break in the weeks that follow.[13, 15, 16, 18] One study followed 389 patients with PVDs but no concurrent retinal breaks or detachments, and 7% developed retinal tears by 6.24 years after PVD diagnosis.[19]

TABLE 1 STAGES* OF POSTERIOR VITREOUS DETACHMENT

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

NOTE: These stages can be studied with optical coherence tomography.[4, 20]

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

Approximately 80% of patients who presented without detected breaks, and then had breaks occur subsequently, had either pigmented cells or hemorrhage in the vitreous or retina at the initial evaluation, or new symptoms that prompted a return visit to the ophthalmologist.[15, 16]

A spontaneous vitreous hemorrhage can be the presenting sign of PVD or may occur during the evolution of the PVD. Two-thirds of patients who present with associated vitreous hemorrhage were found to have at least one break. In this subgroup, one-third had more than one break and approximately 88% of the breaks occurred in the superior quadrants.[21]

EVOLUTION OF RETINAL BREAKS AND LATTICE DEGENERATION

Precursors to RRDs are PVD, asymptomatic retinal breaks, symptomatic retinal breaks, lattice retinal degeneration, and cystic and zonular traction retinal tufts. (See Glossary.) Because spontaneous retinal reattachment is uncommon, nearly all patients with a symptomatic clinical RRD will progressively lose vision unless the detachment is repaired. Currently, more than 95% of uncomplicated RRDs can be successfully repaired, although more than one procedure may be required.[22] The prophylactic treatment of high-risk breaks usually prevents RRD. An early diagnosis of an RRD is also important because the rate of successful reattachment is higher, and the visual results are better when repaired early and especially before the RRD involves the macula.[23, 24] The goal of RRD treatment is to allow patients to maintain or re-establish their abilities to read, work, drive, care for themselves, and maintain their quality of life.[25]

Asymptomatic Retinal Breaks

Asymptomatic operculated holes and atrophic round holes rarely lead to RRD. A 1998 study followed 46 asymptomatic eyes with operculated retinal breaks over an average of 11 years.[26] A 1974 study followed 28 eyes for up to 5 years in subjects where 80% of the fellow eyes had an RRD.[27, 28] All combined, none of the 74 eyes from these studies progressed to RRD during the follow-up period.

Eyes with signs and symptoms of acute PVD may have atrophic retinal breaks with clinical features of chronicity, such as pigment demarcation, suggesting that they are unrelated to the acute vitreoretinal traction from the PVD. Such breaks are considered to be pre-existing rather than symptomatic. Treatment may be considered for these breaks in certain situations, although the literature provides little guidance.[28] Randomized clinical trials are not available; therefore, there is limited evidence to support prophylactic therapy.[28]

Approximately 5% of eyes with asymptomatic horseshoe tears progress to RRD.[26, 29, 30] Horseshoe tears discovered in asymptomatic fellow eyes are less likely than symptomatic horseshoe tears to lead to clinical RRD (See Glossary).

Symptomatic Retinal Breaks

A symptomatic retinal break is defined as a break caused by vitreoretinal traction in a patient typically with a PVD associated with new-onset flashes and/or floaters. At least half of untreated symptomatic retinal breaks with persistent vitreoretinal traction (horseshoe or flap