

Ergebnis: Es bestand eine metabolische (Laktat-)Azidose mit Hypokaliämie. Beidseits lag der Visus bei HBW, die Pupillen waren lichtstarr bei sonst normalen vorderen Augenabschnitten. Funduskopisch zeigte sich ein Papillenödem. Zehn Tage nach Aufnahme betrug der Visus 0,8. Die Pupillen waren lichtreagibel, die Papillen weiterhin unscharf begrenzt, das Gesichtsfeld zeigte unspezifische Defekte, der Farbttest nach Ishihara war unauffällig. Das OCT zeigte eine normale foveale Senke mit normaler Ganglienzellenschicht, allerdings eine, im Verlauf rückläufige Verdickung der peripapillären Nervenfaserschicht.

Schlussfolgerung: Trotz ungünstiger Prognose erreichte der Patient ein gutes Sehvermögen. Um die Mortalitätsrate und das Risiko persistierender Sehstörungen zu reduzieren, soll bei Verdacht auf eine Methanolvergiftung sofort Ethanol verabreicht werden. Alternativ (nicht additiv) ist eine Hemmung der ADH mit Fomepizol oder 4-Methylpyrazol möglich.

Angaben zu potentiellen Interessenkonflikten: Irvandi Irvandi

Honorarleistungen: Nein

Arbeitsverhältnis: Nein

Fördermittel: Nein

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PDo09-04

Elucidating the origin of P100 double peaks in visual evoked potentials of patients with optic neuritis

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Research question: We investigated the origin of P100 double peaks in the visual evoked potential (VEP), seen in a subset of patients with optic neuritis and hypothesized to form because of a partial conduction delay in the optic nerve. We simulated the assumed differential signal timing in healthy participants by presenting asynchronous half-field stimuli.

Methodology: We recorded monocular transient checkerboard reversal VEPs in 20 healthy participants. The stimuli were vertically split to allow for independent manipulation of presentation time of the temporal and nasal half of the stimuli. Full-field VEPs with synchronous and asynchronous (24 ms, 35 ms, 47 ms) half-field presentation as well as temporal and nasal half-field VEPs were recorded. The data were analyzed by using half-field responses to predict full-field recordings with asynchronies, and compare these to the vertical full-field VEPs recorded under the respective stimulation conditions. Both were assessed qualitatively and quantitatively for agreement and for effects of stimulus manipulations.

Results: Double peaks formed consistently with $\geq 80\%$ of traces manifesting the atypical waveform when temporal half-field presentation was delayed by 35 ms and 47 ms. Analysis of the full-field VEPs demonstrated a gradual wave shape shift from broadened and amplitude-reduced single P100 peaks to distinct double peaks with increasing delays. Time between the first and second P100 peak closely mirrored the induced delay time. The full-field VEP and the respective half-field VEPs demonstrated the second P100 peak of the full-field stimulation aligning with the P100 peak of the time-shifted response to temporal half-field stimulation.

Conclusion: Our findings further substantiate the hypothesis of P100 double peaks originating in variations in signal timing, consistent with the notion of partial conduction delays in the optic nerve. In patients with optic neuritis, it may indicate underlying differential de- or remyelination stages within the optic nerve.

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The functional state of the visual analyzer according to electrophysiological indicators in non-arteritic ischemic optic neuropathy

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Non-arteritic ischemic optic neuropathy (NAION) is the second leading cause of irreversible vision loss after glaucoma in individuals.

Was to assess changes in electrophysiological parameters of the visual system in patients with NAION using pattern visual evoked potentials (*P*-VEP) and pattern electroretinography (*P*-ERG).

21 Patients (31 eyes) with NAION were examined. The mean age was 47.5 ± 11.6 years (range: 37–66 years). All patients underwent standard ophthalmic examination. Based on the presence of a central scotoma, patients were divided into two groups: group 1 (8 patients—8 eyes) with central scotoma due to axial bundle damage, and group 2 (3 patients (3 eyes) were with unilateral NAION and 10 patients (20 eyes)—with bilateral NAION) without central scotoma. Electrophysiological studies were conducted by the RetiScan system (Roland Consult, Germany) in accordance with ISCEV standards. *P*-VEPs were recorded using 1° and 15' checkerboard patterns and *P*-ERG by 48' checkerboard pattern. Statistical analysis was performed using IBM SPSS Statistics with significance $p < 0.05$.

Visual acuity in the group 1 was 0.15 ± 0.1 , 2-d group— 0.62 ± 0.3 . No significant differences were found in *P*-VEP latencies (N75 and P100) between the groups. The *P*-VEP P100 amplitude to the 1° stimulus was 9.0 ± 3.3 μ V in group 1 and 13.1 ± 5.3 μ V in group 2 (1.45 times higher, $p = 0.04$). The *P*-VEP P100 amplitude to the 15' stimulus was 8.9 ± 2.7 μ V in group 1 and 13.1 ± 5.3 μ V in group 2 (1.45 times higher, $p = 0.02$). The factor of the axial bundle damage was significant for the 15' VEP amplitude— $F = 4.23$, $p = 0.048$. There was a direct correlation $r = 0.44$ ($p < 0.05$) between the visual acuity and the 15' VEP amplitude.

The bioelectrical activity of ganglion cells of central retina was assessed using *P*-ERG. Latency values (N35, P50, N95) did not differ significantly between groups. The N35-P50 amplitude was 3.5 ± 1.3 μ V in group 1 and 4.5 ± 1.6 μ V in group 2 (1.28 times higher, $p = 0.05$). The P50-N95 amplitude was 4.2 ± 1.4 μ V in group 1 and 5.7 ± 2.4 μ V in group 2, but was not different ($p = 0.09$). These results suggest reduced functional activity of ganglion cells in the central retina region.

Electrophysiological changes in NAION are heterogeneous and depend on the location of damage. In patients with axial bundle depression, *P*-VEP amplitudes were reduced by 1.5 times and *P*-ERG amplitudes by 1.2 times. Latency parameters remained stable.

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