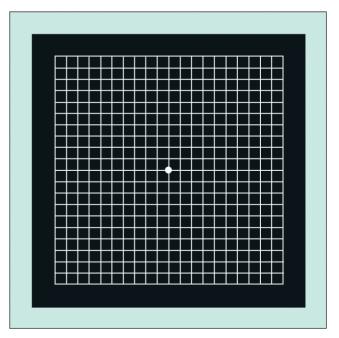


Fig. 1.9 Amsler grid chart (Courtesy of A Franklin)

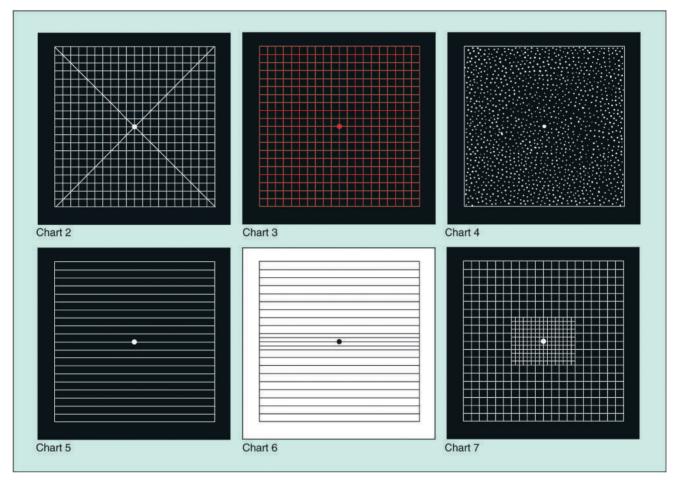


Fig. 1.10 Amsler charts 2–7 (Courtesy of A Franklin)

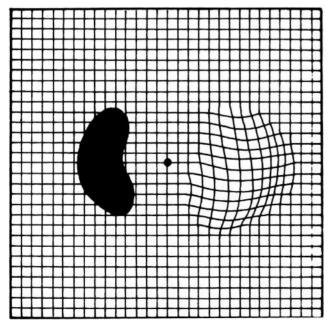


Fig. 1.11 Stylized Amsler recording sheet shows wavy lines indicating metamorphopsia, and a dense scotoma

Light brightness comparison test

This is a test of optic nerve function, which is usually normal in early and moderate retinal disease. It is performed as follows:

- A light from an indirect ophthalmoscope is shone first into the normal eye and then the eye with suspected disease.
- The patient is asked whether the light is symmetrically bright in both eyes.
- In optic neuropathy the patient will report that the light is less bright in the affected eye.
- The patient is asked to assign a relevant value from 1 to 5 to the brightness of the light in the diseased eye, in comparison to the normal eye.

Photostress test

• **Principles.** Photostress testing is a gross test of dark adaptation in which the visual pigments are bleached by light. This causes a temporary state of retinal insensitivity perceived by the patient as a scotoma. The recovery of vision is dependent on the ability of the photoreceptors to re-synthesize visual pigment. The test may be useful in detecting maculopathy

when ophthalmoscopy is equivocal, as in mild cystoid macular oedema or central serous retinopathy. It may also differentiate visual loss caused by macular disease from that caused by an optic nerve lesion.

- Techniques
 - \circ $\;$ The best-corrected distance VA is determined.
 - The patient fixates on the light of a pen torch or an indirect ophthalmoscope held about 3 cm away for about 10 seconds (Fig. 1.12A).
 - The photostress recovery time (PSRT) is the time taken to read any three letters of the pre-test acuity line and is normally between 15 and 30 seconds (Fig. 1.12B).
 - The test is performed on the other, presumably normal, eye and the results are compared.
 - The PSRT is prolonged relative to the normal eye in macular disease (sometimes 50 seconds or more) but not in an optic neuropathy.

Colour vision testing

Introduction

Assessment of colour vision is useful in the evaluation of optic nerve disease and in determining the presence of a congenitally anomalous colour defect. Dyschromatopsia may develop in retinal dystrophies prior to the impairment of other visual parameters. Colour vision depends on three populations of retinal cones, each with a specific peak sensitivity: blue (tritan) at 414–424 nm, green (deuteran) at 522–539 nm and red (protan) at 549–570 nm. Normal colour perception requires all these primary colours to match those within the spectrum. Any given cone pigment may be deficient (e.g. protanomaly – red weakness) or entirely absent (e.g. protanopia – red blindness). Trichromats possess all three types of cones (although not necessarily functioning perfectly), while absence of one or two types of cones renders an individual a dichromat or monochromat, respectively. Most individuals with congenital colour defects are anomalous trichromats and use abnormal proportions of the three primary colours to match those in the light spectrum. Those with red–green deficiency caused by abnormality of red-sensitive cones are protanomalous, those with abnormality of green-sensitive cones are deuteranomalous and those with blue–green deficiency caused by abnormality of bluesensitive cones are tritanomalous. Acquired macular disease tends to produce blue–yellow defects, and optic nerve lesions red–green defects.

Colour vision tests

- The Ishihara test is designed to screen for congenital protan and deuteran defects. It is simple, widely available and frequently used to screen for a red-green colour vision deficit. The test can be used to assess optic nerve function. It consists of a test plate followed by 16 plates, each with a matrix of dots arranged to show a central shape or number that the subject is asked to identify (Fig. 1.13A). A colour-deficient person will only be able to identify some of the figures. Inability to identify the test plate (provided VA is sufficient) indicates non-organic visual loss.
- The City University test consists of 10 plates, each containing a central colour and four peripheral colours (Fig. 1.13B) from which the subject is asked to choose the closest match.

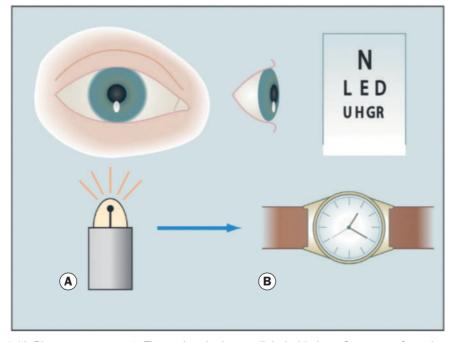


Fig. 1.12 Photostress test. **(A)** The patient looks at a light held about 3 cm away from the eye, for about 10 seconds; **(B)** the photostress recovery time is the time taken to read any three letters of the pre-test acuity line and is normally between 15 and 30 seconds

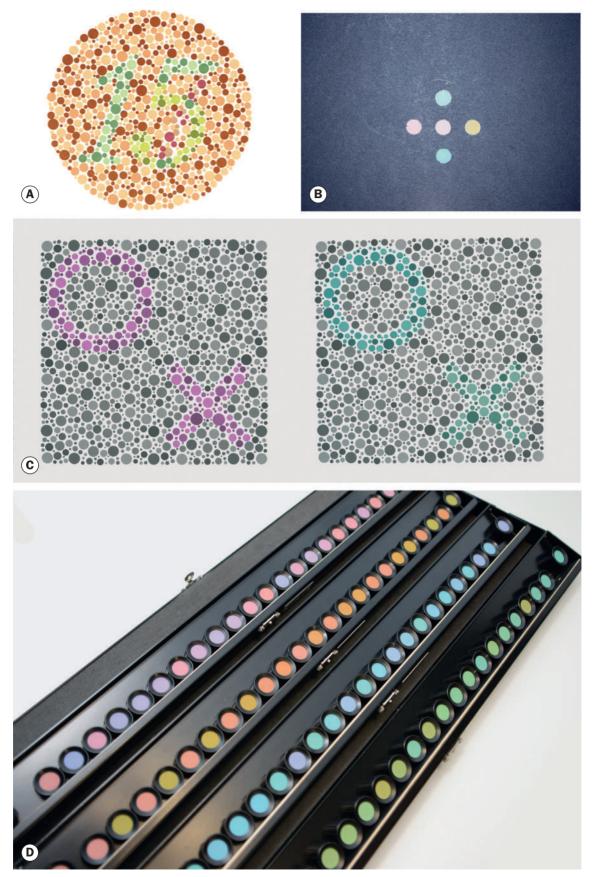


Fig. 1.13 Colour vision tests. (A) Ishihara; (B) City University; (C) Hardy–Rand–Rittler; (D) Farnsworth–Munsell 100-hue test

CHAPTER Examination Techniques

- The Hardy-Rand-Rittler test is similar to the Ishihara, but can detect all three congenital colour defects (Fig. 1.13C).
- The Farnsworth–Munsell 100-hue test is a sensitive but longer test for both congenital and acquired colour defects. Despite the name, it consists of 85 caps of different hues in four racks (Fig. 1.13D). The subject is asked to rearrange randomized caps in order of colour progression, and the findings are recorded on a circular chart. Each of the three forms of dichromatism is characterized by failure in a specific meridian of the chart (Fig. 1.14).

Plus lens test

A temporary hypermetropic shift may occur in some conditions due to an elevation of the sensory retina – the classic example is central serous chorioretinopathy (CSR). A +1.00-dioptre lens will demonstrate the phenomenon.

PERIMETRY

Definitions

- The visual field can be represented as a three-dimensional structure akin to a hill of increasing sensitivity (Fig. 1.15A). The outer aspect extends approximately 50° superiorly, 60° nasally, 70° inferiorly and 90° temporally. VA is sharpest at the very top of the hill (i.e. the fovea) and then declines progressively towards the periphery, the nasal slope being steeper than the temporal. The 'bottomless pit' of the blind spot is located temporally between 10° and 20°, slightly below the horizontal.
- An isopter is a line connecting points of the same sensitivity, and on a two-dimensional isopter plot encloses an area within which a stimulus of a given strength is visible. When the field is represented as a hill, isopters resemble the contour lines on a map (Fig. 1.15B).
- A scotoma is an area of reduced ('relative') or total ('absolute') loss of vision surrounded by a seeing area.
- Luminance is the intensity or 'brightness' of a light stimulus, measured in apostilbs (asb). A higher intensity stimulus has a higher asb value; this is related inversely to sensitivity.
- A **logarithmic** rather than a linear scale is used for stimulus intensity and sensitivity, so that for each log unit intensity changes by a factor of 10. With a log scale, greater significance is given to the lower end of the intensity range. The normal eye has a very large sensitivity range, and assessment of the lower end of the scale is of critical significance so that early damage can be detected. With a linear scale, the lower end would be reduced to a very small portion of a graphical chart axis. The visual system itself operates on close to a logarithmic scale, so using this method more closely matches the physiological situation.
- Decibels. Simple log units are not used in clinical perimetry, but rather 'decibels' (dB), where 10 dB = 1 log unit. Decibels are not true units of luminance but a representation, and vary between visual field machines. Perimetry usually concentrates on the eye's sensitivity rather than the stimulus intensity.

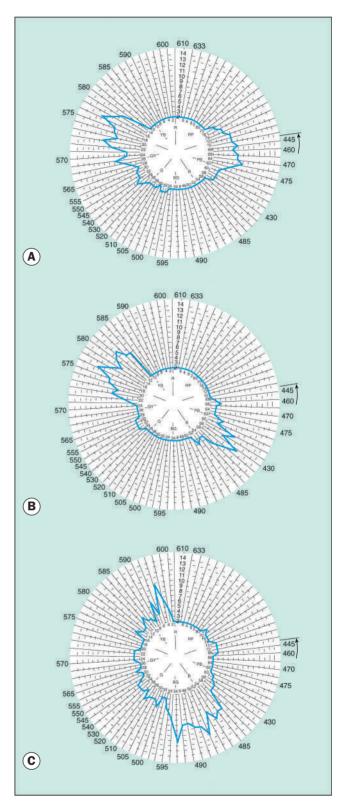


Fig. 1.14 Farnsworth–Munsell test results of colour deficiencies. (A) Protan; (B) deuteron; (C) tritan

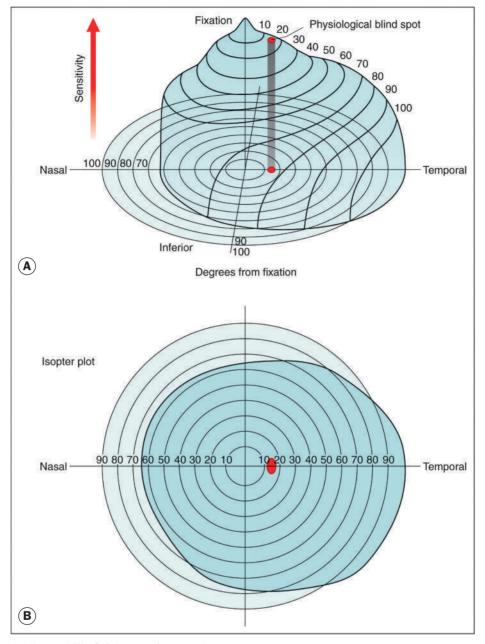


Fig. 1.15 (A) Hill of vision; (B) isopter plot

Therefore, the decibel reading goes up as retinal sensitivity increases, which obviously corresponds to reducing intensity of the perceived stimulus. This makes the assessment of visual fields more intuitive, as a higher number corresponds with higher retinal sensitivity. If the sensitivity of a test location is 20 dB (= 2 log units), a point with a sensitivity of 30 dB would be the more sensitive. The blind spot has a sensitivity of 0 dB. If, on a given machine, seeing a stimulus of 1000 asb gives a value of 10 dB, a stimulus of 100 asb will give 20 dB.

• Differential light sensitivity represents the degree by which the luminance of a target must exceed background luminance in order to be perceived. The visual field is therefore a three-dimensional representation of differential light sensitivity at different points.

- **Threshold** at a given location in the visual field is the brightness of a stimulus at which it can be detected by the subject. It is defined as 'the luminance of a given fixed-location stimulus at which it is seen on 50% of the occasions it is presented'. In practice we usually talk about an eye's *sensitivity* at a given point in the field rather than the stimulus intensity. The threshold sensitivity is highest at the fovea and decreases progressively towards the periphery. After the age of 20 years the sensitivity decreases by about 1 dB per 10 years.
- Background luminance. The retinal sensitivity at any location varies depending on background luminance. Rod

photoreceptors are more sensitive in dim light than cones, and so owing to their preponderance in the peripheral retina, at lower (scotopic) light levels the peripheral retina becomes more sensitive in proportion to the central retina. The hill of vision flattens, with a central crater rather than a peak at the fovea due to the high concentration of cones, which have low sensitivity in scotopic conditions. Some diseases give markedly different field results at different background luminance levels, e.g. in retinitis pigmentosa the field is usually much worse with low background luminance. It should be noted that it takes about 5 minutes to adapt from darkness to bright sunlight and 20–30 minutes from bright sunlight to darkness. The HFA (see below) uses a photopic (preferentially cone) level of background luminance at 31.5 asb.

- Static perimetry. A method of assessing fields, usually automated, in which the location of a stimulus remains fixed, with intensity increased until it is seen by the subject (threshold is reached Fig. 1.16A) or decreased until it is no longer detected.
- **Kinetic (dynamic) perimetry** is now much less commonly performed than static perimetry. A stimulus of constant intensity is moved from a non-seeing area to a seeing area (Fig. 1.16B) at a standardized speed until it is perceived, and the point of perception is recorded on a chart. Points from different meridians are joined to plot an isopter for that stimulus intensity. Stimuli of different intensities are used to produce a contour map of the visual field. Kinetic perimetry can be performed by means of a manual (Goldmann) or an automated

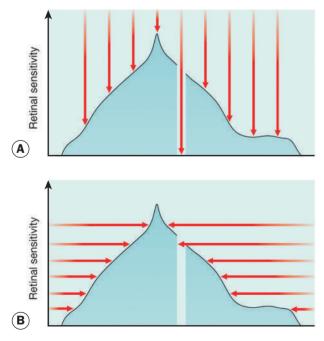


Fig. 1.16 Principles of perimetry. **(A)** Static – stimulus intensity (red arrow) at a single location is increased until perceived – areas of lower sensitivity perceive only stimuli of greater intensity (longer red arrows); **(B)** kinetic – stimulus of constant intensity is moved from a non-seeing area until perceived

perimeter if the latter is equipped with an appropriate software program.

- Manual perimetry involves presentation of a stimulus by the perimetrist, with manual recording of the response. It was formerly the standard method of field testing but has now largely been superseded by automated methods. It is still used occasionally, particularly in cognitively limited patients unable to interact adequately with an automated system, and for dynamic testing of peripheral fields.
- Standard automated perimetry (SAP) is the method used routinely in most clinical situations. Automated perimeters in common use include the Humphrey Field Analyser (HFA), the Octopus, Medmont, Henson and Dicon (Figs 1.17 and 1.18). These predominantly utilize static testing, though software is available on some machines to perform dynamic assessment.

TIP Visual field results should always be used in conjunction with the clinical findings.

Testing algorithms

Threshold

Threshold perimetry is used for detailed assessment of the hill of vision by plotting the threshold luminance value at various locations in the visual field and comparing the results with agematched 'normal' values. A typical automated strategy is to present a stimulus of higher than expected intensity. If seen, the intensity is decreased in steps (e.g. 4 dB) until it is no longer seen ('staircasing'). The stimulus is then increased again (e.g. 2 dB steps) until seen once more (Fig. 1.19). If the stimulus is not seen initially, its intensity is increased in steps until seen. Essentially, the threshold is crossed in one direction with large increments, then crossed again to 'fine-tune' the result with smaller increments. Threshold testing is commonly used for monitoring glaucoma.

Suprathreshold

Suprathreshold perimetry involves testing with stimuli of luminance above the expected normal threshold levels for an agematched population to assess whether these are detected. In other words, testing to check that a subject can see stimuli that would be seen by a normal person of the same age. It enables testing to be carried out rapidly to indicate whether function is grossly normal or not and is usually reserved for screening.

Fast algorithms

In recent years strategies have been introduced with shorter testing times, providing efficiency benefits with little or no detriment to testing accuracy. The HFA offers the SITA (Swedish Interactive Thresholding Algorithm), which uses a database of normal and glaucomatous fields to estimate threshold values and takes responses during the test into account to arrive at adjusted estimates throughout the test. Full-threshold values are obtained at the start of the test for four points. SITA-Standard and SITA-Fast (Fig. 1.20) versions are available. The Octopus Perimeter

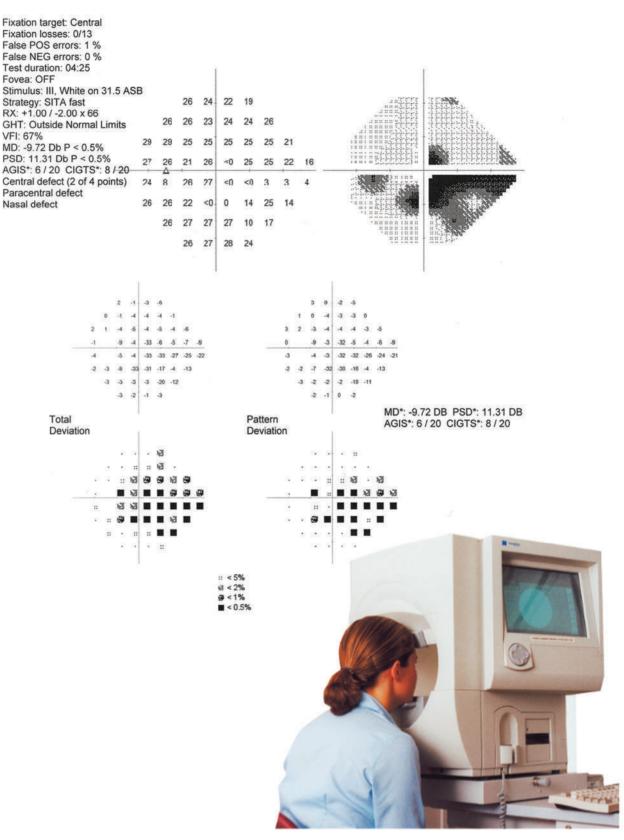


Fig. 1.17 Humphrey perimeter and printout

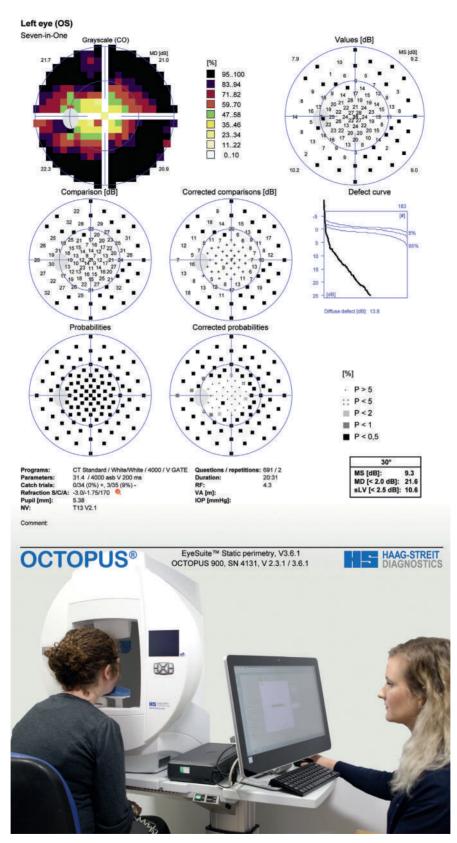


Fig. 1.18 Octopus perimeter and printout

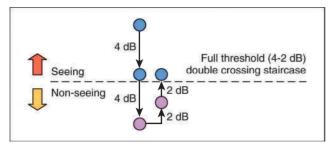


Fig. 1.19 Determination of threshold

uses G-TOP (Glaucoma Tendency Oriented Perimetry), which estimates thresholds based on information gathered from more detailed assessment of adjacent points. TOP presents each stimulus once at each location, instead of 4–6 times per location with a standard technique.

Testing patterns

Glaucoma

- Importance of central area. Most important defects in glaucoma occur centrally – within a 30° radius from the fixation point – so this is the area most commonly tested.
- 24-2 is a routinely used glaucoma-orientated pattern. '24' denotes the extent in degrees to which the field is tested on the temporal side (to 30° on the nasal side). The number after the hyphen (2) describes the pattern of the points tested. 30-2 is an alternative.
- 10-2 is used to assess a central area of radius 10°. Glaucomatous defects here may threaten central vision. The 10-2 pattern facilitates more detailed monitoring of the extent of damage, especially in advanced glaucoma where there is 'split' fixation.
- **Peripheral field.** Patterns that include central and peripheral points (e.g. FF-120) are typically limited to the assessment of neurological defects.
- **Binocular field testing** (e.g. Esterman strategy) is used to assess statutory driving entitlement in many jurisdictions.

Analysis

SAP provides the clinician with an array of clinically relevant information via monitor display or printout. The patient's name and age are confirmed and a check made that any appropriate refractive error compensation was used. General information should be reviewed, such as the type of algorithm performed, the time taken for the test and the order in which the eyes were tested.

Reliability indices

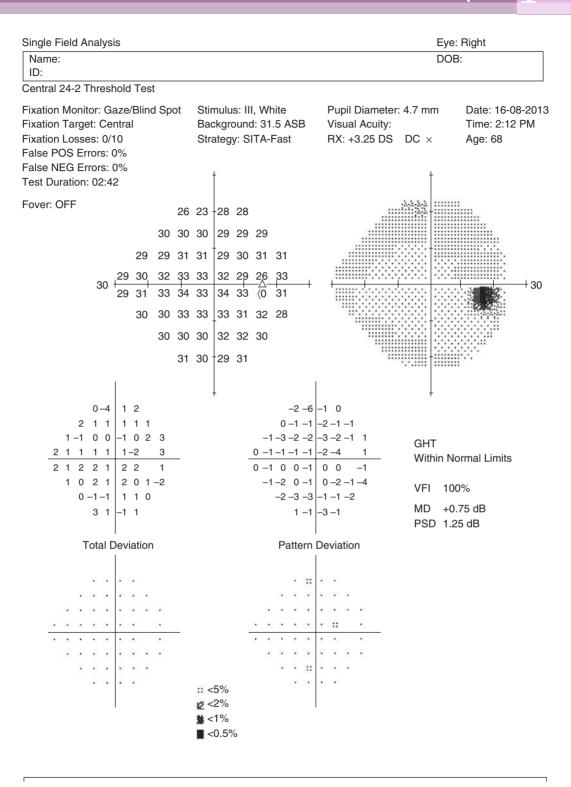
Reliability indices (see Fig. 1.20, top left corner) reflect the extent to which the patient's results are reliable. If significantly unreliable, further evaluation of the visual field printout is pointless. With SITA strategies, false negatives or false positives over about 15% should probably be regarded as significant and with full-threshold strategies, fixation losses over 20% and false positives or negatives over 33%. In patients who consistently fail to achieve good reliability it may be useful to switch to a suprathreshold strategy or kinetic perimetry.

- **Fixation losses** indicate steadiness of gaze during the test. Methods of assessment include presentation of stimuli to the blind spot to ensure no response is recorded, and the use of a 'gaze monitor'.
- False positives are usually assessed by decoupling a stimulus from its accompanying sound. If the sound alone is presented and the patient still responds, a false positive is recorded. With a high false-positive score the grey scale printout appears abnormally pale (Fig. 1.21). In SITA testing, false positives are estimated based on the response time.
- False negatives are registered by presenting a stimulus much brighter than threshold at a location where the threshold has already been determined. If the patient fails to respond, a false negative is recorded. A high false-negative score indicates inattention, tiredness or malingering, but is occasionally an indication of disease severity rather than unreliability. The grey scale printout in individuals with high false-negative responses tends to have a clover leaf shape (Fig. 1.22).

TIP If the reliability indices are poor, be careful when interpreting the results of a visual field test.

Sensitivity values

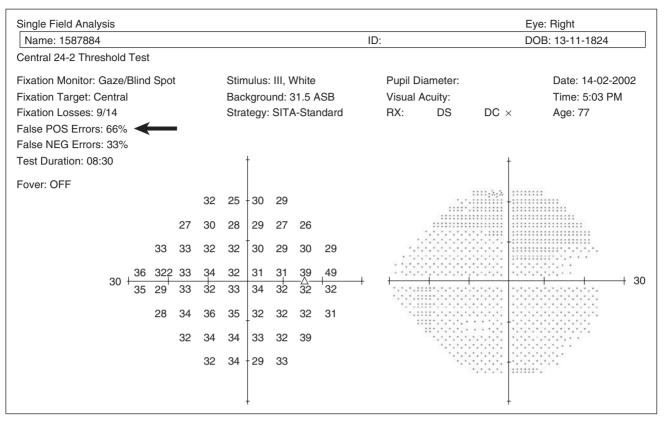
- A numerical display (see Fig. 1.20, upper left display) gives the measured or estimated (depending on strategy) threshold in dB at each point. In a full-threshold strategy, where the threshold is rechecked either as routine or because of an unexpected (>5 dB) result, the second result is shown in brackets next to the first.
- A grey scale represents the numerical display in graphical form (see Fig. 1.20, upper right display) and is the simplest display modality to interpret: decreasing sensitivity is represented by darker tones the physiological blind spot is a darker area in the temporal field typically just below the horizontal axis. Each change in grey scale tone is equivalent to a 5 dB change in sensitivity at that location.
- **Total deviation** (see Fig. 1.20, middle left display) shows the difference between a test-derived threshold at a given point and the normal sensitivity at that point for the general population, correcting for age. Negative values indicate lower than normal sensitivity, positive values higher than normal.
- **Pattern deviation** (see Fig. 1.20, middle right display) is derived from total deviation values adjusted for any generalized decrease in sensitivity in the overall field (e.g. lens opacity), and demonstrates localized defects.
- **Probability value plots** of the total and pattern deviation (see Fig. 1.20, left and right lower displays) are a representation of the percentage (<5% to <0.5%) of the normal population in whom the measured defect at each point would be expected. Darker symbols represent a greater likelihood that a defect is significant.

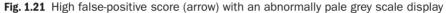


الكمرينية أسرك مقلسا فالتسمير مساطرة أألف

© 2010 Carl Zeiss Meditec HFA II 750-41202-5.1.2/5.1.2

Fig. 1.20 Humphrey perimeter – SITA-Fast printout (see text) (Copyright 2010 Carl Zeiss Meditec)





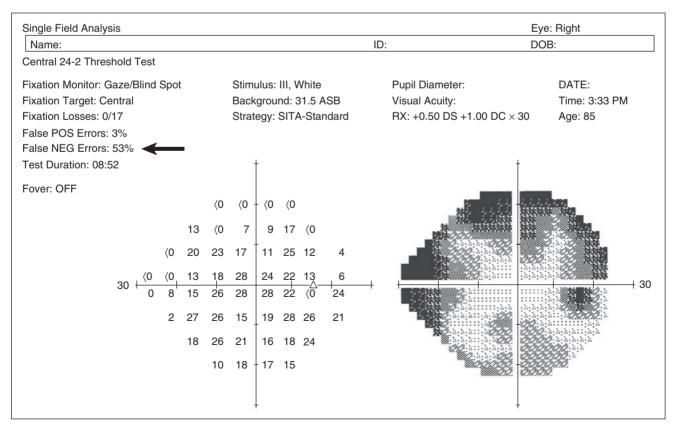


Fig. 1.22 High false-negative score (arrow) with a clover leaf-shaped grey scale display

Summary values

Summary values ('global indices' on the HFA – see Fig. 1.20, right of middle row) represent distilled statistical information, which considers age-matched normal data. These values are principally used to monitor progression of glaucomatous damage rather than for initial diagnosis.

- Visual field index (VFI) in the HFA is a measure of the patient's overall visual field function expressed as a percentage, the normal age-adjusted value being 100%.
- Mean deviation (MD) on the HFA (mean defect on the Octopus) gives an indication of the overall sensitivity of the field. It is derived from averaging the total deviation values.
- **Pattern standard deviation** (**PSD**) is a measure of focal loss or variability within the field and considers any generalized depression in the hill of vision. An increased PSD is therefore a more specific indicator of glaucomatous damage than MD.
- Loss variance (LV) is a summary measure on the Octopus Perimeter similar to PSD.
- **Probability values.** Abnormal summary values are followed by a probability value, representing the percentage likelihood that an abnormal value of this level will occur in a normal subject. The lower the *P* value, the more likely the result is abnormal.
- The glaucoma hemifield test (GHT) compares corresponding areas in the superior and inferior hemifields and relates only to glaucoma.

Computer analysis of serial fields

Computed analysis of serial visual fields for progression is in widespread use. A disadvantage is the requirement for several reliable fields to be carried out before analysis is effective. The quality of available software has been improving steadily, with integrated programs such as GPA (Guided Progression Analysis) on the HFA and several trend analysis options on the Octopus.

High-sensitivity field modalities

SAP tends to detect field damage only after substantial ganglion cell loss is established. Attempts at detecting change at an earlier stage include the adoption of stimuli intended to target specific ganglion cell types.

- Short-wave automated perimetry (SWAP) uses a blue stimulus on a yellow background. Sensitivity to blue light (mediated by blue cone photoreceptors) is adversely affected relatively early in glaucoma. SWAP is more sensitive to early glaucomatous defects but has not been widely adopted because cataract decreases sensitivity to blue light (the brunescent lens acts as a yellow filter) and patients frequently dislike the lengthy test. It is available on newer HFA models.
- Frequency-doubling test (FDT). Large diameter axon (magnocellular) ganglion cells appear to be preferentially lost in early glaucoma. The frequency-doubling illusion is produced when a low spatial frequency sinusoidal grating undergoes high temporal frequency counter phase flicker (>15 Hz). The

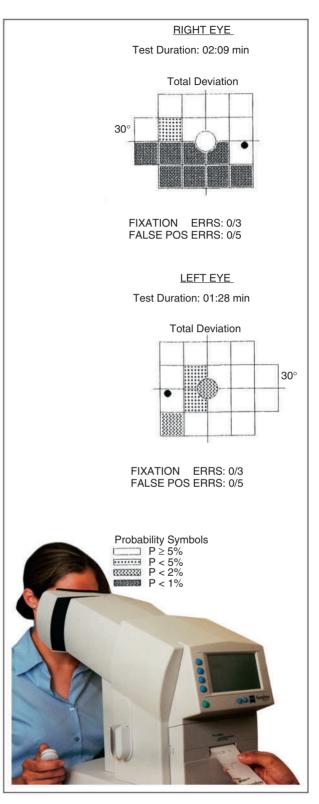


Fig. 1.23 Screening frequency-doubling perimeter with display

rapid alternation in which the light bars become dark and vice versa produces the illusion of the grating having doubled its frequency; magnocellular ganglion cells are believed to mediate the pathways used. Screening (Fig. 1.23) and extended testing (Humphrey Matrix) perimeter versions are available,

the latter being suitable for detailed assessment and monitoring of glaucoma.

Sources of error

- **Inexperienced or unskilled technician.** Though less important with SAP than manual perimetry, correctly setting up the test, explaining the procedure to and reassuring the patient and monitoring performance are fundamental to obtaining an accurate field.
- **Incorrect patient details.** The patient's date of birth must be entered correctly to facilitate appropriate normative database matching.
- Poor patient performance.
- Uncorrected refractive error can cause a significant decrease in central sensitivity. If a patient with hypermetropia who usually wears contact lenses is tested wearing spectacles, this will have the effect of magnifying and enlarging any scotomas as compared with contact lenses. Most perimetry is performed with a stimulus at approximately reading distance, so a near correction should be used for individuals who are presbyopic.
- Spectacle rim artefact. Spectacles can cause rim scotomas if small aperture lenses are used or if incorrectly dispensed (Fig. 1.24). Narrow-aperture trial frame lenses are unsuitable for perimetry.
- Miosis decreases sensitivity in the peripheral field and increases variability in the central field in both normal and glaucomatous eyes. Pupils less than 3 mm in diameter should therefore be dilated prior to perimetry; a consistent mydriatic should be used for serial tests.
- Media opacities (usually cataract) can have a profound effect, exaggerated by miosis.
- **Ptosis**, even if mild, can suppress the superior visual field. Similar effects result from dermatochalasis, prominent eyelashes and deeply set eyes.
- Inadequate retinal adaptation may lead to error if perimetry is performed soon after ophthalmoscopy.

TIP Perimetry is a subjective examination and is not always reliable.

Microperimetry

 Microperimetry is a visual field test that measures retinal sensitivity and fixation behaviour in patients with macular disease and focal glaucoma involving the central visual field.

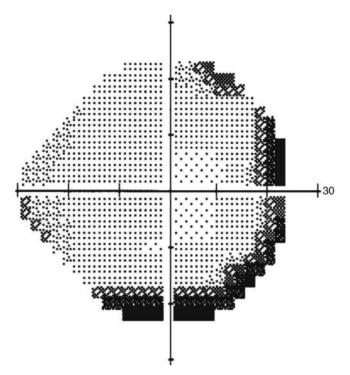


Fig. 1.24 Grey scale display of spectacle rim artefact

It allows an exact correlation between pathology involving the macula and the corresponding functional abnormality. Microperimetry is more sensitive than SAP in detecting subtle abnormality of visual function.

- The MAIA perimeter is a table top instrument (Fig. 1.25). The test is usually undertaken after 20 minutes of adaptation in mesopic conditions. Fundus tracking occurs utilizing a line-scanning laser ophthalmoscope (SLO), with superluminescent diode illumination using a central wavelength of 850 nm.
- Goldmann size 3 stimuli are projected onto the central 9° of the fundus. A 4-2 staircase thresholding technique is used, with each stimulus applied for 200 milliseconds against a white background. Using this instrument normal retinal sensitivity is 18 dB.
- Results are printed, together with reliability indices (fixation loss), probabilities, and retinal sensitivity values, which are colour-coded. Microperimetry is particularly useful in assessing the effect of existing and future therapeutic interventions on the macula. It can also be helpful in patients with early glaucoma, particularly when there is subtle change close to fixation (Fig. 1.26).

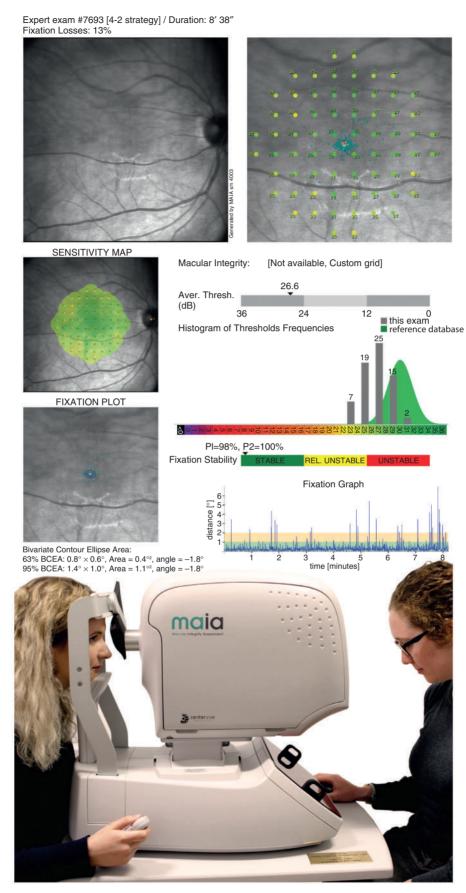


Fig. 1.25 MAIA perimetry and printout

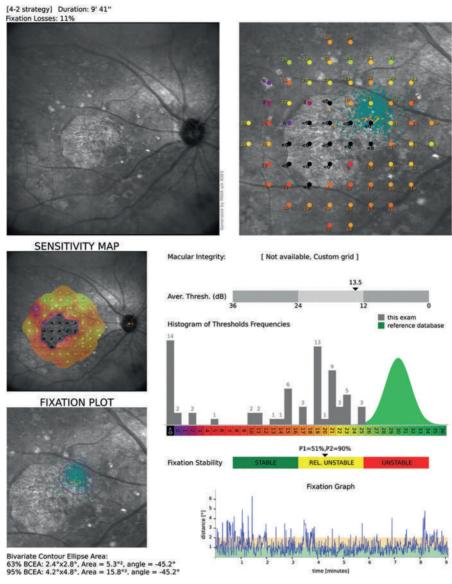


Fig. 1.26 Abnormal microperimetry in a patient with geographic atrophy of the macula

SLIT LAMP BIOMICROSCOPY OF THE ANTERIOR SEGMENT

The purpose of slit lamp examination of the cornea and anterior segment is to determine the position, depth and size of any abnormality (Fig. 1.27).

Direct illumination

Direct illumination with a diffuse light is used to detect gross abnormalities:

- A narrow obliquely directed slit beam is used to visualize a cross-section of the cornea.
- Further narrowing of the beam to a very thin optical section moved across the cornea can determine the depth of a lesion.
- The height of the coaxial beam can be adjusted to measure the horizontal and vertical size of a lesion or associated epithelial defect.

• The use of a red-free filter makes red objects appear black, thereby increasing contrast when observing vascular structures. A cobalt blue filter is normally used in conjunction with fluorescein.

Scleral scatter

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 reflection. A corneal stromal lesion will become illuminated because of forward light scatter. This technique is especially useful to detect subtle stromal haze, or cellular or lipid infiltration.

Retroillumination

Retroillumination uses reflected light from the iris or fundus after pupil dilation to illuminate the cornea. This allows the detection

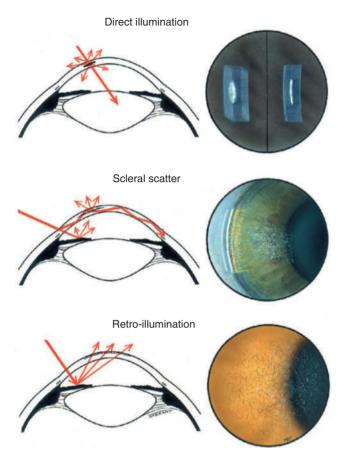


Fig. 1.27 Technique of slit lamp biomicroscopy of the anterior segment

of fine epithelial and endothelial changes, such as epithelial cysts, keratic precipitates and small blood vessels.

Specular reflection

Specular reflection shows abnormalities of the endothelium such as reduced cell density and guttata. Pseudoguttata probably represent reversible endothelial cell oedema and inflammatory cells beneath the endothelial layer.

FUNDUS EXAMINATION

Direct ophthalmoscopy

- 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, systematic hypertension, raised intracranial pressure). The major advantage of direct ophthalmoscopy is that it can be used at the bedside. The image obtained is significantly magnified (15 times normal) (Fig. 1.28).
- The direct ophthalmoscope can be used to examine the 'red' reflex using retroillumination. By placing a +15.00 lens in position and looking at the fundus at a distance

of 15-20 cm, lens and vitreous opacities can be detected (Fig. 1.29).

• The light beam can be used to (a) illuminate the cornea, allowing detection of a foreign body, (b) check the pupil for irregularity, and (c) determine the light reflexes. Some oph-thalmoscopes incorporate a cobalt blue filter, which allows corneal abrasions and ulcers to be seen after the insertion of fluorescein.

TIP A direct ophthalmoscope can be used at the bedside and provides a magnified view of the fundus, but there is no stereopsis and the field of view is small.

Technique

- It is preferable to dilate the pupil if there is no contraindication. However, a good view can be obtained through an undilated pupil by darkening the examination room and by asking the patient to look into the distance.
- When examining the right eye, the clinician should stand at the patient's right side, holding the ophthalmoscope in the right hand. When examining the left eye, the clinician should stand at the left side, holding the ophthalmoscope in the left hand and using the left eye (Fig. 1.30).

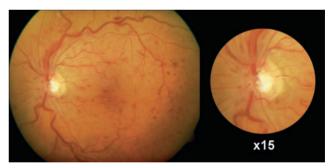


Fig. 1.28 Magnified image obtained from direct ophthalmoscope



Fig. 1.29 Red reflex showing anterior capsular thickening



Fig. 1.30 Technique of direct ophthalmoscopy (see text)

- The 'O' on the illuminated lens dial of the ophthalmoscope should be selected.
- To overcome corneal reflection, the small aperture can be used and the light beam directed towards the edge of the pupil, rather than through the centre. The linear polarizing filter may be helpful.
- The examiner's free hand should rest on the patient's forehead.
- The patient is slowly approached at a slight angle, from the temporal side.
- The light beam is directed at the pupil and the optic disc should come into view at a distance of about 3.5 cm from the eye. If the disc is not in sharp focus, the ophthalmoscope lenses should be rotated into the aperture using the index finger until the disc becomes clearly visible.
- In hyperopic individuals use a plus lens (green) and in myopic individuals a minus lens (red).
- Examine the disc for clarity of outline, colour, elevation and condition of vessels. Check for spontaneous venous pulsation. Follow the vessels as far as possible into the retinal periphery.
- To locate the macula, focus the light on the disc then move the light about two-disc diameters temporally. Alternatively, ask the patient to look at the light.
- The retinal periphery can be examined by asking the patient to look up/down and to the sides.

Slit lamp biomicroscopy

A range of diagnostic contact and non-contact lenses are available for use with the slit lamp. Contact lenses should not be used if a penetrating injury is suspected or in the presence of corneal trauma, hyphaema or corneal infection.

- Non-contact lenses
 - 60 D. High-magnification lens optimized for viewing the posterior pole from a working distance of 13 mm. When estimating optic disc diameter use a correction factor of ×0.88–1.0 for the Volk lens and ×1.0 for the Nikon lens.



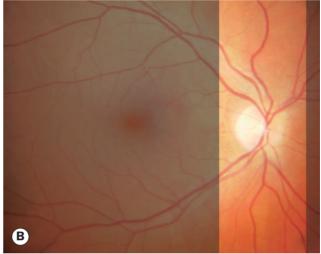


Fig. 1.31 (A) Indirect slit lamp biomicroscopy; (B) fundus view

- 90 D. Wide-field lens with lower magnification and shorter (7 mm) working distance. Can be used with smaller pupils. The correction factor is ×1.3 (Fig. 1.31 A and B).
- 78 D. Intermediate properties; ideal for general-purpose examination. The correction factor is ×1.1.
- Miscellaneous. Numerous other lenses are available, offering qualities such as a very wide field of view and extremely small pupil capability.

Technique

Indirect ophthalmoscopy utilizes a high-power convex lens which is designed to obtain a wide field of view of the fundus. The image is vertically inverted and laterally reversed. The technique is as follows:

- The slit beam is adjusted to a width of about 1/4 of its full round diameter.
- The illumination is set at an angle coaxial with the slit lamp viewing system.
- The magnification and light intensity are adjusted to the lowest settings.