# Clinical usefulness of the vep biology essay

Science, Biology

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\n[/toc]\n \nBACKGROUND: The visual evoked potential (VEP) is an electrical signal generated by the occipital visual cortex in response to stimulation of the retina either by light flashes or pattern stimuli. Since there are various factors affecting the VEP, one among them is the physiological factor ( i. e hormonal factor). Studies show that hormones play an important role in neuronal activity of visual, auditory, olfactory and taste thresholds . AIM & OBJECTIVE: To study the influence of ovarian hormones in the visual evoked potentials. MATERIALS & METHODOLOGY: 26 young female of age group 18-20 were randomly selected from the I year medical students. Informed consent was obtained. The study was approved by institutional ethical committee. Those having regular periods for past 6 months and having 6/6 vision were included in the study. Those having irregular periods, H/O PCOD, and those taking any hormonal treatment were excluded from the study. The phases of menstrual cycle were determined by the detailed history and the latency of p 100 wave of pattern reversal visual evoked potentials were recorded during these phases. RESULTS: Statistical analysis was done using

unpaired t test. When compared with the Luteal phase, during the Follicular phase significant reduction in p 100 latency (101 ± 3. 61 Vs 97. 9 ± 1. 43 ms; p < 0. 0001) were recorded. CONCLUSION: Estrogen is reported to cause a decrease in the visual transmission time by increasing the sensitivity of receptors in the optic pathways to dopamine . The effect of estrogen on the central nervous system seems to be antagonized by progesterone. Therefore reduction in latency during follicular phase is due to oestrogen and prolonged VEP latency in luteal phase is thought to reflect the effect of progesterone. Thus variation in the ovarian steroid hormones have an impact in the excitability of visual system.

## INTRODUCTION:

In 1934, Adrian and Matthew noticed potential changes of the occipital EEG can be observed under stimulation of light. Ciganek developed the first nomenclature for occipital EEG components in 1961. During that same year, Hirsch and colleagues recorded a visual evoked potential (VEP) on the occipital lobe (externally and internally), and they discovered amplitudes recorded along the calcarine fissure were the largest. In 1965, Spehlmann used a checkerboard stimulation to describe human VEPs. An attempt to localize structures in the primary visual pathway was completed by Szikla and colleagues. Halliday and colleagues completed the first clinical investigations using VEP by recording delayed VEPs in a patient with retrobulbar neuritis in 1972. A wide variety of extensive research to improve procedures and theories has been conducted from the 1970s to today1-3. A visual evoked potential (VEP) is an electrophysiological potential that can be extracted, as a signal, from the electrical activity of brain which is recorded

at the scalp. It is caused by a visual stimulus, such as an alternating checkerboard pattern on a computer screen and responses are recorded from electrodes that are placed on the head and observed as a reading on a monitor. These responses originate from the occipital cortex, the area of the brain involved in receiving and interpreting visual signals. The VEP can provide important diagnostic information regarding the functional integrity of the visual system. VEP peak latency and amplitude are used as the parameters. VEP peak latency refers to the time from stimulus onset to the maximum positive or negative deflection. VEP peak latency may also be referred to as ‘ time to peak’ or peak time. The VEP measures the time that it takes for a visual stimulus to travel from the eye to the occipital cortex. It gives us an idea of whether the nerve pathways are abnormal in any way. For example, in multiple sclerosis, the insulating layer around nerve cells in the brain and spinal cord (known as the myelin sheath) can be affected. This means that it takes a longer time for electrical signals to be conducted from the eyes, resulting in an abnormal VEP. A normal VEP can be fairly sensitive in excluding a lesion of the optic nerve, along its pathways in the anterior part of the brain.

## VEP Stimuli:

The diffuse light flash stimulus is rarely used due to the high variability within and across subjects. However, it is beneficial to use this type of stimulus when testing infants or individuals with poor visual acuity. The checkerboard and grating patterns use light and dark squares and stripes, respectively. These squares and stripes are equal in size and are presented to one at a time via a television or computer screen.

## VEP Waves

The VEP nomenclature is determined by using capital letters stating whether the peak is positive (P) or negative (N) followed by a number which indicates the average peak latency for that particular wave. For example, P50 is a wave with a positive peak at approximately 50 ms following stimulus onset. The average amplitude for VEP waves usually falls between 5 and 10 microvolts.

## Types of VEP

Some specific VEPs are: Sweep visual evoked potentialBinocular visual evoked potentialChromatic visual evoked potentialHemi-field visual evoked potentialFlash visual evoked potentialLED Goggle visual evoked potentialMotion visual evoked potentialMultifocal visual evoked potentialMulti-channel visual evoked potentialMulti-frequency visual evoked potentialStereo-elicited visual evoked potentialSteady state visually evoked potential

## Clinical usefulness of the VEP:

The VEP is a standardised and reproducible test of optic nerve functionIt is more sensitive compared to magnetic resonance imaging (MRI) in detecting lesions affecting the visual pathway in front of the optic chiasm (area in the optic pathway where the optic nerve crosses sides)It is usually less costly compared to other investigations such as MRIIf results of the VEP are negative, this can be useful in excluding certain disorders. The VEP is an important test that is very good at detecting problems with the optic nerve and lesions in the anterior part of our visual pathway, before the optic nerves

emerge. However, it is a non-specific test and to determine the exact underlying problem in each patient, a good history and examination is also very important4-7. The human menstrual cycle lasts for 28 days and divided into 4 phases: follicular, ovulatory, luteal and menstrual. The menstrual cycle influences various clinical and neurological conditions such as atopic dermatitis, diabetes, asthma, rheumatoid arthritis, pulmonary edema, myasthenia gravis, multiple sclerosis, aneurysms, meningioma, epilepsy, and migraine may be worse during pre-menstrual phase. EEG also varies during different phases of the menstrual cycle. Since, during the menstrual cycle, there are changes in neuronal activity in auditory, olfactory, and taste thresholds have been documented, this study attempts to study the variation in VEP latency during the various phases of menstrual cycle.

## MATERIALS & METHODOLOGY:

26 young female of age group 18-20 were randomly selected from I year MBBS. Informed consent was obtained. The study was approved by institutional ethical committee. Those having regular periods for past 6 months and having 6/6 vision were included in the study. Those having irregular periods, H/O PCOD, and those taking any hormonal treatment were excluded from the study. The phases of menstrual cycle were determined by the detailed history. The latency of p 100 wave of pattern reversal visual evoked potentials were recorded during these phases. These latencies were recorded by placing the active electrode over the occipital cortex, reference electrode over the vertex and ground electrode placed over the forearm. The visual evoked potentials were recorded in the NEROPERFECT EMG 2000 SYSTEM. RESULTS: Statistical analysis was done using unpaired t test. When

compared with the Luteal phase, during the Follicular phase significant reduction in p 100 latency ($p < 0.0001$) were recorded.

# DISCUSSION:

Prolonged VEP latency during luteal phase indicates that high progesterone levels may have an inhibitory effect on optic nerve conduction velocity . Increased latency on VEP waves is the hallmark of many visual pathway diseases. Excitatory oestrogen increases the sensitivity of the central nervous system to catecholamines by changing the opening frequency of voltage-related L-type calcium channels and augmenting the effect of glutamate; in addition it inhibits the formation of gamma-amino butyric acid (GABA) by the inhibition of glutamate decarboxylase enzyme. It is argued that oestrogen increases transmission in the optic pathways and that oestrogen is responsible for the shorter latency values and higher amplitudes of visual evoked potentials in women. There are different studies on VEP changes in healthy females during the menstrual cycle. Mohsen Azarmina et al8, observed that the Pattern and flash VEPs were performed in 15 healthy women aged 18 to 25 years and they concluded that the prolonged VEP latency on the maximum bleeding day indicates that high progesterone levels may have an inhibitory effect on optic nerve conduction velocity and also Avitabile T et al9 in his study, compared with the follicular phase, during the luteal phase significant reduction in VEP latency and increase in amplitude were recorded in 50 healthy women. Yilmaz H et al , suggested that sex steroids seemed to affect the generation of PRVEPs. The significant decrease in PRVEP latencies when estrogen levels peaked was thought to be due to facilitating effect of estrogen on the neural transmission

of the visual pathways10. Tasman A et al found that the latency of P300 was longer during the ovulatory phase, and they suggested that there may be a small relationship between visual ERP or BAEP and MC phase11 and Studying 23 healthy female subjects with regular menstruation, Kaneda et al12 showed increased latency on flash VEPs associated withlow estrogen and high progesterone levels and Shushtarian et al13 reported prolongation of flash VEP latency in 20 female subjects during a normal cycle. Vingerling et al reported an association between macular degeneration and early menopause14. The effect of estrogen on the central nervous system seems to be antagonized by progesterone and its metabolites, therefore prolonged VEP latency is thought to reflect the effect of progesterone15, 16. The most probable reasons for increased VEP latency during menstruation may be as follows: 1. decrease in blood estrogen levels and diminution of the neuroprotective effect of estrogen15; 2. associated biochemical changes causing anxiety and stress16, 17, thus interfering with concentration on the central target of the monitor; 3. vascular congestion around the optic nerve reducing conduction velocity18. Hence, prolongation of VEP latency during the menstrual cycle in the luteal phase probably reflects the effect of progesterone. Yilmaz H et al19 recorded the monocular pattern reversal visual potentials (PRVEP) of both eyes of 54 post-menopausal women before treatment and of 30 of them after replacement therapy with Tibolon, and of 24 women receiving placebo treatment. They found statistically significant decrease in the mean PRVEP latencies and a statistically significant increase in mean amplitudes after replacement treatment ($P < 0.001$) compared with those before treatment and those after placebo treatment. Hence, VEP

analysis is a useful tool for the study of the actions of gonadal hormones on CNS in humans. While interpretating VEP in females, one should remember the influence of gonadal hormones in the VEP latency during menstrual cycle before thinking of the pathology. CONCLUSION: Estrogen is reported to cause a decrease in the visual transmission time by increasing the sensitivity of receptors in the optic pathways to dopamine. The effect of estrogen on the central nervous system seems to be antagonized by progesterone and , therefore prolonged VEP latency is thought to reflect the effect of progesterone. Thus variation in the ovarian steroid hormones have an impact in the excitability of visual system.