Emotional processing following cortical and subcortical brain damage: contribution of the fronto-striatal circuitry

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The present study examined the differential contribution of cortical and subcortical brain structures in emotional processing by comparing patients with focal cortical lesions ($n = 32$) to those with primarily subcortical dysregulation of the basal ganglia (Parkinson's disease; $n = 14$). A standardized measure of emotional perception (Tübingen Affect Battery) was used. Only patients in the more advanced stages of Parkinson's disease and patients with focal damage to the (right) frontal lobe differed significantly from controls in both facial expression and affective prosody recognition. The findings imply involvement of the fronto-striatal circuitry in emotional processing.

Keywords: Facial expressions, affective prosody, right hemisphere dominance, Parkinson's disease

1. Introduction

Recent investigations with neurological patients have provided evidence of an involvement of neocortical brain areas in nonverbal[1] communication of emotion. Research findings further suggest a dominant role of the right hemisphere in the decoding of AFFECTIVE PROSODY[2] [7, 27, 33, 43, 56, 57, 58, 69] as well as FACIAL EXPRESSIONS [7, 23, 25, 42]. The deficits observed in neurological patients with right hemisphere damage could neither be explained by a general impairment in processing visual-spatial, nonemotional facial or acoustic stimuli [11, 14, 25] nor by a general lack of emotional comprehension [7]. The observed impairments have been interpreted with the destruction and/or disconnection of 'category-specific lexical-semantic representations of nonverbal expressions' [7, p. 1125] in the right hemisphere, which may lead secondarily to a higher distractability for context information, e.g., the semantic content of sentences [32, 66].

The widely accepted notion of a general right hemisphere dominance in the identification of facial and vocal emotions was recently challenged. Several investigations during the last decade could not support a right hemisphere superiority for facial expressions [18, 26, 67, 74], affective prosody [18, 72], or linguistic prosody [31].

In order to resolve some of the uncertainty in the literature with respect to a right hemisphere dominance for emotional prosody processing (which is at least partially due to differences in study designs) Pell and Baum [51] incorporated different degrees of linguistic structure (speech-filtered/phonetic, nonsense/syntactic, semantically well-formed/semantic) into a single experimental design. Tasks involved discrimination and identification of linguistic and emotional prosodic stimuli. Overall, the main finding was

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[1]'Nonverbal' emotional processing refers to the perception/expression of emotions in vocal intonations, facial expressions, and gestures.

[2]'Prosody' is defined as the speech rhythm, the pitch contour, and the accent patterns accompanying vocal utterances [45]. In addition to the discrimination of statement and question contours (*linguistic prosody*), there are intonation patterns differentiating emotional states of the speaker (*affective or emotional prosody*; [1]). Prosodic tasks usually involve either the *discrimination* of two sentences (same/different judgements) or the *identification* of the expressed emotion.

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that neither the patients with right (without neglect) nor left hemisphere damage were impaired compared to controls in discriminating or recognizing affective prosody on either task level, but that patients with left-sided damage showed deficits in semantically biasing tasks. The authors conclude that the results of previous studies reporting significant emotional prosodic deficits following right hemisphere damage are probably confounded by the co-existence of lasting visual neglect behavior in patient groups with temporo-parietal lesions.

Attentional demand of the tasks used may be another intervening factor. For example, Schmitt et al. [61] tested patients with right and left hemisphere damage in a task which required subjects to simultaneously judge the facial expression, the emotional prosody, and the emotional meaning of videorecorded stimulus scenes. The authors intended to simulate naturally occurring social interactions, but the design of the task may have put an attentional over-demand on the reduced attentional resources of the patient groups, especially when the three channels of communication were conflicting.

Besides interhemispheric differences, a differential involvement of INTRAHEMISPHERIC brain areas in the PERCEPTION and PRODUCTION of emotion is still disputed. Ross [56] postulated the existence of an emotional processor in the right hemisphere that functions as an analog to the speech processor in the left hemisphere [28]. According to this theory, anterior brain structures are involved in expressive tasks, whereas posterior areas contribute to stimulus reception. More recent investigations differentiating between patient groups with *anterior and posterior lesions* of the right or left hemisphere failed to report significant differences between these groups [24]; for a summary, see [10]. One exception is the recent study by Hornak et al. [36] who reported significant group differences in the naming of facial expressions between patients with orbital or ventrolateral frontal damage and patients with lesions outside this area (dorsolateral frontal, temporal or parietal). The patient groups did not differ in a (nonstandardized) task which consisted of naming emotional human 'sounds'. In the latter task, both patients with orbital/ventrolateral frontal and non-ventral damage performed significantly inferior to control subjects. The authors explained their results in light of the direct anatomical projections from both visual and auditory temporal cortex areas to the orbitofrontal cortex [6, 46] which serve the function of associating sensory stimuli with reinforcing/punishing attributes.

Recently, there has been support for an important role of the BASAL GANGLIA in emotion processing. Cancelliere and Kertesz [18] concluded from their data that patients with cortical lesions who had *additional* damage to the basal ganglia and/or the anterior temporal lobes showed the most pronounced deficits in emotional judgements, independent of the lesion side. Further evidence is provided by studies describing prosodic and facial comprehension disorders in patients with PARKINSON'S DISEASE (PD), a neurological condition with primarily dysregulation of the basal ganglia. Scott et al. [62] reported reduced performance in identification of affective prosody and facial expressions in a group of patients with PD in (supposedly) more advanced stages of the disease, unfortunately using nonstandardized emotional test material. The performance of patients with PD in the *earliest stage* of the disease, however, did not differ from healthy normal control subjects in standardized emotional tasks [8]. In an attempt to integrate these controversial findings, Pell [50] presented 11 more advanced PD patients with a battery of emotional prosody tasks (using sentences with both semantically congruent and nonsense content). Overall, the performance of PD patients was significantly inferior to control subjects in the four identification tasks (84% correct compared to 90% correct), indicating a degradation of prosodic functions but not a complete loss of prosodic knowledge [62]. Furthermore, Jacobs et al. [37] found significantly impaired performance in PD patients (also supposedly in more advanced stages) compared to control subjects in the *discrimination* of facial emotions and in tasks using emotional imagery. Dysfunction of the direct neuroanatomic connections between the ventral striatum and the limbic system [38] could explain the observed deficits of patients with PD in the processing of 'emotionally or motivationally powerful stimuli' [34, p. 87].

One further theory of emotional processing, the VALENCE HYPOTHESIS, postulates a dominance of the right hemisphere in the processing of negative emotions, whereas the left hemisphere is more involved in the comprehension of positive emotions (for a review, see [64]). The majority of studies with neurological patients, however, have not provided support for this differentiation (see [10]). Since most of these investigations used summary scores across all negative and positive emotions (e.g., pooled across anger, fear, sadness etc. for negative emotions), group dif-

ferences in single emotional categories may have been overlooked. Recently, evidence for the role of the amygdala in recognizing SPECIFIC EMOTIONS signaling threat/arousal (see also [41]) has been provided by several single case-studies presenting patients with selective bilateral damage to the amygdala (Urbach-Wiethe disease, amygdalotomy, encephalitis). The patients presented with selective deficits for the recognition of FEARFUL (and to a lesser extent angry) facial expressions, but not for the naming of other basic emotions in facial expressions or for the identification of famous faces [2, 3]. In this context, it is important to note that patients with *unilateral* damage of the amygdala did not differ from the control group in recognition of any of the facial emotions [3], suggesting a bilateral processing of emotions at the medial temporal lobe level. Using a novel technique for interpolating pairs of facial emotion prototypes ('Identification of morphed facial expressions'), Calder et al. [17] reported impairments not only for the perception of fear, but also for the emotional category anger and to a lesser extent disgust in two other patients with bilateral damage to the amygdala (see also [76]). The same group described impaired recognition of frightened and angry intonations in spoken words [63] in one of the two patients. The patient was also impaired in other complex auditory perception tasks (e.g., perception of linguistic prosody), indicating that the patient's impairment was not entirely restricted to affective prosodic recognition.

Contradictory results come from a study by Hamann et al. [30], who did not report deficits in the identification of facial emotions in two patients with complete bilateral destruction of the amygdala. The differences between research findings may be due to patients' different ages at the onset of lesion. Contrary to the patients in the other investigations, the two patients in the study by Hamann et al. [30] suffered from late-acquired brain lesions and may have established alternative neural networks outside the amygdala, possibly in posterior cortical regions of the right hemisphere [4].

Another line of evidence for the so-called 'separate substrates hypothesis' [17] of different emotions comes from recent PET [47] and fMRI [53] studies in normal control subjects (reporting that fearful stimuli activated the amygdala and disgust stimuli the anterior insular cortex) as well as from preliminary reports of especially severe deficits in the recognition of facial and vocal DISGUST (and to a lesser extent fear) in patients with HUNTINGTON'S DISEASE (HD, [65]) as well as in HD gene carriers [29]. Neocortical degeneration in patients with HD is widespread (involving both the basal ganglia as well as posterior cortex regions), making it difficult to relate the observed deficits in symptomatic patients with HD to a specific brain structure. Studies with HD gene carriers (i.e., clinically pre-symptomatic individuals) are therefore of particular interest with respect to neural substrates of emotion, because basal ganglia structures (caudate nucleus) are affected earliest by the neurodegeneration.

Furthermore, clinically significant disorders of emotional processing can be MODALITY-SPECIFIC (e.g., limited to facial or affective prosodic or gestural processing impairments) or generalized. So far, there is controversy in the literature about whether a central processor exists or whether the different modalities are processed independently. Bowers et al. [13] reported a double dissociation between identification of facial and affective prosodic stimuli in 22% of patients with right hemisphere damage and in 8% of patients with focal lesions of the left hemisphere, suggesting independent modality-specific processors. Evidence of different modalities for facial and affective prosodic perception comes from the study by Hornak et al. [36] who found dissociations between facial and vocal processing in all of their non-ventral damage patients, whereas eight of the 12 patients with orbital/ventrolateral frontal lesions were impaired in both modalities. Contradictory results were reported by Cancelliere and Kertesz [18], who classified 75% of the patients with right-sided lesions and 77.8% of the patients with damage to the left hemisphere as 'global aprosodic', with impairments in both facial and affective prosodic modalities. A problem with the latter study, however, is the minimal criterion for time since lesion (two weeks to three months). Observed deficits during this early stage might be due to nonspecific brain swelling [15], and patients might show complete or partial spontaneous recovery, especially of prosodic functions (e.g., [19]).

In summary, findings concerning affective perception in patients with cortical and/or subcortical dysfunctions remain divergent [39]. The discrepancy might be caused by inclusion of heterogenous patient samples with regard to intrahemispheric lesion site and time interval since brain damage. As many studies used nonstandardized, ad hoc constructed test materials of unknown reliability, it is unclear whether a single, homogeneous function, e.g., emotional processing, was measured. We are not aware of any study directly comparing the results of patients with unilateral focal cortical lesions to patients presenting with

primarily subcortical dysregulation of the basal ganglia (e.g., patients with PD). Furthermore, recent investigations including patients with PD did not differentiate between patients in the earliest and more advanced stages of the disease, despite neuroanatomical considerations as well as research findings (see above) predicting differing results.

The main objective of the present study was to determine whether differential patterns of performance in the comprehension of linguistic/affective prosody and facial expressions can be demonstrated in patients with focal cortical lesions (right versus left, anterior versus posterior) and primarily subcortical dysregulation (e.g., patients with PD) using standardized test material. A German version [16] of the Florida Affect Battery – Revised [12] was developed and used to test these patient groups systematically. The inclusion of patients with PD was based on the theoretical assumption that in the earliest stage of the disease the pathophysiological processes are generally limited to subcortical structures (substantia nigra and basal ganglia), whereas during the course of the disease functionally related neuroanatomical structures (especially the frontal lobes) are involved [49]. This research design therefore allows some degree of estimation of differential contributions of cortical and subcortical brain structures in affective processing. For the patients with PD, it was hypothesized that patients in the earliest stage of the disease would match the performance of normal control subjects, whereas the results of patients in more advanced stages might be more similar to those of patients with frontal lesions.

Also of special interest was the association between performance in recognition of facial expressions and that of performance in affective prosody (both modalities assessed in the *same* study).

In the light of recent developments on neural differentiation of specific emotions, we also wanted to test whether observed deficits are limited to SPECIFIC EMOTIONS, as recently reported for patients with HD/bilateral amygdala lesions [2, 3, 17, 29].

2. Methods

2.1. Subjects

Thirty-two patients with focal cerebral lesions involving the cortex, 14 non-demented patients with PD and two groups of healthy controls (HC) matched to the patients for age, sex, and IQ took part in the following study. All subjects were right-handed (Edinburgh Handedness Inventory, [48]).

The inclusion of separate control groups for the patients with cortical lesions ($n = 10$) and the patients with PD ($n = 12$) was necessary because PD patients were generally older (about 10 years) than the other patient groups. Age might be an intervening factor in affective processing in clinical groups [68] and should be controlled for. Although the majority of studies failed to report sex differences (e.g., [10]) or correlations with intellectual functioning, the patient groups and HC were matched for these variables.

Exclusion criteria for the HC were (a) a history of psychiatric and/or neurological diseases, and (b) the intake of medication affecting the central nervous system at the time of testing. HC were recruited through advertisements in public institutions and university buildings.

2.1.1. Patients with cortical lesions

Of the 32 patients, 16 had lesions confined to the right hemisphere (R), and 16 patients had suffered damage to the left hemisphere (L). In each of these two subgroups, eight patients had focal lesions of the anterior (A) lobes, and eight patients had lesions limited to posterior (P) brain structures. Etiologies comprised cerebral vascular accidents (stroke), brain tumor removal, contusions, temporal lobe resection because of medically refractory epileptic seizures, and gunshot wound. The patients were recruited from the outpatient clinic of the Department of Neurology, University of Tübingen, Germany. All lesion locations were documented by CT- and/or MRI-scans. More detailed information on the extent of the lesion, e.g., more localized sites within the frontal lobe, were not available. In all cases, however, the lesion did not extend beyond the anterior or posterior cortex and was restricted to one hemisphere. Thirteen patients (40.6%) were taking anticonvulsive medication at the time of testing (phenytoin, carbamazepine, valproic acid) with patient groups not differing in the distribution of medication ($\chi^2_{(12)} = 9.45$, $p = 0.66$). The characteristics of the four patient groups and their matched HC are provided in Tables 1 and 2.

Inclusion criteria for the study were: (a) an interval of at least 12 weeks and at maximum eight years since occurrence of the lesion; (b) no clinically significant aphasic symptoms; (c) no history of psychiatric disorders; and (d) no signs of dementia or neglect.

Table 1
Clinical information (patients with cortical lesions)

Case no.	Interval[a]	Etiology[b]	Lesion site
Right anterior damage			
01	66	REF	fronto-lateral
02	03	CVA	frontal (anterior cerebral artery)
03	06	BTR (astrocytoma)	frontal
04	25	BTR (menigeoma)	fronto-lateral
05	15	CVA	fronto-lateral; involvement of basal ganglia
06	17	CVA	frontal (rostral media artery)
07	12	CON	frontal
08	96	BTR (oligoastrocytoma)	frontal
Right posterior damage			
01	07	GSW	temporo-occipital
02	96	BTR (melanoma)	parieto-occipital
03	19	BTR (menigeoma)	temporo-parietal; involvement of basal ganglia
04	25	BTR (astrocytoma)	parietal
05	05	BTR (astrocytoma)	temporal
06	12	CVA	temporo-parieto-occipital
07	24	CON	temporal
08	22	CON	temporal
Left anterior damage			
01	15	CON	frontal
02	33	CON	ventral frontal
03	14	BTR (glioblastoma)	frontal
04	23	CON	frontal
05	84	BTR (oligoastrocytoma)	frontal
06	17	CVA	rostral frontal
07	75	BTR (oligodendroglioma)	frontal
08	22	BTR (mixed glioma)	frontal
Left posterior damage			
01	04	CON	temporo-occipital
02	15	BTR (gioblastoma)	parieto-occipital
03	91	BTR (astrocytoma)	temporo-occipital; involvement of the basal ganglia
04	60	CVA	parietal
05	03	CON	parietal
06	27	CON	temporo-parietal
07	60	BTR (oligoastrocytoma)	temporal
08	15	CVA	temporo-parietal

[a] Time since lesion (in months).
[b] REF = removal of epileptic focus; BTR = brain tumor removal; CVA = cerebrovascular accident; CON = contusion; GSW = gunshot wound.

2.1.2. Patients with PD

Seven patients with PD fulfilled the criteria for stage I (unilateral symptoms), and seven patients were in stage II (bilateral symptoms) according to the Hoehn and Yahr [35] classification system for severity of motor symptoms. All patients were medicated at the time of testing (a standard combination of levodopa, D2-agonist, and MAO-B-inhibitor), none of the patients had undergone surgical treatment for PD (see Table 3 for patient characteristics). The patients were recruited from the outpatient clinic of the Department of Neurology, University of Tübingen, Germany.

2.2. Materials

2.2.1. Neuropsychological background screening

A short neuropsychological screening battery was administered to all subjects to control for general performance deficits which might influence the affective processing test performance. Basic INTELLECTUAL FUNCTIONING (subtests 'Similarities' and 'Picture completion' of the German version of the reduced Wechsler scale; WIP [21]), ATTENTION SPAN ('digit span' [73]) and MOOD ('Bond-Lader Visual Analogue Scales' [9]; German version [22]) were assessed.

Hearing abilities were tested in a short audiometric examination: inclusion criteria for the following study

Table 2
Demographic and neuropsychological data (patients with cortical lesions and healthy controls)

Variable	RA	LA	RP	LP	HC
sample size (n)	8	8	8	8	10
sex					
female/male	4/4	3/5	5/3	2/6	6/4
handedness					
unilateral right	5	8	7	7	10
ambidextrous	3	0	1	1	0
duration since lesion (in months)					
M	28.9	35.4	26.3	34.4	
SD	33.5	28.0	29.2	32.1	
Range	3–96	14–84	5–96	3–91	
age in years					
M	45.8	40.6	48.4	41.8	44.4
SD	19.3	10.1	11.9	14.1	11.5
Range	22–73	18–53	33–69	26–63	27–70
intelligence verbal					
M	113.5	102.5	110.5	107.9	114.7
SD	11.2	10.8	15.6	8.2	16.9
performance					
M	109.3	109.8	101.5	110.9	109.3
SD	10.9	11.4	8.8	14.0	16.5
attention span					
forward					
M	6.1	6.9	5.9	5.5	6.5
SD	0.8	1.0	1.0	0.8	1.3
backward					
M	4.8	4.5	4.8	4.3	5.6
SD	1.3	0.8	1.5	1.7	1.2
mood[a]					
M	40.4	30.8	29.4	40.3	31.1
SD	18.8	11.3	10.9	9.6	13.0

RA = patients with right anterior damage; LA = patients with left anterior damage; RP = patients with right posterior damage; LP = patients with left posterior damage; HC = healthy controls; n = number; M = mean; SD = standard deviation.

[a] Higher scores reflect more depressed mood.

Table 3
Demographic and neuropsychological data (patients with Parkinson's disease and healthy controls)

Variable	Patients with PD		HC
	PD-I	PD-II	
sample size (n)	7	7	12
sex			
female/male	3/4	3/4	8/4
handedness			
right handed	7	6	12
ambidextrous	0	1	
duration of disease (in months)			
M	44.0	62.4	
SD	28.9	29.5	
Range	14–93	24–102	
age in years			
M	44.6	57.6	47.7
SD	8.5	12.8	13.2
Range	33–55	33–72	27–70
intelligence verbal			
M	113.1	119.7	115.8
SD	5.7	15.6	15.7
performance			
M	121.0	114.6	112.0
SD	10.1	19.3	16.2
attention span			
forward			
M	7.3	6.5	6.3
SD	0.8	1.1	1.4
backward			
M	5.3	5.3	5.3
SD	1.5	1.8	1.2
mood[a]			
M	29.1	33.3	30.6
SD	11.9	16.3	12.2

PD = Parkinson's disease; PD-I = patients in the early stage; PD-II = patients in more advanced stage; HC = healthy controls; n = number; M = mean; SD = standard deviation.

[a] Higher scores reflect more depressed mood.

were that all presented frequencies (500 Hz to 8000 Hz tones) had to be identified with a maximum sound pressure level of 30 dB.

2.2.2. Affective stimuli

A highly reliable German version of the 'Florida Affect Battery – Revised' [12] was used to examine the nature of the perceptual affective deficits in the patient groups ('Tübingen Affect Battery': internal consistency by Cronbach's Alpha $= 0.97$ for the complete battery, with Cronbach's Alpha ranging between 0.86 and 0.94 for the three parts of the battery [16]). A detailed description of the subtests as well as information about test construction is provided elsewhere (English version [7]; German version [16]). The battery includes ten subtests (see Table 4): five subtests require discrimination, naming, pointing to, or matching of FACIAL STIMULI; in three subtests the subjects are asked to discriminate or name LINGUISTIC (1 subtest) and AFFECTIVE PROSODIC (2 subtests) sentences; in the remaining two subtests the subjects are instructed to MATCH a facial expression to one of three affective prosodic sentences or vice versa (crossmodal matching). Every subtest is preceded by several practice items to ensure that the task was understood. Both facial and affective prosodic stimuli depicted one of five different basic emotional categories: happiness, anger,

Table 4
Means and standard deviation (percentage correct responses) for the four patient groups with cortical damage, the patients with PD and the healthy control subjects for the 'Tübingen Affect Battery'

Subtest	RP ($n = 8$)		RA ($n = 8$)		LP ($n = 8$)		LA ($n = 8$)		HC-COR ($n = 10$)		PD-I ($n = 7$)		PD-II ($n = 7$)		HC-PD ($n = 12$)	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Facial tasks																
(total score)	84.9	5.4	83.3	14.0	88.1	9.2	84.1	8.6	95.2	2.6	94.1	3.7	89.7	6.5	95.2	2.4
identity discrimination	95.8	7.1	95.8	4.9	98.3	3.2	98.3	3.2	98.6	3.0	99.0	2.6	98.0	3.4	98.8	2.7
emotion discrimination	81.8	7.7	74.0	24.1	85.0	8.6	77.5	9.6	91.3	6.2	91.4	7.3	87.7	7.1	91.6	5.6
name emotional face	80.0	11.7	84.3	13.7	86.6	15.0	78.4	14.1	94.5	6.2	97.0	3.7	88.6	8.4	94.8	5.8
point to emotional face	87.4	7.4	85.9	12.5	90.0	13.3	85.0	12.5	97.2	3.6	93.3	7.7	93.3	7.7	96.5	3.7
match emotional face	79.4	16.6	76.8	25.9	80.6	17.2	81.5	14.2	94.6	5.2	89.6	6.4	81.0	15.9	94.3	5.3
Linguistic prosody	85.4	20.4	89.6	18.2	91.6	10.0	94.9	14.5	100	0.0	96.6	4.3	97.7	3.9	100	0.0
Affective prosody																
(total score)	83.0	11.1	81.8	13.8	84.0	11.0	82.6	11.0	96.8	3.3	93.5	11.2	84.9	11.5	96.8	3.3
affective discrimination	98.4	4.6	94.1	7.5	95.8	9.5	99.1	2.5	100	0.0	99.0	2.6	100	0.0	100	0.0
name emotional prosody	71.2	19.9	80.0	18.0	75.0	17.8	75.9	23.8	94.6	8.2	91.4	14.7	78.1	18.0	94.9	7.6
name incongruent affective prosody																
– incongruent sentences	68.0	27.0	64.0	32.4	68.0	24.3	67.5	19.1	94.9	4.6	86.6	26.1	66.9	24.4	94.1	6.2
– congruent sentences	94.0	6.4	89.3	10.4	97.0	5.6	87.8	13.2	97.6	5.1	96.6	5.9	94.7	9.8	98.0	4.7
Intermodal tasks																
(total score)	77.2	15.5	75.4	22.8	83.4	12.4	77.9	13.6	97.3	3.9	94.8	4.6	83.9	10.9	97.6	4.1
match prosody to faces	73.5	17.1	71.0	26.3	81.9	9.8	76.6	16.6	97.3	4.6	95.3	6.2	79.1	14.2	95.5	6.6
match faces to prosody	80.9	16.0	79.9	20.1	85.0	18.5	79.1	12.5	97.2	3.6	94.3	5.9	88.6	14.7	97.7	3.4

RA = patients with right anterior damage; LA = patients with left anterior damage; RP = patients with right posterior damage; LP = patients with left posterior damage; HC-COR = healthy controls for patients with cortical lesions; PD = Parkinson's disease; PD-I = patients in the early phase; PD-II = patients in more advanced stage; HC-PD = healthy control subjects for patients with PD; n = number; M = mean; SD = standard deviation.

sadness, fear, and neutral expression. Responses could be made in either verbal or nonverbal (pointing to the emotion word on a vertically-arrayed multiple choice display of five emotion words) manner.

For the German version, all sentences of the Florida Affect Battery [12] were translated to match the English original as closely as possible with regard to content and syllable length. A professional actress was instructed to intonate these sentences with the six different emotions (80 sentences with 16 different emotional or emotional neutral meanings) or to speak them as questions (5 sentences). All sentences were recorded (in multiple repetitions) using digital equipment, and were processed with a sampling rate of 25 kHz ('Computerized Speechlab 4300'; Kay Elemetrics Corp., USA). The authors chose the most characteristic presentation with respect to the intended emotion from the multiple versions of each sentence. The selected items were recorded on an audiotape.

In the next step, all 60 facial stimuli (originals taken from the English version) as well as the 85 prosodic sentences were given to 100 normal control subjects (50 women), and the results subjected to an item analysis. Items with negative or near-zero item-whole correlations were excluded. This resulted in a decreased number of items in the German test battery compared with the English version (15 facial identity stimuli, 30 facial expressions, 45 prosodic sentences). Item difficulties ranged between 68 and 100%, a ceiling effect was expected in this sample of normal controls (see [12]).

2.3. Procedure

After the subjects had provided informed consent, the neuropsychological screening battery was administered (about 20 min), followed by presentation of the 'Tübingen Affect Battery' (about 60 min) in a quiet room. The audiotaped recordings were played on a portable tape player through stereo headphones. The tape was paused when the subject did not respond in the 4 sec interval between stimuli. Both a total score (mean score for all 10 subtests) and sub-scores for each of the four parts (facial expression, linguistic prosody, affective prosody, matching) were calculated for each subject. Travel expenses were reimbursed (DM 30,–).

2.4. Data analysis

All scores (percentage correct responses) were analyzed using one-way or two-way analyses of variance (ANOVA). For post hoc comparisons of paired groups, Tukey's HSD tests were performed.

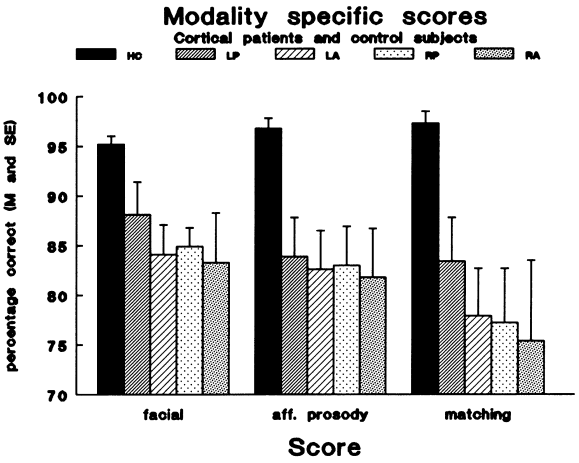

Fig. 1. Percentage correct (mean and standard error) for the modality-specific subscores (facial expressions, affective prosody, matching) of the different cortical patient groups (RA = patients with right anterior damage; RP = patients with right posterior damage; LA = patients with left anterior damage; LP = patients with left posterior damage) and healthy controls (HC).

3. Results

3.1. Patients with cortical lesions

The four patient groups and HC did not differ in the distribution of SEX or HANDEDNESS, nor in AGE, INTELLECTUAL FUNCTIONING, ATTENTION SPAN, or in subjective MOOD (all $p > 0.10$). TIME SINCE LESION ($F(3, 28) = 0.16$, $p = 0.92$) or distribution of LESION ETIOLOGY ($\chi^2_{(12)} = 9.29$, $p = 0.69$) did not differ in the four patient groups.

Group comparisons for the total score and modality-specific scores. Mean and standard deviations for all scores and all groups are displayed in Table 4. ANOVAs revealed significant group differences for the TOTAL score ($F(4, 37) = 3.61, p = 0.01$) as well as for three MODALITY-SPECIFIC (facial expressions, affective prosody, matching) subscores (all $F(4, 37) > 2.98$, $p < 0.03$; see Fig. 1), but not for linguistic prosody ($F(4, 37) = 1.35$, $p = 0.27$). Post hoc analyses (Tukey tests) showed significantly reduced TOTAL performance and significantly lower scores in the MATCHING subscore for both patient groups with right hemisphere lesions (RA, RP) compared to HC (all $p < 0.05$). The RA patient group scored significantly lower than the HC group for FACIAL EXPRESSIONS and AFFECTIVE PROSODY (both $p < 0.05$), patients in the LA group differed from HC in the AFFECTIVE PROSODY subscore ($p = 0.047$). The patient groups did not differ from each other in any of the scores (all $p > 0.81$).

'Double dissociation'. To test the hypothesis that a central processor exists for both modalities (facial expression versus affective prosody), a criterion for 'double dissociation' was developed: test performance for the patient had to be within the range of the HC group in one modality and below the range of the HC in the other modality. Additionally, a critical difference between modality-specific subscores had to be exceeded, e.g., the critical difference had to lie above the greatest difference of the HC (critical difference greater than 4.6%). Results showed that 10 of the 32 patients fulfilled this criterion: six patients presented with a modality-specific impairment for affective prosody (3 LP, 1 LA, 1 RP, 1 RA), and four patients showed specific impairments for facial expressions (2 LA, 2 RP). However, 17 patients showed decreased performance in both modalities ('global affective agnosics'), suggesting dependent processing of both modalities.

Valence hypothesis.[3] An ANOVA with the between-group factor 'group' and the repeated factor 'valence' (angry, happy, sad, frightened, neutral) was conducted (separately for facial expression, affective prosody, and matching scores) to explore whether lesion location is related to performance in specific emotions. A significant interaction of the two factors emerged for facial expressions and affective prosody (both $F(16, 148) > 2.00$, $p \leq 0.02$), but not for the matching condition. The results are presented in Fig. 2.

For FACIAL EXPRESSIONS, significant group differences were found for the *angry* category only ($F(4, 37) = 5.15$, $p = 0.002$). Group differences for *frightened* facial expressions approached significance ($F(4, 37) = 2.28$, $p = 0.08$). Post hoc analyses (Tukey tests) revealed significantly lower scores of the LA and the RP patient groups compared to HC in the anger category (both paired comparisons: $p < 0.01$), but no significant group differences between any of the four patient groups. It should be noted that the identification of angry faces was among the most difficult to recognize emotional category for all patient groups (see Fig. 2).

The significant interaction of 'valence x group' for the AFFECTIVE PROSODY domain was due to significant group differences in the identification of sad and *frightened* intonations (both $F(4, 37) > 4.00$, $p < 0.01$), group differences for *happy* intonations approached significance ($F(4, 37) = 2.41$, $p = 0.07$). Whereas both patient groups with posterior lesions (LP, RP) achieved lower sad scores than the HC group (Tukey tests: both $p \leq 0.02$), scores for the frightened category were significantly reduced for three of the patient groups (LP, LA, RP) compared to the HC group (Tukey tests: all $p \leq 0.03$). No significant group differences emerged between any of the four patient groups. As can be seen in Fig. 2, sad and frightened intonations were *not* the most difficult to recognize emotional categories. The patients' deficit may indicate a failure to process timing information which is the most characteristic acoustic component for both sad and frightened intonations (see Discussion).

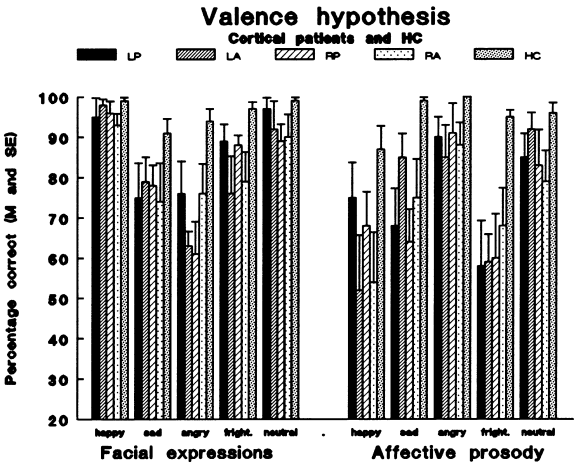

Fig. 2. Percentage correct (mean and standard error) for single emotional categories in facial expressions and affective prosody subtests of the different patient groups and healthy controls (RA = patients with right anterior damage; RP = patients with right posterior damage; LA = patients with left anterior damage; LP = patients with left posterior damage; HC = healthy controls; fright. = frightened).

Etiology of lesion. One possible explanation for the divergence of the present results from those of other research groups (e.g., [7]) could be the heterogeneous lesion etiologies in the present sample. To examine this possibility, the performance of the three most prominent etiological groups (cerebrovascular accident, contusion, brain tumor removal) was compared. ANOVAs revealed significant main effects for both total and composite test scores (all $F(3, 36) > 8.26$, $p < 0.001$). Paired comparisons (Tukey tests) showed that patients with CEREBROVASCULAR ACCIDENTS presented with the lowest performance in both total and subtest scores and differed sig-

[3]The subtests identity and emotion discrimination (facial tasks) as well as affective discrimination (affective prosody tasks) were excluded from valence analyses since findings in patients with cortical lesions indicate that auditory recognition and discrimination may be separate abilities [70, 71].

nificantly from the HC group (all $p < 0.001$) as well as from patients with brain tumor removal (all scores: all $p < 0.02$) or contusion (total score: $p = 0.003$; affective prosody/matching: both $p < 0.01$). Patients following brain tumor removal scored significantly lower than the HC group in both total score ($p = 0.02$) and affective prosody- or matching-subscores (both $p < 0.02$). In summary, patients with cerebrovascular accidents showed the poorest performance.

3.2. Patients with PD

The two patient groups (PD-I and PD-II) and their HC did not differ in distribution of SEX or HANDEDNESS, in INTELLECTUAL FUNCTIONING, MOOD, ATTENTION SPAN or AGE measures (all $p > 0.12$). Patients in the earliest and more advanced stages of the disease were not significantly different with regard to DURATION OF DISEASE ($t(12) = -1.18$, $p = 0.26$).

Group comparisons for the total score and modality-specific scores. ANOVAs revealed significant group differences for both the TOTAL score and modality (FACIAL EXPRESSIONS, AFFECTIVE PROSODY, MATCHING) subscores (all $F(2.23) > 4.00$, $p < 0.03$), but not for the linguistic prosody subscore. Paired group comparisons using Tukey's HSD tests showed that PD-II patients scored significantly lower in all subtests (except linguistic prosody) compared to HC (all $p < 0.03$) and also performed significantly worse than the PD-I group in the matching task ($p = 0.02$). PD-I patients did not differ from HC in any of the measures (see Table 4 for means and standard deviations of all subtests).

Valence hypothesis. A repeated measures ANOVA with 'group' (PD-I, PD-II, NC) as between-group and 'valence' (angry, happy, sad, frightened, neutral) as within-group factors revealed a significant two-way interaction for AFFECTIVE PROSODY ($F(8, 92) = 2.10$, $p < 0.05$; see Fig. 3), but not for the other modalities (facial expressions, matching). The interaction could be explained by group differences for *angry* and *frightened* intonations (both $F(2, 23) > 6.60$, $p < 0.01$). However, group differences for *happy* and *sad* prosody also approached significance (happy: $F(2, 23) = 3.55$, $p = 0.05$; sad: $F(2, 23) = 3.24$, $p = 0.06$). Post hoc analyses (Tukey's HSD test) showed significantly reduced scores of PD-II patients compared to HC in both the angry and the frightened category (both $p \leq 0.01$). For angry prosodic utterances, PD-II patients also scored significantly lower

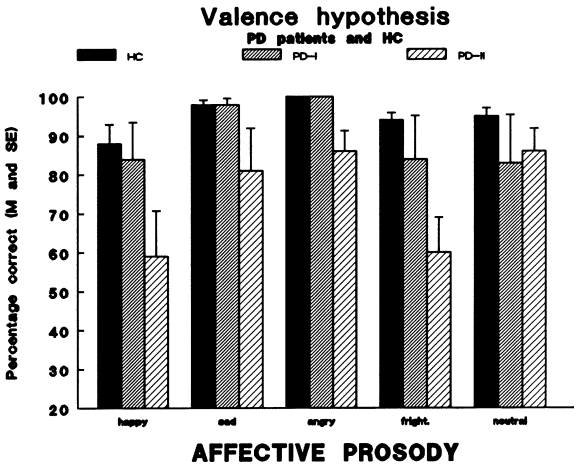

Fig. 3. Figure 3: Percentage correct (mean and standard error) for single emotional categories in the affective prosody subtests of the PD patient groups and healthy controls (PD - I=patients with PD in the early phase; PD-II = patients with PD in more advanced stage; HC = healthy controls; fright. = frightened).

than the PD-I patients (Tukey test: $p = 0.003$). None of the other paired comparisons achieved significance. Recognition of angry intonations was at ceiling level in the HC group and in the PD-I group. The deficit observed in PD-II patients can thus not be explained by increased item difficulty. The deficit in recognizing angry and frightened tones of voice therefore seems to be idiosyncratic for the PD-II patients and may be related to a failure in processing vocal cues of arousal which is the shared acoustic component of these emotions (see Discussion).

4. Discussion

The main aim of the present study was to determine the differential contribution of cortical and subcortical brain structures in emotional processing by comparing patients with focal cerebral lesions and patients with primarily subcortical dysregulation of the basal ganglia (patients with PD). It was assumed that patients with PD with bilateral symptoms would perform similar to patients with focal frontal damage. Before moving on to a general discussion of the results, we will first summarize the main findings for the different patient groups.

Patients with cortical lesions. Results show differential impairment of the patients with cortical lesions compared to HC, with the individual patient groups not significantly differing from one other. Whereas pa-

tients with damage to either anterior or posterior parts of the right hemisphere presented with deficits in both total test score as well as in the intermodal matching subscore compared to HC, only patients with right anterior lesions were impaired in the perception of *facial expressions* compared to HC. The results therefore support an overall RIGHT HEMISPHERE DOMINANCE in the processing of emotional stimulus material. For the affective prosody subscore, however, patients with anterior damage showed the lowest scores, independent of lesion side.

With respect to INTRAHEMISPHERIC LESION site, Ross' assumption of the dominance of the posterior right hemisphere in emotional perception [56] cannot be maintained. Patients with anterior and posterior lesions did not differ and patients with anterior damage showed the strongest impairments in both facial and prosodic emotional recognition, The results for the different etiological groups (cerebrovascular accident, brain tumor removal, contusion) rather suggest an intervening effect of lesion etiology: patients with cerebrovascular accidents showed the lowest test scores. This might be due to the more extensive lesions caused by ischemia compared to brain tumor removal or contusion. Further research should continue to investigate the 'etiology hypothesis'.

The present study also aimed to investigate whether the recognition of facial expressions and affective prosody are dependent or MODALITY-SPECIFIC. Ten of the 32 cortical patients showed a 'double dissociation' of facial and affective prosodic impairments which was, however, not clearly related to lesion site. Further interpretation of this result with regard to distinct processors for facial and affective prosodic perception is therefore not possible. Furthermore, the means of all four patient groups were similar to each other and were lower than those of the HC. One possible explanation could be that brain damage per se, independent of lesion site or side, affects emotional perception.

No significant group differences were found for the *linguistic prosody* subscore. The finding supports the assumption of SEPARATE CORTICAL MODULES FOR LINGUISTIC AND EMOTIONAL PROSODIC FUNCTIONS [52] and indicates that our patients did not present with a general impairment of processing complex auditory signals.

Noteworthy results emerged from the ANALYSES OF SPECIFIC EMOTIONS, indicating that the recognition of angry (and to a lesser extent frightened) FACIAL EXPRESSIONS were most affected in the cortical patients. The impairments were, however, not distinctly related to lesion site and are at variance with the findings by Adolphs et al. [4] that patients with damage to the left hemisphere perform within the normal range for all emotions. Our findings indicate that patients with cortical lesions were especially impaired with respect to facial emotions that are related to arousal and threat and did not show any deficits for happy facial expressions. Further interpretation of our findings is limited by the factor that intrahemispheric lesions of our patient samples were quite extensive and more detailed analysis of the locus of the damage was not possible. For AFFECTIVE PROSODY, both posterior groups presented with impaired recognition of sad intonations compared to HC and all patient groups (except the RA group) achieved lower scores for frightened prosody than the HC group. Different emotions can be described by different sets of acoustic parameters, with speech rate and pitch exerting the strongest effect on listeners' judgements [59, 60]. In the temporal domain, a slow speech rate is perceived as sad and a fast rate as frightened or happy. The specific pattern of deficits thus indicates that the patients may fail to appropriately process timing information in the speech signals. Future studies should address this issue, for example, by systematically varying temporal information in prosodic stimuli.

Patients with PD. A differential pattern of impairment could be found for PD-patients with uni- and bilateral motor symptoms. Patients in the more advanced stage of the disease performed worse in both total test score as well as in the modality-specific subscores (FACIAL EXPRESSIONS, AFFECTIVE PROSODY, MATCHING) than the HC. Furthermore, in the matching subtest, which requires the integration of two different modalities (visual, acoustic), patients in more advanced stages were significantly more impaired than patients in the early stage. Patients with unilateral symptoms did not differ in any of the scores from the HC group, and their scores ranged between those of the patients in more advanced stages and the matched HC.

With respect to SINGLE EMOTIONS, a differential deficit pattern was only observed for the AFFECTIVE PROSODY subtests. PD patients in the more advanced stage were especially impaired in recognizing angry and frightened (and to a lesser extent happy) intonations. The common acoustic feature of these emotions is high mean pitch which is related to the speaker's arousal [60]. It is therefore possible that the PD-II patients present with a failure to process vocal cues of arousal, an issue that warrants attention in future

studies. Since our design did not include the category disgust, our findings in patients with PD cannot directly be compared to those reported in patients with HD [65]. Similarly to patients with HD, the PD-II patients achieved the lowest scores for frightened and happy vocal emotions, but those were also the two most difficult categories for our HC. In summary, our findings do not provide clear evidence for an especially severe deficit for one particular emotion in PD.

As patients and HC did not significantly differ from each other in any of the NEUROPSYCHOLOGICAL background or MOOD variables and patients with lasting visual neglect were excluded (see [51]), the observed differences between the groups in the emotional tasks can neither be explained with general attentional or intellectual impairments, nor with mood changes in the patient groups.

SUMMARIZING the results for all clinical groups, patients in more advanced stages of PD showed the same general pattern of impairment as the patients with (right) anterior cortical lesions. The assumption of an important contribution of the fronto-striatal circuitry in emotional, especially in affective prosodic functions (e.g., [18]) is supported by the present data, and the observed deficits are also consistent with those of Scott et al. [62] and Pell [50] in patients in (presumably) more advanced stages of PD. A predominantly unilateral involvement of the basal ganglia (PD-I patients), however, exerted a weaker effect on patients' performance in emotional processing.

Alexander et al. [5] described two parallel prefrontal loops: the so-called 'dorsolateral prefrontal' circuit, which is associated with spatial memory and executive functions [44], and the 'lateral orbitofrontal' circuit, which receives input from both auditory and visual association areas of the temporal lobe and which contributes to cognitive and emotional functions [20]. A third basal ganglia loop, the 'limbic' circuit, receives input from the amygdala (e.g., via the ventral striatum, linking medial temporal lobe structures to orbitofrontal regions) and is considered to contribute to affective/motivational processes and divided attention. A contribution of these basal ganglia loops, particularly the 'LATERAL ORBITOFRONTAL' circuit with its involvement in emotion-related learning and social behavior [36, 44, 54, 55], could explain the present finding that predominantly patients with anterior cortical lesions as well as patients with more advanced PD presented overall with the strongest deficits in the processing of emotional stimulus material. The present study therefore provided further evidence for an involvement of the fronto-striatal circuitry in emotional functions [40, 75].

Unfortunately, the present investigation was planned and carried out before reports on disgust-specific deficits were reported in HD gene carriers [29] and our stimulus material did not include the emotional category disgust. Future studies with PD patients should therefore address the issue of 'the importance of the basal ganglia in the emotion of disgust' [29, p. 2036]. A further issue should be to examine the possible association of emotional processing and other neuropsychological functions [27].

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