

The International Vitreomacular Traction Study Group Classification of Vitreomacular Adhesion, Traction, and Macular Hole

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Objective: The International Vitreomacular Traction Study (IVTS) Group was convened to develop an optical coherence tomography (OCT)-based anatomic classification system for diseases of the vitreomacular interface (VMI).

Design: The IVTS applied their clinical experience, after reviewing the relevant literature, to support the development of a strictly anatomic OCT-based classification system.

Participants: A panel of vitreoretinal disease experts was the foundation of the International Classification System.

Methods: Before the meeting, panel participants were asked to review 11 articles and to complete 3 questionnaires. The articles were preselected based on searches for comprehensive reviews covering diseases of the VMI. Responses to questionnaires and the group's opinions on definitions specified in the literature were used to guide the discussion.

Main Outcome Measures: Optical coherence tomography-based anatomic definitions and classification of vitreomacular adhesion, vitreomacular traction (VMT), and macular hole.

Results: Vitreomacular adhesion is defined as perifoveal vitreous separation with remaining vitreomacular attachment and unperturbed foveal morphologic features. It is an OCT finding that is almost always the result of normal vitreous aging, which may lead to pathologic conditions. Vitreomacular traction is characterized by anomalous posterior vitreous detachment accompanied by anatomic distortion of the fovea, which may include pseudocysts, macular schisis, cystoid macular edema, and subretinal fluid. Vitreomacular traction can be subclassified by the diameter of vitreous attachment to the macular surface as measured by OCT, with attachment of 1500 μm or less defined as focal and attachment of more than 1500 μm as broad. When associated with other macular disease, VMT is classified as concurrent. Full-thickness macular hole (FTMH) is defined as a foveal lesion with interruption of all retinal layers from the internal limiting membrane to the retinal pigment epithelium. Full-thickness macular hole is primary if caused by vitreous traction or secondary if directly the result of pathologic characteristics other than VMT. Full-thickness macular hole is subclassified by size of the hole as determined by OCT and the presence or absence of VMT.

Conclusions: This classification system will support systematic diagnosis and management by creating a clinically applicable system that is predictive of therapeutic outcomes and is useful for the execution and analysis of clinical studies.

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The vitreous body is the largest structure within the human eye, yet until recently, no method was widely available for adequately visualizing and evaluating the vitreomacular interface (VMI). The evolution from slit-lamp examination to ocular imaging with optical coherence tomography (OCT) has resulted in a variety of competing, and sometimes contradictory, definitions and systems for the classification of VMI diseases such as vitreomacular traction (VMT) and macular holes. Multiple studies have proposed using OCT-based findings to characterize and define VMI conditions; however, there is currently no consensus on their definition and classification, which hinders clinical practice, consistent reporting, and the evaluation of potential therapies to treat these conditions.¹⁻⁴

The emergence of OCT more than 2 decades ago dramatically altered the landscape of vitreoretinal diagnosis by enabling physicians to visualize and monitor the VMI with greater consistency and accuracy than ever before. The capabilities afforded by such advances in imaging technologies, together with the introduction of new therapies, creates a unique opportunity to develop a unifying evidence-based OCT-derived scheme to define and classify the range of diseases affecting the VMI.

Methods

A panel of vitreoretinal disease experts, the International Vitreomacular Traction Study (IVTS) Group, was convened to develop

a consensus system for the classification of diseases of the VMI. The classification system presented herein is meant to facilitate systematic evidence-based identification, monitoring, and management of VMI diseases. The panel met with the goal of creating a system that is simple, easy to remember, evidence based, clinically applicable, predictive of surgical outcomes, and useful for the execution and analysis of clinical studies.

Before the meeting, panel participants were asked to review and respond to 3 sets of materials that included a survey that was used to gauge opinions regarding the classification and staging of VMA, VMT, macular hole, lamellar hole, and macular schisis; 11 journal articles containing published statements regarding diseases of the vitreoretinal interface; and 3 questionnaires consisting of open-ended questions regarding overall impressions of the articles and their usefulness in classifying and staging diseases of the vitreoretinal interface. Articles were selected first based on PubMed searches for comprehensive reviews covering diseases of the VMI. Search terms included *vitreomacular adhesion* (VMA), *macular hole*, and *full-thickness macular hole* (FTMH). From this first round of searches, articles were selected based on degree of relevance as determined by the roundtable chairpersons. Responses to questionnaires, and the group's opinions on definitions specified in the literature, were used to guide the discussion and the composition of the manuscript.

Vitreomacular adhesion, VMT, and FTMH were defined by anatomic criteria that must be present on at least 1 OCT B-scan image and classified by size of attachment or lesion and presence of concomitant retinal or vitreoretinal conditions. Most of the entities in the classification scheme rely on 1 OCT B-scan image to make the diagnosis. However, if only 1 or just a few line scans are looked at, it is possible to miss vitreous attachments or small holes (false negatives). Looking at multiple line scans or the entire cube scan through the macula is recommended to be certain of the presence or absence of the pathologic features.

Natural History of Vitreous Aging

The vitreous consists of approximately 98% water and 2% structural macromolecules.^{5,6} These molecular constituents form a clear, gel-like structure during early embryonic development, which ultimately attains an average volume of 4 ml in adults.

The vitreous is attached to all contiguous structures of the inner eye, including the internal limiting membrane (ILM) of the retina. The vitreous body is contained within a cortex composed of a dense collagen matrix. The ILM primarily is composed of type IV collagen. The extracellular matrix between the ILM and posterior vitreous cortex is rich in glycoproteins, typical extracellular matrix constituents, and a range of collagen types, each of which is purported to play a role in vitreoretinal adhesion.⁷ The posterior vitreous cortex and retinal ILM are bound at their interface by this macromolecular attachment complex, which is composed of fibronectin, laminin, and other extracellular components that form a glue-like matrix.^{8,9} Chondroitin sulfate is present throughout the vitreoretinal interface and also plays a role in mediating hyaluronan–collagen interaction in the gel vitreous.^{10–12} Posterior vitreous detachment (PVD) is the result of a complex and inevitable set of events that occurs as the eye ages. It manifests as gel liquefaction and weakening of vitreoretinal adhesion. Imaging of the VMI with OCT reveals that PVD usually begins in the perifoveal macula.^{1,2,13} Over decades during midlife, there is concurrent liquefaction of the gel and progressive posterior vitreous cortex separation, ultimately leading to nonpathologic PVD in most eyes.

Typically, concurrent vitreoretinal separation initiates at multiple sites throughout the peripheral fundus as well, and the process proceeds for years to decades before coalescing into a final

separation of the vitreous from the macula and optic nerve, resulting in a complete PVD. The completion of vitreopapillary separation, often signaled by the appearance of the Weiss ring, is the acute, often symptomatic end of a years-long process. Inadequate or incomplete vitreoretinal interface separation can result in anomalous PVD with the potential of ensuing VMI pathologic features.

Partial vitreoretinal separation is an almost inevitable stage throughout this process. However, in cases where liquefaction or gel contraction outpaces detachment of the vitreous cortex, an abnormal adhesion of the vitreous cortex to the ILM is present, or a combination of both is present, a range of anomalous macular conditions can ensue that vary according to the strength and position of the remaining attachments.^{13,14} The result of this mismatch is an anomalous PVD. For the purposes of this classification scheme, an anomalous PVD is defined as a partial vitreous detachment with persistent attachment in the macular region featuring an anomalous strength of adhesion to 1 or more structures in the posterior pole, resulting in tractional deformation of retinal tissue. Local anatomic ocular variations, as occur in high myopia, or extrinsic forces such as blunt trauma or intraocular surgery may play a role as well. The purpose of the consensus panel was to define the pathologic progression of anomalous PVD at the VMI based on OCT-derived anatomic findings.

Given our current knowledge of the potential complications of anomalous PVD and the recent emergence of pharmacologic therapy to supplement vitrectomy therapy, a scheme to classify anomalous anatomic configurations of the vitreous and its effects on the macula is needed. This new OCT-based anatomic classification of VMI diseases provides a consistent nomenclature for clinical use, medical and surgical decision making, future studies, and cross-study comparisons (Table 1). The new classification is based on current evidence and is subject to adaptation as further knowledge is gained.

Optical Coherence Tomography–Based Definition and Classification of Vitreomacular Adhesion

Vitreomacular adhesion is a perifoveal vitreous detachment and is defined, as with other terms in this report, by anatomic features detected with OCT. In Uchino's, Gaudric's, and Johnson's classification schemes, VMA is the equivalent of a stage 1 PVD.^{1,2,13,15} Most eyes have complete vitreoretinal adhesion at birth, so the concept of vitreoretinal adhesion and VMA is a normal state. In this OCT-based classification scheme, however, VMA represents a specific stage of vitreous separation wherein partial detachment of the vitreous in the perifoveal area has occurred,

Table 1. The International Vitreomacular Traction Study Classification System for Vitreomacular Adhesion, Traction, and Macular Hole

Classification	Subclassification
Vitreomacular adhesion	Size: focal ($\leq 1500 \mu\text{m}$) or broad ($> 1500 \mu\text{m}$) Isolated or concurrent
VMT	Size: focal ($\leq 1500 \mu\text{m}$) or broad ($> 1500 \mu\text{m}$) Isolated or concurrent
Full-thickness macular hole	Size: small ($\leq 250 \mu\text{m}$), medium ($> 250 - \leq 400 \mu\text{m}$), or large ($> 400 \mu\text{m}$) Status of vitreous: with or without VMT Cause: primary or secondary

VMT = vitreomacular traction.

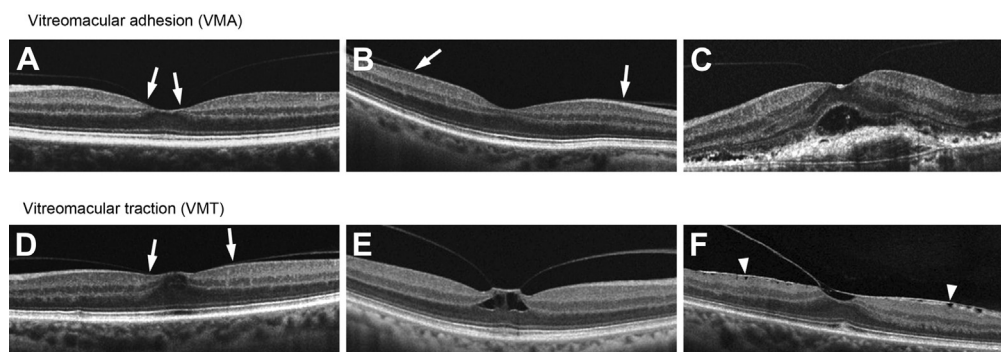


Figure 1. Optical coherence tomography (OCT) scans illustrating (A–C) vitreomacular adhesion (VMA) and (D–F) vitreomacular traction (VMT) according to the International Vitreomacular Traction Study Classification System for Vitreomacular Adhesion, Traction, and Macular Hole. **A**, Focal VMA. The white arrows mark the sites of vitreous attachment. The area of attachment is 1500 μm or less, and there is no detectable change in foveal contour of underlying retinal tissues. **B**, Broad VMA. The white arrows mark the sites of vitreous attachment. The area of attachment is more than 1500 μm , and there is no detectable change in foveal contour of underlying retinal tissues. **C**, Focal VMA with concomitant wet age-related macular degeneration. **D**, Focal VMT. The white arrows mark the sites of vitreous attachment. The area of attachment is 1500 μm or less and is associated with distortion of the foveal surface. **E**, Focal VMT with intrafoveal pseudocyst. **F**, Broad VMT. The area of attachment is more than 1500 μm and is associated with distortion of the foveal surface and elevation of the foveal floor. The VMT also is associated with an epiretinal membrane and macular pucker (white arrowheads).

without retinal abnormalities. Vitreomacular adhesion is characterized by an elevation of the cortical vitreous above the retinal surface, with the vitreous remaining attached within a 3-mm radius of the fovea (defined arbitrarily). The angle between the vitreous and the inner retinal surface is acute, and the retina displays no change in contour or morphologic features on OCT because of the vitreous adhesion. People with VMA generally experience no visual impairment, and the finding is normal in the natural course of PVD. With time, the vitreous may separate spontaneously from the inner retina, usually without incident.

Certain key points are worth noting when considering the definition of VMA. First, this anatomic definition of VMA has been dissociated from symptomatology, because specific visual symptoms are subjective and may be caused by unrelated disease. Second, eyes with VMA may be subclassified by size of the adhesion into either: (1) focal ($\leq 1500 \mu\text{m}$) or (2) broad ($> 1500 \mu\text{m}$; Fig 1A, B). The 1500- μm cutoff has been selected for several reasons. This 1500- μm diameter is a known area of increased vitreous adhesion to the fovea. In addition, this figure has been used routinely to distinguish focal from broad VMA in the published vitreoretinal literature and at most OCT reading centers.^{4,16} It remains unclear whether there is any prognostic difference between focal and broad VMA. When ascertaining the expanse of vitreous attachment, one measures areas in which the adhesion is roughly parallel to the retinal pigment epithelium (RPE). Small regions of dehiscence ($< 1 \text{ mm}$) between the vitreous and neurosensory retina may be present within zones of broad VMA and should be disregarded when classifying VMA as either focal or broad. Eyes with VMA also may have other associated macular abnormalities, including age-related macular degeneration (Fig 1C), retinal vein occlusion, or diabetic macular edema.^{17,18} In these eyes, VMA should be termed *concurrent*, and the term *isolated* should be reserved for cases where no ocular disease is present (Table 1).

Optical Coherence Tomography–Based Definition and Classification of Vitreomacular Traction

Introduction and Definition. The progression of PVD can lead to periods of excessive traction on the macula. Such traction can result in anatomic changes in the contour of the foveal surface, intraretinal pseudocyst formation, elevation of the fovea from the

RPE, or a combination that typically results in reduced or distorted vision.¹⁹ If detectable retinal anatomic changes occur on OCT, with concurrent vitreous status showing perifoveolar PVD, the eye is characterized as having VMT. All of the following anatomic criteria must appear on at least 1 B-mode OCT scan to classify an eye as having VMT: (1) evidence of perifoveal vitreous cortex detachment from the retinal surface; (2) macular attachment of the vitreous cortex within a 3-mm radius of the fovea; and (3) association of attachment with distortion of the foveal surface, intraretinal structural changes, elevation of the fovea above the RPE, or a combination thereof, but no full-thickness interruption of all retinal layers.

Like VMA, VMT can be subclassified into either focal or broad, depending on the width of vitreous attachment (Table 1). Broad areas of attachment with traction can cause generalized thickening of the macula, vascular leakage on fluorescein angiography, macular schisis, and cystoid macular edema. Focal areas of vitreous attachment with traction tend to distort the foveal surface, elevate the foveal floor, form pseudocysts within the central macula, or result in a combination thereof (Fig 1D–F). The presence of pseudocysts usually is associated with diminished visual acuity and visual distortion. After release of traction, pseudocysts generally resolve over time with little remaining visual deficit.²⁰

Relationship to Epiretinal Membrane Formation. With PVD progression, even in the absence of a Weiss ring, residual vitreous tissue often is left on the inner retinal surface. Autopsy studies reveal that residual vitreous remains on the surface of the retina in nearly half of all eyes with PVD.²¹ This condition is called vitreoschisis.²² This residual vitreous may proliferate to form an epiretinal membrane (ERM) at any stage of vitreous separation. Clinical studies uniformly show a high incidence of apparent PVD in eyes with macular pucker. Epiretinal membranes primarily are composed of glial cells and laminocytes (histiocytes) that attach to a scaffold of vitreous cortex remnants on the inner retinal surface.²³ These remnants are thought to be left on the VMI with clinical PVD, and/or represent laminar dehiscence of the posterior vitreous cortex, or both.²² Epiretinal membrane contracture causes macular traction. Further cellular proliferation worsens the ERM, and the combined forces of contracture and vitreoretinal attachment magnify tractional stress on the underlying foveal structure. Delineation on OCT between

Table 2. Correlation between Commonly Used Clinical Macular Hole Stages and the International Vitreomacular Traction Study Classification System for Vitreomacular Adhesion, Traction, and Macular Hole

Full-Thickness Macular Hole Stages in Common Use	International Vitreomacular Traction Study Classification System
Stage 0	VMA
Stage 1: impending macular hole	VMT
Stage 2: small hole	Small or medium FTMH with VMT
Stage 3: large hole	Medium or large FTMH with VMT
Stage 4: FTMH with PVD	Small, medium, or large FTMH without VMT

FTMH = full-thickness macular hole; PVD = posterior vitreous detachment; VMA = vitreomacular adhesion; VMT = vitreomacular traction.

an ERM versus a thickened, attached posterior hyaloid may be difficult, but B-scan ultrasonography could assist in this regard.

Full-Thickness Macular Hole

Introduction and Definition. Full-thickness macular hole is an anatomic defect in the fovea featuring interruption of all neural retinal layers from the ILM to the RPE. Although the definition requires detection on only 1 OCT B-scan, FTMH usually is obvious on several scans through the fovea. When in doubt, a series of closely spaced scans should be examined, because very small FTMHs can be missed in a single line scan.

The actual anatomic opening of the foveal center may arise from several mechanical causes (i.e., tractional foveal cystoid space, breakdown and elevation of central photoreceptors, traction on the inner retina) and may occur with a true retinal operculum. The edge of the hole usually is rounded and may contain intraretinal pseudocysts. The edge also may be thicker than neighboring retinal tissue because of accumulation of intraretinal fluid and may appear slightly elevated from the RPE plane. The hole usually has an hourglass shape that can vary with orientation of the OCT scan. Vitreous may or may not be attached to the edge of the macular hole.

The Gass classification was based on careful clinical examination and divided macular holes into 4 stages.^{24,25} Although the original Gass classification is still quoted widely and adaptations of it are in clinical use, OCT-based anatomic data have added much to our understanding of the pathogenesis and progression of macular hole over the past 2 decades (Table 2).

Optical Coherence Tomography–Based Full-Thickness Macular Hole Classification System (Size of Hole, Presence or Absence of Vitreomacular Traction, Cause). Size of Hole.

Using an OCT-based system, FTMH can be defined anatomically, quantitatively, and reproducibly. The OCT-based measurements of minimum hole width (aperture size; Fig 2A) predict anatomic treatment with both medications and surgery (Pieramici DJ, Boyer DS. The phase III MIVI-TRUST clinical trial data: subgroup responder analysis of a single intravitreal injection of ocriplasmin in patients with full-thickness macular hole. Paper presented at: ARVO Annual Meeting, May 6, 2012; Fort Lauderdale, FL).^{26–28} Aperture size can be measured using the caliper function on spectral-domain OCT devices. The minimum hole width is measured at the narrowest hole point in the mid retina, using the OCT caliper function, as a line drawn roughly parallel to the RPE (Table 1). In this OCT-based anatomic classification system, macular holes are determined to be small, medium, or large based on aperture size.

A small FTMH features an aperture size of less than 250 μm (Fig 2B). The cutoff for small FTMHs at 250 μm is derived from studies showing that these holes are associated with a small rate of spontaneous closure, have a very high closure rate with vitrectomy

(approaching 100%), and of all FTMH classes, are most responsive to pharmacologic vitreolysis (Pieramici DJ, Boyer DS. The phase III MIVI-TRUST clinical trial data: subgroup responder analysis of a single intravitreal injection of ocriplasmin in patients with full-thickness macular hole. Paper presented at: ARVO Annual Meeting, May 6, 2012; Fort Lauderdale, FL).²⁷

A medium FTMH is defined by aperture size from 250 to 400 μm (Fig 2C). Studies exploring postsurgical FTMH closure rates by aperture size consistently show a very high anatomic closure rate (>90% in all recent series) with complete removal of residual hyaloid, with or without ILM peeling.^{26,28} Pharmacologic therapy can be successful as well, but at a lower rate than for small macular holes (Pieramici DJ, Boyer DS. The phase III MIVI-TRUST clinical trial data: subgroup responder analysis of a single intravitreal injection of ocriplasmin in patients with full-thickness macular hole. Paper presented at: ARVO Annual Meeting, May 6, 2012; Fort Lauderdale, FL).²⁹

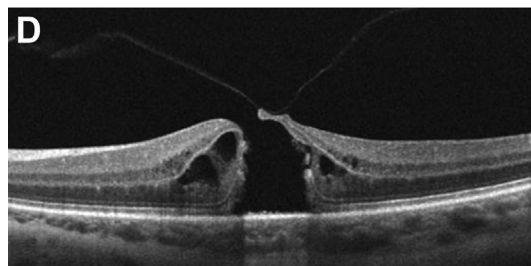
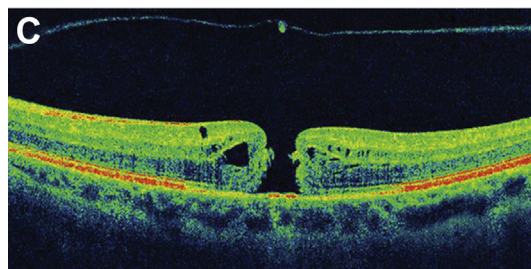
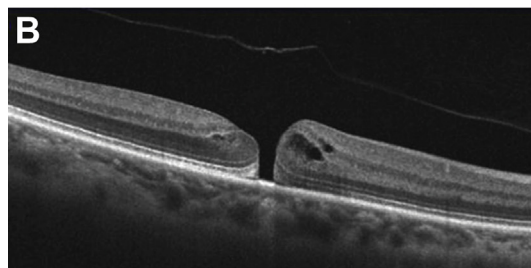
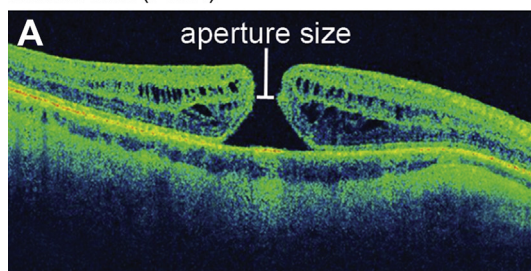
Nearly half of FTMHs are large (diameter >400 μm) at the time of diagnosis (Fig 2D). Vitrectomy with ILM peel is associated with high closure rates (90%–95%), even for these large holes. Without an ILM peel, the vitrectomy success rate is closer to 75%.³⁰ In the few eyes with large FTMH that have undergone pharmacologic vitreolysis, no anatomic success has been recorded.²⁷

Status of the Vitreous: Presence or Absence of Vitreomacular Traction. In the OCT-based anatomic system, FTMHs are categorized secondarily according to absence or presence of vitreous attachment (Table 1). Only macular holes with concurrent VMT should be considered for pharmacologic vitreolysis.

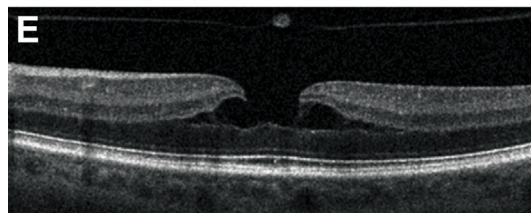
Primary Versus Secondary Full-Thickness Macular Hole. Full-thickness macular hole can be subdivided into primary and secondary forms. Primary FTMH (formerly referred to as idiopathic) results from vitreous traction on the fovea from anomalous PVD (VMT; Fig 3). A secondary FTMH is caused directly by other pathologic features and does not have pre-existing or concurrent VMT. A partial list of conditions that have been shown to result in secondary macular hole includes the following: (1) blunt trauma,^{31–34} (2) lightning strike,³⁵ (3) high myopia (note that some eyes with high myopia demonstrate FTMH with VMT as a precursor),^{36,37} (4) macular schisis,³⁸ (5) macular telangiectasia type 2,³⁹ (6) wet macular degeneration treated with anti-vascular endothelial growth factor therapy (Chaudhry NA, et al. Spontaneous development and closure of full-thickness macular hole during intravitreal anti-VEGF therapy for neovascular age-related macular degeneration. Paper presented at: ARVO Annual Meeting, May 6, 2012; Fort Lauderdale, FL), (7) macroaneurysm,^{40,41} and (8) surgical trauma.^{42,43}

Full-thickness macular hole can occur concomitantly with macular edema that is associated with a variety of retinal diseases, including diabetic macular edema, age-related macular degeneration, retinal vascular occlusions, and uveitis.^{44,45} Some of these cases should be classified as primary macular holes if VMT is

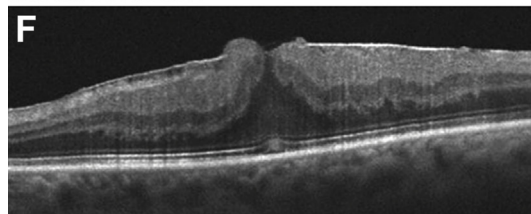
Full-Thickness
Macular Hole (FTMH)



Lamellar
Macular Hole (LMH)



Macular Pseudohole



shown to play a role. In a small number of cases, FTMH may develop in an eye with macular edema without prior or concurrent VMT. Full-thickness macular hole also has been reported to occur after certain intraocular surgical procedures, including vitrectomy for retinal detachment and lens removal.^{42,46} It recently was shown that fovea destabilization resulting from traction-induced damage to the inner fovea, occurring before or coincident with spontaneous vitreofoveal separation, may predispose some eyes to macular hole formation.⁴⁷ For cases in which the vitreous obviously has been removed before formation of a macular hole, the designation of secondary macular hole is probably most accurate.

Impending Macular Hole

A special circumstance exists when an individual develops FTMH in one eye and OCT reveals VMA or VMT in the fellow eye. Studies show that these fellow eyes are at increased risk for development of FTMH.⁴⁸ In the past, the finding of VMA in a fellow eye has been referred to as a stage 0 macular hole, but the term *impending macular hole* should be used instead to describe a case in which FTMH is observed in one eye and VMT is observed on OCT in the fellow eye (Tables 2 and 3). The term *impending macular hole*, despite the connotation of inevitability, does not exclude the possibility of spontaneous resolution.

Lamellar Macular Hole

Lamellar macular hole (LMH) is a partial-thickness foveal defect that typically appears on biomicroscopy as a round or oval, well-circumscribed, reddish lesion. Clinical detection of early LMH may be difficult using biomicroscopy alone. Anatomic OCT-based features of LMH include the following: (1) an irregular foveal contour; (2) a defect in the inner fovea (may not have actual loss of tissue); (3) intraretinal splitting (schisis), typically between the outer plexiform and outer nuclear layers; and (4) maintenance of an intact photoreceptor layer. Lamellar macular hole can be distinguished from FTMH on OCT best by the presence of intact photoreceptors at the base (Fig 2E).

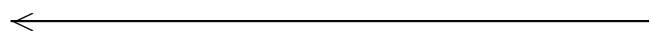


Figure 2. Optical coherence tomography (OCT) scans illustrating examples of full-thickness macular hole (FTMH), lamellar macular hole (LMH), and macular pseudohole according to the International Vitreomacular Traction Study (IVTS) Classification System for Vitreomacular Adhesion, Traction, and Macular Hole. **A**, Full-thickness macular hole can be defined anatomically by OCT-based measurement of minimum hole width or aperture size. The aperture size is measured at the narrowest hole width in the mid retina, using the OCT caliper function, as a line drawn roughly parallel to the retinal pigment epithelium. The OCT scan shows an FTMH with aperture size of 192 μm and without vitreomacular traction (VMT). **B**, Small FTMH without VMT. The foveal lesion interrupts all macular layers from the internal limiting membrane to the retinal pigment epithelium and has a narrowest linear width of 250 μm or less. The vitreous is not attached to the macula or edge of the hole. **C**, Medium FTMH without VMT. The hole has a narrowest linear width of more than 250 μm and 400 μm or less. **D**, Large FTMH with VMT. The narrowest linear width of the hole is more than 400 μm . The vitreous is attached to the edge of the hole. **E**, Optical coherence tomography scan of an LMH with intraretinal splitting (schisis) between the outer plexiform and outer nuclear layers, but with maintenance of the photoreceptor layer. The vitreous is detached with an operculum visible above the hole. **F**, Optical coherence tomography scan illustrating anatomic features that distinguish a macular pseudohole in the IVTS Classification System.

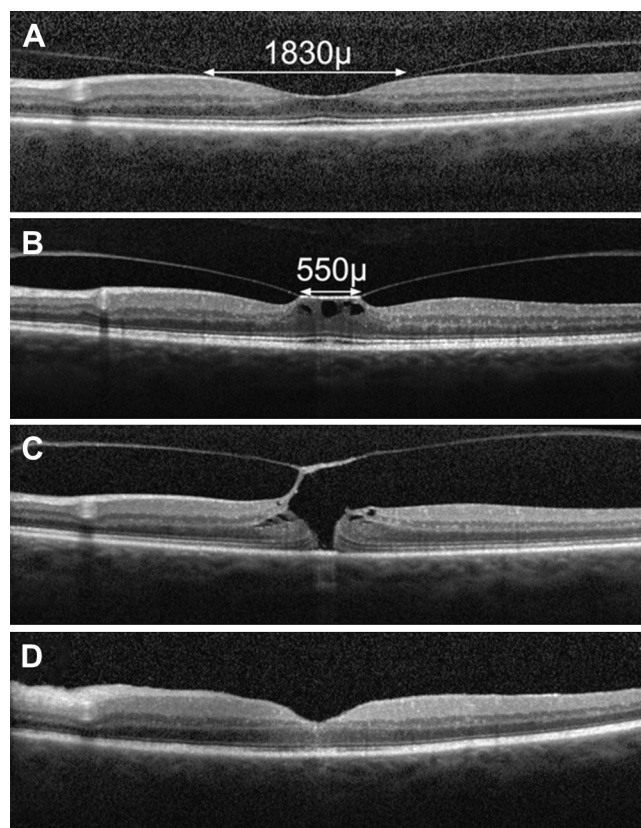


Figure 3. Optical coherence tomography images showing progression of vitreomacular pathologic features. **A**, At presentation, the patient had broad, isolated vitreomacular adhesion (VMA) with the region of attachment having a diameter of 1830 μm . **B**, One year later, she demonstrated mild distortion. The diagnosis was focal, isolated vitreomacular traction (VMT) with a diameter of attachment of 550 μm . Note the change in contour of the inner fovea with cystoid spaces in the inner retinal tissue. **C**, Three months later, the patient demonstrated a primary macular hole. The inner flap of retina was adherent to the vitreous, there were cystoid spaces in the retina, and there was a slight upturn of the inner margins of the hole. **D**, One month after surgical repair that included vitrectomy, membranectomy, and fluid-gas exchange, the macular hole was closed anatomically.

Lamellar macular hole usually progresses slowly and is thought to arise from incomplete FTMH formation, centripetal traction from ERM, or both, although current understanding of LMH evolution and surgical indications remains incomplete.⁴⁹ Forces acting on the central macula include an ERM in some patients and varying anteroposterior and tangential forces from the vitreous in others.^{50,51} Lamellar macular hole usually is associated with reports of mild or limited central vision loss; reading vision may deteriorate over time, typically because of progression of associated ERM. Most patients with LMH have mild metamorphopsia, limited central vision loss, and stable visual acuity.

Vitreopapillary adhesion, where the vitreous remains attached to the optic disc and peripapillary retina with peripheral detachment, is present in approximately half of eyes with LMH, suggesting that persistent vitreous cortical traction plays a role in some eyes.⁵² Lamellar macular hole width may expand more quickly in the presence of vitreopapillary adhesion, because it can pull on an LMH, causing horizontal enlargement and visual impairment.

Lamellar macular hole has been reported in eyes after cataract surgery and in association with concomitant ocular conditions including myopia, uveitis, exudative age-related macular degeneration, and retinal detachment.⁵³ Surgery for LMH remains controversial, and patient selection is crucial. Results of surgical interventions for LMH are variable, with anywhere between 25% and 75% of patients achieving improved visual outcomes, typically because of peeling of the associated ERM.^{51–57} Future prospective studies of pars plana vitrectomy for LMH are warranted to define guidelines for surgical intervention.

Macular Pseudohole

Macular pseudohole is a clinical diagnosis based on slit-lamp biomicroscopic examination of the macula. Specific morphologic features are confirmed best with OCT.⁵⁸ Clinically, a pseudohole appears as a discrete, reddish, round or oval lesion in the fovea that typically is 200 to 400 μm in diameter and similar in appearance to a small or medium FTMH. Slit-lamp examination of the macula can result in a false diagnosis of FTMH, hence the term *pseudohole*. Although a large cystic lesion in the central macula also can mimic a pseudohole, careful biomicroscopy will reveal the difference. Optical coherence tomography with multiple foveal line scans is 100% sensitive in ruling out FTMH.

The advent of OCT has elucidated anatomic changes at the VMI that are associated with the presence of a macular

Table 3. Characteristic Attributes of Clinical Stages of the International Vitreomacular Traction Study Classification System for Vitreomacular Adhesion, Traction, and Macular Hole

Clinical Stages	Attributes	Comments
VMA	Vitreous adhesion to central macula with no demonstrable retinal morphologic changes	Has been called stage 0 in the past when contralateral eye has FTMH; normal appearance on clinical examination; no symptoms
VMT	Vitreous adhesion to central macula with demonstrable changes by OCT but no full thickness tissue dehiscence; may include the following: tissue cavitation, cystoid changes in macula, loss of foveal contour, elevation of fovea above RPE	May or may not have yellow changes in central macula on examination; can be referred to as impending macular hole if FTMH in contralateral eye
Small FTMH	Hole $\leq 250 \mu\text{m}$; may be round or have a flap adherent to vitreous; operculum may or may not be present	Visual acuity may be relatively good; optimal size for successful repair by pharmacologic vitreolysis; very high probability of success with vitrectomy surgery
Medium FTMH	Hole >250 but $\leq 400 \mu\text{m}$; may be round or have a flap adherent to vitreous; operculum may or may not be present	High probability of success with vitrectomy surgery
Large FTMH	Hole $>400 \mu\text{m}$; vitreous more likely to be fully separated from macula	Slightly less probability of successful closure with vitrectomy surgery

FTMH = full-thickness macular hole; OCT = optical coherence tomography; RPE = retinal pigment epithelium; VMA = vitreomacular adhesion; VMT = vitreomacular traction.

Table 4. The IVTS Classification System for Vitreomacular Adhesion, Traction, and Macular Hole

Anatomic State	IVTS Classification System for Vitreomacular Adhesion, Traction, and Macular Hole
VMA	<p>Definition</p> <ul style="list-style-type: none"> Evidence of perifoveal vitreous cortex detachment from the retinal surface Macular attachment of the vitreous cortex within a 3-mm radius of the fovea No detectable change in foveal contour or underlying retinal tissues <p>Classification</p> <ul style="list-style-type: none"> By size of attachment area <ul style="list-style-type: none"> Focal ($\leq 1500 \mu\text{m}$) Broad ($> 1500 \mu\text{m}$, parallel to RPE and may include areas of dehiscence) By presence of concurrent retinal conditions <ul style="list-style-type: none"> Isolated Concurrent
VMT	<p>Definition</p> <ul style="list-style-type: none"> Evidence of perifoveal vitreous cortex detachment from the retinal surface Macular attachment of the vitreous cortex within a 3-mm radius of the fovea Association of attachment with distortion of the foveal surface, intraretinal structural changes, and/or elevation of the fovea above the RPE, but no full-thickness interruption of all retinal layers <p>Classification</p> <ul style="list-style-type: none"> By size of attachment area <ul style="list-style-type: none"> Focal ($\leq 1500 \mu\text{m}$) Broad ($> 1500 \mu\text{m}$, parallel to RPE and may include areas of dehiscence) By presence of concurrent retinal conditions <ul style="list-style-type: none"> Isolated Concurrent
FTMH	<p>Definition</p> <ul style="list-style-type: none"> Full-thickness foveal lesion that interrupts all macular layers from the ILM to the RPE <p>Classification</p> <ul style="list-style-type: none"> By size (horizontally measured linear width across hole at narrowest point, not ILM) <ul style="list-style-type: none"> Small ($\leq 250 \mu\text{m}$) Medium ($> 250 \mu\text{m}$ and $\leq 400 \mu\text{m}$) Large ($> 400 \mu\text{m}$) By presence or absence of VMT By cause <ul style="list-style-type: none"> Primary (initiated by VMT) Secondary (directly due to associated disease or trauma known to cause macular hole in the absence of prior VMT)
LMH	<p>Definition</p> <ul style="list-style-type: none"> Irregular foveal contour Defect in the inner fovea (may not have actual loss of tissue) Intraretinal splitting (schisis), typically between the outer plexiform and outer nuclear layers Maintenance of an intact photoreceptor layer
Macular Pseudohole	<p>Definition</p> <ul style="list-style-type: none"> Invaginated or heaped foveal edges Concomitant ERM with central opening Steep macular contour to the central fovea with near-normal central foveal thickness No loss of retinal tissue

Abbreviations: ERM = epiretinal membrane; FTMH = full-thickness macular hole; ILM = internal limiting membrane; IVTS = International Vitreomacular Traction Study; LMH = lamellar macular hole; RPE = retinal pigment epithelium; VMA = vitreomacular adhesion; VMT = vitreomacular traction.

pseudohole.⁵⁰ Importantly, there is no loss of foveal tissue, as is observed typically with LMH or FTMH. Central foveal thickness usually is normal or slightly thin.⁵⁰ Thus, OCT confirms the diagnosis on the basis of the following 4 characteristics (Fig 2F): (1) invaginated or heaped foveal edges, (2) concomitant ERM with central opening, (3) steep macular contour to the central fovea with near-normal central foveal thickness, and (4) no loss of retinal tissue.

Perhaps the most characteristic feature of macular pseudohole is the presence of a concomitant ERM on the surface of the macula that distorts the foveal contour into a shape with a steep slope; altered light reflex also is observed commonly. Epiretinal membrane plays a causative role in pseudohole, its contraction pulling the underlying retinal tissue toward the center of the fovea.⁵⁸ The result is invagination of the perifoveal retina into

a shape that mimics a hole but contains no actual loss of foveal tissue.

Management of macular pseudohole typically is conservative. If the ERM is associated with a significant decline in vision, pars plana vitrectomy with membrane peeling can be performed. Successful ERM removal often leads to restoration of the normal foveal contour and some improvement in visual acuity.

The new OCT-based anatomic International Classification System for Vitreomacular Adhesion, Traction, and Macular Hole was developed to allow clinicians to speak a common language when discussing diseases of the VMI. It is purely anatomically based, without regard to symptoms or fellow eye status. Definition criteria were dissociated from symptoms, because specific visual symptoms are subjective and may be caused by unrelated disease. The classification system was designed to be simple, easy to

remember, clinically applicable, helpful in predicting therapeutic outcomes, and useful for the execution and analysis of clinical trials. According to the consensus definition, VMA is characterized exclusively by anatomic criteria, including the presence of normal or unperturbed retinal morphologic features, and by extension is not expected to present with visual symptoms in most cases. Vitreomacular interface-related visual systems usually are associated with vitreous traction and distorted foveal morphologic features and, according to the new definitions, therefore would be considered VMT. The panel's consensus outcomes are summarized in Tables 3 and 4. The definitions and classifications are proposed with the understanding that such systems are dynamic and subject to perpetual change as we advance our knowledge of vitreoretinal diseases and their underlying pathophysiologic characteristics.

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