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Research Report

Comparative processing of emotional prosody and semantics following basal ganglia infarcts: ERP evidence of selective impairments for disgust and fear[☆]

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ABSTRACT

There is evidence from neuroimaging and clinical studies that functionally link the basal ganglia to emotional speech processes. However, in most previous studies, explicit tasks were administered. Thus, the underlying mechanisms substantiating emotional speech are not separated from possibly process-related task effects. Therefore, the current study tested emotional speech processing in an event-related potential (ERP) experiment using an implicit emotional processing task (probe verification). The interactive time course of emotional prosody in the context of emotional semantics was investigated using a cross-splicing method. As previously demonstrated, combined prosodic and semantic expectancy violations elicit N400-like negativities irrespective of emotional categories in healthy listeners. In contrast, basal ganglia patients show this negativity only for the emotions of happiness and anger, but not for fear or disgust. The current data serve as first evidence that lesions within the left basal ganglia affect the comparative online processing of fear and disgust prosody and semantics. Furthermore, the data imply that previously reported emotional speech recognition deficits in basal ganglia patients may be due to misaligned processing of emotional prosody and semantics.

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1. Introduction

More than 20 years ago, a review on the relationship between emotional dysfunctions and the basal ganglia was published (Mayeux, 1983). In this review, the author observed that emotional disorders are reported more frequently for basal ganglia disorders than would be expected by chance. Since then accumulating (neuro-)psychological evidence has shown that the basal ganglia play an important role in emotional facial (e.g., Dujardin et al., 2004) and vocal expression

processing (e.g., Breitenstein et al., 2001; Kotz et al., 2003a; Pell and Leonard, 2003). Moreover, in accord with the assumption that “basic” human emotions engage distinct neural networks (e.g., Morris et al., 1996; Phillips et al., 1997; Adolphs, 2002), a small but growing literature suggests that next to the insula, the basal ganglia are engaged in the processing of disgust, in particular (Calder et al., 2000, and see Calder et al., 2001, for an overview). Although this result is controversially discussed there is also evidence that the basal ganglia are involved in the recognition of other basic emotions

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such as anger, fear, or sadness (Calder et al., 2004; Kan et al., 2002; George et al., 1995), indicating that the basal ganglia engage in “negative” emotion processing. Other positions claim that the basal ganglia are generally involved in language processing (see, e.g., Liebermann, 2001, for a review), including linguistic prosodic (e.g., Van Lancker Sidtis et al., 2006), or controlled syntactic (e.g., Kotz et al., 2003b), and lexical-semantic processes (e.g., Cappa and Abutalebi, 1999; Lieberman, 2001; Kotz et al., 2002; but see, e.g., Gotham et al., 1988 for counter evidence).

1.1. A role for the basal ganglia in vocal emotional processing?

Increasing neuroimaging and clinical evidence postulates that the basal ganglia are engaged in the processing of vocal emotions in general and more specifically for certain basic emotions. For instance, in an fMRI study by Morris and colleagues (1999), participants listened to fearful, sad, happy, and neutral non-verbal vocalizations. The authors reported a bilaterally distributed network of brain regions that is involved in the processing of vocal emotions. Next to both temporal and prefrontal areas, enhanced activity to all emotional vocalizations was found in the anterior insula, and the caudate nucleus. Further neuroimaging evidence for a potential role of the basal ganglia during emotional speech processing comes from Kotz et al. (2003a). The authors compared brain activation patterns in healthy participants in response to normal speech and PURR-filtered speech, in which the semantic and syntactic information is removed. Normal speech activates a bilateral temporo-putaminal network, while filtered speech results in bilateral fronto-caudal activation.

These results go hand in hand with data from clinical studies. Patient studies on neurodegenerative basal ganglia disease, such as Parkinson’s, Huntington’s, or Wilson’s disease, also suggest an important role of the basal ganglia in emotional speech processing. For example, some Parkinson studies (e.g., Breitenstein et al., 1998, 2001; Benke et al., 1998) suggest basal ganglia involvement in emotional prosodic processing. Moreover, in a study by Pell and Leonard (2003), patients in the early stage of Parkinson’s disease displayed a range of emotional perception difficulties in discrimination, identification, and feature rating tasks. These data suggest that the basal ganglia may differentially engage in vocal emotional processing dependent on task and stimulus type. Although neurodegenerative changes of the basal ganglia can result in functional deficits that are not directly tied to the basal ganglia, these data contribute to a growing consensus that the basal ganglia are associated with (vocal) emotion processing. Thus, these data should be considered when postulating hypotheses about the functional role of the basal ganglia in emotion processing.

1.2. Basal ganglia involvement in the processing of specific emotions?

The paradigm applied by Pell and Leonard (2003) also allowed inspecting emotion-specific effects. While results showed specific impairments for disgust and sadness in a feature rating task, patients did not suffer from such impairment in

the identification task (Pell and Leonard, 2003). Actually, recognition deficits for facial and vocal expressions of disgust have been reported frequently in patients with neurodegenerative basal ganglia disorders (e.g., Pell and Leonard, 2003; Sprengelmeyer et al., 1996, 1997; Calder et al., 2000; Wang et al., 2003), all pointing to the fact that the basal ganglia may modulate the perception of disgust. For instance, Calder and colleagues (2000) reported data from a patient who suffered from an isolated recognition deficit of facial and vocal disgust expressions. However, next to this recognition deficit, other studies also suggest a deficit for the emotional categories of fear (e.g., Kan et al., 2002) and anger (Calder et al., 2004). Calder and colleagues investigated anger recognition in patients with focal lesions of the ventral striatum and patients with lesions in more dorsal areas of the basal ganglia regions. Their results suggest that lesions to the ventral striatum can lead to severe impairment in facial, but also vocal expressions of anger recognition (Calder et al., 2004). In a study by Kan and co-workers, Parkinson’s patients were reported to suffer from a dynamic facial emotion recognition deficit for disgust and fear, but not during vocal emotion recognition. Our own data also point to the fact that the basal ganglia can be involved in the processing of negative emotional prosody (Paulmann et al., 2005; Dara et al., 2008). For instance, we tested emotional prosody perception in basal ganglia lesion patients using vocal expressions (with and without lexical content) of anger, fear, disgust, and happiness compared to a neutral baseline. While patients showed an above-chance-level recognition for emotional and neutral prosody, categorization of disgust and fear compared to the neutral baseline was deficient. In contrast, categories of anger and happiness were less strongly affected (Paulmann et al., 2005). In sum, the idea that the basal ganglia are involved in the processing of negative emotions is strengthened by some recent data, but the current results motivate further investigations of the basal ganglia and their role in vocal emotion processing.

1.3. Can the functional role of the basal ganglia be specified in emotion processing?

As surveyed above, there is ample behavioral evidence from clinical studies suggesting a key role for the basal ganglia during emotion processing (e.g., Breitenstein et al., 1998, 2001; Pell and Leonard, 2003; Paulmann et al., 2005). In contrast, neurophysiological evidence on emotional processing in clinical populations is still rare. However, one creditable exception is the study by Wieser and colleagues (2006). Early emotion discrimination was investigated in Parkinson’s disease patients by means of event-related potentials (ERPs). In addition, explicit behavioral responses, such as arousal ratings, were recorded for a subset of emotional pictures. Results revealed dissociations between early discrimination and late behavioral responses. In particular, emotional pictures elicited an early posterior negativity (EPN) 220 ms after picture onset that was comparable between patients and healthy controls. In contrast, behavioral rating of highly arousing pictures revealed lower arousal rating in patients than in controls, suggesting that discrimination difficulties arise at later stages of emotion processing in patients. Along similar lines, we investigated emotional speech (with and

without lexical content) processing with ERPs in patients with focal lesions of the basal ganglia (Paulmann et al., 2006). In addition, the same patients were asked to explicitly categorize a subset of the same emotional vocalizations (Paulmann et al., 2005). ERP results show an early and comparable P200 effect in emotional speech processing in basal ganglia patients and healthy controls (Paulmann et al., 2006). However, as reported earlier, at later processing stages, patients showed affected emotional prosodic categorization (Paulmann et al., 2005). Taken together, these data indicate that emotional prosodic processing may be particularly impaired at later processing stages. Alternatively, it can be argued that explicit task instructions (i.e., categorization) may enhance the impairments observed in subcortical patients. Further studies should therefore test if emotional impairments in basal ganglia patients result from task-specific instructions.

Nevertheless, the question arises how discrepancies between early and later emotional discrimination processes can be explained. Here, other non-emotional functions typically linked to the basal ganglia may shed some light on the problem. The basal ganglia have been considered to be involved in general integrational functions (e.g., Kotz et al., 2003b; Friederici et al., 2003; Kyran and Larson, 2001). For instance, Kotz and colleagues (2003b) investigated sentence processing in patients with focal lesions of the basal ganglia. Patients were tested with sentences that included a verb argument structure violation. Previously recorded data from young healthy participants revealed that this type of violation elicits an N400–P600 complex. It is argued that the N400 is elicited because of incorrect semantic–thematic role assignment, while the P600 is elicited because verb information and the syntactic structure of the sentences do not match. Results revealed that basal ganglia patients showed no P600, but an extended N400. The authors suggest that late semantic–thematic integration processes as reflected by the N400 are partially modulated by the basal ganglia. The lack of a P600 implies that the basal ganglia play an important role in late syntactic integration processes (Kotz et al., 2003b). Furthermore, evidence from this group also showed that Parkinson's disease patients suffer from impairments of late syntactic integrational processes rather than from impairments of early automatic syntactic processes, again suggesting that the basal ganglia are involved in syntactic integration (Friederici et al., 2003). Last, in a speech production study, Kyran and Larson (2001) investigated how auditory feedback is used in the control of pitch production by means of a pitch-shift reflex effect in Parkinson's disease patients. Patients and controls were asked to produce vocalizations while the same vocal signals were played back to them via headphones. Critically, their vocalizations were manipulated so that the signal was systematically shifted in pitch and duration. Results showed that patients had significantly longer reflex peak and end times than healthy controls in some of the manipulations. The authors argue that their results demonstrate that Parkinson's disease patients suffer from dysfunctional sensory integration of auditory feedback. In sum, there is evidence that suggests that the basal ganglia are involved in several language-related integration processes, such as syntactic integration, semantic–thematic integration, and sensory integration of auditory feedback.

Clearly, the successful recognition of an emotional stimulus requires the decoder to integrate or combine emotional information from various sources. For instance, to recognize an emotional speech stimulus, the listener has to comparatively process acoustic correlates such as perceived pitch, duration, and intensity in a speech stream (i.e., prosodic information) as well as emotional semantic information. Taking the evidence elaborated above, one may speculate that because of dysfunctions of these integration, or fusion processes, patients with basal ganglia impairments will suffer from emotional speech recognition deficits. To clarify, the discrepancy between successful early emotional differentiation as reflected in the P200 for emotional vocal expressions and the unsuccessful later emotional recognition for the same stimuli may be due to deregulated integration process, which is (at least in part) modulated by the basal ganglia.

1.4. Aims and rationale

To test this hypothesis, i.e., are the basal ganglia involved in the comparative processing of emotional prosodic and emotional semantic information in speech processing, we tested patients with focal lesions of the basal ganglia in an ERP experiment. Specifically, we investigated the time course of comparative processing of emotional prosody and emotional semantics. Previous studies with healthy participants have clearly shown that emotional speech processing relies on the integration and evaluation of verbal and non-verbal emotional cues and can be effectively investigated by means of ERPs (e.g., Bostanov and Kotchoubey, 2004; Wambacq and Jerger, 2004; Schirmer et al., 2002, 2005; Kotz and Paulmann, 2007; Paulmann and Kotz, 2008). Attempts to investigate emotional speech by means of ERPs include cross-modal priming experiments, emotional judgement tasks, or oddball paradigms. However, such paradigms do not allow drawing firm conclusions about prosodic and semantic comparative processes as they unfold in time. However, cross-splicing emotional prosodic and semantic information licenses to investigate their online integration or combination of different (emotional) information channels in the speech signal (see Kotz and Paulmann, 2007). In particular, cross-splicing allows investigating the temporal dynamics of integrative or comparative processes as it permits to temporally synchronize and time lock critical deviation points of (emotional) expectancy. Therefore, we recently used this approach to investigate the time course of emotional prosody with and without emotional semantics. Our results clearly differentiate processing dynamics of emotional prosody and the comparative processing of emotional prosody with semantics irrespective of task (Kotz and Paulmann, 2007; Paulmann and Kotz, 2008), i.e., in explicit and implicit emotional processing tasks. More specifically, we showed that emotional prosody expectancy violations elicit a right-lateralized positivity, while combined emotional prosodic/semantic expectancy violations elicited an early negativity in healthy listeners. Reported results are in line with studies that show processing differences for linguistic prosodic deviance processing and combined prosodic/semantic processing (Astésano et al., 2004). Moreover, we also showed that left-sided basal ganglia patients show the same right-lateralized positivity in response to pure emotional

prosodic expectancy violations (Paulmann et al., 2008a), questioning the fact that the perception of acoustic correlates in emotional speech is impaired. Whether the same is true for the comparative processing of emotional prosody and semantics will be investigated in the current study.

Adopting our previously established cross-splicing paradigm, we aimed to replicate effects of combined emotional prosodic and semantic expectancy deviance processing (reflected in a negative ERP component) in healthy participants and in patients with focal lesions of the basal ganglia. With this approach, we attempt to further specify the processes that may recruit the basal ganglia during emotional speech processing. If the comparative processing of emotional prosody with emotional semantics is impaired in basal ganglia patients, a negative ERP response to combined expectancy violations should be affected. Furthermore, it seems to be particularly important that more than one emotion is tested when aiming to specify whether one particular emotional vocal expression is correlated with the basal ganglia disorder. Here, four emotions (anger, fear, disgust, and happiness) were investigated. If there is a specific deficit for disgust, the combined expectancy violation to the vocal expressions of disgust should elicit different ERP responses than expectancy violations to other emotional categories. Taken together, we aim to specify if the online combination of emotional prosodic and semantic information recruits the basal ganglia during emotional speech processing and if so, if one specific basic emotion is particularly modulating this involvement.

2. Results

ERP mean amplitudes were calculated for each emotional category (anger, disgust, fear, happy) separately. The ERP for matching and mismatching sentences was calculated with a $2 \times 2 \times 4$ design. The factors were the between-subjects factor *Group* (healthy controls/basal ganglia patients) and the repeated within-subjects factors *M* (matching or non-violated/mismatching, or violated sentence) and *Scalp Regions of Interest* (SROI). Each SROI defined a critical region of scalp sites: left frontal (LF): F7 F3 FT7; right frontal: F8 F4 TF8; left central (LC): T7 C3 CP5; right central (RC): T8 C4 CP6; left parietal (LP): P7 P3 O1, right parietal (RP): P4 P8 O2 (see Dien and Santuzzi (2004) for regional averaging). The null hypothesis was rejected for p values smaller than .05. The Huynh–Feldt correction was applied to all repeated measures with greater than one degree of freedom in the numerator (Huynh and Feldt, 1976). ERP time windows were defined based on previous evidence (Kotz and Paulmann, 2007; Paulmann and Kotz, 2008), visual inspection, and consecutive 50-ms time-line analyses (see Handy, 2004).

Behavioral results were not analyzed because previous research (Kotz and Paulmann, 2007) has found no relevant effects for expectancy violations in reaction times or correct responses in healthy participants.

3. ERP results

Starting from sentence onset ERPs for both groups and all emotional categories revealed a N100 increasing in amplitude

extending from anterior to posterior electrode sites, followed by a P200 with larger amplitude at anterior and central than at posterior electrode sites. Following these early components, morphologically differently accentuated effects for unspliced and spliced sentences were observed. These varied with respect to emotional category and participant group. In the following, statistical analyses for the different time windows are listed for each emotional category separately. In the case for disgust, visual inspection and time-line analyses also revealed a significant $M \times \text{Group}$ interaction at a later time window than previous research would suggest for the expected negativity. Thus, a second time window was analyzed accordingly.

3.1. Anger: 400 to 500 ms

A significant effect of M was found ($F_{(1,22)}=5.21$, $p<.05$), revealing more negative-going ERP components for mismatching sentences than for matching sentences. No other main effects or interactions of interest reached significance (all $p>.05$). This result confirms that both groups show the expected negativity in response to semantically and prosodically violated angry sentences.

3.2. Disgust: 400 to 500 ms

The analysis revealed a significant *Group* effect ($F_{(1,22)}=4.71$, $p<.05$), showing a more negative-going amplitude for the healthy control group than for the patient group. No other effects reached significance.¹

3.3. Disgust: 600 to 750 ms

A significant interaction between M and *Group* ($F_{(1,22)}=6.52$, $p<.05$) was found. This interaction allowed for a by *Group* analysis, revealing a significant M effect in the patient group ($F_{(1,11)}=6.51$, $p<.05$), showing a more positive-going ERP component for mismatching sentences than for matching sentences. In contrast, the more negative-going ERP component in response to mismatching sentences that was clearly visible in the ERPs for the control group did not reach significance ($p>.05$). The results revealed significant ERP component differences between the two groups, with the patient group showing a positivity for mismatching sentences.

3.4. Fear: 400 to 500 ms

No significant main effect was found ($p>.05$); however, an interaction between M and *Group* turned out to be marginally significant ($F_{(1,22)}=3.17$, $p<.09$). Because it was of special interest to clarify if ERPs differed between patients and their controls and because visual inspection suggested this difference, a hypothesis-driven by *Group* analysis was carried out.

¹ Because ERP plots clearly revealed a negativity for healthy controls in the time window of 400–500 ms, we carried out analyses by electrode and group. These analyses revealed marginally significant M effects for the control group at electrodes CZ ($p=.0814$), PZ ($p=.0817$), and P4 ($p=.0976$), showing more negative going ERP amplitudes for violated sentences compared to non-violated sentences.

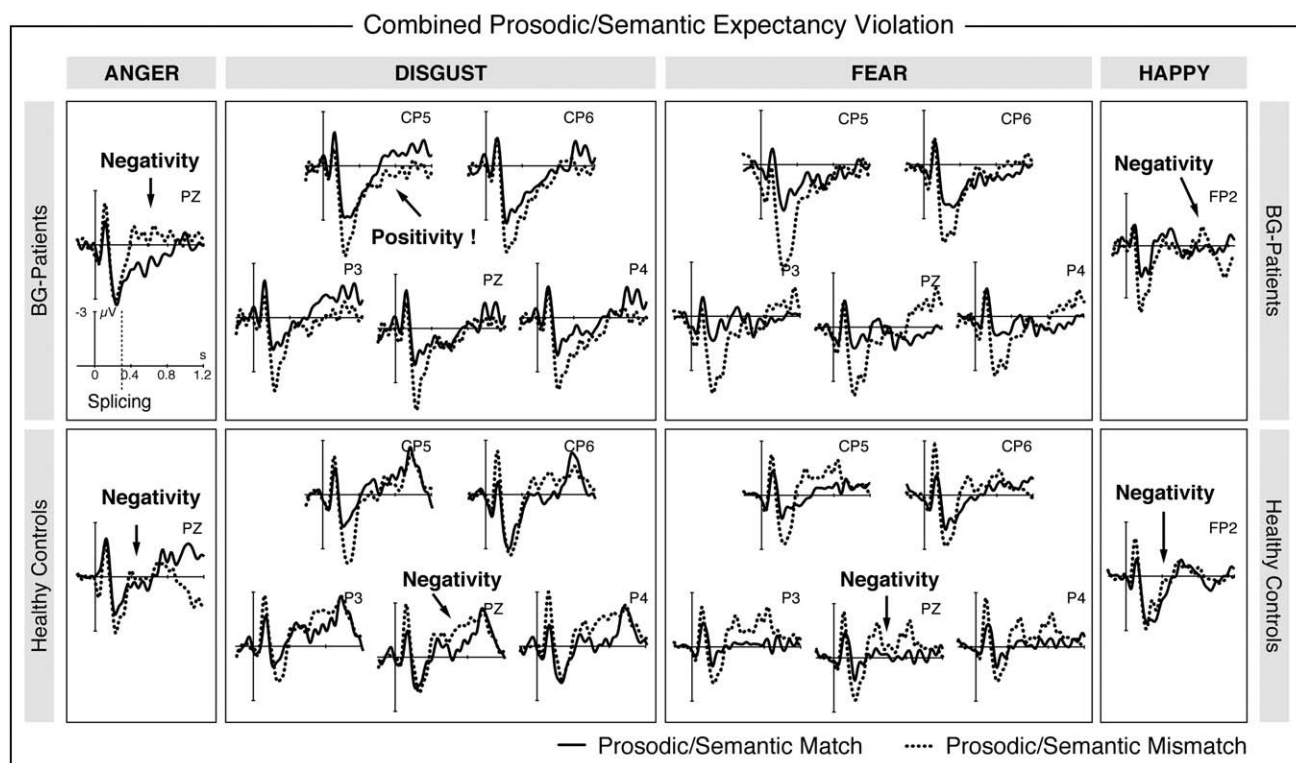


Fig. 1 – Illustration 1 displays the ERP effects elicited by combined emotional prosodic and emotional semantic expectancy violations for basal ganglia patients and healthy controls.

Results revealed a significant M effect in the control group ($F_{(1,11)}=7.54$, $p<.05$), with mismatching sentences showing a more negative ERP component than matching sentences. The same M effect was not found in the patient group ($p=.6154$). In sum, this suggests processing differences between controls and patients for mismatching fearful sentences, with controls showing the expected negative ERP component in response to mismatching fearful sentences.

3.5. Happy: 350 to 450 ms

No significant main effect of M was found. However, the interaction $M \times \text{SROI} \times \text{Group}$ turned out to be significant ($F_{(1,22)}=3.40$, $p<.05$). The stepdown by Group analysis only revealed a borderline significant interaction $M \times \text{SROI}$ in the control group ($F_{(1,11)}=2.10$, $p=.0974$), but not in the patient group ($p=.1488$). Again, as visual inspection clearly revealed a negativity for mismatching sentences for both healthy controls and basal ganglia patients, we carried out hypothesis-driven analyses by electrode channel and group. For the control group, we confirmed a significant M effect at electrode F4 ($F_{(1,11)}=5.39$, $p<.05$) revealing more negative ERP components for mismatching sentences than for matching sentences.² In sum, controls showed negative ERP waveforms in response to semantically

and prosodically mismatching happy sentences between 350 and 450 ms. This effect was found for the patient group only at a slightly later time window (500–600 ms).

3.6. Summary

Taken together, results reveal processing differences for the two groups between semantically and prosodically matching and semantically and prosodically mismatching sentences in the emotional categories of fear and disgust, in particular. For the emotional category of anger, no differences were found, while the negativity found for both groups in the emotional category of happiness differed slightly in latency. Fig. 1 displays the ERP effects for basal ganglia patients and healthy control participants.

4. Discussion

The present study investigated emotional speech processing in patients with focal lesions in the left basal ganglia as well as age- and education-matched healthy controls to further explore the role of the basal ganglia in emotional speech processing. With a previously established cross-splicing approach, we investigated the online combination of emotional prosody with emotional semantics. In particular, we looked at neural responses to expectancy violations of emotional prosody and emotional semantics in an ongoing speech stream. In line with accumulating evidence for basal ganglia involvement in emotional speech processing (e.g., Breitenstein

² For a slightly later time-window of 500 to 600 ms, these analyses also revealed marginally significant effects at CP6 ($p=.0702$) and P8 ($p=.0807$), for basal ganglia patients.

et al., 2001; Kotz et al., 2003a; Pell and Leonard, 2003; Paulmann et al., 2005; Dara et al., 2008), we report impairment in left-sided basal ganglia patients for the processing of emotional prosodic and semantic expectancy violations for disgust and fear, in particular. This was reflected in a missing modulation in the case of fear and a reversed pattern (a positivity rather than a negativity) in the case of disgust.

While many previous studies have investigated the role of the basal ganglia by means of behavioral methods and explicit emotional tasks (e.g., Pell and Leonard, 2003; Breitenstein et al., 1998; Paulmann et al., 2005; Dara et al., 2008), the current ERP study provides online evidence from an implicit emotional processing task. We thereby ensured that the process of interest was not confounded by task demands. More specifically, this approach allowed separating process-correlated task effects from potential emotional processing deficits related to emotional speech processing. Furthermore, most previous studies investigated emotional speech processing in patients with neurodegenerative disorders, such as Parkinson's and Huntington's disease. Here, a patient population suffering from focal lesions in the left basal ganglia was tested. This weakens the argument that previously reported emotional deficits in neurodegenerative disorders may result from cortical dysfunction and strengthens the hypothesis that the basal ganglia are indeed involved in emotional speech processing. In sum, the present study optimizes previous approaches to study the role of the basal ganglia during emotional speech processing. However, before discussing the present results, one caveat of the current results needs to be addressed.

Previous evidence (Kotz and Paulmann, 2007; Paulmann and Kotz, 2008) from healthy young participants revealed that combined emotional prosodic/semantic expectancy violations elicit a more negative-going ERP waveform than non-violated emotional sentences, irrespective of task (Kotz and Paulmann, 2007) or speaker voice (Paulmann and Kotz, 2008). While this evidence was obtained in studies with at least 30 participants, the current study included only 12 participants per group. In addition, we have previously reported age effects on vocal emotion recognition in middle-aged participants (Paulmann et al., 2008b). Moreover, the negativity observed here has been functionally linked to the N400 component (Kotz and Paulmann, 2007; Paulmann and Kotz, 2008). This component varies in amplitude and latency as a function of age (e.g., Federmeier and Kutas, 2005; Kok, 2000). Patients and controls in the current population sample were approximately 25 years older than previously tested participants. These observations may have added to the fact that we failed to find significant results for the emotional categories of disgust and happiness in the control group even though visual inspection of ERPs clearly indicate a more negative-going waveform for mismatching sentences when compared to matching sentences in these categories. In sum, we cannot exclude the possibility that reduced power (i.e., to the limited number and the age of participants) may have influenced the present results. Nevertheless, the current results clearly offer new insights into the functional role of the basal ganglia in emotional speech processing and provide a valuable foundation for further research on the specific role of the basal ganglia in emotional speech processing.

4.1. Comparative processing of emotional prosody and emotional semantics

The current data nicely complement neuroimaging (e.g., Kotz et al., 2003a) and clinical data (e.g., Breitenstein et al., 1998; Dara et al., 2008) for basal ganglia involvement in emotional speech processing. While previous clinical studies explored emotional speech recognition, categorization, or identification (e.g., Pell and Leonard, 2003; Breitenstein et al., 1998; Paulmann et al., 2005; Dara et al., 2008), the present study investigated the comparative online processing of two emotional channels (prosody with semantics) by means of a cross-splicing procedure. The cross-splicing approach offers the unique possibility to time lock the processing of information channels, that is, it allows synchronizing emotional information processing (see Kotz and Paulmann, 2007). In consequence, previously reported impairments in patients with basal ganglia disorder for emotional speech processing can be further differentiated with this approach.

Building on the observation that basal ganglia dysfunction leads to off-line emotional facial (Wieser et al., 2006) and vocal (Paulmann et al., 2005) expression recognition but not to online discrimination deficits in the same patients groups (Wieser et al., 2006; Paulmann et al., 2006), we hypothesized that previously reported off-line recognition difficulties could result from difficulties to comparatively process different emotional channels such as prosody and semantics. The current experimental setup offers the possibility to look at this phenomenon in the auditory modality. Prosodic and semantic expectancy violations to vocal expressions of disgust and fear did not elicit the expected N400-like ERP component in basal ganglia patients. Hence, the present findings provide new evidence for the role of the basal ganglia in emotional prosodic and emotional semantic integration processing. In fact, while the role of the basal ganglia in emotional prosodic processing has been previously reported (e.g., Breitenstein et al., 2001; Kotz et al., 2003a; Pell and Leonard, 2003; Paulmann et al., 2005), there is also evidence that the basal ganglia are involved in semantic processing (see Copland, 2003 for an overview). However, most published work investigated the role of the basal ganglia in lexical-semantic processes, while integration processes have not yet been extensively investigated. Still, in the work of Friederici and colleagues (2003) as well as Kotz and colleagues (2003b), an extended N400 latency for patients (suffering from Parkinson's disease or focal basal ganglia lesions) but not for healthy controls in response to semantic violations can be observed. These data indicate that both lexical selection and semantic integration may be affected in these patients. Thus, there is converging evidence that basal ganglia disorders can also affect semantic meaning integration, a result that is also found for emotional semantic processing (Kotz et al., 2006).

Alternatively, it could be argued that it is not the comparative processing of prosodic and semantic information that is affected in these patients, but rather the deficit could be due to deficient processing of particular acoustic cues (e.g., elevated pitch; Breitenstein et al., 1998). Two observations make this explanation rather unlikely. First of all, previous evidence in healthy participants suggests that neural responses to combined expectancy violations are primarily

semantically driven albeit with an emotional prosodic influence (cf. Kotz and Paulmann, 2007; Paulmann and Kotz, 2008). This was argued based on the observation that pure emotional prosodic expectancy violations elicit a positivity and combined emotional prosodic/semantic expectancy violations elicit an N400-like negativity (Kotz and Paulmann, 2007; Paulmann and Kotz, 2008), suggesting that emotional semantic information may predominate prosodic information processing when the two information are time locked. Importantly, recent evidence from the very same patient group tested here revealed no deficit when processing emotional prosodic expectancy violations, that is, patients showed the same kind of expected positive ERP component in response to violations as healthy controls (Paulmann et al., 2008a). That is, a pure emotional prosodic deviance detection deficit was not confirmed, a result that is supported by recent results by Dara and colleagues (2008). In an intensity rating task, Parkinson's disease patients did not differ from healthy controls, rendering it unlikely that elevated pitch identification (that tends to correlate with arousal/intensity of a stimulus) is affected in these patients. It seems as if processing of highly salient acoustic parameters (such as intensity) or parameter configurations (as required in processing of emotional prosodic expectancy violations) is not per se impaired (see also Dara et al., 2008). Instead, more complex processing (integration of multiple prosodic cues in addition to semantic meaning processing) seems to lead to severe impairments in basal ganglia patients during emotional speech processing. Taken together, the current data complement previous findings and serve as new evidence that the basal ganglia are also engaged in emotional prosodic and semantic comparative processing.

4.2. Selective impairments for vocal expressions of disgust and fear

One question that remains open is why only expectancy violations to disgust and fearful vocalizations fail to elicit an N400-like negativity while expectancy violations to angry and happy (slightly delayed³) vocalizations did not reveal such impairment. One possible explanation is that comparative processing of prosodic/semantic information is only impaired for particular emotions, i.e., only when the processing of an emotional category is modulated by the basal ganglia. Indeed, as previously mentioned, investigations of “basic” emotions, such as anger, disgust, fear, or happiness, postulate that distinct neural networks are engaged processing these emotions (e.g., Adolphs, 2002). For instance, different evidence suggests that the basal ganglia modulate the perception of disgust, in particular (Pell and Leonard, 2003; Sprengelmeyer et al., 1996; Calder et al., 2000), an observation that is supported by the current data set, which failed to find the expected negativity for disgust expectancy violations. A word

of caution is needed though, as the present patient population included 6 patients with additional lesions of the insula, an area implicated in disgust processing (see Introduction). Thus, to verify the consistency of our effects and to exclude the possibility that effects were driven by patients with additional insula lesions in particular, we applied the jackknifing procedure (see Obleser et al., 2006). Although this procedure confirmed the homogeneity of our patient group, we also saw some indication that patients with additional insula lesions showed a different strength of effects in contrast to patients with isolated basal ganglia lesions. Clearly, the present patient population is too small to make extensive claims about the role of the insula in comparative emotional semantic and prosody processing for disgust sentences, but future studies should try to investigate this issue in more depth.

Returning to the idea that distinct neural networks are engaged in processing different emotions (e.g., Adolphs, 2002), there is also evidence that the basal ganglia are involved in the recognition of fear (Kan et al., 2002; Dara et al., 2008). Again, the present findings support basal ganglia involvement for the processing of fearful stimuli, as the expected negative ERP component for combined expectancy violations of fearful sentences was not confirmed in basal ganglia patients. Moreover, in line with evidence from Calder and colleagues (2004), who found impairment in anger recognition from facial but not from vocal expressions, our data revealed no comparative processing deficit from angry vocalizations (however, see Dara et al., 2008, for an off-line anger recognition deficit). This observation speaks against the idea that the basal ganglia are engaged in auditory anger processing, or negative auditory emotion processing in general. Calder et al. (2004) suggested that heterogeneous results are due to different lesion localization within the basal ganglia (ventral vs. dorsal parts), an idea that should be addressed in future studies. In sum, we hypothesize that severe impairment for emotional comparative processes can only be observed if the basal ganglia are involved in processing the particular emotion, that is, the more involvement of the basal ganglia in an (emotional) process, the more apparent the impairment.

Alternatively, heterogeneous results regarding the involvement of specific brain regions in specific emotions gathered from clinical data can be attributed to differently affected brain structures in patients (see also comment above). Clearly, general emotional processing is based on several interconnected cortical and subcortical brain structures, including the amygdala, basal ganglia, orbito-frontal cortex, and other structures (e.g., Adolphs, 2002). In particular, despite reports of specific or greater brain activation for specific emotions, there also seems to be great activation overlap in emotion processing (e.g., Phillips et al., 1998). For instance, Phillips and colleagues (1998) report activation of superior temporal gyrus by four different emotions. Also, the authors report activation of inferior posterior temporal gyrus and middle occipital cortex for both facial and vocal expressions of emotions implying that these structures play an important role in emotion processing per se. Moreover, Johnston et al. (2001) have presented a neural network model for emotional facial expression recognition. The intact network showed recognition accuracy similar to the behavior of healthy participants (over 80% correct emotion identification). However, lesioning the network resulted in a

³ As mentioned earlier, latency differences in the N400 component as observed in aging and patient populations are not an uncommon phenomenon (e.g., Federmeier & Kutas, 2005; Kok, 2000; Friederici et al., 2003). Also, it seems unlikely that the delay in the negativity for patients implies a functional difference as topographic distribution as well as amplitude size is still comparable across the two groups.

general inability to correctly identify emotional facial expressions. Interestingly, negative emotion recognition was more affected than positive emotion or neutral stimuli recognition. The authors thus argued for “a unitary location for the evaluation of emotional valence, or a functionally unitary circuit involving a number of brain areas with a high degree of reciprocal connectivity and activational interaction” (Johnston et al., 2001).

In fact, such a unitary emotional valence evaluation seems rather plausible from an evolutionary perspective. Specifically, the recognition of negative emotions such as fear, disgust, sadness, or anger may encourage a comparable reaction, including autonomic activation, flight, or avoidance behaviors. That is, it can be argued that, as a first step, the brain maximally discriminates between positive and negative emotions to trigger subsequent behavior. Clearly, emotions of the same valence dimension (e.g., fear, anger, disgust) would allocate greater brain area, or neural response overlap, as suggested by some recent fMRI studies (e.g., Phillips et al., 1998). In turn, these similarities could lead to difficulties in discriminating between different emotions of the same valence.

5. Conclusion

The current study was designed to explore the online comparative processing of emotional prosody and semantics in patients with lesions of basal ganglia. In line with the literature that relate the basal ganglia to integrational functions during language processing (e.g., Kotz et al., 2003b; Friederici et al., 2003; Kyran and Larson, 2001), the assumption was that basal ganglia patients would suffer from problems during the combination of the two channels. In particular, based on previous evidence (Calder et al., 2000, 2004), emotional vocalizations of disgust and fear were of central interest. Results revealed impairment in left-sided basal ganglia patients during processing of combined violations to emotional prosody and semantics for disgust and fear but not for happy and angry. Thus, the view that detection of fearful and disgust deviances is hampered in basal ganglia patients is supported. The present data contribute to the discussion that

the basal ganglia are involved in emotional speech processing but also point to the fact that this involvement depends on emotional channel disposability, that is, the fusion of emotional prosody and emotional semantics is of particular importance when processing an emotionally laden stimulus. Thus, the data hint to the fact that previously reported emotional speech recognition deficits in basal ganglia patients may be caused by an online deficit to combine the two emotional channels prosody and semantics.

6. Experimental procedures

6.1. Participants

Twelve German-speaking patients (1 female, all right-handed; mean age: 49.2 years, SD: 17.2) with lesions in the striatum participated in the study after giving informed consent. Lesions resulted from left hemisphere insults: ischemic stroke ($n=3$), embolic stroke ($n=3$), intracerebral bleeding (ICB; $n=6$), or arterio-arterial infarction ($n=1$). The average time since lesion was 4.6 years (range, 1 year 8 months to 7 years and 11 months). Lesion sites were determined by (T1- and T2-weighted) anatomical MRI datasets from a 3.0-T system (Bruker 30/100 Medspec) and evaluated by an experienced neuroanatomist. In addition, 12 healthy controls took part in the experiment. The groups were age, education, and gender matched. Only non-aphasic patients and those who showed no noticeable results on standard neuropsychological testing (e.g., Behavioral Assessment of the Dysexecutive Syndrome [BADS], Wechsler Gedächtnistest [WMR-S]) were included in the current study. See Fig. 2 for a graphical display of a lesion overlay. Individual patient information is listed in Table 1.

6.2. Stimulus material

The base stimulus material consisted of semantically and prosodically matching stimuli spoken in four emotions (anger, fear, disgust, happy) and a neutral baseline. Thirty sentences of each emotion and sentence type were presented, resulting in

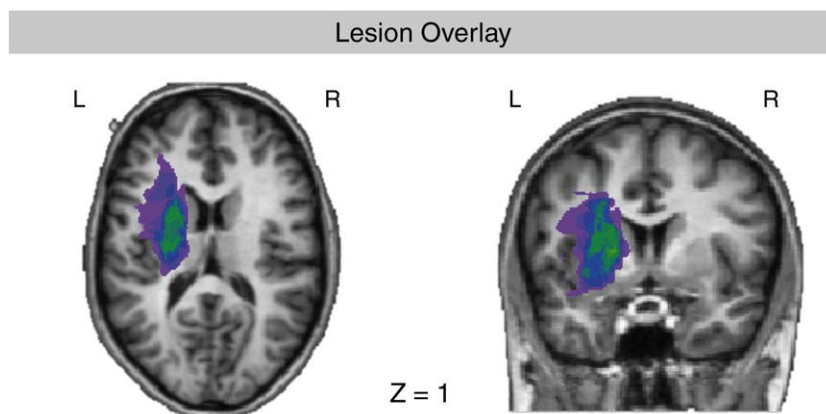


Fig. 2 – The illustration shows an overlay of respective individual patient lesions indicating maximum overlap in the basal ganglia. Displayed is one slice level ($Z1=89$ [originally 1–180]). Green/yellowish shades reveal maximum overlap of lesion sites, whereas purple shades reveal minimal lesion site overlap. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 1 – This table provides the demographic patient information.

Patient	Sex	Age at test (years)	Time since lesion (years)	Etiology	Lesion description
01	m	63	7 years and 4 months	ICB	ant. GPe, ant. IC
02	m	53	6 years and 1 month	ICB	post. Put., GPe, post. EC, IC, lat. Thal.
03	m	48	5 years and 1 month	ICB	Put., GPe, EC, ant. IC, reduced volume of Caud.
04	m	31	5 years and 5 months	Ischemic Infarct	post. Put., Caud. (body), middle Ins., parietal operculum
05	m	68	4 years and 4 months	Ischemic Infarct	Caud. (ant. body), ant. Put., GPe, EC, ant. IC, ant. Ins., preinsular WM
06	f	40	3 years and 3 months	Arterio-Arterial infarct	Caud. (body), Put., GPe, ant. IC, EC, parietal operculum, post. Ins.
07	m	59	4 years and 11 months	Ischemic infarct	Caud. (body), Put., GPe, IC, EC
08	m	66	7 years and 11 months	ICB	Caud., Put.
09	m	33	6 years	Embolic infarct	Put., Caud.
10	m	28	1 year and 8 months	ICB	post. Put., Caud.
11	m	26	3 years and 5 months	ICB	Thal., post. Put., Caud.
12	m	75	4 years and 11 months	Embolic infarct	Caud. (body), Put.,

Lesions resulted from left hemispheric insults. The average time since lesion in the basal ganglia was: 4.6 years (range, 1 year 8 months to 7 years and 11 months). Lesion sites were determined by (T1- and T2-weighted) anatomical MRI datasets from a 3.0 T system (Bruker 30/100 Medspec) and evaluated by an experienced neuroanatomist. Note: m, male; f, female; ICB, intracerebral bleeding; ant., anterior; post., posterior; Caud., caudate nucleus; EC, external capsule system; IC, internal capsule; Ins., insula; GPe, globus pallidus externus; GPI, globus pallidus internus; Put., Putamen; Thal., thalamus; WM, white matter.

150 matching lexical sentences. In addition, the same sentences were presented in a cross-spliced version, that is, combined prosodic and semantic expectancy violations were created by cross-splicing. To this aim, a semantically and prosodically neutral first half (“Er hat”/“Sie hat”, English translation: “He has”/“She has”) of a sentence was cross-spliced to a semanti-

cally and prosodically emotional (angry, disgust, fear, happy) second half of a sentence (mean splicing point at ~300 ms after sentence onset). This splicing procedure resulted in 30 cross-spliced angry, 30 cross-spliced disgust, 30 cross-spliced fearful, and 30 cross-spliced happy sentences (i.e., 120 spliced lexical sentences were presented in total; for a visualization of splicing



Fig. 3 – The illustration explains the splicing procedure.

procedure, see Fig. 3). In addition, we included the same amount of matching and spliced pseudo-sentences (results not reported here). Thus, a total of 540 trials were presented in one session. Emotional prosody valence was obtained in an earlier rating study. In the following, the percentage correct (%) and respective standard deviations (SD) for each emotional category presented are listed: anger: 96% (SD: 3.9), disgust: 93% (SD: 7.0), fear: 69% (SD: 17.4), happy: 79% (SD: 9.7), neutral: 93% (SD: 5.5), pleasant surprise: 42% (SD: 11.4), sad: 76% (SD: 17.2; see Paulmann, Pell and Kotz, 2008b, for rating details).

Sentences were spoken by a trained male speaker in standard High German and were taped with a video camcorder (SONY Digital Video camera Recorder MiniDV DCR-TRV60E) attached to a high-quality clip-on microphone. The video material was digitized, and the voice track was separated from the visual track. Within the current experiment, only the voice material was tested. The voice material was digitized at a 16-bit/44.1-kHz sampling rate, and the amplitudes were normalized. The stimulus material was prosodically analyzed (see Paulmann et al., 2008b).

6.3. Procedure

Participants were seated in a comfortable chair at a distance of 115 cm of a computer monitor. Each participant was tested individually with a two-button panel placed before him/her in a sound-attenuating room. Half of the participants pressed the yes button with their right hand and the no button with their left hand. The other half proceeded vice versa. The sentences were presented via loudspeaker. Directions, with examples, asked participants to listen to the sentence, to read the following word, and to make a decision whether the word had been previously heard in the spoken sentence as quickly as possible. Participants had to respond within a time frame of 8000 ms. The inter-trial interval was 1500 ms. Before the actual start of the experiment, a practice session with 20 trials was carried out.

6.4. ERP recording and data analysis

The electroencephalogram (EEG) was recorded from 32 Ag-AgCl electrodes mounted on a custom-made cap (Electro-Cap International) according to the modified expanded 10–20 system (Nomenclature of the American Electroencephalographic Society, 1991). Signals were recorded continuously with a bandpass between DC and 70 Hz and digitized at a sampling rate of 250 Hz. Electrode resistance was kept under 5 K Ω . The reference electrode was the tip of the nose. Data were re-referenced off-line to linked mastoids. Eye artifact control measures were applied to the raw data of each participant to increase the number of critical trials in each condition (Pfeifer et al., 1995). Subsequently, individual EEG recordings were scanned for additional artifacts on the basis of visual inspection. ERPs were filtered off-line with a digital FIR bandpass filter ranging from 0.298 to 30 Hz (–6 dB cutoff; 1471 points). ERPs were averaged for epochs of 1200 ms starting 200 ms before sentence onset, thus including a 200-ms pre-stimulus baseline. Data quantification was constrained by a time-line analysis of the whole epoch. Based on these systematic statistical tests, previous evidence, and close visual inspection, time windows were defined for further

ERP analyses of mean amplitudes. For graphical display, ERPs were filtered off-line with a 7-Hz low pass filter.

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