Q

- Patients with a PDA are at increased risk of ROP
Patients with a PDA are at increased risk of ROP: True

Birth weight is a greater predictor for ROP than O2 exposure: True; LBW is #1 risk factor

Exposure to ambient light has a small but significant effect on ROP: False; the Light-ROP study found no relationship

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The sexes have roughly equal ROP incidence rates: False; ROP is significantly more common in males

Once the ROP process starts, it usually progresses to an advanced level: False; roughly 80% of ROP arrests spontaneously, without significant sequelae

ROP: True or false

True
Q

- Patients with a **PDA** are at increased risk of ROP True

Why would a Personal Digital Assistant* put someone at increased risk of ROP?

*Do y'all know what a *Personal Digital Assistant* is?
Patients with a **PDA** are at increased risk of ROP **True**

Why would a *Personal Digital Assistant* put someone at increased risk of ROP? In this context, PDA stands for **quack**.
Patients with a PDA are at increased risk of ROP True

Why would a Personal Digital Assistant put someone at increased risk of ROP? In this context, PDA stands for **patent ductus arteriosus**
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Note the pattern…
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Is exposure to supplemental O2 a risk factor at all?
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There is another risk factor that is tied for #1 with LBW—what is it?

LBW is #1 risk factor along with...?
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**Development of significant ROP correlates best with the infant’s postmenstrual age.**

Postmenstrual age equals **one way of measuring infant age** + **another age**.
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What does this indicate about the relationship between timing of ROP development and an infant’s chronologic age? It indicates that younger preemies take longer to develop significant ROP than do older preemies.
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It indicates that younger preemies take longer to develop significant ROP than do older preemies. Consider two infants, one born at gestational age 24 weeks, the other at 27. Neither is expected to develop ROP before postmenstrual age 31 weeks. Thus, the 24-weeker needs to be examined at chronologic age 7 weeks (24+7=31), whereas the 27-weeker should be examined at chronologic age 4 weeks (27+4=31). (We’ll have more to say later about ROP screening, and its timing.)
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What does the term Everest in utero have to do with ROP?
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The term highlights the fact that the gestational environment is profoundly hypoxic compared to life ex utero. Oxygen levels in utero are about what they are at the 26,000 ft level on Everest—the so-called ‘death zone.’ It is under these O₂ conditions that the retinal vasculature is supposed to develop.
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What does this suggest about premature birth and the pathophysiology of ROP?
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The term highlights the fact that the gestational environment is profoundly hypoxic compared to life ex utero. Oxygen levels in utero are about what they are at the 26,000 ft level on Everest—the so-called ‘death zone.’ It is under these $O_2$ conditions that the retinal vasculature is supposed to develop.

What does this suggest about premature birth and the pathophysiology of ROP?
When the preemie experiences normal ex utero $O_2$ levels, further development of the retinal vasculature is suppressed. This leaves more peripheral retinal areas with inadequate oxygenation.
A

ROP: True or false

- Patients with a PDA are at increased risk of ROP True
- Birth weight is a greater predictor for ROP than $O_2$ exposure True; LBW is #1 risk factor
- Exposure to ambient light has a small but significant effect on ROP development False; the *Light-ROP* study found no relationship
- Infants with a R→L cardiac shunt (and subsequent low $O_2$ sat) are protected from ROP False, and this provides strong evidence that excess $P_aO_2$ is not causative

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What does this suggest about premature birth and the pathophysiology of ROP?
When the preemie experiences normal ex utero $O_2$ levels, further development of the retinal vasculature is suppressed. This leaves more peripheral retinal areas with inadequate oxygenation. As they mature, these hypoxic retinal cells do what hypoxic retinal cells tend to do—they produce VEGF. The result is the now-familiar cascade of neovascularization, bleeding, and tractional retinal detachment.
Patients with a PDA are at increased risk of ROP True

Birth weight is a greater predictor for ROP than O₂ True; LBW is #1 risk factor

Exposure to ambient light has a small but significant effect on ROP development False; the Light-ROP study found no relationship

Infants with a R→L cardiac shunt (and subsequent low O₂ sat) are protected from ROP False, and this provides strong evidence that excess PaO₂ is not causative

In other words, ROP is a biphasic disease:

---

(No question yet—keep going)

---

strong evidence that excess PₐO₂ is not causative

*What does the term Everest in utero have to do with ROP?*

The term highlights the fact that the gestational environment is profoundly hypoxic compared to life ex utero. Oxygen levels in utero are about what they are at the 26,000 ft level on Everest—the so-called ‘death zone.’ It is under these O₂ conditions that the retinal vasculature is supposed to develop.

*What does this suggest about premature birth and the pathophysiology of ROP?*

When the preemie experiences normal ex utero O₂ levels, further development of the retinal vasculature is suppressed. This leaves more peripheral retinal areas with inadequate oxygenation. As they mature, these hypoxic retinal cells do what hypoxic retinal cells tend to do—they produce VEGF. The result is the now-familiar cascade of neovascularization, bleeding, and tractional retinal detachment.
Patients with a PDA are at increased risk of ROP True

Birth weight is a greater predictor for ROP than O2 True; LBW is #1 risk factor

Exposure to ambient light has a small but significant effect on ROP development False; the Light-ROP study found no relationship

Infants with a R→L cardiac shunt (and subsequent low O2 sat) are protected from ROP False, and this provides strong evidence that excess PaO2 is not causative

In other words, ROP is a **biphasic disease:**

*(What happens first?)*

strong evidence that excess P_{a}O_{2} is not causative

*What does the term Everest in utero have to do with ROP?*

The term highlights the fact that the gestational environment is profoundly hypoxic compared to life ex utero. Oxygen levels in utero are about what they are at the 26,000 ft level on Everest—the so-called ‘death zone.’ It is under these O2 conditions that the retinal vasculature is supposed to develop.

*What does this suggest about premature birth and the pathophysiology of ROP?*

When the preemie experiences normal ex utero O_{2} levels, further development of the retinal vasculature is suppressed. This leaves more peripheral retinal areas with inadequate oxygenation. As they mature, these hypoxic retinal cells do what hypoxic retinal cells tend to do—they produce VEGF. The result is the now-familiar cascade of neovascularization, bleeding, and tractional retinal detachment.
Patients with a PDA are at increased risk of ROP True
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Exposure to ambient light has a small but significant effect on ROP development False; the Light-ROP study found no relationship
Infants with a R$\rightarrow$L cardiac shunt (and subsequent low $O_2$ sat) are protected from ROP False, and this provides strong evidence that excess $P_aO_2$ is not causative

In other words, ROP is a biphasic disease:
--First, premature birth (+/- supplemental $O_2$) exposes the immature retina to vastly higher-than-normal $O_2$ levels, leading to downregulation of VEGF. This causes the immature retinal vascular tree to stop proliferating.

What does the term Everest in utero have to do with ROP?
The term highlights the fact that the gestational environment is profoundly hypoxic compared to life ex utero. Oxygen levels in utero are about what they are at the 26,000 ft level on Everest—the so-called ‘death zone.’ It is under these $O_2$ conditions that the retinal vasculature is supposed to develop.

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When the preemie experiences normal ex utero $O_2$ levels, further development of the retinal vasculature is suppressed. This leaves more peripheral retinal areas with inadequate oxygenation. As they mature, these hypoxic retinal cells do what hypoxic retinal cells tend to do—they produce VEGF. The result is the now-familiar cascade of neovascularization, bleeding, and tractional retinal detachment.
In other words, ROP is a **biphasic disease**:
--First, premature birth (+/- supplemental O₂) exposes the immature retina to vastly higher-than-normal O₂ levels, leading to **downregulation of VEGF**. This causes the immature retinal vascular tree to **stop proliferating**.

*(What happens later?)*

---

**Strong evidence that excess PₐO₂ is not causative**

*What does the term Everest in utero have to do with ROP?*
The term highlights the fact that the gestational environment is profoundly hypoxic compared to life ex utero. Oxygen levels in utero are about what they are at the 26,000 ft level on Everest—the so-called ‘death zone.’ It is under these O₂ conditions that the retinal vasculature is supposed to develop.

*What does this suggest about premature birth and the pathophysiology of ROP?*
When the preemie experiences normal ex utero O₂ levels, further development of the retinal vasculature is suppressed. This leaves more peripheral retinal areas with inadequate oxygenation. As they mature, these hypoxic retinal cells do what hypoxic retinal cells tend to do—they produce VEGF. The result is the now-familiar cascade of neovascularization, bleeding, and tractional retinal detachment.
Patients with a PDA are at increased risk of ROP True

Birth weight is a greater predictor for ROP than O₂

In other words, ROP is a **biphasic disease**: 
--First, premature birth (+/- supplemental O₂) exposes the immature retina to vastly higher-than-normal O₂ levels, leading to **downregulation of VEGF**. This causes the immature retinal vascular tree to **stop proliferating**.

--Later, the (unvascularized) peripheral retina becomes metabolically active. The lack of vascularization renders the peripheral retina hypoxic, leading to **upregulation of VEGF**. This causes the vascular tree to **start proliferating again**.

strong evidence that excess PₐO₂ is not causative

**What does the term Everest in utero have to do with ROP?**
The term highlights the fact that the gestational environment is profoundly hypoxic compared to life ex utero. Oxygen levels in utero are about what they are at the 26,000 ft level on Everest—the so-called ‘death zone.’ It is under these O₂ conditions that the retinal vasculature is supposed to develop.

**What does this suggest about premature birth and the pathophysiology of ROP?**
When the preemie experiences normal ex utero O₂ levels, further development of the retinal vasculature is suppressed. This leaves more peripheral retinal areas with inadequate oxygenation. As they mature, these hypoxic retinal cells do what hypoxic retinal cells tend to do—they produce VEGF. The result is the now-familiar cascade of neovascularization, bleeding, and tractional retinal detachment.
 Patients with a PDA are at increased risk of ROP True
Birth weight is a greater predictor for ROP than O₂ exposure True; LBW is #1 risk factor
Exposure to ambient light has a small but significant effect on ROP development False; the Light-ROP study found no relationship
Infants with a R→L cardiac shunt (and subsequent low O₂ sat) are protected from ROP False, and this provides strong evidence that excess PₐO₂ is not causative
Whites have a greater risk of ROP than blacks
Patients with a PDA are at increased risk of ROP True
Birth weight is a greater predictor for ROP than $O_2$ exposure True; LBW is #1 risk factor
Exposure to ambient light has a small but significant effect on ROP development False; the Light-ROP study found no relationship
Infants with a $R \rightarrow L$ cardiac shunt (and subsequent low $O_2$ sat) are protected from ROP False, and this provides strong evidence that excess $P_a O_2$ is not causative
Whites have a greater risk of ROP than blacks True
Q

- Patients with a PDA are at increased risk of ROP True
- Birth weight is a greater predictor for ROP than O₂ exposure True; LBW is #1 risk factor
- Exposure to ambient light has a small but significant effect on ROP development False; the Light-ROP study found no relationship
- Infants with a R→L cardiac shunt (and subsequent low O₂ sat) are protected from ROP False, and this provides strong evidence that excess PₐO₂ is not causative
- Whites have a greater risk of ROP than blacks True
- The sexes have roughly equal ROP incidence rates
Q/A

- Patients with a PDA are at increased risk of ROP True
- Birth weight is a greater predictor for ROP than $O_2$ exposure True; LBW is #1 risk factor
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- Whites have a greater risk of ROP than blacks True
- The sexes have roughly equal ROP incidence rates False; ROP is significantly more common in M vs F
Patients with a PDA are at increased risk of ROP True
Birth weight is a greater predictor for ROP than $O_2$ exposure True; LBW is #1 risk factor
Exposure to ambient light has a small but significant effect on ROP development False; the Light-ROP study found no relationship
Infants with a R→L cardiac shunt (and subsequent low $O_2$ sat) are protected from ROP False, and this provides strong evidence that excess $P_a O_2$ is not causative
Whites have a greater risk of ROP than blacks True
The sexes have roughly equal ROP incidence rates False; ROP is significantly more common in males
Patients with a PDA are at increased risk of ROP True
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Infants with a R→L cardiac shunt (and subsequent low O₂ sat) are protected from ROP False, and this provides strong evidence that excess PₐO₂ is not causative
Whites have a greater risk of ROP than blacks True
The sexes have roughly equal ROP incidence rates False; ROP is significantly more common in males
Once the ROP process starts, it usually progresses to an advanced level
Patients with a PDA are at increased risk of ROP True
Birth weight is a greater predictor for ROP than O₂ exposure True; LBW is #1 risk factor
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Whites have a greater risk of ROP than blacks True
The sexes have roughly equal ROP incidence rates False; ROP is significantly more common in males
Once the ROP process starts, it usually progresses to an advanced level False; roughly 80% of ROP arrests spontaneously, without significant sequelae
Patients with a PDA are at increased risk of ROP True

Birth weight is a greater predictor for ROP than O₂ exposure True; LBW is #1 risk factor

Exposure to ambient light has a small but significant effect on ROP development False; the Light-ROP study found no relationship

Infants with a R→L cardiac shunt (and subsequent low O₂ sat) are protected from ROP False, and this provides strong evidence that excess PₐO₂ is not causative

Whites have a greater risk of ROP than blacks True

The sexes have roughly equal ROP incidence rates False; ROP is significantly more common in males

Once the ROP process starts, it usually progresses to an advanced level False; roughly 80% of ROP arrests spontaneously, without significant sequelae
**ROP classification**: Based on pathology criteria, another criterion (called…), another criterion (called…), and another criterion (two words) status:
**ROP classification**: Based on pathology location (zone), appearance (stage), and plus disease status:
**ROP classification**: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1
  - Zone 2
  - Zone 3
**ROP classification**: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - **Zone 1**: Circle around ONH w/ radius 2x disc-fovea distance
  - **Zone 2**
  - **Zone 3**

(ONH = optic nerve head)
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: [Blank]
  - Zone 3

(OD)
**ROP classification**: Based on pathology **location** (zone), **appearance** (stage), and **plus disease** status:

- **Location**
  - **Zone 1**: Circle around ONH w/ radius 2x disc-fovea distance
  - **Zone 2**: Edge of Zone 1 to nasal ora, and around temporally
  - **Zone 3**
ROP classification: Based on pathology location, appearance, and plus disease status:

Location
- Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
- Zone 2: Edge of Zone 1 to nasal ora, and around temporally
- Zone 3: 

Diagram:
- Zone 1
- Zone 2
- (OD)
**ROP classification**: Based on pathology **location** (zone), **appearance** (stage), and **plus disease** status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2
**ROP classification**: Based on pathology location, appearance, and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1:
  - Stage 2
  - Stage 3
  - Stage 4
  - Stage 5
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2
  - Stage 3
  - Stage 4
  - Stage 5
**ROP classification**: Based on pathology location (zone), appearance (stage), and plus disease status:

### Location
- **Zone 1**: Circle around ONH w/ radius 2x disc-fovea distance
- **Zone 2**: Edge of Zone 1 to nasal ora, and around temporally
- **Zone 3**: Residual crescent anterior to Zone 2

### Appearance
- **Stage 1**: Demarcation line
- **Stage 2**: 
- **Stage 3**: 
- **Stage 4**: 
- **Stage 5**: 
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - **Zone 1:** Circle around ONH w/ radius 2x disc-fovea distance
  - **Zone 2:** Edge of Zone 1 to nasal ora, and around temporally
  - **Zone 3:** Residual crescent anterior to Zone 2

- **Appearance**
  - **Stage 1:** Demarcation line
  - **Stage 2:** Elevated line (‘ridge’) +/- small tufts of neo
  - **Stage 3:** Ridge with extensive neo growing through ILM
  - **Stage 4:** Subtotal RD
  - **Stage 5:** Total RD
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

Location
- Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
- Zone 2: Edge of Zone 1 to nasal ora, and around temporally
- Zone 3: Residual crescent anterior to Zone 2

Appearance
- Stage 1: Demarcation line
- Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
- Stage 3:
- Stage 4
- Stage 5
**ROP classification**: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - **Zone 1**: Circle around ONH w/ radius 2x disc-fovea distance
  - **Zone 2**: Edge of Zone 1 to nasal ora, and around temporally
  - **Zone 3**: Residual crescent anterior to Zone 2

- **Appearance**
  - **Stage 1**: Demarcation line
  - **Stage 2**: Elevated line (‘ridge’) +/- small tufts of neo
  - **Stage 3**: Ridge with extensive neo growing through ILM

(ILM = internal limiting membrane)
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line ('ridge') +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - Stage 4: 
  - Stage 5
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - **Zone 1**: Circle around ONH w/ radius 2x disc-fovea distance
  - **Zone 2**: Edge of Zone 1 to nasal ora, and around temporally
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- **Appearance**
  - **Stage 1**: Demarcation line
  - **Stage 2**: Elevated line (‘ridge’) +/- small tufts of neo
  - **Stage 3**: Ridge with extensive neo growing through ILM
  - **Stage 4**: Subtotal RD
  - **Stage 5**
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - **Zone 1**: Circle around ONH w/ radius 2x disc-fovea distance
  - **Zone 2**: Edge of Zone 1 to nasal ora, and around temporally
  - **Zone 3**: Residual crescent anterior to Zone 2

- **Appearance**
  - **Stage 1**: Demarcation line
  - **Stage 2**: Elevated line (‘ridge’) +/- small tufts of neo
  - **Stage 3**: Ridge with extensive neo growing through ILM
  - **Stage 4**: Subtotal RD
  - **Stage 5**

*Stage 4 is divided into two substages:*

*4a*: RD with macula…

*4b*: RD with macula…
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - **Stage 4:** Subtotal RD

**Stage 4 is divided into two substages:**
- 4a: RD with macula...on
- 4b: RD with macula...off
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - Stage 4: Subtotal RD
  - Stage 5: Total RD
**ROP classification**: Based on pathology **location** (zone), **appearance** (stage), and **plus disease** status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - Stage 4: Subtotal RD
  - Stage 5: Total RD
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - Stage 4: Subtotal RD
  - **Stage 5: Total RD**

What description is usually applied to the Stage 5 total RD?
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - Stage 4: Subtotal RD
  - **Stage 5: Total RD**

What description is usually applied to the Stage 5 total RD?
It is described as a ‘funnel’ RD
ROP stages
ROP classification: Based on pathology (location), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - **Stage 4: Subtotal RD**
  - **Stage 5: Total RD**

What are the three basic types of retinal detachment (generally speaking; not specific to ROP)?
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - Stage 4: Subtotal RD
  - Stage 5: Total RD

*What are the three basic types of retinal detachment (generally speaking; not specific to ROP)?*
Rhegmatogenous, exudative and tractional
**ROP classification**: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - **Zone 1**: Circle around ONH w/ radius 2x disc-fovea distance
  - **Zone 2**: Edge of Zone 1 to nasal ora, and around temporally
  - **Zone 3**: Residual crescent anterior to Zone 2

- **Appearance**
  - **Stage 1**: Demarcation line
  - **Stage 2**: Elevated line (‘ridge’) +/- small tufts of neo
  - **Stage 3**: Ridge with extensive neo growing through ILM
  - **Stage 4**: Subtotal RD
  - **Stage 5**: Total RD

What are the three basic types of retinal detachment (generally speaking; not specific to ROP)?
Rhegmatogenous, exudative and tractional

Which sort of RD occurs in ROP?
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - Stage 4: Subtotal RD
  - Stage 5: Total RD

What are the three basic types of retinal detachment (generally speaking; not specific to ROP)?
Rhegmatogenous, exudative and tractional

Which sort of RD occurs in ROP?
Tractional RD (TRD)
- **ROP classification**: Based on pathology location (zone), appearance (stage), and plus disease status:
  - **Location**
    - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
    - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
    - Zone 3: Residual crescent anterior to Zone 2
  - **Appearance**
    - Stage 1: Demarcation line
    - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
    - Stage 3: Ridge with extensive neo growing through ILM
    - Stage 4: Subtotal RD
    - Stage 5: Total RD
  - **Presence/absence of plus disease**
    - *Plus* disease = dilated/tortuous retinal vessels
• **ROP classification**: Based on pathology **location** (zone), **appearance** (stage), and **plus disease** status:

  ● **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

  ● **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - Stage 4: Subtotal RD
  - Stage 5: Total RD

  ● **Presence/absence of plus disease**
  - *Plus disease* = **Dilated/tortuous** retinal vessels
ROP: *Plus* disease
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - Stage 4: Subtotal RD
  - Stage 5: Total RD

- **Presence/absence of plus disease**
  - *Plus disease* = Dilated/tortuous retinal vessels

How dilated/tortuous do the vessels need to be to qualify as plus disease?
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

Location
- **Zone 1**: Circle around ONH w/ radius 2x disc-fovea distance
- **Zone 2**: Edge of Zone 1 to nasal ora, and around temporally
- **Zone 3**: Residual crescent anterior to Zone 2

Appearance
- **Stage 1**: Demarcation line
- **Stage 2**: Elevated line (‘ridge’) +/- small tufts of neo
- **Stage 3**: Ridge with extensive neo growing through ILM
- **Stage 4**: Subtotal RD
- **Stage 5**: Total RD

Presence/absence of plus disease
- **Plus disease** = Dilated/tortuous retinal vessels

How dilated/tortuous do the vessels need to be to qualify as plus disease? A standardized photo exists indicating the ‘official’ amount needed
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - Stage 4: Subtotal RD
  - Stage 5: Total RD

- **Presence/absence of plus disease**
  - *Plus* disease = Dilated/tortuous retinal vessels

How dilated/tortuous do the vessels need to be to qualify as plus disease? A standardized photo exists indicating the ‘official’ amount needed

What if the vessels are definitely dilated/tortuous, but not to the extent indicated in the standardized photo?
ROP classification: Based on pathology (location), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
  - Zone 3: Residual crescent anterior to Zone 2

- **Appearance**
  - Stage 1: Demarcation line
  - Stage 2: Elevated line (‘ridge’) +/- small tufts of neo
  - Stage 3: Ridge with extensive neo growing through ILM
  - Stage 4: Subtotal RD
  - Stage 5: Total RD

- **Presence/absence of plus disease**
  - *Plus* disease = Dilated/tortuous retinal vessels

*How dilated/tortuous do the vessels need to be to qualify as plus disease?*
*What if the vessels are definitely dilated/tortuous, but not to the extent indicated in the standardized photo? This is referred to as Pre-Plus disease*
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
  - Zone 2: Edge of Zone 1 to nasal ora, and around temporally
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    - Indicates arteriovenous shunting is taking place
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**Location**
- **Zone 1**: Circle around ONH w/ radius 2x disc-fovea distance
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**Presence/absence of plus disease**
- *Plus disease* = Dilated/tortuous retinal vessels
  - Indicates arteriovenous shunting is taking place
  - Strong indicator that disease is occurring
ROP classification: Based on pathology location (zone), appearance (stage), and plus disease status:

- **Location**
  - Zone 1: Circle around ONH w/ radius 2x disc-fovea distance
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- **Presence/absence of plus disease**
  - *Plus* disease = Dilated/tortuous retinal vessels
    - Indicates arteriovenous shunting is taking place
    - Strong indicator that disease progression is occurring
This is the *outdated* definition of when to treat ROP (so-called *Threshold disease*):

- 5 contiguous clock hours or 8 noncontiguous hours of *Stage 3* disease (or worse) in Zone I or II, associated with *plus disease*
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*What was the name of the study from which these (now considered outdated) treatment guidelines were developed?*
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*What was the name of the study from which these (now considered outdated) treatment guidelines were developed?*

The CRYO-ROP study
This is the outdated definition of when to treat ROP (so-called Threshold disease):

- 5 contiguous clock hours or 8 noncontiguous hours of Stage 3 disease (or worse) in Zone I or II, associated with plus disease.

What’s wrong with these criteria for treatment? Why don’t we use them anymore?
This is the outdated definition of when to treat ROP (so-called Threshold disease):

- 5 contiguous clock hours or 8 noncontiguous hours of Stage 3 disease (or worse) in Zone I or II, associated with plus disease

What’s wrong with these criteria for treatment? Why don’t we use them anymore? Research indicated that less than 13% of children treated via these criteria went on to have 20/40 or better vision in treated eyes! That’s not a very good outcome, so these criteria have been revised.
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What are the new criteria?
Treatment is indicated if the ROP meets one of three criteria:

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What was the name of the study from which these treatment guidelines were developed?

The ET-ROP (Early Treatment of Retinopathy of Prematurity) study
This is the outdated definition of when to treat ROP (so-called Threshold disease):

- 5 contiguous clock hours or 8 noncontiguous hours of Stage 3 disease (or worse) in Zone I or II, associated with plus disease

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What are the new criteria?

Treatment is indicated if the ROP meets one of three criteria:

1. Zone 1, any Stage, with Plus disease
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The motivating factor behind the **ET-ROP** was to see whether earlier intervention could improve upon these dismal results.

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**What are the new criteria?**

Treatment is indicated if the ROP meets one of three criteria:

1. Zone 1, any Stage, with Plus disease (‘Rush disease’) or
2. Zone 1, Stage 3, with or without Plus disease or
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Per the ET-ROP, disease meeting these criteria are known as what ‘type’ of ROP?
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- 5 contiguous clock hours or 8 noncontiguous hours of Stage 3 disease (or worse) in Zone I or II, associated with plus disease

Note that disease meeting ET-ROP criteria for treatment would not have met threshold under CRYO-ROP criteria. For this reason, the new criteria are sometimes referred to as *'pre-threshold Type I ROP’*

**What are the new criteria?**

Treatment is indicated if the ROP meets one of three criteria:

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By what special name is ‘Zone 1 + Plus disease’ known?

Rush disease

Why is it called Rush disease?

Because these eyes are at especially high risk of very rapid progression to TRD

Which infants are at particular risk for developing Rush disease?

Those weighing under 1000 grams
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Another term is used for aggressive posterior ROP--what is it?

Aggressive Posterior ROP (APROP)

Is APROP simply another name for Rush disease?
While the terms are sometimes used interchangeably, the clinical appearance and behavior of APROP can differ from that of Rush disease. APROP is characterized by the presence of neovascular fronds lying flat on the retinal surface (ie, without a ridge) in Zone 1 or posterior Zone 2. Active A-V shunting is the rule. APROP is notorious for three unfortunate tendencies: 1) Direct progression from Stage 1 to Stage 3 disease; 2) very rapid progression (Stage 1 to 3 or even 4 in a matter of days); 3) a proclivity to recur despite seemingly adequate treatment; and 4) a less-than-robust response to conventional laser treatment.
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What’s wrong with these criteria for treatment? Why don’t we use them anymore? Research indicated that less than 13% of children treated via these criteria went on to have 20/40 or better vision in treated eyes! That’s not a very good outcome, so these criteria have been revised.

What are the new criteria?

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1. Zone 1, any Stage, with Plus disease
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What is the conventional treatment for ROP?

- Ablation (via either cryo or laser) of the avascular retina

Which is preferred, cryo or laser? Most clinicians prefer laser; it is less traumatic to the eye, and less risky (5% of infants undergoing cryo for ROP will have cardiopulmonary arrest!)
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3. Zone 2, Stage 2 or 3, with Plus disease

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ROP: Treatment Considerations

What is the conventional treatment for ROP?

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What are the other advantages of laser over cryo?

- Less bad thing to tissue

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Which is preferred, cryo or laser? Most clinicians prefer laser; it is less traumatic to the eye, and less risky (5% of infants undergoing cryo for ROP will have cardiopulmonary arrest!)

What are the other advantages of laser over cryo?

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Although it must be stressed that laser treatment is not wholly benign—issues with intra-operative apnea and/or adverse cardiac events have been reported, as have sequelae including cataract and glaucoma.
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Is APROP simply another name for Rush disease?
While the terms are sometimes used interchangeably, the clinical appearance and behavior of APROP can differ from that of Rush dz. APROP is characterized by the presence of neovascular fronds lying flat on the retinal surface (ie, without a ridge) in Zone 1 or posterior Zone 2. Active A-V shunting is the rule. APROP is notorious for four unfortunate tendencies:
1) Progressing directly from Stage 1 to Stage 3 disease;
2) very rapid progression--Stage 1 to 3 (or even 4) in a matter of days;
3) a proclivity to recur despite seemingly adequate treatment; and
4) a less-than-robust response to conventional laser treatment

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Another term is used for aggressive posterior ROP--what is it?
It is called Aggressive Posterior ROP (APROP)
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The recurrence rates did not differ statistically.

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ROP: Treatment Considerations

So that’s it then--bevacizumab is the tx of choice for Zone 1 APROP, right?

Not quite. There are a host of criticisms of the BEAT-ROP trial specifically, as well as concerns regarding the use of intravitreal bevacizumab in infants generally.

Identify five specific criticisms re the BEAT-ROP trial

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Another, smaller study found that recurrence after bevacizumab occurred as late as 69 weeks postmenstrual age.

(No question yet—keep going)

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These criteria have been revised. What are the new criteria?

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In addition to the late age at which they occur, in what other important manner can recurrences after bevacizumab differ from those after CLT?
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What's wrong with these criteria for treatment? Why don't we use them anymore?

Research indicated that less than 13% of children treated via these criteria went on to have 20/40 or better vision in treated eyes! That's not a very good outcome, so these criteria have been revised.

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Treatment is indicated if the ROP meets one of three criteria:

1. Zone 1, any Stage, with Plus disease
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What is the conventional treatment for ROP?

Intravitreal bevacizumab

What clinical trial evaluated intravitreal bevacizumab as a primary treatment for ROP?

The BEAT-ROP (Bevacizumab Eliminates Angiogenic Threat in ROP) trial

What was the key finding of the BEAT-ROP trial?

In eyes with Zone 1 APROP, the rate of recurrence after CLT was 42%, whereas the rate after intravitreal bevacizumab was only 6%.

What about in eyes with posterior Zone 2 APROP?

The recurrence rates did not differ statistically.

What about post-treatment development of the immature peripheral retina?

Peripheral vascularization proceeded in an apparently normal fashion in the bevacizumab eyes, but not in the CLT eyes.

So that’s it then--bevacizumab is the tx of choice for Zone 1 APROP, right?

Not quite. There are a host of criticisms of the BEAT-ROP trial specifically, as well as concerns regarding the use of intravitreal bevacizumab in infants generally.

--Dosing. As mentioned, the BEAT-ROP used 0.625 mg of bevacizumab--half the adult dose. However, the vitreous volume of a preemie eye is only about 1/3 that of an adult eye, and some experts believe the study dose was unnecessarily high.

--Length of follow-up. BEAT-ROP infants were followed until age 54 weeks (postmenstrual). While this interval is adequate to capture most cases of post-CLT recurrence, there is evidence suggesting it may miss up to 50% of recurrences after bevacizumab.

--Generalizability. In the BEAT-ROP cohort, >50% of the infants were Hispanic. It is not clear to what extent (if any) this limits the applicability of the data to preemies of other ethnic backgrounds.

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What if the pt develops a TRD--how is that managed? PPV and/or scleral buckle is effective. Only 30% of cases achieve anatomic reattachment; of these, only 25% are still attached at 5 years, and only 10% have ambulatory vision.
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(PPV = Pars plana vitrectomy)
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Once a decision to treat has been made, how long can it be deferred?
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or
3. Zone 2, Stage 2 or 3, with Plus disease

Once a decision to treat has been made, how long can it be deferred? When possible, treatment should be initiated within 72 hours.
**ROP screening**

**Who?**

- Screen all infants…
  - …with a birth weight of less than ___ gm
ROP screening

Who?

- Screen all infants…
  - …with a birth weight of less than 1500 gm
ROP screening

Who?

- Screen all infants…
  - …with a birth weight of less than 1500 gm, and/or
  - …whose gestational age at birth was # weeks or less
ROP: Screening and Follow-Up

- **ROP screening**
  - **Who?**
    - Screen all infants…
      - …with a birth weight of less than 1500 gm, *and/or*
      - …whose gestational age at birth was 30 weeks or less
ROP screening

Who?

Screen all infants…

- …with a birth weight of less than 1500 gm, and/or
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What about infants >1500 gm and/or with gestational age >30 weeks? Should they be screened?
**ROP screening**

**Who?**

- Screen all infants...
  - ...with a birth weight of less than **1500 gm**, and/or
  - ...whose gestational age at birth was **30 weeks or less**

*What about infants >1500 gm and/or with gestational age >30 weeks? Should they be screened?*

Not as a general rule. However, the guidelines state that such infants should be screened if/when their neonatologist feels it is indicated.
ROP screening

Who?
- Screen all infants...
  - ...with a birth weight of less than 1500 gm, and/or
  - ...whose gestational age at birth was 30 weeks or less

When?
- Timing of first screen is a function of pt age (see table)
  - Serious ROP rare before postmenstrual age 31 weeks, so this is the youngest age that requires screening

A single screening exam is sufficient if the retina is fully vascularized OU. Otherwise, 1-3 week follow-up is indicated (depending upon exam findings).
### ROP Screening

**Who?**
- Screen all infants...
  - with a birth weight of less than 1500 gm,
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- Timing of first screen is a function of patient age (see table).

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### Table: ROP: Screening and Follow-Up

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Don’t try and memorize the table! Instead, here is first-screen timing in a nutshell:

No question—proceed when ready
ROP screening

Who?

- Screen all infants
  - with a birth weight of less than 1500 gm,
  - and/or
  - whose gestational age at birth was 30 weeks or less.

When?

- Timing of first screen is a function of pt age (see table)
  - Serious ROP rare before postmenstrual age 31 weeks, so this is the youngest age that requires screening

Don’t try and memorize the table! Instead, here is first-screen timing in a nutshell:
If the infant’s gestational age at birth was 27 weeks or younger, perform first screen at postmenstrual age 31 weeks, or

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### ROP screening

#### Who?
- Screen all infants...
  - ...with a birth weight of less than 1500 gm,
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#### When?
- Timing of first screen is a function of patient age (see table)
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*Don’t try and memorize the table! Instead, here is first-screen timing in a nutshell:* If the infant’s gestational age at birth was 27 weeks or younger, perform first screen at **postmenstrual age 31 weeks**, or If the infant’s gestational age at birth was 28 weeks or older, perform first screen at **chronologic age 4 weeks**

*No question—proceed when ready*
**ROP screening**

**Who?**
- Screen all infants…
  - …with a birth weight of less than 1500 gm, and/or
  - …whose gestational age at birth was 30 weeks or less

**When?**
- Timing of first screen is a function of pt age (see table)
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**How Often?**
- A single screening exam is sufficient if the retina is
**ROP screening**

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- A single screening exam is sufficient if the retina is fully vascularized OU
- Otherwise, follow-up is indicated (depending upon exam findings)
ROP screening

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How Often?

- A single screening exam is sufficient if the retina is fully vascularized OU
- Otherwise, 1 - 3 week follow-up is indicated (depending upon exam findings)
Long-term follow-up: A child with ROP needs periodic follow-up beyond the newborn period because...

- Vitreoretinal traction can lead to RD in the 1st or 2nd decade of life.
Long-term follow-up: A child with ROP needs periodic follow-up beyond the newborn period because…

- Vitreoretinal traction can lead to RD in 1st or 2nd decade
**Long-term follow-up:** A child with ROP needs periodic follow-up beyond the newborn period because...

- **Vitreoretinal traction** can lead to RD in **1st or 2nd decade**.
- Amblyopia can result from **refractive problem**, **pathology**, **macular pathology**, and/or **EOM problem**.
Long-term follow-up: A child with ROP needs periodic follow-up beyond the newborn period because...

- Vitreoretinal traction can lead to RD in 1st or 2nd decade
- Amblyopia can result from high myopia, macular dragging, and/or strabismus
ROP: Macular dragging
**Long-term follow-up**: A child with ROP needs periodic follow-up beyond the newborn period because...

- **Vitreoretinal traction** can lead to RD in **1st or 2nd decade**
- Amblyopia can result from **high myopia**, macular **dragging**, and/or **strabismus**
- Macular dragging can produce **pseudo EOM problem**
**Long-term follow-up**: A child with ROP needs periodic follow-up beyond the newborn period because...

- **Vitreoretinal traction** can lead to RD in 1<sup>st</sup> or 2<sup>nd</sup> decade
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    - Will have positive **angle kappa**, but no **shift on cover testing**.
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Speaking of macular dragging...
In addition to a dragged macula, ROP pts often have a dragged...
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In addition to a dragged macula, ROP pts often have a dragged disc. What three other clinical entities can give a similar picture?

3 things that look like ROP:

1) ?
2) ?  
3) ?

Hints forthcoming…
In addition to a dragged macula, ROP pts often have a dragged disc. **What three other clinical entities can give a similar picture?**

3 things that look like ROP:

1) **Hint: A phakomatosis (buzzterm: ‘Splashed paint’)**
2) ?
3) ?
In addition to a dragged macula, ROP pts often have a dragged disc. What three other clinical entities can give a similar picture?

3 things that look like ROP:

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3 things that look like ROP:

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Hint: A dz of the vitreoretinal interface
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*Hint: Can also look like Rb*
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What is the eponymous name for IP?

Bloch-Sulzberger syndrome

What is the inheritance pattern of IP?

X-linked dominant

What does this pattern portend for its demographics?

Males die in utero, so almost all cases will be females.

We noted that IP is a phakomatosis. By what more on-the-nose term are phakomatoses known?

'Neurocutaneous syndromes.' Most present with multiple lesions in two or more organ systems, usually including the CNS and skin (hence the name).

To what does the buzzterm splashed paint refer?

The appearance of the infant's skin after erythema and bullae develop at age ~1 week.
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Incontinentia pigmenti

Splashed paint refers to the appearance of the infant’s skin after erythema and bullae develop at age ~1 week.
A 4-month old girl with incontinentia pigmenti was admitted for seizures and intracranial hemorrhage. It may be difficult to appreciate the peripheral nonperfusion with RetCam photography alone (A-B), but the findings become clear with RetCam FA
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Incontinentia pigmenti: Splashed-paint appearance
Patients s/p ROP often have a dragged fovea and/or dragged disc.

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In a nutshell, what sort of condition is FEVR?

A vitreoretinal dystrophy

What is the basic retinal problem in FEVR?

The temporal retina fails to vascularize

"The temporal retina fails to vascularize"—that sounds like ROP. In what two ways will FEVR neonates differ from ROP neonates?

FEVR babies will be full-term, and have normal oxygenation status.

What is the inheritance pattern for FEVR?

AD, AR and X-linked forms all exist.
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FEVR: Fundus photo and FA
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**What sort of bug is Toxocara?**

**A roundworm**

**What animals are the principal hosts?**

**Dogs and cats**

**How is the worm acquired by humans?**

**Usually via consumption of contaminated soil**

**What is the classic appearance of the ROP-like lesion?**

**A peripheral retinal mass connected by dense fibrous strands to the optic disc**

**What percent of ocular toxocariasis pts have the ROP-like presentation?**

**About half**
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What sort of bug is Toxocara?
A roundworm

What animals are the principal hosts?
Dogs and cats

How is the worm acquired by humans?
Usually via consumption of contaminated soil

What is the classic appearance of the ROP-like lesion?
A peripheral retinal mass connected by dense fibrous strands to the optic disc

What percent of ocular toxocariasis pts have the ROP-like presentation?
About half
Patients s/p ROP often have a dragged fovea and/or dragged disc. **What three other clinical entities can give a similar picture?**

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**What 3 things that look like ROP:**
- Incontinentia pigmenti
- Familial exudative vitreoretinopathy (FEVR)
- Toxocara chorioretinitis
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**ROP: DDx**

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- *Toxocara chorioretinitis*
Patients s/p ROP often have a dragged fovea and/or dragged disc. What three other clinical entities can give a similar picture?

3 things that look like ROP:

1) Incontinentia pigmenti
2) Familial exudative vitreoretinopathy (FEVR)
3) Toxocara chorioretinitis

Mechanism(s) for disc/foveal dragging in each?

These two share a common mechanism
Patients s/p ROP often have a dragged fovea and/or dragged disc. What three other clinical entities can give a similar picture?

3 things that look like ROP:

1. Incontinentia pigmenti
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Mechanism(s) for disc/foveal dragging in each?
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3 things that look like ROP:
1) Incontinentia pigmenti
2) Familial exudative vitreoretinopathy (FEVR)
3) Toxocara chorioretinitis

Mechanism(s) for disc/foveal dragging in each?

- Mechanism similar to ROP (peripheral neo→retinal traction)
- This one has a different mechanism
Patients s/p ROP often have a dragged fovea and/or dragged disc. What three other clinical entities can give a similar picture?

3 things that look like ROP:
1) *Incontinentia pigmenti*
2) *Familial exudative vitreoretinopathy (FEVR)*
3) *Toxocara chorioretinitis*

Mechanism(s) for disc/foveal dragging in each?

- Mechanism similar to ROP (peripheral neo→retinal traction)
- Disc/foveal dragging due to inflammatory granuloma