

From Print to Practice:  
PVD a common process with potential for ocular morbidity

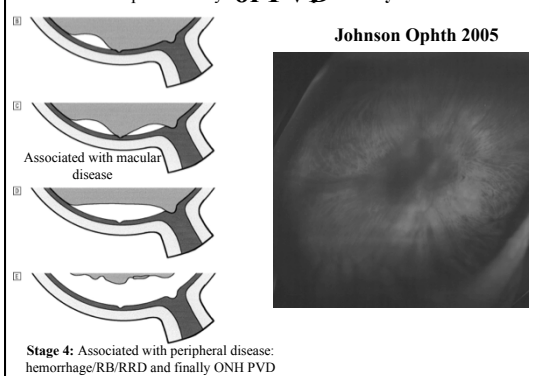
Diana Shechtman OD FAAO

### Financial disclosure

I have received lecture honoraria or serve on the advisory boards or speaker's bureaus of:  
Reichert, Alcon, Allergan, CZ & Zeavision

Uchino, E. et al. Arch Ophthalmol 2001

The PVD process may not be at the end of PVD & likely starts earlier on



Johnson Ophth 2005

Associated with macular disease

Stage 4: Associated with peripheral disease: hemorrhage/RB/RRD and finally ONH PVD

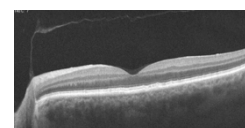
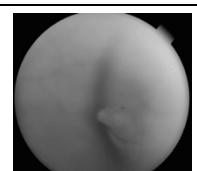
### Anomalous PVD

Vitreoschisis (split within the vitreous)

- ERM
- MH

Partial PVD

- Posterior separation RB/RRD
- Peripheral separation Vitreopapillary traction
- VMTS
- MH, ERM, CME
- Exacerbation of DME, AMD, ME/VO

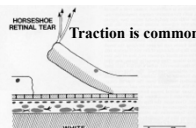
Symptomatic FTs are always treated!

**Based on relationship b/t RB & RD, would you Hold' em or Fold' em?**

Byer's work: symptomatic flap tears lead to RRD in >50% of cases  
Wilkinson Ophth 2000: SYMPTOMATIC FT (flat tears) has the best supportive evidence & overall consensus with regards to treatment.

### Horseshoe or U-shaped Flap Tear (FT) following a PVD

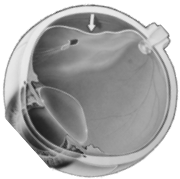
The base faces the Anterior retina



Traction is common

The apex faces the Posterior retina

### Flap tears



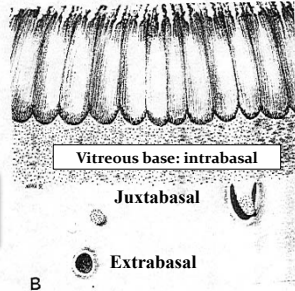
This is a full thickness retinal break w/ attached flap (**TEAR**), resulting from abnormally strong vitreoretinal adhesion

50% of flap tears are located superior

### Location...Location...Location

**Juxtapapillary breaks: WORRY!**  
Traction here results in FT (Highest risk of RRD)

Also take into consideration Superior retinal breaks



Vitreous base: intrabasal  
Juxtabasal  
Extrabasal

### The history of RB and tx

In 1920, Gonin recognized that RBs were the cause of RRDs  
Tx of RD requires sealing of such a **BREAK**  
The question of prophylactic treatment of retinal tears to prevent a retinal detachment was further discussed by Linderl 1934

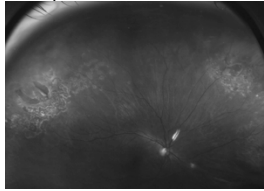
Late '50's, it was recommended to tx all breaks to prevent RD  
Birth of photocoagulation xenon laser (Meyer-Schwickerath)

TODAY...it is **IMPORTANT** to determine when a RB requires referral for tx and when it can be follow

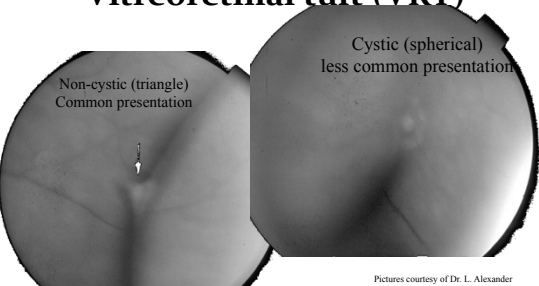
**Complications associated with treatment can occur...**

### THE FACTS

- 6-7% of the general population have a RB
- 1 in 10,000 will lead to a RRD
- RRD incidence is 12 in 100,000



### Vitreoretinal tuft (VRT)



Non-cystic (triangle)  
Common presentation

Cystic (spherical)  
less common presentation

**Retinal Tuft: Risk of RD is <1% (Foos '74) (Byer '81)**  
Most are followed

Pictures courtesy of Dr. L. Alexander

### Operculated holes

Probably arise from cystic retinal tufts

- ✓ Commonly follows vitreous traction, which may brought upon by a PVD
- ✓ 80% associated with PVD

Generally asymptomatic and stable but there are exceptions...

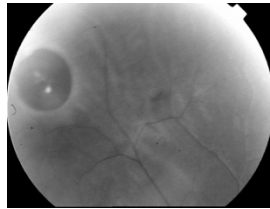
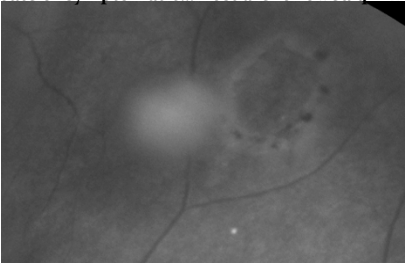
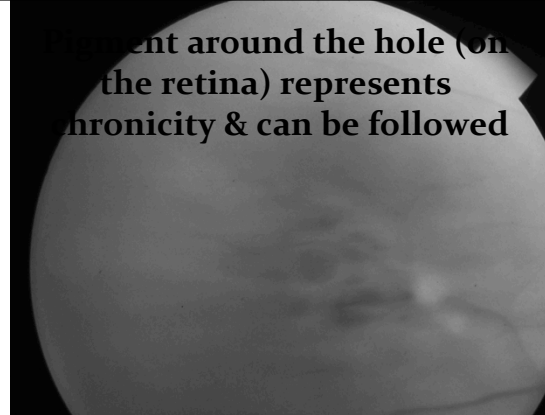


Photo courtesy: D. Hassenpflug, OD

**Operculated hole:**  
 represent a round, full-thickness retinal defect with an associated avulsed piece of retinal tissue in the vitreous cortex  
 unless acute or symptomatic...most are followed q6-12M



...ent around the hole (on  
 the retina) represents  
 chronicity & can be followed



**Scleral depression:  
 The questions**

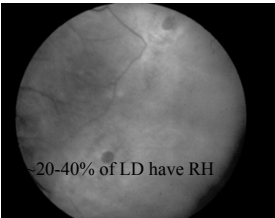
When to use the procedure?  
 To view a more anterior structure or manipulate view of a hidden lesion  
 Viewing a vitreoretinal abnormality in profile  
 DDx (retinal hemorrhage, associated traction, fluid cuff...)  
 To better view a shallow RD/RB

What do you use?  
 Thimble, Q-tip, Scleral Indenter

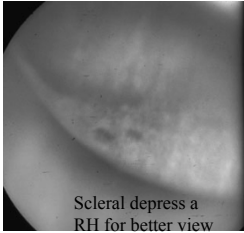
**Making scleral depression  
 an uplifting experience**

Show them the instrument & demonstrate what you will be doing  
 Q-tip is the least intimidating  
 What you say matters  
 Maximum dilation  
 Place the instrument properly

**Atrophic Retinal holes: Most common RB**  
 Yearly monitored (Byer '74, Neumann '72)  
 This is NOT associated with vitreoretinal traction (PVD)  
 The pathogenesis is retinal thinning

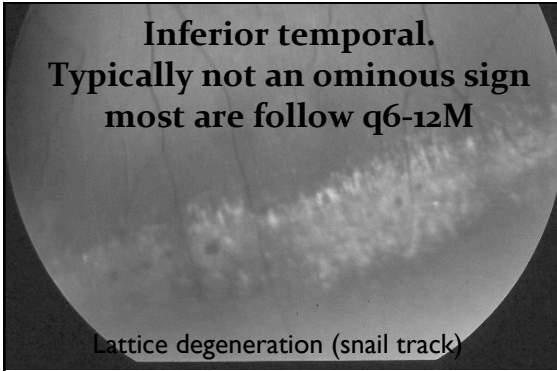


~20-40% of LD have RH



Scleral depress a RH for better view

**Inferior temporal.**  
 Typically not an ominous sign  
 most are follow q6-12M



Lattice degeneration (snail track)

### Lattice degeneration & retinal breaks/detachments

- Although 30% of eyes with RD have Lattice Degen. The chance of LD developing an RD is 1%
- In addition, 89% of RD's from affected eyes occur in areas of normal peripheral retina
- Prophylactic laser in OTHER studies did not prevent tear or RD in fellow eyes... if no PVD present at time of tx:
  - 94% still developed tears & 76% still developed RD

1. Evidence based Prophylactic. Wilkinson C. P. Ophthalmology 1/2000  
 Editorial by Norman Byer, M.D. in same issue  
 2. Chauhan et al. Failure of Prophylactic Retinopexy. Arch Ophth. 7/06

### Common peripheral pathological conditions associated with the onset of an acute PVD include

Retinal break	Likelihood of treatment
Flap tears	Frequently May deserve a consult
Operculated holes	Sometimes Referral depends on symptoms, traction, etc
Atrophic holes	Not likely Look at risk factors

### So, decision making: Retinal breaks that are typically monitored

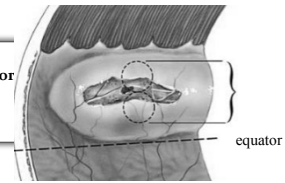
- Atrophic retinal holes
- Asymptomatic operculated holes
- No vitreoretinal traction
- Patient is reliable
- Surrounding retinal pigment

Follow up q6-12M

What is a SUBclinical RD?

**Subclinical RD:** fluid extend >1DD from the RH but NOT > 2DD posterior to the equator (Davis '73)

>30% become CLINICAL RD (Davis '74)



Courtesy of Dr. B. Townsend

### Decision making: RBs that may require consultation

- Larger breaks
- Any symptomatic break
- Flap tears (asymptomatic vs. symptomatic)
- Superior location
- Associated traction
- ?Fluid cuff/subclinical RD

### Guidelines for tx RB:

AOA Guideline  
 Symptomatic retinal breaks deserve a consultation

- Is it symptomatic?

- Symptom is a critical prognostic criteria towards RRD progression
  - >~30% of symptomatic untxed RBs lead to a RRD
    - Colyear '56 & '60, Davis '73, Shea '74
  - Asymptomatic RBs do not show any significant tendency towards RD
    - Byer 1998: 162 cases (16% FT) → 1 subclinical RRD
    - (Neumann '72) (Davis '74)
    - Yet...

**Risk factor towards the development of a retinal break or detachment**

- Is there associated trauma?
  - As high as 80% of traumatic RBs (in one study) were associated with development of a RRD
    - Cooling 1986, Johnson 1991
- Is the pt a moderate-high myope (>3D)?
  - 50% of RD pts are myopes
    - Eye disease control grp '93

☺

**Risk factor towards the development of a retinal break or detachment**

- Is the pt pseudophakia or aphakic?
  - Aphakia accounts for up to 40% of RDs but incidence is ~1%
    - Risk is higher closer to the time after surgery,
      - Younger age, complication during CE surgery, high myopia, presence of PVD
- Has the pt had a yag?
  - Ambler 1988: increase risk <2%

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**Risk factor towards the development of a retinal break or detachment**

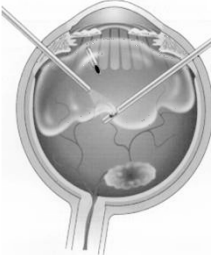
- history of RD in the fellow eye (contralateral eye)?
  - 5-10% of pts with hx of RD will develop RRD in fellow eye (Combs '82, Davis '74, Tornquist '63)
    - Same dynamics
- Is there a STRONG family history of a RD?
  - Snead '94: Stickler's syndrome

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Progression of VMT

Hikichi 1995:  
10% associated with spontaneous PVD, while others remain stable & yet others progress

**VMT: Today's treatment**



PPV & epimacular vitreous cortex aspiration  
Surgery is invasive...

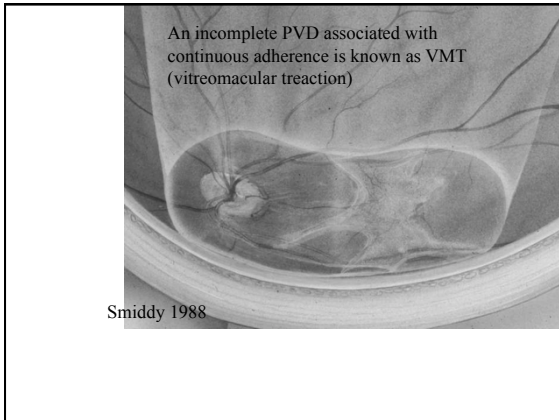
**VMT: when to consider treatment**

The variable factors that one takes into account

- VA & degree of visual symptoms
- Occupation
- Progression
- Associated complications

Yet success depends on:

- Onset
- Associated complications
- VA



### VMT

#### Print to practice Smiddy 1990

VMT may play a role in the pathogenesis of a number of maculopathies & commonly associated with

- Cystoid macular edema (CME)  
MOST COMMON
- Macula hole formation (MH)
- Epiretinal membrane (ERM)
- Exacerbates of macular edema
  - Macular retinoschisis or detachment
  - Myopic foveoschisis

### Vitreofoveal Traction

- OCT defined
- Vitreofoveal attachment

### The role of minimal surface traction

Stage "o" Chan Oph 2004

Central posterior vitreous adhesion to fovea

Increase risk of developing MH (10%)

Pt is asymptomatic but at risk

### Stages of Macular Holes

Gass 1988

I: Pseudocyst associated with traction

IA: foveal detachment (macular cyst)  
Yellow dot stage

IB: Loss of foveal depression  
Donut shaped yellow ring  
TYPICALLY FOLLOWED, IF MILD s/s AND GOOD VISION  
50% resolve on their own

### Stages of Macular Holes

Gass 1988

II: Partial tear in the sensory retina  
<400um

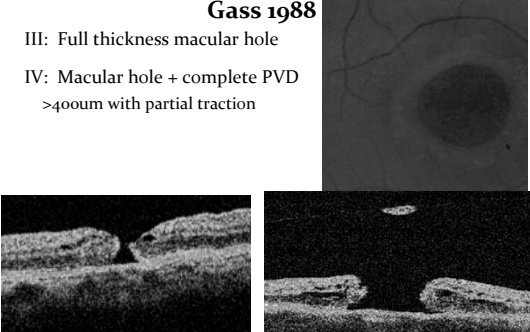
Anything from stage 2 on needs to be refer for treatment

### Stages of Macular Holes

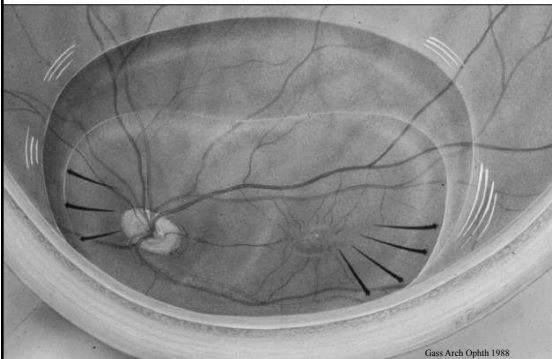
**Gass 1988**

III: Full thickness macular hole

IV: Macular hole + complete PVD  
>400um with partial traction



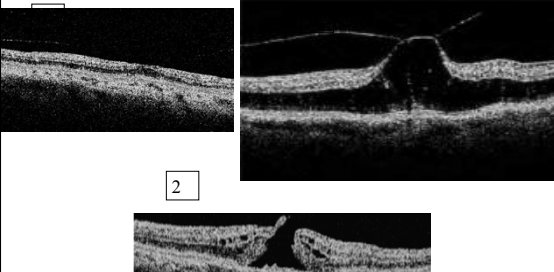
### Old theory: Tangential traction due to shrinkage and contraction of the cortical vitreous



Gass Arch Ophthalm 1988

### OCT has expanded our knowledge about macular hole pathogenesis

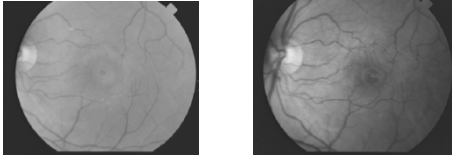
Hee 1995, Gaudrin 1999, Ito 2003



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### MH - Spontaneous closure

3M later: spontaneous closure

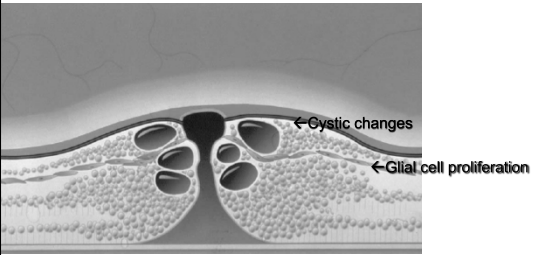


MH 3: 20/70

20/40 BCVA  
glial plug & RPE  $\Delta$ s

Kokame R. Et al. Am J Ophthalmol 2002; 133: 280

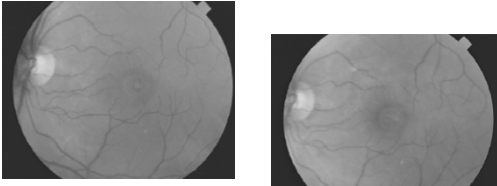
### Glial cell proliferation & closure of a hole



← Cystic changes

← Glial cell proliferation

### MH – Spontaneous reopening



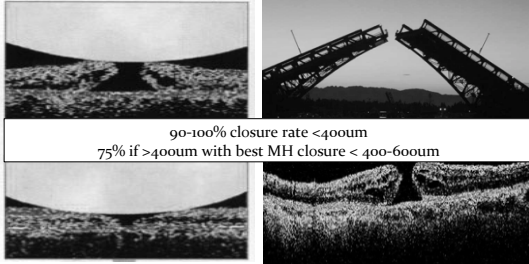
20/60 BCVA, glial remnant (Sup)  
Note surrounding fluid

S/P: surgically repaired 20/20

Kokame R. Et al. Am J Ophthalmol 2002; 133: 280

## Tamponade

Bubble + face down position  
Sealed closure of the hole  
Allows for better glial cell adhesion



90-100% closure rate <400um  
75% if >400um with best MH closure < 400-600um

### Success in tx depends on distinct factors including:

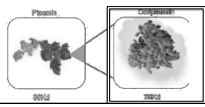
- Stage
  - Earlier stage
- Duration
  - < 6M
- Size of hole
  - < 400 um



### Future VMT tx **microplasmin**

an active molecule similar to plasmin

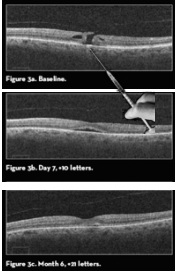
- ◆ Is a recombinant truncated form of human plasmin that is active
  - ◇ functions as a thrombolytic agent causing an enz pharmacological induced vitreolysis
    - > Nonsurgical PVD
    - > The enzymatic agents alter the biochemistry of vitreous
      - Liquefaction of the vitreous occurs
      - LYSIS between vitreous cortex and ILM is the final outcome



### Ocriplasmin (ThromboGenics)

#### Could it be a future tx for symptomatic VMT?

- ◆ MIVI-TRUST phase 3 (n=650)
  - ◇ Inclusion: VMT w VA  $\leq$  20/25 & OCT showing thickness
  - ◇ Microplasmin injection vs placebo
    - > ~30% of pts had resolution of VMT
    - > ~40% of pts with MH had a complete closure
  - ◇ Improve VA/restores structures
    - > Good safety profile




Recently received Priority FDA review

Packo & Jumper ASRS 2010

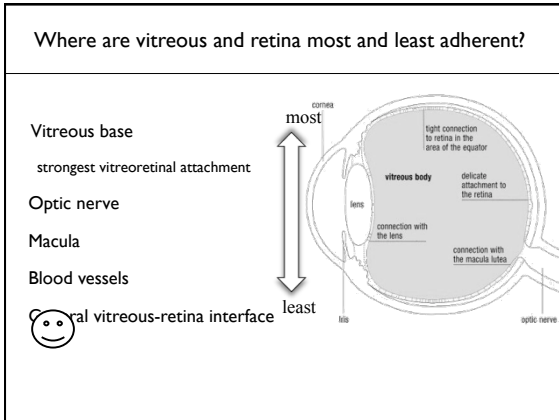
### What about this case?

#### Would you refer given the fact it is 20/50-?



Stage 3-4 require tx in order to improve VA





**VR conditions associated with PVD process**

Complications likely associated with accelerated liquefaction occurs before weakening of VR adhesion

VR traction site	Retinal condition
Retinal vasculature	Retinal or vitreous hemes Avulse retinal vessel
Macula	VMT syndrome &/or MH
Periphery	Operculated Holes/ Breaks RRD

**Posterior Vitreous Detachment**

- Symptoms of flashes/floaters
- Common in pts >65 yo
- As seen by different imaging
- Weiss ring clearly seen on OCT
- Partial PVD seen on B-scan  
– Dynamic process
- Approx 65% of people >70yo
- Approx 85-95% of clinical RRD's arise as result of PVD and traction

**So how to managed the ACUTE PVD?**

- Dilated fundus exam is a MOST! (also consider SD)
- Close Follow-Up
  - f/u for symptomatic acute PVD 2-6wks—3M→yearly
    - Same follow up if symptoms but NO PVD noted
    - Asymptomatic chronic PVD followed annually
  - PRINT→PRACTICE:
    - 15% of symptomatic PVDs have a RB
      - JAMA 2009, Linder 1966, Jaffe '79, Brodley '83, Novak 1984, Byer 1994
  - Educate about onset of news si/s

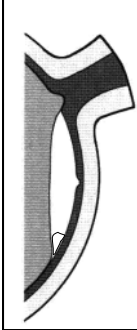
When evaluating an acute PVD, Pigment cells in vitreous or vitreous heme increase the chances of a concomitant retinal break

**From PRINT→PRACTICE**  
**Pigmented cells vitreous**  
Br J Ophthalmol 2000;84:1264

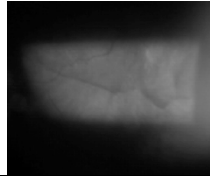
115 Eyes With RRD	96.5% had a PVD	96% had Pigmented cell in the vitreous
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Courtesy of Dr. I. Alexander

### The truth about VH associated with a retinal break



Because of gravity, VH have a tendency to settle inferiorly, An associated RB is not necessary at the location of the VH



ce with the  
on

J Sotka NSUCO