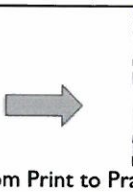
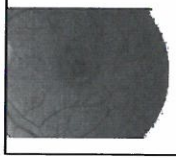
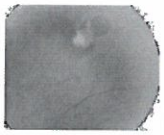




From Print to Practice:
PVD a common process with potential for ocular morbidity

Diana Shechtman OD FAAO

Disclosures

- I am on
  - The speaker's bureau for: Bausch & Lomb, Zeavision, Carl Zeiss Meditec & Alcon
  - Advisory Board for: Allergan, Thrombogenics, Carl Zeiss Meditec, Alcon Nutritional Dept & Arctic Dx

These affiliations will have no affect on the content of this lecture

FACTS

2/3 of pts > 60yo have a PVD

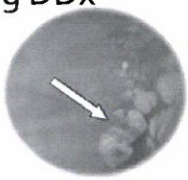
1 in 6 acute PVD → RB

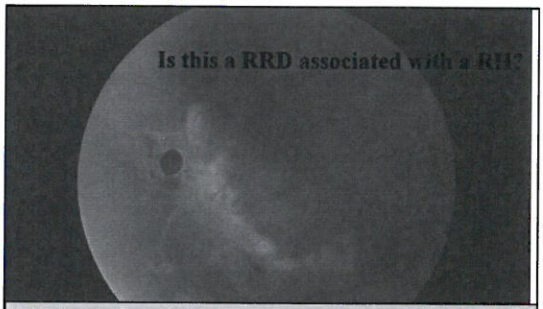
1 in 3 symptomatic RB→RRD

So…which is better: monitor or refer

Start by thinking DDx

- Paving-stone degeneration
  - Missing OUTER retina
- Meridional folds
  - Fold in ora
- Peripheral cystoid degeneration
  - Small intraretinal vesicles that can lead to …
- More…



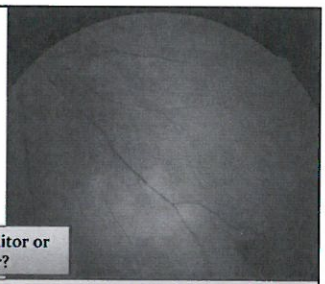


WWOP: 30% of population & most common in pigmented skin pts
Optical phenomenon
May be related to increased density of collagen fibrils at interface with retina.
Tears can occur along border due to traction/vitreous degen in pts>40-50

FEAR FACTOR 101

54 BM
No Flashes
(+) floater

Inferior VIEW…



Why would you monitor or
Why would you refer?

Recently a UK study revealed that asymptomatic RB (in particular inferior) should be followed due to low risk of RRD. Current lit does not provide enough evidence to suggest tx of RB other than symptomatic FT (Wilkinson 2000)

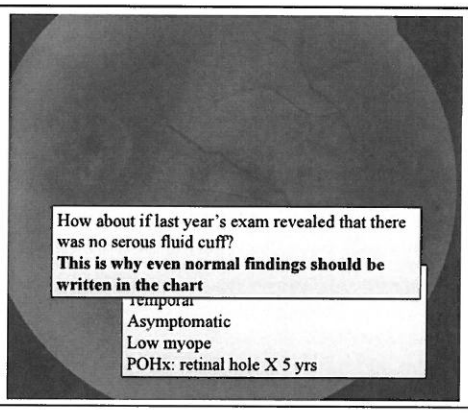
OPHTHLAMOLOGY 9/2013
RD s/p CE

"The risk for RRD is **4-fold** greater **after CE** than in untreated eyes."

Analyzed from a national registry of **202,226 pts**
Eyes s/p CE vs THEIR contralateral eye (w/o CE)

575 RD :
465 RDs operated eye
110 in a nonoperated eye.

Risk was highest w/I 6m but may occur up to 10 yrs later



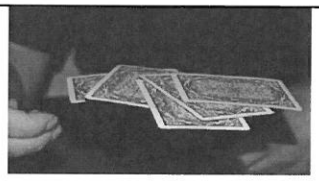
What is likely present in the periphery?

Retinoschisis

Atrophic hole

Flap tear

Congenital hypertrophy of the RPE (CHRPE)

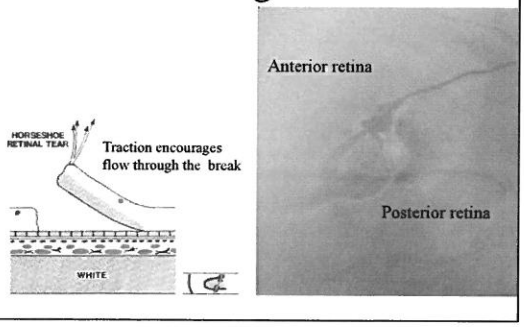


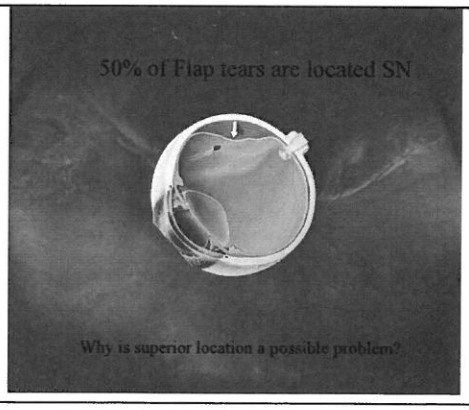
Symptomatic FTs are always treated!

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 Opth 2000: SYMPTOMATIC FT (flat tears) has the best supportive evidence & overall consensus with regards to treatment.

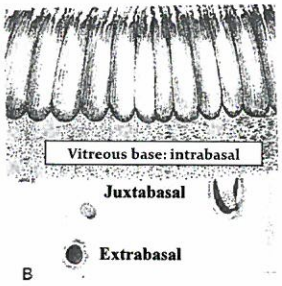
Horseshoe or U-shaped **Flap Tear (FT)** following a PVD



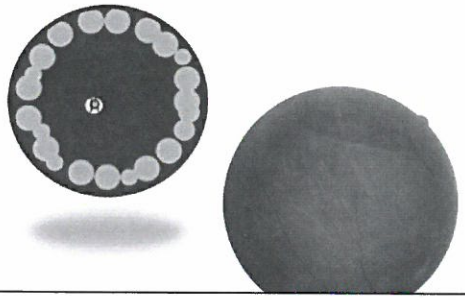


Location...Location...Location

Juxtabasilar breaks: Often this is a location for a FT **(Highest risk of RRD)**



She had continuous RB repairs with development of new ones
Final outcome...She received laser 360 OU



Possible Tear Treatment

- Cryotherapy
  - Transconjunctival COLD application seal
    -
  - destroys choriocapillaris, RPE/retina providing chorioretinal adhesion b/t tear and adjacent retina
    - subretinal space
  - Adhesion not immediate (1-3 wks required)

Possible Tear Treatment

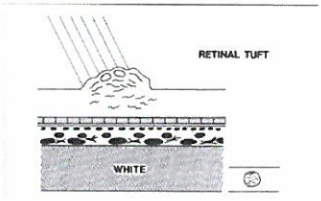
- Laser: chorioretinal adhesion occurs the instant that the laser photocoagulation is applied
  - maximal adhesion occurs 7–10 days later.
  - NOTE: Lightly pigmented pts often do NOT have naturally high pigment in the RPE & laser may be less effective

THE FACTS

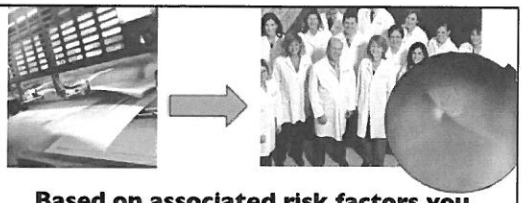
- 6-11% of the general population have RB(s)
  - 1 in 10,000 will lead to a RRD
- Incidence of RD is 12 in 100,000/yr

So the important Question is: "Which RB should we refer?"

Retinal tufts are small areas of gliotic or cystic degeneration of the retina associated with vitreous traction



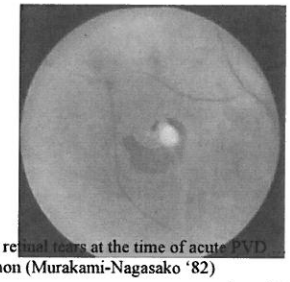
Traction during the PVD can lead to …



Based on associated risk factors you have read, would it be best to monitor or get a consult?

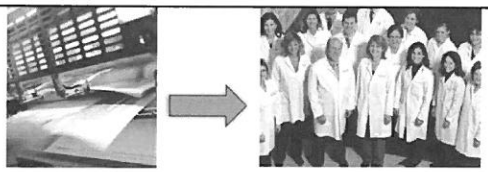
Our presentation: There's fluid & traction
Although <1% of tufts develop RRD,
10% of RD are associated with breaks near cystic tuft (Byer '81)

Retinal Tufts can lead to RBs



Tuft can lead to retinal tears at the time of acute PVD but is is uncommon (Murakami-Nagasako '82)

Courtesy of Dr. L Semes



Based on on the presentation, what would you do?

Would your management change if she was NOT MONOCULAR?

AOA guidelines for operculated holes:
6-12M follow-up

Operculated holes

Probably arise from *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...**

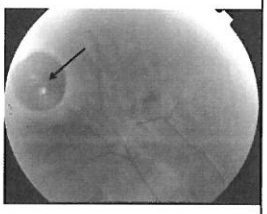
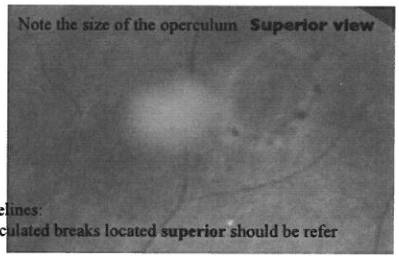


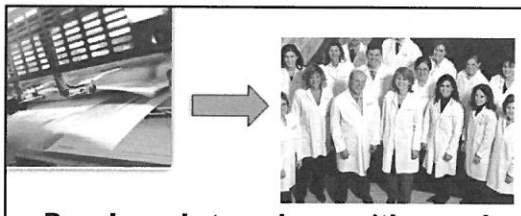
Photo courtesy: D. Hassenpflug, OD

59 WM: Language barrier issues

What to do?



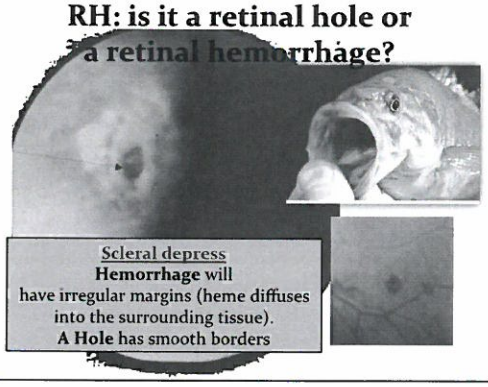
AOA guidelines:
Fresh operculated breaks located **superior** should be refer



Based on what you know with regards to pigment, would it be best to monitor or get a consult?

Pigment indicates it is **chronic** (Morse '75)
Follow yearly

RH: is it a retinal hole or a retinal hemorrhage?



Scleral depress
Hemorrhage will have irregular margins (heme diffuses into the surrounding tissue).
A Hole has smooth borders

Scleral depression: The questions

To view a more anterior structure or manipulate view of a hidden lesion

vitreoretinal abnormality in profile

DDx (retinal hemorrhage, a retinal tuft...)

Is there 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

What you say matters

Maximum dilation

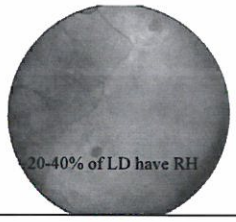
Place the instrument properly...CURVE WITH THE EYE & NOT ON AN ANGLE

Atrophic

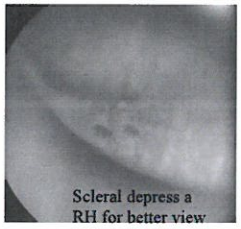
(Byer '74, Neumann '72)

This is NOT associated with vitreoretinal traction (PVD)

The pathogenesis is retinal thinning

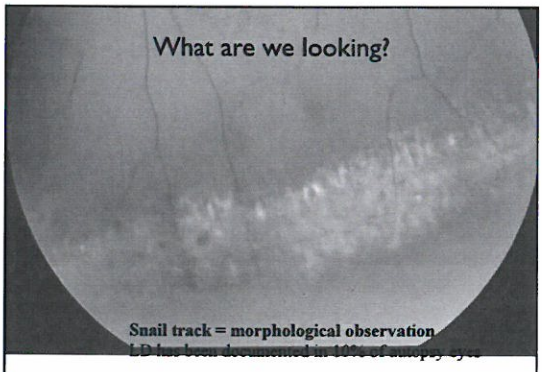


20-40% of LD have RH



Scleral depress a RH for better view

What are we looking?



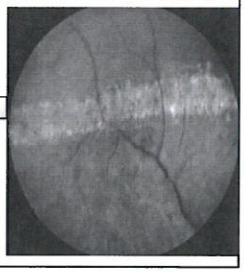
Snail track = morphological observation

LD has been documented in 10% of autopsy eyes

Lattice Degeneration as a Routine Finding

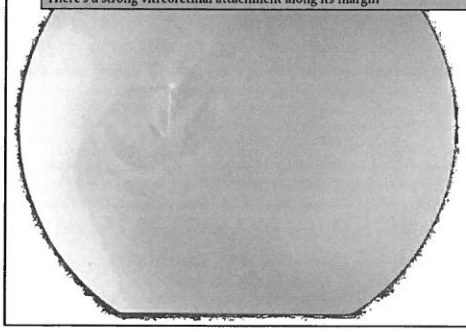
The chance of LD developing an RD is **1%**

How about if he had a hx of **contralateral RRD and** the pt was a **10D myope**?



Tear in a HIGH Myope with LD Hx

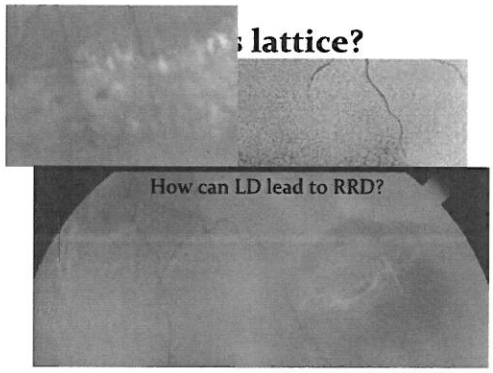
25% cases of LD in HIGH myopes have an associated break
There's a strong vitreoretinal attachment along its margin



True science teaches us to doubt...
Claude Bernard

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

lattice?



How can LD lead to RRD?

So, VR anomalies/breaks can cause RRD but decision to tx is based on which has the highest propensity towards the development of RRD

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

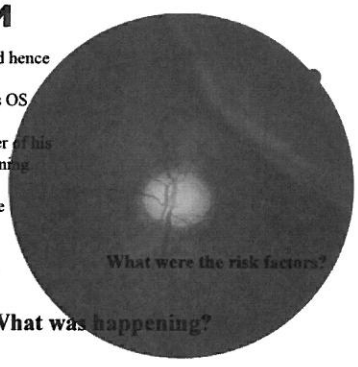
There is NO widespread agreement as to when to trt (**Retina 2007**):
clinical & scientific evidence for laser tx of RB only exists for a few particular situations

Feature	Atrophic hole	Operculated tear	Flap tear
Shape	Round or oval	Round with disc shape operculum floating above the break	U shape with central flap
Vitreous traction			
Location	Far periphery	Far to mid periphery	Far to mid periphery
Retinal or vitreous haemorrhages	Never	Rarely	Often
Symptoms (flashes, floaters)	Never (unless clinically significant RD occurs)	Possible in traction phase (or if clinically significant RD occurs)	Frequent in traction phase (or if clinically significant RD occurs)
Incidence of RD	Rare	1/6[9] (much less if asymptomatic)	1/3[9] To 1/5
Prophylactic treatment of break	Never (for exceptions see lattice)	Rarely	typically

54 BM

CC: Blur vision and hence wants to glasses
Floaters X 4-5 days OS
(-) Flashes
Unable to see corner of his nose since this morning

POHx: 10D Myope
Glaucoma suspect
Trauma X 10 yrs
2 Retinal holes OS
(Dxed last year)



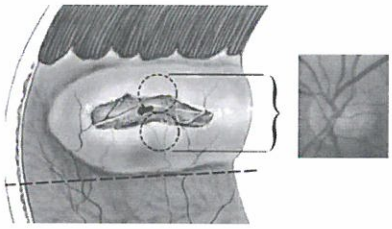
What were the risk factors?

What was happening?

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

Guidelines for tx RB:

AOA Guideline
Symptomatic retinal breaks deserve a consultation

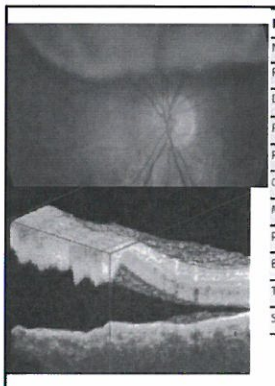
Is it symptomatic?

- Symptom is the most critical prognostic ***criteria determining the likelihood the RB will progression towards RRD***
  - >~30% **of *symptomatic* untxed RBs in phakic eyes 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***
    - ***(Neumman '72: <5% progress) (Davis '74)***
    - ***(Byer '82 n=231 X 1yr)***
    - **Yet...**

Is this a schisis or a RRD?

Retinoschisis
Split within the neurosensory retina
Smooth, shiny **translucent**
Stationary on eye movement
No flashes or floaters
Bilateral usually
7% of population >40 years
70% hypermetropes
Stable absolute scotoma
No breaks **?Holes**
No tobacco dust
Infero-temporal commonest

Likely may be monitored



Rhegmatogenous Retinal detachment
Neurosensory retina separates from RPE
Rippled, irregular **folds**
Drifts or undulates with eye movements
Flashes, floaters, cobwebs
Rarely bilateral simultaneously
0.1% of population
Myopes over-represented
Progressive relative scotoma
Break present (may be difficult to locate)
Tobacco dust
Superior-temporal commonest

REFERAL

When dealing with a RB there are modified strategies based on risk factors

- trauma?
  - *80% of traumatic RBs (in one study) were associated with development of a RRD*
    - *Johnson 1991*
- Is the pt a moderate-high myope (>6D)?
  - ~40% of phakic RD's are seen in myopes
    - *grp '93*

OTHER risk factors

- *Is the pt* pseudophakia or aphakic?
  - to 40% of RDs but incidence is ~0.6-1.7%
  - *higher closer to the time after surgery,*
    - Younger age, complication during CE surgery, high myopia (>26mm axial length), absence of PVD before surgery
- Has the pt had a yag?
  - *Ambler 1988: increase risk <2%*

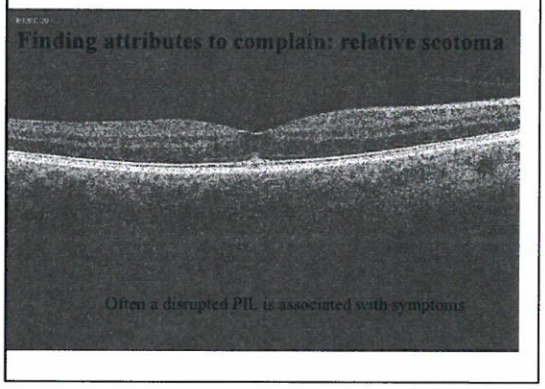
When dealing with a RB there are modified strategies based on risk factors

- 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?
  - Marfan's syndrome, Ehlers-Danlos syndrome, Wagner's syndrome and Stickler's syndrome

Risk Factors: A Review

RB & risk factors that may increase progression towards RRD

- Fluid
- Traction
- Symptomatic
- Trauma
- Myopia
- RD in contralateral eye
- Recent cataract surgery or others
- FHx of RD
- Size & location (Sup) & lifestyle also taken into account



What is the best

Monitor or fill out a

Based on the what you read regarding the evolution of the disease, do you monitor or refer?

RTC q3-6M (VA 20/20)

May remain stable or even resolve

VMT may be relative stable

BPEI study ARVO 2012

N= 38 eyes

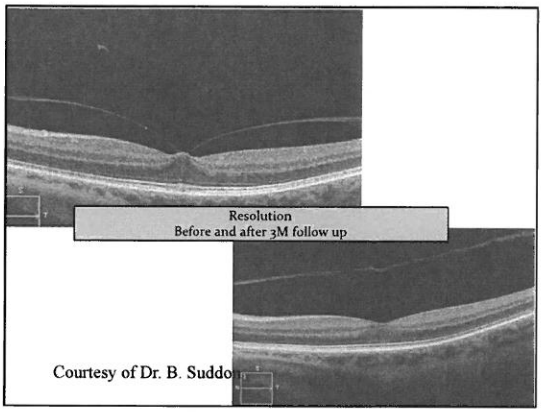
Grades 1-3 VMT w mean BCVA 20/50

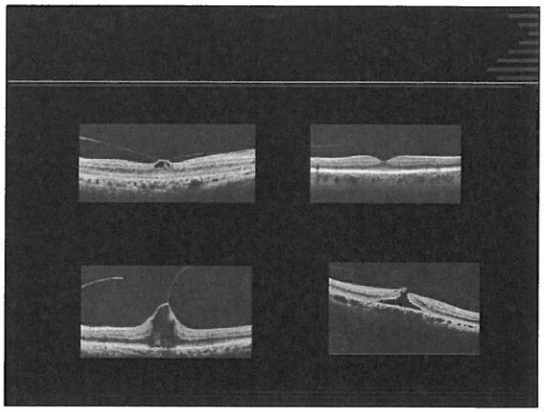
1. VMT
2. cystic
3. fluid

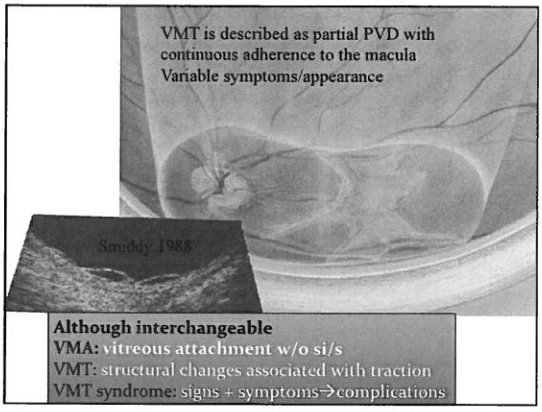
Mean BCVA 20/40

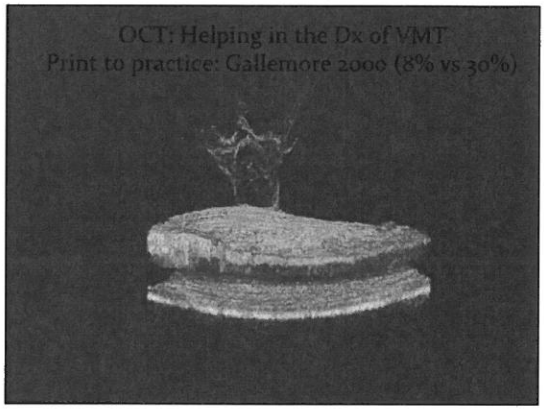
64% (24 eyes) remain stable

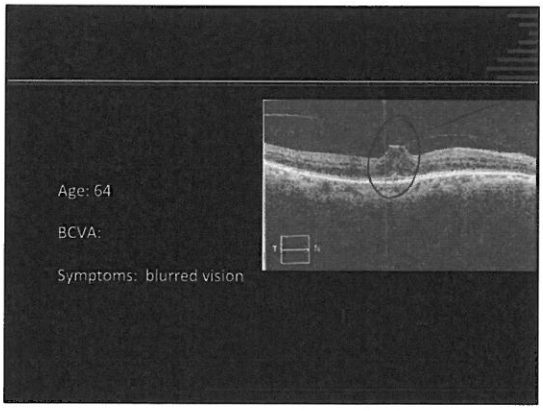
13% (5 eyes) progress & rest progress

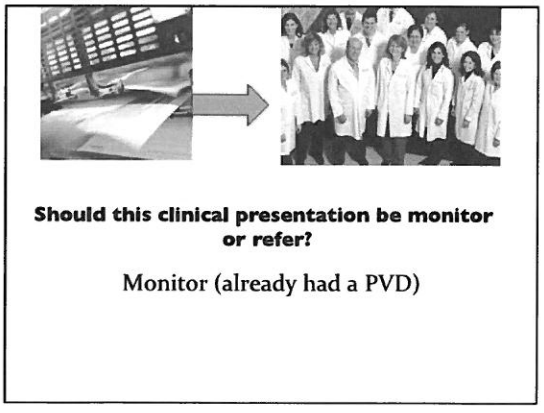

Resolution
Before and after 3M follow up
Courtesy of Dr. B. Suddo




VMT is described as partial PVD with
continuous adherence to the macula
Variable symptoms/appearance
Smiddy 1988
Although interchangeable
VMA: vitreous attachment w/o si/s
VMT: structural changes associated with traction
VMT syndrome: signs + symptoms→complications


OCT: Helping in the Dx of VMT
Print to practice: Gallemore 2000 (8% vs 30%)


Age: 64
BCVA:
Symptoms: blurred vision


Should this clinical presentation be monitor or refer?
Monitor (already had a PVD)

VMT
Print to practice Smiddy 1990

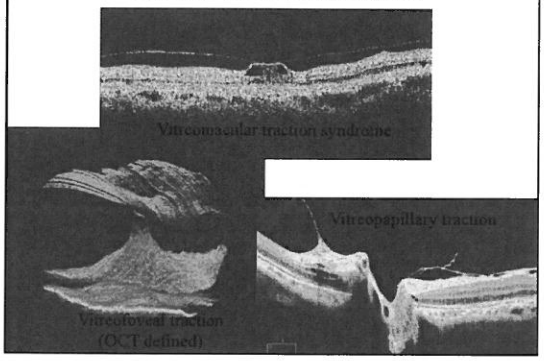
VMT may play a role in the pathogenesis of a number of maculopathies

- Cystoid macular edema (CME)
  - MOST COMMON
- Macula hole formation (MH)
- Epiretinal membrane (ERM)
- Exacerbates of macular edema
  - Macular retinoschisis or detachment
  - Myopic foveoschisis
  - DME (pts w DME are less likely to have a complete PVD)
  - AMD (eyes w wet AMD commonly don't have a complete PVD)

VMT
Print to practice Smiddy 1990

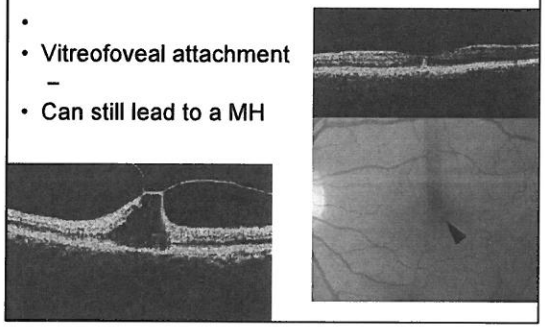
It is classified as a "SPECTRUM" group of disorders caused by incomplete posterior vitreous detachment (PVD) varying in adhesion size/ force

Variable tractional locations

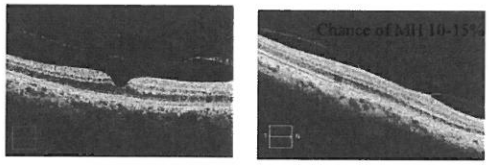


Vitreofoveal Traction

-
- Vitreofoveal attachment
  –
- Can still lead to a MH



Both associated with a contralateral MH4…How does management differ?



No real MH chance

MH

2x more common among women

Unilateral>>>bilateral

Idiopathic

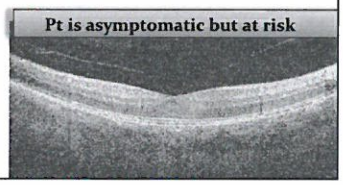
Symptoms may be sudden or gradual

The role of minimal surface traction

Stage "o" Chan Opth 2004

Central posterior vitreous adhesion to fovea

Increase risk of developing MH (10%)



WHEN FOLLOWING A VMT or early MH, IT SHOULD BE DONE INITIALLY IN SHORT INTERVALS…**4 WKS…much like an acute PVD**

Stages of Macular Holes

Gass 1988

I:Pseudocyst associated with traction

Note the outer retinal layers

IA: foveal detachment (macular cyst)

Yellow dot stage

IB: Loss of foveal depression

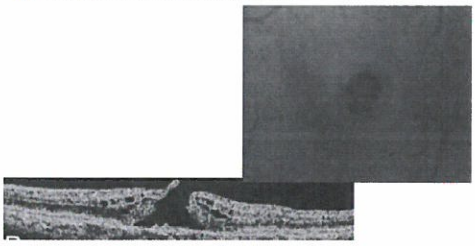
Donut shaped yellow ring

VA 20/20- → 2070

Stages of Macular Holes

Gass 1988

II: Partial tear in the sensory retina



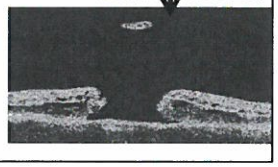
Stages of Macular Holes

Gass 1988

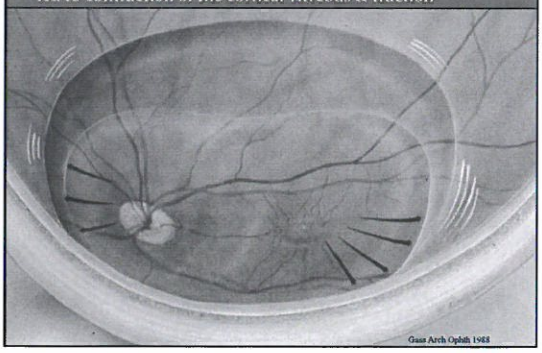
III: Full thickness macular hole

IV: Macular hole + complete PVD

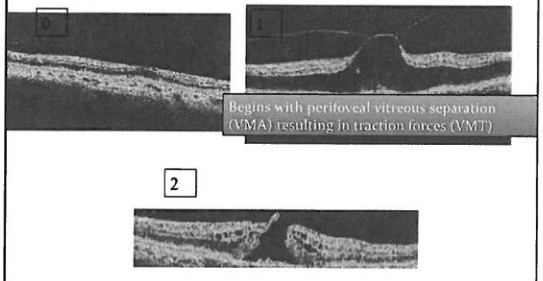
What's the difference?



Old theory: focal shrinkage of vitreous in the macula led to contraction of the cortical vitreous & traction

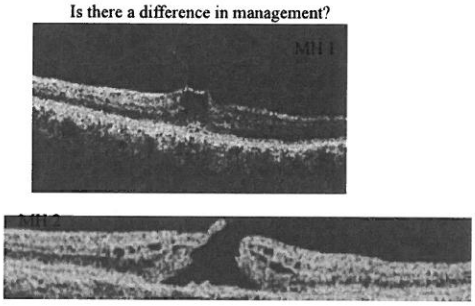


OCT has expanded our knowledge about macular hole pathogenesis
Hee 1995, Gaudrin 1999, Ito 2003

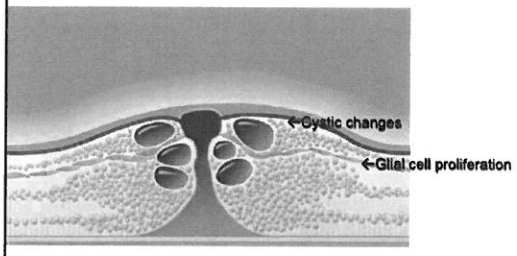


Lets talk optometry: Which is better 1 or 2?

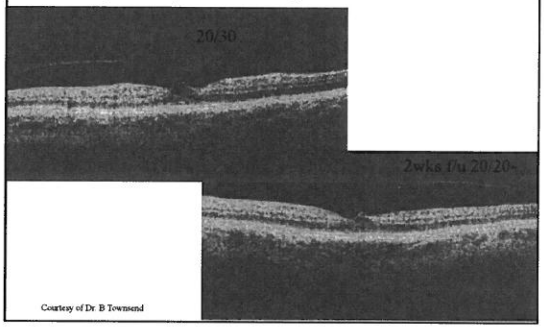
Is there a difference in management?

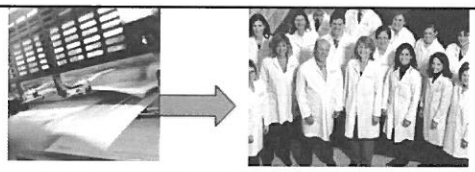


Glial cell proliferation may be associated with spontaneous closure of a hole



Is this a potential intermittent tx?
Pre and post Topical NSAID QID X 2 wks

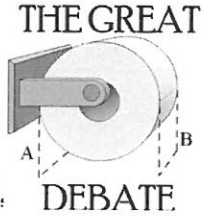




Based upon what you know with regards to a stage 4, what should you do?

REFER
Kelly 1991 (successful tx of MH)
Friedman: Vitrectomy for Treatment of Macular Hole Study Group '97
Visual benefit s/p PPV (for MH 'stage 2-4')
After stage 2, there's <10% of spontaneous resolution

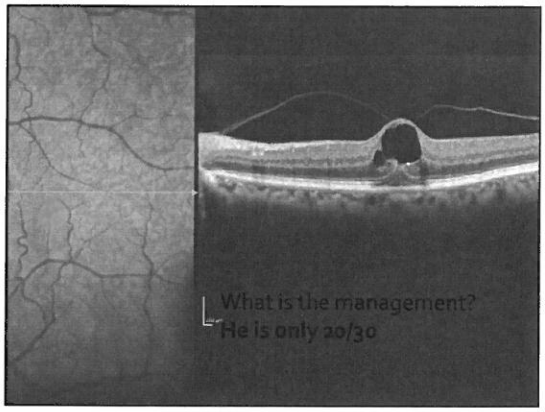
Face down



- Elemenst
  - For yrs we use to believe that best prognosis was w face down position from 10-14 days
  - Recent reports state 7 days is enough
  - <500 um MH may have good prognosis with only semi seated position
  - The GAS may have impact on face down position
    - Short vs long acting gas used
    - Proper gas-air ratio

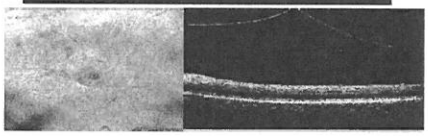
Is facedown position a necessity?
The wine glass phenomena

It has been proposed by tormabre recently that
Cases of complete closure can occur in a lrg pts w/o prone position



What is the management?
He is only 20/30

What is the best



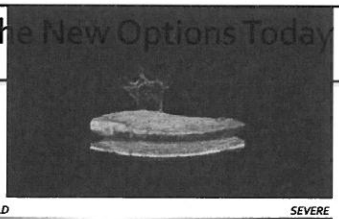
REFER:
Given it may progress (associated with various complications)

MONITOR
Given it may resolve
Previous studies stated 10% resolution but w OCT more recent studies have found up to 40%
Or may even remain stable

Remember risks vs benefits

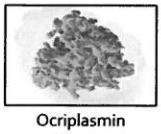
LET US NOT FORGET THE BURDEN ON THE PT. regardless of some (limited cases) reporting full closure w/o prone position, most surgeons advise pts to keep a head down position for 3-7 days

The New Options Today

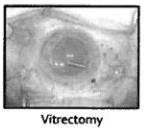


MILD SEVERE

Clinical Monitoring

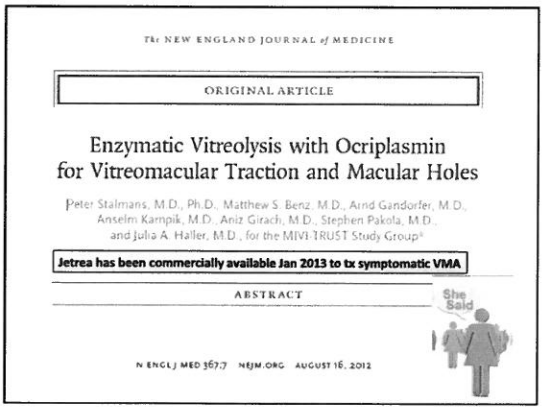


Ocriplasmin
(Jetrea)



Vitrectomy

77



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Enzymatic Vitreolysis with Ocriplasmin for Vitreomacular Traction and Macular Holes

Peter Stalmans, M.D., Ph.D., Matthew S. Benz, M.D., Arnd Gandorfer, M.D., Anselm Kampik, M.D., Aniz Girach, M.D., Stephen Pakola, M.D., and Julia A. Haller, M.D., for the MIVI-TRUST Study Group*

Jetrea has been commercially available Jan 2013 to tx symptomatic VMA

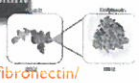
ABSTRACT

She Said

N ENGL J MED 367;7 NEJM.ORG AUGUST 16, 2012

WHAT IS microplasmin

an active molecule similar to plasmin

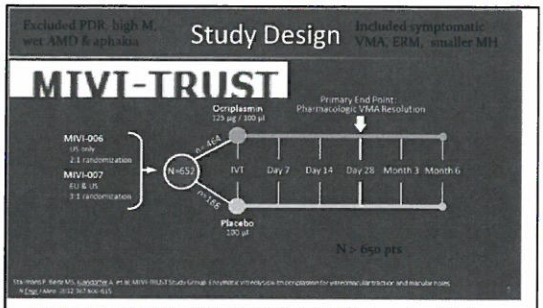


Truncated form of active PLASMIN

recombinant protease with activity against fibronectin/ lamin (components of VR interference)

pharmacological induced vitreolysis

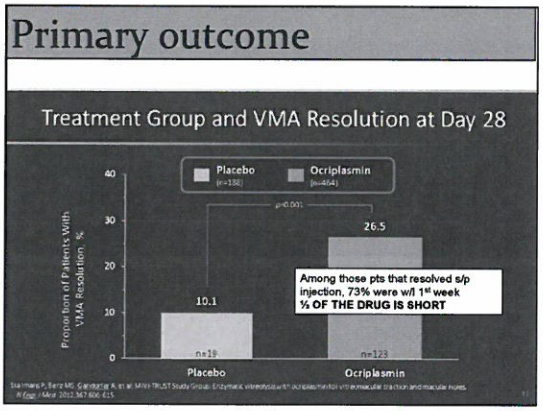
- Nonsurgical PVD
- The enzymatic agents alter the biochemistry of vitreous
  - Liquefaction of the vitreous occurs
  - LYSIS between vitreous cortex and ILM is the final outcome

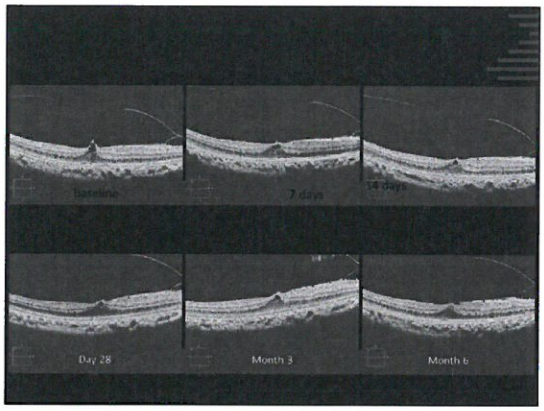


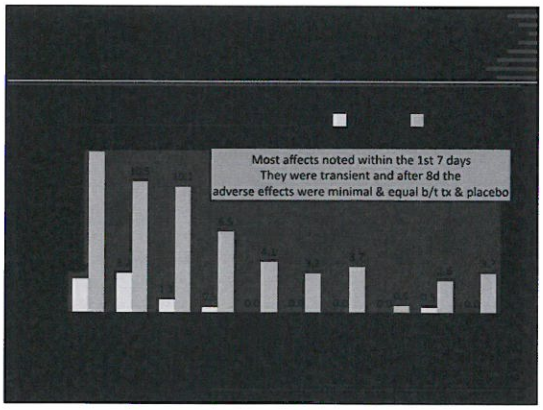
Inclusion: VMT w VA $\leq$ 20/25 & OCT showing thickness

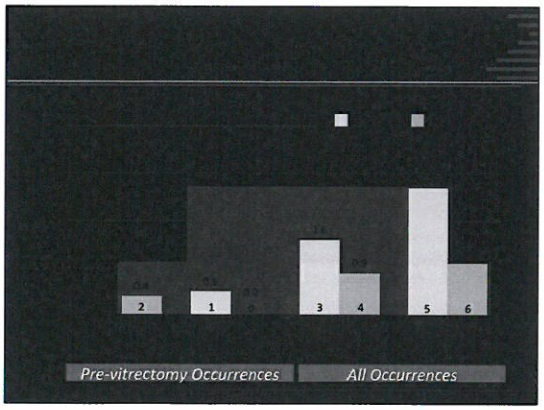
PPV advised if:

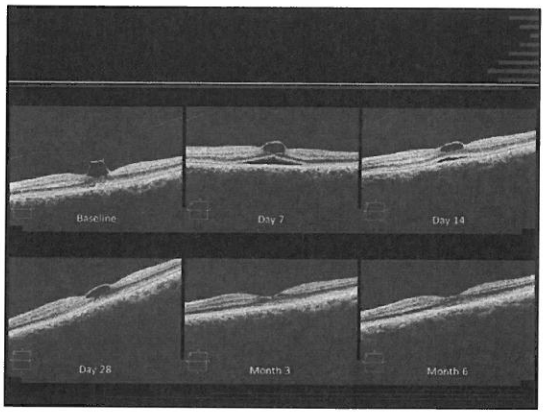
- MD deemed it to be necessary s/p 1M
- VA worsen by 2 lines or No improvement s/p tx





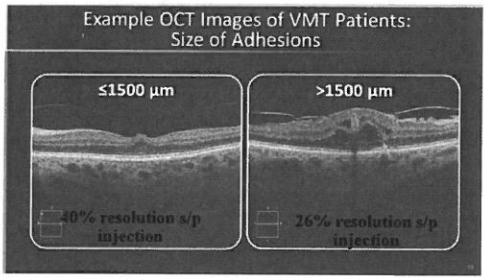


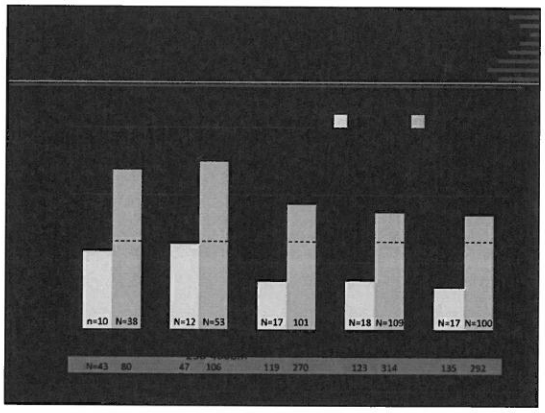




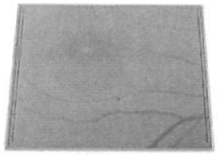
The right pt selection is important

Example OCT Images of VMT Patients:
Size of Adhesions

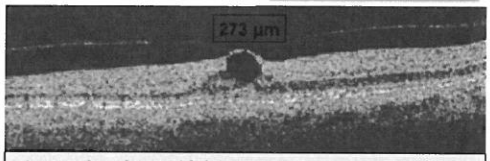




So Is the pt the RIGHT pt for JETREA?



64yo phakic



Increasing the # of (+) features was associated with increase odds VMA resolution

It is all about the dynamics of VR adhesion

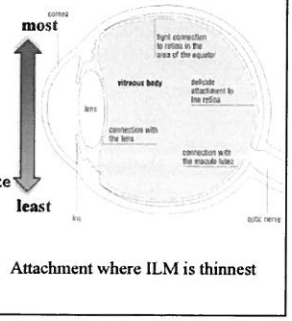
Vitreous base

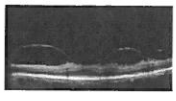
Optic nerve

Macula

Blood vessels

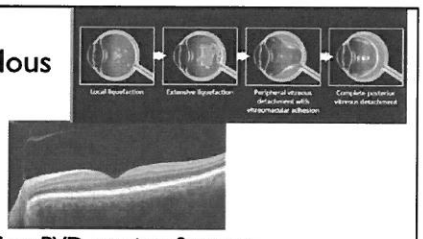
General vitreous-retina interface





Attachment where ILM is thinnest

Anomalous PVD



Complete PVD requires 2 steps:

Vitreous liquefaction

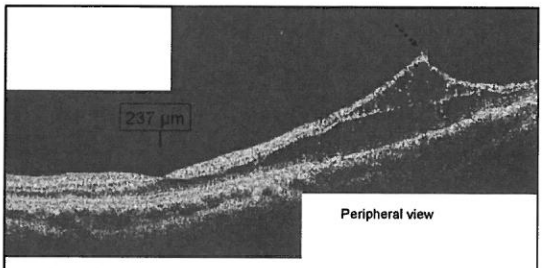
Separation of the vitreous from the ILM

What happens when 1 step occurs without the other...

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	Operculasted Holes/ Breaks RRD



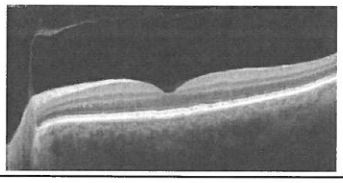
Myopia, inflammation, trauma, inherited VR disease, & other conditions may accelerate liquefaction of the Vitreous in YOUNGER pts (and hence increase the risk of RD/RB)

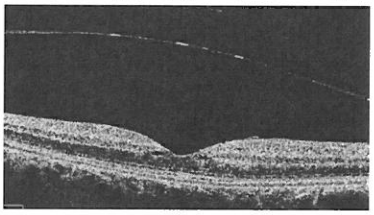
1 in 6 symptomatic PVDs are associated with a RB/RD

DFE

What's the Dx?

What do you needs to be rule out?





Acute PVD is seen

When do you bring pt back?

How long is follow up?

How about this case?

PVD is NOT seen but pt has symptoms…

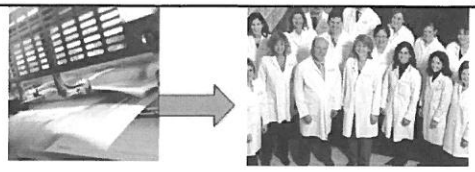
When should you see them back?

REVIEW

- Dilated fundus exam is a MOST! (also consider SD)
- Close Follow-Up
  - f/u in 1-6 wks depend on F/F
    - Then 3-6month →yrly (follow till photopsia resolve: **often described as a temporal arc)**
    - Highest threat is within 1st 3M
  - 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

Regarding PVD disorders, which of the following increases the likelihood of a concomitant RB?

A. Retinal Tuft
B. Pigmented cells in the vitreous
C. Syneresis in the vitreous
D. Pigment on the retina



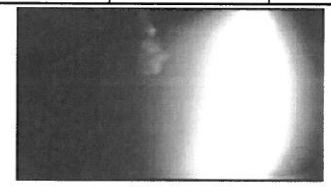
Based on what you read how would you management this case?

>80% of pts with pigmented cells in the vitreous present with a RB (JAMA '09)

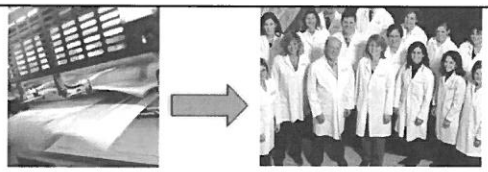
From PRINT→PRACTICE
Pigmented cells vitreous

Br J Ophthalmol 2000;84:1264

115 Eyes With RRD	**96.5% had a PVD**	**96% had Pigmented cell in the vitreous**



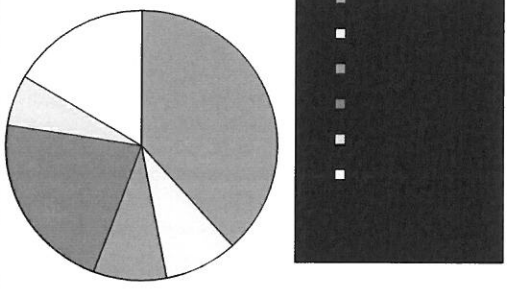
Courtesy of Dr L Alexander



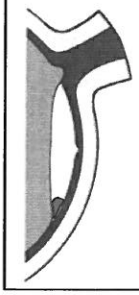
Based on what you read with regards to VH +PVD, what is the best management?

10% of PVD+VH cases can co-exits WITHOUT a RB
Yet, 50-70% of pts w/ PVD + VH also have a RB
Sharma '04

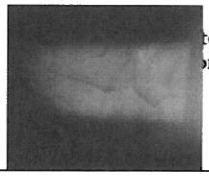
Causes of VH



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



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on

J Sowka NSUCO

Clinical Exam of a Patient with A Symptomatic PVD

- FULL Dilated fundus exam **(use PE/tropicamide)**
- Look specifically at the vitreous
  - +/- pigmented cells in the anterior vitreous
  - +/- VH