

Rebound Intracranial Hypertension

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Objectives

- Define rebound intracranial hypertension
- Discuss epidemiology
- Contrast clinical presentation of SIH and RIH
- List known risk factors
- Evaluate treatment options for RIH
- Explore RIH pathophysiology

Rebound Intracranial Hypertension

Follows procedural treatment of SIH with epidural blood patching, CSF venous fistula embolization or ligation, or surgical dural repair

Reverse orthostatic headache different from the original SIH headache

Resolution of headache following administration of oral acetazolamide

Not better accounted for by another cause of headache

Epidemiology

Overall Incidence

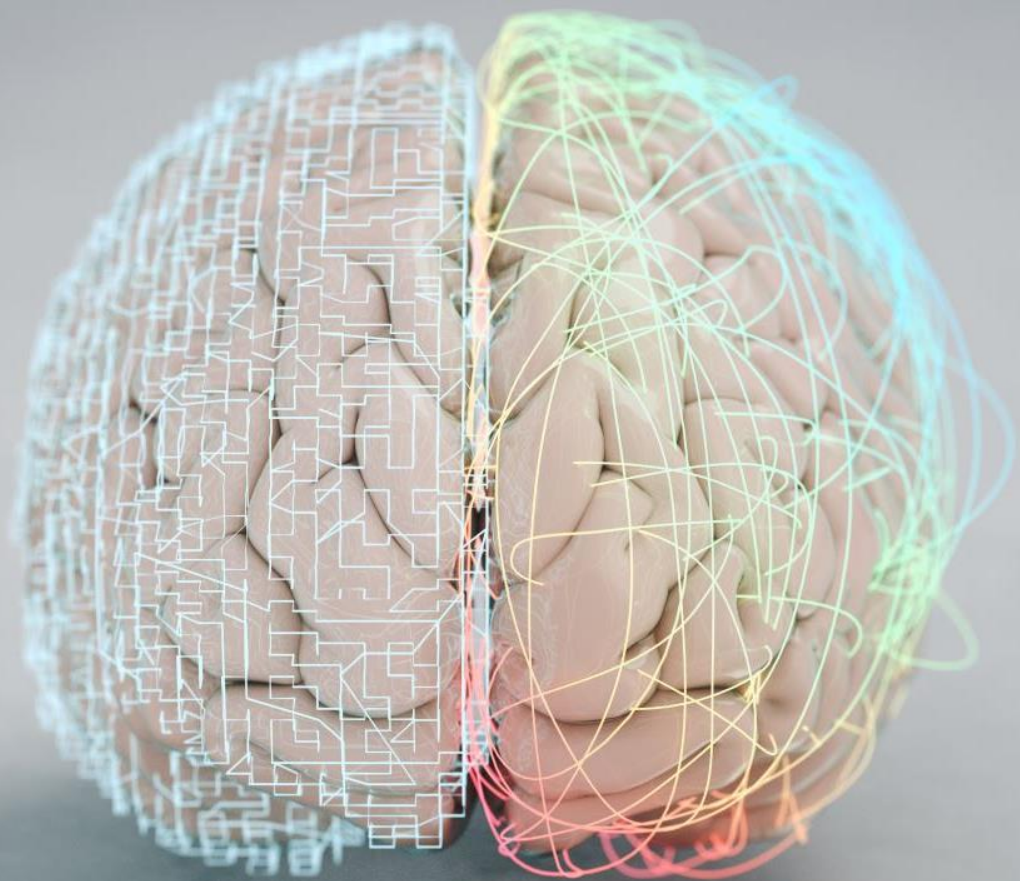
- ~1/4 of patients treated for SIH, female predominant, 5th decade of life

Procedural Relationship

- Slightly higher incidence with surgical vs non-surgical intervention (29.4% vs 21.4%)
- 36% after minimally invasive dural repair
- Not linked to patch volume

CSF Venous Fistula Embolization

- Linked to treatment success?
- 5.2% of patients with complete resolution, 29.7% with improvement but no resolution, and 60% with no improvement of SIH symptoms experienced RIH 3 months post-procedure



Risk Factors

- Extensive extradural CSF collection
- Obesity + CSF Venous Fistula increased risk of developing RIH and papilledema
- IIH, history of, or risk factors of IIH (eg weight gain, untreated OSA)
- Not predictive: brain sag, pre-procedure opening pressure

Schievink WI et al. Spinal CSF-venous fistulas in morbidly and super obese patients with spontaneous intracranial hypotension. *AJNR Am J Neuroradiol.* 2021
Kranz PG et al. Spontaneous intracranial hypotension: pathogenesis, diagnosis, and treatment. *Neuroimaging Clin N Am.* 2019
Sulioti et al. Popping the balloon: Abrupt onset of a spinal CSF leak and spontaneous intracranial hypotension in idiopathic intracranial hypertension, a case report. *Headache.* 2022

Clinical Presentation



66-74% 24-72 hours after procedure
22% 3-7 days following
94% resolve within 3 months

SIH

Suboccipital

Worsened with recumbency

Best in AM after sleep

RIH

Frontal or peri-orbital

Worsened upright

Worsened in AM after sleep

Nausea, vomiting, blurred vision, transient papilledema

Exceptions

RIH presenting
with occipital
or
non-frontal
pain

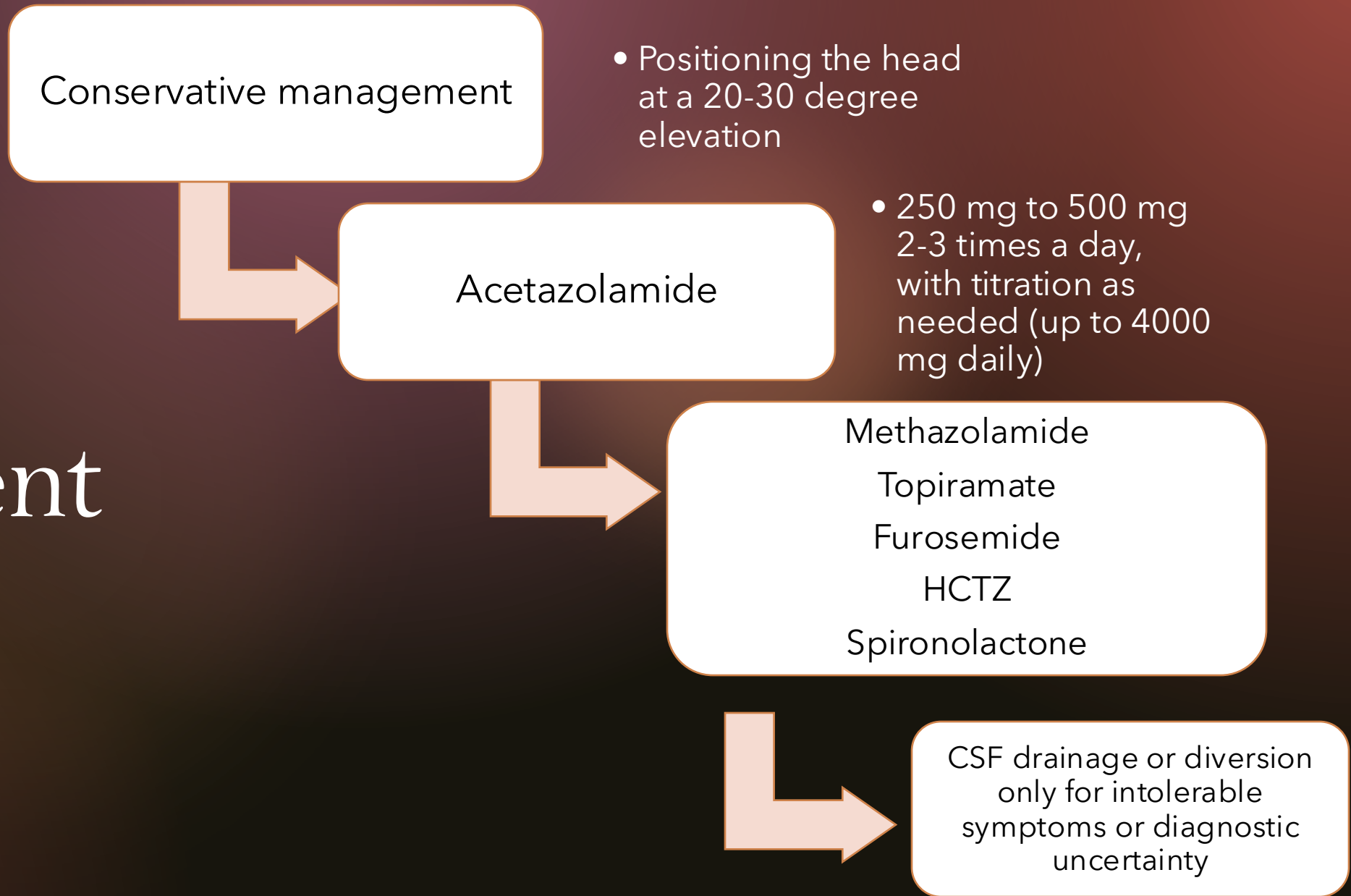
Delay in
symptom
development
(weeks to
months)

SIH presenting
with frontal
pain



Refractory or atypical
presentations of RIH
need workup for other
causes of intracranial
hypertension

Treatment



Treatment Considerations



? Pre-treatment in those with risk factors



Aggressive post-procedural intracranial pressure management of elevated intracranial pressure in patients with IIH-caused cranial CSF leaks has been shown to improve procedural success

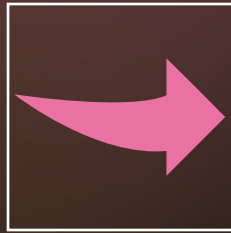


Ferrante et al. found benefit with pre-medication with acetazolamide at the dosage of 250 mg at 18 hours and 6 hours prior to a large-volume untargeted lumbar EBP

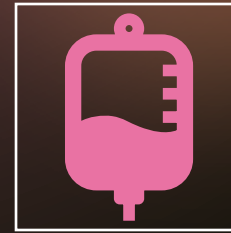
Pathophysiology



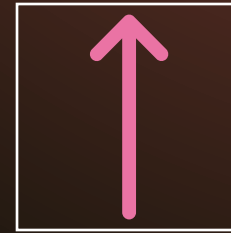
Venous distension



Changes in CSF
reabsorption

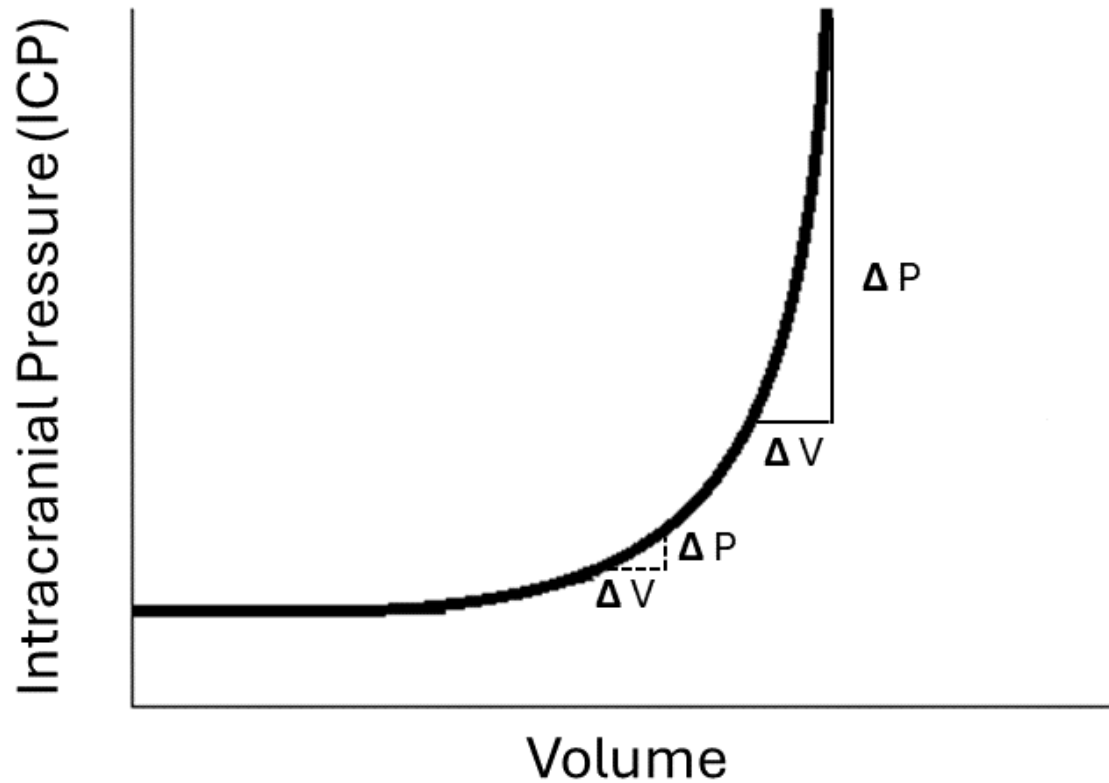


Restriction of cerebral
venous outflow



Underlying IIH

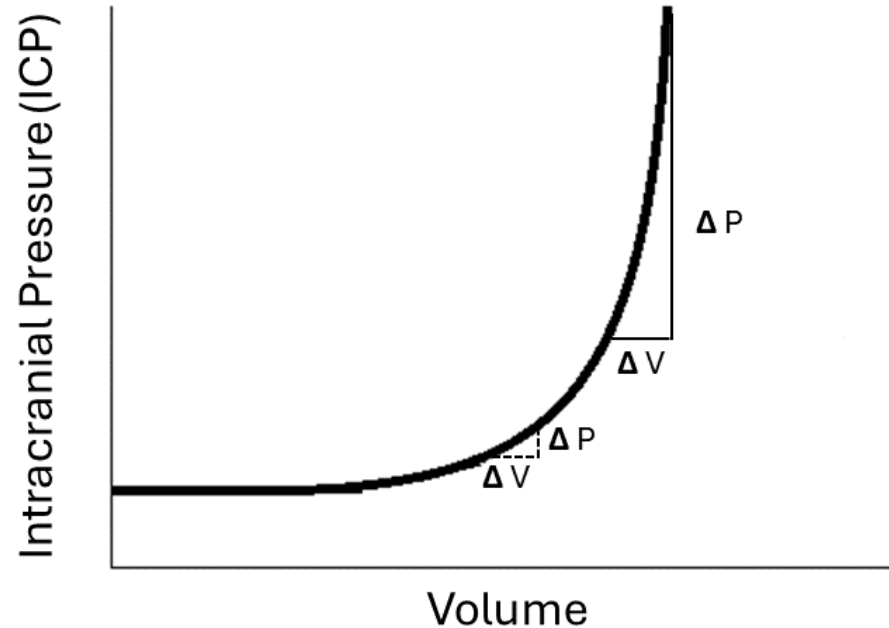
Pathophysiology: Increase in craniospinal elastance?



Elastance measures the pressure response to a known change in volume

Veins as capacitance vessels

- 1) Compensatory increase in venous volume in the spinal epidural macro-veins
- 2) Decrease in transcranial venous outflow



Schievink et al.: $\frac{1}{4}$ of those with focal narrowing in one transverse sinus, and $\frac{1}{2}$ with complete signal gap in one transverse sinus OR any involvement of both transverse sinuses developed RIH

Tsai, Y.-H et al. Noninvasive assessment of intracranial elastance and pressure in spontaneous intracranial hypotension by MRI. J Magn Reson Imaging. 2018

Schievink WI et al. Spinal CSF-venous fistulas in morbidly and super obese patients with spontaneous intracranial hypotension. AJNR Am J Neuroradiol. 2021

Hypothesis: Variability

Reserve of the craniospinal elastance system as a whole

Symptoms occur at the point in which volume shifts resulting from the procedure and/or subsequent healing process and resulting treatment-related fibrosis overwhelm an individual's buffering reserve

Affected by variations in the compliance of the dura itself, which affects CSF outflow resistance

Those with a highly compliant dura and low CSF outflow resistance may not or to a lesser degree experience RIH

Others, who have developed adhesions, scarring, and/or fibrosis, as part of natural or induced dural healing which results in decreased CSF compliance and increased CSF outflow resistance, may be more vulnerable to RIH

Future Research

- Add RIH to the ICHD-4 diagnostic criteria; formal diagnostic code
- Multi-institutional studies assessing the development of RIH at specific time points post-procedure
- Understanding of RIH pathophysiology could help predict response to treatment, risk factors for RIH