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How common is a complaint of headache among IIH patients?
Extremely—in some studies, 99% of IIH patients report a headache

- How severe is the HA?
  - Severe

- Is it worse in the morning, or the evening?
  - Morning

- Does it ever awaken the pt from sleep?
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- Upright vs supine—In which position is the HA in IIH worse?
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**What co-morbidity commonly found in IIH pts greatly complicates the clinical picture?**

- Many of these pts suffer from one of the so-called headache syndromes

Because sometimes a HA is just a HA. That is, it is difficult for the clinician to distinguish between HA secondary to increased ICP (which might require modification of IIH tx) vs HA due to the syndrome.

So-called ‘rebound headache syndrome’ is notorious for occurrence in the IIH population. Two substances are commonly implicated. What are they?

- Analgesics
- Caffeine
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How common are TVOs in IIH?

Very—about 75% of IIH pts experience them.

What is the classic provocative event for TVO?
Change in posture, especially standing up after bending over.

How long do the TVOs last?
Seconds—no more than 30 or so. Afterwards, vision returns to baseline.

What if the pt complains of decreased vision when she gets overheated?
This is Uhthoff’s phenomenon, and is suggestive of multiple sclerosis, not IIH.

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Non-blind spot field loss occurs in over 90% of cases. Other than an enlarged blind spot, what patterns of VF loss occur in IIH? It tends to be rather glaucoma-like, starting with inferior nasal steps, then superior, followed by arcuates.

What about central and ceco-central defects? These are not expected, and suggest that something other than IIH is the culprit.
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Does this mean VF loss doesn’t occur in IIH?

No--to the contrary, VF loss occurs in almost all cases

Ah, you’re referring to enlargement of the blind spot (BS), yes?

Non-blind spot field loss occurs in over 90% of cases.

Other than an enlarged blind spot, what patterns of VF loss occur in IIH?

It tends to be rather glaucoma-like, starting with inferior nasal steps, then superior, followed by arcuates.

What about central and ceco-central defects?

These are not expected, and suggest that something other than IIH is the culprit.

Do VF defects improve/resolve with (successful) treatment?

Depends on which defects we’re talking about. The enlarged blind spot essentially always resolves, whereas the steps/arcuates resolve in about half of cases.
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Does this mean VF loss doesn’t occur in IIH?
No--to the contrary, VF loss occurs in almost all cases.

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No. While such enlargement does occur in essentially all cases, **non-blind spot** field loss occurs in over 90% of cases.

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If the pt complains of decreased vision when she gets overheated, this is Uhthoff’s phenomenon, and is suggestive of multiple sclerosis, not IIH.

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**Refraction.** The enlarged blind spot is usually secondary to a hyperopic shift in the peripapillary receptors (edema elevates these receptors, shortening their effective axial length and thereby producing a local hyperopic shift).

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Is the diplopia horizontal, or vertical? (Esotropia, Incomitant, CN6 palsy)
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Idiopathic Intracranial Hypertension (IIH)
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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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A suspected IIH pt with an obvious CN6 palsy is found to have a small vertical deviation on careful cover testing. Should the diagnosis be questioned?

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Not necessarily. 'Pure' CN6 palsies can manifest small vertical imbalances.
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How prevalent is obesity among IIH pts?
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Very. Over **%** of IIH pts are obese!
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How prevalent is obesity among IIH pts?
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Among all the signs of IIH, what is the status of disc edema?
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What is IIH without papilledema called? It is called 'IIH without papilledema'.

What is the incidence/prevalence? These have not been well established, but it is felt to be quite rare.

How does it present? In the same manner as IIH with papilledema (ie, an obese young-adult female who c/o HA, TVO, tinnitus, etc).

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**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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Caveat emptor: The (latest-in-my-possession) Neuro-Oph book disagrees, suggesting that CT (with proper angiographic protocols) is adequate!
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What structures (there are four of them) must be unremarkable on MR imaging to allow a diagnosis of IIH?
- Enlarged and ‘unfolded’ (ie, straightened) optic nerves
- Empty sella
- Flattened globes
- Collapse of the lateral venous sinuses (aka ‘smooth-walled venous stenosis’)

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What position should the pt be in for the LP?

What are the units of measurement for ICP?

Millimeters or centimeters of water

What is the cutoff value for diagnosing IIH?

This is controversial. Some recommend using 250 (this is the value listed by the Neuro-Oph book). 250 is very specific for increased ICP, but has poor sensitivity. For this reason, many clinicians will accept as diagnostic values as low as 200 if the overall clinical picture is consistent with IIH.
Q/A

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The lateral decubitus is probably preferred (and is the position promoted by the latest version of the BCSC Neuro-Oph book in my possession). That said, other positions can be used if needed, so long as the opening pressure is measured from the height of the anatomical landmark (two words).
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What is its pediatric prevalence?
This has not been well defined, but it is felt to be uncommon
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Yes

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With what common chromosomal condition is IIH associated? Down syndrome

How early can it present? It has been reported in infancy

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This is the consensus of the BCSC *Peds* book along with two other Academy sources. The *Neuro-Oph* book seems to suggest pre-pubescent boys and nonobese children are more likely to develop IIH than are their female and obese counterparts, but this may be the result of poor phrasing on the book’s part. I recommend going with the version in the table below.

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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.

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Head’s up: While couched here in a discussion of Pediatric IIH, note that all the issues of hyper/hypoventilation and their effect on ICP apply to LPs performed on adults as well!
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- This antibiotic is also used as an acne medicine: Minocycline
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- Use *or* withdrawal of this medicine can lead to IIH:
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
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 withdrawal of steroids is well-documented as causing IIH. But does the **use** of steroids do so as well? The latest (in my possession) version of the BCSC Neuro-oph book maintains that it does. But again, more recent literature from the Academy tends to differ, indicating this claim is incorrect. Further, the Neuro-Oph book states that a short course of high-dose steroids ‘may be beneficial’ in severe/fulminant cases. **Caveat emptor, as before.**
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
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Pharmacologic causes of (secondary) IH:
- This (and related) abx are known to cause IIH: *Minocycline*
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- 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*
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- The classic association is ‘polar bear liver:’ *Excess vitamin A*

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:

- This (and related) abx are known to cause IIH: Minocycline
- This antibiotic is also used as an acne medicine: 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?

Cod-liver oil
Pharmacologic causes of (secondary) IH:

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- 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:
Pharmacologic causes of (secondary) IH:
- This (and related) abx are known to cause IIH: Minocycline
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- The classic association is ‘polar bear liver:’ Excess vitamin A
- The test scenario will be a teenager with acne: Isotretinoin
Pharmacologic causes of (secondary) IH:

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

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Note the recurring theme here—hypervitaminosis A. It is a well-established cause of secondary IH!
Pharmacologic causes of (secondary) IH:

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- This antibiotic is also used as an acne medicine: Minocycline
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Note the recurring theme here--hypervitaminosis A. It is a well-established cause of secondary IH!

Can one develop hypervitaminosis A from ingesting excess vitamin-A precursor (ie, beta carotene)?
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
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**Note the recurring theme here—hypervitaminosis A. It is a well-established cause of secondary IH!**

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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?

Note the recurring theme here—hypervitaminosis A. It is a well-established cause of secondary IH!
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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- 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

Note the recurring theme here--hypervitaminosis A. It is a well-established cause of secondary IH!

What will result from high levels of beta-carotene ingestion? Carotenosis, a benign condition the hallmark of which is the development of an orange tint to the skin.
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**
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- 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**

What retinal-related ocular condition is isotretinoin notorious for causing?
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
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What retinal-related ocular condition is isotretinoin notorious for causing?
Nyctalopia
Pharmacologic causes of (secondary) IH:

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

In layman’s terms, what is nyctalopia?
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
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What retinal-related ocular condition is isotretinoin notorious for causing?
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In layman’s terms, what is nyctalopia?
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What other, much less serious ocular condition is associated with isotretinoin use?
Pharmacologic causes of (secondary) IH:

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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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What is the brand-name for nitrofurantoin?
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What is the brand-name for nitrofurantoin?
Macrobid
Pharmacologic causes of (secondary) IH:
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- This antibiotic is used as a DMARD: Minocycline
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- This antibiotic is used for UTIs: Nitrofurantoin
- This antibiotic is no longer available in the US:
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- What medicine is implicated, for which I have failed to come up with an interesting question?
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One population in particular is at risk for levothyroxine-induced IH--who is it?
Pharmacologic causes of (secondary) IH:

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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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- The test scenario will be a teenager with acne: **Isotretinoin**
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- 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*
What is the most appropriate way to monitor the visual status of an IIH patient?
What is the most appropriate way to monitor the visual status of an IIH patient? **Serial VF exams**
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If a patient has mild dz (little edema; minimal VF loss) what are the mainstays of treatment?
What is the most appropriate way to monitor the visual status of an IIH patient? **Serial VF exams**

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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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?*
What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams

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!
What is the most appropriate way to monitor the visual status of an IIH patient? **Serial VF exams**

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?
What is the most appropriate way to monitor the visual status of an IIH patient? **Serial VF exams**

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What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams

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 starting dose of acetazolamide?

Options vary, but 250 or 500 bid is a reasonable starting dose

If the starting dose is ineffective, should the dose be raised? Yes. It should be titrated to effect, or to side-effect intolerance
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What are the classic side effects of acetazolamide?
Q/A

- What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams
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What are the classic side effects of acetazolamide?
--Tingling of fingers, toes and perioral area
--Altered taste of food ('metallic' taste)
--Lassitude
What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams.

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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, thereby producing weight loss.

What is the starting dose of acetazolamide? Opinions vary, but 250 or 500 bid is a reasonable starting dose.

If the starting dose is ineffective, should the dose be raised? Yes. It should be titrated to effect, or to side-effect intolerance.

What are the classic side effects of acetazolamide? -- Tingling of fingers, toes and perioral area -- Altered taste of food (metallic taste) -- Lassitude.
What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams

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

What are the classic side effects of acetazolamide?
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What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams

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

Note: While my Neuro-Oph book considers acetazolamide the first-line drug in IIH, my Glaucoma book, citing possible teratogenicity, states flatly that “oral CAIs should not be used by women in their childbearing years.” Caveat emptor.
What is the most appropriate way to monitor the visual status of an IIH patient? **Serial VF exams**

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?
What is the most appropriate way to monitor the visual status of an IIH patient? **Serial VF exams**

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- And if they continue to worsen? **Topiramate**
What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams

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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?
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--
--
Q/A

- What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams
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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
What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams.

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.
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What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams

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?
What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams.

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.
What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams

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Is ONSF an appropriate intervention if the primary goal is headache amelioration? Yet another point of contention. My Neuro-Oph book declares it is largely ineffective as a tx for HA, whereas a more recent Academy publication says it is effective, but only if the HA is frontal and mild.
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Is ONSF an appropriate intervention if the primary goal is ICP reduction? My Neuro-Oph book declares flatly that it is ineffective in this regard.
What is the most appropriate way to monitor the visual status of an IIH patient? Serial VF exams.

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

- And if they continue to worsen? Topiramate.
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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 many fenestrations scar down, thereby precluding long-term CSF egress.

That said, the phenomenon of scarring provides an alternative explanation—specifically, that circumferential scarring prevents CSF from reaching the ONH, thereby moving the pressure head from the vulnerable circulatory watershed zone that is the ONH to the robustly-perfused retrobulbar region of the nerve. Unfortunately, while this explanation circumvents the problem posed by cessation of CSF egress, it can’t explain those cases in which ONSF 1) does effectively lower ICP, 2) effectively treats HA (note the hedging use of the term largely above), or 3) arrests loss of visual function in the fellow eye.

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Q

**Idiopathic Intracranial Hypertension (IIH)**

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If they continue to have intractable HA? Consider a shunt procedure.

What are examples of shunting procedures employed in managing IIH?

Lumbar-peritoneal
Ventriculo-atrial
Ventriculo-jugular
Ventriculo-peritoneal

Are shunts effective in treating IIH? Generally yes.

What is their main drawback? Over half will fail, and thus require revision (and many will require multiple revisions).

All are in the form ‘space the shunt starts’ – ‘space the shunt ends.’
(There are a total of two starting spaces and three ending spaces.)
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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?

The brief drop in ICP allows the flattened venous sinuses to re-form, thereby re-establishing normal CSF circulatory dynamics. The inevitable re-collapse of the sinuses and subsequent derangement of CSF circulation leads to recurrence of the HA.
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