Q

Which is the more common entity?
A

- Which is the more common entity? CRAO
Q

- Which is the more common entity? **CRAO**
- Which is more likely to be embolic?
Which is the more common entity? **CRAO**

Which is more likely to be embolic? **BRAO**
Q

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
A

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
Calcific; platelet-fibrin; cholesterol
Q

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
Calcific; platelet-fibrin; cholesterol

Emboli composed of cholesterol are known by what eponymous name?
Which is the more common entity? CRAO
Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
Calcific; platelet-fibrin; cholesterol

Emboli composed of cholesterol are known by what eponymous name?
Hollenhorst plaque
BRAO vs CRAO

Hollenhorst plaque
Q

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
Calcific; platelet-fibrin; cholesterol

For each statement, identify which variety is associated:
--Arise from diseased cardiac valves: ?
A

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
Calcific; platelet-fibrin; cholesterol

For each statement, identify which variety is associated:
--Arise from diseased cardiac valves: Calcific
Q

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
Calcific; platelet-fibrin; cholesterol

For each statement, identify which variety is associated:
--Arise from diseased cardiac valves: Calcific
--Arise from carotid arteries: ?
Which is the more common entity? CRAO
Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
Calcific; platelet-fibrin; cholesterol

For each statement, identify which variety is associated:
--Arise from diseased cardiac valves: Calcific
--Arise from carotid arteries: Cholesterol
Q

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
- Calcific; platelet-fibrin; cholesterol

For each statement, identify which variety is associated:
- Arise from diseased cardiac valves: Calcific
- Arise from carotid arteries: Cholesterol
- Associated with large-vessel arteriosclerosis: ?
A

Which is the more common entity? CRAO

Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
Calcific; platelet-fibrin; cholesterol

For each statement, identify which variety is associated:
--Arise from diseased cardiac valves: Calcific
--Arise from carotid arteries: Cholesterol
--Associated with large-vessel arteriosclerosis: Platelet-fibrin
Q

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
Calcific; platelet-fibrin; cholesterol

For each statement, identify which variety is associated:
--Arise from diseased cardiac valves: Calcific
--Arise from carotid arteries: Cholesterol
--Associated with large-vessel arteriosclerosis: Platelet-fibrin

In general terms, where do emboli usually lodge?
Q/A

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?

**Calcific; platelet-fibrin; cholesterol**

*For each statement, identify which variety is associated:*

--Arise from diseased cardiac valves: Calcific
--Arise from carotid arteries: Cholesterol
--Associated with large-vessel arteriosclerosis: Platelet-fibrin

*In general terms, where do emboli usually lodge?*

At a **two words** in the arterial tree
Which is the more common entity? CRAO
Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
Calcific; platelet-fibrin; cholesterol

For each statement, identify which variety is associated:
--Arise from diseased cardiac valves: Calcific
--Arise from carotid arteries: Cholesterol
--Associated with large-vessel arteriosclerosis: Platelet-fibrin

In general terms, where do emboli usually lodge?
At a branch point in the arterial tree
A

Which is the more common entity? CRAO
Which is more likely to be embolic? BRAO

What are the three main varieties of embolus? Calcific; platelet-fibrin; cholesterol

For each statement, identify which variety is associated:
--Arise from diseased cardiac valves: Calcific
--Arise from carotid arteries: Cholesterol
--Associated with large-vessel arteriosclerosis: Platelet-fibrin

In general terms, where do emboli usually lodge?
At a branch point in the arterial tree. That is, an emboli will continue to float along in the bloodstream until it reaches a bifurcation for which it is too large to travel down either fork, and becomes lodged.
BRAO vs CRAO

BRAO with embolus visible at branch point
Which is the more common entity? CRAO

Which is more likely to be embolic? BRAO

What are the three main varieties of embolus?
Calcific; platelet-fibrin; cholesterol

For each statement, identify which variety is associated:
--Arise from diseased cardiac valves: Calcific
--Arise from carotid arteries: Cholesterol
--Associated with large-vessel arteriosclerosis: Platelet-fibrin

In general terms, where do emboli usually lodge?
At a branch point in the arterial tree. That is, an emboli will continue to float along in the bloodstream until it reaches a bifurcation for which it is too large to travel down either fork, and becomes lodged. In CRAO, the embolus usually lodges at the level of the lamina cribrosa; for this reason, an embolus isn’t always visible on the ONH.
Q

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO
- For which is VA usually NLP?
Which is the more common entity? **CRAO**
Which is more likely to be embolic? **BRAO**
For which is VA usually NLP? **Neither**
Q

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO
- For which is VA usually NLP? Neither

If vision is NLP after an arterial occlusion, which artery is implicated?
A

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO
- For which is VA usually NLP? Neither

*If vision is NLP after an arterial occlusion, which artery is implicated?*
*The ophthalmic artery*
Q

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO
- For which is VA usually NLP? Neither
- Which can be associated with migraines?
A

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO
- For which is VA usually NLP? Neither
- Which can be associated with migraines? Both
Q

- Which is the more common entity? **CRAO**
- Which is more likely to be embolic? **BRAO**
- For which is VA usually NLP? **Neither**
- Which can be associated with migraines? **Both**
- Which eventually recannulates, thus restoring blood flow to the affected area?
A

- Which is the more common entity? **CRAO**
- Which is more likely to be embolic? **BRAO**
- For which is VA usually NLP? **Neither**
- Which can be associated with migraines? **Both**
- Which eventually recannulates, thus restoring blood flow to the affected area? **Both**
Q

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO
- For which is VA usually NLP? Neither
- Which can be associated with migraines? Both
- Which eventually recannulates, thus restoring blood flow to the affected area? Both

Does restoration of blood flow reverse the damage wrought by the RAO?
A

- Which is the more common entity? CRAO
- Which is more likely to be embolic? BRAO
- For which is VA usually NLP? Neither
- Which can be associated with migraines? Both
- Which eventually recannulates, thus **restoring blood flow to the affected area**? Both

*Does restoration of blood flow reverse the damage wrought by the RAO?*
Nope—permanent damage is permanent
Which of the following concerning BRAO are true?

- The majority of cases are embolic
Which of the following concerning BRAO are true?

- The majority of cases are embolic ☑️
Which of the following concerning BRAO are true?

- The majority of cases are embolic T

What are the common causes in non-embolic cases?

--?

--?

--?
Which of the following concerning BRAO are true?

- The majority of cases are embolic

What are the common causes in non-embolic cases?
-- Vasospasm
--?
--?
Which of the following concerning BRAO are true?

- The majority of cases are embolic \( T \)

What are the common causes in non-embolic cases?

-- Vasospasm (eg, )
-- ?
-- ?
● Which of the following concerning BRAO are true?
  
  ● The majority of cases are embolic

  What are the common causes in non-embolic cases?
  -- Vasospasm (e.g., migraine)

  --?
  --?
Which of the following concerning BRAO are true?
- The majority of cases are embolic

What are the common causes in non-embolic cases?
- Vasospasm (eg, migraine)
- ?
- ?
Which of the following concerning BRAO are true?

- The majority of cases are embolic T

What are the common causes in non-embolic cases?
- Vasospasm (eg, migraine)
- Inflammation
- ?
Which of the following concerning BRAO are true?

- The majority of cases are embolic (T)

What are the common causes in non-embolic cases?

- Vasospasm (eg, migraine)
- Inflammation (ie, vasculitis)
- ?
Which of the following concerning BRAO are true?

- The majority of cases are embolic **T**

*What are the common causes in non-embolic cases?*

- Vasospasm (eg, migraine)
- Inflammation (ie, vasculitis)
- ?
Which of the following concerning BRAO are true?

- The majority of cases are embolic \( T \)

*What are the common causes in non-embolic cases?*

- Vasospasm (eg, migraine)
- Inflammation (ie, vasculitis)
- Coagulopathy
Which of the following concerning BRAO are true?

- The majority of cases are embolic T

What are the common causes in non-embolic cases?
- Vasospasm (e.g., migraine)
- Inflammation (e.g., vasculitis)

Two recreational drugs are notorious-but-rare causes of vasospastic BRAO. What are they?
--?
--?

Hints forthcoming
Which of the following concerning BRAO are true?

- The majority of cases are embolic

What are the common causes in non-embolic cases?

- Vasospasm (e.g., migraine)
- Inflammation (vasculitis)

Two recreational drugs are notorious-but-rare causes of vasospastic BRAO. What are they?

- ?  ←  Hint: Illegal
- ?
Which of the following concerning BRAO are true?

- The majority of cases are embolic [T]

What are the common causes in non-embolic cases?

- Vasospasm (e.g., migraine)

Two recreational drugs are notorious-but-rare causes of vasospastic BRAO. What are they?

- Cocaine  [Hint: Illegal]
- ?
Which of the following concerning BRAO are true?

- The majority of cases are embolic **T**

*What are the common causes in non-embolic cases?*

- Vasospasm (eg, migraine)

*Two recreational drugs are notorious-but-rare causes of vasospastic BRAO. What are they?*

- Cocaine

- ?  

*Hint: Legal*
Which of the following concerning BRAO are true?

- The majority of cases are embolic **T**

What are the common causes in non-embolic cases?

- **Vasospasm** (eg, migraine)

Two recreational drugs are notorious-but-rare causes of vasospastic BRAO. What are they?

- Cocaine
- Sildenafil **← Hint: Legal**
Which of the following concerning BRAO are true?

- The majority of cases are embolic

What are the common causes in non-embolic cases?

- Vasospasm (e.g., migraine)
- Inflammation (i.e., vasculitis)
- Coagulopathy

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- Cocaine
- Sildenafil

Speaking of non-embolic cases… A young-adult female presents with multiple bilateral BRAOs. This should bring to mind a specific condition—what is it?

Susac syndrome

What's weird about the BRAOs in Susac syndrome (other than that they're multiple and bilateral)?

They occur at non-branch points

In a nutshell, what is the underlying pathology in Susac?

It is an autoimmune condition involving anti-endothelial cell antibodies. The resulting endothelial cell damage attracts thrombotic material, which obliterates the lumen and thus produces (non-branch-point) occlusions.

What are its two classic non-ocular signs/symptoms?

- Sensorineural hearing loss
- Encephalopathy
Which of the following concerning BRAO are true?

- The majority of cases are embolic

What are the common causes in non-embolic cases?

- Vasospasm (e.g., migraine)
- Inflammation (i.e., vasculitis)
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Susac syndrome
Susac syndrome. Retinal whitening and cotton-wool spots along the proximal inferotemporal arcade vessels and within the inferotemporal macula suggestive of branch retinal artery occlusions
Which of the following concerning BRAO are true?

- The majority of cases are embolic

What are the common causes in non-embolic cases?
- Vasospasm (e.g., migraine)
- Inflammation (i.e., vasculitis)
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What’s weird about the BRAOs in Susac syndrome (other than that they’re multiple and bilateral)? They occur at non-branch points
Susac syndrome. Note the area of macular infarct doesn’t correspond to a branch-point blockage
Which of the following concerning BRAO are true?

- The majority of cases are embolic

What are the common causes in non-embolic cases?

- Vasospasm (e.g., migraine)
- Inflammation (i.e., vasculitis)
- Coagulopathy

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- Inflammation (i.e., vasculitis)
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Which of the following concerning BRAO are true?

- The majority of cases are embolic

What are the common causes in non-embolic cases?

- Vasospasm (e.g., migraine)
- Inflammation (e.g., vasculitis)
- Coagulopathy

Two recreational drugs are notorious—but rare causes of vasospastic BRAO. What are they?

- Cocaine
- Sildenafil

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In a nutshell, what is the underlying pathology in Susac? It is an autoimmune condition involving anti-endothelial cell antibodies
● Which of the following concerning BRAO are true?

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What are the common causes in non-embolic cases?

- Vasospasm (e.g., migraine)
- Inflammation (i.e., vasculitis)
- Coagulopathy

Two recreational drugs are notorious—but-rare causes of vasospastic BRAO. What are they?

- Cocaine
- Sildenafil

Speaking of non-embolic cases…A young-adult female presents with multiple bilateral BRAOs. This should bring to mind a specific condition—what is it?

Susac syndrome

What’s weird about the BRAOs in Susac syndrome (other than that they’re multiple and bilateral)? They occur at non-branch points

In a nutshell, what is the underlying pathology in Susac?

It is an autoimmune condition involving anti-endothelial cell antibodies. The resulting endothelial cell damage attracts thrombotic material, which obliterates the lumen and thus produces (non-branch-point) occlusions.
Susac syndrome. Note the multiple areas of arteriolar inflammation and blockage at non-branch points
Which of the following concerning BRAO are true?

- The majority of cases are embolic

What are the common causes in non-embolic cases?
- Vasospasm (e.g., migraine)
- Inflammation (i.e., vasculitis)
- Coagulopathy

Two recreational drugs are notorious—but-rare causes of vasospastic BRAO. What are they?
- Cocaine
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In a nutshell, what is the underlying pathology in Susac?
It is an autoimmune condition involving anti-endothelial cell antibodies. The resulting endothelial cell damage attracts thrombotic material, which obliterates the lumen and thus produces (non-branch-point) occlusions.

What are its two classic non-ocular signs/symptoms?
--?
--?
Which of the following concerning BRAO are true?

- The majority of cases are embolic.

What are the common causes in non-embolic cases?
- Vasospasm (eg, migraine)
- Inflammation (ie, vasculitis)
- Coagulopathy

Two recreational drugs are notorious—but-rare causes of vasospastic BRAO. What are they?
- Cocaine
- Sildenafil

Speaking of non-embolic cases... A young-adult female presents with multiple bilateral BRAOs. This should bring to mind a specific condition—what is it?
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What’s weird about the BRAOs in Susac syndrome (other than that they’re multiple and bilateral)?
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- Sensorineural hearing loss
- Encephalopathy
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- The majority of cases are embolic

What are the common causes in non-embolic cases?

- Vasospasm (e.g., migraine)
- Inflammation (i.e., vasculitis)
- Coagulopathy

Two recreational drugs are notorious—but-rare causes of vasospastic BRAO. What are they?

- Cocaine
- Sildenafil

Speaking of non-embolic cases... A young adult female presents with multiple bilateral BRAOs. This should bring to mind a specific condition—what is it?

Susac syndrome

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What are its two classic non-ocular signs/symptoms?

- Sensorineural hearing loss
- Encephalopathy

What does MRI brain reveal?

- White matter lesions, especially of the corpus callosum
Which of the following concerning BRAO are true?

- The majority of cases are embolic

What are the common causes in non-embolic cases?
- Vasospasm (e.g., migraine)
- Inflammation (e.g., vasculitis)
- Coagulopathy

Two recreational drugs are notorious—but-rare causes of vasospastic BRAO. What are they?
- Cocaine
- Sildenafil

Speaking of non-embolic cases... A young-adult female presents with multiple bilateral BRAOs. This should bring to mind a specific condition—what is it?

Susac syndrome

What's weird about the BRAOs in Susac syndrome (other than that they're multiple and bilateral)? They occur at non-branch points

In a nutshell, what is the underlying pathology in Susac?
It is an autoimmune condition involving anti-endothelial cell antibodies. The resulting endothelial cell damage attracts thrombotic material, which obliterates the lumen and thus produces (non-branch-point) occlusions.

What are its two classic non-ocular signs/symptoms?
- Sensorineural hearing
- Encephalopathy

What does MRI brain reveal?
White matter lesions, esp. of the...
Which of the following concerning BRAO are true?

- The majority of cases are embolic

**What are the common causes in non-embolic cases?**

- Vasospasm (e.g., migraine)
- Inflammation (i.e., vasculitis)
- Coagulopathy

Two recreational drugs are notorious—but-rare causes of vasospastic BRAO. What are they?

- Cocaine
- Sildenafil

Speaking of non-embolic cases... A young-adult female presents with multiple bilateral BRAOs. This should bring to mind a specific condition—what is it?

**Susac syndrome**

What’s weird about the BRAOs in Susac syndrome (other than that they’re multiple and bilateral)?

They occur at non-branch points

In a nutshell, what is the underlying pathology in Susac?

It is an autoimmune condition involving anti-endothelial cell antibodies. The resulting endothelial cell damage attracts thrombotic material, which obliterates the lumen and thus produces (non-branch-point) occlusions.

What are its two classic non-ocular signs/symptoms?

- Sensorineural hearing loss
- Encephalopathy

**What does MRI brain reveal?**

White matter lesions, esp. of the corpus callosum
Susac syndrome: Classic ‘snowball’ lesions of the corpus callosum
Which of the following concerning BRAO are true?

- The majority of cases are embolic T
- About half of cases involve the temporal retina, and half the nasal retina
Which of the following concerning BRAO are true?

- The majority of cases are embolic **T**
- About **98%** of cases involve the temporal retina, and **only 2%** involve the nasal retina **F**
Which of the following concerning BRAO are true?

- The majority of cases are embolic **T**
- About **98% half** of cases involve the temporal retina, and **only 2% half** the nasal retina **F**
- Risk factors include HTN, CAD, and carotid occlusive dz
Which of the following concerning BRAO are true?

- The majority of cases are embolic **T**
- About **half** of cases involve the temporal retina, and **half** the nasal retina **F**
- Risk factors include HTN, CAD, and carotid occlusive dz **T**
Which of the following concerning CRAO are true?

- The majority of cases are embolic
Q/A

Which of the following concerning CRAO are true?

- The majority of cases are embolic.
Which of the following concerning CRAO are true?
- The majority of cases are embolic (only 20% are embolic) F
Which of the following concerning CRAO are true?

- The majority of cases are thrombotic (only 20% are embolic)

What pathologic process is felt to underlie most thrombotic cases?
Which of the following concerning CRAO are true?

- The majority of cases are thrombotic (only 20% are embolic)

What pathologic process is felt to underlie most thrombotic cases? Atherosclerosis
Q

Which of the following concerning CRAO are true?

- The majority of cases are thrombotic (only 20% are embolic)

What pathologic process is felt to underlie most thrombotic cases?
Atherosclerosis

In such cases, where does the thrombus usually form?
Q/A

Which of the following concerning CRAO are true?

- The majority of cases are thrombotic (only 20% are embolic).

What pathologic process is felt to underlie most thrombotic cases?
Atherosclerosis

In such cases, where does the thrombus usually form?
At the level of the lamina cribrosa (same locale as embolic cases)
Which of the following concerning CRAO are true?

- The majority of cases are embolic (only 20% are embolic)

What pathologic process is felt to underlie most thrombotic cases?
Atherosclerosis

In such cases, where does the thrombus usually form?
At the level of the lamina cribrosa (same locale as embolic cases)
Which of the following concerning CRAO are true?

- The majority of cases are **thrombotic** (only 20% are embolic)
- Vision is 20/40+ in ~20% of cases
Which of the following concerning CRAO are true?

- The majority of cases are **thrombotic** (only 20% are embolic) **F**
- Vision is 20/40+ in ~20% of cases **T**
Which of the following concerning CRAO are true?

- The majority of cases are embolic (only 20% are embolic) **F**
- Vision is 20/40+ in ~20% of cases **T**

What is VA in the majority of cases?
Which of the following concerning CRAO are true?

- The majority of cases are embolic **F**
- Vision is 20/40+ in ~20% of cases **T**

What is VA in the majority of cases?
Final vision is 20/400 or so
Q

Which of the following concerning CRAO are true?

- The majority of cases are embolic  \( \text{F} \)
- Vision is 20/40+ in \( \sim 20\% \) of cases  \( \text{T} \)

\text{What is VA in the majority of cases?}
Final vision is 20/400 or so in about \( \_\_\_\% \) of cases
Which of the following concerning CRAO are true?

- The majority of cases are embolic \( F \)
- Vision is 20/40+ in \(~20\%\) of cases \( T \)

What is VA in the majority of cases?
Final vision is 20/400 or so in about \( 2/3 \) of cases
Which of the following concerning CRAO are true?
- The majority of cases are embolic \( F \)
- Vision is 20/40+ in \( \sim 20\% \) of cases \( T \)

What is VA in the majority of cases?
Final vision is 20/400 or so in about \( \frac{2}{3} \) of cases

What accounts for the lucky 20% with 20/40+ VA?
Which of the following concerning CRAO are true?

- The majority of cases are embolic \( \text{F} \)
- Vision is 20/40+ in ~20% of cases \( \text{T} \)

**What is VA in the majority of cases?**
Final vision is 20/400 or so in about \( \frac{2}{3} \) of cases

**What accounts for the lucky 20% with 20/40+ VA?**
The presence of a \( \text{two words} \) supplying the central macula
Which of the following concerning CRAO are true?

- The majority of cases are embolic \( \text{F} \)
- Vision is 20/40+ in \( \sim20\% \) of cases \( \text{T} \)

**What is VA in the majority of cases?**
Final vision is 20/400 or so in about \( \frac{2}{3} \) of cases

**What accounts for the lucky 20% with 20/40+ VA?**
The presence of a cilioretinal artery supplying the central macula
Which of the following concerning CRAO are true?

- The majority of cases are embolic  F
- Vision is 20/40+ in ~20% of cases  T

What is VA in the majority of cases?
Final vision is 20/400 or so in about 2/3 of cases

What accounts for the lucky 20% with 20/40+ VA?
The presence of a cilioretinal artery supplying the central macula

What is a cilioretinal artery?
Q/A

Which of the following concerning CRAO are true?

- The majority of cases are embolic **F**
- Vision is 20/40+ in ~20% of cases **T**

What is VA in the majority of cases?
Final vision is 20/400 or so in about 2/3 of cases

What accounts for the lucky 20% with 20/40+ VA?
The presence of a **cilioretinal artery** supplying the central macula

*What is a cilioretinal artery?*
An artery that services the retina, but instead of arising from the CRA it arises from a short posterior ciliary artery **abb.**
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What is VA?

How many short posterior ciliary arteries are there?

About 20

From what artery do they originate?
The ophthalmic

Where on the eye do they pierce the sclera?
In a ring around the optic nerve
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Which of the following concerning CRAO are true?

- The majority of cases are embolic **F**
- Vision is 20/40+ in ~20% of cases **T**
- GCA accounts for ~2% of cases **T**

*Just CRAO*

*thrombotic (only 20% are embolic)*

*GCA* *(giant cell arteritis)*
Which of the following concerning CRAO are true?

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**A**
Which of the following concerning CRAO are true?

- The majority of cases are embolic \( \text{F} \)
- Vision is 20/40+ in \( \sim 20\% \) of cases \( \text{T} \)
- GCA accounts for \( \sim 2\% \) of cases \( \text{T} \)
- NVI develops in \( \sim 20\% \) of cases \( \text{T} \)

20% seems low. By comparison, what proportion of ischemic CRVOs develop NVI?
Q/A

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But surely CRAO causes greater ischemia than does an ischemic CRVO. Given this, why isn't the proportion of CRAO cases with NVI higher than that of CRVO?
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Because CRAO is too ischemic. Consider: The genesis for neovascularization is the release of cytokines, especially VEGF. CR\( \text{V} \)O produces vast swathes of ischemic cells, which in a desperate attempt to remain alive release copious amounts of VEGF.
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Which of the following concerning CRAO are true?

- The majority of cases are thrombotic (only 20% are embolic) - **F**
- Vision is 20/40+ in ~20% of cases - **T**
- GCA accounts for ~2% of cases - **T**
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- The classic description of its appearance on ophthalmoscopy is *blood and thunder*
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- The classic description of its appearance on ophthalmoscopy is **F**
CRAO: Cherry red spot
What causes the foveola to be extra red in a CRAO?

The classic description of its appearance on ophthalmoscopy is blood and thunder.
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- NVI develops in ~20% of cases: **T**
- The classic description of its appearance on ophthalmoscopy is "blood and thunder": **F**

What causes the foveola to be extra red in a CRAO?

Nothing—that is to say, the color of the foveola is **unchanged** in CRAO.

The classic description of its appearance on ophthalmoscopy is "blood and thunder".

'cherry red spot'
Which of the following concerning CRAO are true?

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- Vision is 20/40+ in \textbf{~20\% of cases} \textbf{T}
- GCA accounts for \textbf{\textless{}2\% of cases} \textbf{T}
- NVI develops in \textbf{\textless{}20\% of cases} \textbf{T}

The classic description of its appearance on ophthalmoscopy is \textbf{blood and thunder F}

\textit{What causes the foveola to be extra red in a CRAO?}

Nothing—that is to say, the color of the foveola is \textbf{unchanged} in CRAO

\textit{Huh? Then what accounts for the cherry red spot appearance?}

The acute hypoxia induced by the CRAO causes the retina to become opaque and edematous—especially in the posterior pole, where the nerve fiber and ganglion cell layers are thickest. But recall that the foveola lacks several of the inner retinal layers. This means that, when a CRAO occurs, there is less edematous tissue in the foveola, and thus it doesn’t whiten nearly as much as the surrounding macula. So, it’s not that the foveola is \textbf{redder}; rather, it’s that the surrounding retina is \textbf{whiter}, and this makes the (normal) foveolar reflex appear \textbf{ redder by comparison}.
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Just CRAO

Foveola
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The classic description of its appearance on ophthalmoscopy is blood and thunder.
OCT of a CRAO. Note the severe retinal edema that largely spares the foveolar region.
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Huh? Then what accounts for the cherry red spot appearance?
The acute hypoxia induced by the CRAO causes the retina to become opaque and edematous, especially in the posterior pole. The ganglion and nerve fiber layers are the deepest retinal layers, and thus are least affected by the hypoxia. This means that, when a CRAO occurs, there is less edematous tissue in the foveola, and thus it doesn’t whiten nearly as much as the surrounding macula. So, it’s not that the foveola is redder; rather, it’s the surrounding retina that is whiter, and this makes the (normal) foveolar reflex appear redder by comparison.

An important aside: Are all retinal layers equally affected by a CRAO?

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Q/A

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The acute hypoxia induced by the CRAO causes the retina to become opaque and edematous, especially in the posterior pole, where the nerve fiber and ganglion cell layers are thickest. This means that the surrounding macula becomes more white, and thus the foveola, which lacks several inner retinal layers, appears redder by comparison.

An important aside: Are all retinal layers equally affected by a CRAO?
No, the inner layers are much more affected than are the outer layers. This is because of the nature of retinal blood supply.

What does that mean?
It means we need to review the vascular supply of the retina...
Which of the following concerning CRAO are true?

- The majority of cases are embolic: F
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The acute hypoxia induced by the CRAO causes the retina to become opaque and edematous, especially in the posterior pole where the nerve fiber and ganglion cell layers are thickest. This means that the foveola, which lacks several of the inner retinal layers, and thus has less edematous tissue, doesn’t white nearly as much as the surrounding macula.

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The vascular supply of the retina is such that the inner layers are much more affected than the outer.
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The acute hypoxia induced by the CRAO causes the retina to become opaque and edematous, especially in the posterior pole where the nerve fiber and ganglion cell layers are thickest. This means that the foveola, which lacks several inner retinal layers, and thus, the inner retinal layers, and thus, the foveola, will not be as edematous, and thus, will not turn white. This results in the foveola appearing redder by comparison.

An important aside: Are all retinal layers equally affected by a CRAO?

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*Why is this?*

Because of the nature of retinal blood supply.
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Huh? Then what accounts for the cherry red spot appearance?

The acute hypoxia induced by the CRAO causes the retina to become opaque and edematous, especially in the posterior pole, where the nerve fiber and ganglion cell layers are thickest. This makes the foveola, which lacks several of the inner retinal layers, appear white. But recall that the foveola lacks several of the inner retinal layers and thus does not become edematous like the surrounding macula.

An important aside: Are all retinal layers equally affected by a CRAO?

No, the inner layers are much more affected than are the outer.

Why is this?

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Why is this?
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What does that mean?
It means we need a short derail to review the vascular supply of the retina.

The classic description of its appearance on ophthalmoscopy is 'blood and thunder'.

Just CRAO
How many blood supplies does the retina receive?
How many blood supplies does the retina receive? Two
What are the sources of the retina’s two blood supplies?

Blood supply:

Just CRAO
What are the sources of the retina's two blood supplies?

Blood supply: Central retinal artery

Blood supply: Choriocapillaris

Just CRAO
What are the layers of the retina?

- **Retinal Layers**
  - Internal limiting membrane
  - Nerve fiber layer
  - Ganglion cell layer
  - Inner plexiform layer
  - Inner nuclear layer
  - Outer plexiform layer (Henle's layer)
  - Outer nuclear layer
  - External limiting membrane
  - Rod/cone inner and outer segments

- **RPE**

- **Bruch’s membrane**

Blood supply:
- **Central retinal artery**
- **Choriocapillaris**

Just CRAO
Retinal Layers
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Bruch’s membrane

Blood supply:
- Central retinal artery

Blood supply:
- Choriocapillaris

Which layers are supplied by each blood supply?

Just CRAO
A

- **Retinal Layers**
  - Internal limiting membrane
  - Nerve fiber layer
  - Ganglion cell layer
  - Inner plexiform layer
  - Inner nuclear layer
  - Outer plexiform layer (Henle’s layer)
  - Outer nuclear layer
  - External limiting membrane
  - Rod/cone inner and outer segments

- **RPE**

- **Bruch’s membrane**

**Blood supply:**
- **Central retinal artery**
  - Inner 2/3 of INL on in
- **Choriocapillaris**
  - Outer 1/3 of INL on out

*Just CRAO*
Retinal Layers

- Internal limiting membrane
- **Nerve fiber layer**
- **Ganglion cell layer**
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Blood supply:
- **Central retinal artery**
- **Choriocapillaris**

- RPE
- Bruch’s membrane

This is why CRAO devastates the inner retina, but not the outer—blood flow to the outer remains largely intact during the event.
Which of the following concerning CRAO are true?

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So, it’s not that the foveola is redder; rather, it’s that the surrounding retina is whiter, and this makes the (normal) foveolar reflex appear redder by comparison.

A less-important aside: Will a cherry-red spot be present in an ophthalmic artery occlusion?

Just CRAO
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The classic description of the appearance on ophthalmoscopy is **blood and thunder**.

A less-important aside: Will a cherry-red spot be present in an ophthalmic artery occlusion?

No

Why not?
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Nothing—that is to say, the color of the foveola is unchanged in CRAO.

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The acute hypoxia induced by the CRAO causes the retina to become opaque and edematous—especially in the posterior pole, where the nerve fiber and ganglion cell layers are thickest. But recall that the foveola lacks several of the inner retinal layers. This means that, when a CRAO occurs, there is less edematous tissue in the foveola, and thus it doesn’t whiten nearly as much as the surrounding macula.

So, it’s not that the foveola is redder; rather, it’s that the surrounding retina is whiter, and this makes the (normal) foveolar reflex appear redder by comparison.

The classic description of this appearance on ophthalmoscopy is blood and thunder.

A less-important aside: Will a cherry-red spot be present in an ophthalmic artery occlusion?
No

Why not?
Because an ophthalmic artery occlusion also bags the choroidal circulation, which is responsible for the foveolar reflex.
Acute ophthalmic-artery occlusion
Which of the following concerning CRAO are true?

- The majority of cases are embolic  \( \text{F} \)
- Vision is 20/40+ in ~20% of cases  \( \text{T} \)
- GCA accounts for ~2% of cases  \( \text{T} \)
- NVI develops in ~20% of cases  \( \text{T} \)
- The classic description of its appearance on ophthalmoscopy is  \( \text{blood and thunder} \)  \( \text{F} \)

An even less-important aside: The appearance of what condition is described with the term blood and thunder?
Which of the following concerning CRAO are true?

- The majority of cases are embolic \( \text{F} \)
- Vision is 20/40+ in \( \sim 20\% \) of cases \( \text{T} \)
- GCA accounts for \( \sim 2\% \) of cases \( \text{T} \)
- NVI develops in \( \sim 20\% \) of cases \( \text{T} \)
- The classic description of its appearance on ophthalmoscopy is \textit{blood and thunder} \( \text{F} \)

An even less-important aside: The appearance of what condition is described with the term \textit{blood and thunder}? CR\textit{VO}
CRVO: ‘Blood and thunder’
CRAO management involves a single overarching goal: Cause an embolus (if present) to pass downstream. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Five specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective 'prongs.'
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream.*
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream.*

Even though only 20% of CRAO are embolic, these are the only ones for which definitive tx is possible. Thus, for management purposes, it is best to treat all CRAOs as if they’re embolic.
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream.* Methods for achieving this goal work via one of two mechanisms: [ ] or [ ].

Two approaches to getting an embolus to pass: [ ]

Four words: [ ]
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature.

**Lower IOP** → **Just CRAO** → **Dilate the retinal vasculature**

Two approaches to getting an embolus to pass:
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they?

**Lower IOP**  
Two approaches to getting an embolus to pass:  

- ?  
- ?  
- ?  
- ?  
- ?  
- ?  

**Dilate the retinal vasculature**
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: *Lowering IOP* or *dilating the retinal vasculature*. Six specific treatments are commonly employed—what are they?

**Lower IOP**
- Carbogen
- Anterior chamber paracentesis
- Diamox
- Sublingual nitro
- Ocular massage
- Glaucoma drops

**Dilate the retinal vasculature**
- Two approaches to getting an embolus to pass:
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

Two approaches to getting an embolus to pass:

- Carbogen
- Diamox
- Sublingual nitro
- Ocular massage
- Anterior chamber paracentesis
- Glaucoma drops

**Dilate the retinal vasculature**

(Start with carbogen and work down the list)

*Just CRAO*
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

- Diamox
- Glaucoma drops
- Sublingual nitro
- Ocular massage
- Glaucoma drops

**Dilate the retinal vasculature**

- Carbogen
- Anterior chamber paracentesis

*Just CRAO*
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream.* Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

- Two approaches to getting an embolus to pass:
  - Diamox
  - Glaucoma drops

**Dilate the retinal vasculature**

- Carbogen
- Anterior chamber paracentesis
- Sublingual nitro
- Ocular massage
- Glaucoma drops

*What is carbogen?*
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

- **Diamox**
- **Sublingual nitro**
- **Ocular massage**
- **Glaucoma drops**

**Dilate the retinal vasculature**

- **Carbogen**

*What is carbogen?*
An admixture of 95% O₂ and 5% CO₂
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

*Carbogen*

*What is carbogen?*
An admixture of 95% O₂ and 5% CO₂

*How does it dilate the retinal vasculature?*
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

- Two approaches to getting an embolus to pass:
  - Lowering IOP
  - Dilating the retinal vasculature

**Dilate the retinal vasculature**

- **Carbogen**
  - *What is carbogen?*
    - An admixture of 95% O₂ and 5% CO₂
  - *How does it dilate the retinal vasculature?*
    - Retinal arterioles are CNS vessels—they dilate in response to increased PaCO₂

- **Anterior chamber paracentesis**

- **Diamox**

- **Sublingual nitro**

- **Ocular massage**

- **Glaucoma drops**
CRAO management involves a single overarching goal: Cause an embolus (if present) to pass downstream. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

**Carbogen**

*What is carbogen?*

An admixture of 95% O₂ and 5% CO₂

*How does it dilate the retinal vasculature?*

Retinal arterioles are CNS vessels—they dilate in response to increased PaCO₂

---

**Glaucoma drops**

*Just CRAO*

Remember the episode when I couldn’t figure out how to solve a problem using the things I had on hand?

I’m fresh out of carbogen. Is there a way to MacGyver some?
CRAO management involves a single overarching goal: Cause an embolus (if present) to pass downstream. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Just CRAO**

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

**Carbogen**

What is carbogen?

An admixture of 95% O2 and 5% CO2

How does it dilate the retinal vasculature?

Retinal arterioles are CNS vessels—they dilate in response to increased PaCO2

I’m fresh out of carbogen. Is there a way to MacGyver some?

Have the patient breathe into a paper bag

**Glaucoma drops**
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream.* Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

Methods for achieving the goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**
- Carbogen
- Anterior chamber paracentesis
- Diamox
- Sublingual nitro
- Ocular massage
- Glaucoma drops

**Dilate the retinal vasculature**
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, *divide the treatments into their respective ‘prongs.’*

**Lower IOP**

- Anterior chamber paracentesis
- Carbogen
- Diamox
- Sublingual nitro
- Ocular massage
- Glaucoma drops

**Dilate the retinal vasculature**

Two approaches to getting an embolus to pass:
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

- Carbogen
- Anterior chamber paracentesis
- Diamox

**Dilate the retinal vasculature**

- Sublingual nitro
- Ocular massage
- Glaucoma drops

Two approaches to getting an embolus to pass:
Q/A

CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**  
1. Carbogen  
2. Diamox  
3. Anterior chamber paracentesis

**Dilate the retinal vasculature**  
1. Sublingual nitro  
2. Ocular massage  
3. Glaucoma drops

Two approaches to getting an embolus to pass:

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**Just CRAO**
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**
- Anterior chamber paracentesis
- Diamox

**Dilate the retinal vasculature**
- Carbogen
- Sublingual nitro
- Ocular massage
- ?

Two approaches to getting an embolus to pass: Just CRAO

Glaucoma drops
CRAO management involves a single overarching goal: \textit{Cause an embolus (if present) to pass downstream}. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? \textbf{Finally, divide the treatments into their respective ‘prongs.’}

- Lower IOP
- Dilate the retinal vasculature

\textbf{Two approaches to getting an embolus to pass:}

- Carbogen
- Diamox
- Ocular massage
- Glaucoma drops
- Sublingual nitro
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

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*Hol up—this section fails to mention the tx modality of*

- Carbogen
- Diamox
- Glaucoma drops
- Anterior chamber paracentesis

Q

Just CRAO

- CRAO
- Retinal vasculature

167
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**
- Two approaches to getting an embolus to pass:
  - **Dilate the retinal vasculature**
  - **Thrombolysis**

**Carbogen**

**Diamox**

**Hol up**—this section fails to mention the tx modality of **thrombolysis**

**Ocular massage**

**Glaucoma drops**

**Anterior chamber paracentesis**
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

Two approaches to getting an embolus to pass:

1. **Lower IOP**
2. **Dilate the retinal vasculature**

**Thrombolysis**

Hol up—this section fails to mention the tx modality of *thrombolysis*, with its three specific treatments of *?*, *?*, and *?*. Question mark.
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

---

**Thrombolysis**

---tPA

---Intra-arterial thrombolysis

---Transvitreal thrombolysis

---

**Carbogen**

**Anterior chamber paracentesis**

---Diamox

---Glaucoma drops

---Ocular massage

---Hol up—this section fails to mention the tx modality of thrombolysis, with its three specific treatments of tPA, intra-arterial thrombolysis, and trans-vitreal thrombolysis.
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream.* Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

**Dilate the retinal vasculature**

--- Thrombolysis

-- tPA

-- Intra-arterial thrombolysis

-- Transvitreal thrombolysis

Hol up—this section fails to mention the tx modality of thrombolysis, with its three specific treatments of tPA, intra-arterial thrombolysis, and trans-vitreal thrombolysis. Why aren’t these covered here?
CRAO management involves a single overarching goal: Cause an embolus (if present) to pass downstream. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

**Thrombolysis**

- tPA
- Intra-arterial thrombolysis
- Transvitreal thrombolysis

Hol up—this section fails to mention the tx modality of thrombolysis, with its three specific treatments of tPA, intra-arterial thrombolysis, and trans-vitreal thrombolysis. Why aren’t these covered here? Because at present they are not recommended by the AAO in its Preferred Practice Guidelines.
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream.* Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

**What is the timeframe of CRAO treatment?**

**Anterior chamber paracentesis**

**Dilate**

**Carbogen**

**Sublingual nitro**

**Ocular massage**

**Glaucoma drops**
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

*What is the timeframe of CRAO treatment?*

Primate studies indicate that retinal cells undergo irreversible ischemic damage within 90 minutes of CRA ligature—a dauntingly small window of opportunity for treatment.
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

What is the timeframe of CRAO treatment?
Primate studies indicate that retinal cells undergo irreversible ischemic damage within 90 minutes of CRA ligature—a dauntingly small window of opportunity for treatment. However, as many CRAO are felt to be incomplete, and as hope springs eternal in the human breast, most clinicians will treat up to 24° after onset.
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

What is the timeframe of CRAO treatment?

Primate studies indicate that retinal cells undergo irreversible ischemic damage within 90 minutes of CRA ligature—a dauntingly small window of opportunity for treatment. However, as many CRAO are felt to be incomplete, and as hope springs eternal in the human breast, most clinicians will treat up to 24 hours after onset.

What other management step must be taken?

The pt should be referred immediately to a stroke center if the event is acute, ie, if s/he has been symptomatic for <24 hrs.

Is this true for BRAO as well?

Yes

Just CRAO
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Q/A**

**Just CRAO**

**What is the timeframe of CRAO treatment?**

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### Lower IOP

Two approaches to getting an embolus to pass:

### Dilate the retinal vasculature

What is the timeframe of CRAO treatment?

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CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Q**

**What is the timeframe of CRAO treatment?**
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**Just CRAO**

**What other management step must be taken?**
The pt should be referred immediately to a stroke center if the event is acute, ie, if s/he has been symptomatic for <24 hrs.

**Is this true for BRAO as well?**

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

**Carbogen**

**Diamox**

**Glaucoma drops**

**Ocular massage**

**Anterior chamber paracentesis**

**Sublingual nitro**
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream.* Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

**Dilate the retinal vasculature**

*What is the timeframe of CRAO treatment?*

Primate studies indicate that retinal cells undergo irreversible ischemic damage within 90 minutes of CRA ligature—a dauntingly small window of opportunity for treatment. However, as many CRAO are felt to be incomplete, and as hope springs eternal in the human breast, most clinicians will treat up to 24 hours after onset.

*Two approaches to getting an embolus to pass:*

1. **Carbogen**
2. **Diamox**
3. **Glaucoma drops**
4. **Sublingual nitro**
5. **Ocular massage**
6. **Anterior chamber paracentesis**

*What other management step must be taken?*

The pt should be referred immediately to a stroke center if the event is acute, ie, if s/he has been symptomatic for <24 hrs.

*Is this true for BRAO as well?*

Yes
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

What is the timeframe of CRAO treatment?

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Is this true for BRAO as well?

Yes

What other management step must be taken?

The pt should be referred immediately to a stroke center if the event is acute, ie, if s/he has been symptomatic for >24 hrs.

What if it’s been longer than 24 hrs?

Yes

Carbogen

Ocular massage

Glaucoma drops

Dilate the retinal vasculature

Carbogen
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature. Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**What is the timeframe of CRAO treatment?**
Primate studies indicate that retinal cells undergo irreversible ischemic damage within 90 minutes of CRA ligature—a dauntingly small window of opportunity for treatment. However, as many CRAO are felt to be incomplete, and as hope springs eternal in the human breast, most clinicians will treat up to 24 hours after onset.

**What other management step must be taken?**
The pt should be referred immediately to a stroke center if the event is acute, ie, if s/he has been symptomatic for >24 hrs.

**Is this true for BRAO as well?**
Yes.

**What if it’s been longer than 24 hrs?**
They should be referred urgently, ie, they need to be stroke-evaluated within amount of time.

**Lower IOP** → **Two approaches to getting an embolus to pass:**

**Dilate the retinal vasculature**
CRAO management involves a single overarching goal: *Cause an embolus (if present) to pass downstream*. Methods for achieving this goal work via one of two mechanisms: Lowering IOP or dilating the retinal vasculature.

Six specific treatments are commonly employed—what are they? Finally, divide the treatments into their respective ‘prongs.’

**What is the timeframe of CRAO treatment?**
Primate studies indicate that retinal cells undergo irreversible ischemic damage within 90 minutes of CRA ligature—a dauntingly small window of opportunity for treatment. However, as many CRAO are felt to be incomplete, and as hope springs eternal in the human breast, most clinicians will treat up to 24 hours after onset.

**Lower IOP**

Two approaches to getting an embolus to pass:

**Dilate the retinal vasculature**

**What other management step must be taken?**

The pt should be referred immediately to a stroke center if the event is acute, ie, if s/he has been symptomatic for >24 hrs.

**Is this true for BRAO as well?**
Yes

**What if it’s been longer than 24 hrs?**
They should be referred urgently, ie, they need to be stroke-evaluated within 1 week.