From Print to Practice:
PVD a common process with potential for ocular morbidity
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The PVD process may not be abrupt & likely starts earlier on

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

Horseshoe or U-shaped Flap Tear (FT) following a PVD
The base faces the Anterior retina
The apex faces the Posterior retina

Based on relationship b/t RB & RD, would you Hold' em or Fold' em?
Symptomatic FTs are always treated!

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.
Flap tears

This is a full thickness retinal break with an attached flap (TEAR), resulting from abnormally strong vitreoretinal adhesion.

50% of flap tears are located superior.

Location...Location...Location

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

Vitreous base: intrabasal
Juxtabasal
Extrabasal

Also take into consideration Superior retinal breaks.

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 Linder 1934.
Late '50s, 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 followed.

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 <0.5% (Foos '74) (Byer '86)
Most are followed

Operculated holes

Probably arise from cystic retinal tufts
- Commonly follows vitreous traction, which may brought upon by a PVD
- V8% associated with PVD

Generally asymptomatic and stable but there are exceptions...
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

Pigment 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 intimating
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 3%
- 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

   Editorial by Norman Byer, M.D. in same issue
2. Chauhan et al. Failure of Prophylactic Retinopexy. Arch Ophth 7/06

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

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

<table>
<thead>
<tr>
<th>Retinal break</th>
<th>Likelihood of treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flap tears</td>
<td>Frequently, may deserve a consult</td>
</tr>
<tr>
<td>Operculated holes</td>
<td>Sometimes, referral depends on symptoms, traction, etc</td>
</tr>
<tr>
<td>Atrophic holes</td>
<td>Not likely, look at risk factors</td>
</tr>
</tbody>
</table>

What is a SUBclinical RD?

Subclinical RD: fluid extends >1DD from the RH but NOT >2DD posterior to the equator (Davis ’73)
>30% become CLINICAL RD (Davis ’74)

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:

- Is it symptomatic?
  - Symptom is a critical prognostic criteria towards RRD progression
  - ~30% of symptomatic untreated RBs lead to a RRD
  - Colyear ’56 & ’60, Davis ’73, Shea ’74
  - Asymptomatic RBs do not show any significant tendency towards RD
  - Byer 1998: sub cases (~10% PT) → subclinical RRD
  - Neumman ’73 (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

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### 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

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### 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
An incomplete PVD associated with continuous adherence is known as VMT (vitreomacular traction).

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

Smiddy 1988

Vitreofoveal Traction

- OCT defined
- Vitreofoveal attachment

The role of minimal surface traction
Stage “0” Chan Opth 2004

Central posterior vitreous adhesion to fovea
Increase risk of developing MH (10%)
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

OCT has expanded our knowledge about macular hole pathogenesis


MH - Spontaneous closure

3M later: spontaneous closure
MH 3: 20/70
20/40 BCVA
Glial plug & RPE △s

Glial cell proliferation & closure of a hole

MH - Spontaneous reopening

20/40 BCVA, glial remnant (Sup)
Note surrounding fluid
S/P: surgically repaired 20/20
**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**

- Is a recombinant truncated form of human plasmin that is active
  - Functions as a thrombolytic agent causing an enzymatic 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 ≤ 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

**What about this case? Would you refer given the fact it is 20/50?**

Stage 3-4 require tx in order to improve VA
Where are vitreous and retina most and least adherent?

<table>
<thead>
<tr>
<th>VR traction site</th>
<th>Retinal condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retinal vasculature</td>
<td>Retinal or vitreous hemes</td>
</tr>
<tr>
<td>Avulsed retinal vessel</td>
<td></td>
</tr>
<tr>
<td>Macula</td>
<td>VMT syndrome &amp;/or MH</td>
</tr>
<tr>
<td>Periphery</td>
<td>Operculated Holes/ Breaks</td>
</tr>
<tr>
<td>RRD</td>
<td></td>
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VR conditions associated with PVD process

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

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
- Educate about onset of news si/s

From PRINT→PRACTICE

Pigmented cells vitreous

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

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

Posterior Vitreous Detachment

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

Courtesy of Dr. L. Alexander
Because of gravity, VH have a tendency to settle inferiorly, an associated RB is not necessary at the location of the VH.