ADHD group and the Non-Treated Learning Disabled/ADHD control group ($p < 0.001$) and none between the Treated Learning Disabled/ADHD group and the normal control group ($p > 0.05$). (4) The topographic mappings of both components were similar at baseline and 8 months later in both control groups ($p > 0.05$). (5) At baseline, school-marks and Mangina-Test performance of Treated Learning Disabled/ADHD were not significantly different than those of the non-Treated Learning Disabled/ADHD ($p > 0.05$) and significantly lower than those of the normal control group ($p < 0.001$). (6) The Treated Learning Disabled/ADHD group in post-treatment condition had significantly higher school-marks and Mangina-Test performance than those of Non-Treated Learning Disabled/ADHD controls ($p < 0.001$) and were similar to those of normal controls 8 months later ($p > 0.05$). (7) School-marks and Mangina-Test performance at baseline for Non-Treated Learning Disabled/ADHD controls were not modified 8 months later ($p > 0.05$) and normal controls maintained their high performance within the same time interval ($p > 0.05$).

Our present results suggest that time and/or maturation *per se* did not play a role in improving ERP topography and cognition in Non-Treated Learning Disabled/ADHD controls. Rather, brain plasticity as expressed in ERP topographic mapping is suggested in the post-treatment improvement in Treated Learning Disabled/ADHD subjects (Mangina and Beuzeron-Mangina, 2004).

In concluding, our findings provide evidence of the impact of the psychophysiological treatment methodology on brain plasticity and regulation as reflected in significantly improved neurophysiology of prefrontal, frontal and posterior brain regions concomitantly with higher school-marks and neuropsychometric performance in the Mangina-Test.

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NEUROPSYCHOPHYSIOLOGICAL PROFILE OF CHILDREN WITH DEVELOPMENTAL DYSLEXIA

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This study describes the neuropsychophysiological profile of children with developmental dyslexia analysed with Reading-Related Potentials, the Mangina-Test (Mangina, 1994, 1998) and the Reading and Spelling Direct Test. Twenty-four normal children (mean age 9.27 ± 0.23 years.) and 20 dyslexic children (mean age 9.44 ± 0.4 years.) participated in the experiment. Reading-Related Potentials (RRPs) were recorded during two different reading-related tasks. The tasks consisted in reading aloud capital and small letters of the Italian alphabet. The letters appeared on a screen after an externally paced or a self-paced button press and lasted for 25 ms. Statistically significant differences of RRPs latency and amplitude were present in different cerebral areas according to task conditions. Furthermore, the RRPs recorded from dyslexic children showed an increase in latency and a reduction in amplitude as compared to controls. The RRPs components associated with the prelexical, lexical and postlexical periods were significantly correlated with the Reading Quotient of the Reading and Spelling Direct Test and the performance score of the Mangina-Test. The obtained differences in the RRPs morphology suggested that dyslexia is not only caused by perceptual deficits, but it is a more complex disorder that also involves defective higher-order cognitive functioning such as attention, phonological skills, verbal-motor coordination, feedback mechanisms and memory.

BRAIN OSCILLATIONS IN CHILD PSYCHIATRY: CONCEPTS AND APPLICATIONS

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Electrical brain oscillations have become a powerful tool to study central mechanisms of information processing. Following neurobiological maturation, the spontaneous electroencephalogram (EEG) undergoes substantial changes with development in children [1]. Event-related oscillations (EROs) depend on the development of the spontaneous EEG, but they are also associated with information processing in an age-specific way reflecting the maturation of functional networks [2–4].

To apply the concepts and methodology of brain oscillations in child psychiatry and to study psychopathophysiological mechanisms leading to deficits in motor behaviour of children with attention deficit hyperactivity disorder (ADHD), tic disorder (TD), and comorbid disorder (ADHD + TD). The major questions were the following: (1) Are there specific deficits in sensory and cognitive information processing in children with hypermotor syndroms? (2) Do the clinical symptoms originate from a maturational delay or do they result from brain dysfunctions? (3) Is comorbidity specific or additive at the level of psychophysiological investigation?

EEG was recorded during selective-attention and CPT tasks. New methods for analysis (Wavelet, neuronal networks) were applied [5]. Single-sweep phase synchronization was also assessed [6–7]. ERO measures were correlated with clinical, neuropsychological, and performance scores.

The spontaneous EEG activity distinguished the neurobiological background of ADHD and TD because different frequency ranges were affected by each disease. EROs indicated that the neurophysiological mechanisms of stimulus processing were altered in children with hyperactivity. That is, in ADHD, perception was deviant as reflected by larger phase-locked gamma EROs. Enhanced theta EROs appeared as a marker for hyperactivity, and they also distinguished comorbid from pure ADHD and TD. EROs from the delta–theta range disclosed deficits in sustained attention in ADHD as indexed by specific time-on-task effects.