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Shortened silent period suggests inhibitory deficits in children with spina bifida

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Background

This study is part of the multidisciplinary research program "Prognosis of Spina Bifida" performed at the Radboud University Nijmegen (The Netherlands). From a macroscopic point of view, spina bifida is a congenital malformation of the central nervous system in which both spinal cord and the brain are involved. However, from a microscopic point of view, little is known about how spina bifida affects the cortical and spinal interneuronal network. Assessment of the silent period (SP) following Transcranial Magnetic Stimulation (TMS) allows the opportunity to investigate inhibitory deficits in this network. Our aim was to investigate if inhibitory deficits are involved in the pathophysiology of spina bifida. Therefore, we studied the SP in children with spina bifida.

Material and methods

TMS was performed in 37 children with spina bifida (mean age 11.2 years, SD 2.8). Of these, 24 subjects were diagnosed with open spinal dysraphism and 13 with closed spinal dysraphism. Motor evoked potentials (MEPs) and SPs were bilaterally recorded from the biceps brachii muscle.

Results

Measurable MEPs and SPs could be obtained in all subjects. The mean durations of the SPs (right mean 66.8 ms, SD 25.3 and left mean 67.6 ms, SD 26.1) were shorter than reference values from the literature (mean 94.0 ms, SD 10.1[1]). The mean duration of the SP did not differ between subjects with open spinal dysraphism and subjects with closed spinal dysraphism. No differences were found in mean duration of the SP between subjects with and without hydrocephalus, Chiari malformation or corpus callosal dysgenesis.

Conclusion

Although the mechanisms for TMS induced silent periods are poorly understood, both cortical and spinal factors appear to be involved. Therefore, a shortened SP in children with spina bifida suggests deficits in the cortical and/or spinal inhibitory mechanisms, even in children with closed spinal dysraphism and in children without macroscopic cerebral malformations. We suggest that these deficits are more likely to be congenital rather than to be a result from secondary damage.

References

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