



## Classifying posterior vitreous detachment: a new way to look at the invisible

J SEBAG

*Br. J. Ophthalmol.* 1997;81;521-

---

Updated information and services can be found at:

<http://bjournal.bmj.com/cgi/content/full/81/7/521>

---

*These include:*

### References

This article cites 6 articles, 2 of which can be accessed free at:

<http://bjournal.bmj.com/cgi/content/full/81/7/521#BIBL>

3 online articles that cite this article can be accessed at:

<http://bjournal.bmj.com/cgi/content/full/81/7/521#otherarticles>

### Rapid responses

You can respond to this article at:

<http://bjournal.bmj.com/cgi/eletter-submit/81/7/521>

### Email alerting service

Receive free email alerts when new articles cite this article - sign up in the box at the top right corner of the article

---

### Notes

---

To order reprints of this article go to:

<http://journals.bmj.com/cgi/reprintform>

To subscribe to *British Journal of Ophthalmology* go to:

<http://journals.bmj.com/subscriptions/>

# BJO

British Journal of Ophthalmology

---

## Editorials

---

### Classifying posterior vitreous detachment: a new way to look at the invisible

Vitreous is the largest structure within the eye, yet our knowledge of its structure, function, and role in disorders of sight is less than for any other ocular structures. This limitation in understanding arises principally from the inability to adequately visualise vitreous clinically and the lack of effective techniques for its study in the laboratory.<sup>1</sup> Furthermore, there has been the notable absence of a systematic approach to characterising changes in this structure with aging and disease. This is strikingly apparent when one considers the lack of a useful system with which to classify the most common event befalling the corpus vitreus—posterior vitreous detachment (PVD). Similar obstacles in our understanding of proliferative vitreoretinopathy, macular holes, retinopathy of prematurity, and proliferative diabetic vitreoretinopathy (PDVR) were overcome with the introduction of clinical classification systems.<sup>2,3</sup> This issue of the *BJO* features an article by Kakehashi *et al* (p 527) that successfully addresses this deficiency in the diagnosis of PVD.

PVD occurs in upwards of two out of three individuals over the age of 65, and is defined as the separation of the dense outer layer of type II collagen fibrils of vitreous, known as the posterior vitreous cortex, from the internal limiting lamina of the retina.<sup>1</sup> The separation begins at the posterior pole and, when uneventful, results solely in the phenomenon of 'floaters'. If, as the separation progresses anteriorly, a site of firm vitreoretinal adherence is encountered, retinal tears and detachment can result from traction upon the peripheral retina. If the separation is anomalous, the posterior vitreous cortex can split, so called 'vitreoschisis', and the outer layer can remain attached to the retina, a phenomenon which probably plays a role in the pathogenesis of macular pucker, PDVR,<sup>4</sup> and possibly macular holes.<sup>5</sup> Considering the impact of these disorders on vision, a better understanding of the role of PVD is imperative. Such an understanding has long been hampered by the lack of uniformity in characterising PVD in the clinical setting.

Kakehashi and colleagues have now developed a classification system, based upon clinical examination by preset lens biomicroscopy,<sup>6</sup> to distinguish different types of PVD and have found a good correlation with certain disor-

ders. Patients who had complete PVD with collapse of the detached vitreous were most commonly either aged or highly myopic. In fact, this type of PVD was found in over 80% of high myopes. Complete PVD without collapse was typical of patients with uveitis or central retinal vein occlusion. Partial PVD with a thickened posterior vitreous cortex was found most commonly in PDVR, where over 90% of such patients had this form of PVD. Partial PVD without thickening was most commonly encountered in aging.

Thus, it appears that this classification system offers an effective means by which to characterise different manifestations of PVD. Insofar as this approach has a reasonably good correlation with different disease states, the classification may prove useful in furthering our understanding of the role of vitreous in various disorders. It may also provide an effective means to evaluate the efficacy of techniques to induce or prevent PVD. Although the authors close by stating that their classification is 'precise', it must be emphasised that their approach is only as precise as permitted by the current methods of examination. Ongoing investigations of laser based methods such as dynamic light scattering,<sup>7</sup> fluorescence reflectometry, and Raman spectroscopy<sup>8</sup> will in the future provide non-invasive vitreous examination of a truly precise nature.

J SEBAG

18821 Delaware Street, Suite 202,  
Huntington Beach, CA 92648, USA

- 1 Sebag J. *The vitreous—structure, function, and pathobiology*. New York: Springer-Verlag, 1989.
- 2 Retina Society Terminology Committee. The classification of retinal detachment with proliferative vitreoretinopathy. *Ophthalmology* 1983;**90**: 121-5.
- 3 Kroll P, Meyer-Rüsenberg HW, Busse H. Vorschlag zur Stadieneinteilung der proliferativen diabetischen Retinopathie. *Fortschr Ophthalmol* 1987;**84**: 360-3.
- 4 Sebag J. Diabetic vitreopathy. *Ophthalmology* 1996;**103**:205-6.
- 5 Sebag J, Wendell R, De Bustros S. Disorders of the vitreo-macular interface. In: Margo C, Hamed L, Mames R, eds. *Diagnostic problems in clinical ophthalmology*. Philadelphia: WB Saunders, 1994:556-62.
- 6 Takehashi M, Trempe CL, Schepens CL. Biomicroscopic evaluation and photography of posterior vitreous detachment. *Arch Ophthalmol* 1980;**98**: 665-8.
- 7 Sebag J, Dunker S, Suh KI, Ansari RA. Dynamic light scattering measurements in vitreous. *Invest Ophthalmol Vis Sci* 1997;**38**(4):S662.
- 8 Sebag J, Nie S, Reiser KA, Charles MA, Yu NT. Raman spectroscopy of human vitreous in proliferative diabetic retinopathy. *Invest Ophthalmol Vis Sci* 1994;**35**:2976-80.