**OPTOMETRIC CLINICAL PRACTICE GUIDELINE**

*Care of the Patient with*

# Retinal Detachment And Related Peripheral Vitreoretinal Disease

American Optometric Association

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Doctors of optometry are independent primary health care providers who examine, diagnose, treat, and manage diseases and disorders of the visual system, the eye, and associated structures as well as diagnose related systemic conditions.

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The mission of the profession of optometry is to fulfill the vision and eye care needs of the public through clinical care, research, and education, all of which enhance the quality of life.

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American Optometric Association

# OPTOMETRIC CLINICAL PRACTICE GUIDELINE
# CARE OF THE PATIENT WITH RETINAL DETACHMENT AND RELATED PERIPHERAL VITREORETINAL DISEASE

## Reference Guide for Clinicians


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## INTRODUCTION

Optometrists, through their clinical education, training, experience, and broad geographic distribution, have the means to provide effective primary eye and vision care services for a significant portion of the American public and are often the first health care practitioners to diagnose patients with diseases of the retina.

This Optometric Clinical Practice Guideline for Care of the Patient with Retinal Detachment and Related Peripheral Vitreoretinal Disease describes appropriate examination and treatment procedures to reduce the risk of potential loss of vision from peripheral retinal problems. It contains recommendations for timely diagnosis, treatment, and, when necessary, referral for consultation with or treatment by another health care provider. The Guideline will assist optometrists in achieving the following goals:

- Diagnose significant or frequently encountered peripheral vitreoretinal diseases and related congenital ocular abnormalities
- Improve the quality of care rendered to patients with retinal diseases and related congenital ocular abnormalities
- Identify patients at risk of developing retinal breaks or detachment
- Minimize the ocular morbidity and severe vision loss related to retinal disease through diligent monitoring and timely consultation or referral
- Monitor the gains obtained through treatment
- Inform and educate patients and other health care practitioners about the complications and prevention of retinal disease and the availability of treatment.

## I. STATEMENT OF THE PROBLEM

A retinal detachment can have devastating visual consequences. The patient with retinal detachment may lose a portion or all of the vision in the involved eye, resulting in a significant reduction in visual performance and an inability to function at his or her occupation and other activities of daily living. Retinal detachment often requires surgical repair, which has inherent risks.

Detection of a retinal detachment requires a thorough evaluation, incorporating a detailed patient history and a stereoscopic examination of the entire retina through a dilated pupil. The evaluation of conditions predisposing to retinal detachment requires knowledge of peripheral vitreoretinal diseases that may lead to detachment.

### A. Description and Classification of Retinal Detachment and Related Peripheral Vitreoretinal Disease

This Guideline presents the most common peripheral retinal diseases associated with retinal detachment (See Appendix Figure 4 for the ICD-9-CM classification of retinal detachment and related peripheral vitreoretinal disease).

#### 1. Retinal Detachment

A retinal detachment is a separation of the sensory retina from the underlying retinal pigment epithelium (RPE). There are numerous variations in the basic pathogenesis of a retinal detachment. They include developmental factors (e.g., myopia and Marfan syndrome) that affect the overall size and shape of the globe,[1] vitreoretinal disorders (e.g., coloboma and retinal dysplasia), metabolic disease (e.g., diabetic retinopathy), vascular disease (e.g., sickle cell disease), trauma, inflammation, degenerative conditions, and neoplasms. Retinal detachments can be classified as rhegmatogenous or nonrhegmatogenous.



##### *a. Rhegmatogenous Retinal Detachment*

The most common type of retinal detachment, rhegmatogenous, results from a break in the sensory retina. The break is most often caused by vitreous traction on the surface of the retina. This traction physically pulls a small section of the sensory retina away from the pigment epithelium, resulting in what is called a "retinal tear." Traction at the site of a tear can initiate retinal detachment surrounding the tear by pulling on the surface of the adjacent retina. The break in the retina may also allow fluid from the vitreous cavity to percolate into the potential subretinal space. Thus, a rhegmatogenous retinal detachment caused by a retinal tear is the result of both vitreous traction and fluid ingress between the sensory retina and the pigment epithelium.

##### *b. Nonrhegmatogenous Retinal Detachment*

The second type of retinal detachment, nonrhegmatogenous, usually results from the accumulation of exudate or transudate in the potential subretinal space, rather than from a retinal break. Sometimes a nonrhegmatogenous retinal detachment is caused by sheer traction, without the production of a retinal tear. Other etiologies of this type of detachment include chorioretinitis, metastatic choroidal tumor, choroidal effusion, retinal angioma, Harada's disease, pars planitis, sympathetic ophthalmia, eclampsia, and trauma.

#### 2. Retinal Breaks

Any discontinuity of the neurosensory retina is called a retinal break. When the break results from vitreous traction, it is referred to as a "tear." When the break results from a focal loss of retinal tissue, it is atrophic and referred to as a retinal "hole." Although there are specific distinctions between holes and tears, these terms are often used interchangeably.[2]

##### *a. Atrophic Retinal Holes*

A retinal break that is not caused by vitreous traction but is most likely produced by an atrophic process in which vascular insufficiency of the

underlying choriocapillaris impairs retinal circulation is an atrophic retinal hole. Thinning and degeneration of blood vessels eventually lead to the clinical appearance of small, round defects in an area of thin, partially opaque sensory retina. The sizes of these holes vary from pinpoint to 1.5 disc diameters (DD).[3] In the attached retina, the holes are more red than adjacent retinal tissue. Due to the obstructed view of the underlying choriocapillaris in a detached retina, the holes appear pinkish, grayish, or clear depending on the view of the underlying choroid. Although they may be found in any region of the fundus, most retinal holes occur in the temporal half of the retina and are usually confined to the region between the equator and the ora serrata.

### *b. Operculated Retinal Tears*

When there is significant vitreous traction in a small, discrete area of the retina, the increased vitreoretinal adhesion can result in an operculated retinal tear. The traction pulls a small plug of sensory retina (an operculum) out of the surrounding retina. The operculum, which can be seen as a whitish, disc-shaped floater over the retinal break, moves upon eye movements because it is attached to the detached vitreous cortex. Because it has been separated from its blood supply, the operculum becomes smaller than the break due to contraction secondary to degeneration. Operculated tears in the attached sensory retina are usually round and appear more red than the surrounding retina. They are generally located between the ora serrata and the equator, more frequently in the temporal half of the retina; however, they may occur in any region of the retina.

### *c. Horseshoe and Linear Retinal Tears*

Significant localized vitreous traction can cause horseshoe (flap) or linear retinal tears. Horseshoe tears, which are much more common than linear tears, are the result of vitreous traction pulling a horseshoe-shaped thin curvilinear flap of sensory retina into the vitreous cavity. Away from its blood supply, this flap which is attached at the anterior margin of the tear contracts and degenerates to become smaller than the break. A tear in the attached sensory retina appears more red than the surrounding retina, and the apex of the tear almost always points to the



posterior pole. Such tears can exist in any region of the peripheral retina; however, they are most often found near the posterior margin of the vitreous base in areas of lattice degeneration, pigment clumps, or retinal tufts.

### *d. Retinal Dialysis*

A retinal tear that occurs at the ora serrata, concentric with the ora, is called a retinal dialysis. Most of these tears are less than 90 degrees, and they are rarely bilateral. The underlying pigment epithelium becomes more visible in the area of the tear due to the loss of the overlying retina. If the edge of the dialysis remains close to the RPE, then the tear may not be discovered unless scleral depression is performed. As the vitreous contracts, the tear becomes more elevated in conjunction with an increase in the associated retinal detachment.

There are two types of retinal dialysis: congenital and post-traumatic. The congenital form, which is found in young people, is spontaneous and is associated with an asymptomatic, slowly progressive retinal detachment. Congenital dialyses usually occur in the inferior temporal region of the retina, and their bilateralism is in marked contrast to those of post-traumatic etiology.[4] Post-traumatic dialyses, which are more common, usually occur in the superior nasal region of the retina; however, a blow to the eye at the temporal limbus may result in an inferior, temporal dialysis.[5] The trauma responsible for the tear may have occurred in the distant past.

## 3. Related Peripheral Vitreoretinal Disease

### *a. Retinal Tufts*

Retinal tufts are small areas of gliotic degeneration of the retina associated with vitreous traction. They may be classified as noncystic, cystic, or zonular traction tufts. Noncystic tufts are short (<0.1 mm), thin, internal projections that are often found in clusters. They are almost always located within the vitreous base but may be found elsewhere in the pre-equatorial retina. Larger than noncystic tufts (>0.1 mm), cystic tufts are nodular projections of retinal tissue that occur either within or

posterior to the vitreous base. About 78 percent occur in the equatorial zone.[6] They can be found in any quadrant of the retina, are often unilateral, and usually occur singly. Zonular traction tufts are gliotic tufts that are pulled in an anterior direction by a zonular fiber and, therefore, appear as thin strands, stretched over the ora serrata. They are usually solitary lesions and are often within the vitreous base. Zonular traction tufts, which are most commonly located in the nasal half of the retina, are attached to the retina less than 0.5 mm posterior to the ora serrata; only rarely are they attached posterior to the vitreous base.[7-9]

Retinal tufts are generally stable in size over time, but they may have slight changes in shape due to continuous vitreous traction. Because of the traction exerted by the overlying vitreous, cystic and zonular traction tufts can be associated with a retinal tear and subsequent retinal detachment. Cystic tufts account for the development of as many as 10 percent of rhegmatogenous retinal detachments.[8,10-13] Atrophic retinal holes from retinal thinning adjacent to cystic tufts rarely result in retinal detachment and carry a risk factor of less than 0.3 percent.[11] Zonular traction tufts may have associated retinal holes at the posterior margin, but these tufts only account for 0.11 percent of retinal tears in autopsied eyes.[6] Because zonular traction tufts are often intrabasal (i.e., occur within the area of the vitreous base), they are rarely associated with retinal detachment.

### *b. Lattice Retinal Degeneration*

Lattice retinal degeneration is a vitreoretinal degeneration that manifests changes in both the retina and the overlying vitreous. The involved retina thins and becomes fibrotic, while the vitreous forms a pocket of liquefaction (lacuna) immediately above the affected area of the retina. In many cases, the degenerative insult to the retina causes the tissue to become hyperpigmented, and in about 12-43 percent of cases, it also causes vessels that cross the lesion to develop white sclerotic walls.[14] Lattice retinal degeneration usually occurs in the far periphery of the retina, and only occasionally in the equatorial region. It is more common adjacent to the superior and inferior meridians.[6] The lesions range from 1 to 4 DD in length and from 0.5 to 1.75 DD in width. In 48.1 percent of cases, lattice degeneration is bilateral and fairly symmetrical.[15] The



number of lesions in one eye can vary from 1 to 19, averaging 2.4 per eye.[16]

Lattice retinal degeneration and retinal detachment have a significant association. Lattice degeneration has been found in 20-35 percent of patients undergoing surgery for rhegmatogenous detachments.[17-22] However, this association does not mean that patients with this condition are likely to develop retinal detachment; in fact, detachment is reported in only 0.3-0.5 percent of patients with lattice degeneration.[14,23]

Progressive thinning of the retina due to the overlying liquefied vitreous and vitreous traction at the edges of the lattice lesions may lead to retinal break formation in up to 25 percent of eyes with lattice degeneration.[19] Thinning of the retina can result in the formation of atrophic holes in as many as 18.2-29.2 percent of cases.[14,19,24,25] In one study, 75 percent of all atrophic holes occurred within the area of lattice degeneration.[26] However, the frequency of retinal detachment caused by atrophic holes in lattice degeneration is relatively low and has been reported to be 2.8 percent,[27] 13.9 percent,[28] and 9.5 percent.[29] One study found that over a 3 to 9 year period, the incidence of atrophic holes in lattice that progressed to retinal detachment was zero.[30]

In a later study, the same author found that of 276 consecutive untreated patients (average followup 11 years) fewer than 1 percent developed detachment.[31] However, there have been two reports of atrophic holes that were responsible for approximately 30-44 percent of retinal detachments associated with lattice degeneration.[23,29] Thus considerable controversy exists concerning risk for retinal detachments as sequelae of atrophic holes in lattice degeneration; however, in the absence of concomitant risk factors, most of these lesions need only be followed and the patient educated about possible complications.

### *c. Snail-Track Degeneration*

A vitreoretinal degeneration similar to lattice retinal degeneration, snail-track degeneration also results in retinal thinning as a result of a pocket of vitreous liquefaction (lacuna) just above the lesion. It appears as a glistening white (frost-like) area of the retina, often with numerous

yellow-white flecks through the lesion. Snail-track lesions are similar in size to those of lattice degeneration, and they occur in the same region of the retina, usually between the equator and the ora serrata. Approximately 80 percent of them occur in a zone between the ora serrata and 2 DD anterior to the equator.[32] Most frequently occurring in the temporal half of the retina, snail-track degeneration has the same propensity to form retinal atrophic holes, tears, and detachments as lattice degeneration. The holes in snail-track degeneration tend to be larger, however.

### *d. Retinoschisis*

Retinoschisis is a splitting of the sensory retina into two layers and the filling of the cavity formed by this process with a rather thick fluid. The lesion is elevated, bullous, and "blister"-like. Its inner layer is smooth and taut; it does not undulate with eye movement. This inner layer may contain white sclerotic blood vessels and perhaps snowflake-like deposits. Whereas a fresh retinal detachment demonstrates a relative scotoma on visual field testing, a retinoschisis lesion is characteristically an absolute sharp-margined scotoma.

Retinoschisis most frequently occurs in the temporal region of the retina, about 70 percent in the inferior and about 25 percent in the superior temporal quadrants.[33] Peripheral cystoid degeneration always occurs anterior to the retinoschisis, and coalescence of the cystoid cavities is believed to be partially responsible for the formation of the lesions.[34] Peripheral vitreous traction often plays a major role in retinoschisis formation. Although this condition is usually stable over time, in some cases, retinoschisis may progress slowly toward the posterior pole.

More than 25 percent of eyes with acquired retinoschisis demonstrate at least one retinal break in one layer of the split retina.[35] Routine autopsy examination of eyes with no history of ocular disease revealed that nearly 1 percent had retinoschisis with outer layer breaks.[34] Holes can develop in either or both of the layers of the retinoschisis. A hole in the inner layer alone does not lead to any complications because it only allows fluid from the vitreous cavity to enter the retinoschisis cavity, which does not increase the size of the lesion. Usually, a hole in the



outer layer is of little consequence because it produces only a localized retinal detachment around the hole. Two large studies showed that retinoschisis with outer layer breaks rarely progressed to retinal detachment.[36,37] Another study found a low prevalence (<16%) of retinal detachment in such cases.[38]

The greatest risk factor for retinal detachment in retinoschisis is the presence of holes in both the inner and outer layers of the retina. Holes in both layers can allow a larger amount of fluid from the vitreous cavity to migrate into the potential subretinal space, therefore, resulting in retinal detachment. However, recent long-term observation studies on the likelihood of producing a retinal detachment have been less than conclusive.[37]

### *e. White-Without-Pressure*

Retinal white-without-pressure (WWOP) is an optical phenomenon in which vitreous traction on the underlying retinal surface changes the retinal coloration from its usual orange-red appearance to a translucent white or gray-white. These color changes may occur in a small isolated area or as broad areas in the peripheral retina. Although WWOP may occur at any retinal location, it occurs more frequently in the superior or temporal areas. It rarely occurs near the posterior pole; however, it is not uncommon to find WWOP at the posterior edge of lattice retinal degeneration. WWOP is generally stable between examinations, but its configuration sometimes changes.[39]

### *f. Meridional Folds and Complexes*

Spindle-shaped rolls of peripheral retinal tissue called meridional folds are caused by vitreous traction. Although they are usually aligned with a dentate process, meridional folds can be found at the end of an ora bay. A meridional complex is a meridional fold associated with an enlarged dentate process and a ciliary process.

Meridional folds can extend posteriorly for 0.6-6.0 mm and are usually found in the superior nasal quadrant of the retina.[6] It is unusual to find peripheral cystoid degeneration adjacent to the folds. Meridional

complexes are also more commonly found in the superior nasal quadrant of the retina. Occasionally, there are small retinal holes next to either of these entities, with retinal excavations posterior to the meridional folds. Even though meridional folds do not increase the risk for retinal detachment, a retinal tear may occur at the posterior edge of a fold in an eye with a detachment.[40-42]

### *g. Peripheral Pigmentary Degeneration and Pigment Clumping*

Peripheral pigmentary degeneration is produced by the proliferation and migration of retinal pigment epithelial cells into the overlying sensory retina.[43] The stimulus necessary to produce this condition is probably inflammation caused by either mechanical disturbances of traction or biochemical irritation. In some cases, there may be a developmental component that causes a benign proliferation of pigment epithelial cells into the overlying retina.[44] Peripheral pigmentary degeneration is usually visible as a pigmentation that ranges from various sizes of pigment clumping to a fine, diffuse darkening of the peripheral retina. Isolated pigment clumps may be located anywhere from the ora serrata region to many disc diameters posterior to the ora.[45]

Peripheral pigmentary degeneration is most common in the superior or inferior temporal regions of the retina. It tends to be bilateral and is without gender preference. It has a slight tendency to occur more frequently in patients under 39 years of age who have WWOP. It is also associated with a higher frequency of lattice degeneration in patients over age 40.[45] The literature contains some conflicting statements regarding the possible association of peripheral pigmentary retinal degeneration with retinal break formation.[45,46]

In extreme cases, peripheral pigmentary degeneration may be confused with retinitis pigmentosa (RP); however, in RP the pigment is found in the midperiphery of the retina. RP, which may be accompanied by attenuated retinal vessels, optic disc pallor, or symptoms of night blindness, is found in younger patients. Although it is rarely indicated, electrodiagnostic or visual field testing will help to rule out RP.



An isolated pigment clump is a small area of increased pigmentation (retinal pigment hyperplasia) that occurs due to traction of the overlying vitreous.[47] These lesions do not have perfectly round, smooth, regular borders because they are produced by episodes of irregular vitreous traction. Pigment clumps are small and initially lightly pigmented, but they become larger and more darkly pigmented over time due to continued vitreous traction. The clumps probably represent sites of increased vitreoretinal adhesion. When the traction increases dramatically, as in a posterior vitreous detachment (PVD) or trauma, the result may be a retinal tear.[17,42]

### *h. Peripheral Retinal Hemorrhage*

The result of bleeding from a retinal artery or vein, a retinal hemorrhage is caused by either weakness or degeneration of a blood vessel wall (e.g., diabetes or hypertension) or by external forces that rupture the vessel wall (e.g., vitreous traction or traumatic insult). A small, isolated area of hemorrhage may indicate a retinal tear. Peripheral retinal hemorrhages appear as round red spots of varying size and number; they form quickly but may take weeks to slowly resolve.

Peripheral retinal hemorrhages are common among the general population. Patients with known vascular diseases, systemic hypertension, diabetes mellitus, and other blood disorders (e.g., anemia,[48,49] anticardiolipid antibody disease,[50,51] thrombocytopenia and other platelet disorders,[52,53] polycythemia,[54] and leukemia[55]) may have peripheral retinal hemorrhages. Likewise, patients with pulmonary disease[56,57] or those on anticoagulant drug therapy may have them. Older persons are more likely than younger ones to develop peripheral hemorrhages with or without a retinal tear due to the increasing incidence of PVD.

### *i. Pars Planitis*

Pars planitis, also known as peripheral or intermediate uveitis, is a chronic inflammation of the peripheral retina and pars plana ciliaris. Early fundus changes consist of yellowish-gray exudates on the peripheral retina, ora serrata, and pars plana. These exudates most

frequently accumulate in the inferior region of the fundus, presumably due to gravitational forces. Progression of this condition leads to coalescence of these exudates into a hazy white plaque over the ora serrata known as a "snowbank." Fluffy white "snowballs" may float in the inferior vitreous. There is often peripheral vasculitis, usually affecting the venules; it is seen as sheathed, attenuated retinal vessels. Progression of pars planitis can lead to continued obliteration of the vessels toward the posterior pole, together with the development of optic atrophy and severe loss of vision. Although the etiology of pars planitis is unknown, some suggested causes are immune reaction to the patient's retina or vitreous cells, multiple sclerosis, and cell-mediated immune disease.[58-62]

Pars planitis usually affects children and young adults, seldom occurring before the age of 5 years or after 30 years of age.[63,64] It can be either asymptomatic or minimally symptomatic, with such complaints as asthenopia, floaters, occasional mild ocular redness, and blurred vision. From 66 to 80 percent of patients demonstrate bilateral involvement.[65-68] Children often do not complain of a slow progressive loss of vision, especially if it involves the upper visual field; therefore, this disease may not be discovered until extensive damage has occurred. Nearly one-half of all patients have serious loss of vision by the time pars planitis is diagnosed.[67]

### *j. Chorioretinal Scar*

The result of an inflammatory process involving the underlying choroid and retina, usually secondary to infection or trauma, chorioretinal scars are white to cream-colored fibrotic areas in the fundus and may appear either excavated or elevated. The margins are often pigmented as a result of reactive pigment proliferation and migration. These scars, which can be located in any region of the fundus, are frequently found close to retinal blood vessels. The overlying vitreous is firmly attached to the scar because of the inflammatory reaction, and sometimes fairly dense vitreous bands travel up into the vitreous body, becoming especially apparent during scleral depression. Significant vitreous traction on such a scar may result in a retinal tear and subsequent detachment.



### *k. Posterior Vitreous Detachment*

When the vitreous cortex separates from the posterior retina and optic disc, PVD occurs. Over time, synchysis (liquefaction) and syneresis (contraction) will cause the vitreous to become more mobile and slowly shrink. Eventually, the tractional force of the moving vitreous becomes great enough to begin the separation of the vitreous cortex from the retinal surface. Although first there may only be a partial or incomplete separation of the vitreous from a localized region over the posterior retina, the tractional forces will become great enough to pull the vitreous from the margin of the optic disc, the area of greatest adherence in the posterior region. Once the vitreous cortex breaks free from the optic disc, the anterior separation will continue until it reaches the posterior margin of the vitreous base.

The forward displacement of the vitreous body results in a retrovitreal space that fills with liquefied vitreous, mostly through an opening (prepapillary hole) in the posterior cortex surrounded by the peripapillary glial ring (known as Weiss's or Gardner's ring) and through small fractures in the posterior cortex. In addition, aqueous can retroflow from the ciliary body to aid in filling this space, which can become even larger than that occupied by the collapsing vitreous body. A large reservoir of liquefied vitreous and aqueous is available to pass into any retinal break and assist in the formation of a retinal detachment.

The forward collapsing and rotational motion of the vitreous body on eye movement can exert substantial tractional forces on isolated areas of increased vitreoretinal adhesion, possibly resulting in retinal tears.[69, 70] As the vitreous body detaches, the force of the potential motion becomes greater as separation proceeds. The more vitreous freed from the inner wall of the eye, the greater its ability to shift with eye movements. Therefore, in any peripheral areas of increased vitreoretinal adhesion the collapsed vitreous can have more sudden, strong "whip-like" anterior-posterior actions.[71, 72] Retinal tears caused by a PVD can be operculated, horseshoe-shaped, or linear. The symptoms of a PVD are floaters, photopsia, blurred vision, glare, and rarely, metamorphopsia.

Rhegmatogenous retinal detachment is a possible sequela of PVD and is a natural aftermath of retinal tear formation. One clinical study found that syneresis and PVD occur in 90 percent of cases of retinal detachment.[73] Continuous vitreous traction on the flap of a retinal tear greatly enhances the chances for retinal detachment due to continuous physical pulling on the sensory retina. The physical traction around a retinal tear is probably more significant in retinal detachment than the reservoir of fluid in the retrovitreal space.

## B. Epidemiology of Retinal Detachment and Related Peripheral Vitreoretinal Disease

### 1. Retinal Detachment

#### *a. Prevalence and Incidence*

The incidence of phakic nontraumatic retinal detachment in the general population is about 1 in 10,000 persons per year (0.01 percent),[74-78] and the inclusion of traumatic retinal detachment only slightly increases this percentage. The incidence of retinal breaks in the general population is about 3.3 percent per year.[6] Therefore, the difference in incidence between retinal breaks and detachments indicates that the chances of developing a phakic nontraumatic retinal detachment from most breaks is fairly low (1:330). Rhegmatogenous retinal detachments are bilateral in about 15 percent of cases.[79,80]

Retinal detachments can occur in persons of any age but are most likely to occur between the ages of 40 and 70 years[81] (average age for males, 57 years; for females, 62).[77] Only 3-4 percent of retinal detachments occur in persons under 16 years of age.[82] Retinal detachments, in general, are more common in males;[81] however, nontraumatic retinal detachments are more common in females (65.1%) than males (55.7%).[83] Retinal detachments are also less frequent in African Americans.[81]

#### *b. Risk Factors*

The most common risk factors for retinal detachments are myopia (40%-55%), aphakia (30%-40%), and ocular trauma (10%-20%).[81] In patients



with aphakia, the incidence of retinal detachment is 1-5 percent; half occur in the first year following surgery.[84-88] Presumably this is secondary to PVDs, which frequently occur during this time.[84,85,89-91] Whereas only about 2.9 percent of the adult population have had cataract surgery, approximately 40 percent of patients with a retinal detachment have had prior cataract extraction.[73] Cataract patients with extracapsular cataract extraction (ECCE) and intraocular lens (IOL) implantation have an incidence of 0 to 2 percent of pseudophakic retinal detachment in the first postoperative year.[73,87,92-98] However, when posterior capsulotomy is performed, it essentially negates the advantage of ECCE and the incidence of detachment increases to 1-3 percent.[99-102] The rate of retinal detachment increases significantly after surgical loss of vitreous.[103] Furthermore, in patients with pre-existing retinal holes, the increase in retinal detachment following cataract extraction is substantial.[104]

The incidence of the most common type of retinal break in pseudophakic detachments--a flap tear--varies from 46 to 90 percent.[105-107] Flap tears are usually small and located at the posterior margin of the vitreous base.[108] Retinal tears are of similar type and location, and retinal detachments have similar morphological characteristics and time of onset in aphakic and pseudophakic eyes.[84,85,106-110] Aphakic eyes often have small retinal breaks located close to the vitreous base.

Eyes with lattice retinal degeneration are at risk for retinal detachment, the incidence of which ranges from 0.3 to 0.5 percent;[14,23] however, lattice degeneration has been reported to be associated with approximately 20 to 30 percent of all rhegmatogenous detachments that have required surgery.[18,29]

High myopia (>8 diopters (D) or >24 mm in axial length) is another risk factor for retinal detachment.[79,111,112] Individuals with high myopia have a prevalence of retinal detachment of 0.7 to 6 percent.[113-115] One study showed that for people with myopia over 5 D who live to be 60 years of age the lifetime risk of developing retinal detachment is 2.4 percent, as compared with 0.06 percent risk for persons with emmetropia who reach that age.[116] Patients with over 8 D of myopia have a significant proportion of retinal detachments but only amount to 1 percent of the general population.[85] Myopic patients who undergo cataract surgery are

especially at risk. However, two separate studies have shown that even myopic patients with asymptomatic breaks are unlikely to progress to retinal detachment[89,117] and, therefore, most do not need to be treated prior to cataract surgery.[118]

Trauma significantly increases the risk for retinal detachment because it can produce a retinal tear or dialysis. Ninety percent of the cases of severe ocular trauma occur in males who, on average, are 25 years younger than those with nontraumatic unilateral retinal detachments.[119] In a series of 160 patients, the time between trauma and detachment was 1 month (30%), 8 months (50%), and 2 years (80%).[120,121] Thus, a long interval after the trauma does not eliminate injury as a potential cause of detachment.

Glaucoma is also a risk factor, and clinical studies have reported that 4-7 percent of patients with chronic open angle glaucoma developed retinal detachments.[122-124] This risk is especially evident in patients with pigmentary dispersion syndrome; retinal detachment has been reported to occur in 6.4 percent of such cases.[125] Finally, miotic therapy for glaucoma may be associated with retinal detachment formation. The risk seems to be greater at the initiation of therapy or with the use of stronger miotic agents.[103]

Patients whose histories include retinal detachment in one eye are at increased risk for retinal detachment in the fellow eye. The prevalence in fellow eyes with no predisposing lesions is about 5 percent; in eyes with predisposing factors, it is 10 percent or greater.[71,74,75,104,126-131] Several studies have shown that when such patients develop retinal tears, retinal detachments follow in the fellow eyes 25-30 percent of cases.[127,130,132] Another study found that phakic fellow eyes in patients who did not receive prophylactic treatment for retinal detachment with lattice degeneration are 2.5 times as likely to develop retinal detachment as eyes that receive treatment.[133]

Although a number of studies have shown the benefit of prophylactically treating the fellow eye,[28,134-136] there is a lack of consensus about which eyes should be treated.[133] When the fellow eye is aphakic, the prevalence of retinal detachment is reported to be 2-3 times higher (i.e., 21-36



percent compared with 10 percent prevalence in phakic eyes).[131,137,138] One study reported that of 100 patients with bilateral surgical aphakia and a rhegmatogenous retinal detachment in one eye, 26 percent of the fellow eyes developed a detachment.[139] Therefore, prophylactic treatment of flap tears in aphakic fellow eyes may be beneficial.[137,140] However, the effect of treating round retinal holes in lattice degeneration of aphakic fellow eyes is far less clear. Moreover, a family history of rhegmatogenous retinal detachment places a person at increased risk of developing a detachment.[20,104,126,129]

## 2. Retinal Breaks

### *a. Prevalence and Incidence*

Retinal tears are found in 1.4 percent of eyes affected with lattice degeneration.[19] They are most common at the posterior and lateral margins of lattice lesions. The tears are generally linear in configuration as they form along the edge of the lesion, but they may progress to a horseshoe (flap) tear. Occasionally, a giant tear develops along the posterior margin of extensive lattice degeneration. When such retinal tears are associated with lattice degeneration, PVDs are usually responsible for causing a tear along the margin of the lesion. Retinal tears have a higher incidence of retinal detachment formation; reportedly about 40 to 75 percent of tractional tears associated with lattice have progressed to retinal detachment.[23,29,31]

The incidence of retinal holes and tears is 12.1 percent in patients with high myopia.[113] One study of 156 retinal breaks found that 77 percent were atrophic holes; 13 percent, operculated tears; 10 percent, horseshoe tears.[141] Lattice degeneration has a reported prevalence of atrophic holes that varies from 18.2 to 42 percent,[14,19,31] and in one study 75 percent of all atrophic holes were found in lattice degeneration.[26] Operculated tears and horseshoe and linear tears are more likely to be found in patients with PVD, aphakia, pseudophakia, high myopia, or vitreoretinal degeneration, and in those active in contact sports.

Retinal dialyses are uncommon. The post-traumatic type is more common than the congenital form of dialysis. Males are affected more frequently than females by both the congenital and post-traumatic forms.

### *b. Risk Factors*

Retinal tears are associated with PVD, aphakia, pseudophakia, high myopia,[113] vitreoretinal degeneration, and trauma. Syneresis and PVD are the two most important factors in the development of retinal tears. In one study of 23 eyes with peripheral breaks, 21 demonstrated more syneresis than 21 age-matched controls.[142] Most such tears occur anterior to the equator. The frequency of retinal breaks in eyes with a PVD has been reported to be between 8 and 15 percent.[142-150] One study reported finding PVD in 80 percent of eyes with retinal tears.[151]

Congenital and hereditary risk factors (e.g., idiopathic weakness of the peripheral retina and X-linked juvenile retinoschisis) are well known in the development of retinal dialyses. There are no other known risk factors for developing a retinal dialysis other than involvement in a sport or occupation in which head trauma is a frequent occurrence.

Lattice degeneration[14,19,31] and myopia[113] are risk factors for the development of atrophic holes.

## 3. Related Peripheral Vitreoretinal Disease

### *a. Prevalence and Incidence*

**Retinal tufts**. Noncystic retinal tufts are present in about 72 percent of the adult population. They are bilateral in half of the cases; thus they are present in 59 percent of adult eyes. Cystic tufts, found in about 5 percent of the adult population, are bilateral in only 6 percent of the cases; they are detected in 2.5 percent of adult eyes.[6] Zonular traction tufts, found in about 15 percent of the population, are bilateral in 15 percent of cases; thus they are evident in 9 percent of adult eyes.[9]

**Lattice retinal degeneration**. Lattice degeneration is a common finding in the general population. It has been found in 5-10 percent of autopsied



eyes,[15,19,152] and it has a clinical prevalence of 7.1-10 percent.[16,23,31,45,153] The condition, which begins in the second decade of life, is bilateral in about 50 percent of cases and, therefore, is evident in about 4 percent of affected eyes.[6,45]

**Snail-track degeneration**. Snail-track lesions have been observed in up to 80 percent of eyes with lattice degeneration, although the extent varies considerably.[16]

**Retinoschisis**. The age-related acquired form of retinoschisis is seen in about 4 percent of the population.[33] It is found more commonly in persons over 40 years of age and is rare in those under age 20.[24,38,154] It seems to occur more frequently in females.[155] Advanced retinoschisis, found in approximately 7 percent of people over age 40,[156] is bilateral 82.1 percent of the time.[154]

**White-without-pressure**. WWOP, found in more than 30 percent of normal eyes, has a strong tendency for bilaterality. It occurs in only 5 percent of persons over 20 years of age, while in those over age 70 the frequency of this condition is approximately 66 percent.[157] One study found a prevalence of zero in myopic eyes with the shortest axial length and 54 percent in eyes with an axial length over 33 mm.[45]

**Meridional folds and complexes**. Meridional folds occur in about 26 percent of the population and are bilateral in 55 percent of the cases; thus they are present in 20 percent of all eyes.[6] They are more common in males. Multiple folds are found in 27 percent of affected eyes.[40] Meridional complexes are found in 16 percent of the population. They are bilateral in 58 percent of the affected patients; thus are present in 12 percent of all eyes. Meridional complexes are multiple in 45 percent of affected eyes.[6]

**Peripheral pigmentary degeneration and pigment clumping**. Peripheral pigmentary degeneration has a prevalence of 6 percent in eyes of patients less than 19 years of age, 27 percent in eyes in the age group of 20-39 years, and 41 percent in those over 40 years of age.[45] Myopia is associated with a higher frequency of this condition.[158] One study found peripheral pigmentary degeneration had a prevalence of 16.9 percent in

myopic patients.[113] Another study found that prevalence varied from zero in eyes with 21 mm axial length to 75 percent in eyes with axial lengths in the range of 35 mm.[45] Pigment clumps are commonly found on routine eye examination; the prevalence rates are probably close to that of peripheral pigmentary degeneration.

**Peripheral retinal hemorrhage**. Although peripheral retinal hemorrhages occur commonly, no specific estimate of their prevalence has been reported.

**Pars planitis**. The prevalence of pars planitis was reported to be 7.6 percent and 8 percent for two eye clinic populations,[62,159] although the rate in the general population would be expected to be much smaller. Of patients referred for uveitis, 4.3 to 15.4 percent have pars planitis.[62,159-162] The disease is found equally in both genders.[63] The disease seems to occur more commonly in whites than in African Americans,[62,162] and there have been reports of familial occurrence of pars planitis.[64,65,163]

**Chorioretinal scars**. Males are more likely than females to have chorioretinal scars because they have a higher incidence of ocular trauma.[119] The incidence of postinfective chorioretinal scars is probably equal for males and females. The incidence of scars is higher in older patients because they have had a longer period of exposure to trauma and infection.

**Posterior vitreous detachment**. In one study of autopsied eyes PVD was rare in persons younger than 30 years of age, but the prevalence increased to 10 percent in persons from 30-59 years of age, 27 percent in persons from 60-69 years of age, and 63 percent in persons over 70.[164] Ultrasonography showed PVD in 75 percent of patients over 80 years of age.[165] PVD is symmetric in about 90 percent of patients; the fellow eye usually develops the condition within one or two years.

### *b. Risk Factors*

**Lattice retinal degeneration**. Lattice degeneration affects both genders equally, and refractive error does not seem to be an important factor.[18,153] Some have proposed myopia as a factor in the development of this

[redacted]



condition. It has been reported to occur in 13.2 percent of eyes with high myopia.[113] Another study found that the prevalence of lattice degeneration increased from zero in eyes with short axial lengths of around 21 mm to 16 percent in eyes with long axial lengths (>36 mm).[45] However, others have reported that its occurrence in patients with high myopia is nearly the same as for those with low myopia.[166] Although heredity has been suggested as a factor in lattice degeneration, its role is unclear.[167]

**Snail-track degeneration**. Snail-track degeneration seems to be associated with myopia, and heredity may be a risk factor. One study reported a familial tendency to this condition.[32]

**Retinoschisis**. There are no known risk factors for developing retinoschisis other than age; however, vitreous degeneration (PVD) is found in 60 percent of cases of retinoschisis.[38] This relationship may be coincidental rather than causal.

**White-without-pressure**. In older persons, WWOP is probably related to increased vitreous degeneration. In patients of all ages, it occurs most frequently (22.8%) in myopic patients .[113,168] WWOP has been reported to occur more frequently in African Americans,[168] but this may be due to the increased contrast of WWOP against a dark fundus background.

**Peripheral pigmentary degeneration and pigment clumping**. Lesions of peripheral pigmentary degeneration can occur in persons of any age, but they are more likely to be detected in older adults. Persons most at risk are those with myopia and other conditions causing vitreous degeneration.

**Posterior vitreous detachment**. The normal aging process is the usual mechanism in the development of PVD; however, trauma can also be responsible for its occurrence. This process may be enhanced by head trauma; even a slight bump on the head may initiate PVD in older persons. PVD is more likely to occur in patients with aphakia, myopia, or vitreoretinal degeneration, and in patients whose history includes severe ocular trauma or uveitis.

[redacted]

## C. Clinical Background of Retinal Breaks and Detachment

### 1. Retinal Breaks

#### *a. Natural History*

Atrophic retinal holes are generally stable for long periods. Fluid from the vitreous can percolate through a hole and produce a small localized retinal detachment. Such a detachment produces a cuff of whitish edema. When a pigmented demarcation line occurs at the margin of the detachment, the lesion has usually been present for at least 3 months and is relatively stable.[169] This small detachment usually does not require treatment. Most atrophic retinal holes do not lead to clinically significant retinal detachment because there is no strong vitreous traction; however, a retinal hole has a 7 percent chance of progressing to a retinal detachment.[3]

Operculated retinal tears are also stable for a long time. Fluid from either the vitreous or aqueous can percolate through the break and produce a small surrounding retinal detachment that may require treatment. Most operculated retinal tears do not lead to clinically significant retinal detachments because of release of the vitreous traction to the involved area during the formation of the operculum. However, in one study, symptomatic operculated tears without associated subretinal fluid had a retinal detachment rate of 20 percent; 33 percent of the asymptomatic operculated tears with associated subretinal fluid progressed to clinically significant detachment.[104] No reported cases of asymptomatic operculated tears without associated subretinal fluid progressed to clinically significant retinal detachments.[30,104,170]

Because the vitreous usually remains attached to the apex of the flap and continues to exert traction (which sometimes can be seen as translucent vitreous strands, especially upon scleral depression), horseshoe tears are the leading cause of rhegmatogenous retinal detachments. Symptomatic horseshoe tears have a 30-55 percent chance of progressing to retinal detachment,[19,104,134] and even asymptomatic flap tears with associated subretinal fluid (localized detachment) have a 40 percent chance of progressing to retinal detachment. However, in the absence of subretinal



fluid in asymptomatic flap tears, only 10 percent progressed to retinal detachment.[104] The frequency of these tears increases with age, suggesting a degenerative nature. Lateral tears may develop at the bases of large flap tears due to continued traction on the flap. With time, the lateral tears may enlarge into giant tears; such tears and the associated detachments are then more difficult to repair.

Vitreous hemorrhage, which is frequently concomitant with retinal tears, is the result of the rupture of a retinal vessel during the tearing process. The proximity of a retinal vessel that bridges the break (from the flap to the surrounding retina) may enhance the chances of bleeding into the vitreous due to its continued traction on the vessel.

Retinal dialysis often produces asymptomatic and slowly progressive retinal detachment, characterized by successive demarcation lines. When a detachment is stable in one location for more than 4-6 months, the traction of the detached retina on the attached retina can stimulate reactive hyperplasia of the underlying pigment epithelium. The result is the appearance of a "tidewater" pigment line. A retinal detachment associated with a post-traumatic dialysis may be deceptive in that it often occurs weeks to months following the traumatic event. The tearing process can result in a shearing of retinal vessels that causes a hemorrhage into the vitreous. The retinal dialysis may not be visible initially due to vitreous hemorrhage or retinal edema. Sometimes the trauma induces chorioretinal inflammation at the site of the dialysis, producing a scar and preventing retinal detachment. The posterior edge of a dialysis tends not to form the roll commonly seen in giant retinal tears. The clinician should suspect a retinal dialysis if the retinal detachment extends onto the pars plana. The risk for a retinal detachment from a retinal dialysis is reported to be approximately 20 percent.[18,171]

#### *b. Common Signs, Symptoms, and Complications*

Pigment cells floating in the anterior region of the vitreous cavity, which can be found during biomicroscopic examination, may be a sign of a retinal tear.[172] Known as "tobacco dust" or Shafer's sign, these cells are the result of pigment epithelium exposure to vitreous fluid. The pigment

epithelial cells may dislodge from the RPE and float off into the fluid spaces beneath the detachment or into the vitreous cavity; however, the lack of cells at examination cannot be used as a definite indication that a tear is not present.

Hemorrhage may also have occurred in the vitreous. Vitreous hemorrhage with PVD increases the chance that a retinal tear will occur; about 15 percent of patients with PVD have retinal tears. With vitreous hemorrhage the incidence of tears increases to as high as 70 percent, as opposed to a 2-4 percent incidence of the tears without hemorrhage.[173]

The sudden onset of numerous floaters and the occurrence of flashes of light are hallmarks of a potential retinal tear. Significant vitreous traction on the retina can result in the perception of a flash of light (photopsia), and because retinal tearing usually requires significant physical traction, photopsia is a common finding in the early stages of retinal tear formation. The sudden onset of numerous floaters with a retinal tear is the result of bleeding into the vitreous. The bleeding is produced by the shearing of a retinal blood vessel during the formation of the tear. The red corpuscles floating in the fluid reservoir of the vitreous are very small, and only those adjacent to the retinal surface will cast a shadow significant enough to be detected as black specks moving across the visual field. Inasmuch as most retinal tears are found in the peripheral retina, the floaters usually appear in the periphery of the visual field and move across the central field in a matter of hours.

The complications of a retinal tear are vitreous hemorrhage and retinal detachment. Vitreous hemorrhages are generally small, transient, and resolve without significant sequelae. Larger hemorrhages can cause blurry vision that may take weeks to clear.

## 2. Retinal Detachment

### *a. Natural History*

The vitreous traction usually responsible for producing a tear in the retina creates a portal through which fluid in the vitreous cavity can gain access into the potential space between the sensory retina and the

[redacted]



underlying pigment epithelium. Continuous traction is influential in physically separating the sensory retina from the RPE. When these two forces are strong enough, either alone or in concert, a retinal detachment can begin or an established retinal detachment can progress.

Approximately 30-50 percent of rhegmatogenous retinal detachments are caused by retinal tears demonstrating vitreous traction; only 10 percent result from asymptomatic retinal breaks.[18,104,171] Most retinal detachments occur in the far periphery of the retina, then advance toward the posterior pole and the macula. Because of its peripheral location, a small, early detachment is usually asymptomatic (i.e., the person often is unaware of the loss of an area of peripheral vision).

A nonrhegmatogenous retinal detachment is similar to a rhegmatogenous detachment but lacks a retinal break. Whereas the fluid under a nonrhegmatogenous detachment is rich in proteins, it is more viscous than the fluid found beneath a rhegmatogenous detachment; therefore, it tends to shift with head movement and rarely extends to the ora serrata. The absence of a break upon a retinal detachment should alert the clinician to the possibility that the eye harbors a choroidal tumor, which may be difficult to detect under the retinal detachment. The most common tumors metastatic to the choroid are lung and breast carcinomas.[174] About 75 percent of choroidal malignant melanomas have secondary retinal detachments.[175]

### *b. Common Signs, Symptoms, and Complications*

Early symptoms of retinal detachment (photopsia and/or a sudden shower of floaters due to tearing of a retinal blood vessel) are often the result of vitreous traction. Left unchecked, most clinically significant retinal detachments will eventually enlarge and probably advance toward the macula. As the detachment becomes larger and the resultant visual field defect encroaches on the center of vision, the person will notice what appears to be a "shadow" or "curtain" moving in over his or her vision. When the macula detaches, the person experiences a significant loss of central vision. Because the retina has no pain receptors, neither the tearing nor the physical detachment of the retina is accompanied by pain.

[redacted]

A retinal detachment of recent onset will undulate easily during eye movements. A longstanding detachment does not undulate because of the scarring and contraction of the degenerative sensory retina that occur over time. The further into the vitreous cavity the retina floats, the more elevated it appears during ophthalmoscopy and the greater the number and size of retinal folds in the detachment. In longstanding detachments, the scarring and contraction of the degenerating retina cause the folds to flatten and even disappear with time. A recent detachment appears whitish due to the edema of the outer retinal layers when the sensory retina is separated from its outer blood supply, the choriocapillaris. Longstanding detachments, having undergone degenerative processes, have many characteristics similar to those of retinoschisis (e.g., retinal thinning and a transparent appearance).

### 3. Early Detection and Prevention

Early detection and treatment of a retinal tear may help to prevent the formation of a retinal detachment or the expansion of a pre-existing detachment. Patients at risk should be informed about the symptoms of a retinal tear and retinal detachment (e.g., floaters, flashes, curtain, or shadow) and told to seek care immediately if these symptoms develop. Periodic dilated fundus examinations should be conducted to monitor the status of the retina.

When a retinal detachment is small and located in the periphery of the retina, early detection and treatment can help reduce the risk of vision loss associated with larger or more central detachments. A detachment that is detected early will more likely require less extensive surgery, which is more likely to result in successful reattachment. The most important reason for early detection is to prevent involvement of the posterior pole and detachment of the macula. If the peripheral retina in a symptomatic patient cannot be evaluated adequately, referral to a more experienced examiner is appropriate.[103] Failure to detect a retinal detachment is likely to allow the detachment to become more extensive, increasing the chance of involving the macula. Nearly every eye with a symptomatic retinal detachment will become blind unless the detachment is surgically repaired.

## II. CARE PROCESS

This Guideline describes the optometric care provided to a patient with a retinal detachment or related peripheral vitreoretinal disease. The components of patient care described are not intended to be all inclusive. Professional judgment and individual patient symptoms and findings may have significant impact on the nature, extent, and course of the services provided. Some components of care may be delegated.

### A. Diagnosis of Retinal Detachment and Related Peripheral Vitreoretinal Disease

Evaluation of patients with retinal detachment or related peripheral vitreoretinal disease includes the elements of a comprehensive eye and vision examination.*[*] It may include, but is not limited to, the following areas:

#### 1. Patient History

The clinician should review the patient's present and past history of ocular and systemic disease and elicit information regarding:

- Loss of vision
- Sudden, recent onset of floaters
- Flashing lights
- Loss of peripheral visual field
- Family members with loss of vision or history of retinal disease
- History of trauma.

#### 2. Ocular Examination

The examination for retinal detachment and related peripheral vitreoretinal disease may include, but is not limited to:

[*] Refer to the Optometric Clinical Practice Guideline for Comprehensive Adult Eye and Vision Examination.



- Best corrected visual acuity
- Pupillary responses
- Biomicroscopy
- Binocular indirect ophthalmoscopy, with scleral indentation if indicated
- Tonometry
- Visual field screening (confrontation)
- Retinal drawing or photodocumentation, if indicated.

#### 3. Supplemental Testing

The interpretation of data obtained during the examination may indicate the need for additional testing which may include, but is not limited to:

- Fundus biomicroscopy with Hruby lens, fundus contact lens, or other precorneal condensing lens
- Ultrasonography
- Fluorescein angiography
- Formal visual field testing.

Binocular indirect ophthalmoscopy with pupillary dilation is generally necessary for diagnosis of a peripheral retinal break or detachment. During ophthalmoscopy, the underlying choroidal detail is obscured by the detached retina, a helpful sign in detecting the presence of a retinal detachment. Scleral depression may be needed to detect small, asymptomatic peripheral retinal detachments. The biomicroscope can be used to search for breaks in detachments using a mirrored fundus contact lens, a hand-held precorneal fundus lens, or a wide-field fundus contact lens. A search for all possible retinal breaks should be performed, and any breaks discovered should be accurately drawn on the patient's chart or photodocumented.

The biomicroscopic examination can also be useful in detecting a rhegmatogenous retinal detachment or a retinal tear without a significant associated detachment by the observation of pigment cells floating in the anterior vitreous cavity immediately behind the lens (Shafer's sign or "tobacco dust").[172] These pigment cells can migrate from the uncovered

pigment epithelium, float through the retinal tear in the detachment, and spread into the vitreous cavity; however, the absence of these cells is no guarantee that a retinal tear is not present.

An afferent pupillary defect is common in the eye with a clinically significant retinal detachment, but it is not likely to occur in an eye with a small peripheral detachment. The practitioner should be alert to the possibility of a detached retina when there is a reduction in central visual acuity, which will occur if the detachment involves the macular area.

Sometimes a visual field screening test can aid in the detection of a visual field loss from a clinically significant retinal detachment before pupillary dilation and ophthalmoscopy. However, visual fields are usually ineffective in evaluating patients because measurable field loss occurs only in advanced cases.

Ultrasonography can be used to detect a retinal detachment, especially when the view of the fundus is poor or impossible due to opacification of the ocular media (e.g., dense corneal scars, cataracts, dense pupillary membranes, or dense vitreous scarring or hemorrhage). Ultrasonography can be used to differentiate a retinoschisis from a retinal detachment; a recent retinal detachment is thicker than a retinoschisis and shows a greater sound echo on the monitor screen. Ultrasonography can also help discover a subretinal tumor that may be causing a nonrhegmatogenous retinal detachment.

## B. Management of Retinal Breaks and Detachment

The extent to which an optometrist can provide treatment for retinal breaks or detachments may vary depending on the state's scope of practice laws and regulations and the individual optometrist's certification. Management of the patient with retinal breaks or detachments may require consultation with or referral to a retina specialist, if available, or to a general ophthalmologist who has experience in retinal disease, for those services outside the optometrist's scope of practice.



### 1. Management Strategy for Retinal Breaks

The optometric management of the patient with peripheral vitreoretinal disease is summarized in Appendix Figure 1. The management strategy varies with the type and severity of the retinal break.

Most asymptomatic tears have a low propensity to progress to retinal detachment and, therefore, can generally be followed without treatment, especially in the absence of vitreous traction in the involved area.[30,36,117,176] However, an asymptomatic tear in the at-risk patient with aphakia, pseudophakia with posterior capsule rupture, high myopia, or vitreoretinal degeneration, or in the patient who is active in a contact sport, or one who has a personal or family history of retinal detachment should be referred to a retina specialist or general ophthalmologist experienced in retinal diseases for consideration of retinopexy.

The patient with a symptomatic PVD should be followed at least every 2 to 3 weeks until the condition becomes asymptomatic and no retinal tears can be found. The patient should be advised to return immediately if there is a sudden onset of tiny black specks, if photopsia occurs or increases, or if a curtain appears in his or her visual field, either during the acute phase or at any time later.

Most atrophic retinal holes do not require treatment because they are not associated with vitreous traction.[30,36,117,176] Some studies have shown that atrophic holes in aphakic eyes can be safely followed without treatment.[117,177] Asymptomatic operculated tears (those not associated with vitreous traction) are very unlikely to progress to retinal detachment and generally do not require treatment.[103] However, upon discovery of atrophic retinal holes or asymptomatic operculated tears in patients at risk (e.g., aphakia, pseudophakia, high myopia, or vitreoretinal degeneration, as well as those who are active in contact sports, or who have personal or family histories of retinal detachment), a consultation with an ophthalmologist for possible treatment may be needed.

Autopsy and clinical studies have shown at least one retinal break in approximately 5-15 percent of the general population, but most of the subjects had never developed a retinal detachment.[11,23,24,26,85,148,176,178]

Even though a retinal break is essential in the production of a rhegmatogenous detachment, only about 1 in 70 eyes with a retinal break will develop a retinal detachment.[30,178,179] Peripheral atrophic retinal holes and operculated retinal tears should be drawn on the patient's chart or photodocumented, if observable with the fundus camera, for future reference.

The patient with a symptomatic operculated tear, especially if it involves subretinal fluid (i.e., localized detachment), should also be referred to an ophthalmologist for possible retinopexy. Fresh, operculated tears, which are large and superiorly located or associated with significant vitreous hemorrhage, should also be considered for treatment.[103]

Although simply following asymptomatic flap tears may be sufficient, treatment of the tears is common because of concern that retinal detachment may occur. Flap tears in aphakic or pseudophakic patients should be treated, especially if the tears are large or located posteriorly.[89,117,177] Patients with symptomatic tears, with or without subretinal fluid (localized detachment), should be referred immediately for retinopexy.

Due to increased risk for vitreous hemorrhage from a bridging blood vessel in a retinal tear, some retina specialists believe it necessary to treat the eye with photocoagulation or scleral buckling.[180] Others believe that a bridging blood vessel will hemorrhage, with or without treatment.[181,182]

The patient with a retinal dialysis tear should be referred to a retina specialist or general ophthalmologist experienced in retinal disease. Such a condition usually requires treatment due to its propensity to produce a retinal detachment. Treatment consists of retinal detachment surgery with scleral buckling and cryotherapy or expanding gas injections into the vitreous cavity. A retinal dialysis tear should be drawn on the patient's chart, or photodocumented if the retinal dialysis is observable with the fundus camera, for future reference if the patient declines treatment.

It is important to involve the patient in the treatment decision making process; it reduces anxiety and makes the patient more relaxed and



cooperative.[81] The optometrist should tell the patient that the risk associated with the treatment of a problematic retinal tear is less than the risk of developing a retinal detachment. Prophylactic treatment of a symptomatic retinal tear decreases the likelihood of subsequent retinal detachment to approximately 5 percent (studies vary from 1.4%-7.8%).[28,134,135,169,183-186]

The most common complications of peripheral retinopexy are formation of a macular pucker, which occurs in about 3 percent of cases (range, 0%-2.2%),[20,170,187] and formation of additional retinal tears due to vitreous shrinkage in about 5 percent of cases (range, 2.1%-6.7%).[20,187] The probability of developing a retinal detachment following treatment varies from 1.8 to 6.2 percent.[20] The most frequent cause of a retinal detachment subsequent to prophylactic retinopexy is inadequate treatment of the retina anterior to the flap tear.[170,188,189]

## 2. Management Strategy for Retinal Detachment

The optometric management of the patient with retinal detachment is outlined in Appendix Figure 2.

Initial management of the patient with retinal detachment includes restriction of physical activity and reduction in eye movement. In some cases, bilateral patching can reduce the potential effect of inertial forces caused by head and eye movements that could increase the size of the detached area.

Without surgery, nearly every eye with a symptomatic retinal detachment will become blind. Following the diagnosis of significant retinal detachment, the optometrist should make an immediate referral to a retina specialist for surgery. Even subclinical retinal detachments around breaks (e.g., detachments caused by subretinal fluid that extends at least 1 DD from the break, but no more than 2 DD posterior to the equator) may require treatment since as many as 30 percent of them will progress to clinically significant detachments.[104] However, some longstanding subclinical detachments may simply be followed.[103] Spontaneous reattachment of a retinal detachment rarely occurs. The success rate of

surgical reattachment is high, although more than one procedure may be needed.[20,190-192]

Treatment for retinal detachment consists of creating an effective chorioretinal adhesion to prevent leakage of fluid between the sensory retina and the underlying pigment epithelium. Laser photocoagulation or cryotherapy can be used to create a scar that attaches the surrounding retina to the underlying choroid to prevent a subsequent retinal detachment. A scleral buckle results in close apposition of the retinal tear to the retinopexy treated area and has the added effect of reducing transvitreal traction. Some alternative treatments are pneumatic retinopexy, expanding gases, air injection, silicone oil injections, and vitrectomy, all of which have advocates and have been shown to be effective in certain situations. Whenever the macula is threatened by the advancing detachment, it should be treated as soon as possible to prevent loss of central vision.[192-195]

There is no evidence that lattice degeneration without holes requires treatment, and prophylactic therapy is of little value.[31,103,196] However, a recent study showed that prophylactic treatment of lattice degeneration in the phakic fellow eye of patients with retinal detachment reduced the chances of subsequent detachment from 5.1 to 1.8 percent over 7 years.[196] When retinal tears are found with this condition, it is prudent to refer the patient to a retina specialist or a general ophthalmologist experienced in retinal disease for consideration of appropriate treatment modalities.

Because retinal detachment associated with a retinoschisis is uncommon--and even if a detachment does occur, the progression is very slow--most retinoschises with outer layer breaks need not be treated.[103] Treatment may be considered if the retinoschisis has large, multiple, or posterior breaks, or if breaks in a retinoschisis coincide with a retinoschisis detachment in the fellow eye.[36] However, some clinicians believe that treatment is indicated only if the retinoschisis is associated with progressive, symptomatic retinal detachment.[37] A patient who has retinoschisis with holes in both the inner and outer layers should be referred to a retina specialist for possible treatment. Such treatment consists of cryopexy or laserpexy, which is successful in completely



flattening the lesion in about 50 percent of the cases.[197] When the retinoschisis is advancing toward the macula, the patient should be referred to a retina specialist or a general ophthalmologist experienced in retinal disease for possible prophylactic laser treatment just posterior to the leading margin of the lesion.

### 3. Patient Education

The optometrist should educate the patient about the symptoms of a retinal detachment, retinal tear, or related peripheral vitreoretinal disease and advise him or her to return immediately if the symptoms occur. Prompt recognition of symptoms will increase the chances for successful surgery and better postoperative visual acuity.[102,195] Studies have confirmed that the success rate for retinal detachment repair in the fellow eye of a previous retinal detachment is higher and that the visual acuity results are better with early detection.[131,137]

### 4. Prognosis and Followup

The prognosis for the patient with a retinal break and the need for followup by the optometrist depend on the type and severity of the break. The frequency and composition of evaluation and management visits and the prognosis for patients with specific conditions are summarized in Appendix Figure 3.

#### *a. Retinal Breaks*

The prognosis for patients with atrophic holes is good due to the low propensity to progress to retinal detachment. Patients with atrophic retinal holes should have yearly eye examinations, but those with surrounding localized (<1 DD) retinal detachments should be seen every 6 to 12 months.

The prognosis for patients with operculated tears is good because the propensity to develop a retinal detachment is fairly low. Patients with asymptomatic operculated retinal tears should have yearly eye examinations, and patients with symptomatic tears, who decline referral or treatment, should be seen every 3 to 6 months.

The prognosis for patients with horseshoe or linear tears is guarded because of the high probability that the tears will progress to retinal detachment. Following prophylactic treatment, most clinicians see the patient in 1 week and again in 3 to 6 weeks. Even for patients who are successfully treated, followup examinations are essential.[103] In one study, 7 percent of such patients developed additional breaks, and 95 percent of the breaks occurred in the first 3 months following treatment.[198] Patients with untreated asymptomatic horseshoe or linear retinal tears who decline referral for treatment should have eye examinations every 6 months.

The prognosis for the patient with a retinal dialysis tear is poor because there is a high propensity for progression to retinal detachment. However, the prognosis is generally favorable following surgical treatment.

### *b. Retinal Detachment*

The prognosis for recovery is poor for the involved area of a detached retina due to degeneration of the detached photoreceptors over time. In general, the longer the retina is detached, the greater the possible loss of vision after reattachment of the retina. The prognosis is very poor when the detachment involves the macula. The longer the macula is detached, the poorer the resultant central visual acuity. A postoperative visual acuity of 20/20 is sometimes achievable following reattachment of the macula, but it is rare. Therefore, when a detached macula is reattached within a few days, there is a chance of retaining the pre-existing visual acuity. Even when 20/20 vision is achieved, the patient may experience and complain of metamorphopsia (distorted vision).

A number of complications following scleral buckling procedures have been reported. They include anterior segment ischemia syndrome, corneal damage, ptosis, lagophthalmos, heterotropia, symblepharon, trichiasis, entropion, ectropion, implantation cyst, glaucoma, cataract, delayed intraocular hemorrhage, cystoid macular edema, central retinal artery occlusion, optic atrophy, sympathetic ophthalmia, phthisis bulbi, and proliferative vitreoretinopathy (PVR).[199] Macular pucker occurs in 3 to 8.5 percent of patients with rhegmatogenous retinal detachments who



receive photocoagulation or cryopexy during the detachment surgery.[170,187,200] Patients having tears with rolled edges, equatorial ridges, and contraction scars on the detached retina are more likely to develop a macular pucker after a scleral buckling procedure.[201]

The patient's initial followup visits after retinal detachment surgery are usually performed by the retina specialist. Subsequently, the patient should be evaluated by the referring optometrist within a year of the surgery and yearly thereafter for dilated retinal examination to ascertain the status of the retina and to detect subsequent retinal tears on the buckle or other retinal areas.

## 5. Management of Patients with Severe, Irreversible Vision Loss

The patient who has a retinal detachment with macular involvement or related peripheral retinal disease with macular sequela may suffer permanent central vision loss. Consultation with an optometrist who has advanced training or clinical experience in low vision is advisable. Patients may benefit from low vision rehabilitation that includes the use of specialized optical devices and training.

Patients should be evaluated to determine their potential to benefit from comprehensive low vision rehabilitation that reduces the debilitating effects of vision loss from a previous retinal detachment. This task-oriented evaluation may include, but is not limited to:

- Expanded patient history and needs assessment
- Low vision assessment of visual acuity (including eccentric viewing)
- Low vision refraction
- Binocular function assessment
- Supplemental testing, including visual fields, contrast sensitivity, and color vision
- Response to optical and electro-optical magnification
- Response to selective absorption filters.

Once appropriate optical requirements have been determined, the clinician should educate and train the patient in methods of improving

visual function with and without optical devices. The patient should be encouraged to use prescription optical devices for work, home, and social activities.

The goal of low vision rehabilitation is to reduce ocular morbidity and enhance the quality of life. In addition to optical intervention, the evaluation should include the need for nonoptical devices, special lighting, posture aids, contrast enhancement, enlarged print, and nonvisual methods or devices when appropriate. These devices, which significantly enhance the rehabilitative process, are necessary to complement the use of optical devices.

When indicated, the optometrist should recommend blind rehabilitation, occupational, vocational and independent living counseling services, and psychosocial consultation. Patients should be informed of other resources including agencies that register and provide services and advocacy to individuals with legal blindness or visual impairment. These agencies can provide information regarding large-print and talking books, independent travel aids, and other devices geared to improve the patient's quality of life and ability to function in their own households. The optometrist should provide the patient written documentation of his or her status relating to legal blindness for state and federal (Internal Revenue Service) tax requirements. Local and national support groups for the visually impaired assist many patients in coping with the anxiety and concerns of vision loss. Such groups also provide information regarding resources to help patients function safely and productively in their environment.

## CONCLUSION

The optometrist is in a position to diagnose peripheral retinal conditions that are of great significance to the ocular health of his or her patients. Through early detection and timely treatment, preventive measures can protect and maintain the patient's ocular health and vision. A comprehensive eye examination, including a stereoscopic retinal examination through a dilated pupil, enables the optometrist to diagnose potentially sight-threatening conditions. Management of the patient with peripheral retinal disease involves appropriate documentation, patient followup, and, when appropriate, referral for consultation with or treatment by a retina specialist or a general ophthalmologist experienced in retinal disease.



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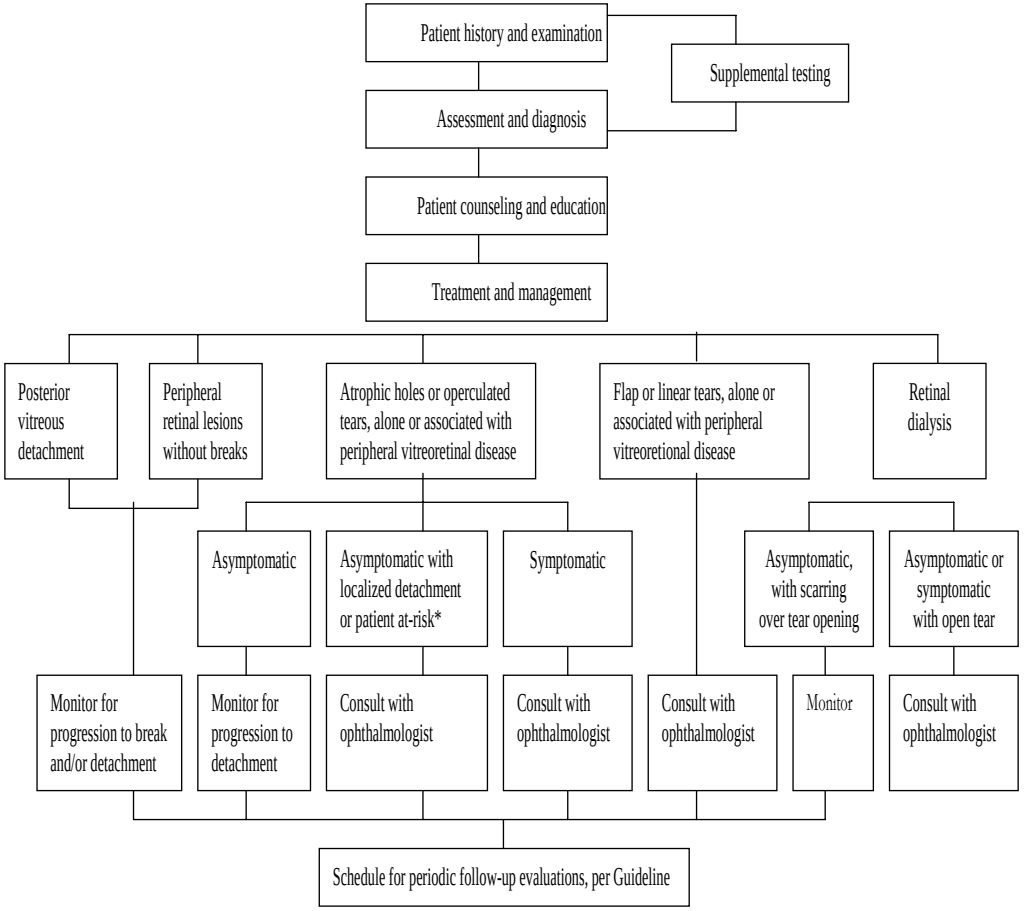
## IV. APPENDIX

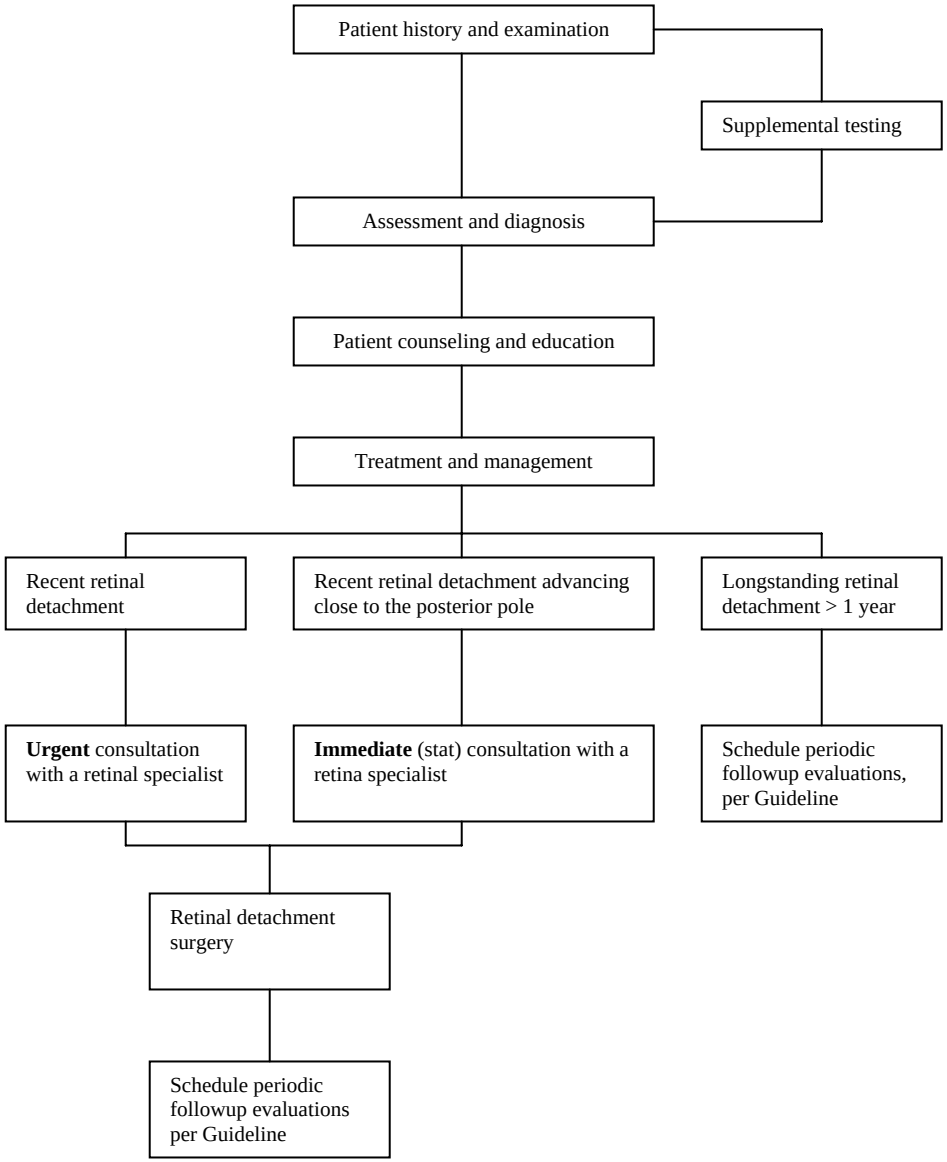
**Figure 1**
**Optometric Management of the Patient with Peripheral Vitreoretinal Disease: A Brief Flowchart**




* Risk Factors: Aphakis, pseudophakia, high myopia, vitreoretinal degeneration, active in sports, or personal or family history of retinal detachment

[redacted]

**Figure 2**
**Optometric Management of the Patient with Retinal Detachment: A Brief Flowchart**

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## Figure 3
## Frequency and Composition of Evaluation and Management Visits for Retinal Detachment and Related Peripheral Vitreoretinal Disease *

| Type of Patient | Frequency of Examination | Patient History | Visual Acuity | Binocular Indirect Ophthalmoscopy | Formal Visual Field Testing | Photo Documentation | Management Plan |
|---|---|---|---|---|---|---|---|
| Posterior vitreous detachment | Every 2 to 3 weeks until photopsia resolves | Yes | Yes | Yes | No | No | Educate patient; observe for possible progression to break or detachment |
| Peripheral retinal lesion without break | Every 6-12 months | Yes | Yes | Yes | No | If possible | Document for future reference; educate patient; observe for possible progression to break or detachment |
| Atrophic hole --Asymptomatic | Annual | Yes | Yes | Yes | No | If possible | Document for future reference; educate patient; observe |
| --Asymptomatic with local detachment or at-risk patient | Every 6-12 months | Yes | Yes | Yes | No | If possible | Document for future reference; educate patient; observe or consult with retina specialist/ophthalmologist |
| Operculated tear--Asymptomatic | Annual | Yes | Yes | Yes | No | If possible | Document for future reference; educate patient; observe |
| --Asymptometic local detachment or at-risk patient | Every 6-12 months | Yes | Yes | Yes | No | If possible | Document for future reference; educate patient; observe or consult with retina specialist/ophthalmologist |
| --Symptomatic | | | | | | | Referral to retina specialist/ophthalmologist |
| Flap or linear tear--Asymptomatic | Every 6 months | Yes | Yes | Yes | No | If possible | Document for future reference; educate patient; observe for possible progression or consult with retina specialist/ophthalmologist |
| --Asymptomatic with local detachment or at-risk patient | | | | | | | Consult with retina specialist/ophthalmologist |
| --Symptomatic | | | | | | | Referral to retina specialist/ophthalmologist |
| Retinal dialysis--Asymptomatic and scarred over | Annual | Yes | Yes | Yes | No | If possible | Document for future reference; educate patient; observe |
| --Asymptomatic or symptomatic with open break | | | | | | | Referral to retina specialist/ophthalmologist |
| Retinal Detachment--Recent symptomatic | | | | | | | Urgent referral to retina specialist/ophthalmologist |
| --Recent, advancing close to posterior pole | | | | | | | Immediate (stat) referral to retina specialist/ophthalmologist |
| --Long-standing detachment (>1 yr) | Annual | Yes | Yes | Yes | Yes | If possible | Document for future reference; educate patient; observe |

**Figure 4**
**ICD-9-CM Classification of**
**Retinal Detachment and Related Peripheral Vitreoretinal Disease**

**Retinal detachments and defects** **361**

**Retinal detachment with retinal defect** **361.0**
Rhegmatogenous retinal detachment
*Excludes: detachment of retinal pigment epithelium (362.42-362.43)*
*retinal detachment (serous) (without defect) (361.2)*

**Retinal detachment with retinal defect, unspecified** **361.00**

**Recent detachment, partial, with single defect** **361.01**

**Recent detachment, partial, with multiple defects** **361.02**

**Recent detachment, partial, with giant tear** **361.03**

**Recent detachment, partial , with retinal dialysis** **361.04**
Dialysis (juvenile) of retina (with detachment)

**Recent detachment, total or subtotal** **361.05**

**Old detachment, partial** **361.06**
Delimited old retinal detachment

**Old detachment, total or subtotal** **361.07**

**Retinoschisis and retinal cysts** **361.1**
Excludes: juvenile retinoschisis (362.73)
microcystoid degeneration of retina (362.62)
parasitic cyst of retina (360.13)

**Retinoschisis, unspecified** **361.10**

**Flat retinoschisis** **361.11**

**Bullous retinoschisis** **361.12**

**Primary retinal cysts** **361.13**

[redacted]



**Secondary retinal cysts** **361.14**

**Other** **361.19**
Pseudocyst of retina

**Serous retinal detachment** **361.2**
Retinal detachment without retinal defect
*Excludes: central serous retinopathy (362.41)*
*retinal pigment epithelium detachment (362.42-362.43)*

**Retinal defects without detachment** **361.3**
*Excludes: chorioretinal scars after surgery for detachment (363.30-363.35)*
peripheral retinal degeneration without defect (362.60-362.66)

**Retinal defect, unspecified** **361.30**
Retinal break(s) NOS

**Round hole of retina without detachment** **361.31**

**Horseshoe tear of retina without detachment** **361.32**
Operculum of retina without mention of detachment

**Multiple defects of retina without detachment** **361.33**

**Other forms of retinal detachment** **361.8**

**Traction detachment of retina** **361.81**
Traction detachment with vitreoretinal organization

**Other** **361.89**

**Unspecified retinal detachment** **361.9**

**Separation of retinal layers** **362.4**
*Excludes: retinal detachment (serous) (361.2)*
*rhegmatogenous (361.00-361.07)*

**Retinal layer separation, unspecified** **362.40**

**Central serous retinopathy** **362.41**

[redacted]

**Serous detachment of retinal pigment epithelium** **362.42**
Exudative detachment of retinal pigment epithelium

**Hemorrhagic detachment of retinal pigment epithelium** **362.43**

**Peripheral retinal degenerations** **362.6**
*Excludes: hereditary retinal degeneration [dystrophy] (362.70-362.77)*
*retinal degeneration with retina defect (361.00-361.07)*

**Peripheral retinal degeneration, unspecified** **362.60**

**Paving stone degeneration** **362.61**

**Microcystoid degeneration** **362.62**
Blessig's cysts Iwanoff's cysts

**Lattice degeneration** **362.63**
Palisade degeneration of retina

**Senile reticular degeneration** **362.64**

**Secondary pigmentary degeneration** **362.65**
Pseudoretinitis pigmentosa

**Secondary vitreoretinal degeneration** **362.66**

**Other retinal disorders** **362.8**
*Excludes: chorioretinal inflammation (363.0-363.2)*
*chorioretinal scars (363.30-363.35)*

**Retinal hemorrhage** **362.81**
Hemorrhage:
preretinal
retinal (deep) (superficial)
subretinal

**Retinal exudates and deposits** **362.82**

**Retinal edema** **362.83**
Retinal:
cotton wool spots
edema (localized) (macular) (peripheral)



**Retinal ischemia** **362.84**

**Retinal nerve fiber bundle defects** **362.85**

**Other retinal disorders** **362.89**

**Unspecified retinal disorder** **362.9**

**Chorioretinal inflammations, scars, and other disorders of choroid** **363**

**Focal chorioretinitis and focal retinochoroiditis** **363.0**
*Excludes: focal chorioretinitis or retinochoroiditis in:*
*histoplasmosis (115.02, 115.12, 115.92)*
*toxoplasmosis (130.2)*
*congenital infection (771.2)*

**Focal chorioretinitis, unspecified** **363.00**
Focal:
choroiditis or chorioretinitis NOS
retinitis or retinochoroiditis NOS

**Focal choroiditis and chorioretinitis, peripheral** **363.04**

**Disseminated chorioretinitis and disseminated retinochoroiditis** **363.1**
*Excludes: disseminated choroiditis or chorioretinitis in secondary syphilis (091.51)*
*neurosyphilitic disseminated retinitis or retinochoroiditis (094.83)*
*retinal (peri)vasculitis (362.18)*

**Disseminated chorioretinitis, unspecified** **363.10**
Disseminated:
choroiditis or chorioretinitis NOS
retinitis or retinochoroiditis NOS

**Disseminated choroiditis and chorioretinitis, peripheral** **363.12**

**Disseminated choroiditis and chorioretinitis, generalized** **363.13**
Use additional code for any underlying disease, as:
Tuberculosis (017.3)

**Other and unspecified forms of chorioretinitis and retinochoroiditis** **363.2**
Excludes: panophthalmitis (360.02)
sympathetic uveitis (360.11)
uveitis NOS (364.3)

**Chorioretinits, unspecified** **363.20**
Choroiditis NOS
Retinitis NOS
Uveitis, posterior NOS

**Pars planitis** **363.21**
Posterior cyclitis

**Chorioretinal scars** **363.3**
Scar (postinflammatory) (postsurgical) (posttraumatic):
choroid
retina

**Chorioretinal scar, unspecified** **363.30**

**Peripheral scars** **363.34**

**Disseminated scars** **363.35**

[redacted]



## Abbreviations of Commonly Used Terms

| | |
|---|---|
| D | Diopter |
| DD | Disc diameters |
| ECCE | Extracapsular cataract extraction |
| IOL | Intraocular lens |
| PVD | Posterior vitreous detachment |
| PVR | Proliferative vitreoretinopathy |
| RP | Retinitis pigmentosa |
| RPE | Retinal pigment epithelium |
| WWOP | White-without-pressure |

[redacted]

## Glossary

**Afferent pupillary defect** A defect of the pupillary reflex characterized by a smaller constriction of both pupils when the affected eye is stimulated by light, as compared with that occurring when the normal eye is stimulated.

**Axial length** The distance from the anterior pole to the posterior pole of the eye.

**Biomicroscopy** Examination of ocular tissue using a bright focal source of light with a slit of variable width and height and a binocular microscope with variable magnification.

**Chorioretinal** A term used to describe both the choroid and retinal layers as a dual unit.

**Choroid** A vascular coat of the eye underneath the retina that nourishes the outer layers of the retina.

**Cryotherapy/Cryopexy** A freezing process used to affix the retina to the underlying choroid in the treatment of retinal breaks, retinal detachment and other retinal conditions.

**Cystoid macular edema (CME)** A swelling of the retina in the macular region caused by serous fluid accumulating in the retinal tissue, usually assuming a petaloid configuration.

**Equator** The circular line that is equally distant from the anterior and posterior poles of the eye.

**Floater** A small opacity above the retina that casts a shadow significant enough to be detected subjectively as a spot or spots that move in the patient's vision.

**Fluorescein angiography** A procedure whereby sodium fluorescein dye is injected intravenously into the vascular circulation and observed as it transits the retina and choroid.



**Fundus** The concave inner wall of the eye seen during ophthalmoscopy.

**Intrabasal** Within the vitreous base (a strongly adherent area on either side of the ora serrata of the vitreous cortex).

**Lacuna** A small space, cavity or depression.

**Laser photocoagulation** Coagulation of tissue in the eye by laser for treatment of retinal detachments, retinal holes, aneurysms, hemorrhages, and neoplasms.

**Macula** The central area of the retina, 3-5 mm in diameter, with the foveal depression in the center.

**Operculum** A disk-shaped torn piece of retinal tissue floating above a retinal break.

**Ophthalmoscopy** Examination of the interior of the eye, using an illumination system involving the light source, lenses, and a prism or mirror, and an observation system, involving a peephole and a set of lenses.

**Ora serrata** The serrated anterior border of the retina.

**Pars plana** The darkly pigmented posterior zone of the ciliary body continuous with the retina at the ora serrata..

**Photopsia** A sensation of instantaneous flashes of light; most commonly indicative of retinal traction.

**Photoreceptors** Receptors in the outermost layer of the sensory retina capable of being activated by light stimuli. Light energy is transformed into electrochemical impulses that are sent to the brain and are then subjectively appreciated as light or vision.

**Pneumatic retinopexy** The injection of air or expanding gases into the eye in the treatment of retinal tears and detachment.

**Posterior vitreous detachment (PVD)** Separation of the vitreous body from its attachment to the retinal surface due to shrinkage from degenerative or inflammatory conditions, trauma, myopia, or age.

**Retina** The light-receptive, innermost coat of the eye that represents the terminal expansion of the optic nerve. It is responsible for sending biochemical impulses to the brain which are interpreted as vision.

**Retinal pigment epithelium (RPE)** The dark layer of the retina between the lamina of Bruch and the neurosensory retina. It is rich in melanin and provides metabolic support to the photoreceptors and outer retinal complex.

**Retinopexy** Surgical procedure for formation of chorioretinal adhesions for correction of retinal breaks, retinal detachment, and other retinal conditions.

**Scleral buckle** Surgical procedure for repair of detachment of the retina in which indentations are made in the sclera over the retinal tears to promote adherence of the retina to the choroid and decrease transvitreal traction.

**Scleral depression** A procedure that combines indentation of the peripheral eye wall with indirect ophthalmoscopy or biomicroscopy to view the peripheral retina.

**Ultrasonography** The location, measurement, or delineation of deep structures in the eye by measuring the reflection of transmission of high frequency or ultrasonic waves.

**Vasculitis** An inflammation of blood vessels.

**Visual acuity** The clearness of vision that depends upon the sharpness of focus of the retinal image and the integrity of the retina and visual pathway.

**Visual field** The area or extent of space visible to an eye in a given position.



**Vitreous humor** The gelatinous, colorless, transparent substance filling the vitreous chamber of the eye; i.e., the space between the crystalline lens, ciliary body, and retina.

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