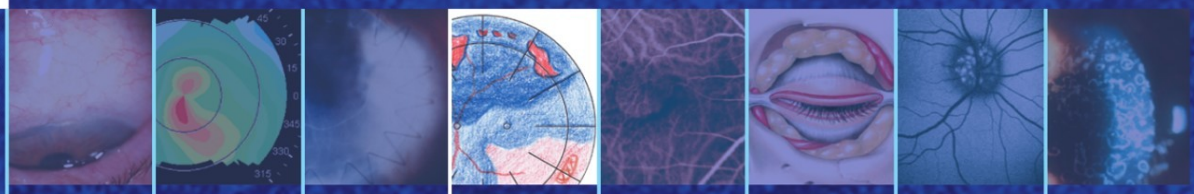
ESSENTIALS IN OPHTHALMOLOGY

G. K. KRIEGLSTEIN · R. N. WEINREB
Series Editors



Glaucoma | Cataract and Refractive Surgery | Uveitis and Immunological Disorders | **Vitreo-retinal Surgery** | Medical Retina | Oculoplastics and Orbit | Paediatric Ophthalmology, Neuro-ophthalmology, Genetics | Cornea and External Eye Disease

Vitreo-retinal Surgery

Edited by
B. KIRCHHOF
D. WONG

Springer

Essentials in Ophthalmology

Vitreo-retinal Surgery

B. Kirchhof D. Wong
Editors

Essentials in Ophthalmology

G. K. Krieglstein R. N. Weinreb
Series Editors

Glaucoma

Cataract and Refractive Surgery

Uveitis and Immunological Disorders

Vitreo-retinal Surgery

Medical Retina

Oculoplastics and Orbit

Pediatric Ophthalmology, Neuro-Ophthalmology, Genetics

Cornea and External Eye Disease

Editors Bernd Kirchhof
David Wong

Vitreo-retinal Surgery

With 122 Figures, Mostly in Colour
and 13 Tables

Springer

Series Editors

Günter K. Krieglstein, MD
Professor and Chairman
Department of Ophthalmology
University of Cologne
Kerpener Straße 62
50924 Cologne
Germany

Robert N. Weinreb, MD
Professor and Director
Hamilton Glaucoma Center
Department of Ophthalmology
University of California at San Diego
9500 Gilman Drive
La Jolla, CA 92093-0946
USA

Volume Editors

Bernd Kirchhof, MD
Professor and Chairman
Department of Vitreo-retinal Surgery
Center for Ophthalmology
University of Cologne
Kerpener Straße 62
50924 Cologne
Germany

David Wong, MD
Professor and Chairman
Department of Ophthalmology
Faculty of Medicine
University of Hong Kong
21 Sassoon Road, Pok Fu Lam
Hong Kong

ISBN-10 3-540-33669-9
Springer Berlin Heidelberg NewYork

ISBN-13 978-3-540-33669-3
Springer Berlin Heidelberg NewYork

ISSN 1612-3212

Library of Congress Control Number: 2006935425

Editor: Marion Philipp, Heidelberg, Germany
Desk Editor: Martina Himberger, Heidelberg, Germany
Production: LE-TeX Jelonek, Schmidt & Vöckler GbR, Leipzig, Germany
Cover Design: Erich Kirchner, Heidelberg, Germany

Printed on acid-free paper
24/3100Wa 5 4 3 2 1 0

Foreword

The series *Essentials in Ophthalmology* was initiated two years ago to expedite the timely transfer of new information in vision science and evidence-based medicine into clinical practice. We thought that this prospicient idea would be moved and guided by a resolute commitment to excellence. It is reasonable to now update our readers with what has been achieved.

The immediate goal was to transfer information through a high quality quarterly publication in which ophthalmology would be represented by eight subspecialties. In this regard, each issue has had a subspecialty theme and has been overseen by two internationally recognized volume editors, who in turn have invited a bevy of experts to discuss clinically relevant and appropriate topics. Summaries of clinically relevant information have been provided throughout each chapter.

Each subspecialty area now has been covered once, and the response to the first eight volumes in the series has been enthusiastically positive. With the start of the second cycle of subspecialty coverage, the dissemination of practical information will be continued as we learn more about the emerging advances in various ophthalmic subspecialties that can be applied to obtain the best possible care of our patients. Moreover, we will continue to highlight clinically relevant information and maintain our commitment to excellence.

G. K. Krieglstein
R. N. Weinreb
Series Editors

Preface

This second edition promises to challenge our ideas about vitreoretinal surgery. As expected, some new and innovative techniques are described, the most exciting of which is the use of free retinal pigment epithelium patch grafts in patients with atrophic macular degeneration. We are interested in the advances that have made this surgery possible and look forward to the long-term outcomes. In the last couple of years, surgeons have become increasingly convinced by the advantages conferred by smaller gauge instruments, brighter and even safer lights, combined with an innovative viewing system to make routine surgery quicker and less traumatic for the patients. The use of 25-gauge for tumor biopsies is an exciting new development that represents a huge advantage over needle biopsies. The indication and the rationale for biopsies in cases of suspected uveal melanoma are presented.

There will always of course be difficult clinical decisions; choices have to be made despite the absence of good data based on randomized trials. A good example of a dilemma is whether to perform early vitrectomy for postoperative endophthalmitis. For this edition, we have opposing views from two surgeons. You simply have to read and decide for yourself. There are other controversies such as vitrectomy for floaters: the preparation in terms of counseling, patient selection, and pitfalls are well covered in a chapter on the subject.

The chapter on macular holes examines the evidence behind our current practice. Despite the high success rate, a minority of patients will fail to respond to conventional surgery and a novel approach with heavy silicone oil is presented.

Sometimes, the skill of a surgeon is to know when not to operate. Asymptomatic inferior retinal detachment is another controversial area explored in a reasoned fashion, though no one knows for sure the cause of optical coherence tomography findings of subclinical macular retinal detachments.

Then we come to the big topic of tamponading. The need to apply scleral buckling, to adopt postoperative head-down posturing, or to use long-acting gas for inferior retinal breaks are being challenged as more rhegmatogenous retinal detachments are treated using primary vitrectomy and air alone. There is renewed interest in the techniques for draining subretinal fluid and the chapter on slippage explains how unwanted retinal folds can be avoided, whilst offering an easy technique for the exchange of fluids and the injection of silicone oil.

We hope you will enjoy reading this book as much as we have preparing it.

Bernd Kirchhof
David Wong
Editors

Contents

Chapter 1
Macular Holes
James Bainbridge, Zdenek Gregor

1.1 Epidemiology 1
1.2 Pathogenesis 1
1.3 Clinical Staging 2
1.4 Natural History 3
1.5 Risk of Full-Thickness Macular Hole in the Fellow Eye 4
1.6 Symptoms and Signs 4
1.7 Fluorescein Angiography and Fundus Autofluorescence 5
1.8 Optical Coherence Tomography 6
1.9 Surgical Management of Macular Holes 6
1.10 Technique for Conventional Macular Hole Surgery 7
1.11 Tamponade Agents and Postoperative Posturing .. 8
1.12 Biological Adjuncts to Macular Hole Surgery 9
1.13 Technique for Intraoperative Application of Biological Adjuncts 10
1.14 Inner Limiting Membrane Peeling 10
1.15 Technique for Inner Limiting Membrane Peeling 11
1.16 Vital Staining to Facilitate Inner Limiting Membrane Peeling 11
1.17 Indocyanine Green 11
1.18 Trypan Blue 12
1.19 Technique for Inner Limiting Membrane Staining 13
1.20 Complications of Macular Hole Surgery 13
1.21 Prognosis Following Macular Hole Surgery 13

Chapter 2
Heavy Silicone Oil for Persistent Macular Holes
Stanislao Rizzo, Federica Genovesi-Ebert

2.1. Introduction 19
2.1.1 Basics 19
2.1.2 Therapeutic Options 19
2.1.2.1 Observation 20
2.1.2.2 Laser Photocoagulation and Outpatient FGEX 20
2.1.2.3 Re-Operations 20
2.1.3 Rationale of HSO Endotamponading 22
2.2 Treatment of Persistent Macular Holes 22
2.2.1 Heavier Than Water Silicone Oils 22
2.2.2 Surgical Technique 24
2.2.3 HSO Removal 25
2.3 Conclusions 25

Chapter 3
The Role of Combined Adjunctive 5-Fluorouracil and Low Molecular Weight Heparin in Proliferative Vitreoretinopathy Prevention
David G. Charteris, D. Wong

3.1 Introduction 33
3.2 PVR Pathobiology 34
3.2.1 PVR Development 34
3.2.2 PVR Components 34
3.3 Adjunctive Agents 34
3.3.1 5-Fluorouracil 34
3.3.2 Low Molecular Weight Heparin 35
3.3.3 Initial Clinical Studies 35
3.4 Clinical Trials of Combined Adjunctive 5FU and LMWH .. 35
3.4.1 Adjunctive Regime 35

3.4.2 High-Risk Retinal Detachments in Eyes Undergoing Vitrectomy and Gas Exchange (PVR 1) .. 36
3.4.3 Established PVR in Eyes Undergoing Vitrectomy and Silicone Oil Exchange (PVR 2) 36
3.4.4 Unselected Primary Retinal Detachments in Eyes Undergoing Vitrectomy and Gas Exchange (PVR 3) ... 36
3.4.5 Macular Translocation with 360° Retinotomy 37
3.5 Implications for Clinical Use .. 37
3.5.1 Uncomplicated Primary Retinal Detachments 37
3.5.2 Established PVR 37
3.5.3 High-Risk Retinal Detachments 37
3.5.4 Intraocular Trauma Patients Undergoing Vitrectomy Surgery 37

Chapter 4
Slippage of the Retina: What Causes It and How Can It Be Prevented?
David Wong

4.1 Introduction 41
4.2 What is Slippage? 41
4.2.1 Surface Tension and Interfacial Tension 41
4.2.2 Shape of Endotamponade Bubble 42
4.2.3 Surface Property of the Retina 43
4.2.4 Fluid–Air Exchange 43
4.3 Setting the Scene for Slippage 44
4.3.1 Perfluorocarbon Liquids and the "Doughnut" of Fluid 44
4.3.2 Seeing and Doing 46
4.3.3 Air Versus Silicone Oil Exchange for PFCL 46
4.3.4 Oil on Water? No, Water on Oil! 47
4.4 Overfilling and Complete Elimination of Aqueous 48
4.5 Injection of Silicone Oil 48

Chapter 5
Complete and Early Vitrectomy for Endophthalmitis (CEVE) as Today's Alternative to the Endophthalmitis Vitrectomy Study
Ferenc Kuhn, Giampaolo Gini

5.1 Introduction and Definitions . 54
5.1.1 Introduction 54
5.1.2 Definitions 54
5.2 Etiology and Classification .. 56
5.3 Pathophysiology, Organisms, and Diagnostics in Brief 56
5.3.1 Pathophysiology 56
5.3.2 Organisms 56
5.3.3 Diagnostics 56
5.3.3.1 Clinical 56
5.3.3.2 Other 57
5.4 Principles of Therapy 57
5.5 The EVS 57
5.5.1 Study Design 57
5.5.2 Results 58
5.5.3 Consequences of the EVS Recommendations 58
5.6 Rationale for Performing Complete and Early Vitrectomy for Endophthalmitis 58
5.6.1 Why Perform Vitrectomy? ... 58
5.6.2 Why Perform Early Vitrectomy? 58
5.6.3 Why Perform Complete Vitrectomy? 59
5.7 Complete and Early Vitrectomy for Endophthalmitis: Surgical Steps 59
5.7.1 Initial Steps 59
5.7.2 Cornea 60
5.7.3 Anterior Chamber 60
5.7.4 Pupil 61
5.7.5 (Intraocular) Lens 61
5.7.6 Posterior Lens Capsule 61
5.7.7 Vitrectomy 61
5.7.8 When to Stop Vitreous Removal? 63
5.7.9 Retina 63
5.7.10 Enucleation/Evisceration 64
5.7.11 Pharmacological Treatment .. 64

5.8 Surgical Decision-Making and Complications 64
5.8.1 Decision-Making 64
5.8.2 Complications and Their Management 64
5.8.2.1 Retinal Break 64
5.8.2.2 Retinal Detachment 65
5.8.2.3 Silicone Oil as a Long-Term Tamponade 65
5.9 Results with CEVE and Their Comparison with the EVS ... 66
5.10 Summary and Recommendations 67

Chapter 6
Treatment of Acute Bacterial Endophthalmitis After Cataract Surgery Without Vitrectomy
Thomas Theelen, Maurits A.D. Tilanus

6.1 Introduction 70
6.1.1 Basics 70
6.1.2 Pathophysiology 70
6.1.2.1 Phases of Infection 70
6.1.3 Clinical Diagnosis 71
6.1.3.1 Role of Ultrasonography 72
6.1.4 Microbial Spectrum 72
6.2 Therapeutical Approaches .. 75
6.2.1 Basics 75
6.2.2 Early Pars Plana Vitrectomy .. 75
6.2.3 Nonvitrectomizing Endophthalmitis Treatment .. 76
6.3 Emergency Management of Endophthalmitis After Cataract Surgery 76
6.3.1 Surgical Technique 76
6.3.2 Equipment for the Emergency Management of Endophthalmitis 77
6.3.3 Treatment Protocol 77
6.3.4 Treatment Outcome in Endophthalmitis Without Immediate Vitrectomy 77
6.3.4.1 Introduction 77
6.3.4.2 Results in Our Department .. 79
6.3.4.3 Discussion 80
6.4 Conclusion 81

Chapter 7
New Instruments in Vitrectomy
Masahito Ohji, Yasuo Tano

7.1 Introduction 85
7.2 Sutureless Transconjunctival Vitrectomy 85
7.2.1 25-Gauge Vitrectomy System 85
7.2.2 Instruments 86
7.2.2.1 Trocars 86
7.2.2.2 Vitreous Cutter 86
7.2.2.3 Light Pipe 87
7.2.2.4 Other 25-Gauge Instruments 87
7.2.2.5 Other Instruments 87
7.2.3 Surgical Procedures of the 25-Gauge System 89
7.2.3.1 Insert Three Trocars 89
7.2.3.2 Vitrectomy 89
7.2.3.3 Closure of the Wound 89
7.2.4 Advantage and Disadvantage of 25-Gauge System 89
7.3 23-Gauge Vitrectomy System 90
7.4 Xenon Endo-Illumination for Vitrectomy 90
7.4.1 Yellow-Filtered Xenon 92
7.4.2 Wide-Angle Viewing System 92
7.4.3 Adjuncts 95
7.4.3.1 Staining of Internal Limiting Membrane with Indocyanine Green 95
7.4.3.2 Other Dyes Staining the ILM . 95

Chapter 8
25-Gauge Biopsy of Uveal Tumors
Bertil Damato, Carl Groenewald

8.1 Introduction 99
8.1.1 Uveal Tumors 99
8.2 Diagnosis of Choroidal Tumors 99
8.2.1 Ophthalmoscopy 99
8.2.2 Angiography 101
8.2.3 Echography 101
8.2.4 Other Investigations 101
8.3 Current Biopsy Techniques .. 102

8.3.1 Fine Needle Aspiration Biopsy 102
8.3.2 Trans-Scleral Tumor Biopsy . 102
8.3.3 20-Gauge Vitreous Cutter .. 102
8.3.4 Trans-Vitreal Incisional Biopsy 102
8.3.5 25-Gauge Biopsy Technique 104
8.4 Preoperative Management . 104
8.5 Surgical Technique 104
8.6 Care of Specimen 104
8.7 Postoperative Management 104
8.8 Results and Complications . 105
8.8.1 Adequacy of Sample 105
8.8.2 Sampling Error 105
8.8.3 Tumor Seeding 105
8.8.4 Hemorrhage 105
8.8.5 Rhegmatogenous Retinal Detachment 109
8.8.6 Other Complications 109
8.9 Indications and Contraindications 109
8.9.1 Indications 109
8.9.1.1 Diagnosis of Metastasis 109
8.9.1.2 Differentiation Between Metastasis and Melanoma .. 109
8.9.1.3 Differentiation Between Nevus and Melanoma 109
8.9.1.4 Confirmation of Local Recurrence of Melanoma after Conservative Treatment 110
8.9.1.5 Confirmation of Intraocular Lymphoma 110
8.9.1.6 Prognosis 110
8.9.2 Contraindications 113
8.10 Conclusions 113

Chapter 9
Vitrectomy Against Floaters
Hans Hoerauf

9.1 Introduction 115
9.2 Clinical Findings 116
9.3 Symptoms and Natural Course 116
9.4 Diagnostic Methods 117
9.5 Surgical Treatment for Vitreous Floaters: Important Studies 117
9.6 Vitrectomy for Vitreous Floaters Despite Full Visual Acuity 117
9.6.1 Patients 118
9.6.2 Surgical Technique 118
9.6.3 Results 119
9.6.4 Case Report 119
9.7 Analysis of Clinical Studies .. 119
9.8 Alternative Therapeutic Options 120
9.9 Personality Traits 121
9.10 Alternative Assessment of Visual Function 121
9.11 Conclusions 123

Chapter 10
Treatment of Retinal Detachment from Inferior Breaks with Pars Plana Vitrectomy
Vicente Martinez-Castillo, Jose Garcia-Arumi, Anna Boixadera, Miguel A. Zapata

10.1 Introduction 125
10.1.1 Basics 125
10.1.2 Importance of Inferior Breaks When Pars Plana Vitrectomy is Performed ... 126
10.1.3 Development of a Chorioretinal Scar 126
10.1.4 Type of Tamponade Agent for Inferior Breaks 127
10.2 Management of Inferior Breaks with PPV: Recent Publications 127
10.3 PPV Alone with Air Tamponade: Surgical Technique 129
10.3.1 Inclusion Criteria 129
10.3.2 Vitrectomy 129
10.3.3 Drainage of Subretinal Fluid 129
10.3.4 Retinopexy 130
10.3.5 Tamponade Agent 130

Chapter 11
Subclinical Retinal Detachment

Jose Garcia-Arumi, Anna Boixadera, Vicente Martinez-Castillo, Miguel A. Zapata

11.1 Definition *133*
11.2 Natural History *135*
11.2.1 Role of the Vitreous *135*
11.3 Therapeutic Options *136*
11.3.1 Observation *136*
11.3.2 Laser Demarcation *137*
11.3.3 Surgery *137*
11.3.3.1 Pneumatic Retinopexy *137*
11.3.3.2 Scleral Buckling *138*
11.3.3.3 Pars Plana Vitrectomy *138*
11.3.4 Pros and Cons of Treating .. *138*
11.3.4.1 Pros of Treating *138*
11.3.4.2 Cons of Treating *138*
11.4 Current Clinical Practice/ Recommendations *139*
11.5 Subclinical Retinal Detachment Diagnosed with Optical Coherence Tomography After Successful Surgery for Rhegmatogenous Retinal Detachment *139*

Chapter 12
Autologous Translocation of the Choroid and RPE in Patients with Geographic Atrophy

Antonia M. Joussen, Jan van Meurs, Bernd Kirchhof

12.1 Introduction: Pathology and Epidemiology *143*
12.2 Treatment Approaches in Patients with Geographic Atrophy *144*
12.3 Concept of Translocation of a Free Choroidal: RPE Graft (Free Graft Translocation) *144*
12.3.1 Patients and Methods *144*
12.3.2 Free Graft Translocation Surgery in Dry AMD *145*
12.3.3 Results *145*
12.3.3.1 Free Graft Translocation Surgery and Complication Profile *145*
12.3.3.2 Anatomical Outcome: Funduscopic Appearance, Vascularization, and Autofluorescence of the Graft *146*
12.3.3.3 Functional Outcome: Distant Visual Acuity, Reading Ability, Fixation, and Microperimetry During the Postoperative Course .. *147*
12.4 Comment *148*

Contributors

James Bainbridge
Moorfields Eye Hospital
City Road
London EC1V 2PD
UK

Anna Boixadera
Hospital Vall d´ Hebron
Servicio de Oftalmologia
Paseo Vall d´ Hebron, 119 – 121
Barcelona
Spain

David G. Charteris
Moorfields Eye Hospital
City Road
London EC1V 2PD
UK

Bertil Damato
Royal Liverpool University Hospital
Prescot Street
Liverpool L7 8XP
UK

Jose Garcia-Arumi
Instituto de Microcirugía Ocular
C/ Munner nº 10
08022 Barcelona
Spain

Giampaolo Gini
City Hospital
Viale Vittorio Veneto 60
Prato 59100
Italy

Zdenek Gregor
Moorfields Eye Hospital
City Road
London EC1V 2PD
UK

Carl Groenewald
Royal Liverpool University Hospital
Prescot Street
Liverpool L7 8XP
UK

Hans Hoerauf
University Eye Clinic, UK S-H
Campus Lübeck
Lübeck
Germany

Antonia M. Joussen
Department of Ophthalmology
University of Duesseldorf
Moorenstraße 5
40225 Duesseldorf
Germany

Bernd Kirchhof
Department of Vitreo-retinal Surgery
Center for Ophthalmology
University of Cologne
Kerpener Straße 62
50924 Cologne
Germany

Ferenc Kuhn
University of Alabama at Birmingham
1201 11th Avenue South, Suite 300
Birmingham
AL 35205
USA

Vicente Martínez-Castillo
Hospital Vall d´ Hebron
Servicio de Oftalmologia
Paseo Vall d´ Hebron, 119 – 121
Barcelona
Spain

Masahito Ohji
Shiga University of Medical Science
Osaka University Medical School, E-7
Department of Ophthalmology
2-2 Yamidaoka Suita
Osaka 565-0871
Japan

Stanislao Rizzo
Chirurgica Oftalmica Ospedale S. Chiara
Via Roma 55
56100 Pisa
Italy

Yasuo Tano
Shiga University of Medical Science
Osaka University Medical School, E-7
Department of Ophthalmology
2-2 Yamidaoka Suita
Osaka 565-0871
Japan

Thomas Theelen
Academish Ziekenhuis Nijmegen
Philips van Leydenlaan 15
6525 EX Nijmegen
The Netherlands

Maurits A.D. Tilanus
Academish Ziekenhuis Nijmegen
Philips van Leydenlaan 15
6525 EX Nijmegen
The Netherlands

Jan van Meurs
Rotterdam Eye Hospital
Postbus 70030
3000 LM Rotterdam
The Netherlands

David Wong
Department of Ophthalmology
Faculty of Medicine
University of Hong Kong
21 Sassoon Road, Pok Fu Lam
Hong Kong

Miguel A. Zapata
C/ Sta Teresa 70 2º 1ª
08172 Sant Cugat
Spain

Chapter 1

Macular Holes

James Bainbridge, Zdenek Gregor

1

Core Messages

- Idiopathic full-thickness macular holes are believed to result from tangential and antero-posterior traction exerted by the posterior vitreous cortex on the neurosensory retina at the fovea.
- Only a minority of full-thickness macular holes resolve spontaneously and there is a significant risk of macular hole development in the fellow eye.
- Surgical management by removal of the posterior cortical vitreous and long-acting intraocular gas tamponade results in a high rate of anatomical closure and a benefit to visual function.
- Modifications to this surgical technique, proposed with the aim of improving anatomical and visual outcomes, include the use of adjuvants to stimulate glial repair and the dissection of the inner limiting membrane, with or without the aid of vital stains. The value of these modifications is not well-established.
- The importance of prolonged face-down posturing to the rate of macular hole closure is unproven.

1.1 Epidemiology

A macular hole is a full-thickness defect of the neurosensory retina involving the anatomical fovea. Idiopathic full-thickness macular holes are an important cause of central visual loss; these lesions are twice as common in females as in males [1, 2] and three-quarters of affected individuals are over the age of 65 years [1]. The population prevalence of macular holes is estimated as being 0.1% in individuals aged 40 years or older and 0.8% in those aged over 74 years. No significant systemic risk factor for the development of macular holes has been identified although a possible association with the menopause has been described [1].

1.2 Pathogenesis

The pathogenesis of idiopathic full-thickness macular holes is not well understood. Macular holes are commonly idiopathic in etiology, although trauma and high myopia are implicated in a minority of cases. On the basis of clinical observations, ocular imaging, histological studies, and the results of vitrectomy surgery, the mechanism underlying idiopathic macular hole is widely believed to involve tangential as well as antero-posterior traction exerted by the posterior vitreous cortex on the neurosensory retina at the fovea. Evidence of the role of posterior vitreous attachment in the development of macular holes includes its association with an increased risk of macular hole development in the fellow eyes of individuals with unilateral macular holes and with the subsequent enlargement of established holes [3, 4]. There is no consensus on the exact mechanism of vitreo-foveal traction. Tangential traction may be the result of contraction of the prefoveal vitreous cortex following invasion and proliferation of Müller cells [5]. Anteroposterior traction may occur from dynamic tractional forces on an abnormally persistent vitreo-foveal attachment following perifoveal vitreous separation [6, 7]. The role of antero-posterior vitreo-foveal traction is supported by optical coherence tomography (OCT) studies that clearly identify perifoveal posterior hyaloid separation with persistent adherence of the posterior hyaloid to the centre of the fovea [6–8]. A cone-shaped zone of Müller cells, the 'Müller cell cone' forms the

1

central and inner part of the fovea centralis and appears to confer structural support, serving as a plug to bind together the foveolar photoreceptor cells [9]. Vitreo-foveal traction may result in disinsertion of the Müller cell cone from underlying foveolar photoreceptor cells and in the formation of a foveal schisis or "cyst" [6–8]. A dehiscence develops in the roof of the foveal cyst that may extend by centric expansion, or more commonly in a pericentric fashion, to form a crescentic hole that progresses to a horseshoe tear [6]. Complete avulsion of the cyst roof results in a fully detached operculum that is suspended on the posterior vitreous cortex in the prefoveal plane [6, 7, 10]. Opercula primarily comprise vitreous cortex and glial elements with a variable amount of foveal tissue that includes photoreceptor cells in 40% of cases [11]. The photoreceptor layer, which is no longer anchored by the Muller cell cone at the foveola, undergoes passive centrifugal retraction to form a full-thickness retinal dehiscence with centrifugal displacement of xanthophyll [9, 12]. The edge of the hole becomes progressively elevated and a cuff of subretinal fluid develops. In the event of vitreo-foveal separation during the development of the macular hole, the relief of traction may result in regression of a cyst, but spontaneous closure of an established full-thickness macular hole is relatively uncommon [13].

While the majority of age-related macular holes are idiopathic in etiology, full-thickness macular holes may also occur in association with high myopia, following posterior segment surgery such as scleral buckling and pneumatic retinopexy, and following ocular trauma. Traumatic macular holes typically result from blunt injury, but have also been reported following laser injury and lightning strike.

Summary for the Clinician

- Idiopathic full-thickness macular holes are caused by traction exerted by the posterior vitreous cortex on the neurosensory retina at the fovea. Disinsertion of its glial plug results in the formation of a foveal "cyst." Avulsion of the cyst roof results in a free operculum suspended on the posterior vitreous cortex. The neurosensory retina retracts centrifugally to form a full-thickness retinal dehiscence.

1.3 Clinical Staging

Macular hole formation typically evolves through a series of four major stages that were first described according to their biomicroscopic features by Gass (Fig. 1.1, Table 1.1) [5, 14]. Stage 1 ('impending') macular hole is loss of the normal

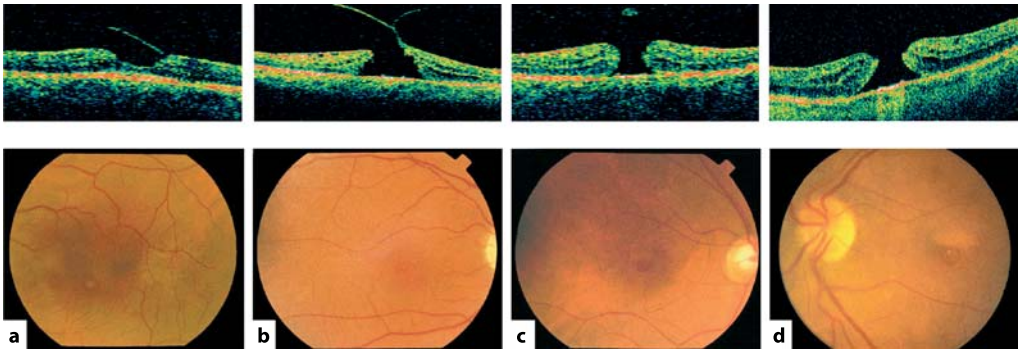


Fig. 1.1 Fundus photographs and OCT images of stage 1–4 macular holes. **a** Stage 1b lesion with a partial-thickness foveal defect. **b** Stage 2 full-thickness hole associated with persistent vitreo-foveal attachment. **c** Stage 3 full-thickness hole with separation of the vitreous from the macula and a fully detached operculum on the posterior hyaloid face. **d** Stage 4 full-thickness hole with complete posterior vitreous separation from the macula and optic disc

Table 1.1 Clinical features and natural history of idiopathic macular holes

	Stage 1	Stage 2	Stage 3	Stage 4
Symptoms	Asymptomatic, or mild metamorphopsia	Metamorphopsia and loss of central vision	Metamorphopsia and loss of central vision	Metamorphopsia and loss of central vision
Visual acuity	20/20–20/60	20/40–20/100	20/60–20/200	20/60–20/400
Biomicroscopy	Loss of foveal depression	Full-thickness retinal defect	Full-thickness retinal defect	Full-thickness retinal defect
	Yellow spot (1a) or yellow ring (1b)	Typically ≤200 μm in diameter	Typically 250–400 μm in diameter	Typically ≥450 μm in diameter
			Operculum may be evident	Operculum may be evident
	Posterior vitreous attached to fovea and optic disc	Posterior vitreous attached to fovea and optic disc	Posterior vitreous typically detached from fovea, but attached to optic disc	Posterior vitreous detached from both fovea and optic disc
Natural history	50% regress 40% progress 10% stabilize	15–21% regress 75% progress	5% regress 30% progress	20% enlarge

foveal depression, associated with the development of a yellow spot (stage 1a) or ring (stage 1b) in the centre of the fovea, changes that reflect the intraretinal schisis that progresses into an intraretinal cyst. A foveal dehiscence may be masked on biomicroscopy (stage 1-b, occult hole) by semi-opaque contracted prefoveolar vitreous cortex bridging the yellow ring. Stage 2 is a small full-thickness hole (≤200 μm), typically with a pericentric configuration, associated with persistent vitreo-foveal attachment. Stage 3 is a larger full-thickness hole (250–400 μm) with a rim of elevated retina and separation of the posterior hyaloid from the macula. A fully detached operculum on the posterior hyaloid may be evident on biomicroscopy. Stage 4 is a full-thickness hole (≥450 μm) with complete posterior vitreous separation from the optic disc, typically demonstrated by the presence of a Weiss ring.

1.4 Natural History

The natural history of macular holes is typically to progress in size and clinical stage, with deterioration in visual acuity that generally stabilizes at the 6/60 to 3/60 level, redistribution of yellow nodular opacities at the level of the retinal pigment epithelium, and the development of retinal pigment epithelial atrophy surrounding the macular hole, resulting in a 'bull's-eye' macular appearance.

An estimated 40% of stage 1 (impending) macular holes progress to full-thickness holes [15], but up to 50% resolve spontaneously [15, 16]. Stage 2 macular holes typically enlarge, with progression to stage 3 in at least 75% of cases [10, 16, 17] and spontaneous closure has been reported in no more than 15–21% [10, 13, 16]. Stage 3 holes progress to stage 4 in approximately 30% of cases over 3 years [16]. Full-thickness holes tend to enlarge modestly; by 25% during the first 12 months and by 29% at 24 months,