



Blink reflex in idiopathic intracranial hypertension patients

Hanan Hosny^a, Mohamed Mabrouk^b, Dalia Gamal^c, Rehab Elanwar^c

^a Department of Clinical Neurophysiology, Beni-Suef University, Cairo, Egypt

^b Department of Neurology Beni-Suef University, Beni-Suef, Egypt

^c Department of Clinical Neurophysiology, Beni-Suef University, Beni-Suef, Egypt

Abstract:

The goal of our study was to clarify the possible effect of increased intracranial pressure on electrophysiological blink reflex in idiopathic intracranial hypertension (IIH) patients. Subjects &

Methods: 40 females; 20 IIH patients and 20 age matched controls, after thorough neurological examination, were subjected to electrically elicited blink reflex (BR). **Results:** There was a significant delay in ipsilateral R2 (R2i) and contralateral R2 (R2c) latencies with 31.6 (± 2.8) msec and 35.2 (± 3.3) msec among cases when compared to controls with 29 (± 2.6) msec and 32.2 (± 2.8) msec respectively with (P-value < 0.001), while R1 latencies were 10.4 (± 1) msec in cases and 10.5 (± 0.6) in controls with no significant difference between the two groups (p-value = 0.726)

Conclusion: the increased intracranial pressure was proved to increase R2i and R2c latencies of blink reflex studies which may suggest subclinical facial nerve dysfunction in IIH patients whether through peripheral nerve compression or at the level of the reflex centers (lateral medulla) in IIH patients.

Keywords: Benign intracranial hypertension, Brain Stem, Blink Reflex, Cerebrospinal Fluid.

1. Introduction:

Idiopathic intracranial hypertension (IIH) is a disorder of elevated cerebrospinal fluid pressure more than 200 mmH₂O, in the non-obese and 250 mmH₂O in the obese patients with normal cerebrospinal fluid (CSF) composition [1].

Symptoms and signs of IIH are related to raised CSF pressure. In most patients, symptoms worsen slowly [2].

The major complications from IIH arise from untreated or treatment-resistant papilloedema. In various case series, the long-term risk of

one's vision being significantly affected by IHH is reported to lie anywhere between 10 and 25% [3].

The common thread here is that the cranial nerves that make nearly a 90° bend (CN II, VI, VII) appear to be susceptible to damage at the site of the bend. The diagnosis of IHH should be viewed with suspicion in patients with ocular motility disturbances other than sixth nerve palsies [4].

Blink reflex (BR) study is useful in detecting abnormalities anywhere along the reflex arc. Impulses are conveyed through polysynaptic medullary pathways both ipsilaterally and contralaterally to the stimulated side of the face, before connecting to the facial nuclei. The crossing takes place in the caudal medulla [5].

2. Aim of work:

Clarifying the effect of increased intracranial pressure on blink reflex in idiopathic intracranial hypertension (IHH) .

3. Patients and Methods:

This was a case control study performed in Beni-Suef university hospital on 40 women (20 cases and 20 matched age healthy controls) after the approval of the Research Committee and the Ethical Committee of the Faculty of Medicine, Beni-Suef University.

3.1 Inclusion criteria:

1. Age: from 18 to 60 years.
2. CT/MRI is normal
3. CSF cytology is normal.

3.2. Exclusion criteria:

1. History of systemic and medical condition considered to affect nervous system e.g. diabetes mellitus, hepatic or renal failure.
2. History of facial nerve palsy, stroke, head trauma or brain tumors.

3.3. All patients were subjected to:

- Thorough general and neurological examination.
- Routine laboratory work-up including complete blood count, fasting blood sugar, liver function and renal function tests.
- Fundus Examination.
- Blink reflex studies were carried out for all study participants using Nihon Kohden machine (Neuropack X1, EMG/EP MEB-2300, Japan).

Recording were performed simultaneously from both sides through active recording electrodes placed on the midpoint of both lower eye lids, the reference electrodes 2-3

cm lateral to the active electrodes and ground electrode in the forehead.

Electrical Stimulation was performed on each side separately with duration of 0.1 msec placed in the superior orbital fissure.

Filters were set to 10 Hz for the high cut filter, 5 KHz for the low cut filter and the notch filter was set at 50 Hz [6].

Statistical methodology

SPSS v. 25 (Statistical Package for Social science) for Windows was used for analysis the mean, standard deviation (SD), median, minimum and maximum.

- Non-significant result when P-value > 0.05.
- Significant results when P-value ≤ 0.05.

4. Results:

• Demographic Data:

This study was conducted on 20 patients and 20 healthy controls. The patient group included 20 IIH female patients and the mean age was 35.4±10.5 years. The control group included 20 female participants matched with the patients in age with mean value 33.8±8.9.

• Clinical Data:

The mean disease duration was 17.3±22.4 weeks. The mean opening pressure was 29.4±24.6 mmH₂O. Error! Not a valid bookmark self-reference.

• Electrophysiological Data

Both patient and healthy controls were subjected to blink reflex studies. The following parameters were taken into consideration in our research R1, R2i and R2c latencies (msec).

The mean values of the blink reflex parameters under study in controls and cases are illustrated in **Table 1**.

Table 1: Blink reflex parameters in cases and control

Items (mean±SD)	Cases (msec)	Controls (msec)	P -value
R1	10.4±1	10.5±0.6	0.726
R2i	31.6±2.8	29±2.6	<0.001*
R2c	35.2±3.3	32.2±2.8	<0.001*

Table (1) showed that was a significant increase in of R2i and R2c latencies among cases than controls with (P-value<0.001).

5. Discussion:

This study was based on that IIH can be associated with single or multiple cranial nerve (CN) palsies, with 39–59% of the patients having some sort of CNs deficit [7]. Although optic and abducent nerves abnormalities are the most commonly reported in IIH, other cranial nerves involvement has been also reported [8]. As cranial nerves (CN) V and VII share in the pathway of the corneal (blink) reflex, so our work was designed to study the findings of blink reflex in patients with increased intracranial pressure compared to normal control group [9].

In our study, there was a significant delay in of R2i and R2c latencies with 31.6 (± 2.8) msec and 35.2 (± 3.3) msec among cases (**figure 1**) comparing to controls with 29 (± 2.6) msec and 32.2 (± 2.8) msec respectively with (P-value <0.001).

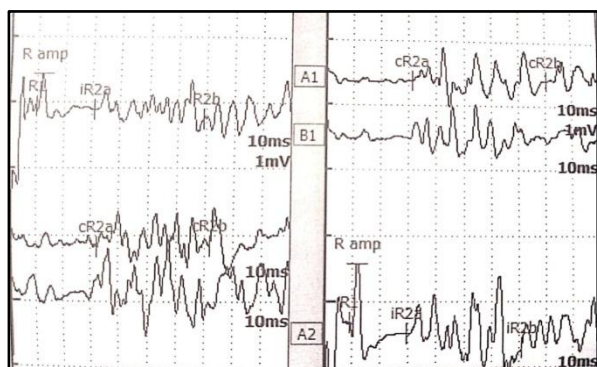


Figure (1) blink reflex in IIH patient

This result may be explained by facial nerve dysfunction; however, it is atypical clinical presentation of IIH [10]. Few confirmed case reports of facial nerve abnormalities in association with IIH have been recorded over years [11].

Although the relationship between facial nerve dysfunction and raised ICP is questionable, theories included a possible compression of the intratemporal segment of the facial nerve by the congested venous plexus accompanying it [12].

On the other hand, some studies reported increased facial nerve excitability and hemifacial spasm*. This was, in contrary to our work, supported by **samanci, 2018** who found that the ipsilateral and contralateral R2

latencies were significantly lower in cases than controls [13].

6. Conclusion:

The increased intracranial pressure resulted in increase R2i and R2c of BR which may suggest subclinical facial nerve dysfunction in IIH patients.

7. References:

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