First Annual Cedars-Sinai Intracranial Hypotension Symposium - October 14, 2017: Rebound Intracranial Hypertension – with Dr. Peter Kranz, Duke University Medical Center

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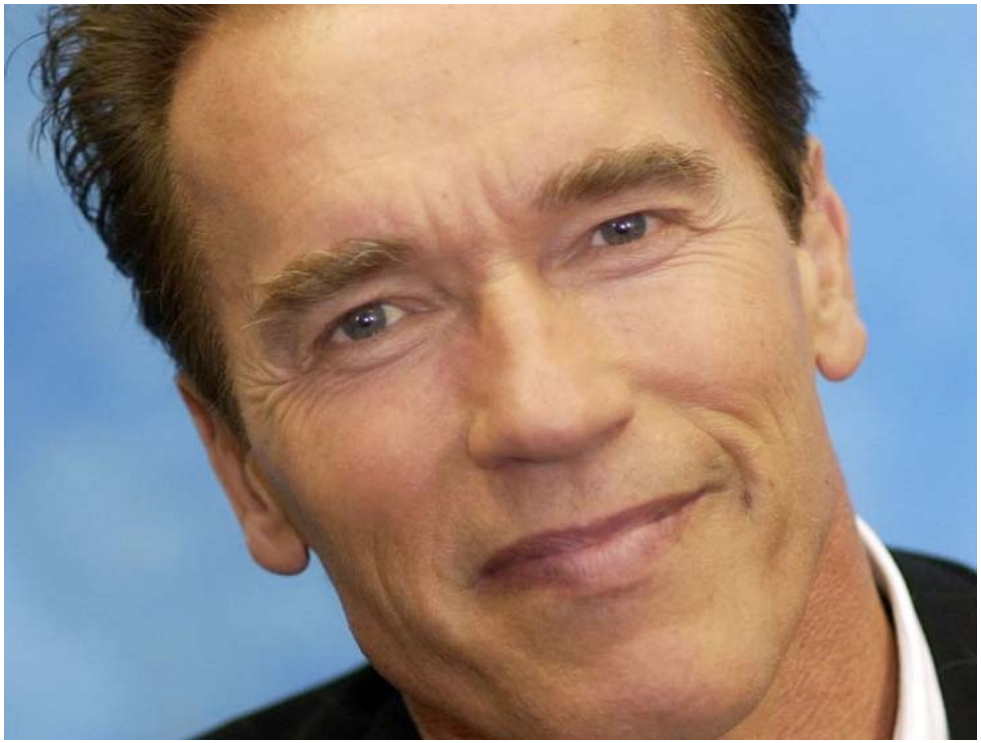
Rebound intracranial hypertension
March 1994



My first patient

John F. DOS: 3-12-1994

41 year-old man

8 year history of orthostatic headaches

MRI brain: brain sagging

CT-myelogram: single lumbar nerve root cyst

Treatment: surgery

Outcome: reverse orthostatic headaches 2nd postop day and visual loss after 6 weeks with papilledema and retinal hemorrhage

:598–605, 1996

Spontaneous spinal cerebrospinal fluid leaks and intracranial hypotension

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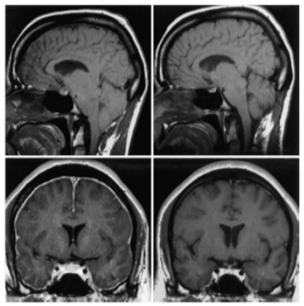


FIG. 4. *Upper Left* and *Right:* Sagittal T_1-weighted magnetic resonance (MR) images revealing caudal displacement of the cerebellum, distortion of the craniomedullary junction, and effacement of the prepontine and perichiasmatic cisterns preoperatively *(upper left)* and significant elevation of the cerebellar tonsils, restoration of the cisterns, and almost complete normalization of the cerebral displacement postoperatively *(upper right)*. *Lower Left* and *Right:* Coronal gadolinium-enhanced T_1-weighted MR images showing intense meningeal enhancement, bowing of the optic chiasm over the pituitary fossa, and reduction of the perichiasmatic cisterns preoperatively *(lower left)* and almost complete resolution of the meningeal enhancement and restoration of the crowding of the optic chiasm postoperatively *(lower right)*.

Case 3. This man initially presented at age 33 years with a 1-year history of progressive, daily occipital headaches. These headaches would invariably go away if he would lie down. A gastroenterological evaluation for associated nausea and vomiting was normal.

Examination. General medical and neurological examinations were normal. Magnetic resonance studies of the head without gadolinium administration revealed that the cerebellar tonsils extended to the level of C-1, but the position of the fourth ventricle was normal. Several lumbar punctures and CSF studies were performed with opening pressures between 0 and 14 cm H_2O, cell counts between 9 and 21/μl, and total protein between 48 and 54 mg/dl. The patient was treated with corticosteroids but showed no improvement. Because of the patient's persistent headaches a decompression of the presumed Chiari I malformation was performed.

The patient recovered well from the surgery and experienced a gradual improvement of the headaches for approximately 4 years; although they never resolved, he was able to lead a relatively normal lifestyle. However, the positional headaches worsened again over the ensuing 3 years. The headaches had been associated with nausea and vomiting, but now the patient also developed hiccups, dizziness, and bowel and bladder incontinence. Neurological examination showed generalized hyperreflexia. Magnetic resonance imaging revealed diffuse meningeal enhancement, displacement of the cerebellar tonsils to the level of C-2, distortion of the craniomedullary junction, and generalized "sagging" of the brain (Fig. 4). A lumbar puncture made with the patient in the lateral decubitus position showed an opening pressure of 6 cm H_2O, total protein of 60 mg/dl, and four nucleated cells/μl. Indium-111 radionucleotide cisternography displayed minimal migration of tracer over the cerebral convexities and an abnormal uptake of tracer in the upper lumbar spine region on the left (Fig. 5). Computerized tomographic myelography was suggestive of a meningeal diverticulum in the left L-2 neural foramen with remodeling of the L-2 pedicle, indicating a long-standing process (Fig. 6 *upper*). In retrospect, an abdominal CT scan performed 8 years previously for the evaluation of associated nausea and vomiting showed the presence of this diverticulum (Fig. 6 *lower*).

Operation. A left L-2 hemilaminectomy and total facetectomy were performed; extradural CSF was encountered immediately after removal of the ligamentum flavum. A meningeal diverticulum with multiple outpouchings was found enveloping the L-2 nerve root. There was significant bleeding from Batson's plexus around the diverticulum, which had eroded the L-2 pedicle. The diverticulum was ligated circumferentially with several sutures without compromising the nerve root. There was no evidence of ongoing CSF leakage.

Postoperative Course. The patient recovered well from surgery with complete resolution of all his symptoms. Leg strength and sensation remained normal. One week after surgery, he noted occasional mild visual blurring. Six weeks postoperatively, examination showed bilateral papilledema with a hemorrhage inferior to the optic disc on the left. The visual symptoms resolved spontaneously and 4 months later the papilledema improved considerably and the hemorrhage resolved. Magnetic resonance imaging of the head revealed resolution of the meningeal enhancement, elevation of the cerebellar tonsils, and marked improvement of the cerebral displacement (Fig. 4).

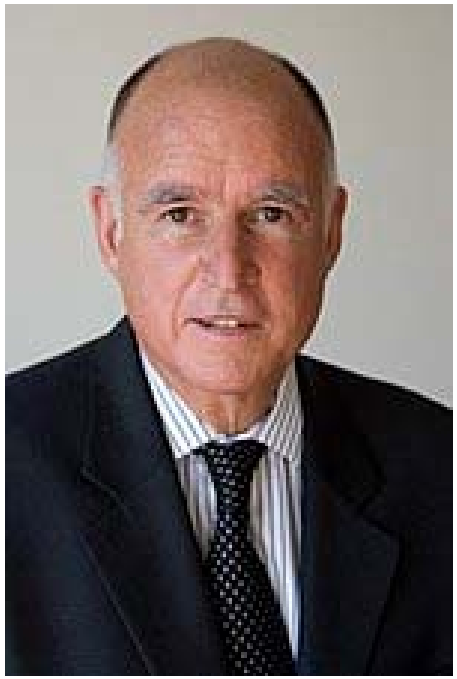
We postulate that the development of papilledema in this patient may have been due to the sudden interruption of the abnormal pathway of CSF resorption, which had been present for many years.

The syndrome of spontaneous intracranial hypotension is characterized by a postural headache that may be associated with a variety of symptoms including posterior neck pain or stiffness, nausea, vomiting, diplopia, visual blurring, tinnitus, vertigo, and local back pain.[2,4,16–18,23,38,39,43,49] The diagnosis is confirmed by lumbar puncture, which reveals a low CSF pressure. However, variable readings of CSF pressure may be obtained over time, possibly indicating that the CSF leak is intermittent. Examination of the CSF itself often shows mild elevations of total protein and increased cell count.

Characteristically, cranial MR imaging studies in pa-

Rebound intracranial hypertension
October 2017

- Routinely diagnosed?



Lack of knowledge of rebound high-pressure headaches

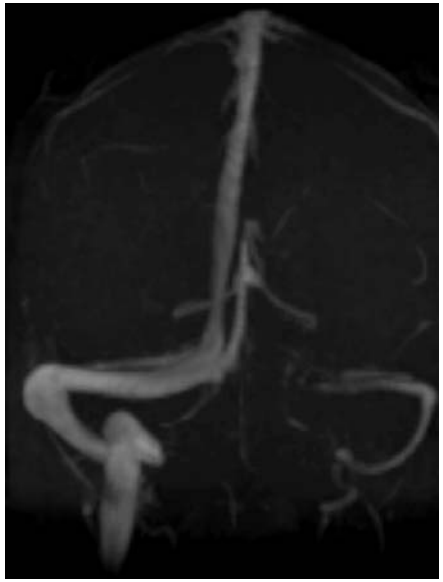
- Frequency: 0-20%
- Treatment: Acetazolamide and ….
- No diagnostic criteria

Rebound high-pressure headache after treatment of spontaneous intracranial hypotension. An MRV study

- 113 consecutive patients with SIH
- Diagnostic criteria for rebound headache
  - a: reverse orthostatic headache
  - b: resolution of headache after Diamox
  - c: not better accounted for by another cause of headache
- MRV scores according to Higgins et al (JNNP 2004)
  - 0: normal
  - 1: one or more areas of focal narrowing
  - 2: one or more signal gaps

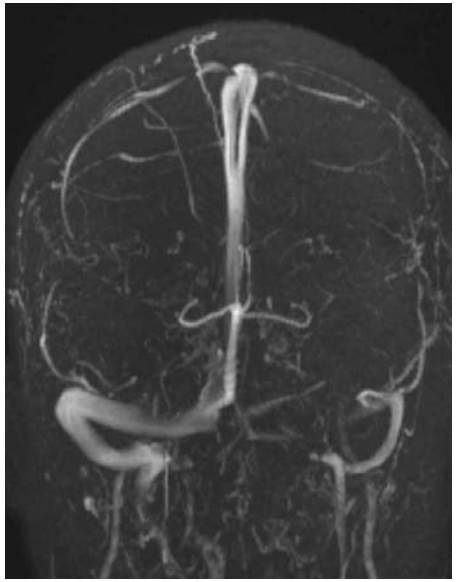
MRV score: 1

1/0



MRV score: 2

0/2



MRV score: 3

1/2

Rebound high-pressure headache after treatment of spontaneous intracranial hypotension. An MRV study

67 women / 46 men
Age: 45.9 (range, 13 – 71 years)

Rebound high-pressure headache: 31 (27.4%)

More common in
a) women (p=0.0474)
b) younger age (p=0.0135)
c) presence of extradural CSF (p=0.0286)

Rebound high-pressure headache after treatment of spontaneous intracranial hypotension. An MRV study

Frequency of rebound high-pressure headache

MRV score	0 (n=42):	14%
	1 (n=34):	24%
	2 (n=34):	44%
	3 (n=3) :	67%
	4 (n=0)	

P=0.0092

Conclusions

- Rebound high-pressure headache is common after treatment for SIH (about one-fourth)

- Related to: Age, sex, presence of extra-dural CSF on spinal imaging, and venous anatomy on MRV

Rebound Intracranial Hypertension

Peter G. Kranz, MD
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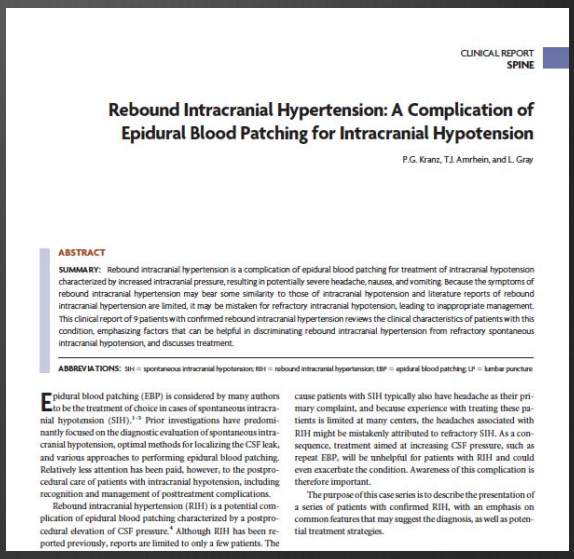
Disclosures

1. No conflict of interest
2. Use of fibrin glue for epidural injection is off label

RIH: What is it?

- New headache type after blood patching
- Characterized by:
  - *HA worse when lying down*
  - *Often change in location – frontal*
  - *Nausea, blurry vision common*
- Increased CSF pressure compared with baseline
  - *May or may not be > 20 cm H_2O*
- Onset immediately after up to days after
  - *Usually worst 24-36 hrs post-patch*



CLINICAL REPORT
SPINE

Rebound Intracranial Hypertension: A Complication of Epidural Blood Patching for Intracranial Hypotension

P.G. Kranz, T.J. Amrhein, and L. Gray

ABSTRACT

SUMMARY: Rebound intracranial hypertension is a complication of epidural blood patching for treatment of intracranial hypotension characterized by increased intracranial pressure, resulting in potentially severe headache, nausea, and vomiting. Because the symptoms of rebound intracranial hypertension may bear some similarity to those of intracranial hypotension and literature reports of rebound intracranial hypertension are limited, it may be mistaken for refractory intracranial hypotension, leading to inappropriate management. This clinical report of 9 patients with confirmed rebound intracranial hypertension reviews the clinical characteristics of patients with this condition, emphasizing factors that can be helpful in discriminating rebound intracranial hypertension from refractory spontaneous intracranial hypotension, and discusses treatment.

ABBREVIATIONS: SIH = spontaneous intracranial hypotension; RIH = rebound intracranial hypertension; EBP = epidural blood patching; LP = lumbar puncture

Epidural blood patching (EBP) is considered by many authors to be the treatment of choice in cases of spontaneous intracranial hypotension (SIH).[1-3] Prior investigations have predominantly focused on the diagnostic evaluation of spontaneous intracranial hypotension, optimal methods for localizing the CSF leak, and various approaches to performing epidural blood patching. Relatively less attention has been paid, however, to the postprocedural care of patients with intracranial hypotension, including recognition and management of posttreatment complications.

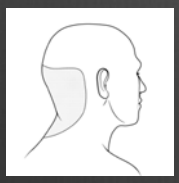
Rebound intracranial hypertension (RIH) is a potential complication of epidural blood patching characterized by a postprocedural elevation of CSF pressure.[4] Although RIH has been reported previously, reports are limited to only a few patients. The cause patients with SIH typically also have headache as their primary complaint, and because experience with treating these patients is limited at many centers, the headaches associated with RIH might be mistakenly attributed to refractory SIH. As a consequence, treatment aimed at increasing CSF pressure, such as repeat EBP, will be unhelpful for patients with RIH and could even exacerbate the condition. Awareness of this complication is therefore important.

The purpose of this case series is to describe the presentation of a series of patients with confirmed RIH, with an emphasis on common features that may suggest the diagnosis, as well as potential treatment strategies.

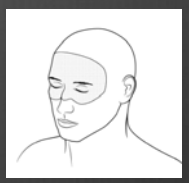
Kranz et al. AJNR 2014. Jun;35(6): 1237-40.

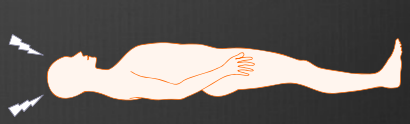
RIH: What is it?

SIH

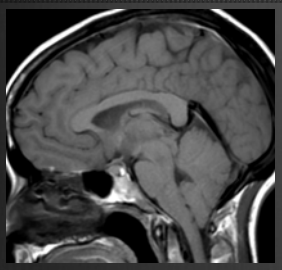


RIH
Rebound Intracranial Hypertension

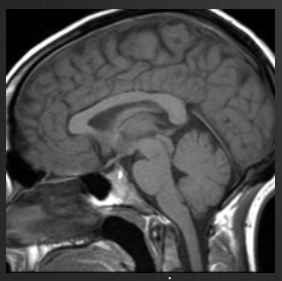




RIH: Examples



Brain MRI = SIH



3 years prior

Myelo = leak

Patch

Initial pressure: **7 cm H_2O**

Overnight developed new sx
- non-positional HA
- Worse with lying down
- N/V, blurred vision

Lumbar puncture (+24 hr)
- Pressure: **28 cm H_2O**
- Drained to 9 cm H_2O

Lumbar puncture (+48 hrs)
- Pressure: **29 cm H_2O**
- Drained to 7 cm H_2O

RIH: Examples

Pre- and post-EBP characteristics of patients with RIH

Patient	Opening Pressure (cm H_2O)		Time from EBP to Repeat LP	Headache Location		Post-EBP N/V[a]	Post-EBP Blurred Vision	Patching Agent	Patch Volume	Duration of Acetazolamide Treatment
	Pre-EBP	Post-EBP		Pre-EBP	Post-EBP					
1	6.8	25.5	6 hours	Occipital	Frontal	Yes	Yes	Fibrin glue	6 mL	5 days
2	N/A	28.0	3 weeks	Retro-orbital	Peri-orbital	No	No	Blood	18 mL	–[b]
3	12.3	31.0	2 hours	Retro-orbital	Retro-orbital	Yes	No	Blood + fibrin glue	16 mL	5 days
4	7.8	31.0	24 hours	Occipital	Holocephalic	Yes	Yes	Blood	18 mL	6 weeks
5	1.0	>55.0	2 hours	Holocephalic	Holocephalic	Yes	Yes	Blood	6 mL	2 weeks
6	15.0	32.0	7 days	Occipital	Frontal	Yes	Yes	Blood	5 mL	3+ years
7	10.8	24.2	24 hours	Occipital	Frontal and peri-orbital	Yes	Yes	Blood	18 mL	4 weeks
8	7.0	22.0	48 hours	Occipital	Frontal	Yes	Yes	Blood	20 mL	6 weeks
9	8.0	22.0	12 months	Occipital	Occipital	Yes	Yes	Blood	42 mL	6 months

Note:— N/A indicates data not available.
[a]Nausea/vomiting.
[b]Received follow-up care at another institution.

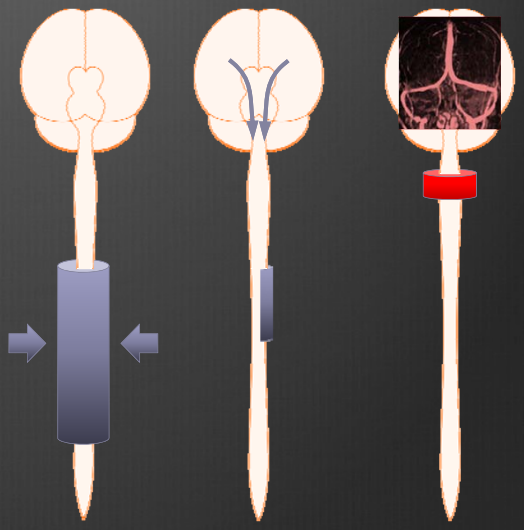
Kranz et al. AJNR 2014. Jun;35(6): 1237-40.

RIH: Why does it happen?

We don't know for sure

Possible mechanisms:

- *"Squeeze" on the sac, displaces CSF*
- *Compensatory ↑ CSF production*
- *Reduced compliance – venous dilation*



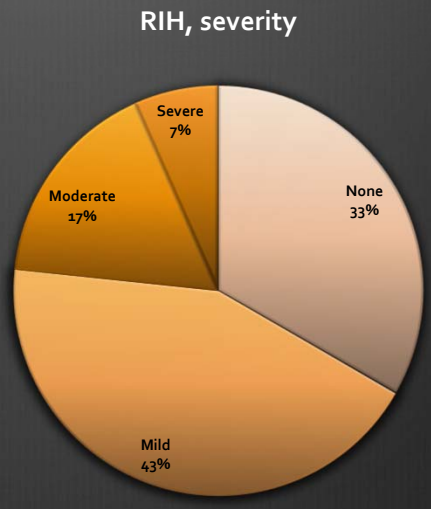
RIH: What is the prevalence?

30 consecutive patients

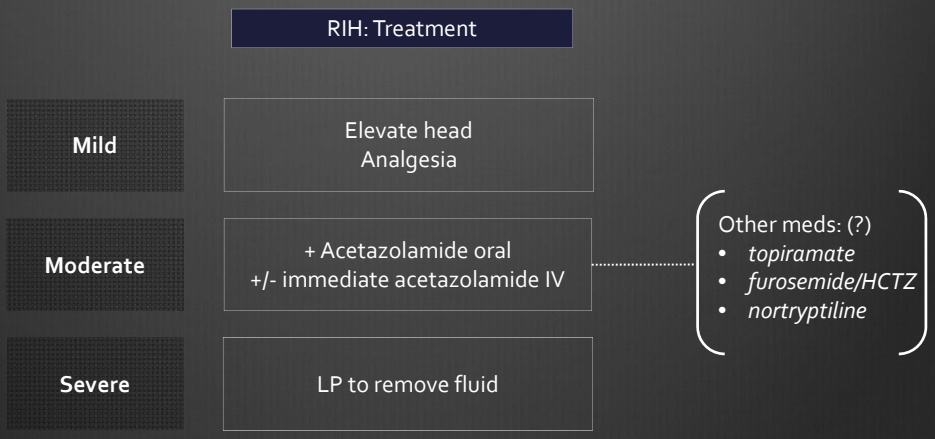
Day +1 after epidural blood patch

Graded RIH:

- 0 = none
- 1 = mild
- 2 = moderate
- 3 = severe



RIH: How to treat



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