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Event-Related Potentials in Response to Facial Affect Recognition in Patients with Schizophrenia

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In the identification of facial expressions related to certain emotions, certain parameters of event-related potentials (ERPs) can be interpreted as the respective indices. Patients with schizophrenia exhibit deficits in facial affect recognition at both behavioral and neural levels. We examined face expression-sensitive ERP components, N170 and N250, which are mostly related to encoding and decoding of the facial emotions, respectively, in 10 patients with schizophrenia and 10 healthy adults matched in age and gender. The mean amplitude and peak latency of the N170 and N250 waveforms, induced by visual stimuli in the form of happy, sad, fearful, and neutral face images, were measured and compared in these two groups. In the patient group, the mean amplitude of the N250 was higher in the right hemisphere (–1.402 mV on average) compared to that in the left hemisphere (–0.814 mV). The independent-sample *t*-test revealed that the N250 latencies were significantly longer in the patient group compared to those in the normal group for all four emotions. We, however, find no significant differences in the mean amplitude and peak latency of the N170 waveform between the two groups. Our study shows that decoding of the facial expressions in patients with schizophrenia is clearly impaired, which may contribute to cognitive and behavioral symptoms of the disease.

Keywords: schizophrenia, face expression, facial affects, event-related potentials (ERPs), N170 and N250 components.

INTRODUCTION

Event-related potentials, or ERPs, are scalp-recorded voltage fluctuations generated in the brain in response to specific events [1]. The ERPs reflect summated activity of postsynaptic potentials generated mostly due to synchronous potential changes in cortical neurons as a direct result of specific sensory, cognitive, or motor events. Despite modern neuroimaging methods, ERPs are being used extensively to study neural processes in response to various stimuli and to test the theories of attention and perception due to their rather high temporal resolution [2]. Different visually induced ERP waveforms include positive components, namely P50, P200, and P300, and negative components, N100, N170, N200, N250, and N300. The ERP components are interpreted based on their amplitudes and latency in response to specific stimuli to determine the intensity of the responses and the time taken for their development, respectively [2]. Shorter latencies are associated with better mental performance, and higher amplitudes of the waveforms are associated with greater attention to a stimulus. As is believed, significantly longer latencies and reduced amplitudes indicate neurobiological abnormalities. Abnormalities in the ERP components have been seen under various neurological conditions, such as dementia, Parkinson's disease, multiple sclerosis, and certain psychiatric disorders including obsessive-compulsive disorder and schizophrenia.

Schizophrenia is a chronic debilitating mental disorder that causes disruptions in cognition, perception, and emotions and is characterized by the manifestation of certain symptoms, such as psychosis, auditory hallucinations, and delusions [3]. Patients with schizophrenia exhibit behavioral alterations, such as impairments in identifying feelings displayed by others through facial expressions and the inability to recognize a familiar face. Such patients have also been found to regulate their own emotions ineffectively. These impairments

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in behavior have been attributed to neurocognitive dysfunctions, which contribute to the cognitive symptoms of the disease [4, 5]. These changes result in poor social functioning, inadequate interpersonal relationships, occupational outcomes, etc., and thus significantly affect the quality of life of the patients [6].

Studies on schizophrenia have reported abnormalities in synchronized oscillatory activity of neurons, which are considered to result in impaired neural activation and temporal coding, which leads to neurocognitive dysfunction [7]. It is, however, debatable as to what extent the emotion recognition deficits represent a generalized or specific form of cognitive impairment in the disorder [8]. The difficulties faced by patients in understanding emotional events can be estimated with psychophysiological, behavioral, and brain imaging studies.

Event-related potentials have been used at present to understand facial emotion perception deficits in schizophrenia since these deficits are closely related to the clinical symptoms of the disease, such as the flat affect, inappropriate affect, and depression [9]. The face expression-sensitive ERP component N170 denotes the time course for structural encoding of the facial features, while N250 is believed to mostly reflect decoding of facial emotions [10]. It was reported that the N170 amplitude is lower in patients with schizophrenia [11, 12]. Schneider et al. [13] observed that patients were unable to discriminate between happy and sad facial expressions and falsely identified the target emotion to be sad; they also showed impairments in identifying happy facial expressions, as compared to healthy individuals [13]. Another study [14] revealed that the N170 amplitude was substantially higher for fearful faces, which appeared in a fearful background, as compared to cases where the subjects were presented with neutral or happy face expressions. These studies suggested that the impairments in emotion recognition in the patients are due to deficits in the early stage of emotion processing, which involves structural encoding of the facial features.

As to the relationship of the N250 to facial emotion recognition in patients with schizophrenia, it should be recognized that the data are contradictory from a few aspects. Mild alterations have been reported in the N250 response in patients, suggesting that this disease exerts a negligible effect on structural encoding of faces [15]. Tamminga et al. [16] observed that the N170 component was normal, while the N250 component was found to be reduced in patients suffering from schizophrenia, suggesting that the impairments in recognition of facial emotions are due to specific deficits in decoding of the facial expressions and not to some deficits in facial feature encoding [16].

The area of interest of our study was examination of the face expression-sensitive ERP components, namely the N170 and N250 waves, in response to facial affect recognition in patients with schizophrenia and healthy participants. Our study aimed to compare the amplitude and latency of these two waveforms between patients with schizophrenia and healthy individuals so as to elucidate what stage of facial affect processing is impaired in patients with schizophrenia.

METHODS

Participants. The study was conducted in the Kasturba Medical College, Manipal, India in the Departments of Physiology and Psychiatry in collaboration with the Department of Speech and Hearing (School of Allied Health Sciences, Manipal). The study sampling consisted of 10 patients with schizophrenia and 10 healthy participants. The participants were between 18 and 55 years of age, and control subjects were matched in age and gender. Patients with schizophrenia, diagnosed according to the DSM-IV (Diagnostic and Statistical Manual of Mental Disorders), were recruited from the Department of Psychiatry, Kasturba Hospital, Manipal. All participants were right-handed. The patients received stable doses of generally used psychotropic drugs during the past six weeks, with no history of hospitalization within the last three months. Possible participations were excluded if they met criteria for the presence of other co-morbid psychiatric disorders and if they received electroconvulsive therapy within the last six months. Healthy participants were volunteers. They were included in the study if they had a General Health Questionnaire score below 1. Possible controls were excluded if they met the criteria for any lifetime psychiatric disorder or if they reported a psychiatric disorder in any of the first-degree relatives. Additional exclusion criteria for both groups included substance abuse (except nicotine), significant visual impairment, or any other neurological disorder and also significant extrapyramidal symptoms interfering with the task

performance. Pregnant women or those in the post-partum state were not included in the study.

ERP Paradigm. All participants performed two different emotion identification tasks while viewing colored pictures of faces with four different facial expressions (based on a procedure developed previously) [10]. The participants were seated comfortably in a chair with armrests, in a quiet dark room, at a distance of 1 m from the computer screen. The pictures of faces displaying emotions for the facial affect recognition task were taken from the Karolinska directed emotional faces (KDEF) stimulus set [17]. The pictures were cropped into an oval frame. External features, such as hair, were also cropped from the images. The cropped images were placed against a uniform black background. The pictures were equalized for their contrast and brightness levels. The participants were asked to identify the emotion of the faces displayed (happy, neutral, fear, or sad) in two separate blocks, in a fixed block order. Each block contained 64 images. The first block contained faces with happy and neutral emotions, while the second block consisted of faces with sad and fearful emotions. Each emotion was repeated four times in a randomized order within the block. Only one image was displayed at a time. The participants were asked to identify the facial emotions using a mouse click response. The presentation of each block began with a blank screen presented for 500 msec. The duration of presentation of the visual stimuli, i.e., the images, was 100 msec with a 1500-msec response window within an interstimulus interval of 2000 msec. Simultaneously, the EEG was recorded. The STIM2s module GENTASK (Compumedics Neuroscan, USA) was used for high-quality stimulus presentation.

EEG Recording. Recordings of the EEGs were acquired with a 32-channel electrode cap (Eazy-Cap) and a SynAmps 2tm amplifier (Compumedics Neuroscan, USA). Impedances of the EEG electrodes were kept below 5 kW. To monitor the eye blinks and other ocular movements independently, additional electrodes were placed in the supraorbital and infraorbital regions. The offline data processing was carried out using EEGLAB version 14.1.1b and MATLAB version 7.10.0.499 [18]. The data were sampled at 1000 sec^{-1}, visually inspected for artefacts, and the latter were removed by rejection. Interpolation was then used to recreate the electrode in EEGLAB [18]. This was followed by FIR filtering of the data with a bandpass of 1 to 30 Hz. The data were epoched from –200 to 800 msec relative to the stimulus onset. Independent component analysis (ICA) and artefact rejection using MARA (multiple artefact rejection algorithm) were done.

Data Analysis. The N170 and N250 waveforms were examined using data collected from 12 out of 32 electrode sites. The electrodes were chosen based on the peak observed amplitudes of the waveforms within a specified time window. The N170 waveform was examined from the averaged data collected at electrode sites P3 and P4 representing the activity in the left and right cerebral hemispheres, respectively. The electrode data from P3, P7, and O1 were averaged with respect to P3, and the data from P4, P8, and O2 were averaged to P4. Thus, the data for the N170 were obtained from the occipital and parietal sites. The N250 waveform was examined from the averaged data collected at electrode sites Fc3 and Fc4, which also represent the activity in the left and right cerebral hemispheres, respectively. The electrode data from Fc3, F3, and C3 were averaged with respect to Fc3, and those from Fc4, F4, and C4 were averaged to Fc4. Thus, the data for the N250 corresponded to the frontal and central electrode sites. A time window of activity of each ERP component was defined separately for normal subjects and for patients, to ensure the coverage of each component based on the peak activity observed by inspection of the averaged ERPs. For normal subjects, the time window was 135 to 175 msec for the N170 and 200 to 250 msec for the N250 ERP component. In patients with schizophrenia, the expected time window was 150 to180 msec for the N170 and 220 to 280 msec for the N250 ERP component. The mean amplitude and peak latency (latency at the highest amplitude within the latency ranges for each component described above) of each ERP component was analyzed separately for each group and then compared.

For each ERP component, a 2×1×2 (group, patients *vs*. controls; task, emotion identification, and hemisphere, right *vs*. left, respectively) repeated-measures analysis of variance (ANOVA) was run. The Greenhouse–Geisser corrections (e) were used in the repeated-measures ANOVA that contained more than one degree of freedom to correct for violations of sphericity. In addition to ANOVA, an independent-sample *t*-test was run to analyze the significance of differences. Numerical values are presented as means ± s.d. Statistical analysis was done in SPSS 15, and a P value of 0.05 was used to determine significant intergroup differences.

RESULTS

Ten healthy participants (8 men and 2 women) and ten patients with schizophrenia (also 8 men and 2 women) participated in this study. The mean N170 and N250 amplitudes and latencies for all tasks in patients with schizophrenia and normal subjects are shown in Table 1. The grand average N170 and N250 waveforms for normal participants and patients with schizophrenia, which were recorded in emotion identification tasks, are presented in Figs. 1 and 2, respectively. The peak N250 latency for various emotions in the right and left hemispheres in normal participants and patients with schizophrenia are shown in Fig. 3.

N170 Mean Amplitude. A 2 (group) × 1(task) × 2 (hemisphere) repeated-measures ANOVA on the N170 amplitude did not reveal a significant main effect of the task (F [3,54] = 1.049, P = 0.378) or hemisphere (F [1,18] = 0.076, P = 0.786). The test, however, revealed a significant main effect for the group (F [1,18] = 5.040, P = 0.038), but the group × task interaction (F [3,54] = 1.733, P = 0.171) and group × hemisphere interaction (F [1,18] = 1.424, P = 0.248) and also other interactions did not reach the significance level.

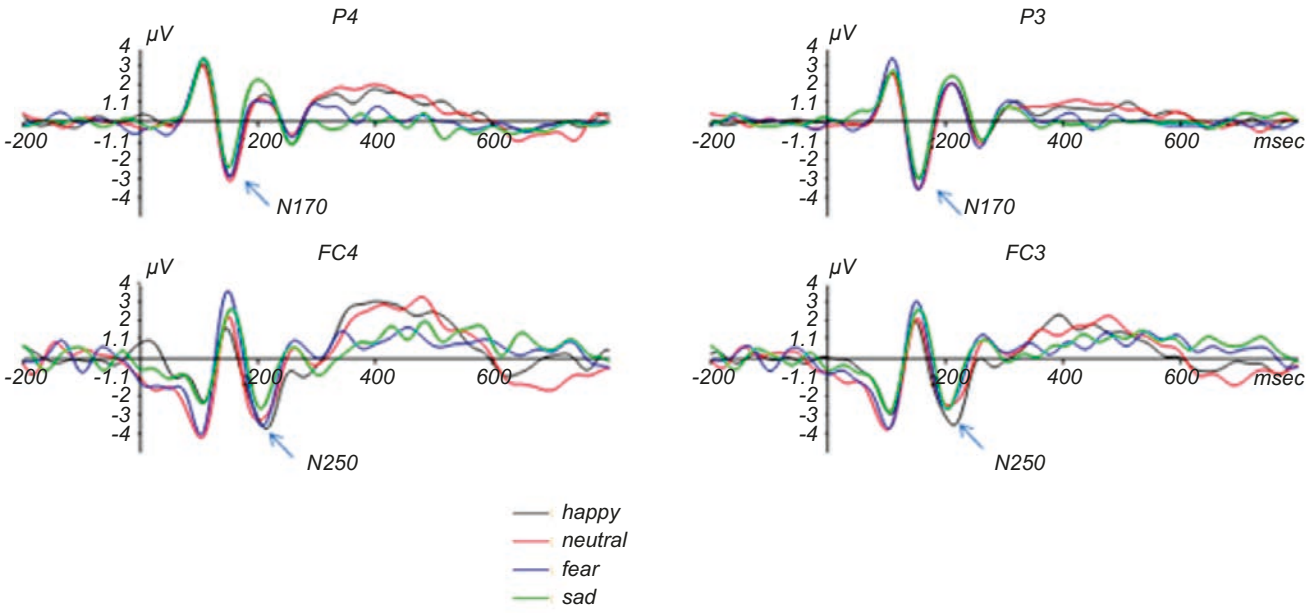
T a b l e 1. Mean N170 and N250 Amplitude and Latency for Patients with Schizophrenia and Normal Subjects, Collapsed Across Tasks

ERP component	Normal (n = 10)	Schizophrenia patients (n = 10)
N170 amplitude, μV	–2.07 ± 0.42	–0.743 ± 0.42
N170 latency, msec	153.86 ± 3.22	157.41 ± 3.22
N250 amplitude, μV	–2.974 ± 0.514	–1.108 ± 0.514
N250 latency, msec	212.12 ± 3.31	233.93 ± 3.31

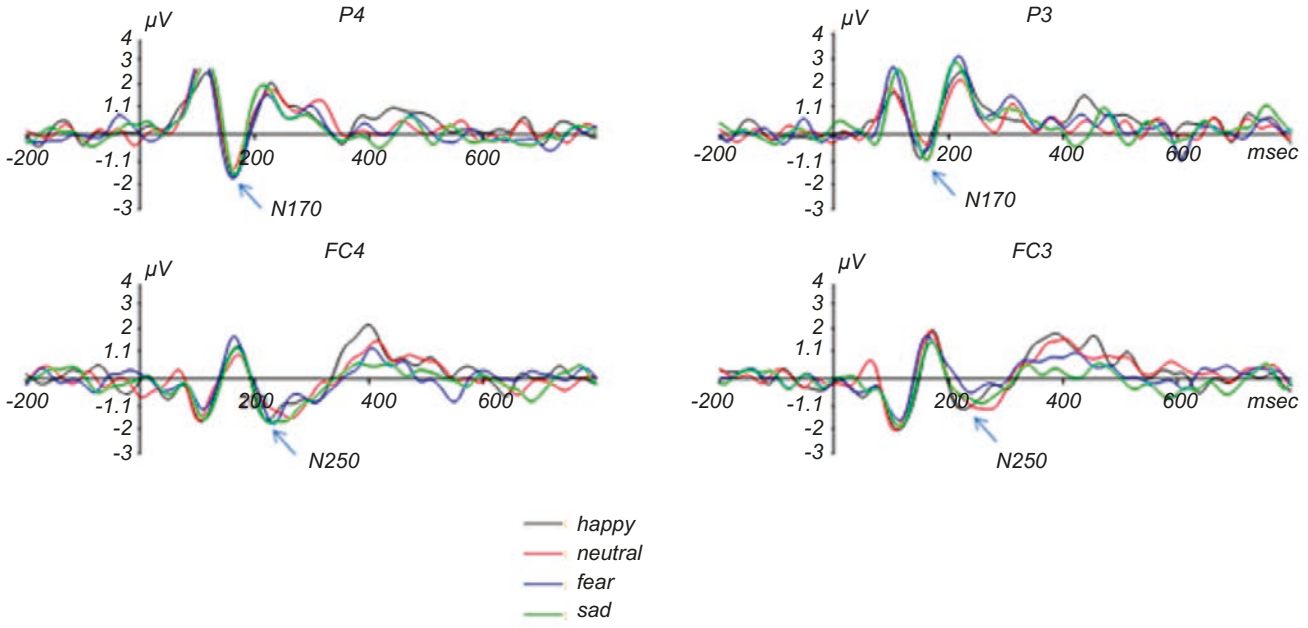
F o o t n o t e. Mean ± standard deviation values are shown.

N170 Peak Latency. A 2 (group) × 1(task) × 2 (hemisphere) repeated-measures ANOVA on the N170 latency did not reveal any significant main effect for the group (F [1,18] = 5.607, P = 0.446), or task, or hemisphere. There was no significant interaction in comparisons of the effects.

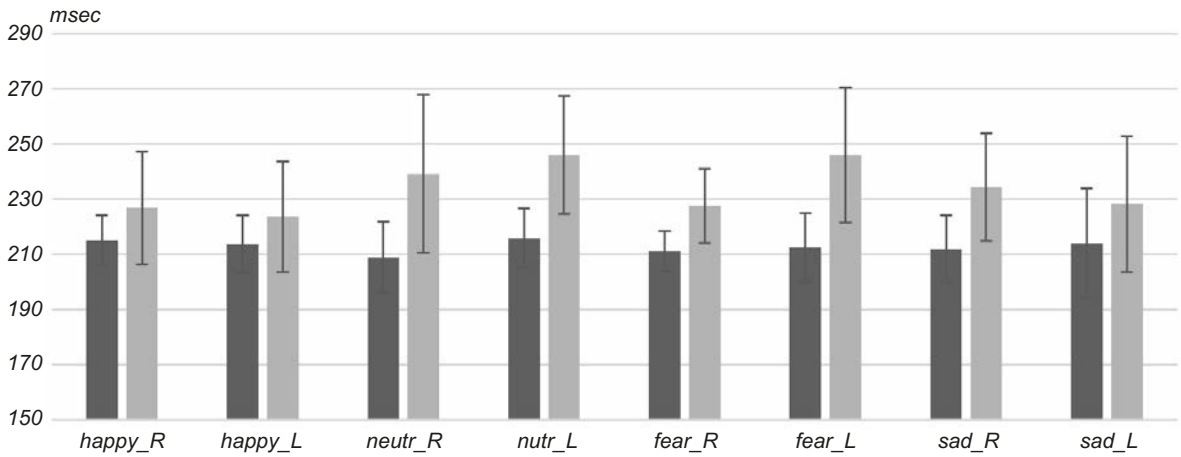
N250 Mean Amplitude. A 2 (group) × 1(task) × 2 (hemisphere) repeated-measures ANOVA on the N250 amplitude did not reveal a significant main effect of the task or hemisphere. The test, however, revealed the existence of a significant main effect for the group (F [1,18] = 6.061, P = 0.024), but the group × task interaction and group × hemisphere interactions were not significant. However, the task × × hemisphere interaction was significant (F [3,54] = = 1.171, P = 0.009). Thus, we have run two repeated-measures on hemisphere × task. Interaction between the hemisphere and task effects in the normal group did not reach the significance level (while was rather close to the latter; F [3,27] = = 1.037, P = 0.080). However, there was a significant interaction between the effects of the hemisphere and task in the patient group (F [3,27] = 0.525,



F i g. 1. Grand average of the N170 and N250 components of the ERPs recorded in the right (P4/FC4) and left cerebral hemispheres (P3/FC3) in response to presentations of happy, neutral, fearful, and sad face images in the normal group.



F i g. 2. Grand average of the N170 and N250 components of the ERPs recorded in the right (P4/FC4) and left cerebral hemispheres (P3/FC3) in response to presentations of happy, neutral, fearful, and sad face images in the patient group.



F i g. 3. Diagrams of the peak N250 latency (msec) in normal and patient groups (light and dark columns, respectively) for various facial emotions; recordings from the right (R) and left (L) hemispheres. Means ± s.d. are shown.

P = 0.019), wherein the mean amplitude of N250 was significantly higher in the right hemisphere (–1.402 mV) compared to that in the left hemisphere (–0.814 mV).

N250 Peak Latency. A 2 (group) × 1(task) × 2 (hemisphere) repeated-measures ANOVA on the N250 latency revealed a significant main effect for the group (F [1,18] = 19.934, $P < 0.001$) and a significant interaction for the task × group (F [3,54] = 3.182, P = 0.031). The independent-sample t-test was then done to further explore the significance of this interaction. There was a significant difference between the N250 latency for happy face expression in the normal (M = 214.3 msec, s.d. = 9.65 msec) and patient group (M = 225.20, s.d. = 19.74; t (38) = = 2.22, P = 0.033). This was also found for the neutral face in the normal (M = 212.3, s.d. = 12.19) and patient group (M = 242.55, s.d. = 24.91; t (38) = 4.88, P = 0.000). There was a significant difference for the fear-expression face in the normal (M = 211.7, s.d. = 9.99) and patient group (M = 236.75, s.d. = 21.46); t (38) = 4.74, P = 0.000). Finally, the respective figures for the sad face, were shorter in the normal group (M = 212.95, s.d. = 16.08) than in

the patient group (M = 231.25, s.d. = 21.87; t (38) =3.02, P = 0.005). These results suggest that the N250 latency is significantly longer in the patient group compared to that in the normal group for all four emotions.

DISCUSSION

In our study, we compared the facial affect recognition abilities between patients with schizophrenia and normal participants. Patients showed an abnormal response concerning the N250 waveform. The latter is the ERP component sensitive to the decoding of facial emotions. At the same time, parameters of the N170, an ERP component, which is sensitive to structural encoding and visual processing of facial features, did not differ significantly from the norm in schizophrenia patients.

N170 Waveform. N170 deficits in schizophrenia during facial affect recognition tasks have been reported previously in some studies [19, 20]. We, however, found that the mean amplitude and latency of the N170 waveform were nearly unchanged in the patients. This finding is consistent with that of Streit at al. [21]. The difference in our results concerning the N170 waveform in the patient group could be due to a difference in the stimulus paradigm or method of analysis, compared to those in other studies where considerable modifications of N170 were found [21]. Some studies have used expressions of only one facial emotion to examine the N170 waveform [11], but we have used four different facial emotions as visual stimuli. Another methodological difference was that previous studies used cognitive oddball tasks, while we have used simple facial emotion identification tasks [22]. A more demanding cognitive nature of such tasks may result in a reduced N170 component in the patients. Certain other studies have used tasks that involve the recognition of familiar *vs*. unfamiliar faces, apart from emotions [23]. Our study focused on the emotion recognition using unfamiliar faces displaying emotions. A difference in the analysis methods used may have also contributed to the difference in the results. Turetsky et al. [15] demonstrated that patients exhibited smaller N170 responses in terms of the amplitude, but these authors examined the global field power, while we measured the mean amplitude. We also utilized different latency windows in examination of our waveforms compared to the studies that found N170 deficits [23].

While many studies lend support to the notion that patients show smaller N170 amplitudes, there are some reports that highlight conflicting findings [24, 25]. Our findings, along with such research, indicated that a deeper understanding is necessary on how the N170 waveform is affected in schizophrenia and what type of the stimulus parameter (in terms of the stimulus duration and specific emotions) has an influence on the N170 waveform in schizophrenia.

N250 Waveform. The main finding of the current study is that we found significant differences in the N250 ERP component in patients with schizophrenia compared to the normal participants. The patients exhibited a longer mean N250 latency compared to the normal group for all four emotional stimuli, i.e., happy, neutral, fear, and sad. The N250 amplitudes in the patient group were noticeably lower than those in the normal participants, but the differences did not reach the significance level. We, however, observed that the amplitude of the waveform was significantly higher in the right hemisphere compared to that in the left hemisphere in the patient group, but such hemispherical asymmetry was not seen in the normal participants. The difference between the normal and patient group was similar to the findings of Frommann et al. [26] who noted a significant variation in the N250 waveform between the two groups, when responses to emotional stimuli were analyzed [26]. Wynn et al. [27] also found similar results related to the N250 waveform.

These findings taken together suggest that schizophrenia considerably affects decoding of facial features, which forms a part of the later stages of facial affect processing. This hypothesis is supported by the fact that the N250 waveform is linked to more advanced stages of facial affect processing. Another study by Wynn et al. [10] predicted a similar result, wherein it was postulated that both encoding and decoding of facial features are adversely affected.

In examination of the patient responses to specific emotion manifestations, the longest latency was observed in response to neutral facial expressions in both hemispheres and for fearful facial expressions in the left hemisphere, while the shortest latency was observed in response to happy facial expressions in both hemispheres. This suggests that the patient group experienced certain difficulties in the decoding processes related to neutral and fearful facial emotions. A study by Leitman et al.

[28] examined processing abnormalities related to fear in schizophrenia. The authors suggested that schizophrenia impacts some specific social outcomes, especially those that are linked to danger and risk [28]. In this context, it becomes harder for patients to identify fear, which is crucial for them to be able to avoid dangerous situations. Based on these findings, the diagnostic role of the N250 can be further evaluated.

Our results also revealed a hemispherical difference in the N250 amplitude in the patient group, wherein the amplitude of this component in the right hemisphere was significantly higher for all emotions compared to the amplitude in the left hemisphere. This difference was not found in the normal participants. Several clinical studies suggested that the right hemisphere of the brain plays a crucial role in the recognition of human emotions, and that the right temporal region mostly mediates identification of facial expressions [22]. Some studies have also shown that schizophrenia causes impairments in the left hemisphere, which results in impaired recognition of emotions in patients. One such study (by Gur et al. [29)] reported that left amygdala activation in the patients, when asked to differentiate between positive and negative emotions, is weaker [29]. Tanaka et al. [30] have established the N250 response to be indicative of memory-related processes in patients. This study reported an increased sensitivity of the N250 in patients only in responses to known faces [30]. Such a differential role played by the cerebral hemispheres in eliciting responses in patients has been correlated to various positive and negative symptoms of schizophrenia. Thus, further research must be conducted in this area of ERP studies.

It is obvious that our study had several obvious limitations. The sample size was rather limited, and the patient population consisted of individuals who exhibited different symptoms, both positive and negative. This circumstance may noticeably influence their ability to recognize facial emotions. In addition to this, the patients were on medication during the study, and it is possible that some differences in medication could affect their cognitive abilities. Further research should be done to analyze the effect of medication on emotion perception and corresponding changes in ERPs, which may help in evaluation of the prognosis.

Schizophrenia is a psychiatric disorder that impairs the ability of the affected individuals to interpret and respond to various emotional cues, which in turn can impair their ease of functioning in society. An increasing need for improvement of the diagnostic methods has led to the study of ERPs in patients with schizophrenia. In our study on facial emotion recognition, we focused on the analysis of the amplitude and latency of two face expression-sensitive ERP components, namely the N170 and N250. These waveforms are believed to be linked to encoding and decoding of facial features, respectively. We observed certain deficits in the N250 waveform in terms of longer latencies in the patients and hemispherical differences in the N250 amplitude in this examined group, suggesting that the deficit is mostly related to the process of decoding of the facial features. The ERP components are possible electrophysiological markers of the underlying facial emotion deficits in schizophrenia and may contribute to the diagnosis, as well as to evaluation of the prognosis in patients with this mental disorder.

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All participants gave written informed consent after receiving a detailed explanation of the study, in a language that they understood, in accordance with the procedures approved by the Institutional Ethical Committee at the Manipal Academy of Higher Education: IEC number 498/2015.

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