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Evidence for a magnocellular disadvantage in early-onset schizophrenia patients: A source analysis of the N80 component visual evoked component.

Promotionsfach: Psychiatrie

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Visual impairments in schizophrenia are suggested to be partly caused by early processing deficits of the magnocellular pathway. This might include disturbed interactions between magnocellular and parvocellular pathways. It has been suggested that the influence of M- on P- pathway (M-priming) might be impaired, which probably affects the faster and detailed visual processing of stimuli. This study sought to test the hypothesis that the M-priming is impaired in schizophrenia. It was carried out studying patients with different ages of onset, a useful design to integrate information processing models and neurodevelopmental models of schizophrenia.

Nine stimulating conditions were used to isolate M- and P-pathways in a pattern reversal VEP paradigm. N80 generators were estimated using a method of source localization, Brain Electrical Source Analysis software (BESA). 40 schizophrenia patients (early onset=19; adult onset=21) were compared to age- and gender-matched healthy controls (early-onset controls=19; adult controls=21). Hypotheses were tested through a bootstrap resample procedure.

The bootstrap analysis yielded significant differences between early-onset schizophrenia patients and its controls. The former obtained reduced amplitudes in response to mixed M-/P-conditions, and normal amplitudes in response to isolated P- and M-biased stimulation. Concerning the latencies, significant differences were observed between adult-onset schizophrenia and adult controls, with prolonged values for adult schizophrenia patients.

The early VEP component N80 seems to be associated to M-priming and showed reduced amplitude in EOS but not in AOS. This points towards an M-priming deficit in EOS and is compatible with neurodevelopmental schizophrenia hypotheses, probably reflecting the asynchronies in the brain maturational abnormalities occurring at different age of illness onset. Future research providing additional evidence about the interactions between visual pathways and the M-priming should be conducted to better understand the mechanisms underlying visual schizophrenia impairments.