Literature review

https://doi.org/10.31288/oftalmolzh202556570

Visual evoked potentials in the diagnosis of optic neuropathies: a literature review

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This review analyzes the utility of visual evoked potentials (VEP) as a method for visual function assessment in optic neuropathies. We systematically reviewed the existing data on the application of several types of VEP (pattern-reversal, pattern-onset/offset, flash-pattern, and chromatic) for objective assessment of the visual pathway in inflammatory, demyelinating, ischemic, toxic, compressive and traumatic optic neuropathies. The review presents the typical changes in VEP parameters (latency, amplitude and morphology) for each of the pathologies considered, and their relationships with clinical manifestations and results of other neuroophthalmological examination techniques such as perimetry and optical coherence tomography. The paper highlights the diagnostic and differential diagnostic value of VEP, especially in challenging cases and for providing an objective approach for characterizing visual function deficiencies.

Key words:

optic neuropathy, visual evoked potentials, electroretinography

Introduction

Optic neuropathies are optic nerve disorders resulting from various pathological processes. The diagnosis is made clinically. Careful anamnesis often points to a possible origin of the optic neuropathy: an acute onset is characteristic of the demyelinating, inflammatory, ischemic or origin traumatic origin, whereas a slow onset is characteristic of compressive, toxic/nutritional or genetic origin.

Typical clinical manifestations include acute or gradual visual loss, visual field defects, abnormal color perception, and relative afferent pupillary defect.

Major signs of the most common types of optic neuropathies

Insufficient blood supply (ischemia) of the optic nerve is observed in ischemic optic neuropathy (ION). ION is primarily of two types: anterior ION and posterior ION, involving the optic nerve head (ONH) and the rest of the optic nerve respectively [1]. Additionally, ION can be classified on the basis of pathogenesis into arteritic (AION – due to giant cell arteritis) and non-arteritic (NAION – due to causes other than giant cell arteritis). AION occurs predominantly in elder patients over age 50, causing a severe vision loss (with a visual acuity of 20/200 or worse) with a residual visual field defect. Moreover, patients with AION usually have symptoms such as appetite loss, malaise, headache, scalp tenderness and tender temporal arteries, jaw pain on mastication (jaw claudication), and generalized muscle or joint aches [2, 3]. NAION is the most common cause of optic neuropathy in adults older than 50. The prevalence of NAION in the United States has been estimated to be anywhere between 2.3 and 10.2 per 100,000 [4], but no data is available regarding the prevalence of NAION in Ukraine. NAION vision loss may be acute, subacute, or stepwise and is not usually associated with pain. Vision loss is less severe than in anterior AION, as about half of patients have a 20/60 visual acuity examination result or better, and visual defects, commonly in the inferior half of the visual field. Fundus examination in the acute stage often exhibits diffuse or segmental optic disc edema on the affected side, predominantly in the superior or inferior regions. Small hemorrhages may also be observable near the optic nerve head [5]. In the early stages of non-arteritic anterior ION, OCT of the RNFL may show thickening relative to the fellow eye. The RNFL thickening decreases rapidly in the subacute phase and up to 6 months after the incidence of anterior ION. Studies with longer follow-up showed no significant decrease in thickness between month 6 and month 12. Studies provided OCT angiography (OCTA) evidence that the superficial retina was more affected than choroid layer in AION. Also, radial peripapillary retinal nerve fibre layer was more affected than the intra-optic-disc area [6, 7].

Typically, patients with first-time, acute optic neuritis (ON) are young adults 20 to 45 years. In a population-based study in Olmsted County, Minnesota [8], the annual age- and sex-adjusted incidence rate was 5.1 per 100,000 person-years from 1985 to 1991, and the age- and sex-

adjusted prevalence rate per 100,000 was 115. Acute demyelinating optic neuritis is the presenting feature (i.e., the first clinical demyelinating event) in 15 to 20 percent of patients with multiple sclerosis (MS), and it occurs at some time during the course of the disease in 50 percent of patients [9]. The most typical signs are vision loss, visual field loss in the form of central or paracentral scotoma of various sizes, and loss of color vision. Only one-third of ON cases present with disc edema and hyperemia, while two-thirds of cases are retrobulbar (the optic disk appears normal). In the acute period of retrobulbar optic neuritis we can see either normal, diminished or increased RNFL thickness, the latter being due to subclinical axonal edema. Approximately 6 months after the acute attack of neuritis it is possible to detect peripapillary RNFL thinning [10].

Common causes of compressive optic neuropathy (CON) include orbital and intracranial meningiomas, pituitary adenomas, intracranial aneurysms, craniopharyngiomas, and gliomas of the anterior visual pathway. In a study by Liu and colleagues [11], the overall age and sex adjusted incidence of CON was 1.14 per 100,000 per year. CON from a tumor or mass usually causes slow, painless, progressive loss of vision. It usually affects one eye and typically affects central vision, except for tumors in the pituitary gland, which can compress the optic chiasm. Compression of the optic chiasm causes bilateral loss of peripheral vision (bitemporal hemianopia). OCT is done to evaluate the optic nerve and may show thickening or atrophy of the optic nerve [12].

Although traumatic optic neuropathy (TON) has become increasingly common due to combat actions in Ukraine, no statistics have been reported on the incidence of TON for the country. According to a systematic literature review by Iranian researchers [13], the overall incidence of TON is 0.7–2.5%, and indirect TON has a higher prevalence than direct TON. It occurs in 0.5% to 5% of all patients with closed head injury and 2.5% of patients with midfacial fractures. Intracanalicular part is the most common site of indirect TON (71.4%), followed by the orbital apex (16.7%) [13]. Clinical findings that help diagnose TON include variable loss of visual acuity (range 20/20 to no light perception), decreased color perception, visual field defects (central or paracentral scotoma, arcuate or hemianopic field defects), and relative afferent pupillary defect. The optic disc appearance will depend on the anatomical site and the timing of injury. With injuries to the optic nerve anterior to the entry point of the central retinal vessels, there is optic disc swelling with associated retinal hemorrhages. With more posterior injuries, which are more common, the fundus can look entirely normal. Optic disc pallor usually develops about 6 weeks following the initial injury [14].

Mechanisms for the generation and recording of visual-evoked poyentials (VEP)

VEP waveforms are extracted from the electro-encephalogram (EEG) by signal averaging. Electrographic brain responses to light-flash exposure were first reported in the early twentieth century. Today VEP is used as a quantitative method of assessing the functional status of the visual pathway from the retinal ganglion cells to the primary visual cortex [15].

The VEP is an important clinical test for assessing the functional integrity of the visual pathway from the retina to the striate cortex (primary visual cortex or V1). As such, this test has been extensively used in the evaluation of ophthalmic, neurological and systemic disease [16]. The VEP is produced from activation of cortical neurons in response to afferent pathway (retina → optic nerve → lateral geniculate body → visual cortex) stimulation, which is recorded with electrodes placed on the scalp (OZ in the 10-20 system) over the occipital cortex.

The major types of VEP are as follows:

1. Pattern reversed VEP (PR-VEP)

Features: A checkerboard or grating stimulus is reversed in contrast over time whilst maintaining a constant mean luminance. A major positive peak is seen around 100ms (P100) [17, 18, 19].

Indications: Assessment of macular pathway function and detection of optic nerve abnormalities. The PR-VEP has relatively low inter- and intra-subject variability [20, 21].

2. Pattern onset/offset VEP (PO-VEP)

Features: A checkerboard appears and disappears on a background with the same mean luminance. The PO-VEP waveform is more complex and variable.

Indications: Assessment of macular pathway function. These stimuli have benefits in the assessment of patients with poor vision and/or low cooperation [22].

3. Flash visual evoked potentials (F-VEP)

Features: VEP is recorded to diffuse flash stimuli. The F-VEP waveform is also complex, with the major N2 and P2 peaks.

Indications: Examination of the generalised visual pathway function, particularly in eyes with poor optical quality where retinal image contrast is degraded. These stimuli do have benefits in the assessment of patients with poor vision, low cooperation, detection of intracranial pathway dysfunction and inter-ocular differences [23, 24, 25, 26].

4. Chromatic VEPs

Features: Chromatic red-green ot blue-yellow stimuli are used to assess parvocellular and koniocellular parallel pathways of the visual system. The stimuli required for testing are technically challenging to achieve and interpret [27].

Indications: Identification of colour processing dysfunction in demyelinating disease [28, 29, 19], Leber hereditary optic neuropathy (LHON) [30], glaucoma [31], and Parkinsons disease [32].

The interpretation of responses is performed by assessing the response amplitude, peak-time/latency, morphology and transoccipital distribution. Abnormalities of the VEP should therefore always be explored with the pattern electroretinogram (PERG) to elaborate on the site and

extent of dysfunction. International standards for clinical VEP should be consulted and results should be interpreted in the context of the clinical picture [33, 34].

VEP results in subjects with particular types of optic neuropathy

Optic nerve disease may be caused by a variety of factors, and VEP can suspect the diagnosis or support the suspected diagnosis in the clinical interpretation of data [35, 36, 37, 38]. Although optic neuritis is most historically associated with MS [39, 40], its presentation, history or PR-VEP changes can have wide differential diagnosis including infectious (i.e. Lyme disease, herpes zoster, toxoplasmosis), toxic/nutritional (i.e. B12 deficiency, ethambutol, or tobacco-alcohol toxicity), compressive (i.e. intracranial tumours), disseminated neurological disorder (i.e. adrenoleukodystrophy, neuromyelitis optica spectrum (NMO) disorder), hereditary optic neuropathies (i.e. LHON or autosomal dominant optic atrophy (ADOA)) or systemic disease (i.e. systemic lupis erythematosus, Sjögrens syndrome or sarcoid), etc. [41].

The early works pioneering the clinical utility of the PR-VEP in optic neuritis came from Halliday et al [42] who demonstrated an increased latency in the PR-VEP as characteristic for conduction delay associated with demyelination. PR-VEP amplitude recovery is more quickly and closely related with the clinical improvement in visual acuity [43, 44, 45].

The multifocal VEP (mfVEP) can assess the functional regions of the optic nerve. They may be of benefit in mild forms of optic neuritis, where topographically only a small segment of nerve axons are affected (i.e. causing a small scotoma or peripheral field defect), the conventional VEP may mask an underlying defect as it is produced from both normal and abnormally functioning axons, whereas the mfVEP may detect this focal loss. However, the technical demands of a mfVEP and its accessibility have limited its widespread use to date [46].

VEP can be useful in patients with early or subclinical TON and for differentiating TON from demyelinating optic neuropathy. VEP usually reveals normal or near normal latency with significantly reduced amplitude of P100. The P50 and N95 components of PERG reflect macular and retinal ganglion cell (RGC) function, respectively. PERG can be useful in a patient with abnormal VEP to identify a macular lesion. Diagnosis is based on the identification of a toxic factor and exclusion of other pathologies giving a similar clinical picture [47, 48].

Compressive optic neuropathies can cause significant disruption to optic nerve and RGC physiology. Intracranial tumours may affect any portion of the visual pathway and, as such, the VEP is well suited to provide assessment in localising the pathway lesion and information of pathway integrity. As such, VEP is a useful tool in the examination of the intracranial visual pathway especially when used in conjunction with the PERG and/or photopic negative response (PhNR). When utilising a transoccipital array of electrodes, one can use the lateralising features of VEP distributions to identify chiasmal and retrochiasmal pathway dysfunction [49].

Seminal studies using F-VEP in patients with homonymous field defects have demonstrated the major positive peak to become altered in lesions relating to the underlying field defect. As such, in lesions of the chiasm, one can observe a ‘crossed’ asymmetry of pattern or F-VEPs, whereby the transoccipital asymmetry will alter its lateralisation dependent on the eye stimulated. These lateralising features have been adopted clinically to identify chiasmal misrouting. The lateralising features of the VEP can be also used in retrochiasmic lesions using a flash or pattern stimulus to produce an ‘uncrossed’ asymmetry [16, 50].

However, hemifield PR-VEPs are a far more advantageous method for investigating these intracranial visual pathway abnormalities [51, 52].

Furthermore, particularly for optic pathway gliomas, VEPs have high sensitivity for detecting functional abnormality, which is particularly advantageous in children for whom behavioural perimetric testing is unreliable although their ability to monitor progression is less certain [53].

PR-VEP amplitude reduction is a hallmark finding in cases of TON and VEP responses can be subnormal even after visual acuity recovery suggesting persistence of subclinical changes. However, VEPs performed in isolation cannot localise the functional deficit to the level of the optic nerve and must be performed in conjunction with pattern and flash ERG to confirm RGC and/or optic nerve functional deficit with a normally functioning outer retina [54].

The VEP abnormalities in ischemic optic neuropathy were first formally introduced by Wilson [55] who demonstrated the amplitude reduction of the PR-VEP, with small or no changes in latency seen. Other studies are in general agreement with Wilson’s amplitude findings, but some studies report varying degrees of latency abnormality, suggesting commensurate conduction delay is not an exclusion to VEP diagnosis of ION [56]. Further, utilising a small check width PR-VEP was found to have higher sensitivity in the detection of optic nerve dysfunction. Overall, a fair conclusion now is that ION predominantly affects PR-VEP amplitude, but latency can in some circumstances be affected. The flash VEP is typically lower amplitude with latency changes [57].

Rangaswamy et al [58] demonstrated in 17 patients with NAION that the PhNR amplitude is reduced and its decrease correlated to the reduction in visual field sensitivity. Including a control group, these authors demonstrated in their Receiver Operating Curve that the PhNR has 96% diagnostic accuracy for anterior ION. Interestingly it was also found that the PhNR was reduced in some degree in the asymptomatic eyes, suggesting there may be subtle signs of global RGC dysfunction before clinical signs of anterior ION may appear.

The mfVEP can provide further information regarding optic nerve integrity, particularly as they demonstrate a close relationship between visual field loss and topograph-

ic mfVEP amplitude reduction, although this may overlap with other optic neuropathies [46].

Conclusion

VEP is an objective electrophysiological method that plays an important role in the systematization of clinical and diagnostic features of optic neuropathies. This non-invasive method records the neuronal response of the visual system to stimuli, which is important for assessing the functional integrity of the visual pathways.

Its objectiveness (i.e., independence of the consciousness and attention state of the patient) allows researchers to obtain reliable data that can be used for the diagnosis of various lesions of the optic nerve and associated structures (inflammatory, atrophic, toxic, tumoral, and genetic disease) depending on the characteristics of the stimulus.

VEP can be used as an alternative method to assess visual acuity in non-verbal infants and adults with low intellectual abilities or potential malingering.

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Disclosures

Received: 01.06.2025

Accepted: 25.09.2025

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Author's contribution. *MSK: Investigation, Writing – Original Draft; VAV: Conceptualization; Writing – review & editing; OSS, Methodology, Formal Analysis; LLC: Formal Analysis; OIM, IIS: Investigation. All authors read and approved the final version of the manuscript.*

Funding. *No external funding.*

Conflicts of interest. *The authors declare that they have no conflicts of interest that could influence their opinions on the subject matter or materials described and discussed in this manuscript.*