Abnormalities in the processing of emotional prosody from single words in schizophrenia

Ana P. Pinheiro [a,b], Neguine Rezaii [b], Andréia Rauber [c], Taosheng Liu [d], Paul G. Nestor [e], Robert W. McCarley [b], Óscar F. Gonçalves [a], Margaret A. Niznikiewicz [b]

[a] Neuropsychophysiology Lab, CIPsi, School of Psychology, University of Minho, Braga, Portugal
[b] Clinical Neuroscience Division, Laboratory of Neuroscience, Department of Psychiatry, Boston VA Healthcare System, Brockton Division and Harvard Medical School, Brockton, MA, United States
[c] Catholic University of Pelotas, Pelotas, Brazil
[d] Department of Psychology, Second Military Medical University (SMMU), Shanghai, China
[e] University of Massachusetts, Boston, MA, United States

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ABSTRACT

Background: Abnormalities in emotional prosody processing have been consistently reported in schizophrenia and are related to poor social outcomes. However, the role of stimulus complexity in abnormal emotional prosody processing is still unclear.

Method: We recorded event-related potentials in 16 patients with chronic schizophrenia and 16 healthy controls to investigate: 1) the temporal course of emotional prosody processing; and 2) the relative contribution of prosodic and semantic cues in emotional prosody processing. Stimuli were prosodic single words presented in two conditions: with intelligible (semantic content condition—SCC) and unintelligible semantic content (pure prosody condition—PPC).

Results: Relative to healthy controls, schizophrenia patients showed reduced P50 for happy PPC words, and reduced N100 for both neutral and emotional SCC words and for neutral PPC stimuli. Also, increased P200 was observed in schizophrenia for happy prosody in SCC only. Behavioral results revealed higher error rates in schizophrenia for angry prosody in SCC and for happy prosody in PPC.

Conclusions: Together, these data further demonstrate the interactions between abnormal sensory processes and higher-order processes in bringing about emotional prosody processing dysfunction in schizophrenia. They further suggest that impaired emotional prosody processing is dependent on stimulus complexity.

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1. Introduction

Among the most significant predictors of long-term disability in schizophrenia (e.g., Couture et al., 2006) is impaired detection and recognition of emotions from voice, i.e., emotional prosody [EP]. Affect recognition from both voice and face is an aspect of social cognition, which has been recently recognized as an important predictor of functional outcomes at all stages of schizophrenia pathology: clinical high risk (Addington et al., 2008; Green et al., 2012), first episode (Horan et al., 2012) and chronic schizophrenia (Kee et al., 2003; Kucharska-Pietura et al., 2005; Green et al., 2012). While face processing abnormality in schizophrenia has been well characterized (e.g., Li et al., 2010), voice and prosody processing have been understudied, especially using event-related potential (ERP) approaches, which remain the only tool to examine temporal changes in neurophysiological events that correspond to early stages of analysis of a speech signal. The existing studies on vocal emotional processing include just a handful of behavioral (e.g., Edwards et al., 2001), functional magnetic resonance imaging (fMRI—e.g., Mitchell et al., 2004; Leitman et al., 2011) and ERP investigations (Pinheiro et al., 2012).

In healthy subjects, perception of emotional prosody is thought to reflect three interacting stages: 1) sensory processing of a speech signal; 2) implicit categorization of salient acoustic features into emotional and non-emotional features; and 3) explicit evaluation and assignment of emotional meaning to a speech signal (Schirmer and Kotz, 2006; Paulmann and Kotz, 2008; Paulmann et al., 2010). Event-related potential (ERP) studies demonstrated that the first two stages are indexed by N100 and P200, respectively (Paulmann and Kotz, 2008; Paulmann et al., 2010; Pinheiro et al., 2012).

Despite the importance of a detailed understanding of emotional prosody processing deficits in schizophrenia, few studies have examined these abnormalities and their underlying neural mechanisms are not well understood. Recent studies suggested that sensory-based dysfunction might not exclusively account for abnormal prosody processing in schizophrenia. Instead, an interaction between dysfunctional sensory and higher-order cognitive processes may better explain it (Leitman et al., 2010, 2011; Pinheiro et al., 2012). A recent ERP study provided further evidence for these abnormalities (Pinheiro et al., 2012). This study investigated prosody processing in 15 chronic schizophrenia patients and 15 healthy controls (HC). Additionally, it explored

the relative contributions of prosodic and semantic cues. Stimuli were prosodic sentences with intelligible (semantic content condition—SCC) and unintelligible semantic content (pure prosody condition—PPC). The ERP effects occurred within the first 200 ms from the sentence onset in both groups (Pinheiro et al., 2012), supporting previous studies' results (Paulmann and Kotz, 2008; Paulmann et al., 2010). The results revealed abnormalities in the three stages of prosody processing in schizophrenia, which were more pronounced for prosodic SCC sentences. Less negative N100 suggested abnormal sensory processing of prosodic SCC sentences irrespective of valence. Increased P200 to angry and happy prosodic stimuli in the SCC, and to happy stimuli in the PPC suggested abnormal detection of emotional salience. Behavioral results revealed impaired cognitive evaluation of the emotional significance of angry SCC and neutral PPC sentences.

In view of a critical need for a systematic study of emotional prosody processing in schizophrenia, the current study extended our previous work, by investigating the temporal course of prosody processing using *single words* with both intelligible (SCC) and unintelligible semantic content (PPC). Based on language studies demonstrating differences in the processing of words in a sentence vs. in isolation (e.g., Van Petten, 1995) and effects of phrasal length and complexity on prosodic processing (Wheeldon and Lahiri, 1997; Krivokapi, 2007), we reasoned that prosody processing of sentences may differ from that of single words. For example, the processing of words embedded in a sentence is susceptible to syntactic and semantic constraints imposed by a sentence context, which can modify many aspects of their processing (e.g., Van Petten, 1995). Furthermore, in relation to words in isolation, the processing of a sentence demands more working memory and attention resources, as meaning is built up across the course of the sentence (e.g., Van Petten, 1995). Thus, considering the attentional (Nestor et al., 2001; Laurens et al., 2005) and verbal working memory deficits (Menon et al., 2001; Silver et al., 2003) often reported in schizophrenia, the processing of prosodic information may be more impaired in sentences than in single words.

Because of its excellent temporal resolution, we used ERPs to address the role of stimulus complexity in the first two stages of emotional prosody processing: the sensory processing of prosodic information (N100) and the detection of its emotional salience (P200), both processes not accessible to behavioral probes. We also collected data on accuracy of prosody recognition to shed light on a later stage of emotional prosody processing, i.e. the assignment of emotional meaning to a voice signal. We hypothesized that if impaired prosody processing is not dependent on stimulus complexity, similar abnormalities to those reported in Pinheiro et al. (2012) will be observed in the current study. However, if stimulus complexity matters, we expected less severe prosody processing abnormalities in the single word relative to the sentence prosody processing study.

Considering previous studies demonstrating an association between deficits in emotional prosody recognition and positive symptomatology (Poole et al., 2000; Rossell and Boundy, 2005; Shea et al., 2007), and between increased P200 amplitude for happy prosody and delusions (Pinheiro et al., 2012), we predicted that ERP abnormalities amplitude would be associated with positive symptomatology scores.

2. Method

2.1. Participants

Sixteen patients with a diagnosis of chronic schizophrenia and 17 HC matched for age, handedness and parental socioeconomic status (Hollingshead, 1976) participated in this study (Table 1). Subjects had normal hearing as assessed by audiometry, and normal or corrected to normal vision. Patients were recruited at the Veterans Affairs Hospital, Brockton and HC were recruited from Internet advertisements.

The inclusion criteria were: English as first language; right handedness (Oldfield, 1971); no history of neurological illness; no history of

Table 1
Demographic and clinical characteristics of participants.

Variable	Healthy controls (n = 17)	Schizophrenia patients (n = 16)	p value[a]
Age (years)	48.13 ± 5.66	48.86 ± 7.40	.750
Women, *n*	7	5	
Education (years)	15.18 ± 1.64	14.00 ± 2.42	.119
Subject's SES[b]	2.13 ± 0.81	2.93 ± 1.14	.033[*]
Parental SES	2.44 ± 0.81	2.79 ± 1.53	.434
Handedness[c]	0.81 ± 0.15	0.79 ± 0.21	.848
Neurocognitive data			
Full scale composite score	99.33 ± 12.30	92.79 ± 14.32	.227
Verbal comprehension composite score	99.08 ± 11.47	95.93 ± 15.82	.572
Working memory composite score	105.33 ± 14.22	92.86 ± 12.90	.049[*]
Processing speed composite score	101.17 ± 89.64	89.64 ± 14.87	.107
Clinical data			
Onset age (years)	NA	30.07 ± 11.23	NA
Duration (years)	NA	19.47 ± 10.95	NA
Chlorpromazine EQ (mg)	NA	356.78 ± 294.56	NA
Antipsychotic medication type	NA	Typical (fluphenazine decanoate, proloxin decanoate, haloperidol) = 3; Atypical (risperidone, olanzapine, ziprasidone, quetiapine, aripiprazole) = 11	NA
Other psychotropic medication	NA	Antidepressants (sertraline, citalopram, bupoprion, trazodone) = 4 Benzodiazepines (lorazepam, clonazepam) = 4 Lithium carbonate = 2 Valproic acid = 3	NA
PANSS delusions	NA	4.88 ± 2.16	NA
PANSS conceptual disorganization	NA	2.50 ± 1.10	NA
PANSS hallucinations	NA	4.00 ± 2.19	NA
PANSS positive scale	NA	20.25 ± 8.19	NA
PANSS negative scale	NA	22.88 ± 9.76	NA
PANSS general psychopathology	NA	38.56 ± 11.70	NA
PANSS total psychopathology	NA	81.69 ± 25.92	NA
SANS total	NA	10.59 ± 5.44	NA
SAPS total	NA	9.63 ± 3.05	NA

Notes. All values represent mean ± SD. SES = socioeconomic status; Chlorpromazine EQ = chlorpromazine equivalent dose; NA = not applicable.
[a] Independent samples *t*-test tested for group differences in sociodemographic and neurocognitive measures.
[b] Hollingshead Four-Factor Index of Social Status (Hollingshead, 1976).
[c] Edinburgh Handedness Inventory (Oldfield, 1971).
[*] *p* < 0.05.

DSM-IV diagnosis of drug or alcohol abuse (APA, 2000) in the last year prior to EEG assessment; full scale intelligence quotient (IQ) above 85 (Wechsler, 2008); no hearing, vision or upper body impairment. For HC, additional inclusion criteria were: no history of Axis I–II disorders (First et al., 1995, 2002); no history of Axis I disorder in first or second-degree relatives (Andreasen et al., 1977).

Patients were diagnosed (screened for HC) using the SCID-I and SCID-II (First et al., 1995, 2002). Symptom severity was assessed with the *Positive and Negative Syndrome Scale* (PANSS-Kay et al., 1987), the *Scale for the Assessment of Negative Symptoms* (SANS-Andreasen, 1983) and the *Scale for the Assessment of Positive Symptoms* (SAPS-Andreasen, 1984) (Table 1).

All participants had the procedures fully explained to them and read and signed an informed consent form.