The primary source for this slide-set is the 2021-22 revision of the BCSC Neuro-Oph book. Other sources include an AAO Focal Points issue on IIH, entries on the ONE Network’s Oculofacial Plastic Surgery and Pediatric Ophthalmology Education Centers IIH pages, and the EyeWiki IIH page. (Note that all are Academy sources.)
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*No question—proceed when ready*
You find bilateral disc edema in an obese young-adult female patient who c/o HA and transient visual obscurations. Your working diagnosis is IIH.

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How strong is the association between IIH and obesity?
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How strong is the association between IIH and obesity?

Very—90% of IIH pts are obese
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_How strong is the association between IIH and obesity?_  
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_How common is IIH in food-scarce regions marked by widespread and severe caloric malnutrition?_
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*How strong is the association between IIH and obesity?*
Very—90% of IIH pts are obese

*How common is IIH in food-scarce regions marked by widespread and severe caloric malnutrition?*
IIH is almost unheard of in such areas
You find bilateral disc edema in an obese young-adult female patient who c/o HA and transient visual obscurations. Your working diagnosis is IIH.

How strong is the association between IIH and female gender?
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*Females are more likely than males to develop IIH. Are they also more likely to suffer severe vision loss (SVL)?
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--Male gender
--African-American ancestry
--Morbid obesity
--Severity of papilledema
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What are the other risk factors for SVL in IIH?
--Male gender
--African-American ancestry
--Morbid obesity
--Severe papilledema
--Systemic condition
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What are the other risk factors for SVL in IIH?
--Male gender
--African-American ancestry
--Morbid obesity
--Severe papilledema
--Anemia
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What are the other risk factors for SVL in IIH?
--Male gender
--African-American ancestry
--Morbid obesity
--Severe papilledema

In addition to being a risk factor for SVL in IIH, the *Neuro* book lists *anemia* as a possible *cause* of the condition.
You find bilateral disc edema in an obese young-adult female patient who c/o HA and transient visual obscurations. Your working diagnosis is IIH.

More precisely than ‘young adult,’ during what developmental stage are women at greatest risk of developing IIH?
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More precisely than ‘young adult,’ during what developmental stage are women at greatest risk of developing IIH?
When they are of ‘child-bearing age.’
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When they are of ‘child-bearing age.’ In terms of age, IIH is most likely to occur when a woman is in her twenties.
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*Two other symptoms are especially common in IIH—what are they?*

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--Pulsatile tinnitus
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- Transient visual obscurations
- Pulsatile tinnitus
- Diplopia

Q: Upright vs supine—In which position is the HA in IIH worse?

A: Supine

Is this fact diagnostic of IIH?

No, but it is strongly suggestive that the HA is due to increased ICP (as opposed to other HA etiologies, in which HA intensity is lessened by supine positioning).
You find bilateral disc edema in an obese young-adult female patient who c/o **HA** and transient visual obscurations. Your working diagnosis is IIH.

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You find bilateral disc edema in an obese young-adult female patient who c/o HA and transient visual obscurations. Your working diagnosis is IIH.

Two other symptoms in IIH are:
- Headache
- Transient visual obscurations
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- Diplopia

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No, but it is strongly suggestive that the HA is due to increased ICP (as opposed to other HA etiologies, in which HA intensity is lessened by supine positioning).
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- You find bilateral disc edema in an obese young-adult female patient who c/o HA and **transient visual obscurations**. Your working diagnosis is IIH.

  **Two other symptoms are especially common in IIH—what are they?**
  - Headache
  - **Transient visual obscurations**

  **What is the classic provocative event for TVO?**
You find bilateral disc edema in an obese young-adult female patient who c/o HA and *transient visual obscurations*. Your working diagnosis is IIH.

Two other symptoms are especially common in IIH—what are they?
-- Headache
-- Transient visual obscurations

*What is the classic provocative event for TVO?*
A change in posture, especially after two words

Is increased ICP typically associated with visual complaints other than TVO (eg, decreased acuity, visual field defects, altered color vision)?
Only if it is severe or (especially) longstanding

You find bilateral disc edema in an obese young-adult female patient who c/o HA and transient visual obscurations. Your working diagnosis is IIH.

Two other symptoms are especially common in IIH—what are they?
--Headache
--**Transient visual obscurations**

What is the classic provocative event for TVO?
A change in posture, especially standing up after bending over.
You find bilateral disc edema in an obese young-adult female patient who c/o HA and **transient visual obscurations**. Your working diagnosis is IIH.

**Two other symptoms are especially common in IIH—what are they?**
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**What is the classic provocative event for TVO?**
A change in posture, especially standing up after bending.

**What if the pt complains of decreased vision when she gets overheated?**
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What is the classic provocative event for TVO?
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This is Uhthoff's phenomenon, and is suggestive of a different condition (two words), not IIH.
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No, more on the order of minutes.

**What are two classic overheating events known to provoke Uhthoff’s?**
- A hot shower
- Exercising
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Two other symptoms are especially common in IIH—what are they?

--- Headache
--- Transient visual obscurations
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**How long do the TVOs last?**
Seconds—no more than 30 or so. Afterwards, vision returns to baseline.
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*It should be noted that one specific pattern of VF loss is extremely common in IIH. What is it?*  
An enlarged blind spot secondary to the disc edema.
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--- Headache
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--- **Pulsatile tinnitus**
--- Diplopia

What causes the tinnitus?

Blood being forced through a narrowed portion of a venous sinus

Is the tinnitus usually unilateral, or bilateral?

Unilateral

Can the tinnitus be auscultated, ie, does it produce a pulse-synchronous bruit?

In some cases, yes

What maneuver?

Compression of the ipsilateral jugular vein (either by pressing on it, or when the pt turns her head)
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There are four so-called ‘cardinal symptoms’ of increased intracranial pressure. What are they?
--Headache
--Transient visual obscurations
--Pulse-synchronous tinnitus
--Diplopia

You find bilateral disc edema in an obese young-adult female patient who c/o HA and transient visual obscurations. Your working diagnosis is IIH.

Is the diplopia horizontal, or vertical? Horizontal
Is it an esotropia, or an exotropia? Esotropia
Is it comitant, or incomitant? Incomitant

What is the underlying mechanism? CN6 palsy
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**Q**

- Is the diplopia horizontal, or vertical? **Horizontal**
- Is it an esotropia, or an exotropia? **Esotropia**
- Is it comitant, or incomitant? **Incomitant**
- What is the underlying mechanism? **CN6 palsy**
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**Why do IIH pts get CN6 palsies?**

**CN6 palsy**

**Is the diplopia horizontal, or vertical?**
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Why do IIH pts get CN6 palsies? Blame the increased ICP. Recall that CN6 makes a 90° turn over the apex of the temporal bone as it enters the cavernous sinus. When ICP is elevated, the nerve gets stretched at this location, compromising its function and causing a palsy on that side.
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Another cranial nerve can be palsied—which one?
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**CN7 palsy**

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What is the underlying mechanism?

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CN7 palsy

Another cranial nerve can be palsied—which one?

CN7

Is CN7 palsy as commonly encountered as CN6?
There are four so-called “cardinal symptoms” of increased intracranial pressure. What are they?

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Is CN7 palsy as commonly encountered as CN6?
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Per the *Neuro* book, these are the only neurologic deficits associated with IIH.

Another cranial nerve can be palsied—which one?
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1 However *EyeWiki* says kids with IIH can manifest CN3 and/or CN4 palsies as well.

2 But the *Peds* book makes no mention of CN3/CN4 palsies, so...
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What imaging study (studies) should be ordered?

The current Neuro book asserts that 'all pts with suspected IIH should undergo MRI and MRV'

Both studies (MRI and MRV) have specific 'rule out' goals. What are they?
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IIH has four classic MRI/V abnormalities—what are they?

--?
--?
--?
--?
Q/A

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Are these pathognomonic for IIH, ie, do they 'rule it in'?
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Lumbar puncture (LP)

**Prior to LP, a different sort of study must be performed. What sort is that?**
Brain imaging

**Why must brain imaging be obtained?**
At a minimum, to rule out the presence of a space-occupying lesion that could result in tonsillar herniation when LP is performed.

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The current *Neuro* book contends that 'all pts with suspected IIH should undergo MRI and MRV'.

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**Both studies (MRI and MRV) have specific rule out goals. What are they?**
- **MRI**: Rule out... Mass, hydrocephalus or meningeal lesion
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MRI findings often found in patients with IIH:

*Top left:* Flattening of the posterior aspect of the globe
*Top right:* Partially empty sella
*Bottom left:* Enlargement of the optic nerve sheath.
*Bottom right:* Narrowing of the distal transverse sinus at its junction with the sigmoid sinus (yellow arrow)
MRV in IIH. Focal stenosis at the junction of the distal portion of the transverse sinus sigmoid sinuses. (The smooth, tapering stenosis noted along the right transverse sinus is a common finding as well.)
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**Q**

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**What are the units of measurement for ICP?**
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**What is the cutoff value for diagnosing IIH?**
Idiopathic Intracranial Hypertension (IIH)

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What are the units of measurement for ICP?
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The value 250 is used in the Neuro book, and thus should be one's answer on the OKAP, WQE, and Boards
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The value 250 is used in the *Neuro* book, and thus should be one’s answer on the OKAP, WQE, and Boards. That said, other sources contend this is too high, ie, that it sacrifices sensitivity for specificity. For this reason, many clinicians will accept values as low as 200 if the overall clinical picture is consistent with IIH.
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| Habitus             | Nonobese ≈ obese           | Obese                       |

A child
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Note how post-pubescent pediatric IIH is demographically equivalent to the adult version…

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Because pre-pubescent pediatric IIH is so dissimilar to the adult version, the Neuro book contends that IIH “appears to be a different disorder in prepubertal children”.

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**What is the normal opening pressure in children?**
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What is the normal opening pressure in children?
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Note that the upper limit of normal for children is a little higher than it is for adults (more on this shortly)
Can children develop IIH? Yes

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So, if a child has an opening pressure of, say, 250, does s/he have IIH?
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Hypoventilation

Hypoventilation leads to decreased O2 and increased CO2 levels, both of which induce dilation of the cerebral vasculature, which in turn increases ICP

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Q/A

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Can children develop IIH?
Yes.

With what common chromosomal condition is IIH associated?
Down syndrome.

How early can it present?
It has been reported in infancy, but is extremely rare before age 3 years.

What is its pediatric prevalence?
This has not been well defined.

After what age does it usually present?
11 years.

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Can you think of a condition--very common in obese adults--that is associated with hypoventilation, decreased O₂, and increased CO₂?
Obstructive sleep apnea (OSA).

Is there a relationship between IIH and OSA?
Indeed there is. OSA is on the differential for IIH. Further, if OSA is co-morbid with IIH, OSA will exacerbate it.

What implications does this have for the management of IIH?
The diagnosis of OSA should be considered in all IIH/potential IIH pts, and further testing should be pursued as warranted.

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Can you think of a condition--very common in obese adults--that is associated with hypoventilation, decreased O₂, and increased CO₂?
Obstructive sleep apnea (OSA)

How does hypoventilation increase ICP?
Hypoventilation leads to decreased O₂ and increased CO₂ levels, both of which induce dilation of the cerebral vasculature, which in turn increases ICP.

What effect should this have on your interpretation of opening pressure?
It must be taken into account, and factored into clinical decision-making.

What is the mechanism thought to account for the increase in ICP associated with sedation?
Hypoventilation

How does hypoventilation increase ICP?
Hypoventilation leads to decreased O₂ and increased CO₂ levels, both of which induce dilation of the cerebral vasculature, which in turn increases ICP.

Children often need to be sedated for LP. What effect (if any) does sedation have on opening pressure in kids?
It elevates it as much as 30 mm.

What implications does this have for the management of IIH?
The diagnosis of OSA should be considered in all IIH/potential IIH pts, and further testing should be pursued as warranted.

Is there a relationship between IIH and OSA?
Indeed there is. OSA is on the differential for IIH. Further, if OSA is co-morbid with IIH, OSA will exacerbate it.

What implications does this have for the management of IIH?
The diagnosis of OSA should be considered in all IIH/potential IIH pts, and further testing should be pursued as warranted.
Can children develop IIH?
Yes

With what common chromosomal condition is IIH associated?
Down syndrome

How early can it present?
It has been reported in infancy, but is extremely rare before age 3 years

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**Obstructive sleep apnea (OSA)**

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What is the normal opening pressure in children? It averages about 195 mm; it ranges from 100-280.

So, if a child has an opening pressure of, say, 250, does s/he have IIH? Maybe; your clinical suspicion will have to be your guide.

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What implications does this have for the management of IIH? The diagnosis of OSA should be considered in all IIH/potential IIH pts, and further testing should be pursued as warranted.
Can children develop IIH? Yes

What is its pediatric prevalence? This has not been well defined

How early can it present? It has been reported in infancy, but is extremely rare before age 3 years

After what age does it usually present? 11 years

What is the normal opening pressure in children? It averages about 195 mm; it ranges from 100-280

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*What is the differential for papilledema + HA?*

--IIH
--?
--?
--?
--?
--?
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--IIH
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--Intracranial space-occupying lesion (mass; bleed)
--Malignant hypertension
--Hydrocephalus
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Cerebral venous thrombosis  Extracranial venous obstruction
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--Intracranial space-occupying lesion (mass; bleed)

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**Obstruction of cerebral venous outflow**

The Neuro book mentions three risk factors for thrombosis—what are they?

Cerebral venous thrombosis

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| --? |
| --? |
| --? |
```
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--**Obstruction of cerebral venous outflow**

Cerebral venous thrombosis

| --OCP use  
| --Dehydration  
| --Thrombotic disorders |

Extracranial venous obstruction

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![Diagram showing differential diagnoses for papilledema and headache](diagram.png)

**Cerebral venous thrombosis**
--- OCP use
--- Dehydration
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--- ?
--- ?

*Compression of venous outflow structures might be due to…*
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- **Cerebral venous thrombosis**
  -- OCP use
  -- Dehydration
  -- Thrombotic disorders

- **Extracranial venous obstruction**
  -- Mass
  -- Scarring (e.g., s/p neck surgery)

*Compression of venous outflow structures might be due to...*
There are a number of pharmacologic and nutritional agents for which evidence of a causal relationship with intracranial HTN exists. In this next section, we will identify and discuss these agents.
Pharmacologic causes of (secondary) IH:
- This (and related) abx are known to cause IIH:
Pharmacologic causes of (secondary) IH:

- This (and related) abx are known to cause IIH: **Minocycline**
Pharmacologic causes of (secondary) IH:

- This (and related) abx are known to cause IIH: Minocycline
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What does DMARD stand for?
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What does DMARD stand for?
Disease-Modifying Anti-Rheumatic Drug
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- This antibiotic is also used as an acne medicine: Minocycline
- This antibiotic is used as a DMARD: Minocycline
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Unless you work in an Inuit village, you’re unlikely to see a pt who overindulged in polar bear liver. If a pt has hypervitaminosis A-induced IIH, what liver-based dietary supplement is likely the culprit?
Pharmacologic causes of (secondary) IH:

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- The test scenario will be a teenager with acne: Isotretinoin
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- This acne medicine is a Vitamin A derivative:
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Note the recurring theme here—hypervitaminosis A
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Can one develop hypervitaminosis A from ingesting excess vitamin-A precursor (ie, beta carotene)?
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Can one develop hypervitaminosis A from ingesting excess vitamin-A precursor (ie, beta carotene)?
No. Only the ingestion of pre-formed vitamin A in excess can cause it.
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Q: What will result from high levels of beta-carotene ingestion?
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What retinal-related ocular condition is isotretinoin notorious for causing?
Pharmacologic causes of (secondary) IH:

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*What retinal-related ocular condition is isotretinoin notorious for causing? Nyctalopia*
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**What retinal-related ocular condition is isotretinoin notorious for causing?**
Nyctalopia

**In layman’s terms, what is nyctalopia?**
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**In layman’s terms, what is nyctalopia?**
Night blindness
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What retinal-related ocular condition is isotretinoin notorious for causing? Nyctalopia

In layman’s terms, what is nyctalopia? Night blindness

What other, much less serious ocular condition is associated with isotretinoin use?
Pharmacologic causes of (secondary) IH:

- This (and related) abx are known to cause IIH: Minocycline
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What retinal-related ocular condition is isotretinoin notorious for causing?
Nyctalopia

In layman’s terms, what is nyctalopia?
Night blindness

What other, much less serious ocular condition is associated with isotretinoin use?
Meibomian gland disease, including chalazion development
Pharmacologic causes of (secondary) IH:

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- This antibiotic is used for UTIs:
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- This acne medicine is a Vitamin A derivative: Isotretinoin
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What is the brand-name for nitrofurantoin?
Pharmacologic causes of (secondary) IH:
- This (and related) abx are known to cause IIH: Minocycline
- This antibiotic is also used as an acne medicine: Minocycline
- This antibiotic is used as a DMARD: Minocycline
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- This acne medicine is a Vitamin A derivative: Isotretinoin
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What is the brand-name for nitrofurantoin? Macrobid
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- This antibiotic is no longer available in the US:
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- This acne medicine is a Vitamin A derivative: *Isotretinoin*
- This antibiotic is used for UTIs: *Nitrofurantoin*
- This antibiotic is no longer available in the US: *Nalidixic acid*
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- What medicine is implicated, for which I have failed to come up with an interesting question?
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- This antibiotic is used for UTIs: Nitrofurantoin
- This antibiotic is no longer available in the US: Nalidixic acid
- What medicine is implicated, for which I have failed to come up with an interesting question? Levothyroxine
Pharmacologic causes of (secondary) IH:

- This (and related) abx are known to cause IIH: *Minocycline*
- This antibiotic is also used as an acne medicine: *Minocycline*
- This antibiotic is used as a DMARD: *Minocycline*
- Use or withdrawal of this medicine can lead to IIH: *Steroids*
- The classic association is ‘polar bear liver:’ *Excess vitamin A*
- The test scenario will be a teenager with acne: *Isotretinoin*
- This acne medicine is a Vitamin A derivative: *Isotretinoin*
- This antibiotic is used for UTIs: *Nitrofurantoin*
- This antibiotic is no longer available in the US: *Nalidixic acid*
- What medicine is implicated, for which I have failed to come up with an interesting question? *Levothyroxine*

One population in particular is at risk for levothyroxine-induced IH--who is it?
Pharmacologic causes of (secondary) IH:

- This (and related) abx are known to cause IIH: Minocycline
- This antibiotic is also used as an acne medicine: Minocycline
- This antibiotic is used as a DMARD: Minocycline
- Use or withdrawal of this medicine can lead to IIH: Steroids
- The classic association is ‘polar bear liver:’ Excess vitamin A
- The test scenario will be a teenager with acne: Isotretinoin
- This acne medicine is a Vitamin A derivative: Isotretinoin
- This antibiotic is used for UTIs: Nitrofurantoin
- This antibiotic is no longer available in the US: Nalidixic acid
- What medicine is implicated, for which I have failed to come up with an interesting question? Levothyroxine

One population in particular is at risk for levothyroxine-induced IH--who is it? Children
Pharmacologic causes of (secondary) IH:

- Minocycline?
- Steroids?
- Vitamin A?
- Isotretinoin?
- Nitrofurantoin?
- Nalidixic acid?
- Levothyroxine?

*The Neuro book states the evidence of a connection with IH is strongest for three of these—which three?*
Pharmacologic causes of (secondary) IH:

- Minocycline
- Vitamin A
- Isotretinoin

The Neuro book states the evidence of a connection with IH is strongest for three of these—which three?

The cycline abx, vitamin A, and retinoic acid
Pharmacologic causes of (secondary) IH:

- Minocycline
- Vitamin A
- Isotretinoin

The Neuro book states the evidence of a connection with IH is strongest for three of these—which three?

The cycline abx, vitamin A, and retinoic acid

Again per the Neuro book: Is the connection between these substances and IH firmly established?
Pharmacologic causes of (secondary) IH:

- Minocycline
- Vitamin A
- Isotretinoin

The Neuro book states the evidence of a connection with IH is strongest for three of these—which three?
- The cycline abx, vitamin A, and retinoic acid
- Again per the Neuro book: Is the connection between these substances and IH firmly established?
  No—in fact, it states ‘a clear correlation is lacking’
• Regular f/u is important to ensure what two tx goals are met?
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

What should be checked to assess visual function and the continued viability of the optic nerves?
--?
--?
--?
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

What should be checked to assess visual function and the continued viability of the optic nerves?
--Acuity
--Color vision
--Visual fields
Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

*What should be checked to assess for resolution of ONH edema?*

--?

--?
Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

*What should be checked to assess for resolution of ONH edema?*

--Fundus photos
--OCT RNFL
Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

*What should be checked to assess for resolution of ONH edema?*

--Fundus photos

--OCT RNFL. **But take note**…serial RNFL measurements cannot be relied upon in isolation.
Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

What should be checked to assess for resolution of ONH edema?
--Fundus photos
--OCT RNFL. **But take note…serial RNFL measurements cannot be relied upon in isolation.**

Huh? Serial RNFLs seem like a straightforward way to document resolution of ONH edema. What’s the issue with using them?
● Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

What should be checked to assess for resolution of ONH edema?
--Fundus photos
--OCT RNFL. **But take note…**serial RNFL measurements **cannot be relied upon in isolation.**

_Huh?_ **Serial RNFLs seem like a straightforward way to document resolution of ONH edema.**

What’s the issue with using them?
The issue is, edema resolution is not the only event that could lead to a decline in RNFL measurement over time—so could ONH atrophy, precisely what we’re hoping to avoid.
Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

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--Fundus photos
--OCT RNFL. **But take note…serial RNFL measurements cannot be relied upon in isolation.**

_Huh? Serial RNFLs seem like a straightforward way to document resolution of ONH edema._

**What’s the issue with using them?**
The issue is, edema resolution is not the only event that could lead to a decline in RNFL measurement over time—so could ONH atrophy, precisely what we’re hoping to avoid. **So a decrease in RNFL thickness cannot, of itself, be interpreted as evidencing edema resolution.**
Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

What should be checked to assess for resolution of ONH edema?
--Fundus photos
--OCT RNFL. **But take note…serial RNFL measurements cannot be relied upon in isolation.**

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The issue is, edema resolution is not the only event that could lead to a decline in RNFL measurement over time—so could ONH atrophy, precisely what we’re hoping to avoid. **So a decrease in RNFL thickness cannot, of itself, be interpreted as evidencing edema resolution.**

How could one go about differentiating between RNFL reduction due to retreating edema vs RNFL reduction due to advancing atrophy?
Q/A

Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

What should be checked to assess for resolution of ONH edema?
--Fundus photos
--OCT RNFL. **But take note…serial RNFL measurements cannot be relied upon in isolation.**

_Huh? Serial RNFLs seem like a straightforward way to document resolution of ONH edema. What’s the issue with using them?_

The issue is, edema resolution is not the only event that could lead to a decline in RNFL measurement over time—so could ONH atrophy, precisely what we’re hoping to avoid. **So a decrease in RNFL thickness cannot, of itself, be interpreted as evidencing edema resolution.**

_How could one go about differentiating between RNFL reduction due to retreating edema vs RNFL reduction due to advancing atrophy?_

By simultaneously monitoring (via OCT) the thickness of the retinal [three words]...
Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

What should be checked to assess for resolution of ONH edema?
- Fundus photos
- OCT RNFL. **But take note…serial RNFL measurements cannot be relied upon in isolation.**

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The issue is, edema resolution is not the only event that could lead to a decline in RNFL measurement over time—so could ONH atrophy, precisely what we’re hoping to avoid. **So a decrease in RNFL thickness cannot, of itself, be interpreted as evidencing edema resolution.**

_How could one go about differentiating between RNFL reduction due to retreating edema vs RNFL reduction due to advancing atrophy?_

By simultaneously monitoring (via OCT) the thickness of the retinal **ganglion cell complex (GCC)**.
Q

- Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

What should be checked to assess for resolution of ONH edema?
--Fundus photos
--OCT RNFL. **But take note…**serial RNFL **measurements cannot be relied upon in isolation.**

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The issue is, edema resolution is not the only event that could lead to a decline in RNFL measurement over time—so could ONH atrophy, precisely what we’re hoping to avoid. **So a decrease in RNFL thickness cannot, of itself, be interpreted as evidencing edema resolution.**

*How could one go about differentiating between RNFL reduction due to retreating edema vs RNFL reduction due to advancing atrophy?*
By simultaneously monitoring (via OCT) the thickness of the retinal **ganglion cell complex (GCC)**

*How does knowing GCC thickness help one interpret changes in RNFL thickness?*
Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

What should be checked to assess for resolution of ONH edema?
--Fundus photos
--OCT RNFL. **But take note…serial RNFL measurements cannot be relied upon in isolation.**

*Huh? Serial RNFLs seem like a straightforward way to document resolution of ONH edema.*
What’s the issue with using them?
The issue is, edema resolution is not the only event that could lead to a decline in RNFL measurement over time—so could ONH atrophy, precisely what we’re hoping to avoid. **So a decrease in RNFL thickness cannot, of itself, be interpreted as evidencing edema resolution.**

*How could one go about differentiating between RNFL reduction due to retreating edema vs RNFL reduction due to advancing atrophy?*
By simultaneously monitoring (via OCT) the thickness of the retinal *ganglion cell complex* (GCC)

*How does knowing GCC thickness help one interpret changes in RNFL thickness?*
Because while ONH edema resolution would cause the RNFL to decrease, no concomitant change in retinal GCC thickness should be seen.
A

Idiopathic Intracranial Hypertension (IIH)

- Regular f/u is important to ensure what two tx goals are met? **Edema resolution** and vision maintenance.

What should be checked to assess for resolution of ONH edema?
--Fundus photos
--OCT RNFL. **But take note…serial RNFL measurements cannot be relied upon in isolation.**

_Huh? Serial RNFLs seem like a straightforward way to document resolution of ONH edema._
What’s the issue with using them?
The issue is, edema resolution is not the only event that could lead to a decline in RNFL measurement over time—so could ONH atrophy, precisely what we’re hoping to avoid. So a decrease in RNFL thickness cannot, of itself, be interpreted as evidencing edema resolution.

*How could one go about differentiating between RNFL reduction due to retreating edema vs RNFL reduction due to advancing atrophy?*
By simultaneously monitoring (via OCT) the thickness of the retinal **ganglion cell complex** (GCC).

_How does knowing GCC thickness help one interpret changes in RNFL thickness?*_
Because while ONH edema resolution would cause the RNFL to decrease, no concomitant change in retinal GCC thickness should be seen. In contrast, atrophy of the ONH would cause both the RNFL and the GCC to thin over time.
Regular f/u is important to ensure what two tx goals are met? **Edema resolution and vision maintenance**

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment?
• Regular f/u is important to ensure what two tx goals are met? **Edema resolution and vision maintenance**

• If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? **Weight loss +/- symptomatic HA relief**
• Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance
• If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? **Weight loss** +/- symptomatic HA relief

How much weight must be lost to ameliorate IIH?
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.

How much weight must be lost to ameliorate IIH? Loss of as little as 5% may be enough!
• Regular f/u is important to ensure what two tx goals are met? **Edema resolution and vision maintenance**

• If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? **Weight loss +/- symptomatic HA relief**

• If a patient has moderate dz, or fails the above, what is added?
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide.
Q

- Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance
- If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief
- If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

What is the dose of acetazolamide?
• Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance

• If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief

• If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

*What is the dose of acetazolamide?*
1-4 g/day
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

What is the dose of acetazolamide?
1-4 g/day

What are the classic side effects of acetazolamide?
--?
--?
--?
Q/A

- Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.
- If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.
- If a patient has moderate dz, or fails the above, what is added? PO acetazolamide.

**What is the dose of acetazolamide?**
1-4 g/day

**What are the classic side effects of acetazolamide?**
--Tingling of fingers, toes and perioral area
--?
--?
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

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If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide.

*What is the dose of acetazolamide?*
1-4 g/day

*What are the classic side effects of acetazolamide?*
--Tingling of fingers, toes and perioral area
--Altered taste of food (described as ___ )
--?
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

What is the dose of acetazolamide?
1-4 g/day

What are the classic side effects of acetazolamide?
--Tingling of fingers, toes and perioral area
--Altered taste of food (described as ‘metallic’)
--?
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

What is the dose of acetazolamide?
1-4 g/day

What are the classic side effects of acetazolamide?
--Tingling of fingers, toes and perioral area
--Altered taste of food (described as ‘metallic’)
--Lassitude
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide.

Why is the altered-taste side effect not necessarily a bad thing?

- Tingling of fingers, toes and perioral area (described as ‘metallic’)
- Lassitude

Acetazolamide?
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide.

**Why is the altered-taste side effect not necessarily a bad thing?**
Because it might lead to reduced caloric intake, leading in turn to weight loss.

---

**Altered taste of food**
-- Tingling of fingers, toes and perioral area (described as ‘metallic’)
-- Lassitude
Regular f/u is important to ensure what two tx goals are met? **Edema resolution and vision maintenance**

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? **Weight loss +/- symptomatic HA relief**

If a patient has moderate dz, or fails the above, what is added? **PO acetazolamide**

And if they continue to worsen?
Regular f/u is important to ensure what two tx goals are met? **Edema resolution and vision maintenance**

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? **Weight loss +/- symptomatic HA relief**

If a patient has moderate dz, or fails the above, what is added? **PO acetazolamide**

And if they continue to worsen? **Topiramate**
Q/A

- Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance

- If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief

- If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

- And if they continue to worsen? **Topiramate**

*With regard to treating IIH, topiramate has three things (properties) going for it. What are they?*

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--?

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Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide.

And if they continue to worsen? **Topiramate**

*With regard to treating IIH, topiramate has three things (properties) going for it. What are they?*

--Like acetazolamide, it has **mechanism of action** (three words) effects

--?

--?
Q/A

- Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance
- If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief
- If a patient has moderate dz, or fails the above, what is added? PO acetazolamide
- And if they continue to worsen? **Topiramate**

*With regard to treating IIH, topiramate has three things (properties) going for it. What are they?*
- Like acetazolamide, it has carbonic anhydrase inhibition effects
- ?
- ?
Q/A

- Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance
- If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief
- If a patient has moderate dz, or fails the above, what is added? PO acetazolamide
- And if they continue to worsen? **Topiramate**

*With regard to treating IIH, topiramate has three things (properties) going for it. What are they?*
- Like acetazolamide, it has carbonic anhydrase inhibition effects
- With respect to HA control it has a [two words] effect
- ?
Q/A

- Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance
- If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief
- If a patient has moderate dz, or fails the above, what is added? PO acetazolamide
- And if they continue to worsen? Topiramate

*With regard to treating IIH, topiramate has three things (properties) going for it. What are they?*
--Like acetazolamide, it has carbonic anhydrase inhibition effects
--With respect to HA control it has a direct analgesic effect
--?
• Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance

• If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief

• If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

• And if they continue to worsen? Topiramate

*With regard to treating IIH, topiramate has three things (properties) going for it. What are they?
--Like acetazolamide, it has carbonic anhydrase inhibition effects
--With respect to HA control it has a direct analgesic effect
--It acts as an appetite suppressant, and thus will promote weight loss*
Q/A

- Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance
- If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief
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- And if they continue to worsen? **Topiramate**

*With regard to treating IIH, topiramate has three things (properties) going for it.*
*What are they?*
--Like acetazolamide, it has carbonic anhydrase inhibition effects
--With respect to HA control it has a direct analgesic effect
--It acts as an appetite suppressant, and thus will promote weight loss
Idiopathic Intracranial Hypertension (IIH)

- Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance
- If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief
- If a patient has moderate dz, or fails the above, what is added? PO acetazolamide
- And if they continue to worsen? Topiramate

If acetazolamide and topiramate are contraindicated, not tolerated, or only partially effective, what drug can be used or added?
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide.

And if they continue to worsen? Topiramate.

If acetazolamide and topiramate are contraindicated, not tolerated, or only partially effective, what drug can be used or added? Furosemide.
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

And if they continue to worsen? Topiramate

What about steroids—should they be tried?
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide.

And if they continue to worsen? Topiramate.

What about steroids—should they be tried? Per the Neuro book, only in severe cases, and in a limited manner (short course; high-dose; delivered IV).
Regular f/u is important to ensure what two tx goals are met? **Edema resolution and vision maintenance**

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? **Weight loss +/- symptomatic HA relief**

If a patient has moderate dz, or fails the above, what is added? **PO acetazolamide**

And if they continue to worsen? **Topiramate**

If they continue to lose VF?
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

And if they continue to worsen? Topiramate

If they continue to lose VF? Consider ON sheath fenestration (ONSF)
**Idiopathic Intracranial Hypertension (IIH)**

- Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.
- If a patient has mild dz (little edema; minimal VF loss), what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.
- If a patient has moderate dz, or fails the above, what is added? PO acetazolamide.
- And if they continue to worsen? Topiramate.
- If they continue to lose VF? Consider ON sheath fenestration (ONSF)

*Is ONSF an appropriate intervention if the primary goal is HA amelioration?*
- Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

- If a patient has mild dz (little edema; minimal VF loss), what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.

- If a patient has moderate dz or fails the above, what is added? PO acetazolamide.

- And if they continue to worsen? Topiramate.

- If they continue to lose VF? Consider ON sheath fenestration (ONSF).

Is ONSF an appropriate intervention if the primary goal is HA amelioration? Per the Neuro book, ONSF “does not reliably treat HA.”

Is ONSF effective at reducing ICP? No (this is probably why it doesn’t treat HA well). Oh well, you can’t have it all I guess. At least it’s an effective long-term tx for preventing vision loss, right? The Neuro book states its long-term success rate in this regard is “unclear.” Is it at least safe??!! Not to the extent one would like—it carries a complication rate of 10-15% (including a 1-2% chance of vision loss, ironically).
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

And if they continue to worsen? Topiramate

If they continue to lose VF? Consider **ON sheath fenestration (ONSF)**

*Is ONSF an appropriate intervention if the primary goal is HA amelioration?* Per the *Neuro* book, ONSF “does not reliably treat HA”

*Is ONSF effective at reducing ICP?*
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

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And if they continue to worsen? Topiramate.

Is ONSF an appropriate intervention if the primary goal is HA amelioration? Per the Neuro book, ONSF “does not reliably treat HA”.

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If they continue to lose VF? Consider ON sheath fenestration (ONSF)
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance

If a patient has mild dz (little edema; minimal VF loss), what are the mainstays of treatment? Weight loss +/- symptomatic HA relief

If a patient has moderate dz, or fails the above, what is added? PO acetazolamide

And if they continue to worsen? Topiramate

If they continue to lose VF? Consider ON sheath fenestration (ONSF)

Is ONSF an appropriate intervention if the primary goal is HA amelioration? Per the Neuro book, ONSF “does not reliably treat HA”

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If a patient has moderate dz, or fails the above, what is added? PO acetazolamide.

And if they continue to worsen? Topiramate.

If they continue to lose VF? Consider ON sheath fenestration (ONSF).

Is ONSF an appropriate intervention if the primary goal is HA amelioration? Per the Neuro book, ONSF “does not reliably treat HA”.

Is ONSF effective at reducing ICP? No (this is probably why it doesn’t treat HA well).

Oh well, you can’t have it all I guess. At least it’s an effective long-term tx for preventing vision loss, right?

The Neuro book states its long-term success rate in this regard is “unclear.”

Is it at least safe??!! Not to the extent one would like—it carries a complication rate of 10-15% (including a 1-2% chance of vision loss, ironically).
Regular f/u is important to ensure what two tx goals are met? Edema resolution and vision maintenance.

If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment? Weight loss +/- symptomatic HA relief.

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And if they continue to worsen? Topiramate.

If they continue to lose VF? Consider **ON sheath fenestration (ONSF)**.

**Is ONSF an appropriate intervention if the primary goal is HA amelioration?**
Per the *Neuro* book, ONSF “does not reliably treat HA”

**Is ONSF effective at reducing ICP?**
No (this is probably why it doesn’t treat HA well)

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What is the mechanism by which ONSF arrests loss of visual function?

This is controversial. The obvious answer is that the fenestration allows CSF to percolate out of the subarachnoid space, thereby reducing pressure--the brain-equivalent of a trab. (It's not for nothing that IIH has been called 'glaucoma of the brain.') However, this explanation is problematic, as studies indicate fenestrations often scar down, thus rendering long-term CSF egress impossible.

That said, the phenomenon of scarring provides a neat explanation for the ability of ONSF to arrest loss of visual function—namely, that circumferential scarring at the surgery site prevents CSF from reaching the ONH, thereby relocating the 'pressure head' from the vulnerable circulatory watershed zone at the ONH to the robustly-perfused retrobulbar region of the nerve.
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Are shunts effective in treating IIH?

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Are shunts effective in treating IIH? Generally yes.

What is their main drawback? A significant portion will fail, and thus require revision (and many will require multiple revisions).

Why would you consider ON sheath fenestration (ONSF)? Consider a shunt or stenting procedure.
A

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What about repeated LPs—is that an appropriate treatment? The Neuro book flatly states it is not.

If they continue to have intractable HA? Consider a shunt or stenting procedure or repeated LPs? No.
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After an LP, how long does it take for ICP to return to its pre-procedure level?

About 90 minutes

But some pts experience HA relief for day to weeks after an LP. How is this possible if ICP returns to baseline in an hour and a half?

Probably because the drop in ICP induced by the procedure allows the flattened venous sinuses to re-cannulate, which in turn re-establishes normal CSF circulatory dynamics. The inevitable re-collapse of the sinuses (with its accompanying re-derangement of CSF circulation) leads to recurrence of the HA.
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