Changes In Quantitative Pupillary Reactivity During Diagnosis And Treatment Of Symptomatic Intracranial Hypotension

Mayne E MD PhD, Hirsch K MD, Mlynash M MD, Hobbs K MD, Venkatasubramanian C. MD, MSc.
Department of Neurology & Neurological Sciences, Stanford University
Correspondence: chitrav@stanford.edu

INTRODUCTION

- Pupillary light response (PLR) evaluates cranial nerves II, III and midbrain function.
- Quantitative infrared pupillometry objectively measures PLR as NPI (neurological pupillary index).
- Increases in ICP decrease NPI.
- We hypothesized that similar changes in NPI could be seen with intracranial hypotension (IH) because of midbrain distortion, despite low intracranial pressures.
- We determined the sequential changes in NPI in IH before and after treatment.

METHODS

- We identified four patients who were monitored with pupillometry for clinical care at the time of IH diagnosis and treatment.
- Quantitative pupillometry was performed with the NPi-200 Pupillometer (Neuroptics, Irvine, CA)
- IH was diagnosed with a compatible history, exam and characteristic neuroimaging findings. (Fig. 1)
- Patients' NPI at baseline, during symptomatic IH and after treatment were compared using non-parametric statistical methods including related samples Friedman's two-way ANOVA by ranks test.

RESULTS

Figure 1: Sagittal MRI of patient # 3.
Pre-treatment MRI (left) shows flattening of ventral pons and prominent dural venous sinuses. After repair of skull fracture (right), the pons shape is restored and dural sinuses are less prominent.

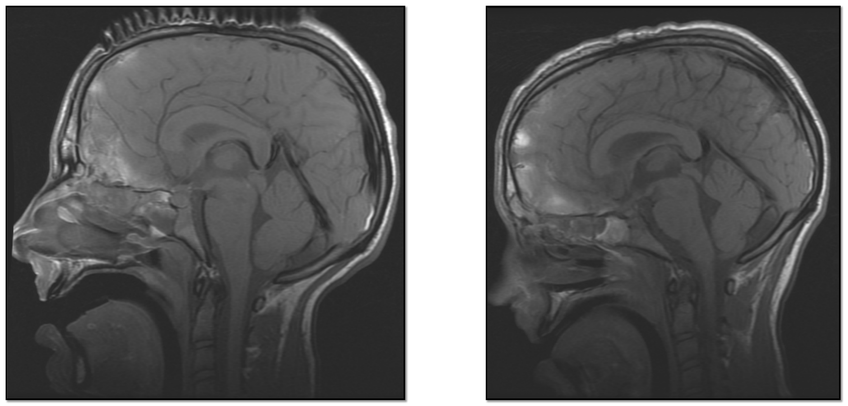
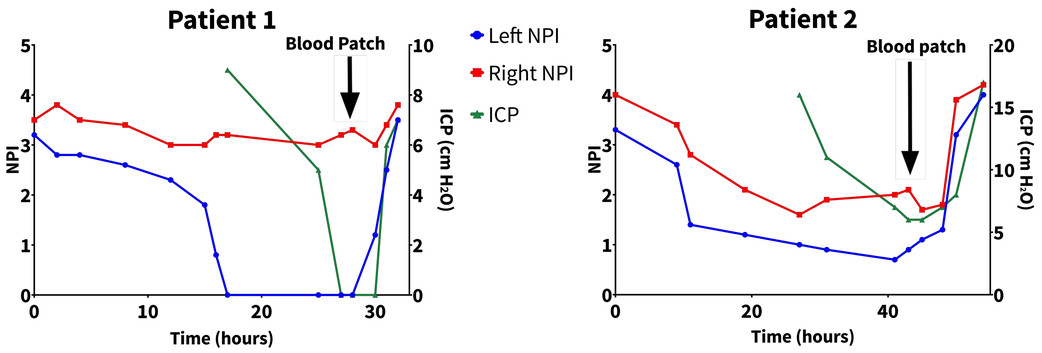


Figure 2: Pupillometry and ICP during symptomatic IH and after treatment



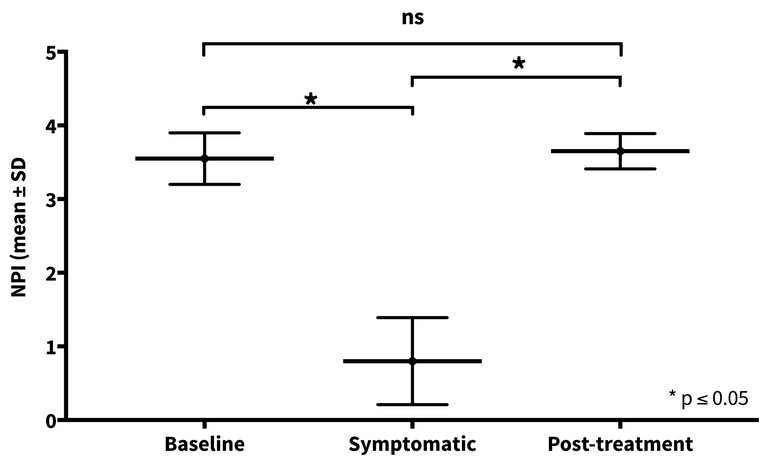
Patient 1: Iatrogenic IH from lumbar drain
49 F w/ L olfactory groove meningioma. Lumbar drain placed during resection, removed post-op. Post-op somnolence 2/2 L frontal extradural hematoma, requiring evacuation and EVD. S/p EVD, brief improvement followed by neurological deterioration temporally associated with downtrending left NPI and ICP with minimal CSF drainage. IH ascribed to CSF leak from lumbar drain site. Trendelenberg, EVD clamping, and blood patch normalized exam and ICP.

Patient 2: Iatrogenic IH from lumbar drain.
47 F with left olfactory neuroblastoma excised via trans-sphenoidal approach with intra-operative lumbar drain, removed post-op. Progressive somnolence and bilateral extensor posturing with worsening NPI's (L > R) and declining ICP, neuroimaging suggestive of IH. Exam, NPI, ICP and imaging improved after blood patch.

Patient 3: Traumatic IH leak due to skull fracture.
22 M w/ basal skull fracture and CSF leak from right ear. Had R decompressive hemicraniectomy for refractory ICP. S/p hemi-crani, had b/l extensor posturing and progressive loss of upper brainstem reflexes, w/ worsening NPI's (R > L) despite "improved" ICPs. Brainstem reflexes regained when laid flat and IH from CSF leak suspected. Brainstem reflexes, NPI and ICP normalized with repair of skull fracture.

Patient 4: Iatrogenic IH from lumbar drain
32 M, HH 2, Fisher 3 aSAH s/p clipping w/ difficult intraop lumbar drain placement, removed post-op. Post-op day 12, had progressive obtundation despite successful intra-arterial treatment of vasospasm and normal TCD's. NPI's (R > L) worsening w/ radiographic evidence of IH. Blood patch normalized exam and NPI's.

Figure 3: Mean NPI at baseline, during symptomatic IH, and after treatment



Mean baseline NPI was normal (defined as >3) and declined in one or both eyes concurrent with clinical deterioration in the 24-48 hours preceding definitive diagnosis. All patients underwent treatment for CSF leak with epidural blood patch or fracture repair, with return of NPI > 3 within 5 hours of treatment. The baseline, symptomatic and post treatment NPI's differed significantly (3.55±0.35 vs 0.80±0.59 vs 3.65±0.24, mean +/- SD, pre-treatment vs nadir vs post-treatment, p=0.05). Both baseline and post treatment NPI's differed from the NPI nadir (p=0.068) but there was no significant difference between baseline and post-treatment NPI (p = 0.71).

CONCLUSIONS

- Impairment of the pupillary light reactivity as measured by NPI occurs early during IH and precedes clinical and ICP worsening with brisk improvement after treatment.
- PLR impairment is attributable to stretching of the 3rd cranial nerve +/- midbrain distortion.
- Management of intracranial hyper- and hypotension differ markedly. Our results emphasize the importance of evaluating the clinical context before attributing pupillary/ NPI changes to increased ICP.
- Automated pupillometry provides a non-invasive, bedside tool for monitoring progression and treatment of intracranial hypotension.

The authors have no disclosures.