

Psychological Medicine, 2003, **33**, 1453–1462. © 2003 Cambridge University Press
DOI: 10.1017/S0033291703008134 Printed in the United Kingdom

Kinematical analysis of emotionally induced facial expressions in patients with obsessive–compulsive disorder¹

R. MERGL,² M. VOGEL, P. MAVROGIORGOU, C. GÖBEL, M. ZAUDIG,
U. HEGERL AND G. JUCKEL

From the Department of Psychiatry, Ludwig-Maximilians-Universität München, Laboratory of Clinical Neurophysiology, München; Pfizer Inc., Karlsruhe; Psychosomatic Hospital Windach, Windach; and Department of Psychiatry, Charité, Humboldt Universität zu Berlin, Berlin, Germany

ABSTRACT

Background. Motor function is deficient in many patients with obsessive–compulsive disorder (OCD), especially in the face. To investigate subtle motor dysfunction, kinematical analysis of emotional facial expressions can be used. Our aim was to investigate facial movements in response to humorous film stimuli in OCD patients.

Method. Kinematical analysis of facial movements was performed. Ultrasound markers at defined points of the face provided exact measurement of facial movements, while subjects watched a humorous movie ('Mr Bean'). Thirty-four OCD patients (19 male, 15 female; mean (s.d.) age: 35.8 (11.5) years; mean (s.d.) total Y-BOCS score: 25.5 (5.9)) were studied in unmedicated state and after a 10-week treatment with the SSRI sertraline. Thirty-four healthy controls (19 male, 15 female; mean (s.d.) age: 37.5 (13.1) years) were also investigated.

Results. At baseline, OCD patients showed significantly slower velocity at the beginning of laughing than healthy controls and a reduced laughing frequency. There was a significant negative correlation between laughing frequency and severity of OCD symptoms. Ten weeks later a significant increase of laughing frequency and initial velocity during laughing was found.

Conclusions. Execution of adequate facial reactions to humour is abnormally slow in OCD patients. Susceptibility of OCD patients with regard to emotional stimuli is less pronounced than in healthy subjects. This phenomenon is closely correlated to OCD symptoms and is state-dependent.

INTRODUCTION

Obsessive–compulsive disorder (OCD) is a prevalent psychiatric disease (Rasmussen & Eisen, 1990), with intrusive thoughts (obsessions) and ritualistic repetitive behaviours (compulsions) as the main symptoms. Many OCD patients suffer from motor disturbances: abnormal involuntary movements are found in

about one-third of OCD patients, disturbances of fine motor coordination were present in 53.7% of OCD patients (Hollander *et al.* 1990; Mavrogiorgou *et al.* 2001).

As in patients with Parkinson's disease, some of OCD patients have been reported to exhibit abnormal facial activity, including rigidity of the face and a mask-like face (Schilder, 1938). Schilder's case reports are interesting because they suggest the presence of basal ganglia dysfunction in OCD. There are several findings suggestive of this, including brain-imaging reports that show reduced caudate volume in many patients with OCD (Luxenberg *et al.*

¹ Presented in part at the 7th World Congress of Biological Psychiatry, Berlin, Germany, 1–6 July 2001.

² Address for correspondence: Dr Roland Mergl, Department of Psychiatry, Ludwig-Maximilians-Universität München, Laboratory of Clinical Neurophysiology, Nussbaumstrasse 7, D-80336 München, Germany.

Table 1. Description of the sample

Variables	OCD patients (N = 34)	Healthy controls (N = 34)	Intergroup comparison
Age (years), mean (s.d.)	35.82 (11.54)	37.53 (13.08)	$t = -0.57$ NS
Gender (male/female)	19/15	19/15	—
Dominant hand (right/left)	31/3	28/6	$\chi^2 = 0.64$ NS
High school level (yes/no)	10/24	26/8	$\chi^2 = 17.58$ ***
Age at the onset of OCD (years), mean (s.d.)	22.11 (12.09)	—	—
Duration of disease, mean (s.d.)	13.72 (9.51)	—	—
Positive family history (yes/no)	17/17	—	—

*** $P < 0.001$; NS, not significant.

1988; Robinson *et al.* 1995), association of OCD with basal ganglia disorders, as in Sydenham's chorea (Swedo *et al.* 1989) and Tourette syndrome (Pauls *et al.* 1986), and higher prevalence of motor signs reflecting basal ganglia dysfunction in OCD patients, as compared to healthy controls (Hymas *et al.* 1998).

Analysis of emotionally induced facial movements is a promising tool for the assessment of abnormal facial activation in OCD. Facial expressions of basic emotions (e.g. joy, anger, disgust) result from movements of the facial muscles that are innervated by the facial nucleus, which is highly connected to the limbic system (Rinn, 1984). The nucleus accumbens, the so-called limbic-motor interface (Mogenson *et al.* 1980) plays a relevant role in starting motor programmes elicited by emotional processes in the limbic system (Heimer *et al.* 1982). Since the serotonergic system is involved in the modulation of neuronal excitability of the nucleus accumbens (Filip & Cunningham, 2002) and of the motor system (Jacobs & Fornal, 1997) as well as in the pathogenesis of OCD (compare Hegerl & Mavrogiorgou, 1999), it may be speculated that involuntary motor disturbances in OCD patients may be triggered by disturbances of serotonergic neurotransmission. Other neurotransmitter systems like dopamine (McDougle *et al.* 1990) might also be involved in these disturbances.

To investigate facial expression in OCD, objective measures are needed, which provide analysis of starting a facial movement. This can be performed by using a new computer-based and very sensitive method (see Juckel & Polzer, 1998; Padberg *et al.* 2001), providing the exact registration of the initial velocity of oro-facial (laughing) movements elicited by emotional

(humorous) stimuli. In this context, the problematical idiosyncrasies of humour (especially high inter-individual variability of sense of humour) must be taken into account. Laughing has been proposed to be a biological and psychological act that can be found in humans as well as great apes (Provine, 2000). Considering the brain mechanisms of laughter, laughing movements have been induced by electrical stimulation at specific cortical sites including the anterior cingulate and the orbitofrontal cortex (Sem-Jacobsen, 1968), the basal temporal lobe (Arroyo *et al.* 1993) as well as the anterior part of the supplementary motor area (Fried *et al.* 1998). Since abnormal or lack of facial responses to emotional stimuli have negative implications to well-being and social interaction (Schulman-Green, 1999) we planned to investigate facial movements in response to humorous film stimuli in healthy subjects and OCD patients tested before and during therapy with a serotonin reuptake inhibitor (sertraline), using a kinematical approach. To our knowledge, it is the first study that examines laughter responses to humorous stimuli in OCD patients with the use of ultrasonic markers.

METHOD

Subjects

Thirty-four adult patients, who met DSM-IV criteria for OCD, participated in the study. The healthy controls were volunteers who were asked by mail to participate in our study. Their addresses have been obtained from the registration office. The normal controls were comparable to the patients with regard to age, gender and handedness (Table 1). They were not medicated and they had no DSM-IV diagnosis

of mental disorders. Exclusion criteria for the healthy subjects included medical disease at study time.

All subjects gave written consent according to the guidelines found in the Declaration of Helsinki (World Medical Association, 1997). At the time of the study all patients were treated in the Psychosomatic Hospital, Windach. The severity of OCD symptoms was assessed using the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) (Goodman *et al.* 1989*a, b*), their degree of depression by use of the Hamilton Depression Rating scale (17-item version) (HDRS) (Hamilton & Guy, 1976) and their severity of disease was evaluated with the Clinical Global Impressions severity and improvement scores (CGI) (Guy, 1976). At baseline, the OCD patients had a mean (s.d.) total Y-BOCS score of 25.5 (5.9), with their score for obsessions being 13.4 (3.0), for compulsions 12.1 (3.7). Their mean (s.d.) HDRS score was 13.4 (6.6) and their mean (s.d.) CGI score was 5.5 (1.1).

All OCD patients were medication free for 14 days prior to the investigations. No patient had been treated with long-acting psychotropic drugs known to have effects on motor control even several weeks after termination of treatment (e.g. neuroleptics). The OCD patients had neither focal neurological disease nor any other major medical disease at time of the study. Obsessions with respect to laughing, watching films, or being observed or being studied have not been found in the patients' sample.

Study design

All OCD patients were screened in the first 2 days after admission into the hospital. After baseline, the OCD patients were treated with sertraline (daily oral dose: 50 mg; after 4 and 7 weeks, the dose could be raised by 50 mg, respectively) and a semi-standardized behaviour therapy (Hauke *et al.* 1998). Ten weeks later, the facial investigations were repeated.

Kinematical analysis of facial expression

Analysis of facial movements was performed as described previously (Padberg *et al.* 2001), using an active device for the measurement of movements (CMS 70; ZEBRIS Ltd., Tübingen, Germany). Special microphones registered high-frequency ultrasonic signals (35 kHz) emitted by

special markers. These signals were registered online with a sampling rate maximally of 200 Hz/number of markers. The digital evaluation of these signals was performed offline, thus allowing the exact, three-dimensional registration of the coordinates of the ultrasonic markers with very high temporal (a few milliseconds) and spatial resolution (0.1 mm). Disturbing head movements were extracted using one reference marker positioned on a tape at the forehead and serving as a reference to differentiate head from facial movements. The ultrasonic markers were fixed by double-faced adhesive tapes to the right and left corner of the mouth, thus providing registration of facial movements caused by activity of the zygomaticus major and minor, risorius and depressor anguli oris muscles. Two further markers were fixed to the left and right medial inferior rim of the eye to record activity of the orbicularis oculi muscle. All these muscles are innervated by the nervus facialis. The experimenter had been trained to place the markers in the same spots before the start of the study in order to ensure comparability of measurements.

The subject sat on a comfortable chair in front of a device used to record the ultrasonic signals emitted by the facial markers, a video-camera and a television screen (for presentation of the emotional stimuli). Two 'Mr Bean' sketches were shown to induce the positive emotion 'humour'. After presentation of the sketches (maximal duration, 9 min), the subject was asked to assess how funny the sketches were, using a visual analogue scale (VAS) ('degree of funniness'; length: 16 cm).

During this investigation, facial activity was registered, using the ultrasonic measurement system and simultaneously video-recording of the subject's face was performed in order to assign the emotional reactions to the kinematical data exactly. In order to draw a clear dividing line between involuntary facial activity elicited by emotional stimuli ('Mr Bean') and voluntary movements of the mouth angles and the eyes, the subjects were asked to stretch their mouth angles and to close their eyes tightly after presentation of the sketches. After the investigation, the humorous movie, video-recording of the face and facial activity, as measured by the ultrasonic markers, were synchronized using a frame code generator, thus allowing exact analysis

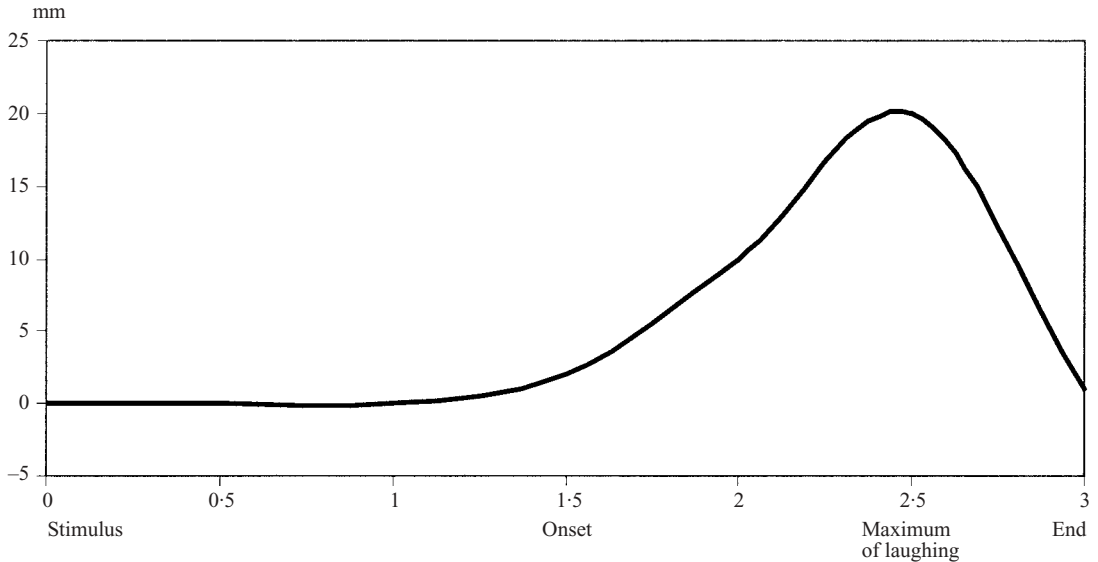


FIG. 1. Schematic representation of laughing movements after presentation of an emotional stimulus.

of the dynamics of laughing facial movements closely connected to the emotional stimuli.

Offline analysis of facial activity has been established by a special computer software (3DA). The digitalized signals of each ultrasonic marker were graphically represented as a spatio-temporal function and filtered. Artefacts have been corrected; a special algorithm representing sliding averaging of data during simultaneous data recording (compare Hermsdörfer *et al.* 1996) provided valid filtering of the data. 'Laughing' has been evaluated in five film sequences by visually detecting synchronous changes of facial activity in the video-recordings and subsequent kinematical analyses of facial movements in the correspondent time interval. Three independent normal raters had previously assessed these film sequences to be especially humorous by use of a visual analogue scale. The laughing movement can be graphically represented as a spatio-temporal measuring curve using 3DA (Fig. 1).

In a first step, frequency of laughing, the number of laughing reactions to the movie, related to the length of the movie (N/min movie), has been determined for each subject. In a second step, the movements of the markers, corresponding to 'laughing' (controlled by a simultaneous video-recording of the face) have

been analysed. The following kinematical main variables shown to be sensitive indicators of facial movements elicited by positive emotion in previous studies (e.g. Juckel *et al.* 1999; Padberg *et al.* 2001; Juckel, 2002) have been computed for each marker using 3DA: the initial velocity of the laughing movement, defined as the velocity (mm/s) of each marker at the beginning of laughing and calculated as slope through the start and maximum (space and time) point of the laughing movement (mean of three); emotional reaction time (ERT) i.e. time between a humorous stimulus in the film (as defined by healthy subjects prior to the study) and the reaction, i.e. onset of a laughing movement of the left and right corner of the mouth (mean of five humorous stimuli per movie) (in seconds).

Statistical evaluation

Mean kinematical parameters of facial expression were analysed separately, using the statistical package SPSS for Windows (version 10.0). The dependent variables were submitted to a 2×2 analysis of variance (MANOVA), in order to compare between two groups (OCD patients, healthy controls) with respect to the corner of the mouth and the rim of the eye (left, right). Greenhouse–Geisser correction of the degrees of freedom was applied. Main effects of

Table 2. Mean facial parameter scores for the right and left side by diagnostic group

	OCD patients (N=34)		Healthy controls (N=34)		Group P	Group × side P
	Right Mean (s.d.)	Left Mean (s.d.)	Right Mean (s.d.)	Left Mean (s.d.)		
Initial velocity (mm/s)						
Mouth angle marker (laughing)	5.76 (3.03)	5.64 (2.75)	8.57 (5.67)	8.26 (4.12)	**	NS
Stretching of the mouth angles	18.51 (9.63)	16.94 (7.08)	22.14 (14.99)	20.64 (15.20)	NS	NS
Eye marker (laughing)	3.50 (1.83)	3.66 (1.85)	5.33 (2.72)	5.35 (2.11)	**	NS
Tight closing of the eyes	18.84 (7.66)	18.85 (8.73)	22.34 (11.89)	21.42 (11.39)	NS	NS

Two-way variance analysis for repeated measures was computed for the initial velocity of facial movements.

** $P < 0.01$; NS, not significant.

the group reflect abnormal facial activity, group and side interactions indicate abnormal motor asymmetries in patients with OCD.

The influence of age, age at the onset of OCD and the duration of disease on kinematical parameters of facial expression was studied by means of Pearson's correlation. We also computed Spearman-Brown correlations between clinical rating scale scores (Y-BOCS, HAMD-17, CGI total scores) and the initial velocity of laughing. Chi-square analysis was performed in order to answer the question whether the initial velocity of laughing is independent from gender or family history of psychiatric disease.

Changes of facial activity during treatment were studied in OCD patients by applying *t* tests for paired samples (baseline *versus* 10th week).

In view of the number of main outcome measures (initial velocity of laughing, emotional reaction time and laughing frequency) raising the statistical problem of multiple comparisons, in all statistical analyses, the significance level was alpha-adjusted and taken at 0.0167 (0.05/3).

RESULTS

OCD patients *versus* healthy controls

Initial velocity of laughing and voluntary facial movements

Mean kinematical scores of facial expression (initial velocity of different facial movements) are represented in Table 2. Analysis of variance (compare Table 2) and *post hoc t* tests revealed that OCD patients exhibited significantly slower initial velocities of involuntary facial movements ('laughing') than healthy controls (left eye, $t(1, 45) = -2.83$, $P = 0.007$; right eye, $t(1, 45) = -2.56$, $P = 0.014$; left angle of the mouth, $t(1, 66) = -3.08$, $P = 0.003$; right angle of the

mouth $t(1, 66) = -2.55$, $P = 0.013$) (Fig. 2), while the group differences in voluntary movements (closing of the eyes; stretching of the mouth angles) were not significant ($-1.14 \leq t(1, 40 \leq 56) \leq -0.83$; $0.26 \leq P \leq 0.42$).

Regarding motor asymmetries (differences between right and left markers in the initial velocity of movements of the face), anomalous side differences could not be found for patients with OCD, as reflected by non-significant group × side interactions in the MANOVA (Table 2).

In line with these results, *post hoc t* tests for paired samples did not reveal any significant differences between right and left markers for the initial velocity of facial movements under different task conditions (laughing, stretching of the mouth angles, tight closing of the eyes) for patients with OCD ($-1.40 \leq t(18 \leq 33) \leq 1.39$; $0.17 \leq P \leq 0.996$) and healthy controls ($-1.25 \leq t(21 \leq 33) \leq 0.05$; $0.23 \leq P \leq 0.96$).

Emotional reaction time

Differences between OCD patients and healthy controls regarding emotional reaction time were not significant (left corner of the mouth, $t(1, 65) = -0.89$, $P = 0.38$; right corner of the mouth, $t(1, 65) = -1.03$, $P = 0.31$), with the OCD patients exhibiting somewhat lower values (mean ERT for the left corner of the mouth (s.d.) = 1.20 (0.53) s; right corner of the mouth = 1.19 (0.53) s) than the healthy controls (mean ERT for the left corner of the mouth (s.d.) = 1.33 (0.63) s; right corner of the mouth = 1.34 (0.64) s).

Laughing frequency

The mean VAS scores were comparable in OCD (mean VAS score (s.d.) = 10.03 (3.39); range, 2.4–15.5) and healthy subjects (mean VAS score

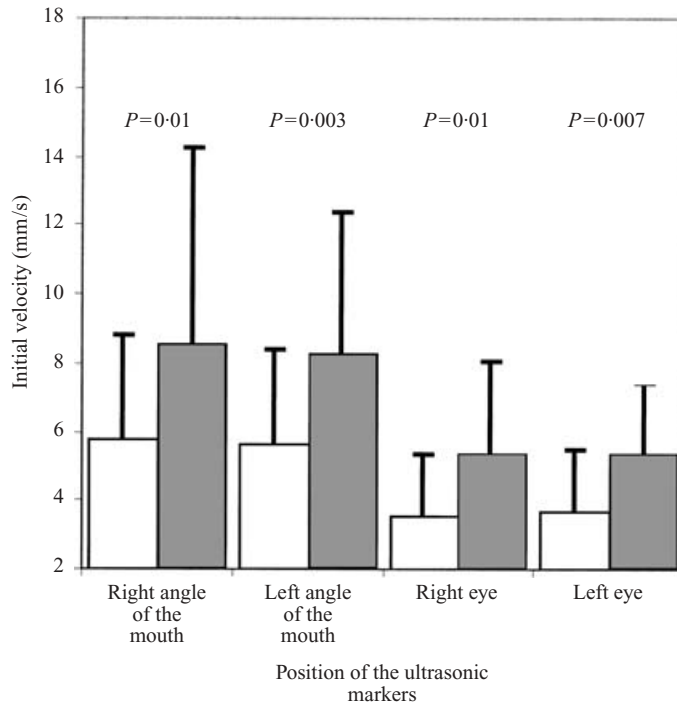


Fig. 2. Mean (s.d.) initial velocity of laughing movements elicited by watching of a humorous movie ('Mr Bean') in 34 patients with OCD (□) and 34 healthy controls (■), as measured by use of ultrasonic markers at different positions of the face (right and left angle of the mouth; medial inferior rims of the left and the right eye). *P* values indicate that healthy subjects laughed significantly faster than OCD patients in this situation.

(s.d.) = 11.32(3.20); range, 0.2–15.1), suggesting that both groups assessed the movies to be humorous in similar intensity ($t(1, 64) = -1.59$, $P = 0.12$). However, frequency of laughing reactions was significantly lower in OCD patients (mean laughing frequency (s.d.) = 13.85(7.79); range, 3–26), as compared to healthy controls (mean laughing frequency (s.d.) = 24.41(10.15); range, 3–45) ($t(1, 66) = -4.81$, $P \leq 0.001$).

Besides the documented statistically significant differences, there was no clinical or interpersonal significance noted in the differences in facial movements found with this ultrasound technique (like sense of oddness on patient recruitment that might reflect decreased 'humorous stimuli' responses).

Correlation of facial movements with demographic and clinical factors in patients with OCD symptoms

In OCD patients, Pearson's correlations between facial and emotional parameters (VAS,

emotional reaction time, laughing frequency and initial velocity of different facial movements) and three other variables (age, age at onset of disease and duration of OCD) indicated that there was no significant association between these variables (age, $-0.24 \leq r_p \leq 0.35$; $0.13 \leq P \leq 0.87$; age at onset of disease, $-0.20 \leq r_p \leq 0.29$; $0.22 \leq P \leq 0.93$; duration of OCD, $-0.23 \leq r_p \leq 0.23$; $0.20 \leq P \leq 0.84$).

In line with these findings, comparison of 17 OCD patients with early onset of disease (<21 years) with 17 OCD patients with later onset of OCD (>21 years) demonstrated that both groups exhibited comparable aspects of facial expression ($-1.29 \leq t(1, 17 \leq 32) \leq 1.91$; $0.07 \leq P \leq 0.93$).

As revealed by *t* tests for independent samples the effects of gender, educational level and family history of psychiatric disease on facial parameters (including VAS) were rather low in patients with OCD (gender, $-0.92 \leq t(1, 17 \leq 32) \leq 1.32$; $0.20 \leq P \leq 0.95$; high school

level, $-0.83 \leq t(1, 17 \leq 31) \leq 1.19$; $0.24 \leq P \leq 0.87$; family history of psychiatric disease, $-1.54 \leq t(1, 17 \leq 32) \leq 1.76$; $0.09 \leq P \leq 0.97$).

Baseline scores on psychopathological rating scales (Y-BOCS, HAMD-17, CGI) were correlated with facial parameters by Spearman's analysis. The higher the total baseline Y-BOCS score, the lower the laughing frequency ($r_s = -0.43$, $P = 0.011$) and the higher the initial velocity of voluntary movements of the right angle of the mouth ($r_s = 0.43$, $P = 0.02$). The latter variable showed also significantly positive correlation with the baseline Y-BOCS scores for the intensity of obsessions ($r_s = 0.40$, $P = 0.02$). Moreover, there were significant negative Spearman-Brown correlations between the total baseline Y-BOCS score for the severity of compulsions and the visual analogue scale ($r_s = -0.45$, $P = 0.008$) and the laughing frequency at baseline ($r_s = -0.52$, $P = 0.002$).

The total baseline HAMD-17 score did not significantly correlate with parameters of facial movements ($-0.18 \leq r_s \leq 0.34$; $0.06 \leq P \leq 0.99$). The higher the baseline CGI score, the lower laughing frequency at baseline ($r_s = -0.46$, $P = 0.006$). Other significant associations between the baseline CGI score and variables of facial expression could not be detected.

DISCUSSION

Interpretations

Abnormal facial expression in patients with OCD

We have shown that OCD patients exhibited significantly slower initial velocities of involuntary laughing movements than healthy controls, with initial velocity of voluntary facial movements (like closing of the eyes) being comparable in both groups. Patients with OCD and healthy controls did not significantly differ in laterality of voluntary or involuntary facial movements. So, distinctive facial features seem not to be restricted to one side in patients with OCD.

Emotional reaction time and judgements of the humour of the movie cannot account for slower initial velocity of involuntary facial movements in OCD patients because they were comparable with the normal controls. One could assume that slower reactions on emotional stimuli in OCD ('subcortical' laughing) resulted

from disturbances of attention. However, this explanation is not convincing because there were no significant differences between OCD patients and healthy controls in the initial velocity of voluntary facial movements in 'cortical' tasks (e.g. stretching of the mouth angles). Instead, level of education, which was much higher in healthy subjects as compared to OCD patients, might represent an important confounding variable. However, it must be emphasized that slower facial reactions on film stimuli in OCD patients were independent of the level of education.

Further analysis revealed that all subjects (patients and controls) were susceptible regarding humour (minimum laughing frequency = 3). However, healthy subjects laughed significantly more frequently than OCD patients in the experimental situation. OCD patients seemed to be less susceptible than healthy controls with regard to emotional stimuli. Laughing frequency and initial velocity of 'laughing' movements were not significantly correlated in OCD patients. Therefore, they seem to reflect different aspects of abnormal facial expression: low susceptibility regarding emotional stimuli and abnormally slow execution of adequate motor reactions on humorous stimuli. The latter finding may reflect hypomimia (low degree of involuntary movements of the face) as part of a parkinsonoid in subclinical intensity. In fact, older studies (Schilder, 1938) suggest that hypomimia is a quite frequent symptom in OCD patients.

Transformation of the feeling of 'humour' into the motor reaction 'laughing' itself was not disturbed in OCD patients (as indicated by normal emotional reaction time). The morphological substrate of this process might be the nucleus accumbens, the so-called limbic-motor interface (Mogenson *et al.* 1980). Since the commencement of these motor programmes seems to be comparable in OCD patients and healthy controls, it is unlikely that functional disturbances exist of the limbic-motor interface in OCD patients. In fact, functional disturbances or structural lesions of the nucleus accumbens have, so far, not been reported in OCD patients.

Instead, it can be assumed that basal ganglia dysfunction underlies disturbances of facial expression (especially hypomimia) in OCD patients since hypomimia is a well-known

phenomenon in patients with Parkinson's disease. Moreover, several studies suggest that basal ganglia dysfunction is an important factor in the pathophysiology of OCD (Saxena *et al.* 1998). However, it is also important to account for the role of transmitter systems in OCD pathophysiology: serotonin (Hegerl & Mavrogiorgou, 1999); dopamine (McDougle *et al.* 1990); and acetylcholine (Lucey *et al.* 1993).

Correlates of abnormal facial expression in patients with OCD

The initial velocity of facial reactions on emotional stimuli was independent from age, gender, educational level and some clinical variables, namely duration of OCD, age at onset of OCD, family history of psychiatric disease and severity of depression in OCD. However, higher severity of disease (higher baseline CGI score) went along with lower laughing frequency at baseline. The latter variable was also associated with the Y-BOCS score. Higher initial velocity of voluntary movements of the right angle of the mouth was associated with higher intensity of obsessions. However, this finding should be interpreted with much caution because it was a single finding and correlation was rather low ($r_s \leq 0.43$).

Overall, initial velocity of involuntary 'laughing' movements of the face seems to be independent from psychopathology in OCD patients. In contrast, laughing frequency was found to be closely associated with OCD symptoms. Therefore, susceptibility regarding emotional stimuli seems to be lower in OCD patients who are severely ill compared to patients with OCD symptoms of mild to moderate severity.

Changes of facial movements

In an exploratory analysis, changes of facial movements during treatment with sertraline and behavioural therapy in OCD patients had been analysed and a significant increase of laughing frequency ($P < 0.001$), emotional reaction time ($P < 0.05$) and the initial velocity of involuntary movements of the right eye during laughing ($P < 0.05$) had been found. Moreover, the amount of clinical remission (as reflected by positive Y-BOCS difference scores) was related to increase in the initial velocity of 'laughing'

movements of the left eye ($r_s = 0.35$, $P < 0.05$) in patients with OCD.

Facial muscle function in OCD and other psychiatric diseases

Overall, our results (normal emotional reaction time, low laughing frequency, slower initial velocity of involuntary facial movements, but normal initial velocity of voluntary facial movements in patients with OCD) primarily reflect hypomimia in OCD patients. In this regard, relevant differences between OCD patients, schizophrenic patients and patients with depression are apparent: unmedicated schizophrenic patients exhibited significantly faster initial velocity of laughing than controls (Juckel *et al.* 1999), with this finding representing the phenomenon of facial disintegration (Heimann & Spoerri, 1957). In contrast, depressed patients were characterized by slower initial velocity of voluntary and involuntary facial movements (Juckel, 2002) that reflect psychomotor retardation. The latter phenomenon is quite frequent in patients with depression (compare Sabbe & Hulstijn, 2001). Our results suggest that only involuntary movements of the face are essentially disturbed in OCD patients, these disturbances are not an epiphenomenon of depression in OCD and they do not resemble the signs of facial disintegration in schizophrenic patients.

Limitations

Our findings are interesting, but should be viewed with caution. First, humour is a phenomenon with problematic idiosyncrasies and it is well-known that responses to humorous movies vary considerably with individuals. Secondly, since both sertraline and semi-standardized behaviour therapy were given over our 10-week study, it is impossible to know if sertraline had any independent effect, let alone how much of the effect was actually related to serotonin function. In order to clarify how much of the apparent increase in facial muscle movements could be accounted for simply by the passage of time, or the fact that a different film was shown at baseline and 10 weeks later, it would have been necessary to retest the healthy volunteers. In addition, the lack of an adequate placebo treatment group (like placebo pill and

waiting-list behaviour therapy) or untreated patient controls does not allow us to conclude that a serotonin-enhancing therapy induced follow-up changes of facial parameters in OCD. Also, the OCD patients were admitted for the total duration of the study (10 weeks), which might have affected their response to humour. In summary, it is not clear whether practice, film version or treatment are responsible for the follow-up changes of facial expression in OCD patients. Further methodological limits of our study that need more investigation are: our sample size was quite small; and, we did not directly compare OCD patients with other psychiatric or neurological patient populations.

Future directions

Further studies of OCD subgroups are needed to develop a more differentiated picture of emotionally induced facial expressions in OCD patients. To examine whether basal ganglia dysfunction represents the morphological substrate of abnormal facial expression in OCD, it will be important to investigate the association of kinematical parameters of facial expression with findings from brain imaging. Finally, another study with a more rigorous design (double-blind, placebo-controlled, randomized; OCD patients and healthy controls tested twice with half the group being shown one film version and the other half the other version) should be conducted to address the question of whether follow-up changes of facial movements in OCD patients are due to serotonin-enhancing therapy.

This study was kindly supported by Pfizer Germany, Inc. The evaluation of the present data was performed by Michael Vogel as part of his M.D. thesis. We gratefully acknowledge the staff of the Psychosomatic Hospital Windach as well as the OCD patients and healthy subjects for their willingness to participate in our study.

REFERENCES

- Arroyo, S., Lesser, R. P., Gordon, B., Uematsu, S., Hart, J., Schwerdt, P., Andreasson, K. & Fisher, R. S. (1993). Mirth, laughter and gelastic seizures. *Brain* **116**, 757–780.
- Filip, M. & Cunningham, K. A. (2002). Serotonin 5-HT(2C) receptors in nucleus accumbens regulate expression of the hyperlocomotive and discriminative stimulus effects of cocaine. *Pharmacology, Biochemistry and Behavior* **71**, 745–756.
- Fried, I., Wilson, C. L., MacDonald, K. A. & Behnke, E. J. (1998). Electric current stimulates laughter. *Nature* **391**, 650.
- Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., Heninger, G. R. & Charney, D. S. (1989a). The Yale–Brown Obsessive–Compulsive Scale. I. Development, use and reliability. *Archives of General Psychiatry* **46**, 1006–1011.
- Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Delgado, P., Heninger, G. R. & Charney, D. S. (1989b). The Yale–Brown Obsessive–Compulsive Scale. II. Validity. *Archives of General Psychiatry* **46**, 1012–1016.
- Guy, W. (1976). *ECDEU Assessment Manual for Psychopharmacology*. Department of Health, Education and Welfare: Washington, DC.
- Hamilton, M. & Guy, W. (1976). *Hamilton Depression Scale*. Beltz: Berlin.
- Hauke, W., Ruppert, S., Unger, H., Manzinger, H., Schlehle, B. & Zaudig, M. (1998). Multimodale stationäre Verhaltenstherapie bei Zwangsstörungen – Teil 2: Die therapeutische Praxis. *Psychotherapie in Psychiatrie, Psychotherapeutischer Medizin und Klinischer Psychologie* **3**, 224–233.
- Hegerl, U. & Mavrogiorgou, P. (1999). Die Zwangsstörung aus neurobiologischer Sicht. *Verhaltenstherapie und Verhaltensmedizin* **20**, 435–447.
- Heimann, H. & Spoerri, T. (1957). Das Ausdruckssyndrom der mimischen Desintegration bei chronischen Schizophrenen. *Schweizer Medizinische Wochenschrift* **35/36**, 1126–1132.
- Heimer, L., Switzer, R. D. & Van Hoesen, G. W. (1982). Ventral striatum and ventral pallidum; components of the motor system? *Trends in Neurosciences* **5**, 83–87.
- Hermisdörfer, J., Wack, S., Mai, N. & Marquardt, C. (1996). *Dreidimensionale Bewegungsmessung zur Analyse der Handfunktion*. EKN – Entwicklungsgruppe Klinische Neuropsychologie: München.
- Hollander, E., Schiffman, E., Cohen, B., Rivera-Stein, M. A., Rosen, W., Gorman, J. M., Fyer, A. J., Papp, L. & Liebowitz, M. R. (1990). Signs of central nervous system dysfunction in obsessive–compulsive disorder. *Archives of General Psychiatry* **47**, 27–32.
- Hymas, N. F. S. (1998). The neurology of obsessive–compulsive disorder. *CNS Spectrums* **3**, 22–28.
- Jacobs, B. L. & Fornal, C. A. (1997). Serotonin and motor activity. *Current Opinion in Neurobiology* **7**, 820–825.
- Juckel, G. (2003). Beeinträchtigungen von Mimik und Emotionsausdruck bei affektiven Störungen. In *Bewegungsstörungen bei psychischen Erkrankungen* (ed. T. Jahn and H. Förstl). Springer: Heidelberg. (In the press.)
- Juckel, G. & Polzer, U. (1998). Fine analysis of abnormal facial expressions in chronic schizophrenic patients – a pilot study. *German Journal of Psychiatry* **1**, 6–9.
- Juckel, G., Präßl, A., Froschmayer, S., Mavrogiorgou, P., Tigges, P., Möller, H.-J. & Hegerl, U. (1999). ‘Im Gesicht lesen lernen’ – Mimikanalyse schizophrener Patienten. In *Schizophrenie – eine affektive Erkrankung?* (ed. W. Machleidt, H. Haltenhof and P. Garlipp), pp. 127–134. Schattauer: Stuttgart.
- Lucey, J. V., Butcher, G., Clare, A. W. & Dinan, T. G. (1993). Elevated growth hormone responses to pyridostigmine in obsessive–compulsive disorder: evidence of cholinergic supersensitivity. *American Journal of Psychiatry* **150**, 961–962.
- Luxenberg, J. S., Swedo, S. E., Flament, M. F., Friedland, R. P., Rapoport, J. L. & Rapoport, S. I. (1988). Neuroanatomical abnormalities in obsessive–compulsive disorder. *American Journal of Psychiatry* **145**, 1089–1093.
- McDougle, C. J., Goodman, W. K., Price, L. H., Delgado, P. L., Krystal, J. H., Charney, D. S. & Heninger, G. R. (1990). Neuroleptic addition in fluvoxamine-refractory obsessive–compulsive disorder. *American Journal of Psychiatry* **147**, 650–654.
- Mavrogiorgou, P., Mergl, R., Tigges, P., El Hussein, J., Schröter, A., Juckel, G., Zaudig, M. & Hegerl, U. (2001). Kinematical analysis of handwriting movements in patients with obsessive–compulsive disorder. *Journal of Neurology, Neurosurgery and Psychiatry* **70**, 605–612.

- Mogenson, G. J., Johns, D. L. & Yim, C. C. (1980). From motivation to action: functional interface between the limbic system and the motor system. *Progress in Neurobiology* **14**, 69–97.
- Padberg, F., Juckel, G., Präbl, A., Zwanzger, P., Mavrogiorgou, P., Hegerl, U., Hampel, H. & Möller, H.-J. (2001). Prefrontal cortex modulation of mood and emotionally induced facial expressions: a transcranial magnetic stimulation study. *Journal of Neuro-psychiatry and Clinical Neurosciences* **13**, 206–212.
- Pauls, D. L., Towbin, K. E., Leckman, J. F., Zahner, G. E. & Cohen, D. J. (1986). Gilles de la Tourette's syndrome and obsessive-compulsive disorder. Evidence supporting a genetic relationship. *Archives of General Psychiatry* **43**, 1180–1182.
- Provine, R. R. (2000). *Laughter – A Scientific Investigation*. Viking: New York.
- Rasmussen, S. A. & Eisen, J. L. (1990). Epidemiology of obsessive-compulsive disorder. *Journal of Clinical Psychiatry* **51** (suppl.), 10–13.
- Rinn, W. E. (1984). The neuropsychology of facial expression – a review of neurological and psychological mechanisms for producing facial expressions. *Psychological Bulletin* **95**, 52–77.
- Robinson, D., Wu, H., Munne, R. A., Ashtari, M., Alvir, J. M., Lerner, G., Koreen, A., Cole, K. & Bogerts, B. (1995). Reduced caudate nucleus volume in obsessive-compulsive disorder. *Archives of General Psychiatry* **52**, 393–398.
- Sabbe, B. & Hulstijn, W. (2001). Psychomotor disturbances in schizophrenia and depressive disorders. *Current Opinion in Psychiatry* **14**, 597–603.
- Saxena, S., Brody, A. L., Schwartz, J. M. & Baxter, L. R. (1998). Neuroimaging and frontal-subcortical circuitry in obsessive-compulsive disorder. *British Journal of Psychiatry* **35** (suppl.), 26–37.
- Schilder, P. (1938). The organic background of obsessions and compulsions. *American Journal of Psychiatry* **94**, 1397–1416.
- Schulman-Green, D. J. (1999). Deceiving appearances. Communicating with facially inexpressive older adults. *Journal of Gerontology and Nursing* **25**, 40–43.
- Sem-Jacobsen, C. W. (1968). Changes in mood. In *Depth-electrographic Stimulation of the Human Brain and Behavior* (ed. W. H. Gantt), pp. 127–138. Charles C. Thomas: Springfield.
- Swedo, S. E., Rapoport, J. L., Cheslow, D. L., Leonard, H. L., Ayoub, E. M., Hosier, D. M. & Wald, E. R. (1989). High prevalence of obsessive-compulsive symptoms in patients with Sydenham's chorea. *American Journal of Psychiatry* **146**, 246–249.
- World Medical Association (1997). World Medical Association declaration of Helsinki. Recommendations guiding physicians in biomedical research involving human subjects. *Journal of the American Medical Association* **277**, 925–926.