Flash visual evoked potentials in healthy infants

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INTRODUCTION

Investigation of visual system in children, especially in infants, may be challenging and often needs implementation of neurophysiologic methods, such as visual evoked potentials (VEPs) [1-2]. Maturation of the visual pathways takes months after the birth, with its final stages being at 3–5y. Newborns prefer visual patterns resembling a human face; 6wk old infants already start to distinguish internal configuration of the mother’s face, detect the living object’s movements and prefer human movements[3-4]. It is argued that visual processing in newborns aged less than 2mo goes on subcortical level, as visual cortex is quite immature yet [1].

In infants, it’s more reasonable to use flash visual evoked potentials (fVEPs), as in this case there is no need in fixed stare on checkerboard pattern, routinely used in pattern VEPs [5]. Wave continuity P2–N2–P3 is supposed to be the main VEPs complex, with its average latency in adults being 124–130 ms, and amplitude = 7–8 μV [5]. In preterm infants fVEPs waves, which may be analyzed properly, start to appear at 24wk postmenstrual age [6].

In first year of life fVEPs parameters in healthy infants, acquired in different laboratories, vary significantly. Some authors reports mean P2 latency 100 ms and N3 150 ms [7]; main cortical peak latency 100–170 ms and amplitude 2–15 μV [8]; average P100 latency (pattern VEPs) 179–180 ms [9]; mean P2 latency 170 ± 16 ms and amplitude 6.26 ± 5.46 μV [10]; P3 latency 145 ms [11]; P2 latency 155 ± 30 ms and amplitude 5.9 ± 4.3 ms [12], mean N2 latency 185.98 ± 31 ms and its amplitude 1.78 ± 1.5 μV [13]. As it may be seen from the data summarized in Table 1, difference in values is vast; have to be noted, that different authors attributed same waves on the fVEPs as different P and N peaks, thus contributing to differences of the data presented.

As may be seen from the data presented, main issue is not only the latency of the cortical wave; amplitude also vary in the range 1.78–15 μV, according to different authors. Thus, despite seemingly well-established normative data for fVEPs, in infants this data needs further exploration.

SUBJECTS AND METHODS

Our study was conducted among healthy children (n = 34, 68
Table 1 Latencies and amplitudes of the main cortical wave in healthy infants, according to different authors

<table>
<thead>
<tr>
<th>Authors</th>
<th>Main wave analyzed</th>
<th>Latency (ms)</th>
<th>Amplitude (µV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pompe et al(2011)</td>
<td>N/A</td>
<td>100–170</td>
<td>2–15</td>
</tr>
<tr>
<td>Muñoz et al(2009)</td>
<td>P100</td>
<td>179–180</td>
<td>N/A</td>
</tr>
<tr>
<td>Feng et al (2013)</td>
<td>P2</td>
<td>170±16</td>
<td>6.26±5.46</td>
</tr>
<tr>
<td>Kasimov et al(2010)</td>
<td>P3</td>
<td>145</td>
<td>N/A</td>
</tr>
<tr>
<td>Brinciotti et al (2009)</td>
<td>P2</td>
<td>155±30</td>
<td>5.9±4.3</td>
</tr>
</tbody>
</table>

Table 2 Amplitudes and latencies of the N2/P3 complex in the group

<table>
<thead>
<tr>
<th>Parameter</th>
<th>OD</th>
<th>OS</th>
</tr>
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<tbody>
<tr>
<td>P3 latency (ms)</td>
<td>138.9±15.6</td>
<td>140.8±17.2</td>
</tr>
<tr>
<td>P3 amplitude (µV)</td>
<td>8.48±5.46</td>
<td>7.91±5.06</td>
</tr>
<tr>
<td>Latencies asymmetry (ms)</td>
<td>6.75±4.19</td>
<td></td>
</tr>
<tr>
<td>Amplitudes asymmetry (µV)</td>
<td>1.9±1.4</td>
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DISCUSSION

Latency asymmetry which was seen in our population may happen due to incomplete myelination of the visual pathways. Apart from myelination, important role for the proper functioning of the visual system plays development of temporal lobe and basal ganglia; in infants this process varies between healthy individuals. Absence of the significant difference of the amplitudes between the eyes may be due to small size of the head of the infant or possibly unspecific conduction of the excitation between the brain parts. Also it has to be stressed that there are no solid proof of the cortical genesis of the so-called cortical waves on the fVEPs in the infants of the first 3 mo of life. It was demonstrated that in 100% of the infants with periventricular leukomalacia main waves of fVEPs were registered, then in children with subcortical lesions no proper peaks were seen. Also fVEPs were insufficient in detecting parieto-occipital lobe involvement in infants after significant hypoglycaemia as compared to healthy controls, which may point to the subcortical levels of generation of the main peaks of these evoked potentials in the first months of life.

Thus we can assume that in healthy children of the first months of life (7 of our infants were younger than 90 d) N2–
P3 complex may appear due to subcortical centers functioning. In lots of animals which have no cortex at all visual system still functions properly; since 1930s one of the main questions in this field was discussion about the possibility of "midbrain vision" in human\(^7\). It was argued that infants with yet undeveloped higher centers of visual cortex may see due to activation of phylogenetically ancient pathway that has specific functional properties and that interacts with cortical processes\(^8\). Also this pathway may play significant role in vision in adults\(^9\).

Variability of latencies in our population of healthy infants demonstrates that normative data in infants have to be treated with caution. Deviation of latency (or amplitude) from the normative parameters not always have to mean some pathologic process on the way; we assume that more significant is the absence of the waves (unilateral or bilateral). One recent work argues that delay of myelination may be the main cause of latency differences in infants\(^10\).

Our attribution of the main waves complex which we registered as N2/P3, as was demonstrated, may be a matter of debate; anyway, it is certainly the wave complex which reflects the main processing of the visual information in the infant's brain. Its absence may lead to the conclusion that visual system formation is disrupted in some way. In all cases the dynamic investigation is recommended. Pattern visual evoked potentials seems to be more reliable and easy to interpret than IVEPs, so perhaps its implementation in pediatrics as early as possible may be recommended\(^21\).

Thus, in all healthy infants aged less than 1y main cortical wave complex may be registered. Its average latency in our population was 138–140 ms and average amplitude 7–9 μV. Latency or/and amplitude deviation from the established normative data may not be due to pathologic conditions, but may be seen in healthy infants as well. In evaluation of IVEPs in infants caution is needed; real pathologic finding may be not the latency lengthening or amplitudes drop, but presence or absence of the main cortical complex (may be attributed as N2/P3).

REFERENCES
2 Ortinj EL, De Cokk PP, Lagae LG. Visual perception in preterm children; what are we currently measuring? Pediatr Neurol 2011;45 (1);1–10
3 Bushnell IWR, Sai F, Mullin JT. Neonatal recognition of the mother's face. Br J Dev Psychol 1989;7(1);3–15
4 Yoong JM, Johnson SC. Biological motion displays elicit social behavior in 12–month-olds. Child Dev 2009;80(4);1069–1075
7 Kosheley DI, Galantdinov MF, Vachmanyanna AA. The practice of the application of the flash visual evoked potentials for the visual system's evaluation. Vestnik OGU 2014;12(173);181–187
8 Pompe MT, Kranje BS, Breeijel J. The study of chromatic and achromatic VEP in the first year of life. Acta Ophthalmol 2009;87 (s244):0
9 Muñoz CI, JA Moreno S, Sierra FM. Pattern reversal–evoked potentials evolution in preterm babies. Clin Neurophysiol 2009;120(4); e137
10 Feng JJ, Wang WP, Guo SJ, Liu ZW, Xu X. Flash visual evoked potentials in preterm infants. Ophthalmology 2013;120(3);489–494
15 De Regnier RA. Neurophysiologic evaluation of brain function in extremely premature newborn infants. Semin Perinatol 2008;32(1);2–10
16 Hu L, Gu Q, Zhu Z, Yang C, Chen C, Cao Y, Zhou W. Flash visual evoked potentials are not specific enough to identify pericerebral lobe involvement in term neonates after significant hypoglycaemia. Acta Paediatr 2014;103(8);e329–333
18 Tamiotto M, de Gelder B. Neural bases of the non–conscious perception of emotional signals. Nat Rev Neurosci 2010;11(10);697–709
19 Visconti di Oleggio Castello M, Golbini MI. Familiar Face Detection in 180 ms. PLoS One 2015;10(8);e0136548
20 Jethani J, Jethani M. Flash visual evoked potentials in patients with periventricular leukomalacia in children less than 1y of age. Indian J Ophthalmol 2013;61(11);634–635