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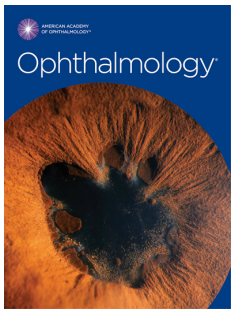

Posterior Vitreous Detachment, Retinal Breaks, and Lattice Degeneration Preferred Practice Pattern®

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Posterior Vitreous Detachment, Retinal Breaks, and Lattice Degeneration Preferred Practice Pattern®

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RETINA/VITREOUS PREFERRED PRACTICE PATTERN® DEVELOPMENT PROCESS AND PARTICIPANTS

The **Retina/Vitreous Preferred Practice Pattern® Panel** members wrote the Posterior Vitreous Detachment, Retinal Breaks, and Lattice Degeneration Preferred Practice Pattern® (PPP) guidelines. The PPP Panel members discussed and reviewed successive drafts of the document, meeting in person twice and conducting other review by e-mail discussion, to develop a consensus over the final version of the document.

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We thank our partners, the Cochrane Eyes and Vision US Satellite (CEV@US), for identifying reliable systematic reviews that we cite and discuss in support of the PPP recommendations.

The Preferred Practice Patterns Committee members reviewed and discussed the document during a meeting in June 2019. The document was edited in response to the discussion and comments.

Preferred Practice Patterns Committee 2019
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The Posterior Vitreous Detachment, Retinal Breaks, and Lattice Degeneration PPP was then sent for review to additional internal and external groups and individuals in July 2019. All those returning comments were required to provide disclosure of relevant relationships with industry to have their comments considered (indicated with an asterisk below). Members of the Retina/Vitreous Preferred Practice Pattern Panel reviewed and discussed these comments and determined revisions to the document.

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FINANCIAL DISCLOSURES

In compliance with the Council of Medical Specialty Societies' Code for Interactions with Companies (available at www.cmss.org/codeforinteractions.aspx), relevant relationships with industry are listed. The Academy has Relationship with Industry Procedures to comply with the Code (available at http://one.aao.org/CE/PracticeGuidelines/PPP.aspx). A majority (88%) of the members of the Retina/Vitreous Preferred Practice Pattern Panel 2018–2019 had no financial relationship to disclose.

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The disclosures of relevant relationships to industry of other reviewers of the document from January to October 2019 are available online at www.aao.org/ppp.

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OBJECTIVES OF PREFERRED PRACTICE PATTERN® GUIDELINES

As a service to its members and the public, the American Academy of Ophthalmology has developed a series of Preferred Practice Pattern® guidelines that **identify characteristics and components of quality eye care.** Appendix 1 describes the core criteria of quality eye care.

The Preferred Practice Pattern® guidelines are based on the best available scientific data as interpreted by panels of knowledgeable health professionals. In some instances, such as when results of carefully conducted clinical trials are available, the data are particularly persuasive and provide clear guidance. In other instances, the panels have to rely on their collective judgment and evaluation of available evidence.

These documents provide guidance for the pattern of practice, not for the care of a particular individual. While they should generally meet the needs of most patients, they cannot possibly best meet the needs of all patients. Adherence to these PPPs will not ensure a successful outcome in every situation. These practice patterns should not be deemed inclusive of all proper methods of care or exclusive of other methods of care reasonably directed at obtaining the best results. It may be necessary to approach different patients' needs in different ways. The physician must make the ultimate judgment about the propriety of the care of a particular patient in light of all of the circumstances presented by that patient. The American Academy of Ophthalmology is available to assist members in resolving ethical dilemmas that arise in the course of ophthalmic practice.

Preferred Practice Pattern® guidelines are not medical standards to be adhered to in all individual situations. The Academy specifically disclaims any and all liability for injury or other damages of any kind, from negligence or otherwise, for any and all claims that may arise out of the use of any recommendations or other information contained herein.

References to certain drugs, instruments, and other products are made for illustrative purposes only and are not intended to constitute an endorsement of such. Such material may include information on applications that are not considered community standard, that reflect indications not included in approved U.S. Food and Drug Administration (FDA) labeling, or that are approved for use only in restricted research settings. The FDA has stated that it is the responsibility of the physician to determine the FDA status of each drug or device he or she wishes to use, and to use them with appropriate patient consent in compliance with applicable law.

Innovation in medicine is essential to ensure the future health of the American public, and the Academy encourages the development of new diagnostic and therapeutic methods that will improve eye care. It is essential to recognize that true medical excellence is achieved only when the patients' needs are the foremost consideration.

All Preferred Practice Pattern® guidelines are reviewed by their parent panel annually or earlier if developments warrant and updated accordingly. To ensure that all PPPs are current, each is valid for 5 years from the approved by date unless superseded by a revision. Preferred Practice Pattern guidelines are funded by the Academy without commercial support. Authors and reviewers of PPPs are volunteers and do not receive any financial compensation for their contributions to the documents. The PPPs are externally reviewed by experts and stakeholders, including consumer representatives, before publication. The PPPs are developed in compliance with the Council of Medical Specialty Societies' Code for Interactions with Companies. The Academy has Relationship with Industry Procedures (available at www.aao.org/about-preferred-practice-patterns) to comply with the Code.

Appendix 2 contains the International Statistical Classification of Diseases and Related Health Problems (ICD) codes for the disease entities that this PPP covers. The intended users of the Posterior Vitreous Detachment, Retinal Breaks, and Lattice Degeneration PPP are ophthalmologists.

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METHODS AND KEY TO RATINGS

Preferred Practice Pattern® guidelines should be clinically relevant and specific enough to provide useful information to practitioners. Where evidence exists to support a recommendation for care, the recommendation should be given an explicit rating that shows the strength of evidence. To accomplish these aims, methods from the Scottish Intercollegiate Guideline Network[1] (SIGN) and the Grading of Recommendations Assessment, Development and Evaluation[2] (GRADE) group are used. GRADE is a systematic approach to grading the strength of the total body of evidence that is available to support recommendations on a specific clinical management issue. Organizations that have adopted GRADE include SIGN, the World Health Organization, the Agency for Healthcare Research and Policy, and the American College of Physicians.[3]

- All studies used to form a recommendation for care are graded for strength of evidence individually, and that grade is listed with the study citation.
- To rate individual studies, a scale based on SIGN[1] is used. The definitions and levels of evidence to rate individual studies are as follows:

I++	High-quality meta-analyses, systematic reviews of randomized controlled trials (RCTs), or RCTs with a very low risk of bias
I+	Well-conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias
I-	Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias
II++	High-quality systematic reviews of case-control or cohort studies High-quality case-control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal
II+	Well-conducted case-control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal
II-	Case-control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal
III	Nonanalytic studies (e.g., case reports, case series)

- Recommendations for care are formed based on the body of the evidence. The body of evidence quality ratings are defined by GRADE[2] as follows:

Good quality	Further research is very unlikely to change our confidence in the estimate of effect
Moderate quality	Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate
Insufficient quality	Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate Any estimate of effect is very uncertain

- Key recommendations for care are defined by GRADE[2] as follows:

Strong recommendation	Used when the desirable effects of an intervention clearly outweigh the undesirable effects or clearly do not
Discretionary recommendation	Used when the trade-offs are less certain—either because of low-quality evidence or because evidence suggests that desirable and undesirable effects are closely balanced

- The Highlighted Findings and Recommendations for Care section lists points determined by the PPP Panel to be of particular importance to vision and quality of life outcomes.
- All recommendations for care in this PPP were rated using the system described above. Ratings are embedded throughout the PPP main text in italics.
- Literature searches to update the PPP were undertaken in April 2018 and June 2019 in PubMed and the Cochrane Library. Complete details of the literature searches are available online at www.aao.org/ppp.

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HIGHLIGHTED FINDINGS AND RECOMMENDATIONS FOR CARE

Acute horseshoe retinal tears and traumatic breaks usually require treatment.

Asymptomatic atrophic or operculated retinal breaks rarely need treatment. More generally, an eye that has atrophic round holes within lattice lesions, has minimal subretinal fluid without progression, or lacks evidence of posterior vitreous detachment (PVD) does not require treatment.

An early diagnosis of a retinal detachment is important because the rate of successful retinal reattachment is higher and the visual results are better when repaired early, especially before the rhegmatogenous retinal detachment (RRD) involves the macula.

Lattice degeneration is present in 6% to 8% of the population and increases the risk of retinal detachment.

Patients presenting with an acute PVD and no retinal breaks have a small chance (~2%) of developing retinal breaks in the weeks that follow. Selected patients, particularly those with any degree of vitreous pigment, vitreous or retinal hemorrhage, or visible vitreoretinal traction, should be asked to return for a second examination promptly if they have new symptoms or within 6 weeks following the onset of PVD symptoms.

Between 5% and 14% of patients found to have an initial retinal break will develop additional breaks during long-term follow-up. Cataract surgery is a risk factor for new retinal breaks.

Treatment of peripheral horseshoe tears should extend to the ora serrata if the tear cannot be surrounded using laser or cryotherapy. The most common cause of failure is inadequate treatment, particularly along the anterior border (where visualization is more difficult).

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INTRODUCTION

DISEASE DEFINITION

Posterior vitreous detachment (PVD) is a separation of the posterior vitreous cortex from the internal limiting membrane of the retina.[4] (See Glossary.) This separation may be complete or partial. Vitreous traction at sites of significant vitreoretinal adhesion is responsible for most retinal breaks that lead to retinal detachment. Retinal breaks are defined as full-thickness defects in the retina. Lattice degeneration is a vitreoretinal degenerative process that predisposes to retinal tears and detachment. It is a peripheral vitreoretinal condition characterized by retinal thinning, overlying vitreous liquefaction, and firm vitreoretinal adhesions at the margins of thinning. Most lattice degenerations are ovoid, with the long axes of lattice running parallel to the ora serrata. Perivascular lattice occurs radially and is typically found adjacent to the retinal vessels. Radial lattice is associated with a much higher risk of retinal detachment than circumferential lattice. Round holes occur frequently within areas of lattice degeneration. Vitreomacular traction (VMT) may develop when the vitreous partially separates from the macula, potentially leading to mechanical distortion of the macula that may correspond to visual symptoms.[4] (See Glossary.)

PATIENT POPULATION

Individuals may present with symptoms or signs suggestive of PVD, retinal breaks, vitreous hemorrhage, retinal detachment, or VMT. A PVD typically occurs between the ages of 45 and 65 in the general population; however, the posterior vitreous may detach earlier in trauma and myopia.[5] Other individuals may not be symptomatic and, based on clinical examination findings, may have an increased risk of retinal detachment as the vitreous separates.

CLINICAL OBJECTIVES

- Identify patients at risk of developing a rhegmatogenous retinal detachment (RRD)
- Examine symptomatic patients who have an acute PVD to detect and treat associated retinal breaks or tears
- Recognize the evolution of retinal breaks and lattice degeneration
- Manage patients at high risk of developing retinal detachment
- Educate high-risk patients about symptoms of PVD, retinal breaks, and retinal detachments as well as the need for periodic follow-up
- Discuss treatment options available for VMT (See Idiopathic Epiretinal Membrane and Vitreomacular Traction PPP)[6]
- Recognize the potential side effects of treatment of VMT (See Idiopathic Epiretinal Membrane and Vitreomacular Traction PPP)[6]

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BACKGROUND

POSTERIOR VITREOUS DETACHMENT

Population-based studies that evaluate incidence and prevalence of PVD are difficult to conduct owing to the lack of definite clinical signs and unreliable clinical tests. A PVD typically occurs between the ages of 45 and 65 in the general population with earlier onset in men than women; however, the posterior vitreous may detach earlier in trauma and myopia, or be precipitated by ophthalmic surgical procedures.[5,7-9] Posterior vitreous detachment leads to vitreous traction at the vitreous base and in areas of lattice degeneration, and thereby, secondarily, is thought to cause most symptomatic retinal breaks that may lead to an RRD. The symptoms of a PVD include light flashes (photopsias) and floaters (myodesopias), and patients with such symptoms are at a higher risk for retinal detachment.[10-14] The stages of a PVD are described in Table 1.[4] Patients typically report the light flashes characteristic of a PVD as being most noticeable in the dark. Such photopsias are likely the result of vitreous traction on the retina as the vitreous separates from the posterior retina toward the vitreous base. The floaters may be due to blood from a torn or avulsed retinal vessel, condensations of vitreous collagen, or the epipapillary glial tissue (Weiss ring) that is torn from the optic nerve head and area adjacent to the optic nerve head. Between 8% and 22% of patients with acute PVD symptoms have a retinal tear at the time of the initial examination.[15-18] There is a direct correlation between the amount of vitreous hemorrhage and the likelihood of a retinal tear.[19] Patients with an acute PVD who have no reported retinal breaks on presentation have a 2% to 5% chance of experiencing a detected (missed or new) break in the weeks that follow.[13,16,20]

TABLE 1 STAGES* OF POSTERIOR VITREOUS DETACHMENT

Stage 1	Perifoveal separation with adhesion of vitreous to the fovea
Stage 2	Complete separation of vitreous from the macula
Stage 3	Extensive vitreous separation with adhesion of vitreous to the disc
Stage 4	Complete posterior vitreous detachment

NOTE: These stages can be studied with optical coherence tomography.[4,21]

* The proposed staging levels may not imply a linear, staged progression of a posterior vitreous detachment.

Approximately 80% of patients who presented without detected breaks, and then had breaks occur subsequently, had either pigmented cells or hemorrhage in the vitreous or retina at the initial evaluation, or new symptoms that prompted a return visit to the ophthalmologist.[16]

A spontaneous vitreous hemorrhage can be the presenting sign of PVD or may occur during the evolution of the PVD. Two-thirds of patients who present with associated vitreous hemorrhage were found to have at least one break. In this subgroup, one-third had more than one break and approximately 88% of the breaks occurred in the superior quadrants.[22]

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EVOLUTION OF RETINAL BREAKS AND LATTICE DEGENERATION

Precursors to RRDs are PVD, asymptomatic retinal breaks, symptomatic retinal breaks, lattice degeneration, and cystic and zonular traction retinal tufts. (See Glossary.) Because spontaneous retinal reattachment is rare, nearly all patients with a symptomatic clinical RRD will progressively lose vision unless the detachment is repaired. Currently, more than 95% of uncomplicated RRDs can be successfully repaired, although more than one procedure may be required.[23] The prophylactic treatment of high-risk breaks usually prevents RRD. An early diagnosis of an RRD is also important because the rate of successful reattachment is higher and the visual results are better when repaired early and especially before the RRD involves the macula.[15,17] The goal of RRD treatment is to allow patients to maintain their abilities to read, work, drive, care for themselves, and maintain their quality of life.[18]

Asymptomatic Retinal Breaks

Asymptomatic operculated holes and atrophic round holes rarely lead to retinal detachment. Byer followed 46 asymptomatic eyes with operculated retinal breaks over an average of 11 years.[24] Davis followed 28 eyes for up to 5 years in subjects where 80% of the fellow eyes had a retinal detachment.[25,26] All combined, none of the 74 eyes from these studies progressed to retinal detachment during the follow-up period.

Eyes with signs and symptoms of acute PVD may have atrophic retinal breaks with clinical features, suggesting that they are unrelated to the acute vitreoretinal traction from the PVD. Such breaks are considered to be pre-existing rather than symptomatic. Treatment may be considered for these breaks in certain situations, although the literature provides little guidance.[26] Randomized clinical trials are not available; therefore, there is limited evidence to support prophylactic therapy.[26]

Approximately 5% of eyes with asymptomatic horseshoe tears progress to retinal detachment.[24,27,28] Horseshoe tears discovered in asymptomatic fellow eyes are less likely than symptomatic horseshoe tears to lead to clinical retinal detachment (See Glossary). Patients should be encouraged to have follow-up. (See Follow-up Evaluation under Surgical Management.)

Symptomatic Retinal Breaks

A symptomatic retinal break is defined as a break caused by vitreoretinal traction in a patient with a new PVD associated with new-onset flashes and/or floaters. At least half of untreated symptomatic retinal breaks with persistent vitreoretinal traction (horseshoe or flap tears) will lead to a clinical retinal detachment unless treatment is applied.[25,29,30] (See Glossary.) Treatment by prompt creation of a chorioretinal adhesion around these symptomatic tears reduces the risk of retinal detachment to less than 5%.[29-34] Traumatic dialyses and tears along the vitreous base are managed similarly to symptomatic tears. Symptomatic operculated breaks usually do not

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progress to a clinical retinal detachment unless the vitreous remains adherent to the retina surrounding the break.[25,30]

Lattice Degeneration

Generally, atrophic round holes within lattice lesions that are accompanied by minimal subretinal fluid and no PVD do not require treatment. However, lattice degeneration is a risk factor for developing an RRD either from round holes without PVD or tractional-related holes associated with PVD. Small asymptomatic peripheral retinal detachments occurring secondary to retinal holes in areas of lattice degeneration are termed subclinical detachments (see Glossary).[35,36] Although these can enlarge and progress to clinical retinal detachments, they have been shown by observation alone to have a low likelihood of progression in most patients.[35] Prophylactic or interventional treatment should be considered when the detachments are documented to become symptomatic, increase in size, or show other signs of progression.[27,37]

One analysis studied 423 eyes with lattice degeneration in 276 patients over a period averaging nearly 11 years.[37] Of these, 150 eyes (35%) had atrophic holes in lattice, and 10 of these 150 eyes had subretinal fluid extending more than 1 disc diameter from the break (subclinical retinal detachment). Six other eyes developed new subclinical retinal detachments during follow-up. Clinical retinal detachments developed in 3 of the 423 eyes. Two were due to round retinal holes in lattice lesions of patients in their mid-20s and one was due to a symptomatic tractional tear. These data indicate that patients with lattice degeneration with or without round holes are at a *very low risk* for progression to clinical retinal detachment without a previous RRD in the fellow eye.

More commonly, RRD occurs in eyes with lattice degeneration when a PVD induces a horseshoe tear. Such tears should be treated using either laser demarcation or cryotherapy.[27,37]

INCIDENCE OF RHEGMATOGENOUS RETINAL DETACHMENT

The annual incidence of RRD is approximately 10 to 18 per 100,000 persons.[38-40] Of these, 20% to 40% have had cataract surgery and 10% have had ocular trauma.[23,41,42] In a recent study from the Netherlands, the annual RRD incidence was 18 per 100,000 people (95% CI, 11–19), with a peak incidence of 53 per 100,000 people (95% CI, 29–57) between 55 and 59 years of age. The rate of bilateral RRD was 1.7%. Prior cataract surgery was reported in 34% of RRD eyes.[40]

RISK FACTORS FOR RHEGMATOGENOUS RETINAL DETACHMENT

Aside from retinal breaks, risk factors for RRD include myopia, lattice degeneration, cataract or other intraocular surgery, neodymium yttrium-aluminum-garnet (Nd:YAG) laser surgery, trauma, a history of RRD in the other eye, certain genetic disorders such as Stickler syndrome, or family history of retinal detachments in a first-degree relative. Combinations of these factors may increase the risk.

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Myopia

More than half of nontraumatic RRD occurs in myopic eyes.[43] As axial length increases, the risk of RRD increases proportionately. One study found that individuals with low myopia (1–3 diopters) have a fourfold risk of RRD,[43] and higher levels of myopia have higher risks compared with nonmyopic individuals.[43,44]

Lattice Degeneration

Lattice degeneration is present in 6% to 8% of the population and increases the risk of retinal detachment.[37,45] Approximately 20% to 30% of patients with RRD have lattice degeneration.[37] Perivascular or radial lattice is associated with a higher risk of retinal tear or detachment formation.[37] Perivascular lattice it is also frequently seen in Stickler syndrome.[46]

Cataract Surgery

The overall risk of RRD after cataract surgery is approximately 1%.[47-50] The following conditions have been reported to increase the risk of RRD after cataract surgery: axial myopia, pre-existing vitreoretinal disease, male gender, younger age, vitreous prolapse into the anterior chamber, vitreous loss (ruptured posterior capsule/zonules), and spontaneous extension of the capsulotomy at the time of surgery.[51,52] One study suggests that in the absence of a posterior capsular tear at the time of cataract surgery, subsequent Nd:YAG laser capsulotomy may not increase the risk of retinal detachment.[53] Other studies suggest that Nd:YAG laser capsulotomy is associated with a fourfold increase in the risk of RRD, especially in myopic patients.[41,42,54-61]

Outside of complications at the time of surgery, risk of RRD after cataract surgery usually occurs 1 to 2 years later. A 5-year study using B-scan ultrasonography reported that it was the postoperative onset of a PVD that was the major risk factor for RRD (not the presence or absence of lattice) after cataract surgery and that the majority of eyes after cataract surgery that did not have a pre-existing PVD developed one at a median of 7 months after surgery. Consequently, one can extrapolate that it is the absence of a PVD (in higher risk eyes such as those with myopia and lattice) at the time of cataract surgery that is the major risk of RRD later.[60]

Trauma

Patients with blunt or penetrating ocular injuries that have altered the structure of the vitreous or retina are at increased risk of RRD.[62] Vitreoretinal interface changes caused by trauma may be detected at the time of injury or even many years later.

Rhegmatogenous Retinal Detachment in the Fellow Eye

Patients with a history of nontraumatic detachment in one eye have a 10% increased risk of developing RRD in the fellow eye, since pathologic vitreoretinal changes are frequently bilateral.[27,39,63-65] The fellow eye in a patient with pseudophakic retinal detachment is also at higher risk of developing a retinal detachment, whether the fellow eye is phakic or

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pseudophakic. Phakic fellow eyes in patients with pseudophakic retinal detachment have a 7% risk of RRD, suggesting that the risk of developing RRD should not be attributed to cataract surgery alone.[66]

Other Risk Factors

Other risk factors that have been reported include prior retinopathy of prematurity[67] and Stickler syndrome.[68,69]

There are case reports of retinal detachment in patients who have had keratorefractive surgery; however, large studies have not shown an increased risk in patients when compared with eyes of a similar refractive error.[70,71] It remains possible that the risk of vitreoretinal pathology is different among particular keratorefractive techniques.[9] Retinal detachment following refractive lens exchange in patients with high myopia showed a cumulative increase from 2% to 8% over a 7-year incidence.[72] Phakic intraocular lenses have not been associated with increased risk of retinal detachment compared with other intraocular interventions in highly myopic patients.[71,73,74]

CARE PROCESS

PATIENT OUTCOME CRITERIA

For management and treatment for PVD and RRD, the following outcomes are important:

- Prevention of visual loss and functional impairment
- Maintenance of quality of life

DIAGNOSIS

The initial evaluation of a patient with risk factors for retinal detachment or symptoms of a PVD involves detection of vitreous pigment cells or debris and includes a thorough peripheral examination looking for retinal tears or holes. It also includes all aspects of the comprehensive adult medical eye evaluation,[75] with particular attention to those aspects relevant to PVD, retinal breaks, and lattice degeneration. The ophthalmologist should also consider other causes of vitreous cells or debris (e.g., uveitis, infection, inflammation, neoplasia).

History

A patient history should include the following elements:

- Symptoms of PVD[10-14]
- Family history of retinal detachment, genetic disorders (e.g., Stickler syndrome)[68,69]
- Prior eye trauma[62]
- Myopia[43,76]
- History of ocular surgery, including refractive lens exchange and cataract surgery[41,42,64,77-79]

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- History of YAG laser capsulotomy
- History of an intravitreal injection[80]

Ophthalmic Examination

The eye examination should include the following elements:

- Confrontation visual field examination
- Visual acuity testing
- Pupillary assessment for the presence of a relative afferent pupillary defect
- Examination of the vitreous for hemorrhage, detachment, and pigmented cells[10-14,16,81]
- Careful examination of the peripheral fundus using scleral depression[82] (see Table 3)

There are no symptoms that can reliably distinguish between a PVD with or without an associated retinal break; therefore, a peripheral retinal examination is required.[82] The preferred method of evaluating patients for peripheral vitreoretinal pathology is to use an indirect ophthalmoscope combined with scleral depression.[83] Many patients with retinal tears have blood and pigmented cells in the anterior vitreous. In fully dilated eyes, slit-lamp biomicroscopy with a mirrored contact lens or a condensing lens is an alternative method in fully dilated eyes instead of a scleral depressed indirect examination of the peripheral retina.

Diagnostic Tests

Optical coherence tomography may be helpful to evaluate and stage the PVD.[4,21,84] If media opacity or patient cooperation precludes an adequate examination of the peripheral retina, B-scan ultrasonography should be performed to search for retinal tears, RRD, mass lesions, or other causes of vitreous hemorrhage.[85] Bilateral patching and/or elevation of the head while sleeping may be used when attempting to clear the vitreous hemorrhage.[86] If no abnormalities are found, frequent follow-up examinations are recommended (i.e., every 1–2 weeks initially). Wide-field color photography can detect some peripheral retinal breaks but does not replace careful ophthalmoscopy and may be useful in patients not able to tolerate the exam.

Even if the vitreous hemorrhage is sufficiently dense to obscure the posterior pole, the peripheral retina frequently can be examined using indirect ophthalmoscopy and scleral depression. Patients who present with vitreous hemorrhage sufficient to obscure all retinal details and have a negative B-scan ultrasonographic evaluation should be followed closely. Often, patients are seen weekly until the vitreous hemorrhage resolves or until a thorough indirect ophthalmoscopic depressed peripheral exam can be done to rule out an underlying retinal tear. When a retinal tear is suspected, repeat ultrasonographic examination should be performed within 1 to 2 weeks of the initial evaluation. There is considerable variation in the reported sensitivity (44%–100%) of B-scan ultrasonography for detecting retinal tears in cases of PVD-associated fundus-obscuring vitreous hemorrhage.[85,87-89] Early vitrectomy (usually defined as within 7 days of presentation) for dense PVD-associated vitreous hemorrhage has been reported to have a low rate of complications and may be considered to reduce the risk of

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vision loss occurring secondary to macula-involving retinal detachment.[89-91] Prompt intervention is indicated if there is a tear seen on ultrasonography and the vitreous cavity precludes a view.

MANAGEMENT

Prevention

There are no effective methods of preventing the vitreous syneresis and liquefaction that lead to a PVD and possibly an RRD. If factors associated with an increased risk of retinal detachment are discovered during a routine eye examination in an asymptomatic patient, a careful peripheral fundus examination is recommended. Patients at high risk should also be educated about the symptoms of PVD and retinal detachment as well as about the value of periodic follow-up examinations.[14] Patients with retinal or vitreous hemorrhage have an increased risk of multiple retinal tears.[92] Moreover, a systematic review performed in 2012 found that there is also no strong evidence in the literature to support or refute the use of 360-degree laser intervention in the fellow eyes of patients with a unilateral giant retinal tear.[93]

Pharmacotherapy for the management of VMT has been developed. In a placebo-controlled trial of microplasmin (a precursor of ocriplasmin) to induce a PVD, intravitreal injection of 125 micrograms of microplasmin led to a moderate increase in the likelihood of induction and progression of PVD (10% vs. 31%).[84] Please refer to the Idiopathic ERM and VMT PPP for a detailed discussion.[6] The analysis showed that ocriplasmin was better than sham or placebo for inducing PVD, although adverse events were more common in the treated group. In addition, 20% still needed pars plana vitrectomy within 6 months.[94]

Pneumatic vitreolysis is also used to induce a PVD. In a meta-analysis, pneumatic vitreolysis approached similar release rates of pars plana vitrectomy and was more effective than ocriplasmin by day 28.[95] Complications of this procedure are typically related to PVD formation and include retinal tear, retinal detachment, epiretinal membrane, and lamellar or macular hole formation.[94,95]

Surgical Management

It is essential that clinical personnel be familiar with the symptoms of PVD and retinal detachment and that they recognize the need for urgent ophthalmologic evaluation of symptomatic patients.[14] Patients with symptoms of possible or suspected PVD or retinal detachment and related disorders should be examined as soon as is feasible by an ophthalmologist skilled in binocular indirect ophthalmoscopy and supplementary techniques. Patients with retinal breaks or detachments should be treated by an ophthalmologist with experience in the management of these conditions. A Cochrane systematic review found low to very low certainty evidence indicating little or no difference between pars plana vitrectomy and scleral buckling in anatomical and visual acuity outcomes.[96]

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Posterior vitreous detachment symptoms (e.g., flashes and floaters) usually diminish over time, sometimes requiring several months. Appropriate reassurance and precautions regarding the symptoms of retinal detachment should be given. However, some patients may be debilitated in the absence of tears or detachments in the retina. The impact of floaters or floater-related visual symptoms may have an adverse effect on a person's vision-related quality of life. Pars plana vitrectomy is an option if symptomatic floaters are still bothersome after several months. In fact, it has been documented using contrast sensitivity function, that patients with a PVD have a significant reduction in contrast sensitivity function.[97] Laser treatments and pharmacotherapies have been proposed to decrease these symptoms; however, such therapies currently lack sufficient evidence to support their use.[98] Pars plana vitrectomy has been used for removal of floaters, and improvement in contrast sensitivity function has been documented.[99] In a recent review of series that compared pars plana vitrectomy and Nd:YAG laser for floaters,[100] pars plana vitrectomy showed evidence of greater patient satisfaction compared with only moderate resolution of symptoms following the Nd:YAG laser procedure. Another study found YAG vitreolysis to yield greater improvement in symptoms than sham laser.[101]

The goal of treatment for retinal breaks is to create a firm chorioretinal adhesion in the attached retina immediately adjacent to and surrounding the retinal tear using cryotherapy or laser photocoagulation surgery to halt the progression of subretinal fluid from detaching the neurosensory retina.

Treatment of peripheral horseshoe tears should be extended to the ora serrata if the tear cannot be surrounded using laser or cryotherapy.[31,102,103] The most common cause of failure in treating horseshoe tears is failure to adequately treat the tear, particularly at the anterior border. Continued vitreous traction may extend the tear beyond the treated area and allow fluid to dissect through the subretinal space to cause a clinical retinal detachment.[31,102,103] Treatment of dialyses must extend over the entire length of the dialysis, reaching the ora serrata beyond each horn or end of the dialysis.

Sufficient evidence exists to warrant treating acute, symptomatic horseshoe tears.[25] There is insufficient evidence for management of other vitreoretinal abnormalities. A Cochrane systematic review found that in making the decision to treat other vitreoretinal abnormalities, including lattice degeneration and asymptomatic retinal breaks, that the risks that treatment would be unnecessary, ineffective, or harmful must be weighed against the possible benefit of reducing the rate of subsequent retinal detachment.[26] *(I+, Good quality, Strong recommendation)* Table 2 summarizes recommendations for management. A Cochrane systematic review in 2014 shows that no randomized controlled clinical trials have been performed to support treatment of asymptomatic retinal breaks of lattice degeneration.[26] There is no level 1 evidence to support the use of prophylactic laser to areas of lattice degeneration prior to anterior segment surgery.[63,65] A primary limitation of prophylactic therapy is that

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causative breaks leading to retinal detachment often occur during a PVD in areas that appear normal prior to the PVD.[65]

TABLE 2 MANAGEMENT RECOMMENDATIONS

Type of Lesion	Treatment*
Acute symptomatic horseshoe tears	Treat promptly[29-34]
Acute symptomatic operculated holes	Treatment may not be necessary
Acute symptomatic dialyses	Treat promptly
Traumatic retinal breaks	Usually treated
Asymptomatic horseshoe tears (without subclinical RD)	Consider treatment unless there are signs of chronicity[27]
Asymptomatic operculated tears	Treatment is rarely recommended
Asymptomatic atrophic round holes	Treatment is rarely recommended
Asymptomatic lattice degeneration without holes	Not treated unless PVD causes a horseshoe tear
Asymptomatic lattice degeneration with holes	Usually does not require treatment
Asymptomatic dialyses	No consensus on treatment and insufficient evidence to guide management
Eyes with atrophic holes or lattice degeneration where the fellow eye has had an RD	No consensus on treatment and insufficient evidence to guide management

PVD = posterior vitreous detachment; RD = retinal detachment

* There is insufficient evidence to recommend prophylaxis of asymptomatic retinal breaks for patients undergoing cataract surgery.

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The surgeon should inform the patient of the risks, benefits, and alternatives to surgery.[104,105] The treating surgeon is responsible for formulating a postoperative care plan and should inform the patient of these arrangements.[104,105]

Retinal detachments may occur in spite of appropriate therapy. Traction is an important component and may pull the tear from the treated area, especially when there are larger breaks or bridging retinal blood vessels. The laser- or cryotherapy-induced treatment adhesion (chorioretinal scar) may not be firm or complete for up to 1 month following treatment.[31,33,102] Furthermore, 10% to 16% of patients will develop additional breaks during long-term follow-up.[33,106,107] Pseudophakic patients are more likely to require retreatment or to develop new breaks.[33]

Complications of Treatment

Proliferation of the epiretinal membrane (ERM), or macular pucker, has been occasionally observed following treatment for a retinal break; however, a direct cause and effect relationship of treatment of a retinal break to ERM remains unclear, since an ERM may also occur spontaneously following the PVD. (See Glossary.) In one long-term follow-up study, the percentage of eyes that developed macular pucker after treatment of retinal breaks was no greater than the percentage of eyes observed to have macular pucker before treatment.[31] Therefore, the method of creating a chorioretinal adhesion may be unrelated to the incidence of postoperative macular pucker.[108]

Follow-up Evaluation

The guidelines in Table 3 are recommendations for the timing of re-evaluation in the absence of additional symptoms. Patients with new symptoms or a change in symptoms may require more frequent evaluation. Patients with no positive findings at the initial examination should be seen at the intervals recommended in the Comprehensive Adult Medical Eye Evaluation PPP.[75] All patients with risk factors should be advised to contact their ophthalmologist promptly if new symptoms such as flashes, floaters, peripheral visual field loss, or decreased visual acuity develop.[41,42,77,109]

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TABLE 3 RECOMMENDED GUIDELINES FOR FOLLOW-UP

Type of Lesion	Follow-up Interval
Symptomatic PVD with no retinal break	Depending on symptoms, risk factors, and clinical findings, patients may be followed within 2 months, then 6–12 months
Symptomatic PVD with no retinal break but with some vitreous or retinal hemorrhage	Depending on the severity of the retinal hemorrhage, 1–2 weeks For vitreous hemorrhage, weekly until resolved. Ultrasonography to check for retinal tears
Acute symptomatic horseshoe tears	1–2 weeks after treatment, then 4–6 weeks, then 3–6 months, then annually
Acute symptomatic operculated holes	2–4 weeks, then 1–3 months, then 6–12 months, then annually
Acute symptomatic dialyses	1–2 weeks after treatment, then 4–6 weeks, then 3–6 months, then annually
Traumatic retinal breaks	1–2 weeks after treatment, then 4–6 weeks, then 3–6 months, then annually
Asymptomatic horseshoe tears	1–4 weeks, then 2–4 months, then 6–12 months, then annually
Asymptomatic operculated holes	1–4 months, then 6–12 months, then annually
Asymptomatic atrophic round holes	1–2 years
Asymptomatic lattice degeneration without holes	Annually
Asymptomatic lattice degeneration with holes	Annually
Asymptomatic dialyses	• If untreated, 1–4 weeks, then 3 months, then 6 months, then every 6 months • If treated, 1–2 weeks after treatment, then 4–6 weeks, then 3–6 months, then annually
Eyes with atrophic holes, lattice degeneration, or asymptomatic horseshoe tears in patients who have had a retinal detachment in the fellow eye	Every 6–12 months

PVD = posterior vitreous detachment

Younger myopic patients who have lattice degeneration with holes need regular follow-up visits to monitor for subclinical retinal detachments that may slowly enlarge to become clinical retinal detachments. Treatment should be considered if the detachments progress in size.[27,37]

Patients presenting with an acute PVD and no retinal breaks have a small chance (approximately 2%) of developing retinal breaks in the weeks that follow.[13] Thus, selected patients, particularly those with any degree of vitreous pigment, vitreous or retinal hemorrhage, or visible vitreoretinal traction, should be asked to return for a second examination within 6 weeks following the onset of symptoms.[13,107]

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History at Follow-up Encounter

A patient history should identify changes in the following:

- Visual symptoms[10-14,81]
- Interval history of eye trauma, intraocular injection, or intraocular surgery[42,62]

Ophthalmic Examination

The eye examination should emphasize the following elements:

- Measurement of visual acuity
- Evaluation of the vitreous status, with attention to the presence of pigment, hemorrhage, or syneresis[10-14,16,81]
- Examination of the peripheral fundus using scleral depression[82] or a fundus contact or non–contact lens using the slit-lamp biomicroscope
- Wide-field photography maybe helpful but does not replace careful ophthalmoscopy
- Optical coherence tomography if VMT is present[4,21,84]
- B-scan ultrasonography when the media is opaque[85]

If the treatment for treated patients appears satisfactory at the first follow-up visit at 1 to 2 weeks, indirect ophthalmoscopy and scleral depression at 2 to 6 weeks will determine the adequacy of the chorioretinal scar, especially around the anterior boundary of the tear. If the tear and the accompanying subretinal fluid are not completely surrounded by the chorioretinal scar, additional treatment should be administered. At any postoperative visit, additional treatment should be considered if subretinal fluid has accumulated beyond the edge of treatment.[31,33]

Even when a patient has had adequate treatment, additional examinations are important. Between 5% and 14% of patients found to have an initial retinal break will develop additional breaks during long-term follow-up. These statistics appear to be similar regardless of how the initial breaks were treated.[33,106] New breaks may be particularly likely in eyes that have had cataract surgery.[33]

COUNSELING AND REFERRAL

All patients at increased risk of retinal detachment should be instructed to notify their ophthalmologist as soon as possible if they have a substantial change in symptoms, such as an increase in floaters, loss of visual field, or decrease in visual acuity.[41,42,77,109] If patients are familiar with the symptoms of retinal tears or detachment, they may be more likely to report promptly, thus improving the opportunity for successful treatment and subsequent visual results.[17] Patients who undergo refractive surgery to reduce myopia should be informed that they remain at risk of RRD despite reduction of their refractive error.

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SOCIOECONOMIC CONSIDERATIONS

Limited data exist on the socioeconomic impacts of PVD, retinal breaks, or lattice degeneration. However, research on the impact of the symptoms of these conditions (e.g., vitreous floaters) has suggested that vitreous symptoms may have an unfavorable effect on a patient's vision-related quality of life.[110,111] The modeled cost of evaluating a patient with PVD and treating associated pathology in the facility/hospital (nonfacility/Ambulatory Surgery Centers)-based setting was $65 to $190 ($25–$71) depending on whether a single or two-examination protocol was used. The cost per quality-adjusted life year (QALY) saved was $255 to $638/QALY ($100–$293/QALY). Treatment of a symptomatic horseshoe tear resulted in a net cost savings of $1,749 ($1,314) and improved utility, whereas treatment of an asymptomatic horseshoe tear resulted in $2,981/QALY ($1,436/QALY). Treatment of asymptomatic lattice degeneration in an eye in which the fellow eye had a history of RD resulted in $4,414/QALY ($2,187/QALY).[112]

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APPENDIX 1. QUALITY OF OPHTHALMIC CARE CORE CRITERIA

Providing quality care
is the physician's foremost ethical obligation, and is
the basis of public trust in physicians.
AMA Board of Trustees, 1986

Quality ophthalmic care is provided in a manner and with the skill that is consistent with the best interests of the patient. The discussion that follows characterizes the core elements of such care.

The ophthalmologist is first and foremost a physician. As such, the ophthalmologist demonstrates compassion and concern for the individual, and utilizes the science and art of medicine to help alleviate patient fear and suffering. The ophthalmologist strives to develop and maintain clinical skills at the highest feasible level, consistent with the needs of patients, through training and continuing education. The ophthalmologist evaluates those skills and medical knowledge in relation to the needs of the patient and responds accordingly. The ophthalmologist also ensures that needy patients receive necessary care directly or through referral to appropriate persons and facilities that will provide such care, and he or she supports activities that promote health and prevent disease and disability.

The ophthalmologist recognizes that disease places patients in a disadvantaged, dependent state. The ophthalmologist respects the dignity and integrity of his or her patients and does not exploit their vulnerability.

Quality ophthalmic care has the following optimal attributes, among others.

- The essence of quality care is a meaningful partnership relationship between patient and physician. The ophthalmologist strives to communicate effectively with his or her patients, listening carefully to their needs and concerns. In turn, the ophthalmologist educates his or her patients about the nature and prognosis of their condition and about proper and appropriate therapeutic modalities. This is to ensure their meaningful participation (appropriate to their unique physical, intellectual, and emotional state) in decisions affecting their management and care, to improve their motivation and compliance with the agreed plan of treatment, and to help alleviate their fears and concerns.
- The ophthalmologist uses his or her best judgment in choosing and timing appropriate diagnostic and therapeutic modalities as well as the frequency of evaluation and follow-up, with due regard to the urgency and nature of the patient's condition and unique needs and desires.
- The ophthalmologist carries out only those procedures for which he or she is adequately trained, experienced, and competent, or, when necessary, is assisted by someone who is, depending on the urgency of the problem and availability and accessibility of alternative providers.
- Patients are assured access to, and continuity of, needed and appropriate ophthalmic care, which can be described as follows.
  - The ophthalmologist treats patients with due regard to timeliness, appropriateness, and his or her own ability to provide such care.
  - The operating ophthalmologist makes adequate provision for appropriate pre- and postoperative patient care.
  - When the ophthalmologist is unavailable for his or her patient, he or she provides appropriate alternate ophthalmic care, with adequate mechanisms for informing patients of the existence of such care and procedures for obtaining it.
  - The ophthalmologist refers patients to other ophthalmologists and eye care providers based on the timeliness and appropriateness of such referral, the patient's needs, the competence and qualifications of the person to whom the referral is made, and access and availability.

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- The ophthalmologist seeks appropriate consultation with due regard to the nature of the ocular or other medical or surgical problem. Consultants are suggested for their skill, competence, and accessibility. They receive as complete and accurate an accounting of the problem as necessary to provide efficient and effective advice or intervention, and in turn they respond in an adequate and timely manner. The ophthalmologist maintains complete and accurate medical records.
- On appropriate request, the ophthalmologist provides a full and accurate rendering of the patient's records in his or her possession.
- The ophthalmologist reviews the results of consultations and laboratory tests in a timely and effective manner and takes appropriate actions.
- The ophthalmologist and those who assist in providing care identify themselves and their profession.
- For patients whose conditions fail to respond to treatment and for whom further treatment is unavailable, the ophthalmologist provides proper professional support, counseling, rehabilitative and social services, and referral as appropriate and accessible.

- Prior to therapeutic or invasive diagnostic procedures, the ophthalmologist becomes appropriately conversant with the patient's condition by collecting pertinent historical information and performing relevant preoperative examinations. Additionally, he or she enables the patient to reach a fully informed decision by providing an accurate and truthful explanation of the diagnosis; the nature, purpose, risks, benefits, and probability of success of the proposed treatment and of alternative treatment; and the risks and benefits of no treatment.
- The ophthalmologist adopts new technology (e.g., drugs, devices, surgical techniques) in judicious fashion, appropriate to the cost and potential benefit relative to existing alternatives and to its demonstrated safety and efficacy.
- The ophthalmologist enhances the quality of care he or she provides by periodically reviewing and assessing his or her personal performance in relation to established standards, and by revising or altering his or her practices and techniques appropriately.
- The ophthalmologist improves ophthalmic care by communicating to colleagues, through appropriate professional channels, knowledge gained through clinical research and practice. This includes alerting colleagues of instances of unusual or unexpected rates of complications and problems related to new drugs, devices, or procedures.
- The ophthalmologist provides care in suitably staffed and equipped facilities adequate to deal with potential ocular and systemic complications requiring immediate attention.
- The ophthalmologist also provides ophthalmic care in a manner that is cost effective without unacceptably compromising accepted standards of quality.

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APPENDIX 2. INTERNATIONAL STATISTICAL CLASSIFICATION OF DISEASES AND RELATED HEALTH PROBLEMS (ICD) CODES

Precursors to rhegmatogenous retinal detachment and related entities with the following ICD-9 and ICD-10 classifications (see Glossary):

	ICD-9 CM	ICD-10 CM
Rhegmatogenous retinal detachment:		
Break, unspecified	361.00	H33.00-
Break, giant	361.03	H33.03-
Break, multiple	361.02	H33.02-
Break, single	361.01	H33.01-
Vitreous detachment/degeneration	379.21	H43.81-
Retinal break without detachment:		
Retinal break, unspecified	361.30	H33.30-
Horseshoe tear	361.32	H33.31-
Multiple	361.33	H33.33-
Round hole	361.31	H33.32-
Multiple defects of retina without detachment	361.33	H33.33-
Horseshoe tear of retina without detachment	361.32	H33.31-
Operculated break without detachment	361.32	H33.31-
Round hole without detachment	361.31	H33.32-
Retinal dialysis	361.04	H33.04-
Lattice degeneration of the retina	362.63	H35.41-

ICD = International Classification of Diseases; CM = Clinical Modification used in the United States; (–) = 1, right eye; 2, left eye; 3, bilateral

Additional Information for ICD-10 Codes:

- Certain ICD-10 CM categories have applicable 7th characters. The applicable 7th character is required for all codes within the category, or as the notes in the Tabular List instruct. The 7th character must always be the 7th character in the data field. If a code that requires a 7th character is not 6 characters, a placeholder X must be used to fill in the empty characters.
- For bilateral sites, the final character of the codes in the ICD-10 CM indicates laterality. If no bilateral code is provided and the condition is bilateral, separate codes for both the left and right side should be assigned. Unspecified codes should be used only when there is no other code option available.
- When the diagnosis code specifies laterality, regardless of which digit it is found in (i.e., 4th digit, 5th digit, or 6th digit):
    - Right is always 1
    - Left is always 2
    - Bilateral is always 3

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GLOSSARY

Atrophic retinal breaks or holes: Full-thickness retinal defects, unrelated to vitreoretinal traction. These can occur within lattice lesions or in areas of the retina that appear otherwise normal.

Clinical retinal detachment: A retinal detachment that either impairs a portion of the visual field or extends more than 2 disc diameters posterior to the equator.

Cystic retinal tufts: Small congenital lesions of the peripheral retina. They are slightly elevated and usually whitish in color with variable surrounding pigmentation. They are firmly attached to the overlying vitreous cortex and are sometimes a cause of retinal tears following PVD.

Epiretinal membrane (ERM): See Macular pucker.

ERM: See Macular pucker.

Flap tear: A horseshoe tear.

Horseshoe tear: A retinal tear caused by vitreoretinal traction on the retina. The tear is horseshoe shaped owing to a flap of torn tissue that remains attached to the detached vitreous gel.

ICD-9: International Statistical Classification of Diseases and Related Health Problems, Ninth Edition.

ICD-10: International Statistical Classification of Diseases and Related Health Problems, Tenth Edition.

Lattice degeneration: A peripheral vitreoretinal lesion characterized by retinal thinning, overlying vitreous liquefaction, and firm vitreoretinal adhesions at its margins. Most lesions are ovoid with long axes parallel to the ora serrata. Round holes frequently occur within the lattice lesion unassociated with PVD. If horseshoe tears are present, they are seen at the development of PVD and usually are observed at the margins of lattice lesions.

Macular pucker: Distortion of the retina in the macular region due to proliferation and contraction of a fibrocellular membrane on the inner surface of the retina.

Operculated retinal tear or break: A defect in the retina caused by vitreoretinal traction at the site of the lesion. The traction pulls a circular or oval piece of retinal tissue (the operculum) free from the retinal surface. If this occurs during PVD, all traction in the vicinity of the retinal break is usually eliminated.

Posterior vitreous detachment (PVD): A separation of the posterior vitreous cortex from the internal surface of the retina. This usually occurs as an acute event after substantial age-related liquefaction in the vitreous gel; the separation usually extends rapidly to the posterior margin of the vitreous base in all quadrants. Adhesions between the vitreous cortex and retina or retinal blood vessels may cause retinal breaks and/or vessel rupture. Vitreous hemorrhage and/or localized intraretinal hemorrhage may accompany this event. Posterior vitreous detachment is diagnosed by slit-lamp biomicroscopy, which will usually show a prominent plane defining the posterior vitreous face. The presence of a glial annulus in the vitreous cavity (Weiss ring) is strong evidence of PVD.

PVD: See Posterior vitreous detachment.

Retinal breaks: Full-thickness defects in the retina. Those caused by vitreoretinal traction are usually called tears. Those that are round and unassociated with vitreoretinal traction are usually called holes.

Retinal dialysis: A specific type of crescentic peripheral retinal break at the ora serrata, usually associated with trauma.

Rhegmatogenous retinal detachment (RRD): A separation of the retina from the retinal pigment epithelium caused by fluid passing from the vitreous cavity into the subretinal space through a break in the retina (from Greek *rhegma*, "rent").

Round retinal hole: A round, full-thickness defect or break in the retina, unassociated with vitreoretinal traction.

RRD: See Rhegmatogenous retinal detachment.

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Stickler syndrome: The most common inherited vitreoretinal and systemic disorder associated with RRD. Ocular features include (1) high myopia; (2) retrolental, transvitreal, and epiretinal membranes and strands; (3) chorioretinal pigment alterations; (4) lattice degeneration, often with a perivascular component that extends posteriorly; and (5) various other abnormalities including glaucoma and cataract. Systemic features include a generalized skeletal dysplasia, often with a marfanoid habitus, flattened facies, high arched or cleft palate, hearing loss, and other extracranial skeletal anomalies, many of which can be very subtle. The inheritance pattern is autosomal dominant, and a gene defect has been related to COL2A1.

Subclinical retinal detachment: A retinal detachment that extends more than 1 disc diameter from the posterior edge of the retinal break, less than 2 disc diameters from the equator, and does not impair the field of vision.

Vitreoretinal adhesion (VMA): A firm attachment between the cortical vitreous and the inner surface of the retina. Condensed vitreous strands adhering to the retina may sometimes be visualized using biomicroscopy or indirect ophthalmoscopy and scleral depression. Traction of the vitreous on the retina during PVD may cause retinal breaks to occur at these sites.

Vitreomacular traction (VMT): Partial vitreous separation from the retina resulting in mechanical distortion of the macula.

VMA: See Vitreomacular adhesion

VMT: See Vitreomacular traction

Zonular traction retinal tufts: Small congenital lesions of the peripheral retina caused by thickened zonules that have been displaced posteriorly to the anterior retina.

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LITERATURE SEARCHES FOR THIS PPP

Literature searches of the PubMed and Cochrane databases were conducted in April 2018; the search strategies are provided at www.aao.org/ppp. Specific limited update searches were conducted after June 2019.

(Retinal Detachment/epidemiology[mh]) AND (rhegmatogenous retinal detachment[tiab])

(Retinal Detachment/etiology[MAJR:noexp] OR Retinal Perforations/etiology[MAJR:noexp] OR Vitreous Detachment/etiology[MAJR:noexp] OR Retinal Degeneration/etiology[MAJR:noexp]) AND (rhegmatogenous retinal detachment[tiab] OR posterior vitreous detachment[tiab] OR retinal break*[tiab] OR lattice degeneration[tiab])

(Retinal Detachment/diagnosis[MAJR:noexp] OR Retinal Perforations/diagnosis[MAJR:noexp] OR Vitreous Detachment/diagnosis[MAJR:noexp] OR Retinal Degeneration/diagnosis[MAJR:noexp]) AND (posterior vitreous detachment[tiab] OR retinal break*[tiab] OR lattice degeneration[tiab])

(Retinal Detachment[MAJR:noexp] OR Retinal Perforations[MAJR:noexp] OR Vitreous Detachment[MAJR:noexp] OR Retinal Degeneration[MAJR:noexp]) AND (Risk Factors[mh]) AND (rhegmatogenous retinal detachment[tiab] OR posterior vitreous detachment[tiab] OR retinal break*[tiab] OR lattice degeneration[tiab] OR cataract*[tiab] OR trauma*[tiab] OR injur*[tiab] OR fellow[tiab] OR retinopathy of prematurity[tiab] OR Stickler[tiab] OR keratorefractive[tiab] OR refractive lens exchange*[tiab] OR phakic intraocular lens*[tiab])

(Retinal Detachment/surgery[MAJR:noexp] OR Retinal Detachment/therapy[MAJR:noexp] OR Retinal Detachment/drug therapy[MAJR:noexp] OR Retinal Perforations/surgery[MAJR:noexp] OR Retinal Perforations/therapy[MAJR:noexp] OR Retinal Perforations/drug therapy[MAJR:noexp] OR Vitreous Detachment/surgery[MAJR:noexp] OR Vitreous Detachment/therapy [MAJR:noexp] OR Vitreous Detachment/drug therapy[MAJR:noexp] OR Retinal Degeneration/surgery[MAJR:noexp] OR Retinal Degeneration/therapy[MAJR:noexp] OR Retinal Degeneration/drug therapy[MAJR:noexp]) AND (posterior vitreous detachment[tiab] OR retinal break*[tiab] OR lattice degeneration[tiab])

(Retinal Detachment/surgery[MAJR:noexp] OR Retinal Detachment/therapy[MAJR:noexp] OR Retinal Detachment/drug therapy[MAJR:noexp] OR Retinal Perforations/surgery[MAJR:noexp] OR Retinal Perforations/therapy[MAJR:noexp] OR Retinal Perforations/drug therapy[MAJR:noexp] OR Vitreous Detachment/surgery[MAJR:noexp] OR Vitreous Detachment/therapy [MAJR:noexp] OR Vitreous Detachment/drug therapy[MAJR:noexp] OR Retinal Degeneration/surgery[MAJR:noexp] OR Retinal Degeneration/therapy[MAJR:noexp] OR Retinal Degeneration/drug therapy[MAJR:noexp]) AND (posterior vitreous detachment[tiab] OR retinal break*[tiab] OR lattice degeneration[tiab])

(Retinal Detachment [MAJR:noexp] OR Retinal Perforations [MAJR:noexp] OR Vitreous Detachment [MAJR:noexp] OR Retinal Degeneration[MAJR:noexp]) AND (posterior vitreous detachment[tiab] OR retinal break*[tiab] OR lattice degeneration[tiab])

(posterior vitreous detachment[tiab] OR retinal break*[tiab] OR lattice degeneration[tiab]) AND ((review*[tiab] AND (literature[tiab] OR systematic[tiab] OR search*[tiab])) OR meta-analysis[tiab])

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(Retinal Detachment [mh] OR Retinal Perforations [mh] OR Vitreous Detachment [mh] OR Retinal Degeneration[mh]) AND (Quality of Life[mh]) AND (rhegmatogenous retinal detachment[tiab] OR posterior vitreous detachment[tiab] OR retinal break*[tiab] OR lattice degeneration[tiab])

(Retinal Detachment [mh] OR Retinal Perforations [mh] OR Vitreous Detachment [mh] OR Retinal Degeneration[mh]) AND (Cost-Benefit Analysis[mh] OR Cost of Illness[mh]) AND (posterior vitreous detachment[tiab] OR retinal break*[tiab] OR lattice degeneration[tiab])

(Retinal Detachment/economics [mh] OR Retinal Perforations/economics [mh] OR Vitreous Detachment/economics [mh] OR Retinal Degeneration/economics[mh]) AND (posterior vitreous detachment[tiab] OR retinal break*[tiab] OR lattice degeneration[tiab])

(Retinal Detachment[mh:noexp] OR Retinal Perforations[mh:noexp] OR Vitreous Detachment[mh:noexp] OR Retinal Degeneration[mh:noexp]) AND (Postoperative Complications[mh]) AND (posterior vitreous detachment[tiab] OR retinal break*[tiab] OR lattice degeneration[tiab])

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