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Preface

Originally written on assignment from the American Academy of Ophthalmology, this monograph is now updated under ownership of Oxford University Press. This edition constitutes a substantial reorganization with large amounts of new material. It is a thorough revision with emphasis on conciseness.

Rather than document each point with specific references to the literature, a list of Selected References is included at the end of each chapter for those who would like to pursue further research.

The work is based on a review of the literature and on our personal experience. We have attempted to acknowledge controversy where it exists, and to present alternative points of view. The monograph is sufficiently brief to encourage cover-to-cover reading, but it has a detailed table of contents and an index to facilitate locating specific information. The book is not an all-inclusive treatise; rather, it is intended as an introduction to retinal detachment for the ophthalmology resident or as a concise review and reference manual for the practicing ophthalmologist.

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PART I Principles

1 History of Surgery for Retinal Detachment  3
  Foundations of Retinal Detachment Surgery  3
  Discovery of Retinal Detachment  3
  Establishing the Etiology of Detachment  3
  Gonin’s Detachment Repair  4
  Development of Modern Surgical Procedures  5
  Scleral Buckling  5
  Cryopexy and Laser  5
  Pneumatic Retinopexy  6
  Vitrectomy for Retinal Detachment  6
  Retinal Detachment Surgery Today  6

2 Pathogenesis, Epidemiology, and Natural Course of Retinal Detachment  9
  Types of Retinal Detachment  9
  Rhegmatogenous Retinal Detachments  9
  Exudative Retinal Detachments  10
  Tractional Retinal Detachments  10
  Role of the Vitreous in Pathogenesis of Rhegmatogenous Retinal Detachment  10
  Vitreous Liquefaction  10
  Myopia  11
Contents

Trauma 11
Inflammation 12
Vitreous Detachment 12
Vitreoretinal Traction 14
Liquid Currents 15
Retinal Breaks 15
Primary and Secondary Breaks 16
Types of Breaks 16
Horseshoe tears 16
Operculated breaks 18
Atrophic holes 18
Dialyses 19
Postnecrotic holes 20
Retinal breaks due to proliferative diabetic retinopathy 21
Macular holes 22
Distribution of Breaks 22
Lesions Associated with Retinal Breaks 23
Congenital anomalies 23
Peripheral cystoid degeneration 25
Degenerative retinoschisis 25
Lattice degeneration 27
Epidemiology of Retinal Detachment 30
Variables Regarding Epidemiology 31
Age 31
Sex 31
Race 31
Heredity 31
Bilaterality 31
Systemic and Genetic Conditions Associated with Retinal Detachment 32
Classification of Retinal Detachments 32
Pathology of the Detached Retina 33
Proliferative Vitreoretinopathy 33
Intraretinal Macrocysts 35
Demarcation Lines 36
Natural History of Untreated Detachment 36
Summary 37

3 Ophthalmoscopy 41
Characteristics of Indirect and Direct Ophthalmoscopy 41
Differences in Visualization 41
Magnification and resolution 41
Field of view 43
Illumination 44
Stereopsis and depth of focus 45
Practical Advantages of the Indirect Ophthalmoscope 45
  Opacities in the ocular media 45
  Children and uncooperative adults 45
  Working distance 46
  Scleral depression 46
Role of Direct Ophthalmoscopy 46
The Indirect Ophthalmoscopic Image 46

Basic Indirect Ophthalmoscopy Techniques 46
Instrumentation 47
  Choice of indirect ophthalmoscope 47
  Adjusting the indirect ophthalmoscope 47
  Choice of condensing lenses 48
Preparation of the Patient 49
  Pupillary dilation 49
  Dilation in infants 50
  Medications to be avoided 50
  Position of the patient 50
  Keeping the other eye open and controlling eye movements 50
First Steps in Observing the Fundus 51
  Holding the condensing lens 51
  Bringing the fundus into view 52
  Troublesome reflexes 53
  Shifting from one part of the fundus to the next 54
Orientation and Drawing 55
  Drawing the inverted image 57
  Position of observer and lesion being examined 58
Fundus charts 58

Examination Through a Small Pupil 59
  Circumstances presenting a narrow optical aperture 59
  Indirect viewing systems 61
  Using the indirect ophthalmoscope with a small pupil 62
  Using the indirect laser with a small pupil 62

Scleral Depression 62
  Purposes of Scleral Depression 63
  Technique of Scleral Depression 63
    Choice of depressor 63
    Initiating scleral depression 64
    Moving the axis of depression 66
    Causes of discomfort 66
    Scleral depression in the horizontal meridia 67
    Scleral depression nasally 68
    Examining the far periphery 68
    Rolling the depressor 68
  Sequence of scleral depression examination 70

Scleral Depression in the Operating Room 72
Summary 73
4 Evaluation and Management  75

Ocular Evaluation  75

History Relevant to Retinal Detachment  75
  Flashes of light  75
  Floaters  76
  Visual field defects  77
  Decreased central acuity  78
  Trauma  78
  Ocular diseases  78
  Systemic diseases  78
  Family history  78

General Ocular Examination  79
  Visual acuity  79
  External examination  79
  Ocular motility  79
  Pupillary reactions  79
  Anterior segment biomicroscopy  80
  Tonometry  80

Retinal Examination  80
  Binocular Indirect Ophthalmoscopy  80
  Posterior Segment Biomicroscopy  81

Ancillary Tests  83
  Perimetry  83
  Ultrasonography  83
  Laser test  84

Preparation for Surgery  85
  Patient Counseling  85
  Urgency of Surgery and Macular Detachment  86

Complicating Preexisting Conditions  88
  Managing miosis  88
  Corneal opacities  88
  Cataract  88
  Vitreous opacities  89
  External disease  89
  Glaucoma  89
  Uveitis  89
  Choroidal detachment  90

Health Management in Preparation for Surgery  90
  Preparation for operating room procedures  90
  Preparation for office procedures  91

Postoperative Management  91
  Activity Restrictions and Positioning  91
  Postoperative Medications  92
  Follow-up Examinations  92
  If the Retina Fails to Settle  92
    Postoperative photocoagulation or cryopexy  92
    Reoperation  94
5 Establishing the Diagnosis  97

Fundus Changes Unrelated to Retinal Detachment  97
Choroidal vasculature  97
Ora serrata  98
Chorioretinal degeneration  100
Reticular pigmentation degeneration  100
Equatorial drusen  100
Cobblestone degeneration  100
Retinal whitening  103
Pars plana cysts  104
Retinal erosion  104
Pigment clumps  105
Hemiretinal differences  106

Fundus Changes Related to Retinal Detachment  106
Findings Suggestive of Retinal Detachment  106
Detection of Retinal Breaks  106
   Distinguishing retinal breaks from retinal hemorrhage  108
Proliferative Vitreoretinopathy  109

Nonrhegmatogenous Retinal Detachment  116
Distinguishing Serous from Rhegmatogenous Detachment  116
Causes of Serous Retinal Detachments  119
   Choroidal tumors with serous retinal detachment  119
   Inflammatory serous detachment  119
   Vascular lesions with serous detachment  121
   Congenital causes of serous detachment  121
   Macular lesions with serous detachment  121
Distinguishing Tractional from Rhegmatogenous Detachment  121
Causes of Tractional Retinal Detachment  122

Lesions Simulating Retinal Detachment  122
   Retinoschisis  122
   Intraretinal macrocysts  123
   Choroidal detachment  123
   Choroidal tumors  125
   Vitreous opacities  125
   White-with-pressure and white-without-pressure  125

Summary  126

PART II Practice

6 Prevention of Retinal Detachment  129
Risk Factors for Retinal Detachment  130
Symptomatic Eyes  131
   Tears with Persistent Vitreoretinal Traction  131
   Symptomatic horseshoe-shaped tears  131
Symptomatic round tears 131
Breaks Unassociated with Persistent Vitreoretinal Traction 132
Symptomatic operculated retinal tears 132
Symptomatic atrophic retinal holes and precursors of retinal breaks 132

Asymptomatic Eyes 133
Asymptomatic Non-fellow Eyes at High Risk 133
Asymptomatic myopic non-fellow eyes 133
Asymptomatic nonphakic non-fellow eyes 133
Family history of retinal detachment 134
Stickler’s syndrome 134
Asymptomatic Precursors of Retinal Breaks without High-Risk Features 134
Asymptomatic lattice degeneration 135
Asymptomatic cystic retinal tufts 136
Asymptomatic degenerative retinoschisis 136
Asymptomatic Retinal Breaks 136

Patients with Retinal Detachment in the Fellow Eye 137
Asymptomatic Phakic Fellow Eyes 137
Asymptomatic phakic fellow eyes with lattice degeneration 137
Asymptomatic phakic fellow eyes with cystic retinal tufts 139
Asymptomatic phakic fellow eyes with degenerative retinoschisis 139
Asymptomatic phakic fellow eyes with retinal breaks 140
Asymptomatic phakic eyes with a history of giant retinal tear in the fellow eye 140
Asymptomatic Aphakic and Pseudophakic Fellow Eyes 140
Asymptomatic non-phakic fellow eyes with lattice degeneration 141
Asymptomatic non-phakic fellow eyes with retinal breaks 141
Asymptomatic non-phakic eyes with a history of giant retinal tear in the fellow eye 142

Treatment to Prevent Retinal Detachment 142
Cryotherapy 142
Laser Photocoagulation 143
Results of Treatment 144
Flap tears 145
Lattice degeneration 145
Retinal holes 145
Patients with previous retinal detachment in the fellow eye 145
Complications of Treatment 145

Summary 146

7 Scleral Buckling 149
Anatomical and Physiological Effects of Scleral Buckles 150
Pathophysiology of Rhegmatogenous Retinal Detachment 150
Reattachment Forces Influenced by Scleral Buckles 150
Principles of Scleral Buckling 151
Scleral Buckle Configuration 151
The Scleral Buckling Operation 153
Prep and Drape 153
Conjunctival Incision and Isolation of Rectus Muscles 154
Localization of Breaks 155
Thermal Treatment 156
Cryotherapy 156
Diathermy 158
Intraoperative laser photocoagulation 158
Buckling Materials 159
Segmental episcleral buckles 159
Encircling episcleral buckles 160
Thin Sclera 163
Intrascleral Buckles 163
Management of Subretinal Fluid 164
Non-drainage technique 165
Assuring perfusion of the central retinal artery 165
Drainage technique 166
Adjustment of Scleral Buckle 168
Accessory Techniques 169
Intravitreal injection of balanced salt solution 170
Intravitreal gas injection 170
Gas–fluid exchange 171
Postoperative laser photocoagulation 171
Closure of Incisions 171
Common Complications of Scleral Buckling 172
Selected Intraoperative Complications 172
Corneal complications 172
Pupillary complications 172
Scleral perforation with suture needles 172
Complications of draining subretinal fluid 173
“Fish-Mouthing” of retinal breaks 174
Complications of intravitreal gas injections 174
Selected Postoperative Complications 174
Increased intraocular pressure 174
Endophthalmitis 175
Choroidal detachment 175
Later periocular infection and implant extrusion 176
Cystoid macular edema 177
Epimacular proliferation 177
Proliferative vitreoretinopathy 178
Recurrent retinal detachment 179
Altered refractive error 179
Strabismus 179
Summary 180

8 Pneumatic Retinopexy 181
Intraocular Gases 181
Choice of Gases 181
What size gas bubble is needed? 183
How long should the bubble remain in the eye? 183
Why Gas Works 184

**Preoperative Evaluation** 185

**Indications and Contraindications** 186

Limits to Indications 186

- Extent of breaks 186
- Inferior breaks 187
- Proliferative vitreoretinopathy 187
- Cloudy media 187
- Inability to maintain positioning 187
- Glaucoma 187
- Lattice degeneration 188
- Aphakia/pseudophakia 188

Relative Indications for Pneumatic Retinopexy 188

- Retinal detachment imminently threatening the fovea 188
- Macular holes and other posterior retinal breaks 188
- Redetachment following scleral buckling 188
- Filtering blebs 188
- Isolated tears under the superior rectus 189
- Contraindications to general anesthesia 189
- Optic pit with macular detachment 189
- Extensively scarred conjunctiva 189
- Very thin sclera 189
- Need to retain emmetropia or to prevent anisometropia 189
- Cosmetic concerns 189
- Operating room not available 189
- Limited financial resources 189

**Operative Technique** 190

Anesthesia 190

Retinopexy 190

- Cryopexy versus laser 190

Sterilizing the Eye 191

Preparing the Gas 191

Making Room for the Gas 191

- Paracentesis 192
- Ocular massage 192

Injecting the Gas 193

After Gas Injection 195

- Is the central retinal artery occluded? 195
- Is a single gas bubble present or are there multiple small bubbles (“fish eggs”)? 195
- Is the bubble mobile within the vitreous or is it trapped at the injection site? 195

**Special Procedures** 196

- Steamroller 196
- Fish Eggs 197

Gas Entrapment at the Injection Site 198

**Summary of Procedure** 199

**Postoperative Management** 199
9 Vitrectomy for Retinal Detachment 205

Vitrectomy Techniques for Routine Retinal Detachments 206

External Steps of the Procedure 206
- Prep and drape 206
- Opening incisions 206

Internal Steps of the Procedure 207
- Removal of vitreous gel 207
- Installation of heavy perfluorocarbon liquids 208
- Identification and marking of retinal breaks 208
- Internal drainage of subretinal fluid 210
- Air infusion 210
- Laser treatment of retinal breaks 211
- Placement of scleral buckle 211
- Closure of sclerotomies and peritomies 212

Vitrectomy Techniques for Complicated Cases 212

Retinal Detachment and Proliferative Diabetic Retinopathy 212
- Vitrectomy for PDR and retinal detachment 214
- Removal of fibrovascular PDR tissue 215
- Treating retinal breaks in PDR 217

Retinal Detachment and Proliferative Vitreoretinopathy 217
- Scleral buckle for PVR 219
- Removal of vitreous 219
- Removal of epiretinal PVR membranes 219
- Retinotomy and retinectomy in PVR 221
- Marking retinal breaks 222
- Internal drainage of fluid and vitreous replacement 222

Giant Retinal Tear 223

Results of Vitrectomy 226

Complications of Vitrectomy 226

Summary 227

10 Selection of Surgery for “Routine” Retinal Detachment 229

Surgery for Common Types of Retinal Detachment 230

Scleral Buckling 231
- Advantages of scleral buckling 232
- Disadvantages of scleral buckling 232

Pneumatic Retinopexy 232
PART I

Principles
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The evolution of the retinal reattachment operation is one of the most remarkable chapters in the history of ophthalmology. Gonin’s operation for repair of the detached retina ranks with Daviel’s cataract extraction, von Graefe’s peripheral iridectomy, and Machemer’s vitrectomy as one of history’s most important surgical treatments for blinding eye diseases.

FOUNDATIONS OF RETINAL DETACHMENT SURGERY

DISCOVERY OF RETINAL DETACHMENT
The entity of retinal detachment was recognized early in the eighteenth century by de Saint-Yves, who reported the gross pathologic examination of an eye with a detached retina. The first clinical description did not appear until almost a century later, in 1817, when Beer detected a retinal detachment without the benefit of an ophthalmoscope. Von Helmholtz’s invention of the direct ophthalmoscope in 1851 was a giant step forward in diagnostic technique, and a rapid succession of ophthalmoscopic observations of retinal detachments soon followed. In the same year, Coccius reported the ophthalmoscopic detection of breaks in the detached retina.

ESTABLISHING THE ETIOLOGY OF DETACHMENT
Von Graefe theorized in 1858 that retinal detachment was caused by a serous effusion from the choroid into the subretinal space. When he observed a retinal break, he assumed that it was secondary to the detachment and represented the eye’s attempt to cure itself. Supposing that the development of a break would
allow the subretinal fluid to pass from the subretinal space into the vitreous cavity, he attempted unsuccessfully to treat detachments with deliberate incision of the retina.

Girard-Teulon invented the reflecting binocular indirect ophthalmoscope in 1861. This potentially important contribution was generally overlooked by the profession, and more than 80 years transpired before Schepens developed the self-illuminating binocular indirect ophthalmoscope.

In 1869 Iwanoff described the entity of posterior vitreous detachment, which is now recognized as a prerequisite to the development of most retinal detachments. The following year de Wecker suggested that retinal breaks cause detachment due to the resultant passage of vitreous fluid through the break into the subretinal space. Unfortunately, his accurate interpretation was not generally accepted. In 1882 Leber reported his observation of retinal breaks in 14 of 27 retinal detachments, and he correctly inferred the role of vitreous traction in the pathogenesis of breaks. Unfortunately, he later altered this opinion. In 1889 Deutschmann treated a detachment by closing the retinal break with ignipuncture. However, the value of this procedure was not appreciated at the time, and it was discarded for several decades.

**Gonin's Detachment Repair**

Jules Gonin (1870–1935; Figure 1–1) investigated the works of Leber and became convinced of the causal role of retinal breaks in the pathogenesis of retinal detachment. Resurrecting Deutschmann’s surgical approach, Gonin treated retinal breaks

with a red-hot searing probe, which was plunged into the vitreous cavity. The probe, manufactured for burning designs in wood, was obtained at a toy store. He first reported his results in 1923 and subsequently reported the surgical cure of 20 of 30 cases of retinal detachment. With the publication of 34 papers in various journals over the ensuing years, and with the 1934 publication of his classic text, *Retinal Detachment*, Gonin established his new treatment as the standard of care, and he has rightfully become accepted as the father of retinal detachment surgery.

Gonin’s procedure was markedly improved in the early 1930s when Weve and Larsson independently developed the use of diathermy in place of the red-hot penetrating technique. This provided a means of treating a wider area around retinal breaks and avoided the need for ultra-precise localization of breaks required with the Gonin technique. In 1933 Lindner treated retinal detachment by shortening the axial length with a full-thickness scleral resection, an adaptation of the scleral resection originally developed by Müller.

**DEVELOPMENT OF MODERN SURGICAL PROCEDURES**

**Scleral Buckling**

Scleral buckling was first described in 1937 by Jess, but this brief mention in the literature was overlooked until Custodis developed the procedure 12 years later. In 1949 Shapland supplanted Lindner’s full-thickness resection with a lamellar scleral resection. Custodis’ segmental scleral buckle was extended by the development of an encircling scleral buckle by Schepens in the early 1950s. His encircling polyethylene tube was later replaced with silicone rubber to minimize the complication of scleral erosion. In the early 1960s Lincoff introduced the silicone sponge for use with segmental buckles described by Custodis, and in 1979 he described retinal reattachment with a temporary external balloon buckle.

In 1945 Schepens invented the modern binocular indirect ophthalmoscope, augmented by the use of scleral depression originally introduced by Trantas; this equipment and technique have represented the standard of care for scleral buckling since the early 1950s.

**Cryopexy and Laser**

Although Bietti reported the use of cryosurgery for retinal detachment in 1933, it was Lincoff who developed and popularized this valuable method in 1964. This freezing technique minimizes damage to conjunctiva, muscle, and sclera, rendering the dissection of scleral flaps unnecessary.

In 1956 Meyer-Schwickerath introduced xenon arc photocoagulation to achieve a chorioretinal adhesion. Since an adhesive burn can only be obtained when an attached retina is treated with photocoagulation, this therapy was originally employed for conditions other than retinal detachment. However, the introduction of lasers and vitrectomy techniques with intraoperative reattachment of the retina and also pneumatic retinopexy (with laser applied after the retina is reattached)
has resulted in the laser being commonly employed in contemporary reattachment surgery.

**Pneumatic Retinopexy**

First performed by Ohm in 1911, the use of intravitreal air injection for retinal detachment was developed by Rosengren in 1938. Years later, Chawla, Fineberg, Vygantas, Norton, and Lincoff brought intravitreal gas into common usage, combined with or following scleral buckling or vitrectomy.

Pneumatic retinopexy is a gas injection procedure for retinal detachment, performed in the office without scleral buckling, vitrectomy, drainage of subretinal fluid, or conjunctival incision. In 1983 G. Brinton presented the first cases, and in 1985 Hilton and Grizzard published the first report using the procedure they named, “pneumatic retinopexy.” This procedure (and a similar one described concurrently by Dominguez) modified Kreissig’s and Lincoff’s technique for treating retinal tears in the posterior pole. Tornambe, Hilton, and others brought pneumatic retinopexy into the mainstream of retinal surgery.

**Vitrectomy for Retinal Detachment**

A surgical approach to the vitreous, an important adjunct in selected cases of retinal detachment, was pioneered by Shafer in 1950 with his method of vitreous transplantation. This important branch of surgery was expanded by Cibis in 1962 with his introduction of intravitreal silicone oil injection. The development of the vitreous infusion suction cutter (VISC) by Machemer in 1971 and the use of intravitreal sulfur hexafluoride gas by Norton in 1973 further expanded the role of vitreous surgery in the management of retinal detachment. In recent years, improvements in vitreotomy instrumentation, the development of wide-angle microscopic viewing systems, the use of perfluorocarbon liquids, and the development of microincisional techniques have resulted in vitrectomy becoming a routine contemporary procedure for the treatment of primary, as well as complex, retinal detachment.

**Retinal Detachment Surgery Today**

Three different surgical techniques are widely employed to reattach the retina: scleral buckling, pneumatic retinopexy, and vitrectomy; these are discussed in Chapters 7, 8, and 9, respectively. Although they are frequently single procedures, a combination of techniques may be required in selected cases. A discussion of preoperative factors that may favor the selection of a particular technique is presented in Chapter 10. Prevention of retinal detachment by treating selected lesions with photocoagulation or cryopexy is discussed in Chapter 6.

**Selected References**


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Retinal detachment does not result from a single, specific disease; rather, numerous disease processes can result in the presence of subretinal fluid.

**TYPES OF RETINAL DETACHMENT**

The three general categories of retinal detachments are termed *rhegmatogenous*, *exudative*, and *tractional*. Rhegmatogenous detachments are sometimes referred to as *primary* detachments, while both exudative and tractional detachments are called *secondary* or *nonrhegmatogenous* detachments.

The three types of retinal detachments are not mutually exclusive. For example, detachments associated with proliferative vitreoretinopathy or proliferative diabetic retinopathy may exhibit both rhegmatogenous and tractional features. However, excluding the section on differential diagnosis in Chapter 5, the scope of this book is limited to rhegmatogenous retinal detachments. Accordingly, throughout the book, the term *retinal detachment* refers to the rhegmatogenous type, unless another type is specifically mentioned.

**RHEGMATOGENOUS RETINAL DETACHMENTS**

Rhegmatogenous detachments (from the Greek *rhegma*, meaning rent, rupture, or fissure) are the most common form of retinal detachment. They are caused by a break in the retina through which fluid passes from the vitreous cavity into the subretinal space. The responsible break(s) can be identified preoperatively in more than 90% of cases, but occasionally the presence of a minute, unseen break must be assumed.
Exudative Retinal Detachments

Exudative detachments, also called serous detachments, are due to an associated problem that produces subretinal fluid without a retinal break. This underlying problem usually involves the choroid as a tumor or an inflammatory disorder.

Tractional Retinal Detachments

Tractional detachments occur when pathologic vitreoretinal adhesions or membranes mechanically pull the retina away from the pigment epithelium without a retinal break. The most common causes include proliferative diabetic retinopathy, cicatricial retinopathy of prematurity, proliferative sickle retinopathy, and penetrating trauma. Retinal breaks may subsequently develop, resulting in a combined tractional and rhegmatogenous detachment.

Role of the Vitreous in Pathogenesis of Rhegmatogenous Retinal Detachment

The essential requirements for a rhegmatogenous retinal detachment include a retinal break and low-viscosity vitreous liquids capable of passing through the break into the subretinal space. Vitreous changes usually precede development of important defects in the retina. The usual pathologic sequence causing retinal detachment is vitreous liquefaction followed by a posterior vitreous detachment (PVD) that causes traction at the site of significant vitreoretinal adhesion with a subsequent retinal tear. Fluids from the vitreous cavity then pass through the tear into the subretinal space (Figure 2–1), augmented by currents within the vitreous cavity caused by rotary eye movements. Although a total PVD is usually seen, many detachments occur with partial vitreous detachment, and evidence of posterior vitreous detachment may not be seen.

Any ocular condition associated with an increased prevalence of vitreous liquefaction, posterior vitreous detachment, or an increased number or extent of vitreoretinal adhesions and traction is likely to be associated with a higher incidence of retinal detachment. As will be discussed later, most eyes with retinal breaks do not develop retinal detachment because existing physiologic forces are sufficient to hold the retina in place.

Vitreous Liquefaction

In early life, the vitreous body is a homogeneous gel consisting of a network of collagen fibrils separated from one another by macromolecules of hyaluronic acid. The density of fibrils is relatively higher near the retina, called the vitreous cortex, but is highest at the vitreous base, the zone of firm anterior vitreoretinal attachment.

Aging of the human vitreous (synchisis senilis) is characterized by liquefaction of the gel and the development of progressively enlarging pools of fluid (lacunae) within the gel. These optically empty liquid spaces continue to coalesce with advancing age, and extensive liquefaction within the vitreous cavity leads
to both a reduction in the shock-absorbing capabilities and the stability of the gel (Figure 2–2). Accelerated vitreous liquefaction is associated with significant myopia, surgical and nonsurgical trauma, intraocular inflammation, and a variety of additional congenital, inherited, or acquired ocular disorders.

**Myopia**

There is abundant evidence that the vitreous gel in myopic eyes has a substantially increased liquid component compared to emmetropic and hyperopic eyes. This is associated with reduced vitreous viscosity and stability.

**Trauma**

Significant blunt or penetrating trauma can damage the vitreous or retina and cause immediate or late changes that increase the odds of subsequent retinal detachment. Blunt trauma can cause accelerated vitreous liquefaction, as well as retinal tears, dialyses, or postnecrotic holes. In eyes with penetrating trauma, dense fibrocellular bands may develop within the vitreous gel and result in traction causing retinal breaks and detachment.

Surgical trauma may contribute to changes in the vitreous that increase the likelihood of retinal detachment. Because the posterior capsule is left intact in the majority of modern cataract operations, the vitreous remains relatively undamaged following uncomplicated surgery. However, subsequent posterior capsulotomy by Nd:YAG laser accelerates the loss of hyaluronic acid, increasing the incidence of vitreous liquefaction and detachment and subsequent retinal tears and detachment.
Inflammation

Intraocular inflammation may predispose to retinal detachment by causing vitreous liquefaction and detachment or by the development of transvitreal membranes, particularly cyclitic membranes. In addition, retinitis can cause severe retinal thinning and associated vitreous liquefaction.

Vitreous Detachment

Vitreous detachment, usually termed posterior vitreous detachment (PVD), usually occurs as an acute event following significant liquefaction of the vitreous gel. The precipitating event is probably a break in the posterior cortical vitreous in the region of the macula, followed by the passage of intravitreal fluid into the space between the cortical vitreous and retina (Figure 2–3). Characteristically, this rapid movement of fluid and the associated collapse of the remaining structure of the gel result in extensive separation of the vitreous gel from the retina posterior to the vitreous base, especially in the superior quadrants.

As the vitreous detaches from around the disc, it may pull loose a glial annulus (Weiss’ ring), which the patient may see as a prominent floater near the visual axis. This is generally considered to be pathognomonic for posterior vitreous detachment (Figure 2–4). With collapse of the vitreous gel, the remaining formed vitreous assumes a position in the inferior aspect of the globe. Because the vitreous remains firmly attached anteriorly, the pull of the collapsed gel frequently creates a fine circumferential retinal fold or ridge near the ora at the posterior limit of the vitreous base.

As noted above, the incidence of vitreous detachment is age related. In one study, slit-lamp examination revealed that 65% of patients older than 65 had vitreous detachment. However, vitreous surgical experience has shown that PVD is frequently misdiagnosed, and in a large series of autopsy eyes, only 22% of eyes
had vitreous detachment by age 65. This number increased to 60% by age 75. Complete and incomplete PVD may cause vitreoretinal traction sufficient to create a retinal break.

The classic symptoms associated with a PVD are the sudden appearance of “floaters” and “flashes.” The former are due to shadows cast by the suddenly collapsed gel,
vitreous hemorrhage, or glial tissue. Flashes are termed photopsias and appear to be due to vitreoretinal traction. The chance of a retinal tear due to PVD in eyes with these sudden symptoms is approximately 15% to 25%, and there is a direct relationship between the amount of vitreous hemorrhage and the likelihood of a tear.

**Vitreoretinal Traction**

Following complete or partial PVD, gravitational traction forces are important and are probably responsible for the predominance of retinal tears in the superior quadrants. However, rotational eye movements, which exert strong forces on all vitreoretinal adhesions, are also important causes of vitreoretinal traction. When the eye rotates, the inertia of the detached vitreous gel causes it to lag behind the rotation of the eye wall and the retina. The retina at the site of a vitreoretinal adhesion exerts force on the vitreous gel, causing the adjacent vitreous to rotate (Figure 2–5). The vitreous gel, because of its inertia, exerts an equal and opposite force on the retina, and this can cause a retinal break or separate the retina further from the pigment epithelium if a break is already present. When the rotational eye movement stops, the vitreous gel continues its internal movement and exerts vitreoretinal traction in the opposite direction.

![Figure 2–5. Rotational eye movements cause vitreoretinal traction. When the eye rotates (large arrow), the detached vitreous gel lags behind the rotation of the eye wall and the retina. The retina at the site of a vitreoretinal adhesion exerts force on the vitreous gel, causing the adjacent vitreous to rotate (arrow). The vitreous gel exerts an equal and opposite force on the retina, causing a retinal break or separating the retina further from the pigment epithelium if a break is already present. Liquid currents within the vitreous gel aggravate the movement of the gel, whereas those in the subretinal space promote extension of the subretinal fluid (arrows).](image-url)
In addition to gravitational and inertial forces, vitreoretinal traction can be caused by contracture of intraocular fibroproliferative tissue associated with retinal vascular proliferative disorders, trauma, and other conditions. This type of traction does not always create a retinal break. Instead, a traction retinal detachment may be produced, and there are classical features that frequently distinguish this type of detachment from the rhegmatogenous variety (see Chapter 5, page 116). Sometimes significant vitreoretinal traction initially causes a localized traction detachment that later becomes more extensive due to the development of a retinal break.

In the normal adult eye, the vitreous adheres firmly to the retina at the vitreoretinal symphysis, or vitreous base. The zone of firm attachment is only 3 or 4 mm wide and straddles the ora serrata. A less firm adhesion of cortical vitreous to the retina is located around the optic disc and surrounding the macula.

Occasionally, a tongue of vitreoretinal adhesion extends posteriorly from the vitreous base as far as the equator. These invisible lesions are responsible for most horseshoe tears, but they are usually not apparent until the tear develops. Vitreoretinal attachment and traction are also found along retinal vessels and are accordingly termed vitreovascular attachments. This accounts for the frequent clinical presentation of a retinal tear with a vitreous hemorrhage and supports the clinical dictum that a vitreous hemorrhage implies a retinal tear until proven otherwise. Pathologic adhesions frequently occur at the margin of a patch of lattice degeneration or at the site of previous chorioretinal inflammation.

**Liquid Currents**

Continued flow of liquid vitreous through a retinal break into the subretinal space is necessary to maintain a rhegmatogenous retinal detachment, because subretinal fluid is continually absorbed from the subretinal space. This flow is encouraged by rotary eye movements that cause liquid currents in the vitreous to dissect beneath the edge of a retinal break into the subretinal space (Figure 2–5). Eye movements also have an inertial effect causing liquid currents in the subretinal fluid, and these favor extension of the retinal detachment.

**Retinal Breaks**

Most retinal breaks do not lead to clinical retinal detachment. Several studies of eye bank eyes and nonselected clinical patients’ eyes have revealed a 5% to 7% prevalence of retinal breaks in the general population. Most of these breaks are small atrophic holes covered by the vitreous base near the ora, and they carry a low risk of subsequent retinal detachment. Equatorial horseshoe tears, which are associated with higher risk, are much less common.

A break in the retina is an essential feature of rhegmatogenous retinal detachment. In addition, liquid in the vitreous cavity must have access to the break. If retinal breaks and posterior vitreous detachment are common, why does clinical detachment of the retina occur so infrequently? Whether or not significant fluid vitreous passes through a retinal break depends upon the balance of forces acting at the
edge of the break. Forces normally responsible for maintaining retinal attachment include negative pressure in the subretinal space created by the metabolic pump of the retinal pigment epithelium and the relatively higher oncotic pressure in the choroid, interdigitation of the pigment epithelial cell processes and the outer segment of photoreceptors, and mucopolysaccharide “glue” between the pigment epithelium and the sensory retina. Retinal detachment occurs when forces favoring adherence of the retina are overwhelmed by forces promoting an accumulation of subretinal fluid.

**Primary and Secondary Breaks**

Retinal breaks are termed *primary* when they are responsible for the production of the detachment. They are termed *secondary* when they are present preoperatively but are not responsible for the detachment and are subsequently lifted up by subretinal fluid. Secondary breaks also include those that develop in the retina after it detaches. There have been a few rare cases of secondary breaks appearing at the edge of paving-stone degeneration. Secondary breaks have also been seen at the site of old chorioretinal scars resulting from either inflammation or photocoagulation. A secondary break can also occur at the time of photocoagulation if excessive power is used. All breaks, both primary and secondary, should be treated at the time of surgery.

**Types of Breaks**

Retinal breaks may be subdivided into *tears*, *holes*, and *dialyses* (Table 2–1). Tears are produced by traction on the retina, whereas holes are due to a gradual thinning of the retina (Figure 2–6). Tears usually occur suddenly, with the retina frequently appearing completely normal before the acute event. Atrophic holes appear to develop slowly, whereas traumatic dialyses probably occur acutely. Most breaks causing retinal detachment are associated with vitreoretinal traction in the vicinity of the break(s). Dialyses usually feature traction on the retina immediately posterior to the break, and if traction is confined to the retina anterior to the dialysis, a *giant tear* is more likely to evolve.

**Horseshoe tears**

Also referred to as *flap* or *U-shaped* tears, horseshoe tears occur in most cases at the irregular posterior margin of the vitreous base during posterior vitreous detachment. The flap thus remains adherent to the posterior vitreous surface following

<table>
<thead>
<tr>
<th>Type</th>
<th>Cause</th>
<th>Shape</th>
<th>Usual Location</th>
<th>Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tear</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Horseshoe</td>
<td>Tractional</td>
<td>Flap</td>
<td>Superior (esp. temporal)</td>
<td>Acute</td>
</tr>
<tr>
<td>Operculated</td>
<td>Tractional</td>
<td>Round</td>
<td>Superior (more posterior)</td>
<td>Acute</td>
</tr>
<tr>
<td><strong>Hole</strong></td>
<td>Degenerative/atrophic</td>
<td>Round</td>
<td>Temporal</td>
<td>Gradual</td>
</tr>
<tr>
<td><strong>Dialysis</strong></td>
<td>Traumatic or familial</td>
<td>Linear</td>
<td>Inferotemporal</td>
<td>Acute</td>
</tr>
</tbody>
</table>
the creation of a tear (Figures 2–6A, 2–7). Essential conditions for the production of a horseshoe tear are a preexisting vitreoretinal adhesion and traction applied to this point via the vitreous. Because of the associated vitreous traction, flap tears frequently lead to detachment. Horseshoe tears are most common in middle age and appear most often near the equator of the eye.
Operculated breaks
Operculated breaks are technically tears because free opercula are produced by a mechanism identical to the one involved in horseshoe tears. However, the flap has been torn free from the retina at the anterior edge of the flap; therefore, the operculum is separated from the retina and adheres to the posterior hyaloid membrane (Figure 2–6B and 2–8). Because the vitreoretinal traction is usually no longer adherent to the surrounding retina, operculated retinal breaks very rarely lead to retinal detachment unless vitreoretinal traction persists in the vicinity of the tear.

The breaks are caused by isolated areas of vitreoretinal adhesion and are not contiguous to the vitreous base. Operculated retinal breaks tend to be more posterior than horseshoe tears, and occasionally an operculated break is found posterior to a horseshoe tear in the same meridian.

Atrophic holes
Atrophic holes are due to the gradual thinning of the retina, often in association with lattice degeneration (Figure 2–6C and 2–9). These small holes occur in the middle of the lattice patch and are distinguished from the retinal tears that occur in full-thickness retina at the margin of the lattice patch. The holes are due to atrophy, but the tears are related to vitreoretinal adhesions, which are universally found at the margin of lattice patches. Still, a progressive accumulation of subretinal fluid is usually due to vitreoretinal traction on the lattice lesions containing atrophic retinal holes.

Retinoschisis is frequently accompanied by atrophic holes. Inner-layer holes are generally small and difficult to see. Outer-layer holes, which are larger and therefore more easily seen, are much more significant in the pathogenesis of retinal detachment (Figure 2–6D).
Atrophic holes sometimes occur in diffuse chorioretinal atrophy, such as in the equatorial zone of the highly myopic eye (Figure 2–9). In rare instances, they are caused by the rupture of a cystoid space in peripheral cystoid degeneration. However, in these cases, the holes seldom produce retinal detachments, because those at the extreme periphery are usually covered by the base of the vitreous.

**Dialyses**

A retinal dialysis is a circumferential linear tear, the anterior margin of which is at or near the ora serrata (Figure 2–6E and 2–10). The retina is thinnest and least
developed at the ora, especially in the inferior temporal quadrant. Dialyses occur at any age, but they are particularly common in youth—hence the well-known clinical entity of inferior temporal dialysis of the young.

About 75% of retinal breaks that occur after blunt ocular trauma are retinal dialyses. The dialysis is thought to result from the marked deformation of the globe, which, in association with the relatively inelastic vitreous base, tears the peripheral retina.

A pathognomonic sign of trauma, avulsion of the vitreous base, is sometimes seen in these cases. This constitutes a circumferentially torn ribbon of the nonpigmented epithelium of the pars plana and the peripheral retina. The ribbon with its adherent vitreous tends to drape down over the peripheral retina.

Penetrating trauma often causes a dense vitreous band to form along the track of the injury, and the band may contract, which subsequently detaches the retina.

In dialyses, the vitreous gel generally remains adherent to the posterior edge of the torn retina, where it exerts some degree of vitreoretinal traction. In this regard, dialyses are different from the vast majority of other retinal tears in which the vitreous is attached to the anterior flap. A dialysis occurring in association with vitreous attached to the anterior flap (Figure 2–6F) behaves much differently than a routine dialysis, and such cases frequently extend to become giant retinal tears (Figure 2–11).

**Postnecrotic holes**

Postnecrotic holes are breaks that develop following retinitis or trauma. Cytomegalovirus (CMV) is a common cause of infectious retinitis in immunologically deficient patients, such as patients on immunosuppressive therapy, fetuses in utero, and patients with acquired immune deficiency syndrome (AIDS). Patients with poor immune function due to AIDS may develop CMV retinitis, and of those who do, retinal detachment occurs in about 17%. These detachments typically
are associated with multiple tiny postnecrotic holes in the thin areas of previous inflammation (Figure 2–12).

Similar findings may be present with other forms of infectious retinitis, such as the acute retinal necrosis (ARN) syndrome. Postcontusional retinal necrosis with breaks also may develop following blunt trauma.

**Retinal breaks due to proliferative diabetic retinopathy**

Vitreous changes are instrumental in the production of atypical holes causing rhegmatogenous retinal detachment in proliferative diabetic retinopathy. Numerous vitreoretinal adhesions form before separation of the posterior hyaloid, which typically occurs slowly, as opposed to senescent PVD. As the vitreous detaches, the
retina may be focally elevated as a traction retinal detachment. Occasionally, the retina splits, producing secondary retinoschisis, especially along retinal vessels. Progression of this process may produce retinal breaks, which are usually located just anterior to the margin of the vitreous separation (Figure 2–13). The typical rhegmatogenous detachment of proliferative diabetic retinopathy starts as a detachment of the posterior retina with small traction-related breaks, from which the retinal detachment subsequently spreads anteriorly to the equator or even the ora serrata.

**Macular holes**

Macular holes may be traction-induced or atrophic, operculated or nonoperculated. Gass has postulated that they often develop as a result of tangential traction from the vitreous cortex at the margins of the foveal pit, although other studies have demonstrated a “micro” or “partial” PVD as being responsible. Macular holes may also be degenerative or may result from coalescence of intraretinal cystoid spaces. Trauma may also induce macular holes. The rare macular breaks that produce detachments are usually associated with high myopia or trauma (Figure 2–14).

**Distribution of Breaks**

The distribution of retinal breaks throughout the quadrants of the fundus is different for each type of break. Horseshoe tears are most common in the superior temporal quadrant; the second most susceptible site is the superior nasal quadrant. Operculated tears are also located most frequently in the superior quadrants, although they tend to occur more posteriorly than horseshoe tears. Atrophic retinal breaks are also usually located in the superior temporal quadrant, but the second

![Figure 2–13. Retinal detachment associated with proliferative diabetic retinopathy. The retinal break is not visible, but it lies just peripheral to a site of vitreous traction upon fibrovascular tissue.](image-url)
most common quadrant is the inferior temporal. Dialyses are found most frequently in the inferior temporal quadrant (Table 2–1). The preponderance of superior temporal breaks in adults is not seen in juveniles because of the relatively high incidence of inferior temporal dialyses among youths (Table 2–2; Figure 2–15).

In one large series of retinal breaks, 12% were found near the ora, 28% between the ora and the equator, 45% near the equator, 14% posterior to the equator, and 1% at the macula. In the typical pseudophakic detachment, one or more small retinal breaks are seen near the ora along the posterior margin of the vitreous base, whereas equatorial breaks are more common in phakic eyes (Table 2–3). In approximately 50% of retinal detachments, there is only one break. When there are multiple breaks, they are found to be within 90 degrees of one another in 75% of the affected eyes.

**Lesions Associated with Retinal Breaks**

Congenital anomalies and peripheral cystoid degeneration rarely lead to clinically significant retinal breaks. Although inner- or outer-layer breaks may develop in degenerative retinoschisis, it is the outer-layer breaks that occasionally produce a clinical detachment of the retina. Retinal breaks with lattice degeneration are common, although the great majority of these breaks do not lead to retinal detachment. Still, the condition is sufficiently common that 20% to 30% of retinal detachments are associated with lattice lesions. Most retinal tears that cause retinal detachment occur where vitreoretinal adhesions are invisible and the retina appears normal before vitreous detachment.

**Congenital anomalies**

Peripheral retinal variations may be associated with vitreoretinal adherence and traction. Cystic retinal tufts (congenital retinal rosettes, granular patches, and

Figure 2–14. Retinal detachment associated with a macular hole in a highly myopic eye.
Table 2–2. Retinal Break Location vs. Age

<table>
<thead>
<tr>
<th>Age</th>
<th>Typical Location of Breaks</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;20</td>
<td>Temporal</td>
</tr>
<tr>
<td>&gt;40</td>
<td>Superior</td>
</tr>
</tbody>
</table>

Figure 2–15. Quadratic distribution of retinal breaks by age group.

glial spheres) and zonular traction tufts display such attachment, so that a retinal tear may be produced with or without posterior vitreous detachment. Cystic retinal tufts frequently lie posterior to the vitreous base, and as visible sites of vitreoretinal adhesion, they are important locations at which tears occur.
Peripheral cystoid degeneration

Peripheral cystoid degeneration, occasionally seen in youth, is virtually universal in the far periphery of the senescent eye (Figure 2–16). Minimal cystoid degeneration is normally located immediately posterior to the ora serrata, and in more advanced cases it might extend as far posterior as the equator. This ubiquitous finding is generally insignificant. In rare cases both the inner and the outer layers of a cyst rupture, creating a through-and-through retinal hole. The hole seldom leads to detachment because it is frequently covered by the vitreous base.

The cystic process begins in the outer plexiform layer but with enlargement can extend from the inner to the outer limiting membranes, with stretching of the Müller cell fibers that bridge the cystic space. When the stretched glial fibers give way, a frank splitting of the retina, referred to as degenerative retinoschisis, is produced.

Degenerative retinoschisis

Degenerative retinoschisis results from the progression of peripheral cystoid degeneration and is usually bilateral. As the glial septa rupture and the small cysts coalesce, a splitting occurs (Figure 2–6D and 2–17). Approximately 5% of the adult population has been found to have retinoschisis, generally of the flat type. The splitting may occur in any quadrant but is most common in the inferior temporal quadrant. Although splitting might extend posteriorly toward the macula, the most frequent avenue of extension is by circumferential progression around the periphery.

The inner layer of the bullous schisis cavity is extremely thin and characteristically exhibits multiple white flecks of uncertain etiology known as snowflakes. Blood vessels coursing over the dome of the retinoschisis frequently become sclerotic and are recognized by their white color. Inner-layer breaks are difficult to visualize with the ophthalmoscope; they tend to be multiple and are relatively small. The less common outer-layer break is more readily seen ophthalmoscopically because it tends to range from 1 to 5 disc diameters in size and is often found posterior to the equator (Figure 2–17).

The outer layer usually remains adherent to the retinal pigment epithelium, and the rods and cones may be relatively well preserved though synaptically disconnected. As in peripheral cystoid degeneration, a mucopolysaccharide substance exists within the schisis cavity that has been found to be sensitive to hyaluronidase.

When retinoschisis produces a retinal detachment, the subretinal fluid is derived from both the schisis and the vitreous cavities. The outer-layer breaks are usually

<table>
<thead>
<tr>
<th>Lens Status</th>
<th>Typical Location</th>
<th>Type of Break</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phakic</td>
<td>Equatorial</td>
<td>Horseshoe</td>
</tr>
<tr>
<td>Pseudophakic or aphakic</td>
<td>Near ora</td>
<td>Small; round or horseshoe</td>
</tr>
</tbody>
</table>

Table 2–3. Distribution of Retinal Breaks
Figure 2–16. Peripheral cystoid degeneration. (A) Gross photograph of peripheral distribution immediately posterior to the ora serrata. (B) Photomicrograph. Cystoid spaces typically develop in the outer plexiform layer. (Courtesy of Hans E. Grossniklaus, MD.)

Figure 2–17. Retinal detachment associated with retinoschisis. Three large outer layer holes (with rolled edges) are responsible. (Courtesy of H. Richard McDonald, MD.)
visible, but there may be tiny inner-layer breaks that are not clinically detectable. Outer-layer breaks are required for the development of detachment. Such detachments do not depend on the presence or absence of inner-layer breaks.

**Lattice degeneration**

Lattice degeneration is the most important visible lesion associated with subsequent retinal detachment. It initially involves the vitreous cortex and the inner layers of the retina (Figure 2–6C). As the lesion progresses, the outer layers also become involved, and there may be sufficient thinning to cause through-and-through retinal holes (Figure 2–18).

The most consistent ophthalmoscopic feature of lattice degeneration is a circumscribed patch of excavation of the inner retinal surface. The patch is generally circumferential, oblong, and equatorial or pre-equatorial. Lattice degeneration varies in extent from a single isolated patch to almost continuous lesions around the equatorial zone of all four quadrants. There is usually just one plane of involvement between the ora and the equator, but occasionally there are two or three parallel rows, or tiers, of circumferentially oriented lattice. Infrequently, the lattice is radial in its orientation, in which case it is generally in a perivascular distribution posterior to the equator.

Ophthalmoscopic examination reveals many variations in the appearance of lattice degeneration. In one form, referred to as *snail-track degeneration*, the lesion is completely white and almost reflective, with tiny, dot-like features on its surface (Figure 2–19). In most areas of lattice, the degenerative process is sufficiently deep.
to involve the pigment epithelium; there is derangement of pigment, with pigment clumping and loss. In some lesions, there are fine, branching white lines due to sclerosis of the blood vessels passing through the lesion (Figure 2–20). (The term lattice is derived from this characteristic.) In general, fluorescein angiography of lattice lesions reveals a zone of relative avascularity.

There may be small, round, atrophic holes due to retinal thinning in the patch of lattice, but retinal tears at the edge of the patch due to vitreoretinal adhesions are more dangerous. These tears usually develop along the posterior edge of the lesion or at either end of the lattice lesion (Figure 2–21).

If some degree of posterior vitreous detachment is not present, only a small amount of fluid can pass through a lattice hole. This fluid is derived from the small pocket of vitreous liquefaction that overlies such lesions. A small amount of stable subretinal fluid, which is usually confined within the lattice patch, is therefore not regarded with the same concern as extensive subretinal fluid. However, a slowly progressive accumulation is seen with some eyes with lattice degeneration containing atrophic holes, especially those who are highly myopic, and this is usually associated with at least a modest vitreous detachment and vitreoretinal traction upon the lattice lesion(s). Retinal tears are accompanied by more complete posterior vitreous detachment.

Lattice degeneration is found in all age groups, and new patches of lattice occur rarely after the first decade of life. Changes within the lesion do occur, however, and many progressive features have been noted. Longitudinal observation has documented the subsequent appearance of pigmentation, branching white lines, retinal breaks, and subretinal fluid.

In one large series of 800 autopsy and 100 clinical eyes, lattice degeneration was found to be bilateral in 48% of cases, and more than one lesion was present in 50% of the 900 eyes examined. The lesions were parallel to the ora in 68% of

Figure 2–19. Lattice degeneration with a “snail-track” appearance. (Courtesy of Norman E. Byer, MD.)
the eyes, oblique in 25%, and perpendicular in 7%. Lattice oriented perpendicular to the ora was usually quite posterior. Pigment clumping occurred in 92% of the lesions, retinal holes in 18%, white vessels in 7%, and retinal tears in 1.4%. The incidence of myopia in the series was higher than in the general population. As with myopia, there is a tendency for lattice to run in families. In another series of 104 asymptomatic patients with lattice degeneration, a 5-year follow-up disclosed

Figure 2–20. Sclerotic retinal vessels passing through lattice lesions are responsible for the “lattice-like” appearance. (Courtesy of Norman E. Byer, MD.)

Figure 2–21. Horseshoe tears associated with lattice degeneration usually occur along the posterior and/or lateral edges of the lattice lesions. (Courtesy of Norman E. Byer, MD.)
that 0.2% had retinal detachment. If such a study were continued for 50 years, a prevalence of 2% could be expected.

**EPIDEMIOLOGY OF RETINAL DETACHMENT**

Reliable information regarding the incidence and prevalence of retinal detachment is difficult to find because of the mobility of today’s population. Several epidemiologic studies of relatively confined groups reveal an annual incidence of about one retinal detachment in 10,000. Assuming an average life expectancy of 74 years, the prevalence is approximately 0.7%.

The most common features associated with retinal detachment are myopia, pseudophakia, lattice degeneration, and trauma. Approximately 40% to 55% of all detachment patients have myopia, and the amount of myopia is directly related to the likelihood of detachment. Twenty to thirty percent of retinal detachments are associated with lattice degeneration, and in 10% to 20%, the eyes have suffered direct ocular trauma. Approximately 30% to 40% of detachments are associated with a history of cataract surgery, and detachment is more likely if the vitreous gel has been involved by surgical complications or Nd:YAG capsulotomy.

Traumatic detachments are most common in youth, myopic detachments occur most frequently among people older than 25, and the incidence of pseudophakic detachments rises with each decade of advancing age (Figure 2–22). The risk factors are not mutually exclusive, and highly myopic eyes with lattice degeneration that undergo cataract surgery appear to be at relatively high risk.

![Figure 2–22. Age distribution of five types of retinal detachment. (Redrawn with permission from S. Karger AG, Basel, from Hilton GF, Norton EWD: Juvenile retinal detachment. *Mod Probl Ophthalmol* 1969;8:325–341.)](image-url)
**Variables Regarding Epidemiology**

**Age**
The most common ages for retinal detachment are between 40 and 80 years, although there is a small increase in rate due to trauma or hereditary factors in the teens (Figure 2–23). In rare instances, detachment is discovered in a newborn, and occasionally it occurs as late as the ninth or tenth decade of life.

**Sex**
Approximately 60% of detachments occur in males. The incidence remains higher for males even when data are corrected for ocular trauma, which is much more common among males than females.

**Race**
The incidence of detachment is reported to be relatively high among Asians and Jews, and relatively low among people of African descent. One study of Native Americans revealed an incidence essentially equal to that of Caucasians.

**Heredity**
Primarily because myopia and lattice degeneration have hereditary tendencies, retinal detachment also has some hereditary predisposition. A positive family history of retinal detachment is a relevant risk factor, but most cases are sporadic.

**Bilaterality**
Approximately 15% of detachment patients ultimately develop detachment in the second eye. The effect on bilaterality of prophylactic treatment, such as cryopexy of retinal breaks in the second eye at the time of detachment in the first eye, has not

**Figure 2–23.** Age distribution of retinal detachment patients with small juvenile mode and prominent middle-age mode. (Redrawn with permission of S. Karger AG, Basel, from Hilton GF, Norton EWD: Juvenile retinal detachment. *Mod Probl Ophthalmol* 1969;8:325–341.)
been optimally evaluated. Bilateral detachments are more common in pseudophakic patients, with an incidence as high as 25% to 30%.

**SYSTEMIC AND GENETIC CONDITIONS ASSOCIATED WITH RETINAL DETACHMENT**

Rhegmatogenous detachments can result from retinal breaks associated with Marfan syndrome, Ehlers-Danlos syndrome, Wagner’s vitreoretinal degeneration, Stickler syndrome, Pierre Robin syndrome, familial exudative vitreoretinopathy, juvenile retinoschisis, proliferative diabetic retinopathy, proliferative sickle retinopathy, retinopathy of prematurity, atopic dermatitis, acute retinal necrosis, and cytomegalovirus virus retinitis.

**CLASSIFICATION OF RETINAL DETACHMENTS**

Retinal detachments may be classified according to a variety of morphologic findings (Table 2–4). There may be considerable overlap in some of these categories,

<table>
<thead>
<tr>
<th>Classification of Detachment</th>
<th>Size of Break</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphology of Break</td>
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<tr>
<td>Horseshoe</td>
<td>Small</td>
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<td>Round or oval</td>
<td>Moderate</td>
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<td>Operculated</td>
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<td>Atrophic</td>
<td>Giant</td>
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<td>Irregular</td>
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<td>Etiology of break</td>
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<td>Total</td>
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<td>Detachment of pars plana</td>
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<tr>
<td>Near ora serrata</td>
<td>epithelium</td>
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</tr>
<tr>
<td></td>
<td>Linear and angular strands on</td>
</tr>
<tr>
<td></td>
<td>outer surface of detached retina</td>
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<tr>
<td></td>
<td>(retina fibrosis)</td>
</tr>
<tr>
<td></td>
<td>Microcystic degeneration of retina</td>
</tr>
<tr>
<td></td>
<td>Thinning of retina</td>
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<tr>
<td></td>
<td>Macular edema</td>
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<tr>
<td></td>
<td>Macular hole</td>
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<tr>
<td></td>
<td>Proliferative vitreoretinopathy</td>
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<tr>
<td></td>
<td>(PVR)</td>
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</table>
as a variety of retinal breaks can coexist in the same case. Atypical rhegmatogenous retinal detachments are commonly seen in eyes with proliferative diabetic retinopathy.

**PATHOLOGY OF THE DETACHED RETINA**

The outer layers of the detached retina undergo the greatest alteration due to separation from the blood supply of the choriocapillaris. Experimental work with the owl monkey has revealed degenerative changes in the outer segments of the receptor cells within a few days after detachment. The discs of the outer segments become ruptured and disorganized and, in time, assume bizarre shapes. In the normal retina, shed fragments of photoreceptor outer segments are phagocytosed by the retinal pigment epithelium. These lamellar inclusion bodies are referred to as phagosomes.

In retinal detachment, this process is disrupted so that phagosomes disappear from the retinal pigment epithelium. Both proliferative and degenerative changes occur in the retinal pigment epithelium. Changes visible with an electron microscope include migration of the pigment granules from the anterior processes of the retinal pigment epithelial cells posteriorly and thickening of Bruch’s membrane. After surgical reattachment, the receptor cell outer segments regenerate, the discs assume a more normal pattern, and phagosomes reappear in the retinal pigment epithelial cells.

With long-standing detachments, the inner and outer nuclear layers of the detached retina undergo degeneration, become thinner, and may ultimately fuse into one layer. Although the inner retina is well preserved initially, the ganglion cells decrease in number in long-standing detachments and may eventually disappear. Other chronic changes include hyalinization of both retinal vessels and the choriocapillaris. Additional pathologic findings in eyes with chronic retinal detachments include proliferative vitreoretinopathy, intraretinal macrocysts, and demarcation lines.

**Proliferative Vitreoretinopathy**

The most ominous and clinically significant finding in retinal detachment is the presence of proliferative vitreoretinopathy (PVR), the process that is responsible for the vast majority of surgical failures of retinal reattachment surgery. The consequence of cell migration and elaboration of collagen is the formation of membranes involving the inner and outer surfaces of the retina, as well as the vitreous. In time, and under the influence of mediators of inflammation, the membranes contract, distorting the retina into folds (Figure 2–24). Localized contracture in the periphery is referred to as a *star fold* (Figure 2–25), and a similar process in the posterior pole is referred to as a *macular pucker* (Figure 2–26). Two more recent classification systems for PVR have come into use and, though imperfect, they have improved our evaluation of retinal detachment and its therapy (see Chapter 5, page 104).
Figure 2–24. Total retinal detachment associated with proliferative vitreoretinopathy (PVR). The retina is pulled into fixed folds by the membranes on the surface of the retina.

Figure 2–25. A star fold located temporal to the macula is due to localized epiretinal membranes that cause surface traction on the detached retina.

Figure 2–26. A “macular pucker” (or “epimacular proliferation”) is due to an epiretinal membrane localized to the central area of the retina.
INTRARETINAL MACROCYSTS

Large intraretinal cystoid spaces may develop over many months in long-standing retinal detachments (Figure 2–27). They may develop in the posterior pole but are more frequent in the equatorial zone. Retinal detachments caused by inferior retinal breaks, such as the classic inferior temporal dialysis of the young, are generally slow in their progression and thus may be present long enough to develop

Figure 2–27. Intraretinal cysts are a sign of chronic retinal detachment. (A) A large intraretinal cyst in detached retina. (B) Following reattachment, the cyst has spontaneously resolved.
macrocysts before the detachment becomes symptomatic. Conversely, macrocysts are relatively rare in retinal detachments caused by superior breaks. With surgical reattachment, the macrocysts regress rapidly (Figure 2–27B).

**DEMARCATION LINES**

If the boundary of a retinal detachment remains stable for a long time, pigment epithelial hyperplasia occurs at the junction between the detached and attached retina. It is generally accepted that a minimum of 3 months is required to generate a demarcation line. The to-and-fro undulations of the retina probably have an irritating effect on the retinal pigment epithelium at the boundary and induce a proliferative change, which subsequently evolves to fibrous metaplasia.

Most demarcation lines have prominent pigmentation (Figure 2–28), but occasionally the pigment granules are lost and nonpigmented demarcation lines result. In rare cases, calcification of the line occurs in long-standing detachments. Demarcation lines may create a permanent barrier to the further progression of the detachment, but often the detachment extends through the barrier and progresses posteriorly. Several concentric demarcation lines may appear, indicating intermittent extension and temporary stability in the course of detachment.

**NATURAL HISTORY OF UNTREATED DETACHMENT**

A limited retinal detachment left untreated may follow one of four outcomes:

1. Usually, most untreated clinical rhegmatogenous detachments progress to near total or total detachment and blindness.
Occasionally, a detachment remains indefinitely as a subtotal detachment with stable borders and the creation of demarcation lines. This is most apt to occur in detachments caused by inferior breaks, particularly small breaks or dialyses.

3. Rarely, subretinal fluid due to a superior retinal break settles inferiorly away from the break, and the site of the original break flattens.

4. Very rarely, spontaneous reattachment occurs, usually associated with a very small break and excellent presumed “pumping” of the retinal pigment epithelium or closure of the break by scar tissue (Figure 2–29).

Retinal detachment is usually associated with decreased intraocular pressure secondary to increased resorption of fluid from the subretinal space. Uveitis or traction on the ciliary body by proliferative vitreoretinopathy may also decrease the production of aqueous humor to the point of hypotony and eventual phthisis. Occasionally, the low-grade uveitis that accompanies retinal detachment damages the trabecular meshwork enough to produce elevated intraocular pressure. Rubeosis iridis may also develop in long-standing detachment, resulting in neovascular glaucoma. When a long-standing detachment is repaired, glaucoma may replace relative hypotony due to damage to the trabecular meshwork.

Clinically, proliferative vitreoretinopathy is seen most often after surgery, but it is also observed as the end result of the natural history of unoperated retinal detachment. Cataract may also develop as a late effect of retinal detachment.

**SUMMARY**

Retinal detachments may be caused by traction or by exudation from a choroidal tumor or inflammation, but most detachments are caused by retinal tears
(rhegmatogenous detachments). Vitreous liquefaction and vitreous detachment can lead to traction on the retina with subsequent retinal tear formation. Liquid currents within the vitreous cavity along with persistent traction promote retinal detachment.

Horseshoe tears and retinal dialyses have a much higher likelihood of leading to retinal detachment than atrophic or operculated retinal breaks, and not all tears require prophylactic treatment. Retinal breaks are most common in superior and temporal quadrants and less likely in the inferior and nasal retinal periphery. The most common risk factors for development of retinal detachment are myopia, history of cataract surgery, lattice degeneration, ocular trauma, and a personal or family history of retinal detachment. Various other ocular, systemic, and genetic conditions can also lead to retinal detachment. Combinations of risk factors increase the likelihood of retinal detachment.

Retinoschisis is to be distinguished from and may occasionally lead to retinal detachment. Proliferative vitreoretinopathy, intraretinal macrocysts, and demarcation lines may accompany chronic retinal detachment. Untreated retinal detachment usually progresses, leading to retinal deterioration and eventual blindness.

SELECTED REFERENCES


Indirect viewing systems, including the binocular indirect ophthalmoscope and slit lamp biomicroscopy through an indirect lens, have become the standard of care for management of retinal detachments. Comparison with direct ophthalmoscopy illustrates the capabilities of indirect systems. The technique of indirect ophthalmoscopy with scleral depression is presented.\(^1\)

**CHARACTERISTICS OF INDIRECT AND DIRECT OPHTHALMOSCOPY**

The characteristics of direct and indirect ophthalmoscopy are compared in Table 3–1. Figure 3–1A shows the optical principles of direct ophthalmoscopy, and Figure 3–1B illustrates the optics of the indirect method. Substantial clinical differences between the two methods are due to the differences in optical characteristics.

**DIFFERENCES IN VISUALIZATION**

*Magnification and resolution*

The direct ophthalmoscope offers 14X magnification compared with 3X with the indirect using the usual +20 diopter lens. However, this does not mean the direct device has an equal advantage in resolution. Resolution is a function of how close

\(^1\) This chapter has been substantially edited and condensed from *Highlights of Ophthalmology* 1966, 179–257, ML Rosenthal & S Fradin.
Figure 3–1. (A) Optical principles of direct method of ophthalmoscopy. (B) Optics of indirect ophthalmoscopy.

Table 3–1. Features of Direct vs. Binocular Indirect Ophthalmoscopy

<table>
<thead>
<tr>
<th>Feature</th>
<th>Direct</th>
<th>Indirect (+20 lens)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Magnification</td>
<td>14X</td>
<td>3X</td>
</tr>
<tr>
<td>Field diameter</td>
<td>2DD</td>
<td>9DD</td>
</tr>
<tr>
<td>Ratio of area of field</td>
<td>1</td>
<td>20</td>
</tr>
<tr>
<td>Illumination</td>
<td>Limited</td>
<td>High</td>
</tr>
<tr>
<td>Depth of focus</td>
<td>Small</td>
<td>Large</td>
</tr>
<tr>
<td>Stereopsis</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Orientation of image</td>
<td>Direct</td>
<td>Inverted, reversed</td>
</tr>
<tr>
<td>View of periphery</td>
<td>Limited</td>
<td>Full</td>
</tr>
<tr>
<td>Scleral indentation</td>
<td>Difficult</td>
<td>Easy</td>
</tr>
</tbody>
</table>

together two points can be and remain distinguished as separate when viewed through an optical system. The visualization of detail that an optical system permits is a function of its resolving power and not its magnification. Resolution is a function of the light available at the points to be resolved and of the quality of the optical components of the system. Magnification plays a role only if the resolution of the optical system exceeds the resolution of the observing human eye at a given level of magnification. Too much magnification of a poorly resolved image results in a loss of detail, such as if one were to examine a halftone newspaper photograph under a microscope.

With the direct method, the greater the degree of myopia, the higher the magnification of the fundus image and the smaller the field of view. In very high myopes, the field of view with the direct instrument becomes very limited.
High cylindric errors strongly and adversely affect the image of direct ophthalmoscopy because the high magnification of the system also magnifies the effects of refractive errors on the image. With the indirect method, the lower magnification minimizes this effect. Furthermore, the condensing lens can be tilted slightly to overcome astigmatic aberrations. Examination of the retinal periphery entails the travel of light obliquely through the cornea and lens, introducing cylindrical aberrations that are likewise problematic with the direct ophthalmoscope and easily overcome with the indirect.

Due to high illumination, binocular viewing, and high-quality optics, a good indirect ophthalmoscope and aspheric lens provide good resolution in spite of low magnification. Substantial advantages gained include a very wide field of view, stereoscopy, large depth of focus, and dynamic examination capability. During the early stages of learning indirect ophthalmoscopy, it is essential to accept that one has to work with a smaller image size; after a time, one ceases to be troubled by it. After enough experience with indirect ophthalmoscopy, one is rarely aided in an evaluation of detail by increased magnification. However, if higher magnification is needed for examining a specific lesion, this can be achieved in several ways:

Magnification is increased by moving the examiner’s head closer to the patient’s eye (rather than examining at nearly arm’s length as is usual). However, this is difficult if the pupil is not well dilated. Using a lower power condensing lens, such as a 14 diopter lens, also provides more magnification, but is also more difficult with a poorly dilated pupil (Table 3–2). The most common way to increase magnification while maintaining the advantages of indirect viewing is to use a slit lamp biomicroscope with a 60- to 90-diopter lens (Figure 3–5). Higher magnification is also achieved by using a slit lamp with a contact lens with or without reflecting mirrors.

**Field of view**

Figure 3–2A shows what an observer sees with a direct ophthalmoscope in an emmetropic eye, in comparison with Figure 3–2B, which shows the field of view with indirect ophthalmoscopy. This is a 20-fold difference in field size, and it makes a huge difference in the facility of the instrument, especially for the diagnosis and treatment of retinal detachment. The topography of the detached retina may be complex with multiple folds, and the view with the direct ophthalmoscope

<table>
<thead>
<tr>
<th>Diopters</th>
<th>Used with</th>
<th>Magnification</th>
<th>Static Field of View</th>
<th>Working Distance from Cornea (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>14</td>
<td>BIO²</td>
<td>4.2 X</td>
<td>38 degrees</td>
<td>72</td>
</tr>
<tr>
<td>20</td>
<td>BIO</td>
<td>3.0 X</td>
<td>50 degrees</td>
<td>47</td>
</tr>
<tr>
<td>28</td>
<td>BIO</td>
<td>2.1 X</td>
<td>58 degrees</td>
<td>27</td>
</tr>
<tr>
<td>60</td>
<td>Slit lamp</td>
<td>1.0 X³</td>
<td>88 degrees⁴</td>
<td>10</td>
</tr>
<tr>
<td>78</td>
<td>Slit lamp</td>
<td>0.8 X³</td>
<td>98 degrees⁴</td>
<td>7</td>
</tr>
<tr>
<td>90</td>
<td>Slit lamp</td>
<td>0.75 X³</td>
<td>94 degrees⁴</td>
<td>5</td>
</tr>
</tbody>
</table>

¹ Static (instantaneous) field of view varies depending on the size of the lens.
² BIO = binocular indirect ophthalmoscope.
³ Magnification is increased as a function of the magnifying power of the slit lamp.
⁴ Using a slit lamp, the portion of the potential static field of view that is illuminated and visualized at one time is limited by the magnification system.
Figure 3–2. (A) Shows single field of view in emmetropic eye with direct ophthalmoscope. This field is approximately 10° in diameter. (B) Shows single field of view in same eye seen with indirect ophthalmoscope. This field is approximately 37° in diameter. Since the ratio of areas of these fields is proportional to the square of radii of the fields, it follows that the field in Figure B covers an area 14 times as great as that in Figure A. The indirect method permits examination of the disc, macula, and perimacular retinal vessels at one time. Contrast between these two fields would be even more striking if the eye being examined has 20D of myopia. In such case, Figure A would show the optic disc occupying the entire field of view, while Figure B would be unchanged. Ratio of areas seen would therefore be much greater than 14-to-1.

is difficult to interpret. The field of view is so small that one sees only a small part of the convoluted retina in any one field, and it is difficult to relate each separate view to adjacent areas.

When examining for retinal detachment, perhaps even more important than the field of view is the viewable field. Direct ophthalmoscopy permits the study of approximately 60% to 70% of the total fundus area in a well-dilated, emmetropic eye (Figure 3–3). In aphakia it may be possible to visualize more than this area, whereas in myopia less than 60% of the fundus can be seen. Thus, peripheral examination is very difficult, and as explained above, even when the periphery can be seen with the direct ophthalmoscope, the image is very blurry. Piecing together what one sees is even harder, so in practical terms, the direct ophthalmoscope is rarely used to examine beyond the posterior pole. Since 30% of the retina lies anterior to the equator, failure to study this region will result in overlooking serious pathology in many, if not most, cases. Diseases such as senile retinoschisis, peripheral uveitis, and most retinal tears and detachments defy evaluation by any other technique.

Illumination

Image brightness of a direct is low due to limited power output. Direct ophthalmoscopes operated by batteries provide about one-half watt of illumination. Instruments operated through transformers deliver several times this amount, but never more than several watts. Indirect ophthalmoscopes can deliver up to 18
watts of output. Better illumination results in improved resolution and improved performance in the presence of media opacities.

**Stereopsis and depth of focus**

The image with the usual direct ophthalmoscope is not stereoscopic and has essentially no depth of focus, whereas the indirect ophthalmoscope affords a stereoscopic view with an excellent depth of focus. These qualities of the image are invaluable in interpreting lesions with depth, including retinal detachments, choroidal tumors, and staphylomas.

**Practical Advantages of the Indirect Ophthalmoscope**

**Opacities in the ocular media**

Opacities in the ocular media of the patient’s eye, either in the cornea, lens, or vitreous, have a much more deleterious effect on the image seen through direct ophthalmoscopy. With indirect ophthalmoscopy, only a narrow path of relative clarity is required to allow visualization of the retina, and one can generally see through even diffuse opacities such as marked cataractous lens changes. Consequently, the clarity of the indirect ophthalmoscopic image provides a poor measure of a cataract’s density. With the indirect instrument, visually significant macular degeneration may be detected in spite of a dense cataract, perhaps contraindicating lens extraction or at least making the surgeon and patient realistic about the visual prognosis.

**Children and uncooperative adults**

With uncooperative young children and disoriented adults, or those with nystagmus, it is often not possible to examine the fundus with the direct method.
With the indirect ophthalmoscope, since the field of view is so much larger, even a wildly gyrating disc can usually be observed satisfactorily.

**Working distance**
The working distance of the direct ophthalmoscope from the patient’s eye is only several inches, while the indirect is used at almost arm’s length, an important advantage in maintaining a sterile field in the operating room. Children generally react favorably to the more impersonal distance of indirect examination. The greater working distance of the indirect also allows the examiner to shift his attention rapidly from one eye to the other, facilitating a quick and accurate comparison of the two eyes.

**Scleral depression**
One of the main advantages of indirect ophthalmoscopy is the ability to perform a dynamic examination of the peripheral retina using scleral depression, as described later in this chapter.

**Role of Direct Ophthalmoscopy**
Most retina surgeons dilate the pupil and examine the posterior pole of the retina with slit lamp biomicroscopy using a 78 or 90 diopter lens or a contact lens, and they use indirect ophthalmoscopy to examine the periphery. If this equipment and the expertise to use it are available, the direct ophthalmoscope offers no added benefit for the dilated patient. In the undilated eye, the direct ophthalmoscope can be useful in providing a limited view of the posterior pole.

**The Indirect Ophthalmoscopic Image**
In indirect ophthalmoscopy we examine an inverted, real image. An aerial fundus image is formed by the condensing lens close to the focal plane of the lens, between the lens and the observer. The dioptric power of the condensing lens will determine how close the lens has to be held to the patient’s eye in order to produce an image of the fundus—the higher the power, the closer it is held to the eye. The observer wears refractive correction for distance if needed.

The magnification of the image in indirect ophthalmoscopy is little affected by the patient’s refractive error, but is determined primarily by the power of the condensing lens. A +20 diopter lens affords approximately 3X power magnification with approximately a 37° field of view (see Figure 3–2B). The entire fundus area, including the ora serrata and part of the pars plana of the ciliary body, can usually be examined with this method using scleral depression.

**Basic Indirect Ophthalmoscopy Techniques**
Binocular indirect ophthalmoscopy is essential for a detailed and complete examination of the peripheral fundus. However, many good ophthalmologists throughout
the world do not master this technique. The acquisition of proficiency in this method of examination is admittedly difficult and requires many hours of patient practice. Instruction in its use is best obtained from an instructor endowed with great patience. Techniques are presented here to help in developing this important proficiency.

**INSTRUMENTATION**

**Choice of indirect ophthalmoscope**

Many retina surgeons prefer small-pupil indirect ophthalmoscopes for use with all patients. When examining the far periphery, the eye is tilted and therefore the pupillary aperture is tilted and oval, making it in effect a small pupil even if the patient is well dilated.

Good small-pupil indirect devices have two features that improve the performance of the instrument when the pupillary aperture is narrow: First, the size of the patch of light is adjustable. When viewing through a small pupil, a large patch of light provides no more illumination of the fundus than a smaller patch, but it causes additional light reflections that hamper the view.

Second, the proximity of the axis of the incident light and viewer’s line of sight for each eye is adjustable. To obtain a binocular view, three paths of light must be able to enter the patient’s pupil simultaneously: the line of sight of the viewer’s right eye, that of the left eye, and the illuminating beam of light. At the front of the headpiece of the indirect are three mirrors that direct these three paths of light into the eye. A satisfactory indirect ophthalmoscope should provide an adjustment that allows these three mirrors to draw closer to each other for small pupil use or farther apart for use with a well-dilated pupil. In the latter situation, the wide-spread placement of the mirrors allows a greater degree of stereopsis and minimizes bothersome reflections.

**Adjusting the indirect ophthalmoscope**

Indirect ophthalmoscopes consist of a light source mounted on the head, ocular lenses containing plus power, and mirrors to enable the observer to sight down a narrow axis. The eyepieces can be adjusted for individual interpupillary distances. Several screws permit movement of the headpiece on several axes, allowing fine adjustment of the light direction. These details are shown in Figure 3–4.

Much of the usual frustration experienced when first using the binocular indirect ophthalmoscope can be obviated by carefully adjusting the instrument to one’s own head. The headband should fit comfortably on the head without undue tightness, with the top strap taking some of the weight of the instrument. Knurled knobs on the instrument are loosened to allow adjustment of the eye-piece upward or downward and toward or away from the face while simultaneously adjusting the angle that the eye-piece makes with the plane of the face. The field of view is optimized by adjusting the eyepieces as close to the examiner’s pupils or eyeglasses as possible. During this adjustment, it is helpful to shine the light on one’s outstretched hand to ensure that the light centers in the field of view at that distance. One eye and then the other should be closed to check this,
adjusting the interpupillary distance of the oculars as needed. With both eyes open, the light is then adjusted vertically by moving the small knurled shaft that controls the mirror.

A comfortable, single, binocular view of the patch of light should be obtained following these adjustments. The sensation of strain from induced phoria due to faulty adjustment of interpupillary distance causes headaches that might be falsely attributed to the weight of the instrument. An experienced observer rarely notices the weight of the instrument, even after long periods of use. If diplopia persists even after careful attention to the above directions, the examiner should have his fusional amplitudes checked and improved with exercises if indicated.

**Choice of condensing lenses**

The most commonly used condensing lens is a 50-millimeter-diameter double-aspheric +20 diopter lens. The higher the power of the condensing lens, the less magnified the image will be and the wider the field of view. Lower-power lenses have to be held farther from the patient’s eye. A 20D lens gives a good compromise between field size and magnification, and permits a convenient working distance from the patient’s eye.

Lenses of 28-D or 30D power provide a substantial advantage when examining patients with poorly dilating pupils or patients with extremely complicated retinal topography (Figure 3–5). The lowest power that is practical to use in binocular indirect ophthalmoscopy is about 14D, but lenses below 20D are not commonly used. These lenses offer the advantage of higher magnification, but 78 or 90 diop-ter lenses are usually used with a slit lamp when higher magnification is desired (Figure 3–5).

Aspheric lenses minimize image distortion, a difference that is especially pronounced in higher power lenses. All lenses used for indirect ophthalmoscopy should have coated surfaces to reduce light reflexes, as these can be very bothersome.
Care should be taken in cleaning these lenses. The coatings are easily scratched by rubbing a dry lens with tissues not made for lens cleaning. Any dirt or fingerprints on the lens surfaces cause great interference with the view of the fundus, causing more difficulty than if the opacities were present in the vitreous. The condensing lens must be kept scrupulously clean and free of fingerprints.

### Preparation of the Patient

It is useless to attempt to do critical indirect ophthalmoscopy on a patient whose pupil is not widely dilated or who is seated in a lighted room. The patient should be warned that the light is intense but that no harm can come to him or her from this bright light. The patient should understand that pressure on the eyelids from scleral depression may cause some discomfort.

**Pupillary dilation**

The pupil should be dilated to the maximum possible diameter. Mydriatics, such as 2.5% or 10% phenylephrine, used alone are totally inadequate; the moment the bright light is projected into the eye, the strong stimulation to sphincter contraction will overcome the action of the dilator muscle, and the pupil will become miotic. On the other hand, cycloplegics used alone, while much more satisfactory than mydriatics alone, do not give maximum dilation but they do result in a dilation that is not affected by the strong light.

The most satisfactory dilation is achieved by the use of any cycloplegic plus 10% phenylephrine drops. This combination produces a wide and lasting result. The choice of a cycloplegic is determined by how long the examiner wishes the pupil to remain dilated, not by how widely he wishes it dilated.

For routine fundus study, tropicamide 1% plus phenylephrine 2.5% is satisfactory. Cyclopentolate 1% gives longer acting cycloplegia if desired. Scopolamine 0.25%, homatropine 5%, and atropine 1% are less commonly used for routine
retinal exams. Phenylephrine 10% can provide a little stronger mydriasis than 2.5%, but the higher concentration is particularly prone to cause an elevation in blood pressure. Punctal occlusion after instillation is indicated if phenylephrine 10% is used in patients with a history of hypertension.

Maximal dilation may be impossible in some eyes due to posterior synechiae, secondary membranes, sphincter damage, or other causes. The positioning and the fine movements of the condensing lens become critical in such patients, so they are not easy subjects for the inexperienced observer to examine. An experienced examiner can see through a miotic pupil but with increased difficulty.

**Dilation in infants**

When dilating the eyes of infants or young children, care must be taken to avoid systemic complications. Repeated use of cyclopentolate can lead to abdominal distension in infants. Phenylephrine 10% can present high systemic absorption relative to body size in young children and should be avoided. Dilation in the neonatal intensive care unit is frequently performed with cyclopentolate 0.2% combined with phenylephrine 1%.

**Medications to be avoided**

Nothing that might cause corneal hazing should be put in the eye prior to retinal examination. Topical anesthetics such as tetracaine or cocaine often result in epithelial edema and may make the appreciation of fine detail more difficult. Ointments of any kind should not be used, as they make the lids slippery and cause blurring of the image. Indirect ophthalmoscopy should be performed prior to slit lamp biomicroscopy with a contact lens, especially when Goniosol is used.

**Position of the patient**

Optimally, the patient should be positioned lying flat on a reclining exam chair or stretcher (Figure 3–6). Although much of the retina can be examined with the patient in a seated position, the supine position offers great advantages. The examiner has much more flexibility to position his head where needed when he is standing over a supine patient. The examiner’s arms are more relaxed when examining the supine patient, and the patient’s head is more relaxed and stable. The headrest should be about at the height of the examiner’s hips. An adjustable headrest accommodates for dorsal kyphosis and avoids flexion of the neck (see Figure 3–6).

If the examiner wants to check for shifting subretinal fluid, unroll the flap of a superior giant break, or shift an overhanging superior bulla to gain a view of the fovea, it may be helpful to position the patient’s head such that the top of the head is as low as possible. The supine patient’s headrest is lowered below the plane of the reclined chair or table, and the neck is hyperextended to achieve this position.

**Keeping the other eye open and controlling eye movements**

The examiner holds apart the lids of the eye being examined while simultaneously holding the condensing lens. If the patient closes the other eye, Bell’s reflex will cause the examined eye to roll up also. It is useless to have the patient hold the fellow eye open with his hand since this only intensifies the Bell’s reflex. Constant
reassurance and reminding are necessary to keep the patient from allowing the opposite eye to close. As he becomes more light adapted, this tendency decreases. Good cycloplegia is the most important single factor in getting cooperation in this regard, since the eye with inadequate cycloplegia is more photophobic.

Some patients have poor voluntary control of eye movements. In such cases, it is advisable to have the patient hold out his own thumb as a fixation object and look at it (Figure 3–7). The proprioceptive impulses originating from the arm serve to enable even a blind patient to cooperate.

**First Steps in Observing the Fundus**

**Holding the condensing lens**

The first impulse is to hold the condensing lens in the hand with which one ordinarily writes. This may make the initial viewing of the fundus easier but may make
holding the scleral depressor harder. It is frequently advisable to use the writing hand for the scleral depressor and the other hand for holding the lens because it may be relatively difficult for most people to use the scleral depressor in the non-writing hand. Either way, one should consistently use the same hand.

The precise manner of holding the condensing lens is of critical importance (Figure 3–8). It should be grasped between the tip of the flexed index finger and the ball of the extended thumb. The wrist should be flexed moderately and the third, fourth, and fifth fingers should be extended. The extended third or fourth finger is used to hold the upper or lower lid of the patient—which lid depends on the side of the patient one is standing on. The scleral depressor or the thumb of the opposite hand is used to retract the lid not held by the third finger (Figure 3–9).

The extended third or fourth finger acts as a pivot that enables the observer to tilt the lens in all planes merely by rocking the forearm on the tip of the finger. The lens can be moved with critical control closer to or farther away from the eye (see Figure 3–8B) by increasing or decreasing the flexion of the index finger. If the lens is incorrectly grasped between the ball of the index finger or the terminal joint of that finger and the ball of the thumb, it is difficult to make the fine adjustments in lens position so essential to critical scanning of the fundus.

**Bringing the fundus into view**

To obtain the initial fundus view, the light intensity on the scope should be turned down to one-half maximum output by adjusting the transformer. The light should be directed onto the patient’s eye without the condensing lens being in place, to initiate light adaptation. After a few seconds, the patient should be asked to look up so that the light will be thrown onto the superior fundus and away from the macula. The lens is interposed about one inch from the patient’s eye, keeping his
head at arm’s length from the lens. The lens is now slowly moved away from the patient’s eye by increasing the flexion of the index finger. When the lens is at a proper distance away, it will fill with the image of the fundus, which should be clear and striking in its stereopsis. No matter how often one may perform this examination, one is struck by the clarity and beauty of the image of the fundus.

**Troublesome reflexes**

Reflexes from the condensing lens surface may be troublesome (Figure 3–10), particularly for novice examiners. These reflexes correspond to images of the ophthalmoscope light bulb formed by the anterior and posterior condensing lens surfaces. These reflexes can be made to move in opposite directions from each other by slightly
tilting the lens (see Figures 3–10B,C). Thus, they can be moved away from the center of the lens so that they do not obscure the object being studied (Figure 3–11).

**Shifting from one part of the fundus to the next**

Small lateral movements of the lens can be made without losing the image of the fundus filling the lens. Such movements do not enable you to see another field of view. In fact, if the movements laterally are too great, the image is lost. These fine movements bring out parallactic movements of objects in the fundus, and this provides added perception of depth. In order to get from one part of the fundus to another, more than mere movement of the lens is needed.

The entire image system can be considered to consist of a number of vital elements arranged on a straight line (Figure 3–12). These elements are the observer’s macula, the eyepiece of the ophthalmoscope, the center of the condensing lens, the patient’s pupil, and the object observed in the fundus. All of these elements must be kept on a rigid axis or the image is lost. To move from one point in the patient’s retina to another, the entire axis must be pivoted like a lever, with the fulcrum or fixed point being the patient’s pupil. Thus, the observer must move his head and tilt the lens simultaneously by using the third or fourth finger as a pivot. The importance of the proper holding of the lens should be apparent. One must be able to maintain the lens at the correct distance from the patient’s eye and still tilt it smoothly in all directions at the same time. To do this, the pivoting finger must be kept rigid (Figure 3–13).

The coordinated movement of the observer’s head and the tilting of the lens is a maneuver that takes considerable practice. The head is not moved on the neck but, rather, the whole torso is moved from side to side and forward and backward while the lens is appropriately tilted (Figure 3–14). It is essential to practice this maneuver until it is completely natural and automatic.

This activity is frequently a major stumbling block in learning indirect ophthalmoscopy. Some ophthalmologists never master this step and consequently do not continue to progress. In order to see the fundus, these ophthalmologists will have the patient look in one direction and then observe the fundus field in view. They then have the patient look in another direction and again find the image of the fundus and examine it. This results in seeing only isolated areas of the fundus. Not only is

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**Figure 3–10.** (A) With condensing lens held perpendicular to visual axis as shown, anterior reflex and smaller posterior reflex are in center of lens, causing maximum interference with vision. (B) Lens has been tilted to left, causing anterior reflex to move to right and posterior to move to left. Observer can look between them. (C) Reflexes can be moved vertically by tilting lens forward. Anterior reflex always moves in direction of lens tilt, and posterior reflex away from it.
it inevitable that large areas of the retina will be missed in this way, but one cannot also ever gain an overall appreciation of fundus topography, and it makes virtually hopeless the task of scleral depression. It is therefore essential that the “sweeping” of the fundus be mastered. To develop this skill, practice following a vessel from the disc to a point as far anterior as can be seen by the observer’s movements alone should be attempted. This vessel should then be followed back to the disc.

**Orientation and Drawing**

The fact that the image seen in the condensing lens is inverted is confusing at first. This challenge can be overcome by a simple technique.

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**Figure 3–11.** (A) Shows observer viewing area of macula. Lens is shown perpendicular to visual axis. In this position, light reflexes are superimposed and view of macula is obscured. It is necessary to tilt lens with reference to visual axis. (B) Observer is shown viewing equatorial area of eye. Lens is correctly tilted to eliminate effect of light reflexes. Lens should be tilted at a slightly more acute angle to visual axis when observing extreme periphery of eye than when viewing posterior pole to compensate for oblique astigmatism. With experience, this correction is made automatically to clear image.

**Figure 3–12.** Alignment of eyepiece, condensing lens, pupil, and scleral depression on visual axis.
Figure 3–13. Previously described technique of holding lens is shown. Importance of extended third finger is apparent, since it is the pivot on which precise control of rotation of visual axis must depend. In this instance, same finger holds upper lid open, and thumb of opposite hand or scleral depressor held in opposite hand holds lower lid.

Figure 3–14. Axis is formed by examiner’s visual axis (split by prisms in headpiece), condensing lens, patient’s pupil, and area of fundus under study. Fulcrum of this axis is patient’s pupil. In order to observe another part of fundus, observer’s head must move and lens tilt in such a way that the new axis also has its fulcrum at the patient’s pupil.
**Drawing the inverted image**

Just as constant practice of a foreign language is the key to incorporating it into one’s subconscious thought processes, so drawing of the fundus is the key technique to mastery of the inverted image. If you cannot draw a fundus lesion, you do not really see it. It is not necessary to be artistic in any sense to make useful fundus sketches. The important thing is not the sketch itself but the process of making it, which forces one to perceive detail that cannot be interpreted in any other way. The problem of orientation in the fundus will be solved by learning to accurately draw the image that is seen in the condensing lens.

The trick is to take the drawing chart and invert it on the patient’s chest. Figure 3–15 shows the relationship of the fundus chart to the patient’s eye. It will be seen that the 6-o’clock position on the chart faces up toward the patient’s head, with 12-o’clock toward the feet. Obviously, the 9-o’clock and 3-o’clock meridians on the fundus chart are also reversed.

It should now be possible to accurately reproduce the picture that is seen in the condensing lens exactly as it appears, since the image in the lens now corresponds exactly to the drawing chart (Figure 3–16). No attempt should be made to think in terms such as superior, inferior, or temporal or nasal when performing this operation initially. No matter on what side of the patient’s eye the observer stands, whatever appears closest to the observer in the condensing lens (down in the lens) is peripheral in the fundus compared with an object that is farther away from the observer in the condensing lens (up in the lens).

![Figure 3–15. Chart is placed, inverted, on supine patient’s chest. It can be seen that 12-o’clock position on chart corresponds to 6-o’clock position on patient’s eye. Likewise, 6-o’clock position on chart corresponds to 12-o’clock position on patient’s eye. However, when fundus is viewed through condensing lens, inverted image in lens will exactly correspond to orientation of chart. Information in lens image can be directly drawn on inverted chart.](image-url)
Figure 3–16. Left eye is being examined, with inverted image shown. In drawing this image, it should be copied just as it is seen onto an inverted drawing chart. Inverted image is drawn on inverted chart, thus preserving normal relationships.

Position of observer and lesion being examined

One should attempt, when drawing a specific lesion on the fundus chart, to stand 180° away from the entity being observed. This will serve to simplify the observer’s understanding of the proper meridian along which to draw the lesion on the chart. If a lesion at the 11-o’clock position in the patient’s fundus is detected, one should stand inferiorly and look superiorly toward that location (Figure 3–17A). Looking at the drawing chart, one can see that he/she is standing near the meridian being observed, that is, the 11-o’clock meridian (Figure 3–17B). If a lesion is observed at the 3-o’clock position, the examiner will be standing at the 9-o’clock meridian in reference to the patient’s eye but at the 3-o’clock meridian on the fundus chart.

The rule is to draw the image seen in the lens on the part of the fundus chart that is closest (see Figure 3–17). An inverted drawing of an inverted image is created. By repeating this process over and over again, one develops a subconscious familiarity with relationships of fundus landmarks in the condensing lens (Figure 3–18). When a sufficient number of drawings have been made, and for most observers this is about 25, the problem of orientation disappears and one is no longer troubled by the inverted image.

Fundus charts

Drawings of the retinal findings have obvious clinical and legal importance. Standard fundus drawing charts can be obtained from many sources. These charts often include three concentric circles representing the equator, the ora serrata, and the anterior limit of the pars plana (Figures 3–19).

An inherent misrepresentation on any drawing results from reduction of the three-dimensional eye to a two-dimensional drawing. The more peripheral the lesion is, the more substantial this inaccuracy becomes.

Colored pencils or electronic medical record systems capable of capturing color drawings are useful in giving a clear, concise reproduction of fundus pathology.
The colors used for various lesions are not universally standardized, and practices vary from surgeon to surgeon, but each surgeon should establish a consistent color coding practice. Table 3–3 is an attempt to outline some of the most commonly used color codes. Because a color can have more than one meaning and because of variations in the color code, it is important to label items in a retinal drawing.

**EXAMINATION THROUGH A SMALL PUPIL**

*Circumstances presenting a narrow optical aperture*

Some pupils cannot be widely dilated. Posterior synechiae formation from uveitis or prior surgery are common causes of poor pupillary dilation. Damage to the iris from prior surgery or injury may also limit dilation. Chronic use of topical miotics
Figure 3–18. In large figure on top we note direct view of right fundus, which includes superotemporal retinal detachment. Small circles within larger one represent three fields to be studied with indirect ophthalmoscope; labeled a, b, and c. Three circles below large figure (labeled A, B, and C) show appearance in condensing lens of respective fundus fields with image inverted. In large figure on bottom, it should be noted that in view C, ora serrata is found in lower part of lens, since it is most peripheral, and in view A, disc is found in upper part of lens, since it is most posterior. These fields are drawn on an inverted fundus chart as indicated.

Figure 3–19. Chart contains three concentric circles. Inner circle represents equator, middle circle represents ora serrata, and outer circle represents region of ciliary processes. Band between middle and outer circles is pars plana. Small circle in center of chart represents disc. Chart for right eye is usually drawn to the left and chart for left eye is usually drawn to the right, since this represents how the examiner sees the patient, and keeps the relationships of the two drawings anatomically correct relative to each other.
or the presence of an iris-fixated intraocular lens implant may prevent adequate dilation. In an intensive care setting in which observation of pupillary reactions for neurological monitoring is required, pharmacological dilation of the pupil may be contraindicated. Lack of time or medications to dilate the pupil well can necessitate examination through a constricted pupil.

Other conditions can mimic the circumstance of a small pupil. A pseudophakic eye with a small clear aperture through capsular opacities can necessitate small-pupil examination techniques, even though the pupil dilates well. In the presence of irregular media opacities, such as corneal scars, posterior subcapsular cataracts, and patchy vitreous hemorrhage, small-pupil examination techniques can be of great assistance. When examining in the far periphery, visualization must occur through a very tilted pupillary aperture, which also mimics the condition of a small pupil.

**Indirect viewing systems**

Indirect visualization systems are generally those which result in an inverted image. These include the indirect ophthalmoscope, slit lamp biomicroscopy through a 60- to 90-diopter noncontact lens or an indirect contact lens, and wide-angle contact and noncontact surgical viewing systems. (In the surgical setting, the indirect image is reinverted back to an erect image.)

Indirect systems generally provide remarkable visualization in spite of a narrow optical aperture. When presented with a small pupil, an indirect system provides a substantial advantage.
Using the indirect ophthalmoscope with a small pupil

Examination through a small pupil can challenge the most experienced observer. Before attempting small-pupil examination, establish proficiency with the basic techniques of indirect ophthalmoscopy in well-dilated eyes.

Using the following techniques of indirect ophthalmoscopy can dramatically improve visualization through a narrow optical aperture:

1. Use a small-pupil indirect ophthalmoscope (see “Choice of Indirect Ophthalmoscope” on page 47).
2. Use a higher-power condensing lens, such as a 28- or 30-diopter lens.
3. Reduce the size of the light patch from the indirect device.
4. Narrow the distance between the optical apertures.
5. Make sure the condensing lens is clean with intact anti-reflective coatings.
6. Examine at full arm’s length from the patient.
7. Minimize the light intensity if the pupil is reactive to light.
9. Optimize alignment of the optical system.
10. Examine in a dark room.

Using the indirect laser with a small pupil

Laser systems connected to an indirect ophthalmoscope present a significant advantage in that they allow delivery of laser treatment to the far periphery of the retina. They also improve treatment through a small pupil.

Indirect ophthalmoscopic lasers add an additional optical pathway to the visual system: the path of the laser beam. This path must also fit through the entry pupil in order to apply laser to the retina and avoid misdirected laser applications.

Lasers differ significantly in their ability to treat through a small pupil, and also in their ease of use in the far periphery. Select a laser that has a highly coaxial optical system, meaning that the angle of entry of the laser approximates the angle of the viewing and illumination axes.

This can be tested with the indirect headset in place and the illumination light and laser aiming beam turned on. Shine these lights on an outstretched hand while viewing through the oculars of the headset. Move the hand closer to and farther from the eyes. The less the aiming beam of the laser moves up and down in the field of view during this maneuver, the more coaxial the laser system is. Lasers that are mounted to small-pupil indirect ophthalmoscopes are also advantageous.

When lasering the peripheral retina, a horizontally oval pupillary aperture presents. Delivery of laser to the periphery can be facilitated by tilting the examiner’s head horizontally to align with the widest dimension of the pupil.

SCLERAL DEPRESSION

Only when the student has mastered the technique of fundus drawing is he/she ready to attempt scleral depression. If the student has not mastered the problems
of moving the hand–head axis, he cannot possibly line up an additional element, the depressor, on the axis (see Figure 3–12).

**PURPOSES OF SCLERAL DEPRESSION**

Scleral depression has two main functions. The first is to make visible that part of the fundus that lies anterior to the equator. This region is ordinarily not visible at all without the use of scleral depression. A second and perhaps even more important function is to enable the observer to “palpate” the retina, and to examine the peripheral retina dynamically and from multiple angles in ways that can only be appreciated with scleral depression.

The technique enables visualization of a retinal flap as it rises from the crest of the indenter mound (Figure 3–20). A red spot might be shown to be a retinal break instead of a retinal hemorrhage (Figure 3–21); an ill-defined gray area might be revealed as an excavation; a white spot might turn out to be a tuft of tissue rising from the retinal surface; and so on. A raised lesion can be differentiated from a depressed lesion, and a hemorrhage or foreign body can be determined to lie on or anterior to the retina.

**TECHNIQUE OF SCLERAL DEPRESSION**

**Choice of depressor**

Scleral depressors are metal or plastic shafts with knob-like tips of two different sizes on each end (Figure 3–22). The broader tip is suited for more anterior depression, while the narrower tip allows more posterior depression. An alternative is a small, curved shaft and tip mounted on a thimble. It can be held between the

![Figure 3–20. Flap of retinal tear, clearly seen as it arises from crest of mound created by scleral indenter. (Reproduced with permission of Medcom, Inc., from Conor O’Malley, MD, FACS, and Patrick O’Malley, MD, FACS: “The Peripheral Fundus of the Eye.”)]
thumb and index finger, or it can be placed upon the index or middle finger as shown in Figure 3–23.

**Initiating scleral depression**

Using one common method, the scleral depressor is grasped between the thumb and the index finger, and the patient is asked to look down. The depressor is applied to the upper lid, without pressure, at the posterior tarsal margin (Figure 3–24A). The patient is then asked to look up and, as the upper lid retracts, the depressor is slid posteriorly, tangential to the surface of the globe (Figure 3–25).
Figure 3–22. Scleral depressor.

Figure 3–23. Two techniques for manipulating thimble scleral depressor are shown. (A) Shows method that is easier to learn for most people because it closely resembles the way pencil or ophthalmic surgical instrument is held. (B) This method is better when third finger is needed to hold patient’s lid.

Figure 3–24. (A) Shows depressor being applied to posterior tarsal margin of patient’s upper eyelid, with patient looking down. (B) With patient looking up and depressor introduced, examiner is in position to see superior periphery.
The depressor now lies against the globe, but no pressure is being applied to the globe. The light from the ophthalmoscope is projected onto the fundus superiorly, the condensing lens is interposed, and the fundus at the equator is brought into view (Figure 3–24B). The depressor is now gently pressed against the globe at the equatorial region and, if it is applied in the correct meridian, a grayish mound comes into view from the lower part of the lens. A small adjustment in the lens should bring into clear focus the image of this indented part of the fundus. Slowly, the depressor is slid anteriorly under direct visual control. The ora serrata should slide into view in the inferior part of the lens.

**Moving the axis of depression**

Once the image of the indented part of the fundus is in view, one moves the depressor, the lens, and the observer’s head in a coordinated manner to move from one part of the retinal periphery to another. It is important to emphasize that the observer’s eye, the eyepiece of the ophthalmoscope, the condensing lens, the patient’s pupil, the object being studied in the fundus, and the scleral depressor all must lie on a straight line as indicated in Figure 3–12. The depressor should be applied in a direction as parallel to this axis as possible. It is not satisfactory to depress with the instrument at a great angle to this viewing axis.

**Causes of discomfort**

The amount of pressure used for scleral depression is about the same as that used when estimating intraocular pressure by taction. Most beginners use excessive pressure. When poor alignment precludes a view of the depressed retina, the novice usually presses more vigorously, resulting in patient pain and lack of cooperation. Instead, the examiner should assess the alignment of the depressor with the other elements of the optical system. If the depressor is aligned correctly, little or no intentional pressure on the eye is needed to visualize the mound of the depressor. Too much pressure causes discomfort to the patient, who subsequently
squeezes the eyelids, and visualization may become impossible. A person who is skilled with this technique can examine even young children without causing significant discomfort.

Frequently the beginner applies his depressor too far anteriorly. This causes considerable discomfort to the patient and, of course, will not permit visualization of the retina.

**Scleral depression in the horizontal meridia**

In the majority of instances, scleral depression should be performed through the lids. Even the 9-o’clock and 3-o’clock meridians can be examined in most patients by careful application of the depressor on the superior lids (Figure 3–26). If the lid is lax, it can be dragged down slightly with the depressor to enable these meridians to be examined. In some cases, however, it may be necessary to examine the 9-o’clock and 3-o’clock meridians by direct application of the scleral depressor to the bulbar conjunctiva (Figure 3–27). If this is necessary, it will be observed that even less pressure is necessary to see the fundus than if one applies the pressure through the lids. A drop of a suitable topical anesthetic will enable this maneuver to be performed. In sensitive patients, a cotton-tipped applicator soaked in topical anesthetic can be used as the depressor. Since topical anesthetics cause corneal epithelial cloudiness, this portion of the examination should be performed after the majority of the fundus examination has been completed.

It is never necessary to use a topical anesthetic when applying a scleral depressor to the lids. The amount of pressure necessary to see the superior periphery is

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**Figure 3–26.** (A) Shows the approximately six positions of depressor on lids sufficient to provide view of entire periphery, with exception of 9- and 3-o’clock positions. (B) Shows how, in most patients, it is possible to drag upper lid down enough to visualize nasal horizontal meridian. (C) Shows how it is usually possible to drag upper lid down enough to examine temporal horizontal meridian.
somewhat less than that necessary to see the inferior periphery due to the somewhat greater thickness of the inferior lid.

**Scleral depression nasally**

When performing scleral depression in the far periphery in temporal meridia, it helps to have the patient look in a temporal direction. However, this is not so when examining in nasal meridia. If the patient looks in a nasal direction, the nasal peripheral retina becomes buried into the bones of the orbit. Rather than having the patient look in a nasal direction, turn the patient’s head about 30 degrees nasally in order to perform scleral depression in the nasal periphery.

**Examining the far periphery**

In examining the ora serrata region, it is usually necessary to have the patient look toward the meridian one wishes to examine. However, it is important to emphasize the necessity for the observer to move his own body freely in examining the retinal periphery. In examining the ora serrata, it is usually necessary to tilt the condensing lens somewhat forward into a plane more nearly parallel to the iris. With this technique, it should be possible, in an eye whose pupil dilates normally, to examine the ora serrata for 360° and the posterior one third of the pars plana ciliaris.

**Rolling the depressor**

It must be emphasized that scleral depression is a dynamic and not a static technique. Significant help in interpreting fundus pathology is obtained by changes
in the image produced by movements of the scleral depressor. The experienced observer is constantly moving the depressor with fine massaging movements anteriorly, posteriorly, and also sideways. These slight movements enable the observer to examine lesions from many angles. This technique is sometimes referred to as “rolling” a lesion. It is this dynamic aspect of scleral depression that enables one to pick up lesions in the fundus that are invisible without scleral depression.

While performing scleral depression, a faint circumferential white line, which marks the posterior aspect of the vitreous base, can frequently be observed. A break in this line should be carefully “rolled,” inspecting it as a possible retinal break (Figure 3–28).

The use of depression allows the examiner to detect and differentiate raised lesions from flat lesions and to determine the difference between a hemorrhage and a retinal break. It also enables one to detect a retinal break that may not be visible until the depressor is applied under it (Figure 3–29A,B). Figure 3–30 illustrates how the movement of the depressor produces changes in the fundus mound, which in turn alter the view of the lesion in question. Figure 3–30A shows the appearance of the lesion without the use of the scleral depressor. Figure 3–30B shows the appearance of the problem with the scleral depressor in place, with the apex of the mound at approximately the equator. Well down on the anterior slope of the mound, a round red lesion that is difficult to identify can nevertheless be detected. After the depressor is slid anteriorly, the nondescript red spot is brought up to the crest of the mound. At this point, it is recognized as a break in the retina (see Figure 3–30C). The depression has blanched the margins of the retinal

![Figure 3–28. Retinal tear interrupts white line of posterior vitreous base.](image)
break and increased the contrast between the edges of the break and the subjacent choroid.

**Sequence of scleral depression examination**

In order to be certain that no area of the fundus escapes evaluation, a system of examination is essential. Otherwise, lesions of considerable size and importance may be overlooked. One can start by examining the fundus posterior to the

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**Figure 3–29.** Elevated lesion seen in (A) is confirmed to be a retinal tear by moving the depressor, as seen in (B).
equator in all quadrants, and then proceeding to a 360-degree depressed examination of the periphery beyond the equator.

Since scleral depression nasally is more difficult than temporally, it helps to examine the temporal periphery first. This allows the patient time to accommodate to the bright lights, and also allows scleral depression to soften the eye a little in preparation for examination nasally. If the examiner is standing on the right side of the patient, it is convenient to begin by examining the left eye first. Starting at 12 o’clock and moving clockwise, scleral depression of the left temporal periphery is performed. To examine the 12-o’clock periphery of the left eye, the examiner stands to the right side of the supine patient’s legs, with the patient looking up. Examining around the 3 o’clock periphery, he/she stands to the right of the patient’s head, patient looking left. Moving to examine at 6 o’clock,
he/she has shifted position to stand at the top of the patient’s head as the patient looks down. As he/she starts to examine inferonasally, the patient’s head is turned about 30 degrees in a nasal direction, with the patient still looking down rather than down and right. The 9-o’clock meridian is examined with the patient looking straight ahead, with the examiner standing to the left of the patient’s head. The patient looks up for the superonasal exam, and when exam is completed back to 12 o’clock, the examiner is standing to the left of the patient’s legs. He/she is then in position to begin examination of the right eye, starting at 12 o’clock and moving temporally to 9 o’clock. The exam continues as with the left eye, this time turning the head to the left to examine the nasal periphery. When examination of the right eye is completed, the examiner is back at the right of the patient’s legs where the process began.

If the primary pathology is in the left eye, it may be preferable to begin with the exam of the right eye, allowing time for the examiner to dark adapt and the patient to get used to the exam.

**Scleral Depression in the Operating Room**

Scleral depression in the operating room is usually performed with a marking depressor with one end designed to leave a mark by temporarily dehydrating the sclera (Figure 3–31). The side of the marking tip is used to locate the retinal break. Then the shaft of the depressor is turned into position directly over the tear, or over the edge of the tear that one wishes to mark. The scleral surface is then exposed, the mark is found, and a marking pen is used to make the mark permanent. (See also Chapter 7, page 155.)

Some surgeons prefer diathermy for marking retinal tears. The fundus lesion is first localized with a cotton-tipped applicator or scleral depressor. Some surgeons then have an Arruga retractor introduced by an assistant to straddle the location. The cotton stick or depressor is then removed, without moving the eye. A flat diathermy electrode is then introduced under ophthalmoscopic control, and scleral depression is performed with it until the mound in the fundus caused by the electrode is positioned to the examiner’s satisfaction. The current is then turned on

![Figure 3–31. Marking scleral depressor.](image-url)
and a burn on the sclera results. If the retina was not too highly detached, a retinal burn will also result, corresponding exactly to the scleral burn.

**SUMMARY**

Indirect viewing systems present an inverted image but enable high-resolution, wide-field binocular imaging extending into the far periphery of the retina. With appropriate techniques and equipment, excellent visualization may be possible, even with poor pupillary dilation or media opacities.

Mastery of the skill of binocular indirect ophthalmoscopy with scleral depression is essential for examining the peripheral retina, and diagnosing and treating retinal detachment. The dynamic technique of scleral depression, how to examine through a small pupil, and ways to ensure patient comfort and cooperation are presented.

**SELECTED REFERENCES**


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Evaluation of a patient for retinal detachment includes a thorough history and a complete ocular exam, including measurement of visual acuity, external examination, ocular motility testing, testing of pupillary reactions, anterior-segment biomicroscopy, tonometry, and binocular indirect ophthalmoscopy with scleral depression. Posterior-segment biomicroscopy, perimetry, and ultrasonography are also sometimes required.

OCULAR EVALUATION

Rhegmatogenous retinal detachment is a diagnosis generally made by clinical examination of the retina alone, but a full history, ocular examination, and sometimes selected ancillary tests are also important parts of the evaluation (Figure 4–1).

HISTORY RELEVANT TO RETINAL DETACHMENT

The symptoms of retinal detachment include flashes of light, new floaters, visual field defect, decreased visual acuity, metamorphopsia, and rarely, defective color vision.

Flashes of light

The perception of light flashes, or photopsia, is due to the production of phosphenes by pathophysiologic stimulation of the retina. The retina is activated by light but is also capable of responding to mechanical disturbances. In fact, the most common cause of light flashes is posterior vitreous detachment. As the
vitreous separates from the retinal surface, the retina is disturbed mechanically, stimulating a sensation of light. This perception is more marked if there are focal vitreoretinal adhesions. Generally, vitreous separation is benign and may almost be regarded as normal in the senescent eye. In approximately 12% of symptomatic posterior vitreous detachments, however, a careful search of the periphery reveals a tear of the retina.

If the flashes are associated with floaters, it is wise to assume that a retinal tear exists, until proved otherwise. These symptoms demand a prompt and careful examination of the periphery with binocular indirect ophthalmoscopy and scleral indentation. The patient’s localization of the photopsia is of little value in predicting the location of the vitreoretinal pathology. If no breaks are evident in the first examination after symptomatic vitreous detachment, they rarely appear at a later date.

If there is no associated hemorrhage or other pathologic condition, the patient needs counseling only. However, if pigment or blood is detected in the vitreous, a follow-up examination is often required. It is prudent to forewarn patients about the symptoms of retinal detachment. Flashes alone or floaters alone are less significant than if they occur together, in which case they are more likely to be associated with a retinal break.

The light scintillations of an ocular migraine are correctly identified by the typical history. However, ophthalmoscopic examination may be required to rule out a retinal cause.

**Floaters**

While a few floaters (muscae volitantes) are experienced almost universally by the general population, the acute onset of new floaters requires careful examination. A patient describing hundreds of tiny black specks is virtually pathognomonic of vitreous hemorrhage. The sudden appearance of one large floater near the visual axis is ordinarily caused by posterior vitreous separation in which the glial annulus
(Weiss’ ring) of the posterior vitreous has pulled loose from its peripapillary location. The appearance of numerous curvilinear opacities within the visual field generally indicates vitreous degeneration. Small vitreous collagen fibrils resulting from degeneration coalesce into large, coarse fibers, which cast curvilinear shadows on the retina.

The most significant cause of floaters is vitreous hemorrhage, and the characteristic numerous tiny black dots may be followed in a few hours by “cobwebs” as the blood forms irregular clots. The cause of acute vitreous hemorrhage must be assumed to be a retinal tear, until proved otherwise. There is a direct relationship between the amount of vitreous hemorrhage and the likelihood of a retinal tear being present.

If the superior fundus cannot be adequately visualized, bilateral patching should be considered. The patient should be provided with patches for both eyes and told to apply them with tape when going to bed. They should not be removed until the patient returns for reevaluation the following morning, so a caregiver must be involved in this process. The eye should then be promptly dilated, and the patch should be immediately reapplied after the drops are instilled. Examination is carried out following adequate dilatation. This practice will usually allow blood to sufficiently settle so that the superior half of the retina can be adequately observed; this is where the great majority of breaks occur.

Although examination by ultrasonography is frequently recommended in this situation, detection of a retinal tear without detachment is a genuine art not perfected by most. If a retinal detachment is discovered in an eye with a poor view that prevents discovery of a retinal tear, the eye should be managed with vitrectomy. Otherwise, continued patching should be considered, but if this is unsuccessful after another 24 hours, serial ultrasound studies can be considered.

**Visual field defects**

Awareness by the patient of a visual field defect or shadow is often the first symptom of post-equatorial retinal detachment. Detachments anterior to the equator have no effect on the visual field and cannot be demonstrated with confrontation visual fields or perimetry. Detachments posterior to the equator can usually be so demonstrated, but patients rarely notice a field defect until the detachment encroaches on the posterior pole. Patients tend to be less aware of superior than inferior field defects, and those patients with inferior retinal detachments may be totally unaware of superior field loss until the fovea is involved. High bulbar detachments cause dense field defects; flat detachments produce relative field defects.

Visual field examination is highly specific for localizing retinal detachments. When a patient with extensive retinal detachment is being examined, it is frequently helpful to inquire about the position of the field defect when first seen, as a clue to the location of the primary retinal break. For instance, if a patient with a total retinal detachment has a history of an inferior nasal field defect that later enlarged to involve the total field, the finding of a retinal break in the superior temporal periphery is likely.
Increased central acuity

Although the duration of retinal detachment symptoms is estimated from the time of the onset of the visual field defect, the duration of foveal involvement dates from the time of decreased central acuity. The prognosis for recovery of central vision is approximately correlated with the duration of foveal detachment. Even a brief detachment of the fovea will usually permanently reduce the best corrected visual acuity after successful reattachment to the level of about 20/50 (6/15). Therefore, a retinal detachment threatening imminent detachment of the fovea should either be treated urgently, or the patient should be bilaterally patched until surgery can be scheduled. Unfortunately, peripheral field loss may escape a patient's notice, so that medical help is frequently not sought until central vision is lost.

Trauma

The patient should be queried about a history of trauma, either accidental or surgical. The examiner should carefully record the details of trauma in the patient's chart, remembering that it is a legal document that might later be reviewed. The time, place, and nature of the accident should be entered. Direct trauma to the globe should be clearly differentiated from any indirect trauma to the head or elsewhere in the body. Details of previous surgery should be noted, particularly cataract extraction, Nd:YAG capsulotomy, intraocular foreign body removal, and retinal procedures. Frequently, a patient denies any trauma during the initial examination but recalls some remote trauma a few weeks later. The denial and recollection should be recorded under the date that each is reported by the patient.

Ocular diseases

The history must include questions regarding previous ocular diseases, such as uveitis, vitreous hemorrhage, amblyopia, glaucoma, or diabetic retinopathy. Any symptom of retinal detachment can be mimicked by other disease processes. Light flashes might be due to posterior vitreous detachment, floaters might be due to age-related vitreous degeneration or uveitis, and visual field defects might be due to vascular occlusion or vitreous hemorrhage.

Systemic diseases

A careful history of the patient’s general health includes specific questions about systemic disorders that are sometimes associated with retinal detachment, including diabetes, tumors, angiomatosis of the central nervous system, sickle cell disease, leukemia, and eclampsia.

Family history

Although most retinal detachments occur as sporadic events, certain families are susceptible to retinal detachment. A family history of retinal detachment is a clue to prognosis and frequently indicates the need for examination of other family members. The history influences decisions regarding prophylactic treatment of retinal breaks.
**GENERAL OCULAR EXAMINATION**

**Visual acuity**
The best corrected visual acuity should be recorded for each eye. Generally, the patient’s current eyeglasses or contact lenses suffice, but if there is any doubt, a refraction should be performed. Visual acuity is usually measured with the Snellen test. Low vision may be recorded with a decreasing numerator of the Snellen fraction, such as 5/200 or 2/200. Because the examiner’s fingers are grossly equivalent to the 20/200 letter, counting fingers at a distance of 5 feet is approximately equivalent to 5/200. The visual acuity of illiterate or non-English-speaking patients or of preliterate children can be measured with the E chart.

Decreased visual acuity always accompanies extension of the retinal detachment into the fovea, but it does not necessarily mean detachment of the fovea. Poor central vision may also be due to antecedent disease of the macula, opacities of the media, optic nerve disease, or amblyopia.

**External examination**
The examiner should record the status of the brows, lashes, and lids. Accurate recording of the preoperative state of these structures is a requisite base for postoperative evaluation of the external anatomy. The early postoperative period may be characterized by pseudoptosis, lid edema, chemosis, and, rarely, permanent ptosis.

**Ocular motility**
Retinal reattachment surgery is occasionally accompanied by temporary changes in the function of the extraocular muscles, and in rare cases the alterations are permanent. Therefore, a preoperative record of the state of ocular motility is important. Although there are many tests of motility, the cover test and versions usually suffice. But when the vision of one or both eyes is poor, the cover test is not reliable. In such a case, the examiner should simply note the position of the light reflection of the flashlight with reference to the visual axis of each cornea (Hirschberg test). Each millimeter deviation of the corneal light reflex from the visual axis is equivalent to approximately 12 prism diopters ($\Delta$). This test can be refined by selection of the appropriate prism to restore the light reflex to the normal visual axis (Krimsky test).

**Pupillary reactions**
Pupillary reactions should be noted, and the size of the maximally dilated pupil recorded. Subtle differences in the direct light reflex might not be apparent unless a comparison is made between the two eyes with a swinging flashlight (Marcus Gunn test), by which the direct pupillary reflex is studied in comparison to the consensual reflex. A test result is positive when the pupil dilates as the light is directed into it, indicating that the direct reflex is weaker than the consensual. A defect in the afferent pupillomotor system, including retinal detachment, resulting in a positive test result is referred to as an *afferent pupillary defect*. 
**Anterior segment biomicroscopy**

The cornea is usually clear in retinal detachment, but occasionally there is enough hypotony to create folds in Descemet’s membrane. Anterior uveitis is rarely sufficient to produce keratic precipitates. Mild flare and cells are frequently noted in the anterior chamber, and occasionally the reaction is marked. The depth of the anterior chamber should be noted. The angle of the chamber can be estimated with the slit-lamp beam near the limbus. If the peripheral anterior chamber seems unusually shallow, gonioscopy is indicated.

Any opacities of the lens should be noted. An objective assessment of the effect of opacities on visual acuity is more accurate with use of the direct ophthalmoscope. A posterior subcapsular cataract might interfere with visual acuity, but it often does not prevent a thorough examination of the fundus periphery. On the other hand, peripheral cortical opacities may not interfere with visual acuity, but they may seriously impair examination of the periphery and conceal the presence of peripheral retinal breaks. The vitreous should be examined for evidence of vitreous detachment, hemorrhage, inflammatory cells, or pigment.

If there has been no previous intraocular disturbance (such as uveitis, trauma, or intraocular surgery), the presence of pigment in the vitreous (“tobacco dust”) is very suggestive of a retinal break. Pigmented cells from the retinal pigment epithelium may pass through a retinal break into the vitreous cavity. Retinal breaks have been found in more than 70% of eyes with “tobacco dust” if there is no other obvious explanation for the presence of pigment in the vitreous.

The indirect ophthalmoscope can be used for examining the anterior segment when a slit lamp is not available. With the examiner’s eyes positioned just 8 to 10 inches from the patient, the condensing lens functions as a magnifying loupe that provides an erect real image.

**Tonometry**

The intraocular tension should be recorded for both eyes before the pressure is artificially lowered by the massage effect of scleral indentation (scleral depression). Usually, an eye with retinal detachment is relatively hypotonic, and the pressure may be as much as 10 mm Hg less than the unaffected eye. Occasionally, the hypotony is so profound that no tension can be recorded. Still rarer is the patient who has a paradoxically elevated intraocular pressure in the presence of retinal detachment (Schwartz syndrome). Either of these extremes is usually relieved by retinal reattachment.

**RETINAL EXAMINATION**

**Binocular Indirect Ophthalmoscopy**

Retinal surgeons rely primarily on the binocular indirect ophthalmoscope for diagnosing retinal detachment. Its main features are contrasted with those of the direct ophthalmoscope in Table 3–1. Despite its low magnification and inverted image, the binocular indirect ophthalmoscope is the instrument of choice due to its large
field of view, high illumination, depth of focus, stereopsis, and especially its ease of use with the scleral depressor. The technique of binocular indirect ophthalmoscopy and scleral indentation is discussed in Chapter 3.

**POSTERIOR SEGMENT BIOMICROSCOPY**

Biomicroscopic examination of the posterior segment is accomplished by the use of a flat macular contact lens, a mirrored contact lens (Figures 4–2 and 4–3), an indirect wide-field contact lens, or a 60- to 90-diopter noncontact indirect lens. The Hruby noncontact technique has been superseded by the noncontact indirect lenses. The biomicroscope provides stereopsis, high illumination, high magnification, and the great advantage of a slit beam optical section. Routine use of biomicroscopy of the peripheral retina is not required for every retinal detachment, but in selected cases it reveals valuable information not obtainable by any other method.

Through the flat central portion of a contact lens, it is possible to examine the central and posterior vitreous with high resolution. There is no better way to examine the optic nerve head and the fine details of macular anatomy. The technique is particularly valuable in the search for posterior retinal breaks, which are particularly difficult to locate in the staphyloma of highly myopic eyes or in the retinal detachment of proliferative diabetic retinopathy.

**Figure 4–2.** Goldmann’s three-mirror lens. (Reproduced with permission from Cockerham WD, Schepens CL: Technique of vitreous cavity examination. In: Symposium on Retina and Retinal Surgery [Transactions of the New Orleans Academy of Ophthalmology]. St Louis: CV Mosby Co; 1969:66–89.)
Vitreoretinal relationships in the periphery can be examined with mirrored lenses or with indirect lenses. Mirrored lenses have the advantage of a shallow depth of focus, which is valuable when a questionable retinal break cannot be clearly defined by ophthalmoscopy. By precise focus on the sensory retina, the presence of a break can be detected. With difficulty, scleral depression can be performed in combination with biomicroscopic evaluation of the peripheral retina (Figure 4–4).

Biomicroscopy is the best way to define the critical role of the detached vitreous cortex in proliferative diabetic retinopathy. The mechanism of elevation of previously flat neovascularization, avulsion of retinal vessels, and tractional retinal detachment can be clearly seen.

Biomicroscopy is also valuable for the preoperative evaluation of a patient with a giant retinal tear. The possible attachment of formed vitreous to the flap of the tear or the presence of formed vitreous behind the retina is a preoperative clue to the prognosis, and suggests the best technique of surgical management.

Binocular indirect ophthalmoscopy should be employed first to obtain a panoramic view of the entire posterior segment. Contact lens biomicroscopy can then be used to define specific details; that is, the indirect ophthalmoscope can be used to see the “forest,” and the slit lamp to see the individual “trees.” Exclusive reliance on slit lamp examination of the retina is not recommended.
ANCILLARY TESTS

Perimetry
Most cases of retinal detachment can be evaluated adequately without perimetry, but there are certain instances in which it is helpful, and others in which it may be important. The visual fields should be examined by confrontation, and if there is any question about the correlation of the field defect with the detachment, formal perimetry may be helpful. Perimetry is particularly indicated if there is antecedent disease of the optic nerve, particularly glaucomatous cupping.

Perimetry may also be helpful in the differential diagnosis of retinoschisis. Relatively flat retinoschisis can be readily differentiated from shallow retinal detachment in that the former invariably causes an absolute field defect, while the latter causes only a relative defect. Perimetry is less specific for bullous detachment, because an absolute field defect could be found in either detachment or retinoschisis. Perimetry may be helpful with a miotic pupil. As previously mentioned, perimetry only discloses disease posterior to the equator.

Ultrasonography
When confronted with opaque media, the physician can obtain valuable information with ultrasonography—both A-scan and B-scan. The technique can reveal both rhegmatogenous and nonrhegmatogenous detachments, such as those secondary to malignant melanoma of the choroid (Figure 4–5). Retinal tears in the absence of retinal detachment can often be detected as well.
A detached retina always remains attached at the optic nerve. This feature of insertion at the shadow of the optic nerve is easily observable with B-scan and helps distinguish retinal detachment from posterior vitreous separation or vitreous hemorrhage (Figure 4–6). A standardized A-scan can also be helpful in making this distinction.

**Laser test**

The distinction between retinal detachment and retinoschisis can usually be made by retinal examination alone. However, at times this can be a difficult diagnosis to make; especially when retinal detachment and retinoschisis coexist, it can be difficult to tell the extent of each.

The laser test can be helpful in this instance. Laser intensity is adjusted to create a medium-intensity laser spot in normal retina. This same intensity is then applied to the area of the retina in question. Where retinoschisis is present, a white spot will result, but where retinal detachment is present, there will be no visible reaction to the laser.
Patient Counseling

Once the diagnosis of rhegmatogenous retinal detachment has been made and full examination has been performed, the patient and family are counseled regarding the nature of the diagnosis and its planned treatment. Informed consent is both a medical and a legal requirement, and an informed patient is apt to be more cooperative and better reconciled to the therapeutic options and outcome.
The prognosis is presented, including the possible need for reoperation and the possibility that even with reoperations the retina may not ultimately be repairable. If the macula is not detached, the physician should explain that some patients (5%–10%) may lose a few lines of visual acuity. If the macula is detached, the ophthalmologist should explain that full visual recovery should not be expected.

Surgical and nonsurgical alternatives are reviewed. The major complications of retinal detachment surgery (including the rare possibility of blindness) should be mentioned, with a thorough discussion if the patient desires. The patient’s financial obligations should be explained.

**URGENCY OF SURGERY AND MACULAR DETACHMENT**

The term “macular detachment” is usually used to refer specifically to detachment of the center of the macula, the fovea. If macular detachment occurs or has occurred, even if only briefly, the visual acuity will usually not return to 20/20 (6/6). Average best corrected visual acuity is about 20/50 (6/15), but the range of outcomes is quite broad.

If the macula has not yet been detached, a judgment should be made about the imminence of this adverse event. Proximity of the detachment to the fovea, degree of bullousness of the detachment, direction from which the detachment approaches the macula, size and location of the retinal break(s), and duration of symptoms are all factors that are considered in making this judgment (Figure 4–7).

The rate of extension of detachment tends to be higher if the break is superior or temporal, and lower if the break is inferior or nasal. Equatorial tears tend to cause more rapid progression than tears near the ora serrata. Large retinal tears are usually associated with a more rapid progression of detachment than small

![Figure 4–7. Bullous retinal detachment threatening to detach the macula (M). (Reproduced with permission from Pneumatic Retinopexy: A Collaborative Report of the First 100 Cases: Hilton et al. Ophthalmology 1987;94:307–314.)](image_url)
tears. However, even large dialyses in the young tend to progress slowly. Bullous detachments tend to progress more rapidly than shallow ones.

If there is a risk that a currently attached fovea may soon detach, prompt surgical intervention is in order. Pneumatic retinopexy has a possible advantage in this instance since, as an office-based procedure, it can usually be performed sooner than an operating room procedure. Also, pneumatic retinopexy offers the opportunity to use the injected gas bubble to press the detachment away from the fovea by appropriate positioning (see “Steamroller” in Chapter 8, page 196).

Until surgery can be performed, progression of the detachment can be slowed, halted, or even reversed by having the patient stay at bed rest with both eyes patched. Some surgeons believe that if the detachment is superior, the head of the bed should be flat and the patient’s head should be turned to the side that makes the detachment lowermost. Such measures can buy some needed time while preparations are made for surgery. If a nondrainage procedure is being considered, bilateral patching before surgery may also be helpful to encourage the absorption of subretinal fluid.

Normal visual acuity rules out the existence of a detached macula, but poor vision does not prove that the macula is detached. Poor vision may also be due to antecedent macular or optic nerve disease, amblyopia, or opacities of the media. Subtle degrees of macular detachment are best evaluated stereoscopically with the slit lamp and contact lens and with ocular coherence tomography (OCT) if necessary. The luteal pigment of the macula, xanthophyll, is not readily apparent in the normal macula, but becomes more evident as a yellow color against the background of intraretinal edema when detachment includes the macula.

Macular detachment is not usually due to or associated with macular holes. Macular breaks as a cause of retinal detachment are usually seen in association with the staphyloma of high myopia or following trauma (Figure 4–8).

Figure 4–8. Retinal detachment caused by macular break in high myopia.
If the macula is already detached, the urgency is not high, but slow deterioration of the detached retina occurs over time. Extent and elevation of the detached retina also usually progress, potentially making the surgical procedure more difficult. Some cases are chronic and less urgent, but for most macula-off retinal detachments we prefer to schedule surgery within a week.

**Complicating Preexisting Conditions**

**Managing miosis**

Poor pupillary dilation may occur because of posterior synechiae or chronic miotic use. Visualization of the retina may be sufficiently impaired that examination and surgical repair of the detachment is compromised. Use of small-pupil examination techniques will often help (see “Examination through a Small Pupil” in Chapter 3), but sometimes this is insufficient.

One very effective solution to this problem is the intraoperative placement of iris retracting hooks, usually used in conjunction with vitrectomy. Four small incisions into the anterior chamber are made at the limbus at the points of a square. The prolene hooks are inserted to catch the edges of the iris and retract it outward.

Another option is photocoagulation of the retina, or photomydriasis. Laser application in at least eight meridia is sometimes sufficient to achieve adequate dilation. Laser spots are placed midway between the pupillary margin and the iris root, causing contraction of the dilator muscle. If pupillary fibrosis is present, it may be amenable to Nd:YAG laser transection.

The effect of pupillary dilation is enhanced by subconjunctival injection of a mydriatic-cycloplegic combination. Care should be used with subconjunctival phenylephrine injections to avoid significant elevation in blood pressure.

**Corneal opacities**

Corneal opacities are rarely sufficient to preclude retinal surgery, but keratoplasty should be considered, if necessary. The corneal and retinal surgery may be performed at the same time, facilitated by use of a temporary keratoprosthesis during the retinal procedure. Edema of the corneal epithelium can substantially impair visualization, but this can be resolved simply by scraping this layer during surgery.

**Cataract**

Limited opacities of the lens are common in patients with retinal detachment, but the binocular indirect ophthalmoscope usually enables adequate examination. The most common site of cortical opacities is in the inferior nasal quadrant where, fortunately, retinal breaks occur least frequently. The obscuring effect of a moderate degree of nuclear sclerosis can be overcome with the indirect ophthalmoscope at maximum voltage. One can often see around limited central posterior subcapsular (PSC) opacification with the indirect, but when PSC is diffuse, adequate visualization may be impossible.

If, in the presence of a significant cataract, a fairly adequate ophthalmoscopic examination is possible and reveals that the distribution of the subretinal fluid is
adequately explained by the apparent retinal breaks, it is reasonable to proceed
with retinal surgery without removing the cataract. However, in many such cases,
if any question about the strength of the cataract wound from prior surgery, the
surgeon should place additional mattress sutures across the surgical incision site.

Peripheral capsular remnants following cataract surgery may also compromise
the visibility of the retinal periphery. Vitrectomy may occasionally be necessary to
allow widening of the capsular opening.

**Vitreous opacities**

Vitreous opacities, especially hemorrhage, are rather common with retinal detach-
ment, but usually adequate examination of the retina is possible. Vitreous hemor-
rhage may be somewhat cleared by binocular patching and positioning the patient
to allow blood to settle away from the area of the suspected retinal break, as men-
tioned earlier.

If a retinal break that explains the detachment is seen, it is frequently best to
proceed with scleral buckling instead of vitrectomy in a phakic eye. In an aphakic
or pseudophakic eye, vitrectomy may be the procedure of choice.

If the fundus cannot be seen in spite of patching, the eye should be examined
by ultrasonography. If a retinal detachment is demonstrated, the surgeon should
proceed with a pars plana vitrectomy and repair of the detachment.

**External disease**

Retinal surgery is not recommended in the presence of infection. Most retinal
detachments can be deferred for several days until external infection is brought
under control with appropriate antibiotic therapy. Postponement may not be nec-
essary for mild blepharitis.

**Glaucoma**

Preoperative gonioscopy is indicated when a history or signs of elevated intraoc-
ular pressure exist. The presence of open-angle glaucoma prior to retinal detach-
ment generally poses no problem. Miotics should be withheld prior to surgery.

Although retinal detachment usually produces a modest degree of hypotony,
in rare cases the detachment causes secondary elevation of the intraocular pres-
sure, which tends to be refractory to any treatment until the retina is reattached
(Schwartz syndrome). In the absence of significant trabecular damage from uveitis,
the intraocular pressure normalizes postoperatively.

**Uveitis**

A minimal degree of flare and cells in the anterior chamber is common in retinal
detachment and requires no medical treatment. Marked uveitis, however, should
be vigorously treated with cycloplegic drops, as well as topical and/or systemic
corticosteroids, as indicated. Surgery is usually delayed for a few days. Of course, care must be taken to ensure that a rhegmatogenous retinal detachment is indeed present and to rule out a nonrhegmatogenous detachment secondary to uveitis.

**Choroidal detachment**

Retinal detachment must always be differentiated from choroidal detachment, especially in patients who have recently undergone intraocular surgery or experienced other ocular trauma. However, rhegmatogenous retinal detachment and choroidal detachment occasionally coexist. The choroidal detachment is generally associated with hypotony, which induces further choroidal detachment, perpetuating the cycle. Such eyes often have significant uveitis as well.

The anatomic results of retinal reattachment surgery in the presence of choroidal detachment and uveitis are relatively poor, due to a relatively high incidence of postoperative proliferative vitreoretinopathy. In such cases, treatment with systemic and topical corticosteroids for 1 or 2 weeks is sometimes beneficial, but prolonged deferment of the retinal surgery is not recommended. If the choroidal detachment is not in the area of the retinal break, the surgeon should simply proceed with retinal surgery. If, however, the choroidal detachment is in the area of the retinal break or in the area where subretinal fluid must be drained, the surgeon should begin the operation with drainage of the suprachoroidal fluid. Normal intraocular pressure can be maintained during drainage with use of a pars plana cannula for the continuous infusion of a balanced salt solution into the vitreous cavity. The surgeon may then proceed immediately with conventional retinal reattachment surgery.

**Health Management in Preparation for Surgery**

**Preparation for operating room procedures**

In preparation for scleral buckling or vitrectomy, a general preoperative physical examination is indicated to ensure that the patient is prepared for the physical stress of the surgical intervention and the associated anesthetic. It is important to know of chronic or acute medical problems before starting the procedure. Preoperative laboratory evaluation is ordered predicated on existing diseases and risk factors. If poorly controlled medical problems exist, it may be appropriate to postpone surgery for a time while these problems are addressed. However, retinal reattachment surgery is rarely deemed elective, and it is likely not appropriate to wait for an extended time while optimal control is established.

Except in instances of high urgency (such as intraocular infection), time is allowed for the stomach to empty prior to surgery. When a referring doctor calls to send a retinal detachment patient for prompt consultation, the retina surgeon will often ask that the patient be advised not to eat or drink anything en route, in case same-day surgery will be indicated.

If the patient is using anticoagulant medications such as warfarin, it may be appropriate to consider, in consultation with the prescribing physician, discontinuing them prior to surgery. However, many surgeons choose to proceed without modifying warfarin treatment. Surgery for retinal detachment is not usually delayed to reverse antiplatelet medications such as aspirin and clopidogrel (Plavix).
Scleral buckling and vitrectomy procedures are most frequently performed under local anesthesia, but general anesthesia is sometimes preferred. Variables regarding this decision include surgeon and patient preference, expected duration of the procedure, and the patient’s health and mental status. In selected cases, topical anesthesia may be considered.

**Preparation for office procedures**

Pneumatic retinopexy is generally performed as an office procedure under local or topical anesthesia. The medical history is relevant, but a full physical exam and preoperative laboratory testing is usually not necessary. An empty stomach is not required. The risk of hemorrhage with pneumatic retinopexy is low, even for a patient on warfarin.

Retinal photocoagulation usually requires no anesthesia for indirect laser, although topical anesthesia is applied if a contact lens is used with slit lamp laser delivery. Sometimes patient sensitivity requires a retrobulbar anesthetic. Cryopexy usually requires topical, subconjunctival, peribulbar, or retrobulbar anesthesia; an empty stomach is not required.

**POSTOPERATIVE MANAGEMENT**

At the time of discharge after surgery, it is useful to supply the patient with written postoperative instructions. The majority of patients probably would have a satisfactory surgical result without most of these restrictions. However, imposing them routinely seems justified because they can be important for the occasional patient.

**Activity Restrictions and Positioning**

Surgeon preferences regarding postoperative restrictions will differ, but the following guidelines may be helpful. Since most reattachment procedures are performed on an outpatient basis, restrictions in general have become less stringent over the years.

As a general rule, postoperative restrictions are most important for the first week, or until subretinal fluid has reabsorbed. The majority of patients are able to return to work within 1 to 2 weeks, but if a patient’s occupation requires clear binocular vision or significant physical activity, convalescence may be longer.

Early ambulation minimizes postoperative problems such as decubitus ulcers, thrombophlebitis, and generalized weakness. Unless gas has been injected into the vitreous, it is usually not necessary for the patient to maintain a set head position, but chemosis and lid edema are minimized if the patient rests on the unoperated side. It is generally acknowledged that ocular rest encourages the absorption of subretinal fluid. However, watching television or reading involves relatively small eye movements, and is not restricted by most surgeons.

Most patients do well with patching of the affected eye only for 1 to 3 days. Change the eye pad at least daily, or whenever wet.

Stooping, bending, and heavy lifting are often restricted initially following surgery, especially if there has been intraocular hemorrhage, but the value of these practices is uncertain.
If gas has been injected into the vitreous, the patient must be warned not to fly until the bubble is relatively small. Specific positioning of the head to place the causative retinal breaks uppermost is prescribed. Longer restrictions on activity may be called for, particularly if a long-acting gas bubble is used.

**Postoperative Medications**

Patients who have had general anesthesia may have nausea and vomiting for a time after surgery. An antiemetic, such as promethazine, droperidol, or prochlorperazine, may be helpful.

Pain is rarely a problem for retinal patients. Meperidine, 50 to 100 mg, is seldom required beyond the first postoperative day. Propoxyphene napsylate with acetaminophen is an adequate analgesic thereafter. Unexpectedly severe or increasing pain calls for prompt evaluation to rule out endophthalmitis, scleral abscess, or markedly increased intraocular pressure.

Antibiotics and corticosteroids, either topical or systemic, are not mandatory for routine cases, but many surgeons prefer a combination of drugs such as neomycin, polymyxin B, and dexamethasone (Maxitrol) three times a day. Topical scopolamine 0.25% or homatropine 5% help prevent posterior synechiae formation and add comfort in the presence of inflammation. These may be particularly helpful in pseudophakic eyes, especially in diabetic patients. Daily use of a cycloplegic and an antibiotic–corticosteroid combination for 2 weeks is sufficient. In the occasional case in which significant inflammation is noted, appropriate corticosteroid medication should be prescribed.

**Follow-up Examinations**

As a rough guideline, the routine patient should be examined on the first or second postoperative day, at 1 and 3 weeks, and at 2 and 6 months, after which annual examinations are recommended. However, more frequent examinations are frequently necessary following pneumatic retinopexy, for giant retinal tears, or other high risk cases, and particularly if complications develop. Therefore, no firm guidelines can be recommended and follow-up examinations are scheduled on a case-by-case basis. Examination should include an interval history, best corrected visual acuity, tonometry, biomicroscopy, and thorough binocular indirect ophthalmoscopy. Annual examinations help assure the discovery of such asymptomatic problems as new retinal breaks, peripheral detachment, erosion of the implant, or glaucoma.

Following an encircling scleral buckle, a refractive shift is often noted, which generally stabilizes within 2 to 3 months and, if indicated, a permanent spectacle lens may be ordered at that time.

**If the Retina Fails to Settle**

*Postoperative photocoagulation or cryopexy*

Persistent leakage of fluid through a retinal break without evidence of settling is an indication for intervention. If the break is only minimally elevated from the
retinal pigment epithelium, supplemental photocoagulation or cryopexy may be considered. The goal is to induce swelling of the retina and choroid, thereby closing the edges of the break and allowing the subretinal fluid to be absorbed and adhesions to permanently seal the break (Figure 4–9).

If a gas bubble is present in the eye, all that may be necessary is to prescribe a new head position in order to get the retina to settle. Supplemental laser or cryo would then be applied. In other situations, injection of a small expansile gas bubble will provide closure of the open retinal break and allow laser therapy to be performed.

Where any subretinal fluid persists, it is difficult to get retinal burns with photocoagulation, and the tendency is to turn up the power of the laser. Care must be taken to avoid treating with too much intensity, or retinal breaks or hemorrhage may ensue. When photocoagulation is to be performed, it is recommended that the physician patch the patient bilaterally and restrict activity before the procedure to encourage as much settling as possible. Scleral depression at the time of laser application may assist in bringing the retina in apposition to the

![Figure 4–9. Postoperative photocoagulation. (A) Inferior leakage of subretinal fluid, anteriorly from horseshoe break and across buckle in inferior temporal quadrant. (B) Photocoagulation around horseshoe tear and at 3:30-o’clock position. (C) Reattachment of retina after photocoagulation.](image-url)
retinal pigment epithelium. Retrobulbar anesthesia may be necessary because of tenderness. Two or three rows of contiguous lesions should be placed around the leaking break.

Supplemental cryopexy is impeded by the presence of an overlying scleral buckle, but it is not as hindered by residual subretinal fluid. Still, the fluid should be shallow enough that supplemental treatment will allow the retina to settle.

Maximal restriction of activity and proper positioning following supplemental laser application may preclude the need for surgical reoperation in some cases. If the response to treatment is not adequate, the surgeon should not hesitate to proceed with a gas injection or full reoperation.

**Reoperation**

If there is more than minimal postoperative nonsettling subretinal fluid around the leaking break, photocoagulation or cryopexy will likely not be successful. If the leaking break is in the superior eight clock hours of the retina, one may consider pneumatic retinopexy (see Chapter 8). Pneumatic retinopexy can provide a simple salvage for selected failing scleral buckling procedures, avoiding the need to revise the buckle or perform vitrectomy. If pneumatic retinopexy is not appropriate, reoperation with scleral buckling (Chapter 7) or vitrectomy (Chapter 9) can be considered. If the break has remained open for more than a few days after surgery, supplemental cryopexy or photocoagulation should be applied around the break.

**Chronic persistent subretinal fluid**

Sometimes limited subretinal fluid will persist but show no evidence of progression. If there is no open retinal break, the macula is not detached, and the amount of subretinal fluid is not increasing, in most cases intervention is not required. Shallow subretinal fluid may persist in the inferior retina for many years. Multiple small, shallow blisters of residual fluid may also persist chronically after retinal detachment repair.

**SUMMARY**

Knowledge of the patient’s health status along with a thorough evaluation of the eye and retina provide an important basis for the diagnosis and treatment of retinal detachment. Principles of preoperative and postoperative patient management focus on detecting, avoiding, and treating potential complications.

Patients at risk for retinal detachment should be told to be seen promptly if new-onset flashes, floaters, or visual field defects develop. If examination shows detachment likely to extend soon into the macula, prompt surgery is indicated, possibly with preoperative bilateral patching and positioning.

**SELECTED REFERENCES**


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Establishing the Diagnosis

The differential diagnosis of rhegmatogenous retinal detachment includes secondary (nonrhegmatogenous) retinal detachment and other entities that may simulate a retinal detachment. Nonrhegmatogenous detachments are categorized as exudative (serous) and tractional detachments. Conditions that may be mistaken for retinal detachment include retinoschisis, choroidal detachment or tumors, and vitreous membranes. Sometimes benign findings in the peripheral retina are mistaken for retinal breaks.

FUNDUS CHANGES UNRELATED TO RETINAL DETACHMENT

The most prominent feature of the fundus is the optic nerve head or disc, the only place in the human body that affords a direct view of a tract of the central nervous system. The foveola, the functional center of the fundus, is located in the center of the fovea, which has a diameter of about 5°. The macula is centered on the fovea and has a diameter of about 17°. The multiple branches of the central retinal artery are readily identified by their bright red color and relatively narrow caliber. The multiple tributaries of the central retinal vein are recognized by their dark red color and relatively wider caliber.

Choroidal vasculature

In a darkly pigmented fundus, the choroidal vessels in the posterior pole can be obscured from view, but in an eye with minimal pigment, they are readily visible.
The venous tributaries of the choroid that make up the vortex veins are usually easily seen. The most prominent features of the choroidal venous system are the vortex ampullae, of which there are usually four (but sometimes more). They are located approximately in the 1-, 5-, 7-, and 11-o’clock meridians, just posterior to the equator. The horizontal meridians are usually identifiable by their radially oriented, long posterior ciliary nerves, and infrequently the long posterior ciliary artery can be seen adjacent to the nerve. The nerve is relatively broad and has a yellow color, and the artery is identifiable by its red color. The artery is usually inferior to the nerve temporally, and superior to it nasally (Figure 5–1).

**Ora serrata**

The ora serrata is the anterior limit of the sensory retina. It is characterized nasally by prominent ora teeth, which point anteriorly. Between each pair of teeth is an ora bay. There are approximately 48 ora teeth per eye. The pattern of serration is not present temporally, where the ora teeth are small or absent (Figure 5–2).

Occasionally, a white line is evident on the retina just posterior to the ora serrata, representing the posterior limit of the vitreous base. A similar circumferential white line representing the anterior limit of the vitreous base is occasionally visible in the middle of the pars plana.

The normal fusion of the sensory retina and retinal pigment epithelium along the ora serrata is referred to as *retinochoroidal adhesion*. This zone is 3 or 4 mm

![Figure 5–1. Normal fundus with principal structures labeled. (Reproduced from Rutnin U, Schepens CL: Fundus appearance in normal eyes. *Am J Ophthalmol* 1967;64:821–852, 1040–1078. Published with permission from the *American Journal of Ophthalmology*. Copyright by the Ophthalmic Publishing Company.)](image-url)
wide in the temporal periphery, but less than 1 mm wide in the nasal periphery. It is pigmented, a feature easily demonstrated by transillumination. The anterior progression of a retinal detachment is generally halted by this adhesion, but occasionally the subretinal fluid breaks through to create a detachment of the pars plana, in which there is separation of the nonpigmented and pigmented epithelial layers. Detachment of the pars plana is more common on the nasal side, apparently because of the narrower retinochoroidal adhesion. Detachment of the pars plana epithelium is an important feature, and the pars plana must be carefully examined with scleral indentation to detect the presence of breaks in the area, particularly along the anterior limit of the vitreous base.

The periphery of the fundus extends from the equator to the anterior limit of the pars plana, constituting about one third of the total fundus. It is the most important area as far as detachment-producing lesions are concerned.

The equator is an important landmark for the recording of retinal findings, but the beginning ophthalmoscopist might have difficulty identifying it. It is helpful to recall that the distance from the ora to the equator is approximately 4 disc diameters and that the equator is just slightly anterior to the vortex ampullae.

Proceeding forward from the tip of an ora tooth, the examiner may find a faint line extending anteriorly into the corresponding valley of the pars plicata. Such lines are known as striae ciliares. The transition axis between the deep nasal ora bays and the shallow temporal bays is a line that extends approximately from the

Figure 5–2. (A) Temporal ora serrata. (B) Nasal ora serrata.
11- to the 5-o’clock position in the right eye and from the 1- to the 7-o’clock position in the left. A fold of redundant retina is commonly associated with the ora teeth, particularly in the superior nasal quadrant. Often found in an ora bay, these meridional folds are usually benign, but occasionally there is an associated retinal break, in most instances at the posterior limit of the fold.

Variations of the morphology of ora teeth include forked teeth, bridging teeth, giant teeth, and ring-shaped teeth (Figure 5–3). Occasionally, an ora bay is surrounded by retinal tissue, and such an enclosed bay must be differentiated from a true retinal break, which it mimics. Sometimes, a deep ora bay is seen nasally at the horizontal meridian. A dense, shiny-white spherical structure is sometimes seen near the ora serrata. Known as an ora pearl, it has no clinical significance despite its remarkable appearance (Figure 5–4A).

Tufts of redundant glial tissue at or near the ora are called cystic retinal tufts, but also have been termed congenital retinal rosettes, glial spheres, and granular tissue. Rarely, an elevated tuft of retinal tissue points toward the anterior segment. This malformation is due to traction from a zonule and is therefore called a zonular traction tuft. Changes in the fundus thought to be clinically insignificant in the production of retinal detachment include chorioretinal degeneration near the ora, reticular pigmentary degeneration, equatorial drusen, paving-stone degeneration, whitening of the retina, pars plana cysts, retinal erosion, and pigment clumps (see Figure 5–4).

Chorioretinal degeneration

Chorioretinal degeneration is the term for a stippled salt-and-pepper type of pigment alteration that often occurs adjacent to the ora. Infrequently, degeneration is also found more posteriorly toward the equator and, when located between the ora and the equator, it apparently halts the posterior progression of more anteriorly located peripheral cystoid degeneration (see Figure 5–4B).

Reticular pigmentary degeneration

This condition occurs in the aging eye, and the frequency increases with advancing age. Also called honeycomb chorioretinal degeneration, it is caused by retinal pigment epithelial degeneration and is most prominent along the nasal equator. The pigment granules migrate into a pattern that looks like a fishnet or honeycomb (Figures 5–5 and 5–4C). The course of the degeneration is benign.

Equatorial drusen

Drusen are more common in the equator than in the macula. Equatorial drusen may be associated with reticular pigmentary degeneration and are most often found in the elderly. They usually occur in the nasal periphery and are either punctate or geographic (Figure 5–4D).

Cobblestone degeneration

Also known as paving-stone degeneration or peripheral chorioretinal atrophy, cobblestone degeneration is frequently found in the fundus periphery, usually in
Figure 5–3. Morphologic variations of ora serrata region in normal fundi. (A) Nasal periphery with marked cystoid degeneration near horizontal meridian, giant tooth, and deep ora bay over long ciliary nerve. (B) Temporal periphery with marked cystoid degeneration near horizontal meridian, deep ora bay over long ciliary nerve, with forked tooth (above) and tooth with pearl at its base (below). (C) Upper temporal periphery with bridging tooth covered with cystoid degeneration; granular globule (above) and white granular patch with large cystoid cavity on its anterior border (below). (D) Nasal periphery near horizontal meridian, with marked cystoid degeneration; ring tooth (closed ring). (E) Temporal periphery near horizontal meridian; deep ora bay over long ciliary nerve; tooth above deep bay had part of tip broken off; ring tooth (hole-in-the-tooth; above). (F) Nasal periphery, near horizontal meridian, with ring tooth (open ring) and deep ora bay over long ciliary nerve. In next bay (below), two tiny tags. (Reproduced from Rutnin U, Schepens CL: Fundus appearance in normal eyes. *Am J Ophthalmol* 1967;64:821–852, 1040–1078. Published with permission from the *American Journal of Ophthalmology*. Copyright by the Ophthalmic Publishing Company.)
the inferior periphery (Figures 5–6 and 5–4E). These lesions consist of circumscribed areas of atrophy in which there is loss of the outer layers of the retina, retinal pigment epithelium, choriocapillaris, and most of the choroidal vessels. Therefore, in an ophthalmoscopic examination of these lesions, the white sclera is revealed, with perhaps a few intact choroidal vessels crossing the defect. Clumps of pigment may be noted at the margin of each lesion.

An isolated lesion is 1 or 2 disc diameters in size, but adjacent lesions may coalesce to form an elongated lesion with a scalloped margin parallel to the ora. Sometimes the posterior edge of a large area of confluent paving-stones creates the impression of a serrated dividing line and consequently has been called a pseudo-ora (Figure 5–6). Rare secondary retinal breaks at the site of paving-stone lesions have been reported, but this form of degeneration is not a significant cause of primary retinal breaks.

**Retinal whitening**

A common condition, whitening of the retina is ordinarily apparent without pressure from the scleral indentor, and this fact is referred to by the notation

---

**Figure 5–4.** Changes of fundus periphery thought to be clinically insignificant. (A) Composite picture of temporal periphery shows size, shape, and location of pearls. (B) Chorioretinal degeneration at ora serrata. (C) Reticular pigmentary degeneration. (D) Equatorial drusen. (E) Paving-stone degeneration. (F) White-with/without-pressure. (G) Pars plana cysts. (H) Retinal erosion. (Reproduced from Rutnin U, Schepens CL: Fundus appearance in normal eyes. *Am J Ophthalmol* 1967;64:821–852, 1040–1078. Published with permission from the *American Journal of Ophthalmology*. Copyright by the Ophthalmic Publishing Company.)

**Figure 5–5.** Fluorescein angiogram of reticular pigmentary degeneration.
“white-without-pressure.” But if it is evident only when pressure is applied, it is described as “white-with-pressure.” Areas of peripheral whitening sometimes assume rather bizarre forms with geographic outlines, and they are not always connected with the ora serrata. Generally, the areas of whiteness are anterior to the equator, but occasionally they extend to the posterior pole. When examined with scleral indentation, they are discovered to be flat, which differentiates them from retinal detachment or retinoschisis (Figure 5–4F). Infrequently, a round area of normal fundus is surrounded by whitening, creating the incorrect impression of a retinal hole surrounded by a shallow retinal detachment (Figure 5–7).

Whitening is assumed to result from alterations in the anatomy of the vitreoretinal interface. It is more marked in pigmented races and is virtually universal among patients of African descent.

**Pars plana cysts**

Pars plana cysts are relatively common (Figures 5–8 and 5–4G). They average from 1 to 3 disc diameters in size and appear transparent. Scleral indentation clearly reveals their elevation. Pars plana cysts are usually multiple, bilateral, located temporally, and limited to the posterior half of the pars plana. They are clinically benign and do not evolve into retinoschisis.

**Retinal erosion**

Isolated areas of loss of substance in the inner retinal layers are referred to as *retinal erosion*. These focal lesions are curiosities and do not predispose to retinal detachment (Figure 5–4H).
Pigment clumps

Small pigment clumps frequently noted in the peripheral fundus are generally insignificant. Occasionally, however, a vitreoretinal adhesion associated with pigment clumps does appear, and this adhesion may produce a retinal tear. The prognosis for pigment clumps without tears is good, and prophylactic therapy is not indicated.
Hemiretinal differences

The temporal periphery is the most common site of lattice degeneration, retinal breaks, pars plana cysts, dialysis of the young, and degenerative retinoschisis. The nasal periphery is the most common site of prominent ora teeth, meridional folds at the ora, granular tissue, and detachment of the pars plana.

FUNDUS CHANGES RELATED TO RETINAL DETACHMENT

Findings Suggestive of Retinal Detachment

An obvious detachment is recognizable by the marked elevation of the retina. The retina appears translucent or semiopaque, its blood vessels are relatively dark, it may lie in folds, and the detached retina may undulate.

A shallow, slightly elevated detachment of the retina can be more difficult to diagnose. Stereopsis afforded by the binocular indirect ophthalmoscope enhances recognition and proper evaluation of the characteristics unique to each detachment. It is helpful to examine the normal attached retina and then compare it with the adjacent area in question for any changes in retinal transparency that may suggest detachment. With fundus biomicroscopy, a shadow of the retinal vessels is cast on the pigment epithelium. A small degree of shadow formation may be found in the normal retina, but prominent separation between the retinal vessel and its underlying shadow is the clue to a shallow detachment.

The detached retina may assume an “orange-peel appearance,” which is best seen when the retina is viewed in light reflected from the choroid. If a shallow detachment in the periphery is suspected, scleral indentation usually enables visualization of the subtle retinal separation.

One of the best ways to recognize the presence of subtle retinal detachment is to pay attention to the clarity of choroidal markings. In areas where the retina is detached, choroidal vessels and pigment variations are relatively obscured. During routine examination of the peripheral retina, this is often the finding alerting the examiner to the possibility that detachment may exist, calling for closer inspection.

The limits of the retinal detachment should be recorded on the fundus chart. In the search for retinal breaks, the distribution of fluid is a valuable clue to the location of the break(s). It should not be assumed that the detachment is limited by the ora serrata, and a careful search for detachment of the pars plana is required. The limit of the detachment may be accentuated by the presence of a demarcation line, either pigmented or nonpigmented (Figure 5–9). The presence of a demarcation line also calls for careful inspection for current or resolved retinal detachment.

Detection of Retinal Breaks

The entire retina should be carefully examined for retinal breaks by binocular indirect ophthalmoscopy, supplemented by scleral indentation for the periphery.
While breaks may be found in any area, the distribution of the subretinal fluid is a clue to the most likely location of a primary retinal break (Figure 5–10). If one superior quadrant is detached, the break is apt to be near the upper edge of the detachment. When the superior half of the retina is detached, the break is most likely near the 12-o’clock meridian. An inferior quadrant detachment usually has the break near the upper edge of the detachment or in the meridian bisecting the area of detachment. If the inferior half is symmetrically detached, the break could
be anywhere within the detachment, but when the fluid is higher on one side of an inferior detachment than on the other, the break is usually on the higher side.

In a total retinal detachment, the break is often between the 10- and 2-o’clock meridians. If there are inferior bullae, the examiner should assume that a retinal break is above the horizontal meridian. In the presence of a demarcation line, the break is often found in the meridian that bisects the demarcated area.

The history may provide a clue to the location of a break. When the detachment has progressed rapidly, the break is usually superior, fairly large, and probably located nearer the equator than the ora. If the history suggests slow progression of the detachment, a small, inferior, or extremely peripheral break should be sought. The quadrant of first detectable field loss is a valuable indication of the location of the primary break, particularly if the detachment has become total. Special attention should be paid to all areas of abnormality, particularly lattice degeneration, meridional folds, pigmentation, opercula, and hemorrhage.

Distinguishing retinal breaks from retinal hemorrhage

A reddish lesion may be either a retinal hole or a retinal hemorrhage. Careful examination will reveal a homogeneous red color for retinal hemorrhage, somewhat denser than the orangish-red choroid seen through a retinal break. Most important, while both lesions are similar in color, a hemorrhage has a solid appearance compared to the translucent quality of a retinal void. Parallax in ophthalmoscopy is helpful because retinal hemorrhage is localized in the plane of the retina, whereas the apparent redness of a hole is in the plane of the pigment epithelium. The most valuable technique for differentiation is scleral indentation, which allows the examiner to change the illumination of the underlying choroid. These changes will show through a retinal hole but will not change the appearance of a retinal hemorrhage.

Table 5–1. Classification of Retinal Detachment with Proliferative Viteroretinopathy, 1983

<table>
<thead>
<tr>
<th>Grade</th>
<th>Name</th>
<th>Clinical Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Minimal</td>
<td>Vitreous haze, vitreous pigment clumps</td>
</tr>
<tr>
<td>B</td>
<td>Moderate</td>
<td>Wrinkling of inner retinal surface, rolled edge of retinal break, retinal stiffness, vessel tortuosity</td>
</tr>
<tr>
<td>C</td>
<td>Marked</td>
<td>Full-thickness fixed retinal folds</td>
</tr>
<tr>
<td></td>
<td>C-1 one quadrant</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C-2 two quadrants</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C-3 three quadrants</td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>Massive</td>
<td>Fixed retinal folds in four quadrants</td>
</tr>
<tr>
<td></td>
<td>D-1 wide funnel shape</td>
<td></td>
</tr>
<tr>
<td></td>
<td>D-2 narrow funnel shape*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>D-2 closed funnel (optic nerve head not visible)</td>
<td></td>
</tr>
</tbody>
</table>

* Existence of narrow funnel shape is evidenced by indirect ophthalmoscopy when anterior end of funnel can be seen within 45° field of 20D condensing lens (Nikon or equivalent).

Source: Retina Society Terminology Committee: The classification of retinal detachment with proliferative vitreoretinopathy *Ophthalmology* 1983;90:121–125. See also Machemer et al., in Selected References.
PROLIFERATIVE VITREORETINOPATHY

The most common cause of failure in retinal surgery is the presence of intravitreal, preretinal, or subretinal membranes. The various gradations of proliferative vitreoretinopathy (PVR), as published by the Retina Society Terminology Committee in 1983 (see Selected References), are summarized in Table 5–1, and Figures 5–11 through 5–17. An updated classification, published by Machemer and associates in 1991 (see Selected References), offers several advantages, including recognition of anterior PVR (Tables 5–2 and 5–3). The 1991 classification has been used in several research publications, but most clinical papers and clinical practices continue to employ the 1983 classification.

The presence of a preretinal membrane can be subtle and may not be directly observed, but its presence can be inferred from the finding of fixed retinal folds—folds that do not undulate with eye movement (Figure 5–18). These folds may be circumferential or meridional. Frequently these folds radiate out in all directions from a central nidus, called a star fold (Figure 5–19). Posterior rolling of the posterior edge of the retinal break is another sign of periretinal organization (Figure 5–20). Far-advanced PVR is recognized by contraction of the retina toward the visual axis, with numerous fixed folds centered about the optic disc (Figure 5–21) and sometimes totally obscuring its view, constituting a D3 PVR (Figure 5–22).

Some long-standing detachments develop subretinal fibrosis. This is clinically recognized by linear, curved, or angular yellow-white lines on the retroretinal surface of the detached retina (Figure 5–23). Severe subretinal fibrosis may form a tight colarette around the optic nerve (Figure 5–24), necessitating a retinotomy with removal of the subretinal band.
Figure 5–12. Proliferative vitreoretinopathy grade B. (A) Surface wrinkling in 7:30-o’clock meridian. (B) Rolled-over posterior edge of retinal break. (Reproduced with permission from Retina Society Terminology Committee: The classification of retinal detachment with proliferative vitreoretinopathy. *Ophthalmology* 1983;90:121–125.)

Figure 5–13. Proliferative vitreoretinopathy grade C. (A) Grade C-1, one quadrant of full-thickness retinal folds. (B) Grade C-2, two quadrants of full-thickness retinal folds. (C) Grade C-3, three quadrants of full-thickness retinal folds. (Reproduced with permission from Retina Society Terminology Committee: The classification of retinal detachment with proliferative vitreoretinopathy. *Ophthalmology* 1983;90:121–125.)
Figure 5–14. Proliferative vitreoretinopathy grade D-1. (A, B) Four quadrants of full-thickness retinal folds with wide retinal funnel configuration. (Reproduced with permission from Retina Society Terminology Committee: The classification of retinal detachment with proliferative vitreoretinopathy. *Ophthalmology* 1983;90:121–125.)

Figure 5–15. Proliferative vitreoretinopathy grade D-2. (A, B) Narrow retinal funnel configuration. (Reproduced with permission from Retina Society Terminology Committee: The classification of retinal detachment with proliferative vitreoretinopathy. *Ophthalmology* 1983;90:121–125.)
Figure 5–16. Proliferative vitreoretinopathy grade D-2. If the anterior entrance to retinal funnel can be visualized within 45° field of +20 D indirect ophthalmoscope lens (Nikon or equivalent), but optic nerve can be seen, funnel is designated as “narrow.” (Reproduced with permission from Retina Society Terminology Committee: The classification of retinal detachment with proliferative vitreoretinopathy. Ophthalmology 1983;90:121–125.)

Figure 5–17. Proliferative vitreoretinopathy grade D-3. (A, B) Closed funnel, obscuring view of optic nerve head. ([A] reproduced with permission from Retina Society Terminology Committee: The classification of retinal detachment with proliferative vitreoretinopathy. Ophthalmology 1983;90:121-125. [B] courtesy of Stephen Gordon.)
Table 5–2. Classification of Proliferative Vitreoretinopathy, 1991

<table>
<thead>
<tr>
<th>Grade</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Vitreous haze, vitreous pigment clumps, pigment clusters on inferior retina</td>
</tr>
<tr>
<td>B</td>
<td>Wrinkling of inner retinal surface, retinal stiffness, vessel tortuosity, rolled and irregular edge of retinal break, decreased mobility of vitreous</td>
</tr>
<tr>
<td>CPI–12</td>
<td>Posterior to equator: focal, diffuse, or circumferential full-thickness folds,* subretinal strands*</td>
</tr>
<tr>
<td>CA1–12</td>
<td>Anterior to equator: focal, diffuse, or circumferential full-thickness folds,* subretinal strands,* anterior displacement,* condensed vitreous with strands</td>
</tr>
</tbody>
</table>

* Expressed in number of clock-hours involved.


Table 5–3. Grade C Proliferative Vitreoretinopathy Described by Contraction Type, 1991

<table>
<thead>
<tr>
<th>Type</th>
<th>Location in Relation to Equator</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal</td>
<td>Posterior</td>
<td>Star folds posterior to vitreous base</td>
</tr>
<tr>
<td>Diffuse</td>
<td>Posterior</td>
<td>Confluent star folds posterior to vitreous base; optic disc may not be visible</td>
</tr>
<tr>
<td>Subretinal</td>
<td>Posterior/anterior</td>
<td>Proliferations under retina: annular strand near disc, linear strands, moth-eaten-appearing sheets</td>
</tr>
<tr>
<td>Circumferential</td>
<td>Anterior</td>
<td>Contraction along posterior edge of vitreous base with central displacement of retina, peripheral retina stretched, posterior retina in radial folds</td>
</tr>
<tr>
<td>Anterior displacement</td>
<td>Anterior</td>
<td>Vitreous base pulled anteriorly by proliferative tissue, peripheral retina trough, ciliary processes may be stretched and may be covered by membrane, iris may be retracted</td>
</tr>
</tbody>
</table>


The presence of PVR in conjunction with retinal detachment may rule out pneumatic retinopexy as the procedure of choice, and significant PVR may necessitate vitrectomy.
Figure 5–18. Mild fixed fold.

Figure 5–19. Star fold.

Figure 5–20. Rolled posterior edge of retinal break (proliferative vitreoretinopathy grade B).
Figure 5–21. Severe proliferative vitreoretinopathy.

Figure 5–22. Severe proliferative vitreoretinopathy (grade D-3).

Figure 5–23. Subretinal fibrosis. (A) Diffuse sheet with strands. (B) Multiple strands. (Courtesy of Elaine L. Chuang, MD.)
NONRHEGMATOGENOUS RETINAL DETACHMENT

Rhegmatogenous retinal detachments must be differentiated from exudative and tractional detachments. The diagnostic features of all three types of retinal detachment are summarized in Table 5–4. More than 90% of all clinical detachments are of the rhegmatogenous type. Rhegmatogenous detachments are termed primary detachments, while exudative and tractional detachments are called secondary or nonrhegmatogenous detachments. The terms serous detachment and exudative detachment are used interchangeably.

The differential diagnosis of nonrhegmatogenous retinal detachment is summarized in Table 5–5.

DISTINGUISHING SEROUS FROM RHEGMATOGENOUS DETACHMENT

If no retinal break can be found to account for the detachment, the possibility of a serous (or tractional) detachment should be entertained. However, in about 10% of rhegmatogenous detachments, no retinal break can be found. Therefore, lack of a visible tear alone does not rule out rhegmatogenous detachment.

Pigmented vitreous cells will usually, but not always, be present with a rhegmatogenous detachment. These cells are seen best with the slit lamp on high intensity, looking posterior to the lens using no auxiliary lens. Recent detachments caused by small retinal breaks may not have these cells initially. Absence of pigmented cells may be part of a collection of features that leads to a suspicion of serous rather than rhegmatogenous detachment.

When serous detachment is suspected, the patient is evaluated for shifting fluid, and the presence of an underlying cause of serous detachment is sought in history, with retinal examination, and potentially with laboratory evaluation. Ultrasound may be helpful.

Serous subretinal fluid usually shifts with changes in position of the head. “Shifting fluid” is defined as a changing of the detachment borders in response
<table>
<thead>
<tr>
<th></th>
<th>Rhegmatogenous (Primary)</th>
<th>Nonrhegmatogenous (Secondary)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>History</strong></td>
<td>Aphakia, myopia, blunt trauma, photopsia, floaters, field defect; generally healthy</td>
<td>Systemic factors such as malignant hypertension, eclampsia, renal failure</td>
</tr>
<tr>
<td></td>
<td>Systemic factors such as malignant hypertension, eclampsia, renal failure</td>
<td>Diabetes, prematurity, penetrating trauma, sickle cell disease</td>
</tr>
<tr>
<td><strong>Retinal break</strong></td>
<td>Identified in 90% to 95% of cases</td>
<td>No break, or coincidental</td>
</tr>
<tr>
<td></td>
<td>Gravity-dependent; extension to ora is variable</td>
<td>No primary break; may develop secondary break</td>
</tr>
<tr>
<td><strong>Extent of detachment</strong></td>
<td>Extends to ora early</td>
<td>Frequently does not extend to ora</td>
</tr>
<tr>
<td><strong>Retinal mobility</strong></td>
<td>Undulating bullae or folds</td>
<td>Smoothly elevated bullae, usually without folds</td>
</tr>
<tr>
<td></td>
<td>Taut retina, concave surface, peaks to traction points</td>
<td>Taut retina, concave surface, peaks to traction points</td>
</tr>
<tr>
<td><strong>Retinal elevation</strong></td>
<td>Low to moderate, seldom extreme</td>
<td>Varies — may be extremely high to approximate lens</td>
</tr>
<tr>
<td></td>
<td>May be extremely high to approximate lens</td>
<td>Elevated to level of focal traction</td>
</tr>
<tr>
<td><strong>Evidence of chronicity</strong></td>
<td>Demarcation line, intraretinal macrocysts, atrophic retina</td>
<td>Usually none</td>
</tr>
<tr>
<td></td>
<td>Present in 70% of cases</td>
<td>Present in trauma cases</td>
</tr>
<tr>
<td><strong>Pigment in vitreous</strong></td>
<td>Present in 70% of cases</td>
<td>Present in trauma cases</td>
</tr>
<tr>
<td><strong>Vitreous changes</strong></td>
<td>Frequently synergetic, posterior vitreous detachment, traction on flap of tear</td>
<td>Usually clear, except in uveitis</td>
</tr>
<tr>
<td></td>
<td>Blocked transillumination if pigmented choroidal lesion present</td>
<td>Vitreoretinal traction</td>
</tr>
<tr>
<td><strong>Subretinal fluid</strong></td>
<td>Clear</td>
<td>Clear</td>
</tr>
<tr>
<td></td>
<td>May be turbid and shift rapidly to dependent location with changes in head position</td>
<td>Clear</td>
</tr>
<tr>
<td><strong>Choroidal mass</strong></td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td><strong>Intraocular pressure</strong></td>
<td>Frequently low</td>
<td>Usually normal</td>
</tr>
<tr>
<td><strong>Transillumination</strong></td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>Blocked transillumination if pigmented choroidal lesion present</td>
<td>Proliferative diabetic retinopathy, retinopathy of prematurity, toxocara, sickle cell retinopathy, post-traumatic vitreous traction</td>
</tr>
<tr>
<td><strong>Examples of conditions causing detachment</strong></td>
<td>Retinal break</td>
<td>Uveitis, metastatic tumor, malignant melanoma, angiomatosis, Coats’ disease, Eale’s disease, Harada’s syndrome, retinoblastoma, choroidal hemangioma, senile exudative maculopathy, optic pit, exudative detachment after cryotherapy or diathermy</td>
</tr>
</tbody>
</table>
to changes in head position; it does not mean movement of the fluid within stable detachment borders. Shifting fluid is common in exudative detachments, and in rare instances is also seen in old rhegmatogenous detachments.

Rhegmatogenous retinal detachment usually has a characteristic undulation or “tobacco-paper wrinkle.” Usually there is a visible distinction between the

| Table 5–5. Differential Diagnosis of Nonrhegmatogenous Retinal Detachment |
|-----------------------------|-------------------------------------------------|
| **Exudative**               |                                                  |
| **Primary tumors**          | Malignant melanoma of choroid                    |
|                             | Hemangioma of choroid                            |
|                             | Retinoblastoma                                   |
| **Metastatic tumors to choroid** | Breast or lung cancer most common                |
| **Inflammation**            | Scleritis                                        |
|                             | Choroiditis (e.g., Harada’s syndrome)            |
|                             | Retinitis (e.g., toxoplasmosis)                   |
| **Vascular disease**        | Angiomatosis of retina (von Hippel’s disease)    |
|                             | Telangiectasia retinae (juvenile and adult Coats’ disease) |
|                             | Eale’s disease                                   |
|                             | Retinal vein occlusion                           |
| **Optic nerve disease**     | Pit or coloboma of optic nerve head with serous detachment of macula |
|                             | Nerve head drusen with serosanguineous detachment of adjacent retina |
| **Congenital disease**      | Nanophthalmos                                    |
|                             | Familial exudative vitreoretinopathy (FEVR)      |
| **Macular disease**         | Central serous chorioretinopathy (rarely can extend to ora serrata) |
|                             | Age-related macular degeneration                 |
|                             | Other causes of disciform detachment             |
|                             | Ocular histoplasmosis, angioid streaks, high myopia |
| **Systemic disease**        | Toxemia                                          |
|                             | Uremia                                           |
|                             | Lupus erythematosus                              |
|                             | Leukemia                                         |
| **Tractional**              | Proliferative diabetic retinopathy               |
|                             | Retinopathy of prematurity                       |
|                             | Sickle cell retinopathy                          |
|                             | Posttraumatic vitreous membranes                 |
|                             | Retinal vein occlusion                           |

Rhegmatogenous retinal detachment usually has a characteristic undulation or “tobacco-paper wrinkle.” Usually there is a visible distinction between the
detached and nondetached portions of the retina (unless total retinal detachment is present or the view is quite poor). With serous retinal detachment, the exact extent of the detachment is not distinct, and shifting of the borders of the detachment should be sought.

**CAUSES OF SEROUS RETINAL DETACHMENT**

**Choroidal tumors with serous retinal detachment**

Choroidal tumors (such as malignant melanoma) may have some associated serous subretinal fluid. Retinal detachments associated with choroidal tumors are usually readily identified by visualizing the tumor under the retina with binocular indirect ophthalmoscopy (Figure 5–25), but in the presence of extensive serous fluid, the causative tumor may be obscured. When serous rather than rhegmatogenous detachment is suspected, ultrasonography can confirm the presence of an underlying tumor. Scleral transillumination can also help, demonstrating a shadow where a pigmented tumor exists.

The distinction is critical because the management is very different. Surgical repair of retinal detachment is contraindicated in this circumstance. If a malignancy is suspected, full systemic evaluation for lesions metastasized from the eye or for a primary source of cancer metastatic to the eye is usually indicated, with subsequent treatment predicated on the findings.

**Inflammatory serous detachment**

Choroiditis, posterior scleritis, and orbital pseudotumor are inflammatory conditions that can cause serous detachment. Harada’s disease (Figure 5–26) and sympathetic ophthalmia are examples of the former. Panretinal photocoagulation or cryopexy can cause inflammation with serous detachment. Infectious etiologies include orbital cellulitis, infected scleral buckle, and retinitis; for example, toxoplasmosis and CMV retinitis.

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**Figure 5–25.** Retinal detachment associated with malignant melanoma. (Courtesy of Devron R. Char, MD.)
An inflammatory detachment is characterized by a transparent retina with an unusually clear view of the underlying choroid. Signs of inflammation with flare and cells are seen during slit-lamp examination of the vitreous; inflammatory lesions of the choroid can usually be seen with indirect ophthalmoscopy. Retinitis is manifested by a fluffy white retinal lesion with turbidity of the vitreous.

Figure 5–26. Vogt-Koyanagi-Harada syndrome with serous retinal detachment.

Figure 5–27. Capillary hemangioma with dilated, tortuous feeder vessel in von Hippel’s disease.
Vascular lesions with serous detachment
Vascular lesions capable of causing serous detachment include Coats’ disease, Eale’s disease, and retinal vein occlusion. Choroidal hemangiomas and retinal angiomatosis as seen in von Hippel’s disease (Figure 5–27) are vascular tumors that can cause serous detachment.

Congenital causes of serous detachment
Nanophthalmos is a congenital condition that can cause uveal effusion and serous retinal detachment. Familial exudative vitreoretinopathy causes exudative and tractional detachment. Congenital optic nerve pits and optic nerve colobomas may also cause serous macular detachments (Figure 5–28).

Macular lesions with serous detachment
Age-related macular detachment with choroidal neovascularization is a common vascular lesion of the macula that occasionally causes extensive exudation and serous detachment. This process can also occur in the periphery with peripheral disciform scarring and exudation. Extensive subretinal hemorrhagic retinal detachment may also occur.

Central serous chorioretinopathy typically involves serous detachment of the macula. Occasionally, serous subretinal fluid will also settle inferiorly into the peripheral fundus.

Distinguishing Tractional from Rhegmatogenous Detachment
Fibrous proliferation on or under the retina may cause retinal detachment in the absence of retinal breaks. The causative fibrous or fibrovascular proliferation is generally clinically evident, but vitreoretinal traction can be subtle. Tractional detachments typically have a concave contour rather than the convex contour of...
rhegmatogenous detachments. The retina is generally tight and on stretch in tractional detachments, whereas rhegmatogenous detachments are more loose and undulating. Tractional detachments may be complicated by the development of a retinal tear with the development of features of rhegmatogenous detachment. Since PVR is primarily a tractional process, rhegmatogenous retinal detachment may likewise develop some features of tractional detachment. Because vitreoretinal traction is the cause of retinal tears, traction is a factor in the development of most rhegmatogenous detachments.

**Causes of Tractional Retinal Detachment**

Tractional detachments are frequently due to vascular proliferation triggered by ischemia; thus they are caused by proliferative diabetic retinopathy, retinal vein occlusion, cicatricial retinopathy of prematurity, or proliferative sickle retinopathy. Penetrating trauma may also cause tractional detachment. Underlying causes of the tractional process should be sought.

The treatment for tractional retinal detachment usually involves a vitrectomy with peeling or dissection of the tractional membranes, and treatment of any coexisting retinal breaks (see Chapter 9). Treatment of underlying conditions (such as diabetes) is emphasized.

**Lesions Simulating Retinal Detachment**

Lesions that might be confused with retinal detachment include degenerative retinoschisis, choroidal detachment, tumors of the choroid, vitreous membranes, and white-with-pressure or white-without-pressure.

**Retinoschisis**

Elevation of the retinal surface may be due to retinoschisis instead of retinal detachment. Retinoschisis constitutes splitting of the sensory retina into two sheets, like separating the two plies of a tissue. It differs from retinal detachment in that the outer retina is still attached to the eye wall. (See also Chapter 2, page 25.)

Retinoschisis can also coexist with retinal detachment, called schisis detachment. When retinoschisis accompanies detachment, it predates the onset of detachment. An attempt should be made to record the extent of the schisis cavity, as well as the area of detached retina. Determining the extent of each where there is overlap is often difficult, but this becomes better defined during cryopexy. A diagnostic feature of retinoschisis is the “white Swiss cheese” appearance of the outer layer of the schistic retina as it is frozen. In contrast, the retinal pigment epithelium deep to an overlying detached retina appears dull orange when viewed during cryopexy.

Type 1 schisis detachment refers to detachment that does not extend beyond the area of retinoschisis, whereas type 2 schisis detachment extends beyond the schisis (Figure 5–29). Type 2 schisis detachments generally require retinal detachment repair, whereas type 1 generally do not. Type 2 schisis detachments accompanied
by extensive schisis generally have a poorer prognosis for surgical repair than detachments without schisis.

Surgical repair of schisis detachments focuses on closing outer layer breaks and full-thickness retinal breaks. Inner layer breaks do not require treatment.

Retinoschisis is classified as flat or bullous. As a rule, flat retinoschisis is not progressive or slowly progressive. Occasionally, bullous retinoschisis will progress and threaten to involve the macula.

**Intraretinal macrocysts**

Intraretinal macrocysts, which are defined as focal secondary retinoschisis, may mimic degenerative retinoschisis. They occur only in areas of long-standing retinal detachment. Intraretinal macrocysts are usually 2 or 3 disc diameters in size and are most often found in the periphery. Macrocysts require no special treatment and disappear after retinal reattachment. (see Figure 2–27)

**Choroidal detachment**

Choroidal detachment is usually bullous, with a smooth rather than undulating contour. Nasal or temporal bullae tend to be larger than superior and inferior bullae. Usually, the brown choroid can be seen immediately beneath the retina, and the translucent appearance seen with a retinal detachment is lacking (Figure 5–30).

In the deep valleys between choroidal bullae, the choroid is tethered to the sclera by the vortex veins or along the course of the long posterior ciliary artery and nerve. The result is a characteristic “hour glass” configuration to the bullae (Figure 5–31). The tethering effect tends to limit the posterior extension of the detachment, and therefore the posterior pole is often spared. While retinal detachment usually extends anteriorly only as far as the ora serrata, detachment of the choroid extends anteriorly to the scleral spur. There may be folds in the retina, but usually there is little or no retinal detachment associated with choroidal detachment.

Most choroidal detachments are serous, but occasionally a hemorrhagic detachment is seen. Serous and hemorrhagic detachments can be readily differentiated by scleral transillumination. There are many causes for choroidal detachment, but the most common is hypotony following intraocular surgery, especially trabeculectomy and sometimes cataract surgery.

"Figure 5–29. Cross-section of type 2 schisis detachment."
Choroidal detachment usually requires no intervention. However, if the anterior chamber angle has been closed because of pressure from behind, resulting in a marked increase in intraocular pressure, surgical drainage of the choroidal detachments may be necessary to reduce the pressure. Also, when choroidal detachment is so massive that it brings the retina from opposite sides of the eye together in the middle of the vitreous cavity, retina-to-retina adhesions may develop over time, and surgical intervention may be necessary to prevent this.
Choroidal tumors
Occasionally, elevated choroidal lesions are confused with retinal detachments. An experienced observer can tell the difference between a choroidal detachment or choroidal mass and a retinal detachment. The choroidal lesion appears more solid, lacking the translucent appearance of a retinal detachment. Choroidal lesions also tend to be smooth in contour, lacking the undulations of retinal detachment (Figure 5–32). However, secondary serous retinal detachment may also be present, which can make the distinction more difficult (as discussed above).

Vitreous opacities
When the view is cloudy, vitreal membranes or hemorrhage may mimic retinal detachment. In contrast to retinal detachment, vitreal membranes are avascular or have abnormal neovascularization, unlike normal retinal vasculature. With cloudy media, ultrasound may be helpful in making the distinction. Sometimes retinal detachment cannot be ruled out. Serial observations can rule out progression, or a vitrectomy may be indicated to clear the media and allow treatment of retinal detachment if present.

White-with-pressure and white-without-pressure
Where the vitreous is adherent to the peripheral retina, the retina may take on a whiter color than the surrounding area, visible at all times or only visible with pressure from scleral depression. This is usually fairly well demarcated and geographic in shape, and may mimic the coloration of a shallow retinal detachment. By placing the area in question on the crest of scleral indentation while examining with an indirect ophthalmoscope, the absence of separation from the underlying retina can be determined.

Figure 5–32. Malignant melanoma.
SUMMARY

Benign peripheral retinal findings are important to recognize in order to distinguish them from retinal detachments or tears and their potential precursors. Findings in early retinal detachment may be subtle and may include loss of choroidal details, slight undulation of the retina, minimal separation of the retina from the mound of the depression, and shadowing of retinal vessels. Scleral depression is important in consistently detecting retinal detachments and breaks. The presence and severity of PVR should also be assessed.

Rhegmatogenous retinal detachment is to be distinguished from serous and tractional retinal detachment, since the management is very different. In nonrhegmatogenous detachments, underlying etiologies should be sought. Retinal detachment is also to be distinguished from retinoschisis, and from choroidal detachments and tumors.

SELECTED REFERENCES


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Retinal detachment is an uncommon disease, affecting approximately 1 in 10,000 people in the general population per year. However, the incidence of retinal breaks is relatively high, affecting 5% to 7% of the population. Obviously, many retinal breaks have minimal, if any, risk for the possible development of a retinal detachment. This includes macular holes that occur as a degenerative process, and asymptomatic, small, round atrophic holes near the ora serrata. However, equatorial horseshoe tears with relevant symptoms progress to retinal detachment in most cases.

Probably all surgeons would agree that a large horseshoe tear near the equator in the superior temporal quadrant, with new-onset symptoms of flashes and floaters and associated vitreous hemorrhage, should be treated prophylactically to avoid retinal detachment. In contrast, most would not advise treatment of a small, round atrophic hole near the inferior ora serrata in an asymptomatic patient with no history of prior detachment. Between these two obvious examples lies a broad spectrum of retinal breaks for which the surgeon must exercise judgment about instituting prophylactic treatment.

Most of the breaks reported in surveys of asymptomatic patients or in autopsy series are of the atrophic type, and only a small proportion are horseshoe tears. Although there are no specific rules for the selection of patients for treatment, and each case has to be judged on its own characteristics, the application of evidence-based medicine to this topic has modified the opinions of many regarding the genuine value of prophylactic therapy for most retinal breaks.

The American Academy of Ophthalmology has used this approach in developing a Preferred Practice Pattern (PPP) entitled “Posterior Vitreous Detachment, Retinal Breaks, and Lattice Degeneration,” the latest version of which was published in
2008. The evidence base described in this PPP will be employed in the following discussion.

RISK FACTORS FOR RETINAL DETACHMENT

Characteristics associated with a relatively high risk of retinal detachment in an eye with visible retinal breaks are listed in Table 6–1. Symptoms and signs of PVD place an eye at particularly high risk. Additional factors include a variety of hereditary, congenital, acquired, and iatrogenic problems.

The risk of retinal detachment is substantially different among subgroups of eyes, a fact that influences interpretation of both natural history data and treatment results. For example, since an acute PVD is the primary cause of most retinal detachments, and since most retinal tears occur during or soon after PVD, it is likely that eyes without a PVD have a higher risk of later retinal detachment than eyes with a history of prior PVD. Similarly, vitreous liquefaction and PVD occur with greater frequency in older patients and in myopic and nonphakic eyes. Thus, data regarding lesions in otherwise normal, young, nonmyopic eyes are not comparable with data from cases with other risk factors that greatly increase the likelihood of PVD. Since more than one factor is often present, data analysis is difficult if all features are not recorded. For example, myopic pseudophakic eyes with lattice degeneration and with a history of retinal detachment in the fellow eye have a substantially greater risk of retinal detachment than otherwise normal eyes with lattice degeneration.

<table>
<thead>
<tr>
<th>Table 6–1. Risk Factors for Rhegmatogenous Retinal Detachment</th>
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<tbody>
<tr>
<td>Symptomatic posterior vitreous detachment (PVD)</td>
</tr>
<tr>
<td>Hereditary/congenital/developmental/degenerative lesions</td>
</tr>
<tr>
<td>Male gender</td>
</tr>
<tr>
<td>Hereditary vitreoretinopathies</td>
</tr>
<tr>
<td>Myopia</td>
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<tr>
<td>Lattice degeneration with and without holes</td>
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<tr>
<td>Cystic retinal tuft</td>
</tr>
<tr>
<td>Degenerative retinoschisis</td>
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<tr>
<td>Retinal breaks</td>
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<tr>
<td>Symptomatic horseshoe tear</td>
</tr>
<tr>
<td>Dialysis</td>
</tr>
<tr>
<td>Prior ocular surgery</td>
</tr>
<tr>
<td>Aphakia/pseudophakia</td>
</tr>
<tr>
<td>Nd:YAG posterior capsulotomy</td>
</tr>
<tr>
<td>Other surgery involving vitreous gel</td>
</tr>
<tr>
<td>Prior trauma</td>
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<tr>
<td>Inflammation</td>
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<tr>
<td>CMV retinitis</td>
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<tr>
<td>Acute retinal necrosis</td>
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<tr>
<td>Other</td>
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<tr>
<td>Fellow eye non-traumatic retinal detachment</td>
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</tbody>
</table>
As noted in the Academy PPP, no prospective randomized trials of therapy to prevent retinal detachment have been performed. The few meaningful published studies of treated and untreated comparable eyes have been retrospective, and most reports regarding prophylactic therapy have simply described results of a treatment series.

This chapter briefly discusses published outcomes regarding both the natural course of lesions that predispose an eye to retinal detachment and results of prophylactic therapy for these lesions. A thorough review of treatment techniques is beyond the scope of this chapter, although they will be briefly discussed along with complications of therapy.

**SYMPTOMATIC EYES**

Patients are considered symptomatic if they describe suddenly increased vitreous floaters and/or photopsia associated with an acute posterior vitreous detachment. Approximately 15% of eyes with an acute symptomatic PVD develop retinal tears of various types. The risk of retinal tears is directly related to the amount of vitreous hemorrhage, and the finding of pigmented cells in the vitreous is a sign associated with a particularly high chance of associated retinal tear(s). In symptomatic eyes, retinal tears associated with persistent vitreoretinal traction are especially likely to cause retinal detachment.

Retinal tears resulting from a symptomatic PVD should be distinguished from preexisting retinal breaks detected after the PVD but not caused by it. Thus, atrophic retinal holes within areas of lattice degeneration are not considered “symptomatic,” even if they were first observed during an examination prompted by symptoms of an acute PVD. Symptomatic retinal tears are subdivided into those with persistent vitreoretinal traction and those in which all traction in the region of the retinal defect has resolved (Figure 6–1).

**TEARS WITH PERSISTENT VITREORETINAL TRACTION**

**Symptomatic horseshoe-shaped tears**

Most symptomatic tears with persistent vitreoretinal traction are horseshoe shaped and have a high risk of causing clinical retinal detachment. That risk has been reported to be in the range of 33% to 55% of cases. Treatment of this type of break substantially reduces the risk of retinal detachment, and immediate prophylactic therapy for these lesions is indicated to prevent an accumulation of subretinal fluid (Academy PPP evidence level A:II). A chorioretinal adhesion is created in flat retina immediately adjacent to localized subretinal fluid.

**Symptomatic round tears**

Rarely, a retinal tear with a free operculum may have persistent vitreoretinal traction as a result of a residual vitreoretinal adhesion near the retinal break, most frequently at the location of a retinal blood vessel. Only two symptomatic operculated retinal breaks have been reported to progress from initial observation to retinal detachment,
and both were associated with persistent vitreoretinal traction on nearby retinal vessels. In unusual cases in which an operculated retinal hole is the only retinal break associated with a clinical retinal detachment, it is presumed that anomalous persistent vitreoretinal adhesions are located in the vicinity of the retinal tear. Treatment of these breaks may be recommended if the presence of a nearby vitreoretinal adhesion cannot be ruled out (Academy PPP evidence level A:III). Failures following treatment of operculated retinal holes have not been reported.

**BREAKS UNASSOCIATED WITH PERSISTENT VITREORETINAL TRACTION**

**Symptomatic operculated retinal tears**

Tears unassociated with persistent vitreoretinal traction in the vicinity of the retinal break have not been reported to progress to clinical retinal detachment. Similarly, although large numbers of these breaks have been treated prophylactically, there are no reports in the literature of a treatment failure. Treatment of this type of retinal break appears to be unnecessary unless the possibility of persistent vitreoretinal traction cannot be excluded (Academy PPP evidence level A:III).

**Symptomatic atrophic retinal holes and precursors of retinal breaks**

Eyes with symptoms and signs of acute PVD frequently contain atrophic retinal breaks that are not due to acute vitreoretinal traction. For the purposes of this discussion, these lesions are considered to be preexisting and not symptomatic and are discussed in a later section. Similarly, precursors of retinal breaks or detachment, including lattice degeneration, cystic retinal tufts, and age-related retinoschisis, are managed as if they were originally discovered in asymptomatic eyes.
ASYMPTOMATIC EYES

Asymptomatic eyes include those with and without additional risk features. Cases in which lesions are diagnosed in a second ("fellow") eye following a detachment in the first eye are discussed in a separate section below.

ASYMPTOMATIC NON-FELLOW EYES AT HIGH RISK

The term "non-fellow eye" indicates absence of a history of retinal detachment in the other eye. Myopia, previous cataract extraction, and a positive family history for retinal detachment are considered as additional risk factors in patients with vitreoretinal lesions believed to predispose a non-fellow eye to retinal detachment. The existence of any of these factors in non-fellow eyes has been associated with an increased enthusiasm for prophylactic therapy, despite the absence of appropriate supporting data.

Asymptomatic myopic non-fellow eyes

Myopia is obviously associated with an increased risk of retinal detachment, and there is a direct correlation between amount of myopia and incidence of retinal detachment. Lattice degeneration associated with retinal holes did not correlate with degree of myopia in one large natural history study, although most slowly progressive clinical retinal detachments associated with lattice degeneration and an absence of extensive PVD occur in young myopic patients, as noted above. There appears to be no increased value for treatment of myopic eyes with lattice degeneration in non-fellow eyes (Academy PPP evidence level A:III), and it is noteworthy that the small favorable effect of preventive treatment of lattice lesions in phakic fellow eyes in one important report could not be demonstrated if the degree of myopia exceeded 6 diopters.

Cystic retinal tufts and degenerative retinoschisis are not more common in myopic eyes, and prophylactic therapy is not recommended in the absence of a progressive subclinical detachment.

Asymptomatic retinal breaks are more common in myopic eyes than in emmetropic or hyperopic cases. However, clinical retinal detachments in these cases are rare in the absence of new symptoms, and prophylactic treatment is usually not advised in non-fellow eyes (Academy PPP evidence level A:III).

Asymptomatic nonphakic non-fellow eyes

Removal of the crystalline lens is associated with a substantial increase in the rate of later retinal tears and detachments, regardless of the method of cataract surgery; this probably is due to vitreous changes in the nonphakic eye. An intact posterior lens capsule appears to be associated with a reduced rate of retinal detachment following cataract surgery, whereas Nd:YAG capsulotomy is clearly associated with an increased risk of subsequent detachment.

The natural course of lattice degeneration in nonphakic non-fellow eyes is not well documented, and results of preventive treatment in these particular cases are not available.
Similarly, meaningful information regarding cystic retinal tufts and degenerative retinoschisis has not been published. Treatment of these lesions in non-fellow eyes is not advised (Academy PPP evidence level A:III).

Asymptomatic retinal breaks in nonphakic non-fellow eyes or eyes undergoing cataract surgery have sometimes been regarded as an indication for prophylactic therapy. However, in one study, 18 retinal breaks in nonmyopic aphakic eyes were followed up for 3 to 7 years, and none detached. In another study of 103 myopic aphakic eyes, 25 asymptomatic retinal breaks were discovered in 19 eyes. Although 6 of the 25 were horseshoe-shaped tears, later retinal detachment occurred in no cases. Treatment of asymptomatic retinal breaks in non-fellow eyes that are non-phakic or scheduled for cataract surgery is usually not recommended (Academy PPP evidence level A:III). Horseshoe-shaped tears should be followed more closely than round breaks.

**Family history of retinal detachment**

Heredity clearly influences the chances of retinal detachment, particularly in families with vitreoretinal degenerative disorders such as Stickler syndrome. Prophylactic therapy is frequently considered in these cases, particularly if retinal detachment has occurred in the primary eye. However, studies have not properly stratified the several high-risk factors associated with retinal detachment and evaluated the natural course and the effects of prophylactic therapy in patients with a familial predisposition to retinal detachment.

**Stickler syndrome**

Individuals at risk for Stickler syndrome due to family history or systemic findings should have a thorough retinal evaluation. Vitreous liquefaction and vitreous membranes are characteristic. Retinal pigmentation in lattice-like patches, retinal tears, and detachments are also common.

Early diagnosis is important, since Stickler patients have about a 50% lifetime risk of retinal detachment. In addition, these patients have a poorer than usual prognosis with surgical repair of detachment. Therefore, all new retinal tears should be treated, and some clinicians recommend prophylactic treatment for all areas of lattice-like degeneration as well, in spite of a lack of data supporting the value of this therapy. Frequent examinations, at least every six months, are recommended, and patients should be well-educated regarding the symptoms of PVD and peripheral field loss.

**Asymptomatic Precursors of Retinal Breaks without High-Risk Features**

Asymptomatic nonmyopic phakic eyes in patients without a personal or family history of nontraumatic retinal detachment are unlikely to develop a retinal detachment, regardless of the presence of vitreoretinal pathology. Nevertheless, prophylactic therapy has sometimes been recommended to treat visible precursors of retinal detachment or retinal breaks.

Precursors of retinal breaks and detachment include lattice degeneration, cystic retinal tufts, and degenerative retinoschisis. Of these, lattice degeneration is clearly
the most important. Both lattice degeneration and cystic retinal tufts can be sites of retinal tears resulting from vitreoretinal traction at the time of PVD. Atrophic retinal holes commonly occur within areas of lattice degeneration and also in the outer layer of degenerative retinoschisis. However, these holes are a relatively infrequent cause of progressive retinal detachment.

**Asymptomatic lattice degeneration**

Lattice degeneration is present in approximately 30% of retinal detachments, and approximately 94% of these detachments occur in primary (non-fellow) eyes. Because lattice lesions are visible and occur in approximately 8% of the population, they have commonly been considered as candidates for prophylactic therapy. However, an important natural history study of 276 patients and 423 involved eyes, followed up for an average of almost 11 years, indicated that lattice lesions in phakic non-fellow eyes were not particularly dangerous. At the end of the follow-up period, atrophic retinal holes were present in 150 eyes (35%).

Subclinical retinal detachments, defined as subretinal fluid extending more than one disc diameter from the break but not posterior to the equator (Figure 6–2), were observed in 10 of the eyes with holes. In six of these eyes the subclinical detachment developed during the observation period, whereas four eyes exhibited the changes at the initial examination. Only one subclinical detachment was considered in need of treatment after a small asymptomatic posterior extension of subretinal fluid.

![Figure 6–2](image.png)

**Figure 6–2.** Subclinical retinal detachments are frequently defined as those associated with subretinal fluid extending more than one disc diameter from the break, but not posterior to the equator.
Four asymptomatic tractional retinal tears were observed in three of these 423 eyes at the initial examination, and symptomatic tractional tears without clinical detachment developed in five additional eyes during follow-up periods of 1.5 to 18 years. Three of five symptomatic and all asymptomatic breaks occurred adjacent to lattice lesions. All symptomatic breaks were successfully treated; no asymptomatic tractional tears were treated, and none changed over follow-up periods of 7, 10, and 15 years. Clinical retinal detachments developed in three of the 423 eyes. Two were due to round retinal holes in lattice lesions of patients in their mid-twenties, and one was due to a symptomatic tractional tear.

These figures clearly indicate that patients with lattice degeneration in a phakic non-fellow eye should usually not be treated prophylactically unless symptoms occur (Academy PPP evidence level A:III). However, retinal detachments associated with vitreoretinal traction upon lattice lesions containing atrophic retinal holes are relatively common in eyes with significant amounts of myopia. A discussion regarding self-examination of peripheral visual fields and periodic follow-up examinations are in order in myopic patients to reduce the chances of macular involvement by slowly progressive detachments resulting from round holes in lattice lesions.

**Asymptomatic cystic retinal tufts**

Retinal tears at sites of cystic retinal tufts may be responsible for as many as 10% of clinical retinal detachments associated with posterior vitreous detachment, and they are also associated with asymptomatic small horseshoe-shaped tears and minimal subretinal fluid in the absence of PVD. The chances of clinical retinal detachment in eyes with cystic retinal tufts have been estimated by one expert to be 1 in 357, and these lesions are not worthy of prophylactic therapy in otherwise normal eyes.

**Asymptomatic degenerative retinoschisis**

Clinical retinal detachments occur in association with degenerative retinoschisis in up to 6% of consecutive detachment cases, and the presence of retinoschisis and outer layer breaks has sometimes been considered an indication for prophylactic therapy. However, a natural course study of 218 eyes in 123 patients demonstrated no clinical retinal detachments during a follow-up period averaging 9.1 years. The vast majority of small subclinical detachments that develop in association with outer layer breaks remain small, and prophylactic therapy is indicated only in the presence of obvious significant progression of subretinal fluid posterior to the equator.

**Asymptomatic Retinal Breaks**

In phakic non-fellow eyes, asymptomatic retinal breaks that are routinely discovered during an evaluation of the peripheral retina are extremely unlikely to lead to clinical retinal detachment, even if they are flap tears and even if posterior vitreous detachment occurs. In one study, during a follow-up period averaging 11 years, asymptomatic retinal breaks were detected in 235 eyes of 196 patients,
and horseshoe-shaped tears were present in 45 cases. Acute PVD occurred in nine eyes without adversely affecting the preexisting breaks, although new horseshoe-shaped tears developed in three cases, and these were promptly treated. Subclinical retinal detachments were observed in 19 eyes (8%). Modest extension of subretinal fluid required therapy in two of these cases, and in a third case a peripheral clinical retinal detachment slowly developed after 14 years of observation.

Prophylactic therapy for asymptomatic retinal breaks in phakic non-fellow eyes is usually not recommended (Academy PPP evidence level A:III). An occasionally observed exception to this rule is an inferior retinal dialysis. These breaks can cause slowly progressive retinal detachments that frequently become symptomatic only after macular involvement.

**PATIENTS WITH RETINAL DETACHMENT IN THE FELLOW EYE**

Pathologic vitreoretinal changes often occur bilaterally, and patients with retinal detachment in one eye have a significantly increased risk of retinal detachment in the other eye. This risk has been estimated as ranging from as low as 9% to as high as 40%. Thus, attempts to prevent retinal detachment in the second eye have received considerable attention. Prospective randomized studies have not been performed, but retrospective data regarding precursors of retinal detachments and asymptomatic retinal breaks have been published. These can be further categorized as phakic and nonphakic fellow eyes.

**ASYMPTOMATIC PHAKIC FELLOW EYES**

Phakic fellow eyes have a lower risk of subsequent retinal detachment than comparable nonphakic eyes.

**Asymptomatic phakic fellow eyes with lattice degeneration**

Lattice degeneration is the most extensively studied indication for prophylactic therapy in fellow eyes. One widely quoted study retrospectively evaluated 388 consecutive cases in which phakic retinal detachment associated with lattice degeneration occurred in one eye and lattice degeneration was present in the second eye. During an average follow-up period of over 7 years, new retinal breaks or detachments occurred in 31 (20%) of 151 untreated eyes. New tears with retinal detachment developed in nine eyes, and new tears without detachment developed in 10 cases. In 10 eyes, new holes developed within areas of lattice degeneration, and atrophic retinal breaks occurred in areas distant from lattice lesions in the remaining two cases.

A reduction in the incidence of new retinal tears and detachments in eyes receiving prophylactic therapy for all lattice lesions was observed. New tears without detachment occurred in five (3.0%) of these fully treated eyes. Retinal detachment occurred in three additional eyes (1.8%), compared with 5.1% in the 151 untreated phakic fellow eyes. The small beneficial effect of treating all lattice lesions was
apparent when follow-up periods of 3, 5, and 7 years were analyzed separately. The beneficial effect was statistically significant for all patient subgroups, except in eyes with myopia of 6 diopters or more, and in eyes with both high myopia and more than 6 clock-hours of lattice degeneration. Importantly, in these subgroups, treatment did not reduce the risk of retinal tears or detachment. Conversely, no detachments occurred after full treatment in eyes with less than 6 clock-hours of lattice degeneration, or with less than 1.25 diopters of myopia. New horseshoe-shaped tears developed in areas unassociated with lattice degeneration in approximately 30% of treated cases.

One expert has estimated that as many as 58% of retinal detachments in eyes with lattice degeneration arise in areas that exhibit no visible vitreoretinal abnormalities (Figure 6–3). Because of this reality, some surgeons have recommended prophylactic therapy featuring the production of laser or cryotherapy burns over 360° of the peripheral retina (Figure 6–4). However, the precise indications, intraocular findings, long-term results, and complications of this form of therapy have not been thoroughly described, and remarkably different success rates have been reported.

Studies of prophylactic therapy of lattice degeneration, with and without holes, in phakic fellow eyes have been of limited value because they have not been prospective and because important information has been missing from available retrospective analyses. In particular, the outcomes have not been studied as a function of the presence of a posterior vitreous detachment. It has been noted by several authors that retinal detachments are unusual in phakic fellow eyes if a PVD was present at the time of the initial examination.

Figure 6–3. Adequate treatment of lattice lesions does not prevent the development of retinal tears at sites of invisible vitreoretinal adhesions.
The relatively low incidence of retinal detachment in untreated cases, the frequency of new tears in normal-appearing retina, the apparent ineffectiveness of therapy in eyes with extensive lattice degeneration and high myopia, and the known success rate following treatment of symptomatic retinal tears and detachments indicate that prophylactic treatment is of limited value in these cases. The apparently modest benefit following treatment of all lattice lesions may be of value in selected patients, such as those with a poor surgical result in the first eye, patients who are incapable of recognizing symptoms of vitreous and/or retinal detachment, or those who live in areas with limited access to ophthalmologic care. In addition, as noted above, myopic patients with atrophic holes in lattice lesions should be evaluated periodically and counseled about loss of peripheral vision, because slowly progressive retinal detachments can occur.

**Asymptomatic phakic fellow eyes with cystic retinal tufts**

Cystic retinal tufts are bilateral in only 6% of cases, so they are not a common cause of bilateral retinal detachment, and there are no data supporting the value of prophylactic therapy.

**Asymptomatic phakic fellow eyes with degenerative retinoschisis**

This is an unusual cause of progressive retinal detachment, but retinoschisis is both common and frequently bilateral. Thus, patients with both retinal detachment and retinoschisis in one eye frequently have retinoschisis in the fellow eye. An evaluation of the literature regarding prophylactic therapy for retinoschisis
in phakic fellow eyes is very difficult because of a lack of complete information regarding the cases Academy PPP evidence level: no consensus. In the unusual case in which outer layer retinal breaks have been responsible for retinal detachment in the first eye, and outer layer breaks and retinoschisis are present in the fellow eye, prophylactic therapy is frequently recommended.

**Asymptomatic phakic fellow eyes with retinal breaks**

Asymptomatic retinal breaks in phakic fellow eyes of patients with previous retinal detachment are frequently cited as an indication for prophylactic therapy. Flap tears appear to be much more likely to cause retinal detachment than round or operculated retinal holes. In one study, retinal breaks were discovered in 186 (19%) of 966 fellow eyes, 28 of which (15%) later developed retinal detachment. Horseshoe-shaped tears were the cause of the detachment in 20 (71%) of the 28 eyes, whereas only 19% of breaks were flap tears in the 158 eyes that did not progress to retinal detachment. However, one author followed 10 untreated asymptomatic horseshoe-shaped tears in phakic fellow eyes, and no retinal detachments occurred. Deficiencies in prior reports have made it difficult to assess both the natural course of asymptomatic retinal breaks that are discovered on an examination of a fellow eye and the results of treatment of these lesions. Most of these breaks are round and located within areas of lattice degeneration, and these cases were discussed earlier. Data regarding therapy for asymptomatic horseshoe-shaped tears in fellow eyes suffer from a lack of details, including the status of the vitreous gel and the relationship between the original retinal break and the cause of subsequent retinal detachment. Still, treatment of horseshoe-shaped tears that are discovered in asymptomatic fellow eyes is sometimes recommended despite the absence of optimal supportive data (Academy PPP evidence level A:III)

**Asymptomatic phakic eyes with a history of giant retinal tear in the fellow eye**

Prophylactic treatment is frequently recommended in phakic fellow eyes in which a non-traumatic giant retinal tear has occurred in the first eye. In one important study, 321 cases were followed for 12 months through 29 years. New giant retinal tears occurred in 14 untreated eyes (4.4%), 13 of which had developed “high-risk features” of high myopia, vitreous degenerative changes, and “white-with-pressure” that increased in extent. In a more recent report, 48 patients were followed for a mean of 84 months after repair of a giant retinal tear in one eye and 360° cryotherapy of the second eye. During the follow-up period, retinal detachment developed in three patients, and a retinal tear alone was observed in a fourth.

**Asymptomatic Aphakic and Pseudophakic Fellow Eyes**

All eyes have an increased risk of retinal detachment after cataract extraction, and the post-cataract surgery risk for fellow eyes in patients with previous retinal detachment in the first eye has been estimated to be 14% to 41%. The chance of detachment is higher if secondary Nd:YAG capsulotomy was required. Thus prophylactic therapy has frequently been recommended for vitreoretinal lesions in
fellow eyes that are nonphakic (aphakic or pseudophakic) or that are scheduled to undergo cataract extraction.

**Asymptomatic nonphakic fellow eyes with lattice degeneration**

Of the precursors of retinal tears that have been considered for prophylactic therapy before or after cataract extraction, only lattice degeneration has been extensively studied, and reviews of the literature have been published. However, no prospective randomized studies have compared the natural course in these cases with outcomes following preventive treatment. As is true of phakic fellow eyes, a major problem in treating only visible pathology is the frequency of new retinal tears that develop in areas of the peripheral retina that appear normal. Although treatment of visible lesions appears to reduce the chances of retinal tears occurring at the treated site, the retinal detachments that frequently develop in these fellow eyes are not prevented by this focal therapy.

Studies of prophylactic treatment of lattice degeneration in fellow nonphakic eyes have not been stratified on the basis of posterior vitreous detachment. In one important study, the critical importance of this variable was evaluated by studying aphakic eyes of patients with aphakic retinal detachment in the primary eye. Retinal detachment subsequently occurred in one (2.3%) of 43 eyes with a PVD in the fellow eye. In the 40 eyes without a previous PVD, retinal detachment later occurred in eight eyes (21%). Similarly, in another study, retinal detachments occurred in five (24%) of 21 aphakic fellow eyes without PVD at the initial examination, but no detachments occurred in 15 additional cases in which a PVD was initially present.

Because of the tendency for new retinal breaks to develop in areas of the retina that appear normal, 360° treatment has been advocated, as noted earlier. In a non-randomized retrospective study of eyes following vitrectomy and silicone oil installation, this form of therapy appeared to be of value following removal of the oil. Still, the risk–benefit ratio of this form of treatment is unknown. Treatment of lattice lesions in nonphakic fellow eyes is frequently performed despite the lack of supportive data. In eyes in which a PVD has previously occurred, it is doubtful if therapy is particularly effective or necessary. The value of various forms of prophylactic treatment in eyes without a PVD will remain debatable until appropriate trials are conducted.

Cystic retinal tufts and degenerative retinoschisis are unusual causes of bilateral retinal detachment, and data discussing the importance of these entities following cataract surgery are not available. They are managed as discussed under Asymptomatic Phakic Fellow Eyes.

**Asymptomatic nonphakic fellow eyes with retinal breaks**

Retinal breaks in nonphakic eyes of patients with a previous retinal detachment in the other eye appear to have a higher rate of causing detachment. One author described asymptomatic retinal breaks in 10 aphakic fellow eyes. Subsequent retinal detachments occurred in five of these cases. Four of the five breaks causing retinal detachment were horseshoe-shaped tears, and the type of the fifth break
was not reported. The literature regarding the value of treating round holes unassociated with lattice lesions is not clear. Treatment can be expected to prevent retinal detachment resulting from the identified break but not detachment resulting from breaks in other areas of the retina. Treatment of asymptomatic horseshoe-shaped tears in aphakic fellow eyes and in fellow eyes scheduled to undergo cataract extraction is usually recommended, despite the absence of supportive data.

**Asymptomatic nonphakic eyes with a history of giant retinal tear in the fellow eye**

Aphakic fellow eyes in nontraumatic giant retinal tear cases have a high risk of retinal detachment. Prophylactic therapy has been recommended for fellow eyes of these patients, particularly if significant vitreous liquefaction and progressive “white-with-pressure” are observed.

**TREATMENT TO PREVENT RETINAL DETACHMENT**

Photocoagulation, cryotherapy, diathermy, and, in certain rare instances, scleral buckling have all been used for prophylactic treatment of retinal breaks without detachment. The choice of treatment technique depends on the surgeon’s experience and the availability of equipment. The laser indirect ophthalmoscope (LIO) with scleral indentation has become a frequent choice where this equipment is available. Alternatively, far anterior tears can be treated with cryotherapy, and more posterior tears can be treated with photocoagulation using a slit-lamp laser with a mirrored contact lens. Diathermy is infrequently used at present because of the need for conjunctival peritomy, but good results can be obtained with it.

**CRYOTHERAPY**

Tanks of compressed gas are connected to a pressure regulator, and tubing directs the gas under pressure to the cryoprobe. The gas is allowed to expand at the tip of the cryo probe, which causes the probe to freeze. When the probe is pressed onto the scleral wall, the freeze will extend into the interior of the eye and achieve the desired thermal burn in the retina and retinal pigment epithelium. Cryotherapy can also be applied directly to the retina through the interior of the eye during vitrectomy, although this practice has rarely been employed since the development of contemporary laser equipment and techniques.

Good anesthesia is obtained by supplementing topical 4% lidocaine with an injection of subconjunctival 1% or 2% lidocaine in the involved quadrants. Retrobulbar anesthesia may be necessary in some patients, but the patient then loses the ability to position the eye in accordance with the surgeon’s instructions.

To apply cryotherapy for a retinal break near the ora, the surgeon instructs the patient to move the eye in the direction of the break. Breaks near the equator are most easily indented with the cryoprobe when the eye is approximately in the primary position. Breaks posterior to the equator can often be reached with the cryoprobe if the patient is instructed to move the eye away from the meridian of the
break and slightly toward the surgeon, who is positioned in the meridian opposite that of the break. More posterior lesions can be reached by a small incision in the conjunctiva but are more easily treated by laser.

A number of applications should be used to surround the break completely with an adequate margin, and treatment of horseshoe tears should extend anteriorly into the vitreous base (Figure 6–5). Treatment should be limited to flat retina and extend only to the margin of the subretinal fluid. Freezing of the exposed pigment epithelium within the tear should particularly be avoided, because this will encourage the migration of retinal pigment epithelial cells into the vitreous cavity. In a patient with lattice degeneration, the treatment should include not only the lattice patch but also adjacent normal retina on all sides of the patch.

If there is significant subretinal fluid associated with the break(s), the patient is generally restricted in activities until some pigment appears around the focal detachment; this generally occurs within 7 to 10 days. Extension of subretinal fluid before an effective chorioretinal adhesion develops may be more common following cryotherapy than after laser photocoagulation, because the adhesion forms more quickly after laser.

**LASER PHOTOCOAGULATION**

For various reasons (including the more rapid development of an adhesive reaction and avoiding any liberation of viable retinal pigment epithelial cells), laser therapy has become a more popular means of creating an adhesion. A laser photocoagulator provides an excellent viewing system, with either the laser indirect ophthalmoscope (LIO) or the binocular biomicroscope and slit lamp used in association with
the Goldmann three-mirror or panfunduscope contact lens. The appropriate end point for these lesions is the development of a dull white reaction within the retina (Figure 6–6). The entire retinal break should be surrounded by a series of photo-coagulation lesions, and treatment should be extended into the vitreous base with flap tears.

**RESULTS OF TREATMENT**

The efficacy of prophylactic treatment obviously depends on the relative risk of detachment before and after treatment. If a retinal detachment ensues within a few days of a prophylactic procedure, it might be argued that the treatment actually precipitated the detachment. But the detachment could very well be due to progression of the disease despite treatment. If the detachment develops several weeks later, it is more likely due to the ongoing, progressive nature of the basic vitreoretinal disease, and is not to be regarded as an iatrogenic complication. While some detachments are caused by the treated breaks, in most cases detachment subsequent to treatment is due to a new break in a different meridian.

Reports of prophylactic therapy results are all somewhat incomplete, as most do not describe information about important variables, including the refractive error, the status of the crystalline lens, the status of the posterior vitreous surface, the type of retinal breaks, and whether there had been a detachment in the fellow eye. In addition, long-term follow-up information is lacking in most reports. In one important study, it was demonstrated that new retinal breaks continued to occur long after the initial prophylactic therapy. In symptomatic eyes that were treated, new retinal breaks were observed in 13% of cases after 3 months, and in 21% of cases 2 years postoperatively. The outcomes in this report were not stratified as a function of the type of retinal break. Another study demonstrated that most detachments that occurred after prophylactic therapy were associated with progression of an incomplete initial PVD.
Flap tears
Horseshoe-shaped tears are most responsible for clinical retinal detachment. Symptomatic horseshoe-shaped tears are much more likely to cause retinal detachment than are asymptomatic tears, but many studies have not distinguished between these groups. However, failure of treatment appears to be more common following therapy of symptomatic cases. Early failures usually are due to vitreoretinal traction forces that cause an accumulation of subretinal fluid before the establishment of an adequate chorioretinal adhesion, or are due to incomplete or inadequate therapy. Treatment should be placed in flat retina immediately adjacent to the location of subretinal fluid, and treatment should extend well anterior to the “horns” of the tear and into the vitreous base.

Lattice degeneration
There are many difficulties in analyzing results of treatment of lattice degeneration, as mentioned earlier. Most new tears following therapy occur in areas not previously treated.

Retinal holes
The prognosis for round holes after treatment is substantially better than that for flap tears with persistent vitreoretinal traction. As noted earlier, round holes unassociated with persistent vitreoretinal traction or with lattice degeneration are usually not treated.

Patients with previous retinal detachment in the fellow eye
Fellow eyes of patients with retinal detachment in a first eye have a substantial risk of retinal detachment, and the risk is even higher after cataract extraction. Aphakia and pseudophakia appear to be statistically significant risk factors for failure after prophylactic therapy for retinal breaks, and treatment of the visible lesions in these cases does not appear to prevent many retinal detachments.

Complications of Treatment
Retinal detachments that occur despite prophylactic therapy are a result of either inadequate adhesion around a retinal break or a new retinal break. Extension of the detachment is considered a complication of therapy if the treatment is inadequate in extent or intensity, and a particularly common cause of failure in treating horseshoe-shaped tears is the absence of an adequate chorioretinal adhesion surrounding the anterior margins of the break, where vitreoretinal traction persists.

New retinal breaks are a complication of prophylactic therapy if the treatment causes excessive damage to the retina, resulting in a break at that location, or if it aggravates vitreous degenerative changes and vitreoretinal traction, causing a tear elsewhere.

Epiretinal proliferation that causes macular pucker has been considered a potential complication of prophylactic therapy, but the association between treatment and membrane formation is uncertain. Symptomatic retinal tears are almost always due to a posterior vitreous detachment, and a PVD is present in more than
90% of eyes with idiopathic epimacular proliferation. Also, when vitreoretinal traction causes a retinal tear, pigment epithelial cells are usually liberated into the vitreous cavity, and these may be a source of subsequent epimacular proliferation. The method of creating a chorioretinal adhesion appears to be unrelated to the incidence of postoperative macular pucker.

### SUMMARY

Although prevention of retinal detachment is an important goal, the genuine value of prophylactic therapy for most vitreoretinal lesions remains unknown because of a lack of appropriate trials. Treatment of symptomatic flap tears is an accepted method of preventing clinical retinal detachments, because the natural course of these breaks and the results of therapy are well documented. In most other instances, treatment of visible abnormal vitreoretinal lesions is of limited value, even in eyes with additional risk features such as high myopia, pseudophakia, and history of retinal detachment in the fellow eye.

This chapter attempts to summarize briefly the literature on this topic (Table 6–2). Specific decisions regarding prophylaxis for a given eye should be made on the basis of the features of the case and expanding medical knowledge. Patients with high-risk features should be made aware of symptoms of posterior vitreous detachment and loss of visual field, and any patient with such symptoms should be promptly evaluated. In addition, periodic evaluations may be indicated.

### SELECTED REFERENCES


Inflammatory detachments are usually treated medically. Some serous detachments, such as choroidal hemangioma, respond to photocoagulation or photodynamic therapy (PDT). Selected traction detachments, such as diabetic or post-traumatic detachments, may be cured with intraocular microsurgery (vitrectomy). Radiation therapy is often used for detachments secondary to metastatic tumors. This chapter is confined to the surgical management of rhegmatogenous detachments with scleral buckling. Alternative methods of repair are discussed in Chapters 8 and 9, and the three techniques are compared in Chapter 10.

Controversy exists regarding the details of the surgical technique, but surgeons generally agree on the three basic steps in closing retinal breaks and reattaching the retina:

1. Conducting thorough preoperative and intraoperative examinations with the goal of locating all retinal breaks and assessing any vitreous traction on the retina.
2. Creating a controlled injury to the retinal pigment epithelium and retina to produce a chorioretinal adhesion surrounding all retinal breaks so that intraocular fluid can no longer reach the subretinal space.
3. Employing an appropriate technique, such as scleral buckling and/or intravitreal gas, to approximate the retinal breaks to the underlying treated retinal pigment epithelium.

If the surgeon follows these basics and applies modern surgical techniques, retinal reattachment may be expected following a single operation in more than 85% of uncomplicated primary detachments, and in more than 95% following additional procedures.
The traditional scleral buckle has served very well since the 1950’s. However, more recent developments have produced a more comprehensive menu for retinal reattachment surgery from which the surgeon may select the appropriate procedure for each case. By the turn of the millennium, surveys had demonstrated that scleral buckling alone was no longer the most popular means of repairing uncomplicated primary retinal detachments. Still, it is a valuable technique that is indicated in many situations.

Temporary scleral buckling can be performed with scleral infolding, gelatin, or orbital balloon. The term *scleral buckling* without a qualifying adjective is generally recognized as referring to a “permanent” scleral buckle with the implantation of a foreign material usually made of silicone.

Successful scleral buckling depends upon a thorough understanding of the anatomical and physiological effects of the procedure. These are substantially different from those associated with Pneumatic Retinopexy (Chapter 8) or Vitrectomy (Chapter 9).

**ANATOMICAL AND PHYSIOLOGICAL EFFECTS OF SCLERAL BUCKLES**

Anatomical changes in the vitreous gel and defects in the retina are of fundamental importance as causes of subsequent retinal detachment, as noted in Chapter 2. Scleral buckling of the eye wall and retina favorably alters pathoanatomy and allows normal physiological forces to reattach the retina.

**PATHOPHYSIOLOGY OF RHEGMA TOGENOUS RETINAL DETACHMENT**

Rhegmatogenous retinal detachment occurs when factors maintaining attachment are overwhelmed by the volume of intravitreal fluid entering the potential subretinal space through retinal breaks, as noted in Chapter 2. Isolated defects in the retina are usually not solely responsible for clinical retinal detachments. In the vast majority of cases, vitreoretinal traction forces upon the retina near the location of the break(s) are required for a progressive accumulation of fluid in the subretinal space. Liquid currents in front of retinal breaks and in the subretinal space contribute secondarily to progression of retinal detachment.

**REATTACHMENT FORCES INFLUENCED BY SCLERAL BUCKLES**

Localized indentation of the sclera, choroid, and pigment epithelium beneath a retinal break alters the anatomical and physiological factors associated with the production of a retinal detachment. The fundamental goal of scleral buckling is the functional closure of all retinal breaks, so that normal physiological forces can maintain a permanent state of attachment. Drainage of subretinal fluid and scleral buckling will usually close the responsible break(s) immediately.

In a non-drainage procedure, functional closure of retinal breaks can result from several beneficial effects of a scleral buckle, including:
1. reduction of vitreoretinal traction by displacing the eye wall and retina centrally;
2. displacement of subretinal fluid away from the location of the retinal break and scleral buckle;
3. postoperative increase in the height of the scleral buckle;
4. approximation of the retinal break and adjacent vitreous gel;
5. increase in resistance to fluid flow through the retinal break, with consequent increase in the relative reattachment forces;
6. alteration in the concave shape of the eyeball, resulting in a change in the effect of intraocular currents that encourage liquid vitreous to enter the subretinal space.

These effects are probably synergistic, and are also important in drainage cases. Although contemporary scleral buckling procedures routinely include the creation of a chorioretinal burn, such an adhesion is not always necessary to maintain retinal reattachment.

PRINCIPLES OF SCLERAL BUCKLING

The most important skill required in surgery for retinal detachment is the ability to detect all retinal breaks and additional areas of vitreoretinal pathology. Scleral buckling is performed to produce functional closure of retinal breaks responsible for retinal detachment and to reduce the chances of recurrent detachment. Various kinds and shapes of silicone are used, including segments of silicone sponge as well as solid silicone shaped into bands for encircling the eye and into additional forms to augment the width and height of the buckle in selected areas (Figure 7–1). The specific configuration of the scleral buckle depends upon a number of factors.

Following localization and treatment of retinal breaks and areas of vitreoretinal degeneration, the silicone buckling element is sutured to the scleral surface. Drainage of subretinal fluid is performed in the majority of cases. Intravitreal gas injection is sometimes employed in conjunction with scleral buckling. Problems encountered at any point of the procedure may require modifications in technique.

SCLERAL BUCKLE CONFIGURATION

The location, number, size, and types of retinal breaks are important variables affecting the selection of a specific buckling technique. Similarly, the presence of vitreoretinal degeneration, with or without retinal breaks, and of significant vitreoretinal traction unassociated with retinal breaks should be considered in the preoperative assessment (Figure 7–2).

If retinal breaks, vitreoretinal degenerative disorders, and significant vitreoretinal traction are present in multiple quadrants, a circumferential buckle is probably favored. A single retinal break unassociated with additional significant problems is usually managed with an isolated segmental buckle, if not with pneumatic retinopexy.
The anterior–posterior dimensions of retinal break(s) and areas of significant vitreoretinal degeneration and vitreoretinal traction are also important considerations in planning a buckling procedure. Scleral buckles should support all edges of the retinal breaks and associated areas of vitreoretinal degeneration. In general, the buckling effect should extend into the zone of the vitreous base to eliminate current and future traction forces.

Figure 7–1. A wide variety of solid silicone and silicone sponge materials are available for scleral buckling. (Courtesy of MIRA, Inc.)
The internal changes caused by scleral buckling are determined by the size, shape, and consistency of the buckling material, the width of the suture bites placed to attach the silicone to the sclera, the tightness of the tied sutures, and the extent to which an encircling element is tightened.

A scleral buckle is associated with a significant displacement of intraocular volume. In order to avoid a large increase in intraocular pressure, drainage of subretinal fluid, paracentesis, or removal of liquid vitreous is usually necessary in cases in which an extensive buckling effect is desired. This is particularly true in eyes with compromised aqueous outflow and in those in which recent anterior segment surgery has been performed.

**THE SCLERAL BUCKLING OPERATION**

The procedure involves routine prepping and draping, conjunctival incision, identification and treatment of all retina breaks and areas of vitreoretinal degeneration, suturing buckling material to the sclera, and additional techniques as indicated.

**PREP AND DRAPE**

For scleral buckling, the face, eyelids, and conjunctiva are prepared with appropriate antiseptic techniques, including povidone iodine solution, and the drapes are applied. The eyelid margins are the part of the operative field most susceptible to infection; therefore, a plastic adhesive drape placed over the opened lids so that the drape adheres to the eyelids and adjacent part of the face is recommended. An incision is made in the plastic drape in the plane of the palpebral fissure, and the free margins of the cut drape are rolled posteriorly over the lid margins and held securely in place with the eyelid speculum.
CONJUNCTIVAL INCISION AND ISOLATION OF RECTUS MUSCLES

A 360-degree limbal conjunctival incision is usually made, but a circumferential incision 4–5 mm posterior to the limbus is sometimes done. The conjunctiva and Tenon’s capsule are fused together near the limbus, so both tissues can be incised together. The peritomy is usually supplemented by two relaxing incisions perpendicular to the limbus over the lateral rectus muscles. A five clock-hour peritomy may be sufficient for a quadrantic detachment when a radial buckle is planned, exposing the two rectus muscles bordering the involved quadrant.

Traction sutures are placed beneath the insertions of the exposed rectus muscles to facilitate positioning the globe (Figure 7–3). Rather heavy suture material, such as 2-0 silk, is recommended. After the traction sutures are placed, the sclera should be examined. It can be exposed for inspection by rotating the globe with two adjacent traction sutures and pushing the intermuscular fascia posteriorly with a cotton-tipped applicator. The surgeon should look for abnormalities such as anomalous vortex veins or scleral thinning. Thinning is usually seen as radial gray lines in the equatorial or pre-equatorial area, particularly superior temporally. These lines are called radial staphylomas, scleral cracks, or scleral dehiscences (Figure 7–4).

After examination of the sclera, the surgeon should carefully examine the retina 360 degrees with binocular indirect ophthalmoscopy and scleral indentation. The investment of a few minutes at this time will pay dividends. Important lesions that were not disclosed preoperatively may become apparent, and occasionally this

Figure 7–3. A 360-degree peritomy 2–3 mm behind the limbus. The four rectus muscles have been exposed and isolated on large sutures. Depending upon surgeon preferences, peritomies can be limited in extent and/or located at the limbus.
examination reveals significant changes that have developed since the preoperative examination.

**Localizing of Breaks**

A localizing mark is made on the sclera overlying the posterior edge of the retinal break(s) with a scleral marking device. The tip of the scleral marker is firmly pressed against the eye for a few seconds, and the pressure creates a temporary black mark on the sclera by dehydrating the sclera. Alternatively, a flat diathermy probe can be used, yielding a light burn. The localization site is immediately touched with a marking pen, because the pressure effect or light scleral burn disappear quickly (Figure 7–5).

If a break is large, marks should ideally be placed at the posterior, anterior, and lateral margins (Figure 7–6). For smaller, routine tears, some surgeons prefer to localize with a single mark placed at the center of the anterior edge of the tear, preferring this location because (1) the anterior edge is the usual site of persistent vitreoretinal traction, (2) the anterior edge is always easier to mark than the posterior border, (3) a buckling effect extending from the anterior edge into the area of the vitreous base is desirable, and (4) it is relatively easy to estimate the amount of buckling effect required more posteriorly to support the respective break(s). Other surgeons prefer a single mark placed at the center of the posterior edge of the tear to help ensure that no part of the tear falls too far back on the posterior slope of the buckle. When positioning the buckle relative to the mark, the surgeon must keep in mind whether the posterior or anterior edge of the tear was marked.

Accurate marking is easily performed if the detachment is sufficiently flat to allow the retinal pigment epithelium to be pressed inward against the sensory retina. If the detachment is highly bullous, this is not possible and the surgeon must try to compensate for the parallax effect. With bullous detachments, the tendency is to localize the break too far posteriorly. If the surgeon is aware of the problem, an adequate anterior compensation can be made.
THERMAL TREATMENT

Diathermy, cryotherapy, and laser photocoagulation have been employed to irritate the choroid and pigment epithelium so that they form chorioretinal adhesions to seal retinal breaks. Cryopexy is most commonly used with scleral buckling.

Cryotherapy

The use of cryotherapy has gained wide acceptance since its reintroduction in 1964. A survey of retinal surgeons revealed that the vast majority of surgeons now employ it for most buckling cases. Although it is possible to apply cryotherapy beneath partial-thickness scleral flaps, it is not necessary to do so.

The goal of cryotherapy is to apply contiguous lesions completely surrounding each retinal break and areas of vitreoretinal degeneration. A single row of...
confluent freeze spots is generally sufficient, although more than one row may be required anteriorly to extend the future adhesion into the vitreous base.

Cryotherapy should be applied while observing the fundus with the ophthalmoscope, using the cryoprobe in place of a scleral depressor (Figure 7–7). A prominent white change develops at the area of retinal freezing. The freezing is generally terminated soon after the surgeon observes the appearance of an ice ball in the plane of the neurosensory retina. In bullous detachments, the ice ball is not allowed to reach the retina itself, and the freezing of the retinal pigment epithelium is discerned by a change of color to dull orange or gray. If portions of the break remain highly elevated during scleral depression with the cryoprobe, treatment can be postponed until some of the subretinal fluid is drained.

A challenge with using cryotherapy is the inability to see what areas of the retina have been treated for many minutes to days following treatment. This can lead to overtreatment if sequential freezes overlap much, and this excessive therapy should be avoided. However, an inadequate adhesion will result if the burns are not confluent in appropriate areas. Thus, the surgeon must form an optimal visual image of the precise limits of prior applications of cryotherapy, and considerable experience is usually required to master this technique.

Cryotherapy burns should extend only to the edges of medium and large retinal breaks from retina surrounding them. If the pigment epithelium lying beneath
the break is included in the cryo burn, an intravitreal dispersion of pigmented cells capable of proliferation can occur (Figure 7–8). For the same reason, scleral depression of a treated area at the edge of a break should not be repeated after the treatment, and localization of retinal breaks should always precede cryotherapy.

Diathermy
Diathermy is no longer employed by most surgeons. It should only be applied through thinned sclera, beneath partial-thickness scleral flaps, to avoid full-thickness destruction of sclera and achieve a more uniform intraocular reaction. The surgeon may apply the diathermy while inspecting the retina with the ophthalmoscope, limiting the intensity to that required to produce a moderately white lesion in the retina. Alternatively, an experienced surgeon can apply diathermy without ophthalmoscopic control, predicting the probable ophthalmoscopic result on the basis of the scleral reaction. Diathermy burns are spaced approximately 1.5 mm apart, producing intermittent pigmentary changes in the fundus that have been referred to as “leopard skin.”

Intraoperative laser photocoagulation
Photocoagulation is not usually used to induce adhesive lesions in detached retina, because the retinal pigment epithelium is not sufficiently close to the retina to cause a burn. Although the surgeon can close retinal breaks with scleral buckling and/or intravitreal gas and then attempt to apply laser photocoagulation, this may
lead to overtreatment. Laser photocoagulation to detached areas is usually easier a
day or so following the operation. Attached retinal breaks may be lasered intraop-
eratively with the laser indirect ophthalmoscope.

**BUCKLING MATERIALS**

Various materials have been used for scleral buckling, including fascia lata, pal-
maris tendon, plantaris tendon, knee cartilage, donor sclera, dura mater, polyviol,
polyethylene, encircling nonabsorbable and absorbable sutures, gelatin, hydrogel,
and silicone. By far the most popular, silicone, is a soft, synthetic rubber material
that is nontoxic and nonallergenic. Readily available, it is produced in a variety
of molded shapes that can be modified by the surgeon and used in either solid
or sponge form (Figure 7–1). Implants can be placed within the sclera or on its
surface. Although they are not technically “implants” in the latter position, the
term has persisted for decades and will be employed in this monograph. Episcleral
implants are currently employed in the vast majority of cases. These can be seg-
mental or encircling. Silicone implants are safe and very different from the silicone
materials used for breast implants.

**Segmental episcleral buckles**

Segmental silicone exoplants are secured to the sclera with 5-0 nonabsorbable syn-
thetic mattress sutures attached to spatula needles with cutting tips. One half to
two-thirds thickness intrascleral passes at least 6 mm long are usually attempted
for sponges and broad exoplants, whereas shorter intrascleral passes can be used to
support encircling bands. The surgeon applies focal pressure with a cotton appli-
cator near the intended suture site to prevent buckling of the sclera in front of the
needle as it is passed through the sclera. Alternatively, a nearby muscle insertion
can be grasped with forceps to provide increased stability and to elevate intraocu-
lar pressure. The location of the needle tip should be visualized at all times during
its passage through the sclera.

Localized scleral buckles may be radially or circumferentially oriented, and a
combination of the two may be considered if more extensive buckling is required.
Radial scleral buckles provide focal support for a retinal tear and minimize the
development of radial retinal folds commonly associated with circumferential
buckles, which shorten the circumference of the eye wall but not the circumference
of the retina. Circumferential buckles provide a zone of support oriented parallel
to the region where vitreous traction is usually most severe, and are an efficient
means of supporting multiple areas of vitreoretinal pathology.

When tightened, the sutures indent the exoplant and underlying eye wall. Using
an exoplant of a given shape, the height of the buckling effect is determined by the
distance between the suture bites and the tightness of the sutures when they are
tied. Calipers are used to measure the distance between the suture bites, and these
should be 2–3 mm wider than the exoplant for a modest buckling effect, and 4–8
mm wider for a relatively high buckle.

When a radially oriented segmental buckle is needed, intrascleral suture limbs
are placed parallel to the meridian of the retinal break and equidistant from its
edges, and the needle is passed so that the knot can be tied posteriorly (Figure 7–9). The size and type of the retinal break usually dictate the width and length of the exoplant. In general, the width of the silicone element should be at least as wide as the edges of the break marked on the sclera, and the buckle length should support both the posterior end of the break and the vitreous base anterior to the break.

If a circumferentially oriented segmental buckle is required, suture limbs are placed parallel to the limbus. The anterior bite is usually placed just anterior to the posterior margin of the vitreous base, a location estimated as lying about 2–3 mm posterior to an imaginary line drawn between the muscle insertions (and forming a portion of the spiral of Tillaux). A silicone element of sufficient width to support both the anterior and posterior edges of the retinal break(s) or other pathology is used, and the posterior suture bite is placed in a position that will produce an optimal buckling effect.

**Encircling episcleral buckles**

If a 360-degree encircling circumferential buckle of modest width and height is needed, an encircling 240, 41, 42, or other style silicone band is passed around the circumference of the globe and beneath the rectus muscles. Sutures are then placed. The band is traditionally anchored with a single mattress suture with bites parallel to the limbus placed in the center of each quadrant (Figure 7–10). Although they are an elegant and effective means of securing a band, scleral tunnels are usually not employed by most surgeons.

Suture bites that straddle a silicone band should be placed just far enough apart to allow the band to move freely beneath the suture, and this distance equals the width of the band plus two times its thickness. Narrower bites inhibit circumferential

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**Figure 7–9.** For a meridional segmental buckle, broad and long mattress sutures are placed perpendicular to the limbus in an effort to place the intended silicone sponge directly beneath the marked and treated retinal break.
movement of the band, particularly if the sutures are pulled tight. Wider bites will allow the band to move anterior to its desired location when its ends are joined. In its proper position, the band is usually intended to support breaks in the region of the posterior edge of the vitreous base, and these are marked and treated before suture placement. In quadrants without retinal breaks, the vitreous base margin can be marked, or its location can be estimated and the anterior suture bite placed about 2–3 mm posterior to the imaginary line mentioned earlier.

When using segmental scleral buckles produced by circumferentially oriented silicone materials, suture bites are always placed in the location overlying the responsible retinal break(s), because the maximum buckle height is produced in this spot. However, with encircling buckles, if additional height may be required in a localized zone, the sutures over the band should be placed elsewhere so that additional elements can easily be inserted beneath the band in that location. If an increased buckling effect is needed in only a small area, a radially oriented piece is used in this situation. If a grooved segment of silicone tire is required because of a need for more extensive circumferential augmentation of the buckle, additional sutures may be required, depending upon the characteristics of the specific case.

If a high, broad 360-degree encircling scleral buckle is required, two broad mattress sutures are placed in each quadrant to accommodate a silicone tire of at least 7 mm width and an overlying silicone band. The anterior suture bite is placed at the estimated location of the ora serrata, and the posterior bite is placed far posteriorly, at a spot dictated by the width of the tire, the desired amount of indentation, and the location of tears. Frequently, this distance is equal to twice the distance from the anterior bite to the marked posterior edge of the retinal break(s) (Figure 7–11). If a vortex vein must be avoided with the posterior suture bite, two small circumferential passes can be made on either side of the vessel.

Figure 7–10. Encircling silicone bands are traditionally fixed with a single mattress suture located in the center of each quadrant. Some surgeons prefer scleral “belt loops” for this purpose.
The ends of an encircling band are joined with a silicone Watzke sleeve, suture, or tantalum clip. Tantalum clips are less popular than the sleeve or suture, primarily because of the extra time required for their use, particularly if later adjustment of the length of the band is required. Some degree of twisting of the band near the elastic sleeve can occur when the band is tightened, and this can be avoided by grasping each end near the sleeve and by keeping the ends flat against the periscleral portion already in place. The ends should be rejoined if twisting causes a narrow edge of the band to indent the sclera, as this can lead to later intrusion problems. Since even minimal twisting of the sleeve can affect the inner morphology of the buckling effect, the sleeve should be located in a quadrant relatively free of significant vitreoretinal pathology.

Alternatively, a 5-0 synthetic suture tied in a single loop around the overlapped ends of the encircling band minimizes twisting while allowing adjustment. To
increase the tension on the band, traction is placed on the two cut ends of the band, while a needle holder grasps the suture and allows it to slip. To decrease tension on the band, traction is placed on the two encircling ends instead.

The final result is a custom-fitted encircling indentation, with all retinal tears mounted on or just anterior to the crest (Figure 7–12). Indentation is modest in the uninvolved meridian of the retina and more pronounced as needed in areas of pathology.

THIN SCLERA

The most common problem requiring a modification in technique is the presence of thin sclera. When this is encountered, scleral suture bites must be placed in positions that are less potentially hazardous. In most such situations, this can be accomplished with suture passes on either side of the ectatic sclera and/or with the use of a wider piece of silicone buckling material. More exotic means of managing thin sclera with donor sclera or tissue glue have been described, but pneumatic retinopexy or vitrectomy without scleral buckling is usually employed if scleral suturing appears to be impossible.

INTRASCLERAL BUCKLES

Rarely used today, intrascleral buckles involve lamellar dissection to create a partial-thickness scleral bed. They usually involve only a limited part of the circumference of the globe, but can vary in length from one to twelve hours of the clock and in width from 4 mm to 12 mm. An encircling band is usually used, attached directly to the surface of the sclera in the areas not undermined, and the ends are joined together with only moderate tension. The implant is placed in the bed of the undermining, and the flaps are then closed over the top with sutures. This “trapdoor procedure” creates a satisfactory buckle (Figure 7–13).
Decisions regarding the drainage of subretinal fluid are among the most difficult associated with scleral buckling procedures, and considerable differences in opinion exist. Drainage is almost never performed if responsible breaks can be easily and almost completely approximated to the pigment epithelium with a non-drainage technique. Drainage is almost always performed if a high and broad encircling scleral buckle is required. However, in most cases the criteria for drainage or non-drainage are less obvious. In eyes in which drainage was considered to be neither clearly unnecessary nor mandatory, a small randomized trial demonstrated comparable results with both techniques.

In most cases, the decision regarding drainage depends upon the size and configuration of retinal tears, the amount of traction, the appearance after the scleral buckle has been elevated beneath the retinal break(s), and the experience of the surgeon regarding the amount of subretinal fluid that can be allowed to remain between the crest of the buckle and the break(s). Most non-drainage procedures are effective if the crest of the buckle is within 3 mm of the respective retinal break. Although this may be relatively easy to accomplish when buckling a single break, the need for more extensive buckling of multiple breaks makes this a more difficult goal and favors a drainage procedure.

Because of complications associated with drainage of subretinal fluid, it is avoided unless considered to be necessary for surgical success. If failure to drain results in persistent subretinal fluid postoperatively, an intravitreal gas injection performed in the office can frequently cause the fluid to settle.

Nevertheless, most surgeons perform transcleral drainage of subretinal fluid in approximately 75% of retinal detachment cases managed with scleral buckling. However, with increasing popularity of primary vitrectomy for retinal detachment, many surgeons now consider vitrectomy in cases which are likely to require

**Management of Subretinal Fluid**

Figure 7–13. Creation of scleral flaps for a segmental “trap door” buckling procedure. Buckling procedures featuring scleral dissection have become much less popular over the past three decades.
drainage, and use drainage in a smaller percentage of scleral buckle cases. Still, drainage and non-drainage techniques play a major role in the management of a routine series of cases, and familiarity with both techniques is essential.

**Non-drainage technique**

Some surgeons perform drainage of subretinal fluid in a large majority of cases, whereas other surgeons drain infrequently. The most common indication for a non-drainage technique is a retinal detachment due to a single break that can be approximated close to the pigment epithelium by scleral depression.

Following the placement of appropriate sutures, the scleral buckle is elevated to the desired height, and the proximity of the retinal break to the surface of the buckle is reevaluated. If the break is not perfectly positioned, the scleral buckle must be adjusted. The breadth of the buckling effect can be extended with additional sutures placed anterior or posterior to those already holding the buckle. If the posterior edge of the break is not positioned on the center of the buckle crest, at least one arm of the mattress suture must be repositioned.

**Assuring perfusion of the central retinal artery**

Unless fluid has been removed from the eye, placement of a scleral buckle will usually raise intraocular pressure so high that the central retinal artery is no longer normally perfused. This is temporarily tolerable, but pressure must be lowered and at least pulsating perfusion restored within five or ten minutes. Whether or not subretinal fluid is drained, some perfusion of the central retinal artery must be confirmed by the end of the case.

It can be difficult to tell whether the central retinal artery is patent. If pulsations of the central retinal artery are observed, perfusion is marginally sufficient. If pulsations are not visualized and perfusion is questioned, additional digital pressure should be applied to the globe to elicit pulsations. If these do not occur, the arterial flow into the eye has probably ceased, and intraocular pressure must be reduced if pulsations do not begin soon. Intraocular pressure can be reduced by drainage of subretinal fluid, paracentesis, aspiration of fluid vitreous, reduction in circumferential contraction of the buckle, or relaxation of sutures causing indentation of the buckle.

Paracentesis is usually performed in non-drainage procedures to achieve timely reduction of intraocular pressure after placement of the buckle, although this may not be necessary if only a single radial buckle is placed. If repeated paracenteses cannot reduce intraocular pressure sufficient to reopen the central retinal artery, pressure can also be reduced by aspirating fluid from the posterior vitreous cavity. However, this maneuver is associated with many more potential complications than paracentesis.

If optimal buckle size and suture width have been employed, the height of the buckle can be increased by a further tightening of mattress sutures after intraocular pressure has returned to relatively normal levels. Sutures are initially tied temporarily to facilitate this adjustment. Perfusion (or at least pulsation) of the central retinal artery must be documented each time that the sutures are tightened; this is particularly important in eyes with reduced outflow facility.
Drainage technique

Drainage of subretinal fluid is performed at a site determined by the configuration of the retinal detachment, where sufficient subretinal fluid allows for safe drainage. Factors considered in the selection of a drainage site include (1) the distribution of subretinal fluid when the eye is in a position at which drainage will be performed, (2) the location and size of the retinal break(s), (3) the location and configuration of the buckle, (4) the vascularity of the choroid, (5) features of vitreoretinal and epiretinal membrane traction, and (6) the ease of exposure of the proposed drainage site.

The optimal locations for drainage are usually just above or below the lateral rectus muscle, because major choroidal vessels are avoided and exposure of sclera is excellent. Choroidal vessels are also avoided by draining on either side of the three remaining rectus muscles, but exposure is frequently more difficult. If possible, drainage is usually performed some distance from retinal breaks (especially large retinal breaks) so that the passage of vitreous through the break(s) and out of the eye can be minimized. The scleral depression effect provided by the buckle or by a cotton-tipped applicator can help prevent this occurrence. In unusual situations in which a large buckling effect is required and very little subretinal fluid exists, drainage can be performed immediately beneath a large tear to allow both subretinal and intravitreal fluids to exit the globe.

Drainage is performed prior to tightening the scleral buckling elements onto the eye, since a high intraocular pressure at the time of drainage increases the risk of complications. A site is usually selected at or slightly anterior to the equator; a location that will ultimately be closed by the exoplant is always preferred. This avoids the need for a preplaced suture at the sclerotomy site, and it facilitates subsequent management of drainage complications, as is noted later. In the situation in which drainage cannot be performed optimally at a site intended to be covered by the buckle, a preplaced suture is employed to close the scleral incision following drainage. This suture is placed after the sclera incision is made, but before the choroid is penetrated.

A 3–4 mm radial incision through the sclera is then performed so that the center of the sclerotomy will be at the appropriate location. All scleral fibers are carefully divided until subtle prolapse of uveal tissue is observed (Figure 7–14). The choroid is then closely inspected for prominent choroidal vessels, using loupes and/or the 20 diopter condensing lens and indirect ophthalmoscope. If large visible vessels cannot be avoided during penetration, a second site nearby is selected, and another scleral incision is performed. If the area of exposed choroid is free of prominent vessels, it is lightly treated with a flat diathermy probe. This causes minimal retraction of the edges of the sclera to improve visualization, and it may reduce the risk of hemorrhage.

All significant traction upon the eye is eliminated to reduce intraocular pressure as much as possible. The choroid is then penetrated with a sharp-tipped conical penetrating diathermy electrode or suture needle. Modest pressure is used to insert the device perpendicular to the surface of the sclera until the subretinal space is entered.

If the conical diathermy electrode is used, this event is usually heralded by a sudden, subtle “pop,” which is usually perceived by touch or observation. Because
of the tapered shape of the electrode, significant amounts of subretinal fluid do not exit the eye until this device is very slowly withdrawn from the eye. The penetrating diathermy electrode is relatively blunt, compared to a suture needle, and penetration of a soft eye with congested choroid may be somewhat difficult. This is managed by modest elevation of intraocular pressure with traction upon the muscle fixation sutures.

An oblique or tangential path of penetration is recommended by some authors to avoid perforating the retina, but this can result in a flap valve of the choroid, which can limit drainage. If a proper drainage site has been selected, penetration of the retina with the tapered diathermy is exceptionally rare, because it is removed prior to the release of significant amounts of subretinal fluid. Lasers have also been employed to drain subretinal fluid, but the expense and time required to use them likely aren’t justified by a significant reduction in the rate of complications.

As the globe softens during drainage, intraocular pressure is very slowly increased to encourage further drainage and to avoid complications associated with hypotony. If a large tear is present, the sclera or the buckle and sclera overlying the break are indented with a cotton applicator. This maintains intraocular pressure and inhibits passage of intravitreal fluid to the subretinal space. Pressure can also be increased by placing applicators on either side of the sclerotomy site and gently pushing them toward the center of the eye; these maneuvers also tend to keep the sclerotomy open. Relatively normal intraocular pressure can also be maintained by indenting the sclera at a location far from the sclerotomy site with a number of cotton applicators.

The drainage site is not touched as long as fluid flows through it. Sudden and significant increases in intraocular pressure are avoided to reduce chances of incarceration of the retina in the sclerotomy, and any sudden cessation of drainage requires immediate closure of the sclerotomy and internal examination of the sclerotomy site with the indirect ophthalmoscope. The appearance of pigment

Figure 7–14. Traditional drainage of subretinal fluid is performed through a scleral incision that exposes bare choroid. A penetrating diathermy pin, suture needle, or other sharp instrument may be employed for this purpose.
granules suspended in the draining subretinal fluid usually indicates that the last of the subretinal fluid is exiting the eye. When drainage ceases, the sclerotomy site is closed by temporarily tying the sutures over an exoplant or by pulling together the ends of an encircling band. If the buckling material does not adequately close the sclerotomy, the scleral incision is closed with a suture prior to significant elevation of intraocular pressure with the buckle.

The eye is quickly inspected following closure of the sclerotomy site and preliminary adjustment of the scleral buckle. The site of drainage is first evaluated for signs of subretinal bleeding, retinal incarceration, and iatrogenic hole formation; management of these relatively unusual intraoperative problems is discussed later. The amount of persistent subretinal fluid is then determined, and the need for further drainage is considered. Significant subretinal fluid is allowed to persist if the optimal amount of buckling nearly closes the retinal break(s).

If drainage of additional subretinal fluid is required, the initial sclerotomy site must be closely evaluated with mobile scleral depression, in which a cotton-tipped applicator is rolled circumferentially beneath the area of drainage. If the pigment epithelium is clearly not in contact with the retina, the sclerotomy site can be reopened by reducing intraocular pressure and/or removing the portion of the exoplant that covers the scleral incision. Additional drainage usually occurs spontaneously, or it can be initiated by gently manipulating the edges of the sclerotomy with applicators or forceps. In some cases, particularly those with exceptionally viscous subretinal fluid, the retina may flatten completely at the site of the sclerotomy, while large amounts of subretinal fluid persist elsewhere. In this situation, additional sclerotomies must be performed if additional drainage is required to produce an adequate buckling effect.

An alternative and increasingly popular method of draining subretinal fluid is to insert a small 25–27-gauge needle into the subretinal space under direct visualization with the indirect ophthalmoscope (Figure 7–15). The needle is usually attached to a tuberculin syringe from which the plunger has been removed. Digital pressure is exerted on the eye or significant intraocular pressure is maintained with traction sutures as the subretinal fluid passively exits the eye. The needle is dynamically positioned to remain within subretinal fluid, and it is slowly retracted as the retina approximates its tip.

**Adjustment of Scleral Buckle**

Following drainage of appropriate amounts of subretinal fluid, an optimal scleral buckling effect is created by adjusting the scleral sutures and the length of the segmental element or the tightness of the encircling band. Broad sutures over the portion of the buckle supporting large retinal breaks are first temporarily tied in a manner intended to provide optimal width and height. If intraocular pressure remains low and the breaks are in optimal position, the sutures are permanently tied. The height of the buckle is adjusted if it is inadequate or excessive. If a fold of retina continues to communicate with an open retinal break (“fish-mouth phenomenon”) following buckle adjustment, a variety of manipulations can be used to solve the dilemma, as is noted later.
If an encircling band has been used in combination with a wider circumferential tire of hard silicone, its ends are overlapped to provide a modest buckling effect supporting the posterior edge of the vitreous base in areas not occupied by the tire. If the encircling band is used without a tire, it is adjusted to create an indentation of somewhat greater height. The degree to which the band should be tightened depends upon the intraocular pressure, the nature and extent of vitreoretinal pathology, and the necessity of a subsequent intravitreal gas injection. If the intraocular pressure remains quite soft following appropriate adjustment of the scleral buckle and the retinal breaks are flat, a balanced salt solution should be injected into the vitreous cavity via the pars plana. Attempts to restore normal pressure with further increases in buckle height can lead to a number of postoperative problems. Indications for gas injections are described below.

The need to replace buckling materials or sutures is quite unusual if appropriate localization of retinal breaks has been performed. However, augmentation of the buckle in certain areas is frequently desired. This can be accomplished with suture adjustments if wide elements are already in place. If an encircling band has been used, and an augmentation of the buckle is needed in an area supported only by the band, hard silicone pieces of an appropriate size may be placed beneath the band; these usually are not sutured.

**Accessory Techniques**

Although scleral buckles are successful in producing a functional closure of retinal breaks in most cases, their effectiveness can be enhanced with accessory techniques, including intravitreal fluid and gas injections, gas–fluid exchanges, and postoperative laser photocoagulation. The first option, injection of a balanced salt
solution, is employed primarily to restore normal intraocular pressure to a hypotonous eye. Intravitreal gas injection and air fluid exchange are usually used to assist in the internal closure of retinal breaks. Laser photocoagulation is usually not used at the time of scleral buckling, but can be employed to create or augment a chorioretinal adhesion postoperatively.

**Intravitreal injection of balanced salt solution**

The solution is drawn into a syringe, and all air is carefully eliminated. A 0.5-inch, 30-gauge disposable needle is recommended. As the surgeon grasps the sclera with a twist pick or forceps, the needle is introduced into the vitreous cavity 3 mm (pseudophakic/aphakic eye) or 4 mm (phakic eye) posterior to the limbus. The tip of the needle is directed toward the geometric center of the globe. In a very soft eye, the tip of the needle may occasionally elevate the pars plana epithelium without perforation. Injection in that situation would produce detachment of the pars plana and retina. This can be avoided by direct visualization of the tip of the needle through the pupil and, when the needle is definitely within the vitreous cavity, the injection can be safely carried out. In phakic eyes, care must be taken to keep the tip of the needle near the middle of the vitreous cavity to avoid touching the lens and causing a subsequent cataract. After injection of the required volume to restore normal intraocular pressure, the needle is withdrawn. The self-sealing wound does not require suturing.

**Intravitreal gas injection**

Gas injections to internally tamponade retinal breaks are commonly performed in association with scleral buckling procedures. Gas is usually injected after the breaks have been treated and well positioned on the scleral buckle. The type and volume of injected gas depend upon the available potential space within the vitreous cavity, as well as the size of retinal break(s) and the desired duration of tamponade.

In the vast majority of cases in which the buckle is in appropriate position, an effective tamponade is necessary for only 24–48 hours. A 1.0 ml bubble of air is usually quite sufficient to tamponade a large retinal break, as a 0.30 ml bubble will maintain contact with a 90-degree arc of the retina. A 1.0 ml bubble of air is not absorbed for three to four days. However, an injection of even 0.3 ml of gas into an eye with normal intraocular pressure will cause a marked increase in intraocular pressure and a transient occlusion of the central retinal artery. Therefore, the eye must be quite soft prior to an injection of a large gas bubble, or a smaller volume of an expansile gas is employed. In cases in which a longer tamponade effect is desired, more insoluble gases such as sulfur hexafluoride ($\text{SF}_6$) and perfluoropropane ($\text{C}_3\text{F}_8$) are used. These possess two potentially favorable characteristics: expansile qualities if injected as pure gas, and longer duration in the eye.

Numerous techniques of gas injection have been described. A simple method is to grasp a muscle insertion to fixate the eye and to penetrate the globe with a 30-gauge needle attached to a tuberculin or 3 ml syringe containing the desired amount and concentration of gas. The injection is performed 4 mm posterior to the limbus in a phakic eye, and 1 mm closer to the limbus in an aphakic or pseudophakic case. The
site of injection is made uppermost so the bubble will tend to remain at the site of the needle tip, to avoid formation of multiple small bubbles. Using indirect ophthalmoscopy, passage of the needle tip through the pars plana epithelium may be confirmed. The needle is then withdrawn enough to leave about 3 mm of the needle in the eye. The predetermined volume of gas is injected moderately rapidly.

The optic nerve is then inspected to document perfusion of the retinal vessels. If pulsations are visualized in a patient with normal blood pressure, no tension-lowering manipulations are performed. If pulsations are not observed and cannot be produced with digital pressure, the intraocular pressure is lowered with a paracentesis if pulsations have not resumed after several minutes. The height of the buckle can also be reduced or some of the gas can be removed if necessary to restore patency of the central retinal artery.

**Gas–fluid exchange**

When a relatively large volume of gas is required and intraocular pressure cannot be adequately lowered by any other means, fluid can be removed from the vitreous cavity prior to gas injection. This is commonly performed during vitreous surgery, but rarely during routine scleral buckling operations. If a total posterior vitreous detachment has been documented preoperatively, and the retina is relatively flat, fluid can be aspirated from the space behind the posterior hyaloid with a 25-gauge needle inserted via the pars plana. It is helpful to have a very small volume of balanced salt solution in the syringe. When the needle is in place, 0.1 ml of solution should be injected. This displaces vitreous gel at the tip of the needle and thereby facilitates the aspiration of fluid vitreous. Alternatively, a vitrectomy-cutting instrument can be used under indirect ophthalmoscopic control. This alternative has the advantage of reducing possible vitreoretinal traction, but involves extra cost and time for setting up equipment. Gas is injected after the eye has been softened by either technique.

**Postoperative laser photocoagulation**

In cases in which cryotherapy can not be appropriately performed or excessive treatment is feared, retinal breaks can be treated with laser therapy after the retina is totally reattached with a scleral buckle. Some surgeons prefer this technique as a routine. In selected cases, recurrent retinal detachments following routine scleral buckling can be repaired by reattaching the retina with a gas bubble, followed later by laser photocoagulation.

**Closure of Incisions**

After placement and adjustment of the scleral buckle have been completed, any excess silicone or relatively sharp edges should be carefully trimmed, especially with very anterior buckles. Irrigation of the operative field with an antibiotic solution is usually performed. Approximately 5–10 ml is drawn into a syringe, and the field is then irrigated using a blunt-tipped needle. The irrigation should be deep in the plane between Tenon’s capsule and the sclera in all opened quadrants. Before any implant material is used, it should be soaked in the same antibiotic solution.
Limbal peritomies are frequently associated with two relaxing incisions perpendicular to the limbus. Each can be closed with one or two interrupted sutures or a running suture. Some postoperative suture discomfort can be eliminated by burying the suture knots in Tenon’s space. Both Tenon’s capsule and the conjunctiva may be closed together.

COMMON COMPLICATIONS OF SCLERAL BUCKLING

Complications have traditionally been divided into intraoperative and postoperative categories. Common intraoperative complications can make the operation more difficult and interfere with the ability to achieve surgical goals in an efficient fashion; familiarity with their management is essential. Complications that follow scleral buckling procedures include factors that affect anatomical and/or visual outcomes and patient satisfaction.

SELECTED INTRAOPERATIVE COMPLICATIONS

Corneal complications

Corneal epithelial edema or trauma to the epithelium impairs the clarity of the cornea and impedes optimal visualization of the retina at surgery. Several factors are responsible for a majority of the cases. It is routine in some operating rooms to instill preoperative topical anesthetics prior to dilating drops, and this practice should be discontinued. Topical anesthetics adversely affect the epithelium. For the same reason, concentrated detergents should not be used in the surgical prep.

Prolonged elevated intraocular pressure during surgery may result in edema of the epithelium, especially in elderly adults with an aging endothelium and in diabetic patients. Some cases respond well to mechanical expression of the edema fluid by pressing firmly on a dry, cotton-tipped applicator as it is rolled across the cornea. This maneuver expresses the edema but does not remove the epithelium. In persistent cases of epithelial edema, the central portion of the epithelium must be removed with gentle strokes of a tilted knife blade, avoiding damage to Bowman’s membrane.

Pupillary complications

Infrequently, there is minimal bleeding into the anterior chamber. As the blood settles onto the anterior lens capsule, the view of the fundus is obscured. A clear view can be regained by pushing the blood into the chamber angle by means of an intracameral injection of sodium hyaluronate.

Intraoperative miosis is due most commonly to hypotony. Intravitreal gas that comes in contact with the iris can also cause miosis. If the pupil does not dilate with cycloplegic and mydriatic drops, intracameral epinephrine and/or iris retractors may be considered.

Scleral perforation with suture needles

Penetration of the sclera with a suture needle most commonly occurs when non-drainage techniques are planned and a long, deep intrascleral pass of the suture
needle is required. Penetration of the globe is usually heralded by the sudden appearance of subretinal fluid at either end of the suture bite. Occasionally, pigment granules and blood are also observed. When an inadvertent penetration is recognized, all traction upon the eye should be immediately released, and the retina should be inspected with the indirect ophthalmoscope. The suture is temporarily left in place to minimize further drainage.

Choroidal hemorrhage is the most common complication of inadvertent penetration, occurring in approximately one-fourth of cases. Usually it is minimal, but if active bleeding is observed, the pressure in the eye should be immediately raised with scleral depression over the bleeding site. If the macula was involved in the detachment, blood can ultimately settle in that location and severely compromise visual acuity. This is best prevented by draining no additional subretinal fluid after the penetration. Postoperatively the patient is positioned so that the blood will settle away from the macula, and intraocular gas may also help express heme away from the macula in a face-down position.

If an inadvertent penetration causes an iatrogenic hole in the attached or detached retina, or if an iatrogenic hole cannot be ruled out, the site is treated with cryotherapy and supported on a modified scleral buckle. If it is near a retinal tear, a wider piece of silicone is sutured in place to support both the break(s) and the penetration site. In the rare circumstance in which formed vitreous passes into the penetration site through a large nearby tear or a new iatrogenic hole, the gel should be amputated at the sclerotomy site, which should be supported on a buckle.

Complications of draining subretinal fluid

This step of a scleral buckling procedure is usually considered the most hazardous. Although drainage complications are not statistically associated with subsequent anatomic failure, they can require a modification of the surgical plan and compromise postoperative visual acuity.

The three classic complications associated with drainage of subretinal fluid are hemorrhage, retinal incarceration, and iatrogenic retinal holes. The first of these is the most common, occurring in approximately 3%–4% of cases. Most hemorrhage is confined to a small area surrounding the sclerotomy site, and additional surgical maneuvers are not required. More significant hemorrhages are managed in a fashion similar to that described following inadvertent penetration of the globe with a suture needle.

Retinal incarceration is observed following drainage in 1%–3% of cases. This usually occurs soon after penetration of the choroid, and is associated with a sudden cessation of drainage. Increased intraocular pressure contributes to this problem. When retinal incarceration is suspected, all traction upon the eye is released, and the fundus is quickly examined with indirect ophthalmoscopy. Usually the incarceration is mild, with only a localized depression in the retina surrounded by radiating striae. The drainage site should be closed immediately with the exoplant or with a suture, then the retina should be more thoroughly evaluated with scleral depression. If an iatrogenic retinal break is discovered, it is treated with light cryotherapy. Incarceration sites are generally supported on a buckle in a manner similar to that described regarding inadvertent suture penetrations.
In the absence of incarceration of the retina, drainage of subretinal fluid rarely causes iatrogenic retinal breaks, but the drainage site is carefully evaluated by indirect ophthalmoscopy following completion of drainage to rule this out.

"Fish-Mouthing" of retinal breaks
This radial folding of the retina at the site of a large horseshoe tear is frequently observed following a circumferential scleral buckling procedure. The open break may prevent reattachment if it is not functionally closed. Increasing the height of the circumferential buckle is tempting but counterproductive, as it worsens the radial folding. The best way to manage this phenomenon is to reduce the height of the buckle beneath the break(s) and to inject an intravitreal gas bubble. Alternatively, an additional radial buckle may help.

Complications of intravitreal gas injections
The two common dilemmas that arise following intravitreal gas injections are difficulties in visualization and elevation of intraocular pressure, and the former can aggravate the management of the latter.

Although the minification of details and altered reflexes due to intravitreal gas bubbles makes visualization more difficult, it is relatively easy to see through the center of a bubble 1 cc or more in volume. Alternatively, the eye can be tilted so that the bubble will float out of the way. A more difficult problem occurs when a multitude of tiny bubbles prevent a meaningful view, and the frequency of this problem is reduced by employing optimal injection techniques as discussed above. If it occurs, the mass of bubbles will frequently float out of the way with appropriate tilting of the eye. Otherwise, a coalescence of bubbles can be encouraged by a series of jerks of the globe and the passage of time.

The optic nerve must always be visualized following an intravitreal injection of gas. Occasionally, extreme repositioning of the patient is required to be certain that the optic nerve and retina are perfused. If perfusion is questionable, increased digital pressure should be placed upon the eye to elicit pulsations. If these do not occur and if they are not visualized after several minutes, pressure-lowering maneuvers should be immediately instituted. The common options include paracentesis and lowering the scleral buckle height. In some pseudophakic cases, paracentesis is best performed via the pars plana.

Selected Postoperative Complications
Increased intraocular pressure
During the early postoperative period, corneal epithelial edema, pain, or pulsation of the central retinal artery can occur due to increased intraocular pressure. Patients likely to develop the complication include narrow-angle glaucoma suspects, elderly patients with enlarged lenses, those who have subluxated lenses, patients with preexisting open-angle glaucoma, those who received relatively large injections of intraocular gas or intracameral injections of sodium hyaluronate, and especially those with prominent postoperative choroidal detachment.
Moderate elevations to 30 mm Hg are frequently transient and usually do not require treatment. Higher pressures may be due to some degree of angle closure. The most common causative mechanism is anterior displacement of the lens and iris by the presence of choroidal detachment or swelling and subsequent compromise of the filtration angle. The condition is usually self-limiting and responds well to intravenous or oral acetazolamide (Diamox). Topical corticosteroids can be useful to discourage the development of peripheral anterior synechiae. If the administration of acetazolamide, topical glaucoma drops, hyperosmotic agents, systemic corticosteroids, and/or cycloplegic drops fails to control the pressure, and the filtration angle is obscured by peripheral iris, it may be necessary to drain the choroidal detachment via posterior sclerotomies to reopen the angle and prevent permanent anterior synechiae.

**Endophthalmitis**

Bacterial endophthalmitis is an exceptionally rare but potentially devastating complication following retinal detachment surgery. Possible routes of infection include the perforation site for draining subretinal fluid, accidental penetration or rupture of the globe, and intraocular injections. Lengthy procedures, extensive scleral surgery, reoperation, and multiple drainage attempts also increase the risk of bacterial contamination and postoperative endophthalmitis.

The earliest clinical symptoms and signs, developing by the third to fifth postoperative day, are pain, chemosis, lid edema, the appearance of localized vitreous opacification or petechial hemorrhages, and acute subretinal exudate at the level of the scleral buckle, noticeable in an examination by indirect ophthalmoscopy. The prompt recognition of such symptoms and signs, even if relatively subtle, is critical to proper management of endophthalmitis, for retinal damage is rapid, and soon the vitreous becomes opaque with inflammatory reaction.

The contemporary management of genuine endophthalmitis is beyond the scope of this chapter. A related condition, “scleral abscess,” usually presents as a sterile vitreous inflammatory reaction combined with evidence of severe presumed infection of the sclera. When this rare syndrome is recognized, the buckling material is removed, and prompt and intense periocular, and sometimes intraocular, antibiotic therapy is instituted.

**Choroidal detachment**

Some degree of choroidal detachment develops in approximately 5% to 10% of scleral buckling procedures. The fluid is assumed to be a transudation from the choroid. Its formation is related in part to advanced age and in part to surgical factors, such as thermal treatment, trauma to the choroid, hypotony, and obstruction of vortex vein outflow (particularly with very broad buckles), with resultant increased intravascular pressure throughout the choroidal vascular system. Although care is taken at surgery to minimize the procedural factors, choroidal detachment is not entirely avoidable.

In a majority of cases, the overlying retina remains in satisfactory apposition to the treated retinal pigment epithelium, and because of eventual spontaneous
resolution of the choroidal detachment, the prognosis is usually good. Choroidal detachments that cause angle closure and glaucoma are a more serious problem, and their management was discussed above. Massive detachments of the choroid may actually touch in the central vitreous (“kissing choroidals”). This problem requires surgical drainage to prevent the possibility of adhesion of retina to retina with subsequent tractional retinal redetachment.

Hemorrhagic choroidal detachments are less frequent than the common serous detachment, and the prognosis is poorer. The loculated hemorrhage is analogous to a hematoma elsewhere. The majority of cases follow a self-limiting course to spontaneous resolution, but some have a considerable chronic inflammatory response characterized by proteinaceous transudation, opacification of the vitreous, glaucoma, and occasional failure of the retina to reattach successfully. In some cases, the hemorrhage passes through the retina to produce a dense vitreous hemorrhage.

Later periocular infection and implant extrusion

The various implant materials and sutures used in retinal detachment surgery are foreign bodies and can become nidi of infection. Normally, the scleral buckling material becomes enveloped within a connective tissue sheath that develops soon after the initial buckling procedure. Therefore, a potential space exists for invasion of organisms between the buckle material and the tissue sheath over the entire extent of the buckle. The organisms responsible are frequently coagulase-positive staphylococci, but may be Gram-negative bacteria. The incidence varies, depending on the surgical technique and observance of the principles of asepsis.

The major clinical indication is pain, which should be considered a symptom of periocular infection until proved otherwise. Other signs are localized inflammation of the conjunctiva, subconjunctival hemorrhage, point tenderness, purulent discharge, and occasionally a draining of the sinus tract through the conjunctiva. Fortunately, associated intraocular involvement is exceptionally rare.

For infections diagnosed after a few weeks, treatment consists of removal of the scleral buckle, identification of the organism, and appropriate antibiotics. For extraocular infections occurring within the first few weeks of surgery, medical treatment may be administered to suppress the infection and gain sufficient time for the retina to firmly reattach before the scleral buckle is removed. Any evidence of intraocular extension of infection, however, demands immediate removal of the buckle. When the retina appears to be reattached and the chorioretinal adhesion matures, removal of the buckle only infrequently leads to redetachment. A second scleral buckling procedure, if required for detachment, may be undertaken as soon as all evidence of residual infection has disappeared.

Exposure of implant materials occurs as a consequence of disintegration of overlying tissues such as from infection, inadequate coverage of scleral implants at surgery, or migration of the implant. Segments of buckle material erode through overlying Tenon’s capsule and conjunctiva (Figure 7–16). Segmental episcleral buckles have a greater tendency to erode than narrower encircling bands. Although clinical signs of bacterial infection may or may not be present, virtually all such cases can be assumed to be infected. Providing the retina is well attached, there is no advantage in attempting to modify the scleral buckle or to patch over the
exposed portion. Most exposed buckles eventually have to be removed. Indications for removal include clinical infection, pain, or a cosmetic problem.

**Cystoid macular edema**

Cystoid macular edema (CME) is a common complication of ocular inflammatory diseases and surgery. Abnormal leakage from perifoveal capillaries causes intraretinal edema that accumulates in a characteristic cystoid pattern. Three to six weeks following successful scleral buckling, some degree of CME can develop in as many as one-third of patients. Pseudophakia is associated with an increased incidence of CME, which is also more likely to develop in older patients. There is no strong relationship between the incidence of CME and preoperative macular detachment, drainage of subretinal fluid, or the type of scleral buckle.

The effect of CME upon final visual acuity is uncertain, primarily because of a lack of studies correlating preoperative vision with a variety of additional variables associated with scleral buckling procedures. Nevertheless, CME contributes to a loss of visual acuity in eyes without preoperative macular detachment, and it probably limits recovery of vision in eyes in which the macula was detached. Eyes with objective signs of intraocular inflammation are treated with topical and periocular corticosteroids. Eyes without visible intraocular inflammation are given a trial of topical steroids and/or non-steroidal anti-inflammatory agents.

**Epimacular proliferation**

Epiretinal membranes that distort or cover the macula are a relatively common cause of disappointing visual acuity following successful scleral buckling surgery. The reported incidence of this problem varies considerably due to varying criteria employed for its diagnosis. Macular puckers have been reported in 2%–17% of successfully buckled cases.

Although the precise cause of macular pucker following reattachment surgery is unknown, it is likely that many epiretinal membranes develop from pigment
epithelial cells that pass through the retinal break(s) into the vitreous cavity. The cells then become attached to the surface of the retina in the posterior pole and subsequently proliferate and contract (Figure 7–17). The development of macular puckers may be associated with a variety of factors, including vitreous hemorrhage and intraocular inflammation associated with preoperative problems and/or operative techniques.

Removal of epiretinal membranes with vitrectomy techniques is the only effective means of treating macular puckers. Surgery is advisable in cases of significant epimacular fibrosis in which relatively poor postoperative visual acuity is associated with a history suggesting a potential for relatively good macular function.

**Proliferative vitreoretinopathy**

Proliferative vitreoretinopathy (PVR) is the only common cause of ultimate failure following retinal reattachment surgery. Unless this occurs, the vast majority of initial failures can be successfully repaired. This cell-mediated process is associated with the production of fibrocellular membranes on the posterior vitreous surface and on both surfaces of the retina (see Chapter 5, page 109). Subsequent contraction of the membranes causes significant shortening of the retina, which prevents reattachment or causes recurrence of detachment, even if all retinal breaks are closed.

Factors which cause a significant breakdown in the blood–aqueous barrier and which allow an increased number of pigment epithelial cells to enter the vitreous cavity are also associated with an increased incidence of PVR. Although cryotherapy has been experimentally implicated as a possible cause of PVR, a positive relationship in appropriately managed cases has not been demonstrated. Still, preoperative intraocular inflammation is clearly related to the incidence of postoperative PVR, and relatively atraumatic surgical techniques are employed in an attempt to reduce the likelihood of postoperative PVR.

**Figure 7–17.** A “macular pucker” following retinal reattachment surgery is due to cellular proliferation and subsequent membrane formation and contraction upon the central retina.
Although scleral buckling procedures alone are successful in the management of selected cases with limited PVR, vitrectomy techniques are routinely employed to reattach retinas associated with more extensive intravitreal and periretinal membranes, and these are discussed in Chapter 9. In combination with vitrectomy, an encircling scleral buckle is also an important component of surgery for the repair of retinal detachments associated with PVR. At the time of vitreous surgery, previously placed segmental buckles are replaced or augmented with encircling procedures, and selected previously placed encircling buckles may be modified in an effort to counteract significant peripheral traction forces.

**Recurrent retinal detachment**

Recurrent or persistent retinal detachment is the most significant complication of scleral buckling, and the severity of this problem is related to its cause. Retinal detachment following scleral buckling surgery occurs in 9%–25% of primary operations, and the vast majority is associated with open retinal breaks.

If postoperative retinal detachment is due to a new tear or an inadequate buckling effect unassociated with proliferative vitreoretinopathy (PVR), modification of the scleral buckle and creation of a chorioretinal adhesion will usually reattach the retina. In selected cases, intraocular injection of a gas bubble, supplemental cryopexy or laser, and appropriate positioning may be sufficient to achieve reattachment and avoid revision of the buckle. If extensive PVR is responsible for the surgical failure, vitrectomy techniques are usually required for a successful reoperation.

**Altered refractive error**

Scleral buckling techniques with an encircling component usually cause a myopic change in the refractive error because of their effect upon axial length. An average increase in axial length of approximately 1 mm induces an average myopic shift of approximately −2.75 diopters.

The effect of radial scleral buckles is usually less significant. Significant astigmatic changes are very unusual unless the buckles are quite anterior, but some degree of astigmatism is occasionally caused by indenting the sclera near the ora serrata.

Although changes in refractive error may be relatively unimportant in eyes with poor postoperative visual acuity, the induction of significant anisometropia can be devastating to some patients, particularly those with excellent postoperative visual acuity and an emmetropic fellow eye. Avoiding anisometropia is an important goal of some alternatives to scleral buckling.

**Strabismus**

Some degree of extraocular muscle imbalance can occur in up to 50% of patients undergoing scleral buckling procedures. Most of these abnormalities are temporary and due to intraoperative muscle damage. Nevertheless, some degree of permanent muscle imbalance is observed in approximately one-fourth of patients after primary buckling operations.

Causes of strabismus include the following: (1) abnormal adhesions between the muscle and the sclera or Tenon’s capsule, (2) injury to the muscle from surgical
trauma, (3) mechanical disturbances due to the location and shape of buckling materials, and (4) problems associated with disinsertion or repositioning of a muscle. Factors associated with postoperative muscle imbalance include placement of a buckle beneath a muscle, size of buckling material beneath a muscle, and reoperations.

Therapy for patients with good bilateral vision usually includes an attempt to prescribe prisms to restore fusion. If this is unsuccessful, surgery is considered. Avoiding postoperative muscle imbalance is a major benefit attributed to alternative reattachment procedures.

**SUMMARY**

Scleral buckling repairs retinal detachments by indenting the sclera under the retinal breaks. Retinal breaks are localized, cryopexy or other thermal treatment is applied to establish a permanent seal around the breaks, and silicone is usually sewn onto the scleral surface. Subretinal fluid may be drained, and gas or fluid may be injected into the eye. Scleral buckling can also be combined with vitrectomy.

Failure to permanently repair the detachment, often with the development of proliferative vitreoretinopathy, is a possible outcome. Potential complications include endophthalmitis, choroidal detachment, increased intraocular pressure, and diplopia.

**SELECTED REFERENCES**


Pneumatic retinopexy (PR) is an office-based, sutureless, no-incision alternative to scleral buckling or vitrectomy for the surgical repair of selected retinal detachments. Cryotherapy is applied around the retinal break(s) to form a permanent seal. A gas bubble is injected into the vitreous cavity, and the patient is positioned so that the bubble closes the retinal break(s), allowing resorption of the subretinal fluid (Figure 8–1A–F). As an alternative to cryotherapy, laser photocoagulation can be applied after the intraocular gas has caused the retina to reattach.

**INTRAOCULAR GASES**

**Choice of Gases**

Sulfur hexafluoride (SF₆) is the gas most frequently used with pneumatic retinopexy. Perfluorocarbon gases such as perfluoropropane (C₃F₈) are sometimes used, and success has also been reported with sterile room air.

In selecting a gas, it is important to understand the longevity and expansion characteristics of the gases. SF₆ doubles in volume within the eye, reaching its maximum size at about 36 hours. It will generally disappear within about 10–14 days, depending on the amount injected. Perfluoropropane nearly quadruples in volume, reaching maximum size in about three days. The bubble will last 30–45 days in the eye. Room air does not expand, but immediately starts to reabsorb. The air bubble will be gone within just a few days (Table 8–1).
Figure 8–1. Pneumatic retinopexy procedure. (A) Volume of subretinal fluid is determined by inflow of fluid vitreous (green arrow) and outflow through retinal pigment epithelial pump into the choroid (red arrow). (B) Area of retinal break is treated with contiguous applications of transconjunctival cryotherapy. (C) With pars plana injection site uppermost, gas bubble is injected into vitreous with 0.5-inch, 30-gauge needle. (D) Head is positioned to place retinal break uppermost, thereby sealing break with intravitreal gas bubble. (E) With break closed, retina is usually reattached by first postoperative day. (F) Gas bubble is spontaneously absorbed. (Published with permission from Hilton GF, Grizzard WS: Pneumatic retinopexy: a two-step out-patient operation without conjunctival incision. Ophthalmology 1986;93:626–641.)
The initial expansion of SF\(_6\) and C\(_3\)F\(_8\) is due to the law of partial pressures and the solubility coefficients of the gases involved. A 100\% SF\(_6\) bubble injected into the eye contains no nitrogen or oxygen, but these gases are dissolved in the fluid around the bubble. Due to the law of partial pressures, nitrogen and oxygen will diffuse into the gas bubble. SF\(_6\) also starts to diffuse out of the gas bubble into the surrounding fluid which contains no SF\(_6\). However, nitrogen and oxygen diffuse across the gas–fluid interface much more quickly than SF\(_6\) because of the relative insolubility of SF\(_6\). The net result is an initial influx of gas molecules into the bubble, expanding its size until partial pressures equilibrate, net influx equals net egress, and maximum expansion is reached. Then the bubble gradually reabsorbs as the SF\(_6\) is slowly dissolved in the surrounding fluid. The diameter of the bubble shrinks at an approximately constant rate until the gas is gone. C\(_3\)F\(_8\) expands more and reabsorbs more slowly because it is even less soluble than SF\(_6\).

The choice of type and amount of gas depends on the following considerations.

**What size gas bubble is needed?**

One must usually plan for a gas bubble more than large enough to cover all detached breaks simultaneously, and keep them covered for three to five days.

Computerized tomography studies on eyes with intravitreal gas bubbles showed that a 0.3 ml gas bubble covers over 45 degrees of arc of the retina (Figure 8–2), but it takes approximately a 1.2 ml bubble to cover 80–90 degrees. A highly myopic eye will require a larger volume of gas than an emmetropic eye to cover the same arc of the retina.

Usually, 0.4 to 0.6 ml of gas is injected into the eye. For room air PR, a larger bubble is generally needed, perhaps 0.8 ml, depending on the characteristics of the case. If it is desired to inject more than 0.6 ml, multiple paracenteses will likely be needed, one before the gas injection, and usually one or more after the gas injection. Alternatively, multiple gas injections may be performed, allowing the return of intraocular pressure toward normal between injections.

**How long should the bubble remain in the eye?**

It is optimal for the gas bubble to cover the break(s) for five days and then disappear as soon as possible. However, good results have been reported with only 3–4 days of tamponade, as with room air. The longevity of air is probably sufficient for most cases, but sometimes the chorioretinal adhesion may not be sufficiently mature when the air has been reabsorbed. Air also forfeits the advantage of post-injection expansion within the eye, necessitating an injection of a large volume.
In most cases, the prolonged longevity of a perfluoropropane bubble is a disadvantage. A lingering gas bubble may induce tears, since movement of the head causes forcible movements of the vitreous when a gas bubble is in the eye. Also, air travel is contraindicated for a longer period of time with C₃F₈. However, it may also eliminate the need to reinject gas if a new break develops, and C₃F₈ allows the injection of a smaller amount of gas initially, thereby reducing the need for paracentesis.

Our gas of choice in most cases is SF₆. We use C₃F₈ for the occasional case which requires an exceptionally large and long-acting gas bubble to tamponade large or widespread breaks. Most of the time we inject 0.4 to 0.6 ml of 100% SF₆.

**Why Gas Works**

The following characteristics of intraocular gases account for their efficacy in reattaching the retina:

1. Surface tension allows the gas bubble to occlude a retinal break instead of passing into the subretinal space. The surface tension of any gas is much higher than that of other substances in the eye. Once the break is occluded,
the retinal pigment epithelial pump can reabsorb the subretinal fluid (Figure 8–1D).

2. Buoyancy of the gas provides the force which pushes the uppermost retina back against the wall of the eye. Apposition of the retina against the retinal pigment epithelium is necessary in order that an adhesion can occur, just as two surfaces to which glue has been applied must be clamped together while the glue dries (Figure 8–1E). When the gas is gone, a permanent seal remains, preventing reopening of the tear (Figure 8–1F).

PREOPERATIVE EVALUATION

Good preoperative evaluation is vital to the success of pneumatic retinopexy. PR is not a good procedure for surgeons lacking in excellent retinal examination skills, for three reasons:

1. Scleral buckling with encirclement may achieve retinal reattachment even if a retinal break is missed. It can be a very “forgiving” procedure in this sense. With pneumatic retinopexy, any missed retinal break might open into a detachment. The presence of a gas bubble within the vitreous cavity causes shifting of the subretinal fluid or vitreous which can open previously attached breaks.

2. When scleral buckling or vitrectomy are performed, the surgeon gets a second look in the operating room with the patient sedated, with full control of the globe, and with open conjunctiva for deep scleral depression. With pneumatic retinopexy, this is not available.

3. Scleral buckling, especially encirclement, supports the peripheral retina, reducing the traction which the vitreous will be able to exert on the retina in the future. Lacking this with pneumatic retinopexy, careful follow-up examinations are required to find any retinal breaks which develop postoperatively.

In addition to a thorough examination of the retina, the preoperative evaluation for possible pneumatic retinopexy should include assessment of the following:

1. Is the patient mentally capable of following positioning directions?
2. Is the patient physically capable of maintaining positioning as needed, especially with regard to neck and back problems?
3. Will it be feasible from the standpoint of the patient’s home situation for the patient to maintain the appropriate position postoperatively?
4. Will the patient be able to return to the surgeon’s office for frequent follow-up as required?
5. Does the patient have plans to travel in the near future, especially by air, which might pose a hazard with an intraocular gas bubble?
6. Does the patient have evidence of severe glaucoma?
7. Has the patient had recent surgery on this eye which would require care to avoid dehiscence of the healing incision?
8. Does the presence of a filtering bleb or a corneal transplant require special handling?
9. Would the induction of increased myopia that generally occurs following an encircling buckle be an advantage or disadvantage given the patient’s refractive status?

Because postoperative patient cooperation is essential, the nature of the procedure and what will be expected of him should be discussed thoroughly with the patient before surgery.

**INDICATIONS AND CONTRAINDICATIONS**

In the multicenter clinical trial which compared pneumatic retinopexy with scleral buckling, cases with the following characteristics were excluded:

1. Breaks larger than one clock-hour or multiple breaks extending over more than one clock-hour of the retina.
2. Breaks in the inferior four clock-hours of the retina.
3. Presence of proliferative vitreoretinopathy grade C or D.
5. Physical disability or mental incompetence precluding maintenance of the required positioning.
6. Severe or uncontrolled glaucoma.

Subsequent experience has demonstrated that selected cases that do not strictly meet these criteria can be successfully treated with pneumatic retinopexy, subject to certain limits.

**LIMITS TO INDICATIONS**

**Extent of breaks**

Clearly, breaks spanning more than one clock-hour can be treated with pneumatic retinopexy. Single or multiple tears or dialyses spanning three clock-hours pose no particular problem. Detached tears six clock-hours apart are difficult to fix with pneumatic retinopexy, although alternating positioning has been used successfully. Even detachments with giant retinal tears have been cured with pneumatic retinopexy, but it is rarely the procedure of choice. The size of the gas bubble should generally reflect the size of the problem, although it is not always necessary to cover all breaks simultaneously with a single bubble.

When deciding whether a case is amenable to pneumatic retinopexy, recognize that there is a difference between attached and detached breaks. In cases where an attached break is present on the opposite side of the eye from the detached breaks, alternate positioning would not be needed. The attached break should probably be treated with laser prior to the gas injection. Care should then be taken, such as by using the steamroller technique (described below) if necessary, to prevent the bubble from pushing the subretinal fluid into the attached break and causing it to
detach. By following these two steps, multiple flat breaks may be ignored in positioning and in determining bubble size.

**Inferior breaks**
Most cases with breaks in the inferior four clock-hours of the eye have been difficult to treat with pneumatic retinopexy. Even for limber patients, it is very difficult to tilt the head below the horizontal for very long. As a rule, a detached break in the inferior four clock-hours represents a contraindication to pneumatic retinopexy.

**Proliferative vitreoretinopathy**
Since pneumatic retinopexy does not relieve traction like scleral buckling or vitrectomy can, significant preoperative traction on a retinal tear is a relative contraindication to the pneumatic procedure. When a tear is adjacent to a star fold, pneumatic retinopexy is usually not the procedure of choice. Mild to moderate proliferative vitreoretinopathy which is distant from any retinal breaks does not necessarily contraindicate pneumatic retinopexy. More severe PVR usually calls for vitrectomy and scleral buckling.

**Cloudy media**
It is important to the success of pneumatic retinopexy that all retinal breaks be identified and treated. Opacities such as peripheral vitreous hemorrhage represent relative contraindications to pneumatic retinopexy. Since pneumatic retinopexy does not seem to jeopardize an eye for future scleral buckling if needed, it may not be unreasonable to use the pneumatic procedure even when opacities obscure part of the attached retina, but this represents a calculated risk.

**Inability to maintain positioning**
Failure to faithfully maintain the appropriate position is probably an important cause of failure of pneumatic retinopexy. It is important to inquire regarding back or neck problems and to assess mental competence before deciding on pneumatic retinopexy. Recognize that some positions are quite easy to maintain, while others require excellent cooperation. Positioning is easiest with tears between 11:00 and 1:00.

**Glaucoma**
Glaucoma has proven to be relatively unimportant as a contraindication to pneumatic retinopexy. The large majority of glaucoma patients can be treated with pneumatic retinopexy without problem. Patients with quite severe glaucoma, such as with splitting of the macular field due to glaucoma, might suffer noticeable damage (even from brief elevations in intraocular pressure), but pneumatic retinopexy can be performed in a manner to avoid elevations in pressure. Except in cases of severely impaired trabecular outflow facility, serial measurements of intraocular pressure following gas injection are not necessary.
Lattice degeneration
In several series, patients with extensive lattice degeneration tended to do rather poorly with pneumatic retinopexy. It does not appear that mild to moderate lattice should be considered a contraindication.

Aphakia/pseudophakia
In some series, aphakic/pseudophakic eyes did poorly with pneumatic retinopexy, but in other reports this was not the case. These eyes, prone to multiple tiny far-peripheral holes, require an especially careful preoperative examination. With peripheral capsular opacities frequently present, the view of the peripheral retina can be quite limited. Such cases should probably not be treated with pneumatic retinopexy. If the peripheral retina can be adequately examined, aphakia and pseudophakia are not contraindications to pneumatic retinopexy. Experience has shown that eyes with an open posterior capsule tend to develop breaks more frequently than eyes with the capsule intact. Like severe lattice degeneration, aphakia/pseudophakia with an open posterior capsule warrants extra caution.

Relative Indications for Pneumatic Retinopexy
Pneumatic retinopexy has particular advantages in the management of several types of cases:

Retinal detachment imminently threatening the fovea
Pneumatic retinopexy allows immediate treatment in urgent situations, avoiding delays inherent in taking a patient to the operating room. Furthermore, by using the steamroller technique described below, PR can be used to sweep subretinal fluid away from the fovea, providing additional protection against foveal detachment.

Macular holes and other posterior retinal breaks
Retinal detachments secondary to posterior retinal breaks are difficult to treat with scleral buckling. For many surgeons, pneumatic retinopexy is the procedure of choice in many of these cases.

Redetachment following scleral buckling
Following scleral buckling, when subretinal fluid accumulates due to a break in the superior eight clock-hours, pneumatic retinopexy is frequently much easier than revising the buckle. Because external cryopexy has difficulty treating through the insulating effect of a scleral buckle, laser treatment is applied after the gas bubble settles the detachment.

Filtering blebs
If a functioning filtering bleb is present, or if an eye may need a filtering procedure in the future, pneumatic retinopexy should be considered to minimize conjunctival scarring and inflammation.
**Isolated tears under the superior rectus**

Placing a segmental buckle under a vertically acting muscle runs the risk of iatrogenic diplopia; this potential complication is eliminated with pneumatic retinopexy.

**Contraindications to general anesthesia**

If performing scleral buckling or vitrectomy under local anesthetic is not an option, medical contraindications to general anesthesia may indicate pneumatic retinopexy as an optimal alternative.

**Optic pit with macular detachment**

Pneumatic retinopexy may be an effective option in the treatment of optic pits with serous macular detachment.

**Extensively scarred conjunctiva**

Conjunctival scarring may make dissection for a scleral buckle difficult, and may make it difficult to get the conjunctiva to cover adequately at the end of the case. These problems are avoided with pneumatic retinopexy.

**Very thin sclera**

With scleral buckling, thin sclera poses a risk of needle penetration or inadequate anchoring of sutures to secure the buckle, problems that are avoided with pneumatic retinopexy or vitrectomy. When ultra-thin sclera is discovered in the operating room, a pneumatic procedure combined with drainage of subretinal fluid can provide a means of repairing relatively extensive detachments, since a very large gas bubble can be obtained.

**Need to retain emmetropia or to prevent anisometropia**

Patients with excellent preoperative vision, particularly if they are uncorrected or have a history of refractive surgery, may consider the loss of emmetropia to be a substantial disadvantage of scleral buckling.

**Cosmetic concerns**

Scleral buckling is associated with a higher risk of ptosis, enophthalmos, and strabismus than that seen after pneumatic retinopexy.

**Operating room not available**

In some settings or at some times, an adequately equipped and staffed operating room may not be available or may require an unacceptable delay. In these instances, an office-based procedure may be a good alternative.

**Limited financial resources**

Pneumatic retinopexy is much less expensive than scleral buckling or vitrectomy because all operating room expenses, ancillary hospital or surgicenter charges, and anesthesia fees are avoided. Preoperative medical and laboratory evaluation is also generally not necessary.
OPERATIVE TECHNIQUE

The technique detailed here is essentially the same as that originally described by Hilton and Grizzard in 1986, with a few modifications. The operation is usually performed in an outpatient department or in the surgeon’s office.

ANESTHESIA

Pneumatic retinopexy can be accomplished with topical, subconjunctival, or retrobulbar anesthesia, depending on the surgeon’s and patient’s preferences. Sensitive patients may do better with a retrobulbar injection.

An injection into the anterior muscle cone will usually produce anesthesia without akinesia initially. This has the advantage of allowing the patient to position his eye to cooperate with cryopexy. After the retrobulbar anesthetic is given, cryopexy is administered promptly before the eye becomes akinetic.

Rarely, general anesthesia (avoiding the use of nitrous oxide) may be indicated if the patient is very young and/or apprehensive.

RETINOPEXY

Pneumatic retinopexy is generally performed in one session with cryopexy applied to the retinal breaks prior to gas injection (Figure 8–1B). An alternative technique involves a two-part procedure, utilizing laser instead of cryotherapy. The first part of the procedure consists of injection of a gas bubble into the vitreous cavity. The patient maintains appropriate head positioning at home, with follow-up in the surgeon’s office. Once the break is reattached, usually the next day, laser treatment is applied.

The photocoagulation is greatly facilitated by use of the laser indirect ophthalmoscope (LIO), which makes it easy to position the patient’s head to move the gas bubble away from the break(s). Treatment can also be applied with a slit lamp laser delivery system by tilting the patient’s head as needed. It is generally best not to attempt to laser until the retina is completely reattached in the treatment area.

Although one can treat the breaks through a moderately large gas bubble, one must be careful not to overtreat. Gas has an insulating effect, conducting heat away from the laser spot at a slower rate than vitreous, which may result in excessive thermal burns with retinal necrosis and hole formation.

Cryopexy versus laser

A one-part procedure with cryopexy is usually preferred over a two-part procedure with laser. Retinal breaks are easier to find when they are detached, and a one-part procedure is usually more convenient.

Certain circumstances might indicate the use of laser instead of cryotherapy. Very posterior breaks are easier to treat with laser than with cryo. The chorioretinal adhesion with laser may be obtained more quickly and firmly than that with cryo. If multiple large breaks are present, laser may be better than extensive cryopexy. When there has been a recent surgical incision in the eye, laser may be safer than cryo because it may be easier to avoid applying pressure to the eye.
Transcleral photocoagulation may provide some advantages over both cryopexy and transpupillary laser. Like transpupillary laser photocoagulation, one can see exactly where the treatment will be applied and where it has been applied. As with cryopexy, it allows treatment in detached retina. Treatment can even be applied through a preexisting buckle.

**Sterilizing the Eye**

A sterile lid speculum is utilized. About six drops of undiluted povidone-iodine solution are instilled directly onto the cornea and conjunctiva, and left in contact with the eye for a few minutes. During this time the gas can be prepared. The injection site is then dried with a sterile cotton-tipped applicator, and the eye is ready for paracentesis and injection of gas.

Be certain to use a povidone-iodine solution which does not contain alcohol or detergent. Preoperative topical antibiotics add nothing to the sterility of a careful sterile prep. Meticulous sterility is mandatory. No cases of endophthalmitis have been reported following pneumatic retinopexy when povidone-iodine solution was used as described above.

**Preparing the Gas**

A pressure-reducing system is attached to the gas cylinder to allow drawing of the gas from a low pressure reservoir. High pressure can blow out the millipore filter and render it useless in sterilizing the gas. A urinary external catheter can be attached to the cylinder, or a step-down valve system can be used. Alternatively, the gas may be drawn into a large syringe and then transferred to a small syringe (Figure 8–3).

The selected gas is drawn through a millipore filter into a 3 ml syringe in a sterile fashion. The tube connecting the gas cylinder with the syringe, including the filter, is flushed through with gas to ensure no dilution with room air. A few milliliters of gas are drawn into the syringe, discarded, and the syringe is filled again. A disposable 30-gauge, one-half inch (12 mm) needle is then placed tightly on the syringe, and excess gas is expelled to leave the exact amount intended for injection. The gas should not be stored in the syringe for more than a few minutes prior to injection because room air will infiltrate the syringe and dilute the gas sample.

**Making Room for the Gas**

The intraocular volume must be decreased before and/or after the gas injection to make room for the gas. We recommend that a paracentesis be performed before the gas injection, especially if the globe has been weakened by surgery or trauma within the past six weeks, or if there is significant glaucomatous optic nerve damage. Laser may be preferred over cryopexy in these cases, since cryopexy may also cause dehiscence of the unhealed wound or raise pressure excessively.

Paracentesis before or after the injection is usually necessary, but prolonged ocular massage, including compression from cryopexy, may be sufficient in some cases.
Paracentesis

The intended paracentesis site should be sterilized with Betadine solution as described above. A 30-gauge, 1/2-inch needle on a 1 ml syringe with the plunger removed is passed obliquely into the anterior chamber through the limbus, staying over the iris with the bevel up. Fluid will flow passively into the syringe. As the flow of fluid slows, gentle pressure on the sclera with a cotton-tipped applicator will facilitate fluid egress. Remove as much fluid as can be obtained, up to about 0.45 ml. Fluid flow can be quite slow at the end, and it may take a few minutes with the needle in the eye to remove sufficient fluid.

If the posterior lens capsule is absent or open widely, paracentesis should not be performed through the limbus to avoid incarceration of vitreous in the limbal needle tract. With plunger in place, pass the needle through the pars plana, then angle it through the posterior capsular opening into the anterior chamber.

Ocular massage

If after paracentesis and gas injection the pressure is too high, the eye may be massaged to reduce intraocular volume. Retropulsion of the eye into the orbit dehydrates the orbital fat, but is less effective at reducing the intraocular volume.
Instead, a scleral depressor is placed against the temporal equator, and the eye is pressed firmly against the bony nasal orbital wall. Firm pressure is applied for 45 seconds, then relaxed for 15 seconds to allow perfusion of the retinal vasculature. This cycle is repeated until the intraocular pressure is low enough. This maneuver causes egress of fluid from the eye, and also stretches the scleral fibers, allowing more ample intraocular volume. Preoperative medications for reducing the intraocular pressure do not help much.

**Injecting the Gas**

An injection site is selected 3–4 mm posterior to the limbus. This site should be away from large, open retinal breaks, highly detached retina, or detached pars plana epithelium. The head of the supine patient is turned 45 degrees to one side to make the injection site uppermost. The needle is then passed into the eye perpendicular to the sclera (Figure 8–4). The needle is pushed 6–8 mm into the eye to ensure that the tip is well into the vitreous, directing the tip away from areas of highly bullous detachment. The needle is then withdrawn until 3 mm of it remains in the eye (Figure 8–5). This will ensure that the tip remains in the vitreous but is shallow enough to prevent multiple small bubbles (“fish

![Figure 8–4. Injecting gas into eye with injection site uppermost.](image-url)
Figure 8–5. Procedure of injecting gas into eye. (A) With injection site uppermost, needle is pushed 6–8 mm into eye to ensure tip is deep in vitreous. (B) Needle is withdrawn until 3 mm of needle remains in eye. Gas is then injected semi-briskly, creating a single bubble.
AFTER GAS INJECTION

Following injection, the eye is examined with the indirect ophthalmoscope to make the following three determinations:

Is the central retinal artery occluded?

Examine the central retinal artery to ensure its patency. Paracentesis or massage may be performed to reestablish patency or strong pulsation of the central retinal artery. Occlusion of the central retinal artery can be safely observed for up to ten minutes. During this time, the intraocular pressure declines and the artery may reopen; if it does not, paracentesis or massage should be performed immediately.

As long as the central artery is open (widely patent or with strong pulsation), measurement of the intraocular pressure has little meaning. The pressure will soon return to normal and not increase, even though the gas is expanding.

Is a single gas bubble present or are there multiple small bubbles (“fish eggs”)?

Fish eggs are undesirable because a small gas bubble can pass through a retinal break into the subretinal space (Figure 8–6). See the section on “Injecting the Gas” above for techniques for avoiding the development of fish eggs, and see the section on “Special Procedures” below for suggestions on management of fish eggs.

Is the bubble mobile within the vitreous or is it trapped at the injection site?

If the bubble is beneath the pars plana epithelium, or trapped in the space bordered by the pars plana, the anterior hyaloid face, and the lens (the canal of Petit), it will not move when the head is turned and will take on a semicircular shape. This has been termed the “donut sign,” the “sausage sign,” or the “bagel sign.” Management of this occurrence is discussed below, under “Special Procedures.”
The “steamroller” technique is now used if indicated (see “Special Procedures” below). We instill antibiotic ointment and patch the eye. The meridian of the retinal break is marked as an arrow on the patch to indicate to the patient and family the head position which is to be maintained. We have a mirror available to show the patient that the head should be positioned so that the arrow is pointing directly at the ceiling. The patient is discharged to home and seen the next day.

**SPECIAL PROCEDURES**

**STEAMROLLER**

If bullous subretinal fluid extends almost to the macula (Figure 8–7A), placement of a bubble against the bullous detachment may cause a macular detachment (Figure 8–7B). This complication can be easily avoided by using the “steamroller” technique.

Following injection of the gas bubble, the patient’s head is turned to a face-down position in such a way as to cause the bubble to traverse the attached retina en route to the macula (Figure 8–7C). Over one to five minutes, the patient’s head position is very gradually changed until the retinal break is uppermost, causing the bubble to roll toward the retinal break, pushing the subretinal fluid back into the vitreous and flattening the retina (Figure 8–7D).

Subretinal fluid will be expressed through the retinal break into the vitreous cavity at a rate depending in part on the size of the break. Since cryopexy causes liberation of pigment epithelial cells, which may cause proliferative vitreoretinopathy if they get in the vitreous cavity, it is recommended that cryopexy not be performed prior to steamrolling.

Whether steamrolling is necessary to prevent macular detachment depends on several factors:

1. How close the detachment is to the macula. Only detachments well within the arcades usually need steamrolling.
2. How bullous the detachment is.
3. How large the gas bubble is.

Possible indications for steamrolling are as follows:

1. Prevention of iatrogenic macular detachment.
2. Prevention of iatrogenic detachment of an attached retinal break.
3. Reduction of subretinal fluid to encourage more rapid resolution of retinal detachment. This might be of use in cases where all retinal breaks cannot be covered at one time by the gas bubble. Also, where large retinal breaks are present, this may minimize the chance of subretinal gas.
4. Reduction of a bullous detachment overhanging the optic nerve, preventing visualization of the central retinal artery during the procedure.
Multiple small gas bubbles ("fish eggs"—Figure 8–8) are usually due to a faulty injection technique. In probable order of importance, the following steps will usually prevent this occurrence:

1. Make sure that the needle is shallowly within the vitreous at the time of injection.
2. Make sure that the injection site is uppermost.
3. Inject with the needle vertical.
4. Inject briskly but not extremely rapidly.

If fish eggs do occur, keep the patient strictly positioned to keep the bubbles away from retinal breaks. If all retinal breaks are small, this may not be necessary, but keep in mind that breaks can stretch a little. The bubbles will usually coalesce spontaneously within 24 hours, and then the patient can adopt a position with the retinal break uppermost.

Some authors recommend inducing fish eggs to coalesce by flicking the eye with a cotton-tipped applicator or gloved finger. Turn the eye so that sclera without underlying retinal breaks is uppermost, and flick this site moderately firmly.

If there is one large bubble with just a few smaller bubbles, usually the above measures are not necessary, but caution is called for in the presence of large tears.

**GAS ENTRAPMENT AT THE INJECTION SITE**

Following gas injection, if the gas bubble remains trapped at the injection site, it is probably trapped in the canal of Petit. If the trapped bubble is small, no treatment is necessary.

Unless there is an immediate threat of the macula detaching, the problem can usually be solved by face-down positioning for 24 hours. This will encourage the trapped anterior gas bubble to break through the anterior hyaloid face by its own buoyancy, aided by its expansion.

If necessary, a large trapped bubble can be removed by passing a 27- or 25-gauge needle back through the injection site. This needle is mounted on a syringe with a
small amount of sterile saline, with the plunger removed. The injection site is positioned uppermost and the needle is passed vertically into the bubble. Sometimes it takes a little manipulating to break the surface tension of the bubble and get it to escape. Most of the gas will escape, bubbling up through the fluid in the syringe. At another site, reinject the gas deeper into the vitreous, with 4–5 mm of the needle in the globe.

**SUMMARY OF PROCEDURE**

The following constitutes a typical order of events:

1. Anesthetic: retrobulbar, subconjunctival, or topical
2. Cryopexy: if one-part procedure, in lieu of laser
3. Sterilization of ocular surface: Betadine solution
4. Paracentesis: limbal, or via pars plana if capsule is open
5. Intravitreal gas injection: 0.4–0.6 ml of SF₆
6. Check for patency of central retinal artery, and perform paracentesis and/or massage if needed
7. Special procedures: for example, steamroller if needed
8. Antibiotic and patch: draw arrow
9. Laser: when retina is reattached, in lieu of cryopexy as two-part procedure

**POSTOPERATIVE MANAGEMENT**

Acetaminophen (Tylenol) may be helpful for postoperative pain control. We recommend a considerable restriction in activity initially, liberalizing day by day as the retina reattaches, the chorioretinal scar matures, and finally the gas bubble reabsorbs. The patient is allowed to return to work two weeks after the procedure, and should be advised not to fly until the bubble is quite small.

If all retinal breaks are closed, the subretinal fluid usually reabsorbs within 24–48 hours. If the fluid is not reabsorbing, a new or missed break exists, the bubble is too small, or the patient has not been positioning properly.

Ensuring proper patient positioning requires considerable effort. It is helpful to explain to the patient why positioning is important, and to demonstrate the position which allows the bubble to close the breaks. The neck strain of an oblique head position can be eased by explaining that sitting with the head tilted 45 degrees to the left is the same as lying on a couch with the head tilted 45 degrees to the right.

Patient positioning is maintained during waking hours for five days; however, three or four days may be adequate. The patient should not sleep face-up, to avoid gas–lens contact in the phakic eye, or ciliary-block glaucoma in the aphakic eye.

Depending on the response to treatment, the patient may be seen on the first postoperative day, then in three days, one week, two weeks, one month, and so
Frequent postoperative exams are indicated, primarily to look for new retinal breaks or detachments. These breaks do not jeopardize the outcome if close follow-up results in early detection and treatment. At least half of these can be cured with an additional office procedure without resorting to scleral buckling.

Inferior subretinal fluid or loculated pockets of subretinal fluid sometimes persist for weeks or months. As long as the fluid is not increasing and the macula is attached, reoperation is not necessary.

**COMPLICATIONS**

**Subretinal Gas**

Subretinal gas in the absence of fish eggs at the time of injection is rare. If fish eggs are noted following injection, one should examine carefully for the presence of subretinal gas. Once the gas bubble expands, it may be more difficult to get it back out of the break it passed through. If a gas bubble does get beneath the retina, it gives the detached retina a pearly, dome-shaped, reflective sheen (Figure 8–9A). Attempt first to massage the bubble back toward the retinal break by scleral depression, assisted by positioning as needed. If this fails and the amount of subretinal gas is large, prompt surgical removal with vitrectomy may be required.

Smaller subretinal bubbles can be managed conservatively (Figure 8–9B). In spite of a small subretinal bubble, the larger intravitreal gas bubble can usually seclude the break from liquid vitreous with strict positioning, and subretinal fluid will reabsorb. The smaller subretinal bubble will reabsorb before the larger vitreous bubble and the detachment can be repaired, injecting additional gas if needed.

**Iatrogenic Macular Detachment**

This preventable complication is avoided by using the steamroller technique as described above.

**New Retinal Breaks**

New or missed retinal breaks following pneumatic retinopexy appear to occur in approximately 13% of eyes. This is similar to the incidence of new retinal breaks following cryopexy or laser, without the injection of intravitreal gas.

The high incidence of new retinal breaks mandates close postoperative follow-up. Most new breaks occur within one month and within three clock-hours of the first break.

In the multicenter trial of pneumatic retinopexy, 96% of eyes with new breaks were successfully reattached. Approximately half of such breaks were managed in the office or outpatient department without scleral buckling. New breaks and detachments can often be treated easily with laser or with pneumatic techniques, without automatically resorting to scleral buckling and/or vitrectomy.
Pneumatic retinopexy does not appear to increase the incidence of proliferative vitreoretinopathy (PVR). In the multicenter clinical trial, PVR occurred in 5% of eyes following scleral buckling, and 3% of eyes following pneumatic retinopexy.

**COMPARISON WITH SCLERAL BUCKLING**

In a review of the worldwide literature on pneumatic retinopexy, statistics on 1,274 cases were compiled. The single-operation success rate was 80%, and 98% were reattached with reoperations, which compares favorably with scleral buckling.

A multicenter, randomized, controlled clinical trial with 198 patients compared pneumatic retinopexy with scleral buckling. The key findings of this study (two-year follow-up) are shown in Table 8–2.
Comparison of the two procedures in this study is summarized as follows:

1. The main disadvantages of pneumatic retinopexy are the reduced single-operation success rate with more frequent need for retreatments, and the need for precise postoperative positioning and close follow-up care.
2. Failure with pneumatic retinopexy does not jeopardize success with subsequent scleral buckling, and final anatomic results were not significantly different.
3. Morbidity was less, and cost was much less with pneumatic retinopexy.
4. Postoperative visual acuity appeared to be better with pneumatic retinopexy than with scleral buckling for eyes in which the macula was detached for less than 14 days ($p = 0.05$).
5. Complications were similar, based on a score system which counted the need for postoperative laser or cryo as a complication.
6. Cataract surgery was required more often with scleral buckling than with pneumatic retinopexy.
7. Scleral buckling is the more versatile procedure, with many detachments not amenable to pneumatic retinopexy. However, pneumatic retinopexy can treat occasional detachments, which scleral buckling cannot, such as detachments with very posterior breaks. For some surgeons, at least 40% of detachments are good candidates for pneumatic retinopexy.

Pneumatic retinopexy is more versatile than the temporary episcleral balloon, since with the former, one is able to treat larger breaks, more widely spread breaks, and more posterior breaks than the balloon can treat. However, the balloon can treat detached inferior breaks, which the bubble cannot. At this time, the temporary episcleral balloon is no longer manufactured.

Pneumatic retinopexy is less expensive than scleral buckling because there is no need for (1) a preoperative history and physical, (2) preoperative laboratory work, (3) an anesthesiologist, (4) operating room expenses, and (5) hospitalization costs. The cost of scleral buckling with its associated expenses may be ten times that required for pneumatic retinopexy.

It should be noted that not all surgeons agree with all of the advantages of pneumatic retinopexy listed above, and there are many surgeons, particularly

### Table 8–2. Comparison of Scleral Buckling with Pneumatic Retinopexy

<table>
<thead>
<tr>
<th></th>
<th>Scleral Buckle</th>
<th>Pneumatic Retinopexy</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anatomic success</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 operation ± laser/cryo</td>
<td>84%</td>
<td>81%</td>
<td>NS</td>
</tr>
<tr>
<td>Final reattachment</td>
<td>98%</td>
<td>99%</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Visual results:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20/50 or better</td>
<td>69%</td>
<td>88%</td>
<td>0.05</td>
</tr>
<tr>
<td><strong>Morbidity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Complications:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cataract</td>
<td>18%</td>
<td>4%</td>
<td>0.05</td>
</tr>
<tr>
<td>New/missed breaks</td>
<td>13%</td>
<td>23%</td>
<td>0.05</td>
</tr>
<tr>
<td>Choroidal detachment</td>
<td>17%</td>
<td>3%</td>
<td>0.001</td>
</tr>
<tr>
<td>Myopic shift</td>
<td>68%</td>
<td>3%</td>
<td>0.001</td>
</tr>
<tr>
<td>PVR</td>
<td>5%</td>
<td>3%</td>
<td>NS</td>
</tr>
</tbody>
</table>
outside the United States, who perform the procedure rarely. Also, microincisional vitrectomy is becoming increasingly popular for treatment of primary retinal detachments (particularly in pseudophakic cases), including patients who may be candidates for pneumatic retinopexy.

SUMMARY

Pneumatic retinopexy is an alternative to scleral buckling or vitrectomy for the surgical repair of selected retinal detachments. A gas bubble is injected into the vitreous cavity, and the patient is positioned so that the bubble closes the retinal break(s), allowing resorption of the subretinal fluid. Laser photocoagulation or cryotherapy is applied around the retinal break(s) to form a permanent seal. The procedure can be done in the office, and no incisions are required.

Pneumatic retinopexy may be appropriate to consider in selected cases without inferior or extensive retinal breaks and without significant proliferative vitreoretinopathy. In selected cases appropriate for PR, lower morbidity, fewer cataracts, decreased expense, and possibly better visual results may be achieved in exchange for a lower single-operation success rate and the need for precise postoperative positioning and close follow-up care.

SELECTED REFERENCES


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Following the introduction of closed vitrectomy techniques by Robert Machemer in the early 1970s, complicated retinal detachments became one of the important indications for vitreous surgery. Most of these were due to proliferative diabetic retinopathy (PDR) or to proliferative vitreoretinopathy (PVR), frequently following failure of routine scleral buckling procedures. As experience in vitreoretinal surgery expanded, the advantages of these techniques in the management of more routine types of retinal detachment became apparent. The popularity of vitrectomy for primary retinal detachments continues to grow, particularly with regard to pseudophakic cases.

Indications for performing a vitrectomy rather than a scleral buckle or a pneumatic retinopexy are summarized in Chapter 10. Virtually all authorities note that a vitrectomy is required (along with a broad scleral buckle) in eyes with severe PVR, and the technique is also clearly indicated for cases due to PDR, detachments associated with major vitreous hemorrhage or scarring from penetrating trauma, and those with giant retinal tears. On the other hand, few would suggest a vitrectomy to repair a very shallow and small retinal detachment due to a single break that could be easily closed with a scleral buckle or pneumatic procedure. Between these two extremes, indications remain a matter of personal choice of the surgeon, and they are influenced by his or her training and experiences with a variety of techniques. Most surveys demonstrate a growing popularity of vitrectomy for an increasing percentage of cases.

The goals of vitrectomy for retinal detachment are to

1. Remove axial opacities such as vitreous hemorrhage or debris.
2. Eliminate vitreoretinal, epiretinal, or subretinal traction.
3. Identify and treat all retinal breaks.
4. Internally reattach the retina.
5. Facilitate placement of a large intraocular tamponade.
6. Avoid complications associated with scleral buckling surgery.

The usual sequence of events includes removal of vitreous gel and epiretinal membranes, identification of retinal breaks, internal removal of subretinal fluid, laser therapy to all responsible breaks and areas of significant vitreoretinal degeneration, and placement of an internal tamponade with gas or silicone oil. Vitrectomy is frequently combined with placement of a scleral buckle.

**VITRECTOMY TECHNIQUES FOR ROUTINE RETINAL DETACHMENTS**

Vitrectomies are routinely performed with an operating microscope, endoillumination, an automated vitreous cutter, a laser photocoagulator, and an accessory viewing system. Thus, the necessary equipment needs are substantially greater and more costly than those required for a pneumatic operation or scleral buckle.

**EXTERNAL STEPS OF THE PROCEDURE**

**Prep and drape**
This is performed in the same fashion as described for scleral buckling in Chapter 7.

**Opening incisions**
If a 360-degree buckle is anticipated, a 360-degree conjunctival peritomy is made (as described in Chapter 7). If only a vitrectomy is planned, a temporal conjunctival incision from mid-quadrant to mid-quadrant is created at the limbus. Some add a radial incision near the lateral rectus muscle. Smaller incisions are also made in a similar manner nasally. If small-gauge (23- or 25-gauge) instruments are used, no conjunctival incisions are made, and the trochars are inserted directly through the conjunctiva.

Sclerotomies are placed 4 mm posterior to the limbus in phakic eyes, and 3.0–3.5 mm posteriorly in pseudophakic or aphakic cases. A 20-gauge myringotomy knife is employed to make the three routine 20-gauge sclerotomies. An infusion cannula is usually sutured to the globe if a routine 20-gauge vitrectomy system is to be employed. The infusion site is traditionally in the inferior temporal quadrant, just below the lateral rectus muscle insertion. Two additional incisions are made just superior to the horizontal rectus muscle insertions, one for a fiberoptic light pipe, and the other for the cutting/aspiration probe, intraocular scissors or forceps, or other instruments.

Few surgeons place trochars at each incision site in 20-gauge cases, but these are routinely employed with smaller gauge systems. The operation is performed in a dark room. Optimal visualization of the posterior segment requires accessory
lenses, and for peripheral vitreoretinal surgery, the introduction of wide-angle viewing systems has greatly improved capabilities.

**INTERNAL STEPS OF THE PROCEDURE**

**Removal of vitreous gel**

A physiologic solution is infused under pressure controlled by the automated machine or by the height of the container of fluid. With suction applied to the cutter, vitreous enters the port, is amputated by the high-frequency cutter, and is slowly aspirated via the tubing connected to the probe (Figure 9–1).

The vitrectomy probe and fiberoptic light pipe may be temporarily withdrawn during the operation to introduce other instruments through the same sclerotomy, or to permit examination of the fundus by means of binocular indirect ophthalmoscopy. In the latter situation, the pars plana sclerotomy sites are temporarily closed with nail-like scleral plugs.

The vitreous gel and associated vitreous hemorrhage are initially removed centrally, where a fluid space is created. The remaining vitreous gel is then excised to remove intravitreal opacities and allow for identification of the posterior vitreous surface. The location of the posterior vitreous surface varies from case to case, ranging from a complete posterior vitreous detachment in many eyes with rhegmatogenous retinal detachment, to multiple residual posterior vitreoretinal adhesions in highly myopic cases, to widespread vitreoretinal adhesions in some eyes with PDR or a history of penetrating trauma. The posterior vitreous surface is initially incised where there is ample separation between the vitreous and the underlying retina (Figure 9–2).

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*Figure 9–1. A standard three-port vitrectomy procedure. The infusion cannula, located at the site of the third incision, is not shown on this illustration.*
After the posterior vitreous gel has been removed, more peripheral vitreous is cautiously excised, and the vitreous base is approached. Vitreous gel adherent to the margins of the break(s) is removed. Sometimes the traction is best relieved by excising the flaps of horseshoe tears. As the vitreous base is approached, its visualization can be enhanced with external scleral depression, usually by the assistant. Alternatively, if a scleral buckle is planned, the encircling band can be tightened to provide an indentation similar to that achieved with scleral depression.

**Installation of heavy perfluorocarbon liquids**

Heavy perfluorocarbon liquids such as perfluorooctane (PFO) may be used at this stage to help reattach the retina. It is injected into the vitreous cavity over the posterior retina. Being heavier than water, it forces subretinal fluid peripherally and out the retinal break(s), pressing the retina against the wall of the eye (Figure 9–3). This aids in identifying breaks, reduces the height of the detachment, allows for laser photocoagulation, and provides relative immobilization of the retina. It is safer to trim vitreous close to the mobile peripheral retinal if the detachment has been stabilized by PFO (Figure 9–4), which is usually infused to the level of the equator. Some surgeons use heavy liquids in most operations, whereas other use it infrequently except in complicated cases. It is almost always removed from the eye before the end of the case.

**Identification and marking of retinal breaks**

The most important step in all forms of retinal reattachment surgery is to identify and treat all retinal breaks. In vitrectomy procedures, retinal breaks are usually
marked with internal diathermy, which leaves a visible white burn in the retina at the edge of the break(s). Since air infusion is often performed before the breaks are treated, it is important to mark the breaks before this step, as they are much more difficult to see in air-filled eyes.

Figure 9–3. Heavy perfluorooctane (PFO) is injected into the vitreous cavity over the posterior retina. This forces subretinal fluid peripherally and out the retinal break(s) (large arrow), pressing the retina against the wall of the eye.

Figure 9–4. It is safer to trim vitreous close to the mobile peripheral retinal and to excise large tear flaps if the retinal detachment has been stabilized by PFO.
Internal drainage of subretinal fluid

Since retinal breaks cannot be treated with laser unless the retinal pigment epithelium (RPE) is in contact with the retina, drainage of subretinal fluid is performed prior to laser therapy. Some surgeons remove subretinal fluid with the assistance of heavy liquids, while others routinely drain during air infusion without PFO. Still others infuse air while draining both subretinal fluid and PFO from the eye. Drainage is usually accomplished by inserting an aspiration needle with a soft flexible tip through an accessible retinal break (Figure 9–5) or simply by pressing fluid out of the subretinal space using PFO and air as described below. Alternatively, fluid can be drained through a small posterior retinotomy which is usually created superior to the optic nerve.

Air infusion

Vitrectomy for routine retinal detachment usually includes infusion of air to internally tamponade the retina. Some surgeons begin the air infusion while the PFO remains posterior to the equator and/or retinal breaks. In this situation, the air pushes residual subretinal fluid toward the equator from the front of the eye, while the PFO pushes it toward the equator from the back, forcing it out of the equatorial break(s). After the anterior retina is completely flat, the PFO is aspirated as the air bubble expands to fill the vitreous cavity (Figure 9–5). A disadvantage of this technique is that small, residual PFO bubbles are relatively difficult to see through an air bubble, and some PFO may inadvertently be left in the eye. By reinstituting some saline fluid, the remaining PFO may be visualized and removed, followed by removal of the saline.

Figure 9–5. Drainage of subretinal fluid is usually accomplished by inserting an aspiration needle with a soft flexible tip through an accessible retinal break. This may be done before air is infused, or during an air–PFO exchange, as shown here.
A decision is made about the type of gas to leave in the eye. Tamponade of all retinal breaks is desirable for several days minimum, so that a mature adhesion will evolve around each of them. In relatively simple cases with superior breaks, air alone may suffice, but in more complicated cases, especially those with large breaks located inferiorly, a larger, long-acting bubble is desirable. In these situations, a dilute mixture of either sulfur hexafluoride (SF₆) or perfluoropropane (C₃F₈) is exchanged for the air. Mixtures with 20% sulfur hexafluoride or 15% perfluoropropane are usually used for a total gas fill since these are nonexpansile mixtures. In very complicated cases, silicone oil is sometimes substituted; this is discussed later.

**Laser treatment of retinal breaks**

Retinal breaks are treated after the retina is internally reattached. This is performed with laser photocoagulation through an endoprobe, via an indirect ophthalmoscopic delivery system, or both. Two to three rows of burns are placed around each break (Figure 9–6). Some surgeons prefer to then create 360 degrees of laser therapy in several rows that straddle the posterior margin of the vitreous base, where any postoperative vitreoretinal traction would be likely to occur.

**Placement of scleral buckle**

With regard to vitreous surgery for routine and uncomplicated retinal detachment, considerable debate persists about the value of a scleral buckle. When employed, a 360-degree encircling band is usually preferred. This is usually secured to support the posterior margin of the vitreous base along with the retinal breaks. If a
buckle is planned, it is frequently placed beneath the four rectus muscles (as noted in Chapter 7) prior to beginning the vitrectomy.

Proponents of a scleral buckle for routine cases believe that it is useful in reducing the chances of later retinal detachment. Others use buckles primarily when inferior breaks are encountered and there is concern about the gas bubble providing a tamponade for a sufficient time for an effective adhesive scar to form around the breaks. Those who do not routinely employ buckles cite the lack of data documenting their value and express concern about adding the complications of buckling to those of vitrectomy.

**Closure of sclerotomies and peritomies**

At the end of the operation, each pars plana 20-gauge incision is closed with a figure-of-eight, 7-0 polyglycolic acid suture. If small-gauge instruments have been employed, the wounds are considered to be self-sealing, and no sutures are required. The conjunctival peritomies are closed in a routine fashion if 20-gauge instruments were used. This is not necessary in cases in which small-gauge devices were employed.

**VITRECTOMY TECHNIQUES FOR COMPLICATED CASES**

Complicated retinal detachments were managed with vitrectomy techniques for many years before more routine cases became common indications for this surgery. The most important of these were retinal detachments associated with PDR or PVR. Detachments due to giant retinal tears are another obvious indication for vitrectomy. This section will briefly discuss techniques that are employed in the management of these three types of complicated retinal detachment, emphasizing differences from the usual sequence of surgical steps mentioned above.

**Retinal Detachment and Proliferative Diabetic Retinopathy**

In proliferative diabetic retinopathy (PDR), extraretinal neovascular and fibrovascular tissue grows almost exclusively along the posterior vitreous surface. This proliferation often causes changes in the vitreous gel that result in a partial posterior vitreous detachment with separation of the cortical vitreous from the retina. The vitreous remains attached to the anterior retina at the vitreous base, and it often exhibits a funnel-shaped configuration that extends between the vitreous base anteriorly to areas of neovascular proliferation posteriorly. The taut posterior vitreous surface causes anteroposterior traction on the areas of vitreoretinal attachment, and usually remains attached at each area of posterior fibrovascular proliferation (Figure 9–7).

The posterior cortical vitreous remains near the plane of the retinal surface, where it bridges from one area of retinal neovascularization to another. When little or no posterior vitreous detachment occurs, the proliferative tissue grows along the plane of the inner retinal surface, and widespread adhesions to the retina may develop (Figure 9–8). This tissue can contract, causing tangential traction on the retina and visual loss from distortion or displacement of the macula.
Fibrovascular tissue growth and secondary changes in the vitreous gel can cause further complications, including hemorrhage, traction retinal detachment, retinal breaks, and rhegmatogenous detachment.

The posterior vitreous surface is therefore of great importance in the pathogenesis of PDR and its secondary complications. Complete removal of the posterior
cortical vitreous and all attached fibrovascular tissue is the major goal in vitrectomy for PDR. If this can be accomplished, retinal reattachment, using the techniques described earlier, can usually be achieved.

The steps in vitrectomy for retinal detachment associated with PDR usually involve an initial partial vitrectomy, removal of the posterior cortical surface and fibrovascular tissue, and treatment of retinal breaks, if they are present.

**Vitrectomy for PDR and retinal detachment**

The posterior vitreous surface is frequently incised in a safe location away from vitreoretinal attachments and/or areas of underlying retinal detachment. If non-clotted blood is present in the preretinal space behind the posterior vitreous surface, it is evacuated using an aspiration device.

Then the posterior vitreous surface is excised around the circumference of its cone-like structure to relieve traction between the vitreous base and the posterior retina. Often, the edges of the posterior vitreous surface separate widely after it is cut, demonstrating that considerable traction was present preoperatively. At this stage, some surgeons prefer to remove as much of the cortical vitreous as possible, except for the anterior portion adjacent to the vitreous base, and posterior remnants near areas of vitreoretinal attachment (Figure 9–9). Others remove the avascular gel at the same time that the fibrovascular tissue itself is excised.

When there is little or no posterior vitreous detachment, the posterior cortical vitreous can be dissected from the retina with a vitreoretinal pick, sharp bent-tipped needle, or myringotomy knife. A larger opening in the separated cortical vitreous is then made with the vitrectomy probe.

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**Figure 9–9.** Some surgeons prefer to remove as much of the cortical vitreous as possible, except for the anterior portion adjacent to the vitreous base and posterior remnants near areas of vitreoretinal attachment.
**Removal of fibrovascular PDR tissue**

“Delamination” and “en bloc” are terms used to describe methods of dissecting epiretinal fibrovascular tissue from the surface of the retina. Using the delamination technique, surgeons initially remove all elevated cortical vitreous that is producing anteroposterior traction on posterior fibrovascular tissue. Vitreoretinal picks and scissors are then used to elevate and divide avascular portions of the more organized posterior vitreous cortex until only islands of tightly adherent fibrovascular tissue remain. Bimanual techniques are used to delaminate those focal membranes that appear to be excisable without major structural damage to the retina (Figure 9–10). Other surgeons prefer the en bloc technique, leaving portions of anterior vitreous gel intact, so that the residual anteroposterior traction will assist in the dissection of epiretinal tissue by elevating its edges (Figure 9–11).

Removing all fibrovascular “islands” is believed to reduce the frequency of postoperative bleeding, contraction of residual epiretinal membranes, recurrence of epiretinal proliferation, and missed small retinal breaks. Therefore, “segmentation” techniques, in which localized islands of epiretinal fibrovascular tissue are left following vitrectomy, have become less popular. Still, segmentation techniques are useful when the combination of relatively mature epiretinal tissue and atrophic retina results in a significant increase in the risk of retinal breaks associated with complete epiretinal membrane dissection, or when vascular membranes are located anteriorly and are difficult to remove safely.

If no segmentation has been performed, the entire organized posterior cortical vitreous is removed in a single piece. Many authors favor an “inside-out” dissection, in which the plane of dissection between the vitreous and retina is initiated

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**Figure 9–10.** Following vitreous removal, the fibrovascular islands are removed with bimanual techniques.
centrally, while others prefer to begin the dissection peripherally. Fibrovascular tissue attached to the optic nerve head is gently avulsed or excised from the nerve after all surrounding attachments to the retina have been eliminated. If retinal mobility associated with retinal detachment interferes with dissecting epiretinal tissue, a small amount of perfluorocarbon liquid is injected onto the posterior retina. This stabilizes the retina and makes delamination easier.

Combinations of techniques are frequently employed, but the goal to remove the posterior cortical surface out to the vitreous base along with all fibrovascular membranes is always the same in these cases. Nonvascularized posterior hyaloid and immature proliferative membranes can frequently be elevated and bluntly dissected from the surface of the retina. Recently proliferating tissue is characterized by neovascularization with little or only translucent fibrous tissue. Older fibrovascular tissue becomes more white and opaque and more firmly adherent to the retina.

Vitreoretinal attachments are often especially firm on detached retina, and thinning of the retina from chronic traction and vascular nonperfusion increases the risk of causing a retinal break during removal. Removal may be impossible if extensive mature tissue and firmer attachments to an underlying atrophic retina are encountered in association with extensive retinal detachment. If surgery is performed relatively early in the proliferative process, it is usually easier to remove neovascularization associated with cortical vitreous from the retina. The most favorable cases are those with widespread separation of the vitreous from the retina with only small posterior zones of fibrovascular tissue adherent to the retina.

Intraoperative hemorrhage during segmentation and delamination is unfortunately not uncommon. Its incidence can be reduced by avoiding segmentation of
highly vascularized membranes. Elevation of intraocular pressure is used to minimize bleeding during the dissection of vascularized tissue. Further elevation of intraocular pressure to a level above systolic blood pressure for 1–2 minutes will frequently stop persistent bleeding. Unimanual (Figure 9–12) or bimanual bipolar diathermy can be applied to sites of persistent bleeding other than the optic nerve. Severe bleeding can be reduced by adding thrombin to the infusion fluid, but this makes removal of preretinal hemorrhage more difficult.

**Treating retinal breaks in PDR**

Retinal breaks are treated before the operation is completed. Subretinal fluid is evacuated through suitable posterior breaks. This flattens the retina against the pigment epithelium, permitting transvitreal laser photocoagulation. Internal drainage of subretinal fluid also demonstrates whether all traction on the retina has been relieved. If the retina tends to redetach rapidly despite release of all fibrovascular tissue, the surgeon should search for thin avascular membranes peripheral to the sites of neovascularization and remove them. Drainage of subretinal fluid is frequently combined with fluid–gas exchange, and laser photocoagulation through the gas bubble is then applied around all retinal breaks.

**Retinal Detachment and Proliferative Vitreoretinopathy**

Proliferative vitreoretinopathy has characteristic features that are distinctive and quite different from features of retinal detachment complicating other conditions, such as PDR or retinal detachment occurring after penetrating injuries. PVR has a wide range of clinical severity, but the underlying anatomic features are remarkably
similar from case to case. A standardized terminology and classification have been developed based on the description of the anatomic changes.

The basic pathologic process in eyes with PVR is growth and contraction of cellular membranes on both sides of the retina, on the posterior vitreous surface, and within the vitreous base. Most variations in clinical appearance among cases are due to differences in the anatomic location and severity of the proliferative process. Contraction of membranes on the inner retinal surface causes distortion and folding of the retina. Early changes due to localized epiretinal membranes are recognized as *star folds*; if these occur in the macula, they constitute *macular puckers*.

Epiretinal proliferation is usually most severe in the posterior pole and the equatorial part of the inferior quadrants, probably because free pigment epithelial cells in the vitreous cavity become attached to the most dependent parts of the retina. Growth and contraction of membranes may be extensive, causing total retinal detachment and marked distortion and immobilization of the entire retinal surface. Ultimately, the retina assumes a narrow funnel shape in the center of the vitreous cavity (Figure 9–13; see also Figures 2–24 and 10–12).

The process is particularly severe if cellular invasion and contraction occur within the vitreous base and over the adjacent pars plana. This usually occurs in the two inferior quadrants, and causes elevation and anterior displacement of the peripheral retina. In extreme cases the retina is dragged onto the pars plicata, and sometimes the retina becomes adherent to the posterior surface of the iris. This results in relative foreshortening of the retina elsewhere, and the retina cannot flatten against the eye wall, even after release of transvitreal traction and removal of posterior epiretinal membranes. This far anterior proliferation may also cover the ciliary processes or cause traction on the ciliary body, contributing to the chronic hypotony that occurs in some cases, despite successful retinal reattachment.

*Figure 9–13. Proliferative vitreoretinopathy (PVR). The posterior retina exhibits fixed folds due to contraction of cellular membranes upon its surface.*
The basic surgical goals in eyes with retinal detachment complicated by PVR are to relieve as much vitreoretinal and epiretinal membrane traction as possible, to close the retinal breaks, and to relieve remaining anterior vitreoretinal traction using a broad scleral buckle.

**Scleral buckle for PVR**

A broad and high scleral buckle is usually made, extending from the ora serrata to the equator. Frequently, a silicone exoplant is used that is 7 mm wide and 3 mm thick, and has a convex surface against the sclera. This produces a rounded buckling effect that is effective in supporting areas of persistent vitreoretinal traction, and in sealing retinal breaks while causing minimal radial folding of the retina. The broad exoplant is extended from 180 to 360 degrees, depending on the extent of the PVR, and is combined with an encircling band 2.5 mm wide placed in the groove of the exoplant. When a scleral buckle is already present, the silicone hardware is revised or replaced if the buckling effect is not of the desired location, shape, and height.

**Removal of vitreous**

The vitrectomy probe is first used to excise the central part of the vitreous gel. The typical taut transvitreal sheet representing the posterior vitreous surface is excised up to the circumferential junction between this membrane and the retina.

**Removal of epiretinal PVR membranes**

The objective is to remove as much of the abnormal tissue as possible and to relieve tangential traction between separate retinal folds without making retinal breaks (Figure 9–14). The rigid, distorted retina thus becomes mobile and can then conform to the contour of the eye wall when the retinal breaks are closed. A vitreoretinal pick is used to engage the epiretinal membrane, and gentle posteroanterior traction is used to peel the membrane from the inner retinal surface. Posteroanterior traction minimizes the risk of causing an iatrogenic retinal break because the retina is thicker posteriorly and the junction of the retina with the optic nerve provides strong counter traction during the dissection. Initial identification of epiretinal membranes is also aided by using blunt instruments to separate adjacent tight retinal folds, thereby exposing the membrane causing the folding. This is usually done with the fiberoptic illuminator and the back surface of the vitrectomy probe or with a vitreoretinal pick.

Removal of epiretinal membranes is greatly aided by the use of intraocular forceps, which are used to grasp the membrane after it has been partially separated with the pick (Figure 9–15). Traction on the membrane can be applied more evenly with forceps, and the membrane is therefore less likely to tear or shred. Sometimes a technique with two forceps or forceps and a pick is used. This requires an ancillary light source, such as a separate fiberoptic device similar to the infusion cannula sutured to the sclera, or a light source attached to the intraocular forceps or the pick.

In some eyes, broad sheets of abnormal tissue are easily separated from the retina. In other cases, the tissue sheets fragment and only small portions can be
removed. Care is taken when applying traction on epiretinal tissue to avoid causing a retinal break. This is especially important when removing epiretinal membranes that are attached near the center of the macula, or membranes in the midperiphery where the retina is thin and easily torn.

Figure 9–14. Vitrectomy for proliferative vitreoretinopathy (PVR).

Figure 9–15. PVR membranes are usually removed with vitreous forceps. Frequently, PFO is injected to immobilize the posterior retina during the dissection.
As the retina becomes relatively mobile, dissection of epiretinal membranes can be facilitated by injecting PFO onto the posterior retina. This immobilizes the retina, making peeling, segmentation, and delamination of the proliferative tissue more efficient. In addition, areas of posterior residual traction can often be more easily identified under the PFO bubble.

Posterior epiretinal membranes are removed and/or bridging traction is released as far peripherally as possible, although this process is often limited by the circumferential ring of vitreoretinal traction that marks the peripheral limit of the posterior vitreous detachment. Removal of as much as possible of the basal cortical vitreous anterior to the ring of circumferential traction is always performed if the anterior component of PVR is severe and the eye is not phakic. This dissection is greatly aided using wide-angle viewing systems and by scleral indentation to improve visualization. Sometimes this is best performed with the coaxial illumination of the operating microscope and without intraocular illumination or an accessory lens.

If the anterior retina and vitreous base have been displaced anteriorly by the contraction of fibrocellular membranes on the vitreous base and peripheral anterior hyaloid, a circumferential incision in the remaining vitreous gel and the abnormal tissue is made with a myringotomy knife, scissors, or vitrectomy instrument. Adhesions between the vitreous base, pars plicata, and posterior iris surface are similarly excised. The relief of significant traction will allow the retina to move more posteriorly into a position in which reattachment is possible.

Retinotomy and retinectomy in PVR

If substantial traction and shortening of the retina persist after extensive removal of membranous tissue, a relaxation retinotomy or retinectomy can be used to allow complete settling of the retina. The decision to perform relaxing retinal surgery is made intraoperatively, after maximal membrane dissection has been completed. The need for this step may not be apparent until an air–fluid exchange has been performed, and tenting of the inferior retina or passage of air into the subretinal space has become apparent. This is most likely to be necessary in eyes with severe PVR and recurrent retinal detachment after a previous vitrectomy and scleral buckle. In many of these cases an adequate dissection of epiretinal membranes is frequently impossible or unsafe, and changes in the existing scleral buckle are usually not attempted.

A retinotomy is performed by enlarging existing retinal breaks or creating a new break so that the edges can separate to relieve traction and allow the retina to flatten. In making a retinotomy, diathermy is first applied along the line of the planned incision to close retinal vessels so that they will not bleed when cut. The incision is made as far anterior as possible but posterior to the contracted retina (Figure 9–16). It should extend well into the normal retina at either end, so that inapparent residual traction forces upon the retina are relieved. At either end of the incision, the cuts can be angled anteriorly to maximize retinal relaxation. A circumferential retinal peritomy made parallel to the equator will assume an ovoid configuration as the traction is relieved.

After a relaxing retinotomy has been made, the anterior flap of retina and the membranes upon it are usually excised to reduce the risks of postoperative
proliferation and contraction. All subretinal fluid is removed during complete fluid–gas exchange. It is essential that the retina flatten completely against the eye wall after the vitreous cavity is filled with gas. If enough residual traction persists so that air passes through an elevated retinotomy into the subretinal space, the retinotomy must be extended until the retinotomy edges and the retina become completely flat. When the posterior and lateral edges of the retinotomy are in firm contact with the pigment epithelium, laser photocoagulation is used to create a confluent zone of chorioretinal adhesion around the edges.

**Marking retinal breaks**

After the objectives of the vitrectomy are completed, all retinal breaks are marked with transvitreal coaxial bipolar diathermy applied directly to the retina. The anterior margin of the break is treated until the tissue turns white. This whitening assists in later identification and treatment of the break(s) after fluid–gas exchange.

**Internal drainage of fluid and vitreous replacement**

If PFO has not been used, internal drainage of subretinal fluid is performed via a retinal break or iatrogenic retinotomy with a soft-tip extrusion needle, as employed for routine cases. After most of the subretinal fluid has been drained, a fluid–gas exchange is begun, intravitreal and residual subretinal fluids are removed from the eye, and total air–fluid exchange is completed. In cases of PVR, the air is always replaced with either a long-acting gas such as perfluoropropane (C₃F₈), or with silicone oil. The latter material does not possess as high a surface tension as gas, but
it does not lose volume in the postoperative period. In some difficult cases in which an inferior retinectomy has been performed, heavy PFO liquids have been left in the eye for several days prior to their removal.

**Giant Retinal Tear**

Giant retinal tears are defined as circumferential retinal breaks of 90 degrees or more. Most such tears are idiopathic, but there is a high rate of bilateral involvement, suggesting a predisposition for this type of tear in certain patients. Idiopathic giant retinal tears occur mainly in males, and there is a high incidence of myopia. The second most common cause of giant retinal tears is blunt trauma. These tears can also result from surgical trauma, such as an anterior vitrectomy, and they occur frequently in patients with Stickler’s syndrome. They can also occur along the posterior edge of large areas of chorioretinal scarring, especially in eyes with the acute retinal necrosis syndrome. The tear usually begins along the posterior margin of the vitreous base because of anterior collapse and traction from the vitreous gel. The tear may also extend posteriorly at one or both ends.

When a circumferential retinal tear extends more than 1 clock-hour, there is a tendency for the mobile posterior edge to become rolled or inverted; this phenomenon is especially common in giant tears (Figure 9–17; see also Figures 2–11 and 10–9). The amount of folding usually increases as the tear enlarges, although it also depends on the amount of liquefaction of the nearby vitreous gel. When a giant tear is caused by blunt trauma, it is due to an immediate mechanical distortion of the globe with avulsion of the vitreous base, and the nearby vitreous gel is
not immediately liquefied. Therefore, accumulation of subretinal fluid may be very gradual, and the posterior flap may not become inverted initially.

Giant retinal tears present special difficulties in surgical treatment. They vary widely in severity depending on the size and location of the break, the position and mobility of the posterior flap of the tear, the number, size, and location of additional retinal breaks, the extent of retinal detachment, the features of the vitreous gel, and any epiretinal membrane formation. The general principles of management of giant retinal tears are to unfold the posterior flap of the tear, flatten it against the eye wall, and seal the tear with a firm adhesion. This may be easily accomplished or quite difficult. Most eyes with an inverted posterior flap require vitrectomy and unfolding of the flap of the giant retinal tear with PFO.

The vitreous is initially excised centrally, and then in the periphery. Any adhesions to the retinal flap are cut, and any epiretinal membranes immobilizing the flap are removed. The anterior flap of the giant tear and adherent vitreous are excised in aphakic and pseudophakic eyes. In phakic eyes the anterior flap is not removed unless it is so large that it can be removed without damaging the lens. After vitrectomy and removal of any epiretinal membranes, the PFO is injected onto the retina in the posterior pole (Figure 9–18). The enlarging “bubble” pushes the peripheral retina outward until it is against the retinal pigment epithelium. A chorioretinal adhesion is then created with laser photocoagulation, although some surgeons defer this step until a successful fluid–air exchange has been performed (Figure 9–19). The PFO is then usually replaced with a nonexpanding, long-lasting air–gas mixture such as 15% C₃F₈, although silicone oil is used in certain cases.

Figure 9–18. After vitrectomy and removal of any epiretinal membranes, PFO is injected onto the retina in the posterior pole to push the peripheral retina outward until it lies against the retinal pigment epithelium.
During the fluid–air exchange, a flexible soft-tipped cannula is used to aspirate intravitreal and subretinal fluid and PFO. It is critical to repeatedly remove all subretinal fluid at the edge of the giant tear. Maintaining a “dry” aqueous-free retina at the edges of the giant tear helps prevent posterior slippage of the retina during this exchange. The fluids in the vitreous cavity are initially removed anterior to the PFO bubble, and residual subretinal fluid is aspirated at the edge of the giant tear. Aspiration of aqueous fluids reaccumulating at the edges of the tear is performed as the aspiration cannula removes all intravitreal fluid posteriorly and replaces it with air. It may be difficult to see small amounts of perfluorocarbon liquid as the air bubble approaches the posterior pole. By temporarily injecting 0.5 to 1 ml of infusion fluid, small residual droplets of PFO become visible, facilitating their removal.

Especially in cases with inferiorly located giant tears, a scleral buckle is usually placed to provide mechanical support for all the peripheral retina. This appears to minimize the risk of recurrent detachment by counteracting later traction on the retinal flap and peripheral retina by epiretinal membranes, supporting areas where unrecognized retinal breaks develop after surgery away from the giant tear, and preventing any anterior recurrent detachment from communicating with and redetaching the posterior retina. The scleral buckle also enhances postoperative identification of the peripheral retina and the edge of the giant tear, and facilitates postoperative photocoagulation if needed. This is particularly useful in phakic eyes.

Giant retinal tears are sometimes complicated by PVR. In these cases the techniques of treating PVR and giant retinal tears are combined. Surgery is often delayed until epiretinal membranes stop proliferating or appear inactive. A high, broad, 360-degree scleral buckle is usually used, because there is generally vitreous
base contraction at the ends of the giant tear and on the opposite side of the eye that cannot be fully relieved or that recur. A lensectomy is performed in phakic eyes with giant tears and PVR, unless there is no anterior component to the epiretinal proliferation. After lensectomy, a complete anterior vitrectomy is performed, with excision of as much tissue in the region of the vitreous base as possible. Meticulous removal of all epiretinal membranes is then performed in a posterior-to-anterior direction to make the retina as mobile as possible. This may be facilitated by injecting a small bubble of perfluorocarbon liquid on the posterior retina to partially reposition it and facilitate identification of epiretinal membranes for easier removal.

Subretinal scar tissue on the exposed outer surface of the retinal flap is also removed to increase the mobility of the retinal flap so that it can be unfolded into a more peripheral location. However, the flap may occasionally remain stiff and infolded, and in some cases excision of the peripheral part of the posterior retinal flap with the vitrectomy cutter is necessary to allow retinal flattening.

RESULTS OF VITRECTOMY

Results of vitrectomy for retinal detachment vary considerably, depending upon case selection. Durable retinal reattachment following vitrectomy for routine retinal detachments ranges from 65% to 100% in various reports, and averages approximately 85%, a figure that is quite comparable to that expected in scleral buckling surgery. One recent prospective randomized, controlled trial comparing these techniques has been performed. However, the results were quite complex and inconsistent with some additional non-controlled data.

Visual results are comparable to those seen following anatomically successful surgery with scleral buckling or pneumatic retinopexy, with the vast majority of patients with macula-off detachments experiencing a significant improvement in vision. Of the multiple factors impacting postoperative vision, preoperative vision is the most important, and ideal trials comparing the postoperative vision obtained with various techniques have not been performed.

COMPLICATIONS OF VITRECTOMY

In phakic eyes, the most important and common complication is that of progressive nuclear sclerosis; this can be expected to occur in the vast majority of cases. Importantly, the complications of altered refractive error and strabismus are quite unusual following vitreous surgery, with the exception of nuclear-sclerotic-induced myopia. Scleral perforation with a suture needle, complications of external subretinal fluid drainage and intravitreal gas injections, fish-mouthing of retinal breaks, and of course implant extrusion (all potential problems with scleral buckling) also do not generally occur with vitrectomy. Increased intraocular pressure, endophthalmitis, PVR, epimacular proliferation, recurrent retinal detachment, and choroidal
detachment all may occur with vitrectomy as well as with scleral buckling. These complications are discussed in Chapter 7.

The most important causes of anatomical failure following retinal reattachment surgery are iatrogenic retinal breaks, new breaks, missed breaks, and PVR. The latter three problems can also be considered to be complications of the disease process that caused retinal detachment. By far the most serious of these is PVR. Severe PVR following vitreous surgery is more likely to feature a serious anterior loop component than PVR after a scleral buckle or pneumatic procedure.

SUMMARY

Vitrectomy techniques represent an elegant means of repairing retinal detachments, and are increasingly employed in the management of routine retinal detachments, particularly nonphakic cases. They are invaluable in the repair of complicated detachment cases, and with microincisional techniques they represent a low-impact method for treatment of less complicated cases. The procedure features endoillumination, high magnification, wide-angle viewing, and vitreous cutting abilities to allow removal of opacities and membranes, unfolding of giant tears, and intraoperative retinal reattachment with the help of gas or perfluorocarbon liquids. Postoperative intraocular tamponade with a total gas fill or with silicone oil is made possible with vitrectomy techniques. Cataracts frequently develop following vitrectomy in phakic eyes.

SELECTED REFERENCES


Selection of Surgery for “Routine” Retinal Detachment

Most rhegmatogenous retinal detachments are blinding disorders unless they are successfully repaired. They were regarded as incurable until the seminal work of Jules Gonin in the 1920s, when an anatomical success rate approaching 50% was first described (see Chapter 1). Anatomical results for routine retinal detachments slowly improved through several decades, reaching the current 85%–90% single-operation success figure for scleral buckling by the 1980s. Unfortunately, a similar improvement in visual results has not occurred because of the profound influence of preoperative macular detachment.

Scleral buckling, once the sole standard of care for uncomplicated cases, has become much less popular worldwide with the development of alternative options starting in the mid 1980s. The most enduring of these are pneumatic retinopexy (PR) (described in Chapter 8) and vitrectomy (described in Chapter 9). Vitrectomy was originally reserved for complicated detachments but became popular for more routine cases as experience and equipment improved. Today, particularly in the United States, scleral buckling, PR, and vitrectomy are standards of care that are widely employed in the management of “routine” or “uncomplicated” retinal detachment, but how frequently each is used varies among different demographic groups. For instance, the popularity of PR varies by geographical location and scleral buckling appears less popular in the hands of relatively young vitreoretinal specialists.

It can be useful to discuss objective clinical criteria that may favor one technique over another. Demarcation, scleral buckling, PR, vitrectomy, and vitrectomy plus scleral buckling have relative indications and contraindications (Table 10–1), as well as limitations and complications. In this brief chapter, clinical factors that may influence the choice of one technique over another, for the types of cases
in which scleral buckling, PR, and/or vitrectomy are neither mandatory nor contraindicated, are discussed. However, it appears clear that we will never universally agree on the “best” operation for a given case, just as a single ice cream flavor will never be favored by all.

**SURGERY FOR COMMON TYPES OF RETINAL DETACHMENT**

There are several relatively common types of uncomplicated retinal detachments (Table 10–2), as well as numerous variables associated with all of them (Table 10–3). Management of retinal detachments with each specific technique is described in Chapters 7, 8, and 9. Complicated detachments are usually managed with vitrectomy techniques, whereas localized, relatively simple cases are usually managed with a “walling-off” (demarcating) procedure employing laser or cryotherapy, with PR, or with a small and localized scleral buckling procedure. Between these two extremes lies a large percentage of cases, probably 50% or more, in which any of the three major options might be considered; combinations of the three are also employed by many surgeons in selected situations. Regardless of technique, if all retinal breaks are surgically closed, and proliferative vitreoretinopathy (PVR) or other more unusual complications do not develop, the procedure will be anatomically successful.
Table 10–2. Common Types of Uncomplicated Retinal Detachments

1. Horseshoe tear(s) with PVD
   a. At margins of lattice lesions
   b. Unassociated with lattice
   c. Along posterior margin of vitreous base
2. Atrophic holes in lattice degeneration
3. Combinations of types 1 and 2
4. Retinal dialysis

The relative indications and contraindications in Table 10–1, the common types of uncomplicated retinal detachments listed in Table 10–2, and the variables contained in Table 10–3 frequently dictate the selection of a specific reattachment procedure. The most important considerations include the location, number, and type of retinal breaks and vitreoretinal degenerative lesions; the relationship between the posterior cortical vitreous and the retina; the clarity of the vitreous cavity; and the status of the crystalline lens.

Table 10–3. Variables Associated with Uncomplicated Retinal Detachments

<table>
<thead>
<tr>
<th>Retinal break(s)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type:</strong></td>
<td>Associated with persistent vitreoretinal traction on edge of break (“horseshoe”)</td>
</tr>
<tr>
<td><strong>Location:</strong></td>
<td>Quadrant</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Posterior surface of cortical vitreous</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total PVD:</strong></td>
<td>Only vitreoretinal traction at vitreous base and near break(s)</td>
</tr>
<tr>
<td><strong>Incomplete PVD:</strong></td>
<td>Residual vitreoretinal apposition and/or traction</td>
</tr>
<tr>
<td></td>
<td>Vitreoretinal traction on lattice lesions</td>
</tr>
<tr>
<td><strong>Location:</strong></td>
<td>Quadrant</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lens status</th>
<th>Clear crystalline lens</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cataract</td>
</tr>
</tbody>
</table>

**Scleral Buckling**

Scleral buckling can be employed in the vast majority of cases in which the retina can be adequately visualized (see Chapter 7). The diminished popularity of this technique is not due to limitations in anatomical success but rather due to the development of alternative techniques that provide acceptable reattachment rates, fewer or different complications, and additional advantages in selected cases.

The most common complications of scleral buckling (other than anatomic failure) do not usually follow PR and vitrectomy. PR offers additional advantages of an office procedure and reduced postoperative discomfort. Vitreous surgery provides a remedy for the most common relative contraindications of buckling, significant vitreous opacification, and posterior retinal breaks.
Advantages of scleral buckling

As the standard of care for decades, scleral buckling’s success and complication rates are relatively well understood. Buckling is usually an extraocular procedure (except for the frequently optional steps of draining subretinal fluid and/or injecting gas), and the risk of endophthalmitis is very low, even when drainage is performed. The cost of equipment and accessory materials is considerably less than for vitrectomy, although much more than for PR. Progressive cataract formation following surgery is much less likely than with vitrectomy. Unlike PR and vitrectomy in general, no special postoperative positioning is usually required with scleral buckling, which may be an important consideration in individuals with arthritis or back trouble.

Disadvantages of scleral buckling

Placement of an encircling buckle frequently induces postoperative myopia. Postoperative muscle imbalance and altered refractive errors are important complications that are more commonly seen following scleral buckling than after PR or vitrectomy. Compared to PR, important disadvantages of scleral buckling include the necessity of performing the procedure in an operating room, with the attendant costs, delays, and additional equipment. More patient morbidity occurs following buckling than after PR. Compared to vitrectomy, significant disadvantages include increased difficulty in the management of very large and/or posterior retinal breaks, and increased patient morbidity following repairs of relatively difficult cases. Whereas experience with the procedure was extensive in the past, a growing number of vitreoretinal training programs appear to be providing less extensive experience with this procedure.

Pneumatic Retinopexy

The classic “ideal” uncomplicated retinal detachment for a pneumatic procedure (PR) is associated with a retinal break or group of breaks located in the upper eight clock-hours of the eye and extending no more than one clock-hour in circumference (see Chapter 8). Although the technique can be employed successfully when breaks are not located either superiorly or close together, fewer surgeons would select the procedure in these instances. Additional features that add to the attractiveness of PR include an apparently total PVD, absence of lattice degeneration and vitreous hemorrhage, and phakic lens status.

PR is associated with approximately a 10% reduction in single-operation anatomical success rate when compared to scleral buckling, but ultimate success following reoperation is not compromised. It, therefore, is a procedure that represents a legitimate standard of care as an option to other forms of reattachment surgery. Interestingly, this operation is considerably more popular in the United States than in Europe or the United Kingdom.

Advantages of pneumatic retinopexy

PR can be performed quickly in an office setting with modest local anesthesia. Patient morbidity is usually less than with alternative operations, and costs are considerably lower with PR. Eyes with recent macular detachment tend to have better visual results with PR than with scleral buckling. Cataract formation does not follow the procedure.
Disadvantages of pneumatic retinopexy

The primary disadvantage of PR is that there are many common types of cases for which it should not be employed (Table 10–1). Most surgeons limit its use to a relatively consistent subset of patients with single superior breaks and few signs of extensive vitreoretinal degenerative disorders. Additionally, even in carefully selected cases, the single-operation anatomic success rate is approximately 10% lower than for scleral buckling. Still, there is no evidence that a failed PR procedure lowers the ultimate anatomic or visual success rate. Postoperative positioning is required, and the patient may not fly by air until most of the gas has resorbed.

Vitrectomy for Primary Retinal Detachment

Until the mid-1980s, vitrectomy was reserved for cases complicated by severe vitreous hemorrhage, PVR, giant tears, and so forth. Since then, this form of surgery has become tremendously popular for more routine cases, particularly for the management of pseudophakic eyes (see Chapter 9).

Advantages of vitrectomy

The primary advantages of vitrectomy include the elimination of media opacities and transvitreal and periretinal membranous traction forces, improved visualization and localization of retinal breaks, internal intraoperative reattachment of the retina, and precise application of adhesive therapy. These steps can usually be accomplished without the complications that are relatively common following scleral buckling.

Disadvantages of vitrectomy

In phakic eyes, the development of nuclear sclerotic cataracts represents a major disadvantage. There is increasing evidence that open-angle glaucoma may develop in pseudophakic vitrectomized eyes over decades following surgery. The costs of this alternative are substantially higher than with PR or scleral buckling. Failure of vitrectomy may be associated with the development of relatively severe forms of PVR, although considerably more research is needed to evaluate this phenomenon. There is a lack of information regarding precise causes of failure following vitrectomy, and as additional data accumulate in regard to this relatively new technique, more answers will hopefully be forthcoming.

TWELVE REPRESENTATIVE CASES

Most retinal detachments can be cured with one of several different techniques. There is no one right way; the choice of procedure is influenced by each surgeon’s background and training. Selected surgical techniques for twelve characteristic types of detachments are discussed here, but they are not the only approaches. Note that in each case, some surgeons prefer vitrectomy with or without scleral buckling over scleral buckling alone if the eye is pseudophakic. In this section, cryotherapy is described, but laser applied postoperatively could be used instead. Scleral buckling, PR, and vitrectomy for retinal detachments are discussed in detail in Chapters 7, 8, and 9, respectively.
1. Quadrantic detachment with one break. Figure 10–1 shows an excellent candidate for PR, in the absence of contraindications not related to the eye. PR avoids placement of a scleral buckle under a vertically acting muscle. It also affords the opportunity to promptly protect the macula from impending detachment, and the “steamroller” technique should be used in this case.

If the tear causing a quadrantic detachment is present elsewhere in the upper eight clock-hours of the periphery, PR is still an excellent choice, although positioning for tears in the oblique and lateral meridia will be more difficult than for a 12-o’clock tear. If the tear is in the inferior four clock-hours, PR is usually contraindicated.

If scleral buckling is used, a silicone sponge is sutured to the surface of the globe, creating a segmental scleral buckle. This is usually placed radially, although if the tear lies under a vertically acting muscle, a segmental circumferential buckle of solid silicone may be preferable. Drainage of subretinal fluid is not usually
necessary, but if the break remains widely open after buckling, the surgeon may release the fluid by untying the sutures to restore normal intraocular pressure and then draining the fluid. If significant fish mouthing occurs, a gas injection may be employed with the buckling procedure if the break is located in the superior two-thirds of the fundus.

2. **Total detachment with one break.** If a single tear is found and there are no other suspicious areas, and if the fundus can be thoroughly examined, PR is still an excellent choice if the tear is in the upper eight clock-hours (Figure 10–2).

Scleral buckling technique differs from case number 1 in that most surgeons prefer to drain subretinal fluid, although many cases have been managed satisfactorily without drainage. An encircling (instead of segmental or pneumatic procedure) should be considered if any of the following conditions is present:

---

**Figure 10–2.** Total retinal detachment with one retinal break.
1. The retinal break is not in the expected position.
2. There is question as to the integrity of the peripheral retina.
3. There is evidence of proliferative vitreoretinopathy.
4. The peripheral retina cannot be fully examined in all areas.

3. *Detachment with multiple breaks at same distance from ora*. PR is an option only if all open breaks are within a several clock-hour arc in the upper eight clock-hours of the eye. Vitrectomy is a reasonable choice in a pseudophakic eye (Figure 10–3).

If scleral buckling is chosen, an encircling buckle is recommended. If the breaks are of average size, they can be adequately managed with a 4-mm silicone band without any additional silicone implants. However, if one or more of the breaks are larger than average, the surgeon may supplement the buckle with a wider piece of silicone placed beneath the band. Each break should be surrounded with

---

**Figure 10–3.** Retinal detachment with multiple retinal breaks located the same distance from ora serrata.
cryotherapy and localized on the sclera. The sutures should then be oriented so as to place the posterior edge of the breaks on the crest of the buckle. Subretinal fluid is usually drained if an encircling procedure is performed.

4. **Detachment with multiple breaks at different distances from ora.** Vitrectomy is a good choice in this instance, especially in a pseudophakic eye and when distances from the ora differ widely. PR is only occasionally useful, when limited breaks are appropriately positioned (Figure 10–4).

Scleral buckling for this case involves a broad, grooved silicone implant employed to place all the breaks on a broad buckle. Each break is marked to ensure they all are adequately supported. The implant is usually used in association with an encircling band. It is sutured to the surface of the globe, with at least one (and

Figure 10–4. Retinal detachment with multiple retinal breaks located different distances from ora.
often with two) broad mattress sutures per quadrant over its extent, and the band is routinely anchored to the sclera in the remaining quadrant(s). Subretinal fluid is usually drained. Another approach to this problem is to use a wide silicone sponge, 7.5–12 mm, in a circumferential orientation to create a wide buckle.

5. “Aphakic detachment” with multiple small ora breaks. In an aphakic/pseudophakic detachment, retinal breaks are sometimes tiny and visualization may be patchy, so some of the breaks may not be found. An encircling buckle with or without vitrectomy is frequently employed. It should be placed immediately posterior to the ora breaks. Drainage of subretinal fluid is usually required (Figure 10–5).

Vitrectomy without scleral buckling is also a common choice. A 360-degree peripheral laser photocoagulation is often applied. If the view is excellent and
breaks are appropriately limited, PR is an option, but it carries a lower single-operation success rate than buckling and/or vitrectomy.

6. *Detachment with peripheral break and macular hole*. Macular holes in this instance are usually secondary cystic changes and not causally related to the rhegmatogenous retinal detachment. These cases are generally managed with no treatment to the apparent macular hole. If, however, fluid reaccumulates around the posterior hole in the early postoperative course, a second procedure—usually PR—is required to close the hole (Figure 10–6).

7. *Detachment due to macular break*. Detachments caused by macular breaks are generally seen in association with high myopia, frequently accompanied by

![Figure 10–6. Retinal detachment with a peripheral retinal break and a macular hole.](image-url)
posterior staphyloma, or in cases of ocular trauma. These breaks are often difficult to visualize. They may be small and irregular or slit-like in configuration, and are usually not located in the center of the fovea, but somewhat eccentrically (Figure 10–7).

PR can be recommended. After 1 day of face-down positioning with gas in the eye, the retina will usually be reattached and the break may be treated with extrafoveal laser photocoagulation. Vitrectomy with fluid–gas exchange may be performed instead of PR, or if PR fails.

If that also fails, the surgeon might use a scleral buckle at the macula, but this is rarely required. A Y-shaped buckle is created from a 3×5-mm sponge, and the ends are sutured near the equator in three quadrants. Alternatively, an implant may be sutured to the sclera of the posterior pole. Drainage of subretinal fluid is usually required.

Figure 10–7. Retinal detachment due to a macular break.
8. **Detachment with retinal dialysis.** A detachment with a retinal dialysis is seen most often in the inferotemporal periphery in juveniles or young adults; however, dialyses may occur in any quadrant at any age. Patients are typically phakic, and scleral buckling is generally the procedure of choice. The posterior margin of the dialysis should be treated with contiguous cryotherapy and by securing a segmental episcleral sponge buckle to the surface of the globe with several mattress sutures. The buckle should be placed just behind the posterior edge of the break. Drainage of subretinal fluid is optional (Figure 10–8).

9. **Detachment with giant break.** A giant break is generally defined as a break spanning 90 degrees or more. Especially where a rolled-over flap exceeding 180 degrees is present, vitrectomy with or without a low, encircling scleral buckle is the procedure of choice (See Chapter 9). The use of heavier-than-water perfluorocarbon

---

**Figure 10–8.** Retinal detachment with retinal dialysis.
liquid greatly facilitates this procedure. After removal of all adherent vitreous and peeling of all membranes, the infolded retina is flattened against the wall of the eye with perfluorocarbon liquid. Photocoagulation is applied entirely surrounding the break. Then a gas–fluid exchange is performed on top of the perfluorocarbon, taking care to remove all water at the edge of the break. Perfluorocarbon is carefully replaced with air while continuing to desiccate the edge of the break and preventing it from slipping posteriorly. Critical postoperative positioning is required is required for 5–10 days (Figure 10–9).

10. Detachment with no apparent break. If no retinal break can be found, a secondary retinal detachment (due to uveitis, tumor, or other entities) should be carefully ruled out prior to surgery. Contiguous cryotherapy is applied in one or two rows starting just posterior to the ora in all detached quadrants. This is because most “unseen”

Figure 10–9. Retinal detachment with giant retinal tear.
breaks are probably located anteriorly along the posterior margin of the vitreous base, where they tend to be smaller and can be harder to see. Depending on the extent of detachment, subretinal fluid is usually drained, and an encircling 4-mm scleral buckle is typically placed between the ora and the equator (Figure 10–10).

Some surgeons prefer vitrectomy in this situation, in both phakic and pseudophakic cases, because installation of heavy vitreous substitutes causes the subretinal fluid to exit the subretinal space via the break(s), which can thus be identified. As a rule, PR is not considered in cases in which the causative break cannot be found, but an exception to this might be made if the gas can cover the entire span of the detachment.

11. Detachment with outer-layer break in retinoschisis. Minimal subretinal fluid requires no treatment. Moderate subretinal fluid (two to four disc diameters) may frequently be managed with cryotherapy or photocoagulation alone. If there is a clinical retinal

Figure 10–10. Retinal detachment with no apparent retinal break.
detachment, each of the outer-layer breaks should be carefully treated with cryotherapy and closed with scleral buckling. Subretinal fluid can be drained as indicated. These maneuvers generally cure the retinal detachment. If the surgeon also wants to collapse the retinoschisis cavity, additional cryotherapy can be applied to cover the entire retinoschisis area. With such cryotherapy, the retinoschisis cavity usually partially collapses after several weeks, and occasionally totally collapses. Inner-layer breaks require no treatment and can safely be ignored (Figure 10–11).

Most surgeons reserve vitrectomy for those cases in which outer-layer breaks are far posterior and their buckling would be difficult. PR generally has a lower rate of success in schisis detachments.

12. Detachment with PVR. This most difficult problem has a limited prognosis and is the most common cause of ultimate failure to reattach the retina, even
after multiple surgeries. Grade C1 or C2 PVR (see Table 5–1) can frequently be cured with a high encircling buckle. For grade C3 or greater, vitrectomy is recommended and is the mainstay of treatment for PVR. PR is contraindicated unless just mild PVR is present and it is well away from the causative break(s) (Figure 10–12).

**ALGORITHM FOR CHOOSING SURGERY FOR RETINAL DETACHMENT**

Figure 10–13 attempts to organize the process of deciding the type of surgery to perform for retinal detachment. It is not meant to be exhaustive, and the particular
circumstances of each individual case must be taken into account in deciding on the right surgery for a given patient. Much depends on the experience and comfort level of the surgeon with each procedure or on what equipment is available. There are many relevant details of a case that are not taken into account in this
algorithm, and it provides only rough guidelines. Again, the concepts presented in this book must be thoroughly understood in order to make an appropriate selection of a surgical procedure. This algorithm reflects only the opinion of the authors and does not establish the standard of care for a given case.

Some limited detachments may need only delimiting laser or cryopexy treatment. This is usually done with a laser indirect ophthalmoscope, but cryopexy can be used as well. There is no consensus regarding which small detachments only need delimiting treatment rather than surgery for the detachment to resolve. Generally, new retinal tears are delimited even if a small area of subretinal fluid surrounds them. Also, a shallow, asymptomatic, or chronic retinal detachment that doesn’t extend posterior to the equator and has a limited circumferential extent would be demarcated rather than repaired. Sometimes such detachments prove to be not progressive.

Assuming that definitive repair is required, the algorithm then goes through a list of contraindications to PR. Typically, if there are no contraindications to PR, this may be the procedure of choice since it is the least morbid procedure and may yield the best visual outcome. However, the surgeon or the patient may choose another approach.

If PR is contraindicated or is not selected, the algorithm goes through findings that may suggest vitrectomy as the procedure of choice. Lacking those, scleral buckling is often the selection. However, preferences vary greatly, and the choice depends on the surgeon and the patient.

CONCLUSION

The fundamental goal of all forms of surgery for retinal detachment is the identification and closure of all responsible retinal breaks; if this can be accomplished without complications, the development of new retinal breaks, and/or the development of PVR, the procedure will be successful. Currently, reattachment surgery is performed using one of the three techniques, or combinations thereof, described in this chapter.

There is relative agreement among surgeons in regard to the use of vitrectomy with or without scleral buckling for complicated retinal detachments. Vitrectomy is also increasing in popularity for noncomplicated pseudophakic retinal detachments, particularly using microincisional techniques. PR may have advantages in the relatively simple cases for which it is an option, although there is wide variation in how broadly that category is defined. Scleral buckling can be useful in a wide spectrum of cases, but the degree to which it is used depends very much on the surgeon’s preference.

There are many factors discussed in this book that may influence the selection of one procedure over another, but we still differ widely in our preferences. Hopefully these issues will be clarified by the development of more meaningful evidence bases in the future.
SELECTED REFERENCES


Acetaminophen (Tylenol), 92, 199
Acetazolamide (Diamox), 175
Acuity, decreased, 78
Acute retinal necrosis (ARN) syndrome, 21, 223
Afferent pupillary defect, 79
Air infusion, 209, 210–211, 210f
Air injection, intravitreal, 6
Altered refractive error, 179
Amblyopia, 78, 87
American Academy of Ophthalmology, 129
Anesthesia:
general, 91, 92
contraindications to, 189
for pneumatic retinopexy, 190
retrobulbar, 91, 94, 142
subconjunctival, 88, 91, 142, 190
 topical, 68f, 91
Anisometropia, 179, 189
Anterior segment biomicroscopy, 80
Aphakic eye, 42, 86. See also
Asymptomatic aphakic fellow eyes; Pseudophakic eyes
pneumatic retinopexy in, 188
retinal detachment with multiple small ora breaks, 238–239, 238f
ARN. See Acute retinal necrosis syndrome
Aspirin, 90
Astigmatism, 179
Asymptomatic aphakic fellow eyes. See also
Aphakic eye
with history of giant retinal tear, 142
with lattice generation, 141
with retinal breaks, 141–142
Asymptomatic degenerative retinoschisis, 136. See also Degenerative retinoschisis; Retinoschisis
Asymptomatic myopic non-fellow eyes, 133. See also Myopia
Asymptomatic nonphakic fellow eyes:
with history of giant retinal tear, 142
with lattice generation, 141
with retinal breaks, 141–142
Asymptomatic nonphakic non-fellow eyes, 133–134
Asymptomatic phakic fellow eyes:
with cystic retinal tufts, 139
with degenerative retinoschisis, 139–140

Note: Page numbers followed by ‘f’ and ‘t’ denote figures and tables, respectively. Drugs are listed under their generic names; when a drug trade name is listed, the reader is referred to the generic name.
Asymptomatic phakic fellow eyes (Cont.)
with history of giant retinal tear, 140
with lattice degeneration, 137–139, 138f, 139f
with retinal breaks, 140
Asymptomatic phakic non-fellow eyes:
at high risk:
  family history, of retinal detachment, 134
  myopic non-fellow eyes, 133
  nonphakic non-fellow eyes, 133–134
  stickler syndrome, 134
retinal breaks, 136–137
precursors without high-risk,
  134–137
cystic retinal tufts, 136
degenerative retinoschisis, 136
lattice generation, 135–136, 135f
Atrophic retinal holes, 18–19, 19f. See also
Retinal holes
Atropine, 49
Bagel sign, 195
Balanced salt solution, intravitreal
  injection of, 170
Bell’s reflex, 50
Binocular ophthalmoscope:
direct, 46
  field of view, 43–44
  illumination, 44–45
  magnification and resolution, 41–43
  optical principles, 42f
  stereopsis and depth of focus, 45
indirect:
  adjusting, 47–48, 48f
  in children and uncooperative adults,
  45–46
  choice of, 47
  condensing lenses, choice of, 48–49, 49f
dilation, in infants, 50
eye movement, 50–51
illumination, 44–45
image, 45f, 46
magnification and resolution, 41–43
medications, avoiding, 50
ocular media, opacities in, 45
optical principles, 42f
patient, preparation, 49
patient’s position, 50, 51f
pupillary dilation, 49–50
scleral depression, 46
stereopsis and depth of focus, 35
working distance, 46
Biomicroscopy, posterior segment,
  81–82, 81f, 82f, 83f
Blunt trauma, 11, 223
Bullous retinal detachment, 86, 86f
Capsulotomy, 11, 30, 78, 133, 140
Cataract, 88–89
Central acuity, decreased, 78
Central retinal artery:
oclusion of, 195
perfusion of, 163
Charles technique, of draining subretinal
  fluid, 168, 169f
Chemosis, 79
Chorioretinal atrophy, peripheral. See
Cobblestone degeneration
Chorioretinal degeneration, 100, 102f
honeycomb, 100
Choroidal detachment, 90, 123, 124f,
  175–176
hemorrhagic, 176
“hour glass” configuration of, 123, 124f
“kissing”, 176
Choroidal hemorrhage, 173
Choroidal tumors, 125
  with serous retinal detachment, 119, 119f
Choroidal vasculature, 97–98, 98f
Clopidogrel (Plavix), 90
Cloudy media, 187
CME. See Cystoid macular edema
Cobblestone degeneration, 100, 102f,
  103, 104f
Cocaine, 50
Condensing lenses, 48–49, 49f
Congenital retinal rosettes, 100
Conjunctival incision, 154–155
Conjunctival scarring, 189
Corneal epithelial edema, 172
Corneal opacities, 88
Corticosteroids, 90, 92, 175, 177
Cryopexy, 5, 88, 90–91, 90f, 119, 142–143,
  190, 196
  with scleral buckling, 156–158,
  157f, 158f
for surgical failures, 92–94
Cyclopentolate, 49
Cycloplegics, 49, 88, 89, 92, 175
Cystic retinal tufts, 23, 100
  asymptomatic phakic fellow eyes
  with, 139
  asymptomatic phakic non-fellow eyes
  with, 136
Cystoid degeneration, 25, 26f
Cystoid macular edema (CME), 177
Cytomegalovirus (CMV) retinitis, 20, 21f, 119

Degenerative retinoschisis, 25–27, 26f. See also Retinoschisis
asymptomatic phakic fellow eyes with, 139–140
asymptomatic phakic non-fellow eyes with, 136
Delamination technique, of vitrectomy, 215, 215f
Demarcation, 36, 36f, 106, 107f, 108
indications and contraindications, 230t
Dexamethasone (Maxitrol), 92
Diathermy, 5, 72, 142, 155, 158, 166–167, 217, 217f, 221
with scleral buckling, 158
Dilation:
in infants, 50
pupillary, 49–50, 88
Donut sign, 195
Drainage, of subretinal fluid, 164, 166–168, 167f, 169f, 210, 210f
intraoperative complications of, 173–174
Draping, 153, 206
Droperidol, 92
Drusen, equatorial, 100, 102f
Emmetropia, 189
Emmetropic eye, 42, 44f
En bloc technique, of vitrectomy, 215, 216f
Encircling episcleral buckles, 160–163, 161f, 162f, 163f
Endophthalmitis, 175,
Epimacular proliferation, 26f, 177–178, 178f
Exudative retinal detachment, 10
diagnostic features of, 117t
Failure of surgery, 92–94, 93f
Familial exudative vitreoretinopathy, 121
Filtering blebs, 188
Fish eggs, 193, 195f, 197–198, 198f
Flap tears. See Horseshoe tears
Flashes of light, 14, 75–76
Floaters, 76–77
Foveal detachment, 78, 188
Fundus:
changes related to retinal detachment, 106–116, 107f
retinal breaks, detection of, 106–108, 107f
retinal hemorrhage, 108
proliferative vitreoretinopathy, 108t, 109–116, 109f, 110f, 111f, 112f, 113t, 114f, 115f, 116f
changes unrelated to retinal detachment, 97–106
chorioretinal degeneration, 100, 102f
choroidal vasculature, 97–98, 98f
cobblestone degeneration, 100, 102f, 103, 104f
equatorial drusen, 100, 102f
hemiretinal differences, 106
ora serrata, 98–100, 117f, 101f, 102f
pars plana cysts, 102f, 104, 105f, 106
pigment clumps, 105
reticular pigmentary degeneration, 100, 102f, 103f
retinal erosion, 102f, 104
retinal whitening, 102f, 103–104, 105f
examination of, 51f
chart, 58–59, 60f, 61f
condensing lenses, holding, 51–52, 53f
initial view, 52–53
inverted image drawing, 57, 57f, 58f
observer's position and lesion, 58, 59f, 60f
orientation, 55
shifting from one part to another, 54–55, 55f, 56f
troublesome reflexes, 53–54, 54f, 55f
Gas, intraocular:
characteristics of, 184–185
choice of, 181, 183
duration and expansion, 183t
entrapment at injecting eye, 198–199
gas bubble, size of, 183
longevity of bubble, 183–184
making room for, 191
determinations after injection, 195
injecting, 193–194, 193f
preparing, 191, 192f
Gas entrapment, at injecting site, 198–199
Gas–fluid exchange, 171
Gas injection, intravitreal, 151, 170–171
intraoperative complications of, 174
Giant retinal tear, 16, 17f, 20, 21f
with edges folding, 223–226, 223f, 224f, 225f
PVR and, 225–226
Glaucoma, 89
pneumatic retinopexy for, 187
Glial spheres, 100
Goniosol, 50
Granular tissue, 100
Hemiretinal differences, 106
Hemorrhagic choroidal detachment, 176.
See also Choroidal detachment
Hirschberg test, 79
Homatropine, 49, 92
Honeycomb chorioretinal degeneration, 100
Horseshoe tears, 16–17, 17f, 18f
Hruby noncontact technique, 81

Iatrogenic macular detachment, 200. See also Macular detachment
Iatrogenic retinal break, 173. See also Retinal breaks
Indirect lenses, 48–49, 49f
comparison of, 43f
Indirect visualization systems, 61
Inflammation, intraocular, 12
Intraocular gases. See Gas, intraocular
Intraocular pressure, increased, 174–175
Intraoperative laser photocoagulation:
with scleral buckling, 158–159
Intraretinal macrocysts, 35–36, 35f, 118–119
Intrascleral buckles, 164, 164f
Intravitreal air injection, 6
Intravitreal gas injection, 151, 170–171 intraoperative complications of, 174
Keratoplasty, 88
Keratoprosthesis, 88
Kissing choroidal detachments, 176
Krimsky test, 79

Laser photocoagulation, 91, 143–144, 144f, 190–191, 211, 224, 225f. See also YAG laser
intraoperative, 158–159
postoperative, 92–94, 93f, 171
for surgical failures, 92–94
transcleral, 191
transpupillary, 191
Laser test, 5–6, 62, 84
Lattice degeneration, 17f, 23, 27–30, 27f, 62f, 145
asymptomatic phakic fellow eyes with, 137–139, 138f, 139f
asymptomatic phakic non-fellow eyes with, 135–136, 135f
asymptotic nonphakic fellow eyes with, 141
horseshoe tears and, 28, 29f
pneumatic retinopexy for, 188
sclerotic retinal vessels and, 27–28, 29f
with snail-track appearance, 27, 28f

Leopard skin, 158
Lid edema, 79
Liquid currents, 15
Loculated hemorrhage, 176

Macrocysts, intraretinal 35–36, 35f, 118–119
Macular detachment, 86–91, 86f, 87f iatrogenic, 200
optic pits with, 189
Macular holes, 22, 23f, 188. See also Retinal holes
Macular pucker, 26f, 33, 34f, 177–178, 178f
Malignant melanoma, 115f, 121f
Marcus Gunn test, 79
Meperidine, 92
Miosis:
intraoperative, 172
managing, 85
Mydriatics, 49
Myopia, 11

Nd:YAG laser. See YAG Laser
Neomycin, 92
Non-drainage, of subretinal fluid, 165
Nonrhegmatogenous retinal detachment, 116–122
diagnostic features of, 117t
differential diagnosis of, 118t
Non-steroidal anti-inflammatory drugs, 177

Ocular coherence tomography (OCT), 87
Ocular diseases, history of, 78
Ocular examination, 79–80
anterior segment biomicroscopy, 80
external examination, 79
ocular motility, 79
pupillary reactions, 79
tonometry, 80
visual acuity, 79

Ocular history, 75–79
central acuity, decreased, 78
family history, 78
fl ashes of light, 75–76
floaters, 76–77
ocular diseases, 78
systemic diseases, 78
trauma, 78
visual field defects, 77
Ocular massage, 192–193
Ocular motility, 79
Opacities in ocular media, 45, 79, 87
Operculated retinal breaks, 17f, 18, 19f

Ophthalmoscopy, 41–73
direct, 46
  field of view, 43–44
  illumination, 44–45
  magnification and resolution, 41–43
  optical principles, 42f
  stereopsis and depth of focus, 45
fundus, examination of, 51f
  chart, 58–59, 60f, 61f
  condensing lenses, holding, 51–52, 53f
  initial view, 52–53
  inverted image drawing, 57, 57f, 58f
  observer’s position and lesion, 58, 59f, 60f
  orientation, 55
  shifting from one part to another, 54–55, 55f, 56f
  troublesome reflexes, 53–54, 54f, 55f
  indirect:
    adjusting, 47–48, 48f
    in children and uncooperative adults, 45–46
    choice of, 47
    condensing lenses, choice of, 48–49, 49f
  dilation, in infants, 50
  eye movement, 50–51
  illumination, 44–45
  image, 45f, 46
  magnification and resolution, 41–43
  medications, avoiding, 50
  ocular media, opacities in, 45
  optical principles, 42f
  patient, preparation, 49
  patient’s position, 50, 51f
  pupillary dilation, 49–50
  scleral depression, 46
  stereopsis and depth of focus, 35
  working distance, 46
small-pupil, examination of:
  narrow optical aperture, 59–61
  using indirect laser, 61
  using indirect ophthalmoscope, 61
  using indirect visualization systems, 61
Optic nerve disease, 79, 87, 118t
Ora pearl, 100
Ora serrata, 98–100, 101f, 102f
  morphologic variations, on normal fundi, 101f
nasal, 99f, 101f
temporal, 99f, 101f
Orbital cellulitis, 119
Paracentesis, 165, 192
Pars plana cysts, 102f, 104, 105, 106
Paving-stone degeneration. See Cobblestone degeneration
PDR. See Proliferative diabetic retinopathy
Perfluorocarbon liquids, 6
  installation of, 208, 209f
Perfluoroocanote (PFO), 208, 209f, 210, 224, 225
Perfluoropropane (C₃F₈), 170, 181, 183, 184, 211, 222, 224
Perimetry, 83
Peritomy, 154
  closure of, 212
PFO. See Perfluoroocanote
Phagosomes, 33
Phakic eye. See Asymptomatic phakic fellow eyes; Asymptomatic phakic non-fellow eyes
Phenylephrine, 49, 50, 88
Photocoagulation. See Laser photocoagulation
Photomydriasis, 88
Photopsia. See Flashes of light
Pigment clumps, 105
Pneumatic retinopexy (PR), 6, 87, 91, 181–203
  advantages of, 232
  complications:
    iatrogenic macular detachment, 200
    new or missed retinal breaks, 200
    proliferative vitreoretinopathy, 201
    subretinal gas bubbles, 200, 201f
  disadvantages of, 232
  indications and constraints, 186–189, 230t
  intraocular gases:
    characteristics of, 184–185
    choice of, 181, 183
    duration and expansion, 183t
    gas bubble, size of, 183
    longevity of bubble, 183–184
  operative technique:
    anesthesia, 190
    cryopexy versus laser photocoagulation, 190–191
    eye, sterilizing, 191
    gas:
      determinations after injection, 195
      injecting, 193–194, 193f
      making room for, 191
      preparing, 191, 192f
      ocular preparing, 192–193
      paracentesis, 192
Pneumatic retinopexy (PR) *(Cont.)*
- postoperative management, 199–200
- preoperative evaluation, 185–186
- procedure, 182f
  - for scleral buckle failures, 94, 188
  - and scleral buckling, comparison between, 201–203, 202t

Polymyxin B, 92

Posterior subcapsular (PSC) cataract, 80, 88. *See also* Cataract

Posterior vitreous detachment (PVD), 10, 12–14, 13f
- symptomatic, 130, 131

Postnecrotic retinal holes, 20–21, 21f. *See also* Retinal holes

Postoperative management:
- activity restrictions and positioning, 91–92
- medications, 92
- PR. *See* Pneumatic retinopexy

Prepping, 153, 206

Prevention of retinal detachment, 129–147
- asymptomatic nonphakic fellow eyes:
  - with history of giant retinal tear, 142
  - with lattice generation, 141
  - with retinal breaks, 141–142
- asymptomatic phakic fellow eyes:
  - with cystic retinal tufts, 139
  - with degenerative retinoschisis, 139–140
  - with history of giant retinal tear, 140
  - with lattice degeneration, 137–139, 138f, 139f
  - with retinal breaks, 140
- asymptomatic phakic non-fellow eyes, 133–137
  - at high risk:
    - family history, of retinal detachment–, 134
    - myopic non-fellow eyes, 133
    - nonphakic non-fellow eyes, 133–134
    - Stickler syndrome, 134
- precursors of retinal breaks without high-risk, 134–137

Symptomatic eyes:
- breaks unassociated with persistent vitreoretinal traction:
  - precursors, 132
  - symptomatic atrophic retinal holes, 132
  - symptomatic operculated retinal tears, 132
  - tears with persistent vitreoretinal traction:
    - symptomatic horseshoe-shaped tears, 131
    - symptomatic round tears, 131–132
    - treatment, 142–144, 146t
    - complications of, 145–146
    - results of, 144–145

Prochlorperazine, 92

Proliferative diabetic retinopathy (PDR), 21–22, 22f, 205
- and traction retinal detachment, 212–217, 213f
- fibrovascular tissue, removal of, 215–217, 215f, 216f, 217f
- retinal breaks, treating, 217
- vitrectomy for, 214, 214f

Proliferative vitreoretinopathy (PVR), 33–34, 34f, 108t, 109–116, 109f, 110f, 111f, 112f, 113t, 114f, 115t, 116f, 178–179, 187, 201, 205
- and retinal detachment, 217–223, 218f, 244–245, 254f
- epiretinal membranes, removal of, 219–221, 220f
- internal drainage of subretinal fluid and vitreous replacement, 222–223
- retinal breaks, marking, 222
- retinectomy in, 221–222, 222f
- retinotomy in, 221–222, 222f
- scleral buckle for, 219
- vitreous, removal of, 219

Promethazine, 92

Prophylactic therapy, 144
- for asymptotic retinal breaks in phakic non-fellow eyes, 134, 137
- of lattice generation, in asymptotic phakic fellow eyes, 137–138, 139f

Propoxyphene, 92

Pseudoora, 103

Pseudophakic eye, 61, 89. *See also* Aphakic eye, Asymptomatic nonphakic fellow eyes; Asymptomatic nonphakic non-fellow eyes

Pneumatic retinopexy in, 188

Pseudoptosis, 79

Ptosis, 79

Pupillary dilation, 49–50, 88

PVD. *See* Posterior vitreous detachment

PVR. *See* Proliferative vitreoretinopathy

Quadrantic retinal detachment with one retinal break, 234–235, 234f. *See also* Retinal detachment
Radial staphylomas, 154, 155f
Rectus muscles, isolation of, 154, 155, 154f, 155f
Recurrent retinal detachment, 92–94, 93f, 179
Reoperation, 94, 179, 188
Reticular pigmentary degeneration, 100, 102f, 103f
Retina breaks, 15–30, 16t, 17f
asymptomatic phakic fellow eyes with, 140
asymptomatic phakic non-fellow eyes with, 136–137
asymptotic nonphakic fellow eyes with, 141–142
atrophic holes, 18–19, 19f
detection of, 106–108, 107f
distribution of, 23–30, 24f, 24t, 25t
due to proliferative diabetic retinopathy, 21–22, 22f
extent of, 186–187
“fish mouthing” of, 168, 174
horseshoe tears, 16–17, 17f, 18f
iatrogenic, 173
identification and marking of, 208–209
inferior, 187
laser treatment of, 211, 211f
localization of, 155–156, 156f
macular holes, 22, 23f
new or missed retinal breaks following pneumatic retinopexy, 200
operculated, 17f, 18, 19f
postnecrotic holes, 20–21, 21f
primary, 16
and retinal hemorrhage, difference between, 108
rolled posterior edge of, 110f, 114f
secondary, 16
Retinal detachment:
bullous, 86, 86f
classification of, 32–33, 32t
diagnosis of, 97–126
discovery of, 3
epidemiology of, 30–32, 30f, 31f
etiology of, 3–4
evaluation and management of patient with, 75–95
with giant retinal tear, 241–242, 242f
history of, 3–7
lesions simulating, 122–125
with macular break, 239–240, 240f
with multiple retinal breaks:
at different distances from ora serrata,
237–238, 237f
at same distance from ora serrata,
236–237, 236f
natural history of, 36–37
with no apparent retinal break, 242–243, 243f
nonrhegmatogenous. See
Nonrhegmatogenous retinal detachment
with outer-layer break in retinoschisis, 243–244, 244f
pathogenesis of, 9–39
pathology of, 33–36
with peripheral break and macular hole, 239, 239f
prevention of. See Prevention of retinal detachment
with proliferative vitreoretinopathy, 244–245, 245f
recurrent, 92–94, 93f, 179, 188
repair, 4–5
with retinal dialysis, 241, 241f
rhegmatogenous. See Rhegmatogenous retinal detachment
selection of surgery for, 229–248
serous. See Serous retinal detachment
surgery. See also Pneumatic retinopexy;
Scleral buckling; Vitrectomy
external disease and, 89
preparation for:
office procedures, 91
operating room procedures, 90–91
patient counseling, 85–86
techniques for, 233–245
algorithm, 245–247, 246f
tractional. See Tractional retinal detachment
types, 9–10
uncomplicated. See Uncomplicated retinal detachment
Retinal dialysis, 19–20
inferior, 20f
with vitreous adherent to anterior edge, 16, 17f, 20, 20f
with vitreous adherent to posterior edge, 17f
Retinal erosion, 102f, 104
Retinal hemorrhage and retinal breaks,
difference between, 108
Retinal holes, 145
atrophic, 17–19, 19f
macular, 21, 22f
postnecrotic, 20, 21f
symptomatic atrophic, 132
Retinal incarceration, 173
Retinal whitening, 102f, 103–104, 105f
white-without-pressure, 102f, 104, 125
white-with-pressure, 102f, 104, 105f, 125
Retina Society Terminology Committee, 108, 109
Retinectomy, in proliferative vitreoretinopathy, 221–222, 222f
Retinitis, 20, 21f, 120
Retinochoroidal adhesion, 98
Retinoschisis, 17f, 18, 122–123
degenerative, 25–27, 26f
asymptomatic phakic fellow eyes with, 139–140
asymptomatic phakic non-fellow eyes with, 136
retinal detachment with outer-layer break in, 243–244, 244f
senile, 42
Retinotomy, in proliferative vitreoretinopathy, 221–222, 222f
Rhegmatogenous retinal detachment, 9, 73. See also Retinal detachment
diagnostic features of, 113t
pathogenesis of, 10–15, 150
liquid currents, 15
posterior vitreous detachment, 10, 12–14, 13f
vitreoretinal traction, 14–15, 14f
vitreous liquefaction, 11–12, 11f
risk factors for, 130–131, 130t
with scleral buckling. See also Scleral buckling
and serous retinal detachment, difference between, 112
and tractional retinal detachment, difference between, 116–117
Sausage sign, 195
Schisis detachment, 122–123. See also Retinoschisis
type 1, 122
type 2, 122, 123f
Schwartz syndrome, 80, 89
Scleral abscess, 175
Scleral buckling, 5, 89, 90, 91, 149–180, 188, 231–232
accessory techniques, 169–171
balanced salt solution, intravitreal injection of, 170
gas–fluid exchange, 171
intravitreal gas injection, 170–171
postoperative laser photocoagulation, 171
adjustment of, 168–169
advantages of, 232
anatomical and physiological effects of, 150–151
complications, intraoperative:
corneal complications, 172
of draining subretinal fluid, 173–174
“fish mouthing” of retinal breaks, 174
of intravitreal gas injections, 174
pupillary complications, 172
scleral perforation with suture needles, 172–173
complications, postoperative:
 altered refractive error, 179
choroidal detachment, 175–176
cystoid macular edema, 177
endophthalmitis, 175
epimacular proliferation, 177–178, 178f
implant extrusion, 176–177, 177f
intraocular pressure, increased, 174–175
later periocular infection, 176–177
proliferative vitreoretinopathy, 178–179
recurrent retinal detachment, 179
strabismus, 179–180
configuration, 151–153, 153f
disadvantages of, 232
incisions, closure of, 171–172
indications and contraindications, 230t
intrascleral buckles, 164, 164f
materials for, 152f, 159–163
encircling episcleral buckles, 160–163, 161f, 162f, 163f
segmental episcleral buckles, 159–160, 160f
operation, 153
conjunctival incision, 154–155
draping, 153
prepping, 153
rectus muscles, isolation of, 154–155, 154f, 155f
placement of, 211–212
and pneumatic retinopexy, comparison between, 201–203, 202t
for proliferative vitreoretinopathy, 219
recurrent or persistent detachment following, 92–94, 93f, 179, 188
retinal breaks, localization of, 155–156, 156f
subretinal fluid, management of, 164–168
central retinal artery, perfusion of, 165
drainage technique, 164, 166–168, 167f, 169f
non-drainage technique, 165
thermal treatment with, 156–159
thin sclera, 163
vitrectomy with:
  - indications and contraindications, 230t
Scleral cracks, 154
Scleral dehiscences, 154
Scleral depression, 46, 62, 121
  - axis of depression, moving, 66
  - discomfort, causes of, 66–67
Sclerotic depressor:
  - choice of, 63–64, 65f
  - rolling, 68–70, 69f, 70f, 71f
  - far periphery, examining, 68
  - in horizontal meridia, 67–68, 67f, 68f
  - initiating, 64–66, 65f
  - nasally, 68
  - in operating room, 72–73, 73f
Scleral perforation with suture needles, 172–173
Sclerotomies, closure of, 212
Scopolamine, 47, 92
Segmental episcleral buckles, 159–160, 160f
Segmentation technique, of vitrectomy, 215–216
Senile retinoschisis, 44. See also Retinoschisis
Serous retinal detachment. See also Retinal detachment
  - choroidal tumors with, 119, 119f
  - congenital causes of, 121
  - inflammatory, 119–120, 120f
  - macular lesions with, 121
  - optic pit with, 121f
  - and rhegmatogenous retinal detachment, difference between, 116, 118–119
  - vascular lesions with, 121
Slit lamp biomicroscope, 43, 46, 61, 83f, 91
Small-pupil, examination or treatment through:
  - using indirect laser, 62
  - using indirect ophthalmoscope, 62
  - using indirect visualization systems, 61
Small-pupil indirect ophthalmoscope, 47, 48f, 88
Snellen test, 79
Snowflakes, 17
Staphylomas, radial, 154, 155f
Star fold, 33, 34f, 109, 114f, 218
Steamroller technique, 186, 196, 197f
Stereopsis, 45, 106
Steroids, 177
Stickler syndrome, 134, 223
Strabismus, 179–180
Striae ciliares, 99
Subclinical retinal detachments, 135, 135f. See also Retinal detachment
Subretinal fibrosis, 109, 109, 115f
  - with tight peripapillary colarette, 116f
Subretinal fluid:
  - chronic persistent, 94
  - internal drainage of, 210, 210f
  - management of, 164–168
    - central retinal artery, perfusion of, 165
    - drainage technique, 164, 166–168, 167f
    - non-drainage technique, 165
Subretinal gas, 200, 201f
Sulfur hexafluoride (SF₆), 170, 181, 183, 184, 211
Symptomatic atrophic retinal holes, 132. See also Retinal holes
Symptomatic eyes, 131–132
  - breaks unassociated with persistent vitreoretinal traction:
    - precursors, 132
    - symptomatic atrophic retinal holes, 132
    - symptomatic operculated retinal tears, 132
  - tears with persistent vitreoretinal traction:
    - symptomatic horseshoe-shaped tears, 131
    - symptomatic round tears, 131–132
Symptomatic horseshoe-shaped tears, 131, 145
Symptomatic operculated retinal tears, 132
Symptomatic round tears, 131–132
Synchisis senilis, 10
Systemic diseases, 78
Tetracaine, 50
Thin sclera, 163, 189
Three-mirror lens, 81f, 82f
Tonometry, 80
Total retinal detachment. See also Retinal detachment
  - with no apparent retinal break, 242–243, 243f
  - with one retinal break, 235–236, 235f
Toxoplasmosis, 119
Trabecular meshwork disease, 159
Trabeculotomy, 196
Trabeculotomy. See also Trabeculotomy
  - with no apparent retinal break, 242–243
  - with one retinal break, 235–236
  - causes of, 122
  - diagnostic features of, 118t
  - and rhegmatogenous retinal detachment, difference between, 121–122
“Trap door” buckling procedure, 163, 164f
Trauma, 11, 20, 78, 223
Tropicamide, 49

Ultrasonography, 77, 83–84, 84f, 85f, 89, 119
Uncomplicated retinal detachment. See also Retinal detachment
indicators and contraindications, 230t
variables associated with, 231f
Untreated detachment, natural history of, 36–37
U-shaped tears. See Horseshoe tears
Uveitis, 37, 80, 89–90
peripheral, 44

Visual acuity, 79, 80, 86, 87, 177
Visual field defects, 77
Vitrectomy, 5, 6, 88, 89, 90, 91, 122, 205–228
air infusion, 210–211, 210f
complications of, 226–227
delamination technique of, 215, 215f
draping, 206
en bloc technique of, 215, 216f
giant retinal tear with edges folding, 223–226, 223f, 224f, 225f
goals of, 205–206
indicators and contraindications, 230t
opening incisions, 206–207
for PDR and retinal detachment, 214, 214f
perfluorocarbon liquids, installation of, 208, 209f
prepping, 206
for primary retinal detachment:
advantages of, 232
disadvantages of, 232
for PVR and retinal detachment, 219–221, 220f
results of, 226
retinal breaks:
identification and marking of, 208–209
laser treatment of, 211, 211f
with scleral buckling, 211–212

indicators and contraindications, 230t
segmentation technique of, 215–216
sclerotomies and peritomies, closure of, 212
subretinal fluid, internal drainage of, 210, 210f
vitreous gel, removal of, 207–208, 207f, 208f
Vitreoretinal degeneration, 152, 153
Vitreoretinal traction, 14–15, 14f, 16, 18, 28, 122, 131, 150, 152
precursors of retinal breaks and, 132
symptomatic atrophic retinal holes and, 132
symptomatic horseshoe-shaped tears with, 131
symptomatic operculated retinal tears with, 132
symptomatic round tears with, 131–132
Vitreous cortex, 10, 27, 82
Vitreous gel, removal of, 207–208, 207f, 208f
Vitreous hemorrhage, 14, 15, 76, 77, 89, 127, 171, 181, 199
Vitreous infusion suction cutter (VISC), 6
Vitreous liquefaction, 10–11, 12, 12f, 130, 134
Vitreous opacities, 89, 125
Vitreovascular attachments, 15
Vogt-Koyanagi-Harada syndrome, 115, 116f
von Hippel's disease, 120f, 121
Warfarin, 90, 91
Weiss’ ring, 12, 13f, 77
White-without-pressure, 102f, 104, 125. See also Retinal whitening
White-with-pressure, 102f, 104, 105f, 125, 140, 142. See also Retinal whitening
YAG laser, 11, 30, 78, 88, 133, 140. See also Laser
Zonular traction tufts, 24, 100