## THE IMPORTANCE OF SEP EXAMINATION IN THE DIAGNOSIS OF HYPOTONIA SYNDROME IN YOUNG CHILDREN

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#### **Abstract**

The purpose of the study. Comparison of electrophysiological (SEP) indicators in young children with muscular hypotonia syndrome and development of SEP criteria for topical diagnosis.

In the period 2022-2023, 105 children with muscular hypotonia syndrome (SMH) of early age (0-3 years) were examined. All patients were divided into 4 groups according to clinical and anamnestic, ultrasound, Doppler and electrophysiological parameters. In the course of this scientific study, new SEP criteria were identified, indicating suprasegmental and segmental changes in the clinical SEP diagnosis of muscular hypotonia syndrome in young children.

Each group was tested for short-latency somatosensory evoked potential by stimulation of the median nerve.

Our investigations revealed that the use of SEP recording methods allows for a realistic assessment of the excitability and conduction function of the structures of the brain and spinal cord. In our patients with central muscle hypotonia, the latency period of N20 and P25 peaks and the increase of N9-N20, N13-N20 intervals were observed. In patients with peripheral genesis of muscle hypotonia, it was found that the latent period of the N13 peak was prolonged, and the N9-N13 and N13-N20 intervals were increased. Lengthening of the latent period of N13, N20, P25 peak components, increase of N9-N13, N13-N20 interval was observed in both central and peripheral lesions. This indicates a violation of conduction from the dorsal medial lemniscal column of the somatosensory pathway to the somatosensory cortex.

This study shows that these methods allow the assessment of the entire nervous system, from the peripheral nerve to the cerebral cortex, and therefore this examination may be useful in determining whether children with muscle hypotonia are of central or peripheral origin.

Keywords: young children, muscular hypotonia syndrome, SEP

Determining the underlying cause of muscle hypotonia remains difficult despite advances in diagnostic laboratories and imaging techniques. Clinical assessment strategies and standardized developmental tests can help distinguish between hypotonia resulting from primary involvement of upper motoneurons and lower motoneuron hypotonia. This is especially important in young children.

Modern neurophysiology studies show that the SEP detects afferent waves from the dorsal column of the spinal cord to the cerebral cortex through the brain stem and spinothalamic pathway in response to stimulation. because of this, the SEP method is considered to be one of the most adequate methods for determining the damage center of the brain and spinal cord.

**Research objective.** Comparison of electrophysiological (SEP) indicators in early childhood with muscle hypotonia syndrome as well as the development of SEP criteria that help in topic diagnosis.

**Research methods.** In the period 2022-2023, 110 children with muscle hypotonia syndrome (MGS) of early age (0-3 years) were examined at the 1st children's Clinical Hospital of Tashkent City. Of this, boys made up 57 (54.2%) and girls made up 48 (45.7%).

All patients were divided into 4 groups according to clinical anamnestic, ultrasonic, dopplerographic and electrophysiological indicators.

Children with MHS of the central type (Group 1) were 31 (29.5%), children with MHS of the peripheral type (Group 2) were 24 (22.8%), mixed-i.e. children with MHS of the Central and peripheral types (group 3) were 46 (43.8%) and children with MHS were of the genealogical type (Group 4) were 4 (3.8%). A control group of 20 healthy children was also formed to compare the indicators obtained. Clinical-anamnestic, ultrasonic, dopplerographic and electrophysiological indicators in all groups of children were studied on the basis of statistical comparison.

The SEP examination was used to record the precise response of the somatosensory pathway from the periphery to the cortex in relation to somatosensory stimulation. It was manifested with different changes in different branches of SEP in head and spinal cord injuries. Peripheral nerves were usually stimulated through the skin, and electrodes were placed on the skin over the selected nerve. Clearer and more reliable responses were obtained by stimulation of the median nerve.

SEP testing was carried out on a 4-channel electromiography from "DEYMED".

The results of a study. The results of the electromyographic examination are presented in Table 1. Short-latensy somatosensory evoked potential examination was conducted in the investigated groups by stimulation of the median nerve. Latency of peak components N9, N11, N13, N20, P25 and interpeak latency of N9-N13, N13-N20 and N9-N20 in SEP examination routinely recorded by stimulation of N.medianus in each group was determined. The results of

the analysis showed that when a short latency somatosensory evoked potential test was performed by stimulating n. medianus in the investigated groups, the latency of the N 9 peak in group 1 was  $8.6 \pm 1.5$ ms, the latency of the N 13 peak latency was  $12.8 \pm 1, 5$ ms, the latent period of the N 20 peak latency was  $25.5 \pm 1.7$  ms, the latency of the P25 peak latency was  $29.2 \pm 0.9$  ms, the inter-peak interval of N9- N13, N9- N20, N13-N20 was 4,2 ms, 16,9 ms, 12,7ms respectively. This indicator indicates the low response of the somatosensory cortex to external influences, that is, damage to the central nervous system. In group 2, unlike group 1, the following results were determined: the latency period of the N9 peak latency was 9.6±0.5ms, the latency period of the N13 peak was 17.1±1.6ms, and the latency of the N20 peak was  $18.9\pm0.7$ ms., the latent period of the P25 peak latency is  $22.2\pm0.9$ , the N9-N13 interval is 7.5; N9-N20 interval 9.3; The N13-N20 interval was 1.8 ms. A prolongation of the latency of the N13 peak from the Cv6 network, indicative of changes in the spinal cord, was indicative injury of spinal cord. In group 3, in all patients, the latent period of peak components N9, N13, P25 significantly ( $16.1\pm0.4$ ;  $26.1\pm1.7$ ;  $30.2\pm0.9$ ) lengthened in the corresponding treatment, N9-N13, N9 -N20, an increase in the N13-N20 interval (6.5; 16.5; 10) was observed (p<0.001). This indicates a violation of conduction from the posterior medial lemniscal column of the somatosensory pathway to the somatosensory cortex. In group 4, the latency of the N9 component is 8.2±1.5 ms, the latency of the N13 peak is 15.1±1.4ms, the latent period of the N20 peak is 25.9±0.7ms, the latency of the P25 peak is 29±1, was 2 ms, N9-N13, N9-N20, N13-N20 interpeak interval showed 6.9 ms, 17.7 ms, 10.8 ms, respectively (p<0.001). These results, similar to the results of group 1, prove a combined injury of the brain and spinal cord. The average latency period of SEP peaks in control group children was observed as follows: N9 peak latency  $9.6 \pm 0.5$  ms, N13 peak latency  $13.1 \pm 0.4$  ms, cortical peak latency  $18.9 \pm 0.4$ 0.7 ms (N20) and was 22.2  $\pm$ 0.9 ms (P25) (Table 1). **Table 1.** 

Table 1. Averages of the latency peaks in somatosensory evoked potential investigation obtained by stimulation of N. Medianus

| Group         | Latency period (ms) |              |                         |              |               |              |
|---------------|---------------------|--------------|-------------------------|--------------|---------------|--------------|
| 20            | N9                  | N13          | N20                     | P25          | N13-N20       | N9-N13       |
| Group 1       | 8,6 🗆 0,29          | 12,64 🗆 0,21 | 25,77□0,15              | 28,7 □0,4    | $12,13\pm0,4$ | $4,04\pm0,8$ |
| Group 2       | 8,7 🗆 0,17          | 16,48 🗆 0,2  | 19,11 🗆 0,27            | 25,07 🗆 0,4  | 2,63±0,8      | 7,78±0,2     |
| Group 3       | 8,7 □ 0,2           | 16,3 🗆 0,12  | 25,07 🗆 0,25            | 28,65 🗆 0,34 | 8,77±0,1      | 7,6±0,3      |
| Group 4       | 7,8±0,5             | 13,65 □0,2   | 24,13 \( \text{0,27} \) | 28,38 🗆 0,2  | 10,48±0,13    | 5,85±0,27    |
| Control group | 8□0,23              | 12,1 🗆 0,4   | 19,1 🗆 0,24             | 24,2 🗆 0,6   | 7±0,2         | 4,1±0,3      |

Our investigations revealed that the use of SEP recording methods allows for a realistic assessment of the excitability and conduction function of the structures of the head and spinal cord. According to electrophysiological studies, the processes of generation of bioelectric activity in muscles and nerve conductors are characterized by a balanced interaction of rising and falling impulse currents at the segmental level, stability of formation and symmetrical

distribution in bilateral biological structures. The obtained information allows discussing the principle of functional unity in the activity of central and peripheral mechanisms of the nervous-muscular system at a new neurophysiological level, which provides greater convenience.

In conclusion, it can be said that the early diagnosis of hypotonia in children of an early age, the identification of hereditary diseases accompanied by hypotonia, comparative reassurance that hypotonia is central or peripheral Genesis and the choice of treatment tactics can help practical doctors, as well as the selection of corrective measures aimed at eliminating changes, as well as improving the effectiveness of treatment.

### References

- 1. Burkova A. S. Classification of perinatal CNS injuries: Method. Recommendations / A. S. Burkova, N. N. Volodin, L. T. Zhurba et al. M., 2005. 40 p.
- 2. Human cortical potentials evoked by stimulation of the median nerve. II. Cytoarchitectonic areas generating long-latency activity. Allison T, McCarthy G, Wood CC, Williamson PD, Spencer DD.J Neurophysiol. 1989 Sep;62(3):711-22. doi: 10.1152/jn.1989.62.3.711.PMID: 2769355
- 3. Generators of short latency human somatosensory-evoked potentials recorded over the spine and scalp. Lee EK, Seyal M.J Clin Neurophysiol. 1998 May;15(3):227-34. doi: 10.1097/00004691-199805000-00006.PMID: 9681560 Review.
- 4. Cortical somatosensory evoked potential amplitudes and clinical outcome after cardiac arrest: a retrospective multicenter study.
- 5. Aalberts N, Westhall E, Johnsen B, Hahn K, Kenda M, Cronberg T, Friberg H, Preuß S, Ploner CJ, Storm C, Nee J, Leithner C, Endisch C.J Neurol. 2023 Aug 28. doi: 10.1007/s00415-023-11951-4. Online ahead of print.PMID: 37639017
- 6. [Somatosensory evoked potentials/fields--exploration of brain function].
- 7. Inoue K, Shirai T, Harada T, Mimori Y, Matsumoto M.Rinsho Byori. 2004 Jan;52(1):77-80.PMID: 14968564 Review. Japanese.
- 8. Igarashi M: Floppy infant syndrome. J Clin Neuromuscul Dis. 2004, 6:69-90.10.1097/00131402-200412000-00003
- 9. Kaur J, Punia S: Floppy infant syndrome: overview. Int J Physiother Res. 2016, 4:1554-63.10.16965/ijpr.2016.134
- 10. Miller VS, Delgado M, Iannaccone ST: Neonatal hypotonia. Semin Neurol. 1993, 13:73-83.10.1055/s-2008-1041110
- 11. Peredo DE, Hannibal MC: The floppy infant: evaluation of hypotonia . Pediatr Rev. 2009,30:66-76. 10.1542/pir.30-9-e66
- 12. Peter Siao, Michelle Kaku. A Clinician's Approach to Peripheral Neuropathy. Semin Neurol 2019; 39(05): 519-530.
- 13. Prasad AN, Prasad C: The floppy infant: contribution of genetic and metabolic disorders .Brain Dev. 2003, 25:457-476. 10.1016/s0387-7604(03)00066-4