B-SCAN ULTRASONOGRAPHIC FINDINGS IN THE STAGES OF IDIOPATHIC MACULAR HOLE*

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ABSTRACT

Purpose: To prospectively evaluate the relationship between the posterior hyaloid membrane (PHM) and the retina in eyes with idiopathic macular hole.

Methods: Ninety-four eyes of 94 consecutive patients with macular hole underwent complete ophthalmologic examination, contact lens biomicroscopy, and B-scan ultrasonography and/or vitreoretinal surgery.

Results: In 93 of 94 patients (99%), the relationship between the PHM and posterior retina could be visualized during echographic examinations or at surgery. Among these 93 patients, the PHM was detectable biomicroscopically in 36 (39%). Persistent PHM attachment to the foveola with partial separation of the PHM from the perifoveal retina was evident with ultrasonography in 5 of 6 patients (83%) with stage 1 hole and in 12 of 18 patients (67%) with stage 2 hole. When axial views were included, separation of the PHM from the perifoveal retina was evident in 13 of 13 patients (100%) with stage 1 and stage 2 hole. Separation of the PHM from the fovea and perifoveal retina with attachment to the peripapillary retina was evident with ultrasonography in 65 of 65 patients (100%) with stage 3 macular hole and pseudo-operculum and was evident biomicroscopically in 22 of the 65 patients (34%) in this group.

Conclusions: These findings suggest that high-resolution axial and paraxial ultrasonographic examination directly on the surface of the eye is more sensitive in detecting separation of the PHM from the retina than biomicroscopy in idiopathic macular holes. The perifoveal detachment of the PHM may be involved in the pathogenesis of macular holes.

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INTRODUCTION

Since Gass described the early stages of idiopathic macular hole,[1] the ophthalmic scientific literature has contained many articles about the diagnosis and treatment of macular holes. In the absence of a pseudo-operculum or operculum, the relationship of the transparent detached posterior hyaloid membrane (PHM) to the stages of macular hole was not included in this landmark clinical description or in its reappraisal.[2] This omission may have been due to the difficulty in visualizing the PHM, even with meticulous contact lens biomicroscopy. Recent studies by our group (unpublished data) and others using B-scan ultrasonography,[3,4] and optical coherence tomography (OCT),[5,6] have described localized vitreomacular separations. For the purposes of this study, we have prospectively evaluated the vitreoretinal relationship with B-scan ultrasonography in eyes presenting with idiopathic macular holes.

SUBJECTS AND METHODS

Ninety-four patients with evolving or full-thickness idiopathic macular holes were evaluated stereoscopically with a slit lamp and fundus contact lens and B-scan ultrasonography and/or operating microscope. The macular holes were classified according to Gass[2] using stereoscopic slit lamp and fundus contact lens examination.

The B-scan examinations were performed with 1 or more instruments: the I^3 unit (Innovative Imaging, Inc, Sacramento, Calif), the Ophthascan S unit (Alcon Surgical, Inc, Fort Worth, Tex), or the Humphrey A/B-scan (Zeiss-Humphrey Instruments, Inc, San Leandro, Calif). After topical anesthetic was administered, the coupling agent methylcellulose was applied to the probe. The probe was placed on the open eye over the nasal limbal conjunctiva or on the cornea opposite the macula, keeping the vitreoretinal echo as perpendicular to the fovea as possible. Longitudinal, transverse, axial, and

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paraxial sections through the macula were studied by using both high and medium gain settings. Dynamic (kinetic) B-scan assessments were recorded on videotape. Static images were printed or photographed. Localized vitreous detachment was diagnosed when a thin, smooth, continuous echodense membrane with minimal movement was detected anterior to the retina. The intraoperative identification of a perifoveal separation of the PHM is made after completion of the core vitrectomy, in cases without vitreous separation from the optic nerve, and consists of visualization of an intact convex curvilinear elevation of an essentially transparent membrane surrounding the fovea. The intraoperative identification of vitreofoveolar adhesion is made when no separation between the PHM and fovea is observed and when the fovea is distorted by traction on the attached PHM.

RESULTS

The patients included 29 men and 65 women whose mean age was 68.3 years (range, 36 to 84 years at the time of the initial examination). All 5 patients in our study who were younger than 60 years of age had myopia of 5 diopters or greater. All patients presented with symptoms that included decreased vision, distortion, or small central spots; patients reported mean symptom duration of 2 months. Presenting visual acuity ranged from 20/20 to 20/400, with a median acuity of 20/80. Twenty-nine (31%) had a stage 2, 3, or 4 macular hole in the fellow eye at presentation.

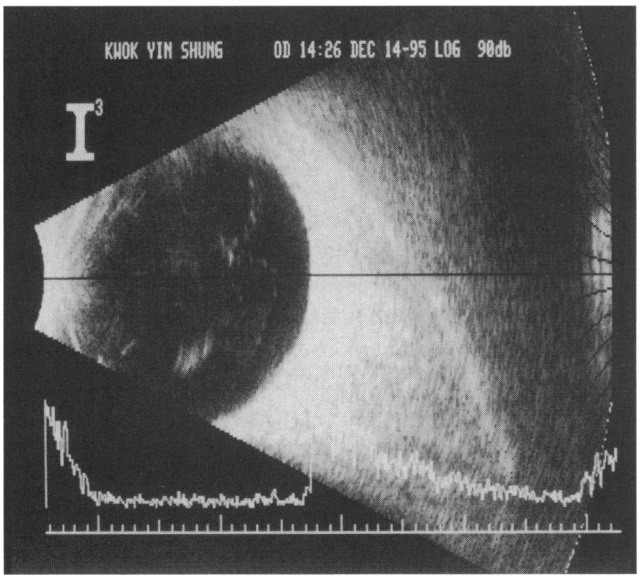

FIGURE 1
Ultrasound of eye with stage 4 macular hole showing echodense linear pattern of posterior hyaloid membrane extending from Weiss ring and variable echolucent space.

STAGE 4 RESULTS

A complete posterior vitreous detachment (PVD), including separation of the vitreopapillary attachment, was present in all 10 eyes with stage 4 macular hole and was evident with both stereoscopic biomicroscopy and B-scan ultrasonography. A mobile prepapillary glial ring (Weiss ring) was visible anterior to the retina both biomicroscopically and echographically. On ultrasound, the attached echodense linear pattern of the PHM extending from the Weiss ring and a variable echolucent space were more noticeable with kinetic assessment (Fig 1).

STAGE 3 RESULTS

This group of 33 patients presented with definite biomicroscopic appearance of a full-thickness stage 3 macular hole, with a visible pseudo-operculum or operculum suspended anterior to the macular hole and no visible Weiss ring. Although the PHM was not visible with stereoscopic fundus contact lens examination in these cases, it was assumed to be separated from the macula because of the suspended nature of the opercula and/or pseudo-opercula in front of the fovea.

In all patients, an echo dense opacity was suspended anterior to the retinal surface and attached to a thinner, less echo dense linear opacity that appeared to connect to the prepapillary glial tissue nasally (Fig 2). The echo dense line was slightly separated from the papillomacular bundle and was separated from the retina by a larger variable distance temporal, superior, and inferior to the fovea.

PSEUDO-OPERCULUM RESULTS

Stereoscopic fundus contact lens examination showed an intact macula with a visible focal condensation or pseudo-operculum suspended anterior to the fovea in the 22 reported elsewhere.[4]

The echographic findings were identical to those in stage 3 macular hole. It was not possible to distinguish an operculum from a pseudo-operculum with echography.

STAGE 2 RESULTS

Details have been submitted elsewhere.[7] Briefly, in the 20 patients classified with stage 2 macular hole, the PHM was visible with fundus contact lens biomicroscopy examination in 4 (20%) of the 20 patients and an operculum was visible in 7 (35%) of the 20 patients. In 1 patient, both an operculum and the PHM were visible; therefore, a total of 10 (50%) of the 20 patients with stage 2 macular hole had at least PHM or an operculum visible with biomicroscopy.

Echographic findings were available for 18 of the 20

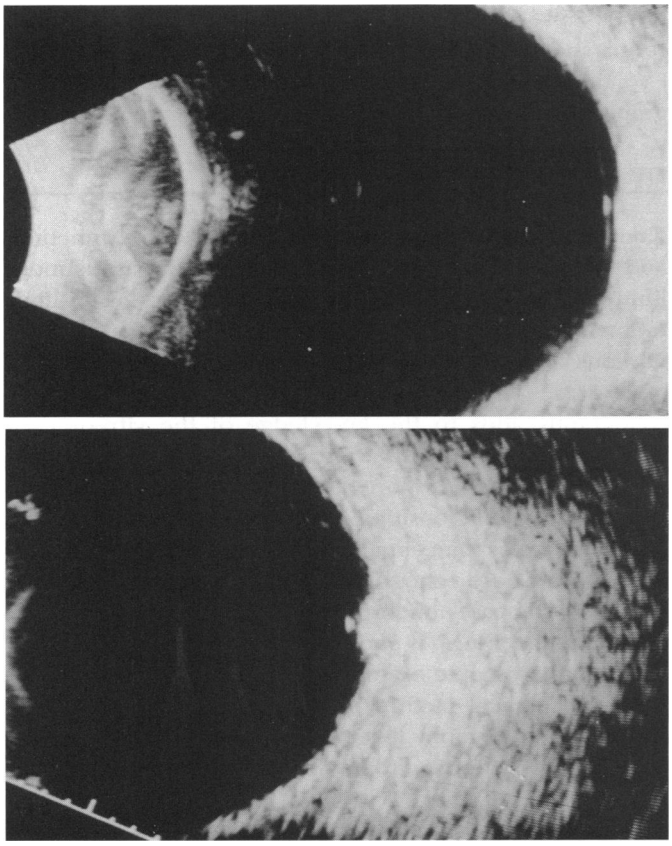

FIGURE 2
Top, medium gain axial view of eye with stage 3 macular hole showing echodense opacity suspended anterior to retinal surface and attached to thinner, less echodense linear opacity. Bottom, low gain longitudinal view of operculum overlying macular hole with rim of subretinal fluid.

patients with stage 2 macular hole (Fig 3). In 12 (67%) of the 18 patients, a visible PHM was separated from the perifoveal retina. When axial and/or paraaxial views were included in the echographic study, the PHM separation from the perifoveal retina was observed in 8 (100%) of 8 patients with stage 2 macular hole. Of the 12 patients with echographically visible PHM, B-scan demonstrated vitreofoveolar adhesion in the 7 patients (58%) without an operculum.

Operative findings in the 20 patients with stage 2 macular hole included a perifoveal separation of the PHM from the retina in 19 (95%). Vitreofoveolar adhesion was observed intraoperatively in 10 (50%) of the 20. One patient had had a spontaneous separation of the PHM from the foveola after initial examination and before surgery.

STAGE 1 RESULTS

Details have been submitted elsewhere.[7] A focal detachment of the fovea was observed with contact lens biomicropscopic examination and classified as either stage 1A or

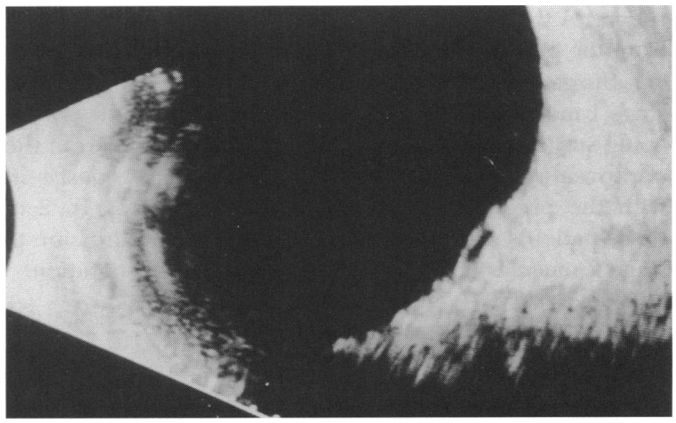

FIGURE 3
Echographic findings in eye with stage 2 macular hole showing visible posterior hyaloid membrane separated from perifoveal retina.

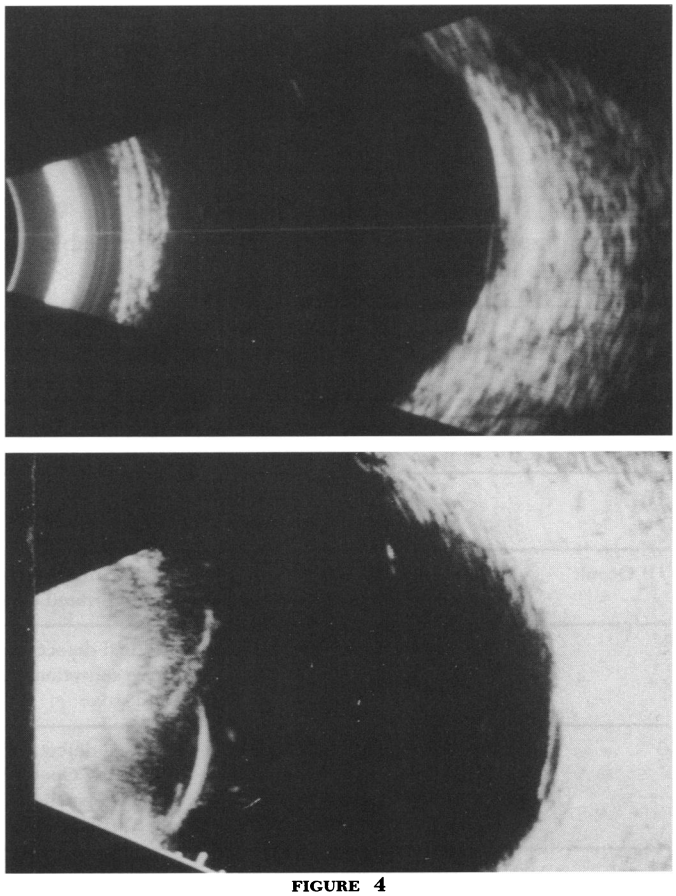

FIGURE 4
Top, image of stage 1 macular hole shows a longitudinal view of a thin echodense linear opacity attached to elevated fovea. Bottom, axial view of stage 1 macular hole.

1B macular holes in all 6 patients (Fig 4). The PHM was not biomicroscopically visible in any of the 6 eyes.

In 5 (83%) of the 6 eyes, B-scan ultrasonography revealed a localized separation of PHM from the perifoveal

retina. In the remaining patient, a separation of the PHM from the perifoveal retina was demonstrated at surgery.

Surgery was performed on 4 of the 6 patients with stage 1 macular hole. The findings in all 4 patients undergoing surgery revealed a separation of the PHM from the perifoveal retina and a vitreofoveolar adhesion, consistent with the preoperative B-scan ultrasonography. In 2 of these patients surgery was performed after progression to stage 2 macular hole, 1 month after initial presentation.

OTHER COMMON ECHOGRAPHIC FINDINGS

In eyes with stage 1, 2, and 3 macular hole and pseudoopercula, 4 consistent echographic observations were made: (1) the PHM remained attached to the peripapillary retina, (2) the long anterior-posterior vitreous fibers moved freely and appeared to be disinserted from the PHM in the macular area, (3) the long anterior-posterior vitreous fibers remained inserted in the peripapillary vitreous ring area, and (4) a localized perifoveal separation of the PHM from the retina was present, which was tethered to the optic disc margin and variably separated from the retina temporally.

In the 10 eyes with stage 4 macular hole, the Weiss ring and the operculum were consistently identifiable on the collapsed PHM as they moved freely anterior to the retina and optic nerve.

Table I summarizes the common clinical and echographic findings in idiopathic macular hole.

DISCUSSION

Knowledge and understanding of the structure, function, and relationship of the vitreous to ocular disease is more limited than that of any other ocular structure. This limitation is largely due to clinical difficulties in visualizing the vitreous and the lack of effective laboratory methods to study it.[5] This report demonstrates that B-scan ultrasonography adds to the knowledge of the vitreoretinal relationship in eyes with idiopathic macular hole. B-scan ultrasonography was more sensitive and more specific in detecting the relationship of the PHM to stages 1, 2, and 3 macular holes than clinical stereoscopic slit-lamp biomicroscopy.[7] These echographic findings were usually confirmed in the cases undergoing surgery. Although visualization of the PHM is more apparent with B-scan than with biomicroscopic examination in all stages of macular hole, the clinical picture of stage 4 provides clues that make ultrasonography less helpful.

Optical coherence tomography has illustrated similar

TABLE I: COMMON CLINICAL AND ECHOGRAPHIC FINDINGS IN IDIOPATHIC MACULAR HOLE*

STAGE	PATHOLOGY	BIOMICROSCOPY	FLUORESCEIN ANGIOGRAPHY	B-SCAN ULTRASONOGRAPHY
1A	Focal detachment of fovea	Yellow dot	No or faint hyperfluorescence	Perifoveal PHM detachment, Vitreofoveal adherence
1B	Larger foveal detachment	Yellow ring, maybe retinal striae	No or faint hyperfluorescence	Perifoveal PHM detachment, Vitreofoveal adherence
1B Occult	Retinal hole hidden by semiopaque vitreous cortex	Yellow ring, may have serrated edge	No or faint hyperfluorescence	Perifoveal PHM detachment, Vitreofoveal adherence
2	Full-thickness macular hole	Retinal defect, may have subretinal fluid and striae	Hyperfluorescent spot	PHM detachment over macula, PHM attached at optic nerve, ±vitreofoveal adherence
3	Full-thickness macular hole, Usually with an operculum or pseudoperculum	Retinal defect with overlying Operculum, yellow opacities at RPE level	Hyperfluorescent spot	PHM detachment over macula PHM attached at optic nerve larger separation temporal, echodense operculum on PHM
Pseudoperculum	Attached intact fovea with separated semi-opaque vitreous	Focal condensation overlying fovea, may cast yellow spot	Normal	PHM detachment over macula, PHM attached at optic nerve, larger separation temporal, echodense pseudoperculum on PHM
4	Full-thickness macular hole	Complete PVD, full-thickness macular hole defect	Hyperfluorescent spot	Complete PVD, echodense operculum on collapsed PHM near Weiss ring raised cuff of macular hole

PHM, posterior hyaloid membrane; PVD, posterior vitreous detachment; RPE, retinal pigment epithelium. *Adapted from Gass.*[2]

perifoveal PHM separation from the internal limiting membrane (ILM) in stage 1 and stage 2 macular holes.[5] The echographic pattern of the PHM in this study and the discrete linear signal (DLS) of the OCT appear to be an image of the same structure. These data make it unlikely that this pattern is an artifact.

In a study by Chauhan and associates,[6] report that OCT demonstrated a DLS in 2 cadaver eyes consistent, wholly or in part, with the posterior cortical vitreous or PHM. In their study of 70 eyes in 35 patients with macular hole, the OCT depicted a DLS from the PHM in 40 eyes (57%) compared with biomicroscopic findings of visible PHM in 16 (23%). Of their 9 patients with bilateral macular holes, 3 patients showed a DLS with a prefoveolar opacity and no Weiss ring. The failure to observe a DLS in the remaining 6 patients with bilateral macular holes may indicate that B-scan ultrasonography is more sensitive than OCT. The images of OCT in stage 1 and stage 2 macular holes are impressive cross-sectional views. The dynamic B-scan images are small and somewhat fleeting and require precise probe positioning. Current OCT technology visualizes the PHM within 2 mm of the retina and is not capable of detecting a PVD in stage 4 macular holes or in other stages where the PHM is further than 2 mm from the retina.

The reports of other methods designed recently to aid in clinical study of the vitreous–preset lens biomicroscopy[9] and scanning laser ophthalmoscope vitreous videography[10]–do not include evaluations of idiopathic macular hole. Macular holes are believed to result from to changes in the vitreoretinal interface, which includes the PHM and the ILM. The almost transparent PHM consists of densely packed collagen fibrils and some flattened spindle-shaped hyalocytes and is 100 to 110 µm thick except over the fovea, where it is thicker.[8] The ILM is principally the basement membrane of the Müller cells. The inner portion of the ILM is the basal lamina proper, the lamina densa, which consists of interlacing, thin, unbranching filaments embedded in a matrix of mucopolysaccharide.[11] Vitreous fibers appear to attach to the lamina densa; however, this attachment may be biochemical.[12,13] A thin electron-lucent space, the lamina rara, is posterior to the lamina densa and is traversed by delicate fibrils, which when present in increased number, are adjacent to attachment plaques in the cytoplasm of the Müller cell. These attachment plaques are present in greater number in areas of clinically known strong vitreoretinal adhesion, such as the vitreous base and possibly the fovea. The ILM varies in thickness and is thinner in areas of strong vitreoretinal adhesion, such as the vitreous base and fovea.[14] The thinness of ILM and the presence of attachment plaques may make the fovea vulnerable to macular hole formation.

The significance of the apparent disinsertion of the long anterior-posterior vitreous fibers from the macular PHM that was observed in this study is not clear. However, these separated fibers may explain the described premacular bursa.[15] In this study, the echographic appearance of the PHM appeared to be intact although variable echoes resulted from folding of the PHM when it was more anterior to the retina, especially temporal to the macula, in stage 3 and in stage 4 holes. Large defects were not observed in the PHM with either biomicroscopy or ultrasonography in this study. The possibility of small defects in the PHM is suggested by the apparent slow accumulation of sub-PHM fluid. An increase in the apparent size of the perifoveal PVD and the elevation of the PHM from the retina was observed with serial echography in 2 patients progressing from stage 1A to 1B macular hole and in 1 patient progressing from stage 1 to pseudo-operculum stage. Three patients with stage 1 impending macular holes observed with OCT have been reported to have progressed to stage 2 macular hole.[16,17] Although 4 patients in this study progressed from stage 3 to stage 4 macular hole, such progression appears to be an infrequent occurrence; the majority of patients appear to maintain the vitreopapillary attachment after full-thickness macular holes occur. If the vitreofoveolar adhesion separates or a small macular hole closes spontaneously, the echographic pattern of pseudo-operculum stage usually is stable but occasionally changes owing to complete posterior vitreous detachment. It is conceivable that a large PHM defect may be required for a sufficient volume of liquefied vitreous to abruptly elevate the PHM sufficiently to cause separation of the vitreopapillary attachment, resulting in a "rhegmatogenous vitreous detachment."[18]

VITREORETINAL SEPARATION

With use of biomicroscopy, complete PVD has been reported in nearly two thirds of otherwise healthy adults between 66 and 86 years of age.[19] However, a recent kinetic B-scan ultrasonography study of 712 essentially healthy eyes in 404 patients revealed a complete PVD in 12 of 105 eyes (11%) in the 65- to 69-year-old age-group and in 56 of 121 eyes (46%) in the 80- to 89-year-old age-group.[20] In our study, where the mean age was 68 years, 10 patients (11%) had a complete PVD. This finding may indicate that biomicroscopy overestimates the rate of complete PVD.

Our prospective study includes 22 patients with pseudo-operculum who were examined with B-scan echography at the start of the study. Of the 29 patients previously reported to have pseudo-operculum, 20 patients had full-thickness macular holes in their fellow eyes.[4] These fellow

eyes are not included because B-scan data were unavailable in 8 of the 20 fellow eyes with macular hole. Of the 12 eyes studied with B-scan, however, the echographic patterns were consistent with the data reported here. Separation of the vitreous cortex from the ILM usually occurs at the vitreoretinal juncture.[21] However, ILM remnants have been observed on the detached posterior cortical vitreous,[22,23] and cortical vitreous has been reported attached to the surface of the ILM after PVD.[24] Although data regarding the frequency of incomplete separation of the posterior cortical vitreous from the ILM are not available, and the incidence of avulsed ILM attached to posterior cortical vitreous remains unknown, both of these conditions exist. The echographic pattern of an avulsed ILM is not known, but this condition could result in confusion with the PHM. Similarly, a schisis of the PHM could be present, creating an echographic pattern that could be confused with the intact PHM.

PATHOGENESIS

The principal hypothesis proposed by Gass for macular hole formation has been tangential traction due to shrinkage of the prefoveal vitreous cortex.[1] Controversy exists regarding the presence or absence of fibrocellular and cellular membrane fragments in vitreous specimens collected at vitrectomy for macular holes. The absence of fibrocellular and cellular membrane fragments reduces the likelihood that cellular contracture is the explanation for traction on the PHM or the fovea.[25] Other hypotheses include cystic degeneration theory,[13] dynamic vitreous traction generated by ocular movement,[13] strong vitreofoveolar adhesion with trampoline-like PHM detachment,[7] and anteronasal papillofoveal traction.[6] These data seem to favor traction on the fovea induced by fluid elevation of the PHM–in other words, a similar mechanism that is presumed to cause round peripheral retinal holes.

At least 3 main variables influence the outcome of the foveolar traction in the pathogenesis of macular holes. One is the degree and direction of traction on the vitreofoveolar adhesion. The elevation of the PHM from the retina may be caused by localized perifoveal fluid, which presumably is similar to subretinal fluid. This elevation of the PHM may generate anterior-posterior traction on the fovea. The vitreous attachment to the optic nerve is much stronger than the attachment to the fovea, and it may limit the elevation of the PHM in the papillomacular area. These data show a frequently greater echographic elevation of the PHM temporal, inferior, and superior to the fovea. This difference in PHM elevation is the most probable explanation of why axial and transverse echographic sections through the macula show the PHM detachment better than do typical longitudinal sections, which include the optic disc and fovea. The other 2 variables not measured by these data but that influence the outcome include the vitreofoveolar adhesion and the ability of the retina to withstand injury or self-repair after vitreofoveolar traction.

CONCLUSION

High-resolution B-scan ultrasonography is commonly available to ophthalmologists around the world. If used with the proper technique, this technology may improve understanding of the relationship between the PHM and the retina in idiopathic macular holes. B-scan ultrasonography is superior to a routine slit-lamp evaluation of the macula and may be more sensitive than OCT in detecting the relationship between the PHM and macular holes. The elevated PHM may generate sufficient traction on the fovea to cause macular holes if the vitreofoveolar adhesion is stronger than the retinal resistance to tear.

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DISCUSSION

DR PAUL E. TORNAMBE. This exhaustive prospective study by VanNewkirk, Johnson, Hughes, Meyer and Byrne evaluated 94 eyes with a detailed clinical examination by premier retinologists, ultrasound examinations by world class technicians, and confirmatory intraoperative observations by pioneers of macular hole surgery. Truly a star-studded cast.

They conclude the ultrasound examination more sensitive than the clinical examination in determining the relationship of the posterior hyaloid membrane (PHM) to the macula, fovea and optic nerve. Their data supports this conclusion. I find this exciting, not only regarding macular holes, but also for other diseases which may involve the vitreomacular interface such as cystoid macular edema, diabetic macular edema, and edema associated with tributary vein occlusions.

The authors' primary conclusion, that the ultrasound evaluation is more sensitive than the clinical evaluation to determine the status of the posterior hyaloid is probably correct, but not surprising. The posterior hyaloid is transparent and the vitreous body may contain large syneretic cavities, which makes visual interpretation confusing.

Using the ultrasound findings, the authors'go 1 step further, concluding that perifoveal traction by the posterior hyaloid membrane is responsible for the pathogenesis of macular holes. It is with their second conclusion that I have a few questions, observations and yet another theory regarding macular hole formation. No one really knows why a macular hole forms. Certainly traction must play a role, but their theory does not completely explain some clinical observations. Macular holes sometimes develop after a posterior vitreous detachment (PVD) has occurred. The incidence of macular hole formation is low in the fellow eye if a PVD is present; but the incidence is not zero. If vitreous attachments to the macula are severed, how can the author's theory explain how eyes with a Weiss ring later develop a macular hole?

We may be too simplistic when we think of a PVD as a pure split of the vitreous from the retina, a separation of the hyaloid from the internal limiting membrane (ILM). Anatomists note the Internal Limiting Membrane of the retina is also the Outer Limiting Membrane of the vitreous. Remnants of the ILM have been found on the detached posterior hyaloid face and remnants of the posterior hyaloid have been found on the ILM. Could these remnants play a role in the pathogenesis of a macular hole?

Sebag[1], using dark field illumination of the vitreous body, demonstrated vitreous fibrils inserting into the macula through a dehiscence of the posterior cortical vitreous over the macula (Fig 1). If localized traction occurs above the macula, these fibrils may partially separate from the ILM, leaving vitreous remnants behind on the surface of the macula. Fibrous metaplasia might form a secondary membrane on the ILM, which could contract forming a foveal dehiscence, a macular hole.

This epimacular membrane could contract while the vitreous still adheres to the nerve head (Stage II, III hole) or could contract after the vitreous has separated from the nerve head. (Stage IV). This would explain how a macular hole could form in an eye, which has a Weiss ring, the hallmark of a complete PVD. (Fig 2)

The schisis of the PHM may not completely separate from the retinal surface in the perifoveal region. This incomplete separation, I believe is what the authors have ultrasonically interpreted as attachments of the PHM in the perifoveal region. (Figure 2B) I don't believe it is this attachment, per se, which results in hole formation, but rather the contraction of the tissue left behind on the surface of the ILM. This membrane is transparent and adherent to the ILM. There are no interfaces therefore it is invisible to ultrasound and OCT. It is also invisible to the clinical examiner and even the surgeon, unless techniques are used to identify it. A new technique I have been using recently involves intraoperative ICG staining of the retinal surface during vitrectomy surgery.

If ICG dye is injected into the vitreous cavity after the

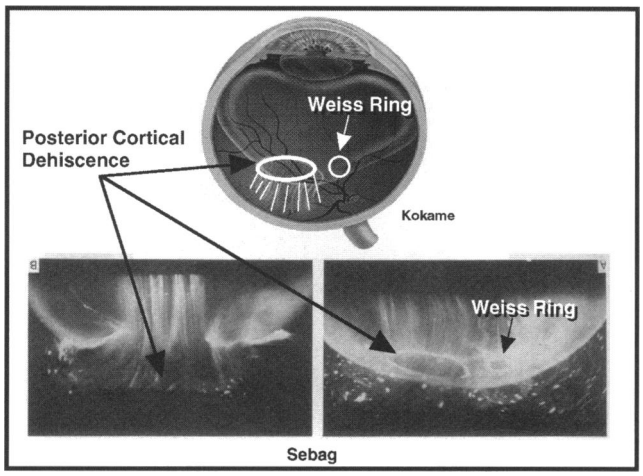

FIGURE 1

Top drawing (courtesy Kokame)and, bottom drawing dark-field slit illumination from Sebag[1] show a broad insertion of vitreous through a dehiscence in posterior cortical hyaloid over macula and in area of Weiss ring.

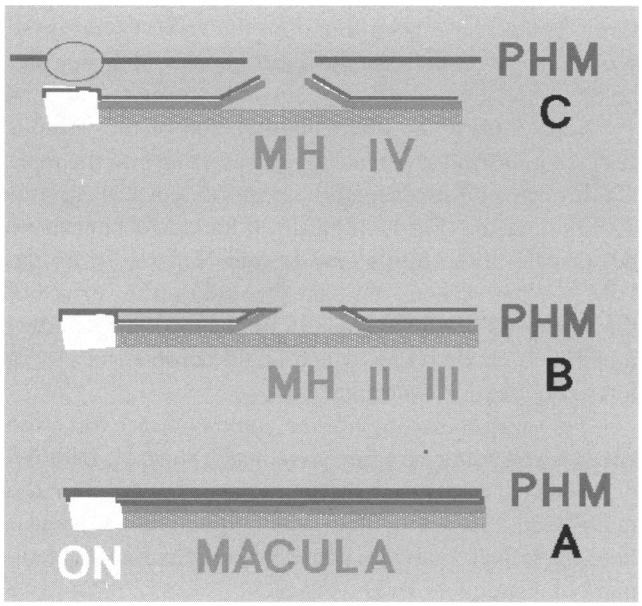

FIGURE 2

Relationship of Posterior Hyaloid Membrane (PHM) to macula and optic nerve. (A) PHM is attached to macula and optic nerve. (B) PHM is separating from surface of macula. PHM is splitting; membrane lying on surface of retina is composed of remnants of posterior hyaloid and secondary cellular proliferation. Contraction of this membrane forms hole. Part of PHM separated from surface of retina is still attached to perifoveal macula and gives ultrasound appearance that this membrane caused macular hole. PHM is still attached to optic nerve (Stage II, III hole). (C) PHM is completely separated from ON and macula. Remnants of PHM still on surface of retina may contract forming a macular hole even after PHM has completely separated from retina (Stage IV hole).

posterior hyaloid is stripped, a green membrane stains in the posterior pole and peripapillary retina. I believe this stained material represents ILM, remnants of the vitreous, and epimacular membranes. During removal, the stained tissue adheres to the edges of the hole. This membrane has a silk-like appearance, and is not simply ILM, which is more rigid. Histopathology shows a cellular proliferation on the surface of the ILM. This membrane can frequently be stripped off the ILM with the Tano diamond dusted scrapper. Those who have noted higher macular hole closure rates using ILM peeling are likely removing the ILM along with this membrane.

I have used ICG in more than 60 vitrectomy operations for an assortment of retinal diseases. No similar perifoveal adhesions were noted with other macular conditions including macular pucker, proliferative vitreoretinopathy, proliferative diabetic retinopathy, background diabetic retinopathy, or cystoid macular edema. It appears that this posterior hyaloid schisis membrane is adherent in the foveal region only in eyes with macular holes. This adhesion may be a genetic trait. In 11 eyes with macular holes operated upon with ICG enhanced membrane removal, all holes closed, without face down positioning.

In summary, the authors do make a good case regarding the superior sensitivity of detecting the PHM over the clinical examination; however, I don't feel their theory explains macular hole formation. I wish to congratulate the authors and hope they will continue to explore this area and expand their investigation to other diseases that involve the vitreoretinal interface.

REFERENCES

1. Sebag, J. *The Vitreous. Structure, Function, and Pathobiology.* New York: Springer-Verlag, 1989 Chapter 4, pg42 1989s
2. Kokame GT, Macular traction associated with a Premacular Circular Dehiscence of the Detached Posterior Vitreous Cortex. *VitreoTech* Vol 5:1 2000

DR D. JACKSON CELEMAN. Mylan is an excellent fly fisherman. Although one can usually rely on a fisherman's count, one does not often hear the techniques that were used. One of the most important aspects of this paper was the effective utilization of new ultrasonic techniques that are evolving rapidly. The use of digital as well as dynamic evaluation techniques are very important. Until recently the use of high frequency techniques has been limited to the anterior segment, but we can now use these for looking at the back of the eye. We can make measurements of tissue thickness to within one micron. Therefore I thank Mylan for showing that ultrasound is as useful as biomicroscopy for looking at the macula, for pointing out that the ultrasonic techniques are evolving and are giving us more and more useful information, and that in the future the combination of ultrasound and OCT are going to make it easier for the surgeon. My question is how does

this benefit the surgeon at the present time?

DR HANS E. GROSSNIKLAUS. Congratulations on a fine paper. How did your study help you to understand the role of tangential vs. anterior-posterior traction in the pathogenesis of idiopathic macular hole formation?

DR MYLAN R. VANNEWKIRK. Thank you; those are excellent questions and comments. Paul's comments are valid. The complexity of the vitreo-retinal junction is beyond my present knowledge and understanding. There are many possibilities including schisis of the posterior hyaloid and schisis of the internal limiting membrane. Histopathology has shown tissue adherent to both of these structures in different situations.

Jack, thank you for your comments. As a vitreous surgeon, the benefit of B-scan ultrasonography has been a better understanding before surgery of the vitreo-retinal anatomy. I encourage every vitreous surgeon to do his or her own at echography because this enables one to better prepare for the case.

Finally, Hans, my experience with these cases has shown that there is an increase in the size of the perifoveal vitreous separation. Without question in many of these cases the anterior-posterior traction is exerted by the accumulation of fluid beneath the posterior hyaloid membrane. I have had a difficult time convincing Don Gass of this idea and have still not been entirely successful. However, he does recognize that this perifoveal detachment exists.