

An Electrophysiological Study of Cognitive and Emotion Processing in Type I Chiari Malformation

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Purpose

Type I Chiari malformation (CMI) is a neurological condition in which the cerebellar tonsils descend into the cervical spinal subarachnoid space resulting in cervico-medullary compression. Early case-control investigations have indicated cognitive deficits in the areas of attention, memory, processing speed, and visuospatial function. The present study further examined cognitive and emotional processing deficits associated with CMI using a dual-task paradigm.

Methods

Nineteen CMI patients were recruited during pre-surgical consultation and 19 matched control participants identified emotional expressions in separate single and asynchronous dual-task designs. To extend earlier behavioral studies of cognitive effects in CMI, we recorded event-related potentials (ERPs) in the dual-task design.

Results

Though response times were slower for CMI patients across the two tasks, behavioral and ERP analyses indicated that patients did not differ from matched controls in the ability to allocate attentional resources between the two tasks. P1 ERP component analyses provided no indication of an emotional arousal deficit in our CMI sample while P3 ERP component analyses suggested a CMI-related deficit in emotional regulation. P3 analysis also yielded evidence for a frontalization of neurophysiological activity in CMI patients. Pain and related depression and anxiety factors accounted for CMI deficits in single-task, but not dual-task, response times.

Conclusions

The present findings suggest that CMI manifests with specific, rather than general, attentional control deficits. While CMI patients did not experience deficits in cognitive control, response times and emotion regulation appear to be affected by CMI. Moreover, while single-task response time was no longer a predictor of CMI status after controlling for chronic pain and related factors, dual-task response time remained a significant predictor of CMI status, suggesting that the CMI-related deficits at least partially stem from CMI pathophysiology rather than chronic pain and related factors.

