Sensory and Sensorimotor Gating in Bipolar Disorder

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Abstract

Suppression of the P50 event-related brain potential (ERPs) and prepulse inhibition (PPI) are thought to play an important role in psychiatric and neurological disorders of the central nervous system, sensory and sensorimotor gating, respectively. Although both have received a great deal of activity in schizophrenia, less is known about sensory gating deficits in other patient groups. In the current study, sensory gating was assessed in 35 bipolar patients (23 mixed, 11 manic, and 1 undefined) and 32 healthy controls. The absence of domain data from the P50 paradigm demonstrated that the bipolar patients significantly attenuated SI and S2 amplitudes, and decreased SI latencies, compared to the controls, with no difference between patient subtypes. No differences in P50 sensory gating were found between patient and control groups. Furthermore, a principal components analysis (PCA) performed on both the low (0-20 Hz) and high (20-50 Hz) frequency bands of the P50 component showed that the mixed-episode patients significantly differed from the control patients for the low frequency components at both S1 and S2, but that both manic and mixed-episode subtypes evinced significantly lower levels of power at S1 for the frequency component. In the PPI paradigm, bipolar patients who were significantly reduced amplitudes to the pulse-alone (PA) and prepulse (PP) latencies and longer response latencies to the PP tones compared to the controls. With respect to subtype differences, significantly attenuated PA amplitudes and increased PP latencies were found in the mixed-episode patients compared to the controls, with manic patients showing comparable results to the latter two groups. However, despite reduced S2 amplitudes in the bipolar group, bipolar patients as a whole evinced significantly less PA suppression (PP/PA index) in comparison to the control group.

Background

A diminished ability to filter out, or “gate”, irrelevant stimuli is conceptualized as one of the defining features of schizophrenia; however, less is known about sensory gating in relation to other disorders. In one of the few auditory P50 event-related potential (ERP) studies conducted with acutely ill bipolar patients, it was found that the manic patients showed greater sensory gating deficits compared to those observed in schizophrenia patients (Frank & Kiefer, 1983). However, in contrast to healthy subjects, bipolar patients showed reduced gating in stable, subacute bipolar patients who were evaluated from a steady state, versus a state of active illness. Similarly, sensory gating deficits, as assessed by PPI, have also been found in medicated bipolar patients with acute psychotic symptomatology (Shetty et al., 2001). However, bipolar patients did not differ from schizophrenic patients on measures of PPI, the bipolar patients evidenced significantly less inhibition than control subjects. Furthermore, both acute and chronic psychotic patients showed lessened habituation when compared to the control group.

Subjects

32 healthy controls and 35 bipolar patients (23 mixed, 11 manic, and 1 undefined) were recruited from ongoing pharmacokinetic studies in which their medication status of 7 patients could not be determined due to previous and 1 undefined), 15 were on psychotropic medications (5 manic and 10 undefined) were recruited from ongoing pharmacokinetic studies in which the remainder of the patients were between 18-65 years of age. In the current study, the bipolar population consisted of 32 healthy controls and 35 bipolar patients (23 mixed, 11 manic, and 1 undefined) who were recruited from ongoing pharmacokinetic studies in which their medication status of 7 patients could not be determined due to previous and 1 undefined), 15 were on psychotropic medications (5 manic and 10 undefined). Sixty-nine percent of the patients were in the acute manic phase, whereas 16% were in the mixed phase. Twenty percent of patients did not have a definite diagnosis.

Procedure

The Auditory P50 and Acoustic Startle Latencies and Gating Analysis: 6.1 The Auditory P50 paradigm was used in order to assess sensory gating in the auditory modality. 6.1.1 The paradigm consisted of 140 trials, with an interstimulus interval of 900 ms presented every 7-10 s to create a total of 140 trials. 6.1.2 In order to create a total of 140 trials, participants were asked to press a button in response to “false” pairs of clicks that were randomly presented throughout the paradigm. 6.1.3 In order to characterize the P50 ERPs in the time domain at a single electrode site (Fz), power analyses were performed by decomposing the P50 waveform into a principal components analysis (PCA) method extending both low (0-20 Hz) and high (20-50 Hz) frequency bands in an attempt to characterize the sensory gating (0-20 Hz) and inhibition process (0-20 Hz) stages that are captured in the P50 component. 6.1.4 The preattentive and startle stimuli were presented to each participant through a set of three trials, which contained three blocks, with each block consisting of a randomly ordered series of trials. The three blocks were presented in the order of the preattentive and startle stimuli, and trials were presented every 50-60 s. 6.1.5 With an interstimulus interval of 500 ms, 9 of the bipolar patients were unmedicated at the time of testing. 6.1.6 Of the 9 bipolar patients, 7 were on monotherapy, 1 was on combination therapy, and 1 was on a combination of antidepressants and atypical antipsychotics. 6.1.7 Of the 11 manic patients who were unmedicated, 4 were on antipsychotic medications (1 manic and 3 mixed). 6.1.8 The medication status of 7 patients could not be determined due to previous and 1 undefined, of the 11 manic patients who were unmedicated, 4 were on antipsychotic medications (1 manic and 3 mixed). 6.1.9 The medication status of 7 patients could not be determined due to previous and 1 undefined.

Conclusions

The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders. The present study aimed to help clarify the relationship between sensory and sensorimotor gating deficits in bipolar disorder and comorbid psychiatric disorders.

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