Maturation of visual cortex – assessment by visual evoked potentials
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Brain evoked potentials measure electrophysiological responses of the central nervous system to a variety of stimuli. Almost any sensory modality can be tested in theory; in clinical practice, however, only few are used on a routine basis. The ones most often encountered are the visual evoked potentials (VEPs), short-latency somatosensory evoked potentials (SEP), and short-latency brainstem auditory evoked responses (BAER, BAEP). Late-evoked responses (cognitive evoked potentials – event related potentials – ERP) are used for studying higher cortical functions, such as P300 wave in Alzheimer disease. Evoked potential amplitudes are usually rather small compared to spontaneous EEG. To resolve these low-amplitude potentials against the background of ongoing EEG and other biological signals, signal averaging is usually required.

The VEPs represent electrical activity of large populations of neurons in the visual cortex and they are usually recorded with electrodes placed on the scalp (non-invasive examination). They test the function of the visual pathway from the retina to the occipital cortex. Their waveform depends on the frequency of stimuli repetition – at rapid rates it is sinusoidal (steady state VEPs), for lower rates it consist of several positive and negative deflections specific for different stimuli (transient VEPs). According to the type of stimulus, flash VEPs, pattern VEPs and motion VEPs are recently performed. The pattern-related VEPs reflect activity of the parvocellular system and the ventral stream of the visual pathway and they do not provide sufficient information about the function of the magnocellular system and the dorsal stream. Their function can be, however, tested by means of motion related VEPs, especially those which are elicited by motion-onset (Kuba et al. 2007). To test the visual pathway in a complex way, both pattern-related and motion-related VEPs must be examined.

All mentioned types of VEPs display some changes during maturation. Whilst maturation of the flash VEPs and pattern-related VEPs is finished by 6 years of age, the motion-onset VEP latencies maturate (shorten) much longer - up to about 18 years of age and then they immediately start to prolong due to ageing processes (Langrova et al. 2006). For that reason, special age-corrected norms are necessary for determination of the VEPs pathology in clinical practice. The motion-onset VEPs not only go through significant latency development but their shape also changes, especially in the VEPs to radial motion stimuli. In young children the dominating peak of the VEP is positive and is followed by very late negative peak. This positive peak diminishes during maturation and the most prominent peak of the “adult” motion-onset VEPs becomes the negative N160 peak. The development of the motion-onset VEP shape complicates diagnostic use of this kind of VEPs in children. Prolonged or inappropriate maturation of the magnocellular pathway / dorsal stream is one of important etiological factors for reading disability (dyslexia), which can be among others diagnosed also by the motion-onset VEPs (Kubova et al. 1996). Explanation how the magnocellular dysfunction can lead to dyslexia is based on a relationship between magnocellular pathway/dorsal stream activity and the control of eye movements.

These and other papers on this topic can be found at: http://www.lfhk.cuni.cz/elf/