

The potential of SEPs recordings to address neurodevelopmental disorders in children

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The pathophysiology of several clinical conditions in humans presents with abnormal brain cortical excitation levels. This is potentially due to either reduced cortical excitability [e.g. all conditions of depressed consciousness] or enhanced cortical excitability [e.g. migraine¹⁻⁴, epilepsy⁵, and dystonia⁶]. We can measure the level of cortical excitability by means of different techniques; some of them are invasive, several others are not. Here we will consider somatosensory evoked potentials [SEPs] as: 1) their use is very common in ordinary clinical activity, 2) technicians are largely independent in recording acquisition, 3) their analysis and interpretation are quite fast, and 4) the technology required is economic. In order to be useful for wide clinical research, any technique has to fulfill these criteria, and SEPs do so.

We usually consider three main neurophysiological parameters of cortical excitability [also sometimes referred to as cortical responsiveness – to somatosensory influx within the cortex] when interpreting SEPs: 1) habituation, 2) recovery cycle, and 3) high frequency oscillations [HFOs].

The first refers to the reduction of the amplitude of the potential [reduction of the amplitude of the evoked peak and of the overall area under the curve] [e.g. the N20 response when stimulation is provided to the arm], when repetitive, non-noxious stimuli are administered. Habituation represents a brain cortical response aimed at avoiding irrelevant consumption of energy for processing stimuli that do not bring any new information. The more habituation phenomena are weak, the more the responsiveness of the cortex is large. Absent or weak habituation stays for abnormal and excessive cortical excitability.

The second refers to the time interval necessary for the somatosensory cortex to respond again to another stimulus, when couples of stimuli are provided. If the second stimulus is provided too soon [few millisecond after the first delivery], the cortex will still be in a depressed condition [in a sort of a repolarization phase that follows the depolarization one that correspond to the cortical response] and the second cortical response will be weak. Modifying inter-stimulus intervals [e.g. 5, 10, 20, 40 milliseconds] we can measure the time interval necessary to the cortex to regain the response amplitude that followed the first stimulus administration. It is usually expressed as percentage of the first response. Again amplitude of the evoked potential [peak amplitude and area under the curve amplitude are considered] directly expresses the level of cortical excitability. The more rapidly the cortex regains the 100% of the first stimulus-dependent amplitude, or the shorter the recovery cycle is, the more the cortex is responsive/excitable.

The third refers to high frequently oscillations [they are micro-wavelets] of the evoked potential. All potentials such as the N20 [for the arms] and P40 [for legs] responses are commonly referred to as low-frequently potentials and they represent a macroscopic neurophysiological phenomenon. HFOs

are micro-phenomena present within the low-frequency response. They are usually divided into two sections: HFOs of the ascending slope of the low-frequency cortical evoked response, and the HFOs of the descending slope. HFOs strictly depend on the bi-directional thalamo-cortical network: the more this system functions properly, the more the background noise present within each sensorial system is low. The more the thalamo-cortical system functions properly, the more the cortical response to the incoming sensorial stimulus will be large. Therefore, HFOs are a neurophysiological parameter of cortical responsiveness/excitability as they are large or reduced on the basis of the cortical excitability state. If the cortex is tonically excited [it is excessively pre-activated] the incoming stimulus will elicit phasic small responses due to excessive background noise; if the cortex is tonically resting [as the thalamo-cortical system works properly] the incoming stimulus will elicit phasic large responses. HFO strictly follow this trend.

We recently published a couple of papers 7-8 where we addressed habituation, recovery cycle, and HFO in typically developing children compared to healthy adults. Several differences emerged between developing children and adults, and all suggested physiological higher cortical excitability during the developing age. The technique appeared easily administrable to even small children, well tolerated, and time-saving [30-40 minutes for all recording sessions].

Many neurodevelopmental disorders are characterized by abnormal sensorial processing. For instance, autism spectrum disorders usually present with exaggerated behavioural responses to sensorial stimuli, spectrum of attention-deficit hyper-activity disorders present with abnormal modulation of sensorial information than can be either excessively determinant or irrelevant in guiding behaviour, developmental coordination disorder determines clumsy motor performance in children and the sensorial underpinnings of this condition is far from being fully understood.

So, which is the potential of wide administration of SEPs recording in neurodevelopmental disorder? It is conceivable that this technique – easy, economic, and well tolerated in children – can help in comprehending better both the pathophysiology of several clinical conditions and the potential targets of therapeutic strategies, pharmacological and rehabilitative. As far as this second goal is concerned in particular, we might aim at identifying potential targets of pharmacological modulation of the thalamo-cortical networks. Monoamines such as serotonin, beta-blockers such as propranolol, aminoacids such as glutamate are only few examples of molecules that are known to modify functioning of the above-mentioned cortical-subcortical network devoted to modulation of the sensorial influx to the cortex. Moreover, proper knowledge of the pathophysiology of sensory processing in neurodevelopmental disorders might help in identifying adequate sensorial experiences for atypically developing children [at school and at home – adequate materials, tasks, etc. – but also outdoor – sports, plays, etc.].

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In conclusion, we are facing potential significant expansion of our understandings of several neurodevelopmental clinical conditions if we will start systematic and wide administration of SEPs to address sensorial processing in atypically developing children. The data than we will collect will likely help in identifying all clinical subtypes of atypical sensory processing and better specific therapeutic approaches.

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