INTRODUCTION

The Korsakoff’s syndrome is defined by several neurocognitive impairments. The best known characteristic is a profound memory impairment (e.g., Kessels et al., 2000), but other cognitive impairments have been reported as well, for example, deficits in attention (Oscar-Berman, 1989) and executive functioning (Pollux et al., 1995). Performance on well-structured, untimed tests of familiar, usually overlearned material often remains intact, while scores decline on tests relying on speed, visuoperceptual and visuospatial organization (Lezak, 1995). Intellectual ability, however, is commonly preserved (Mayes and Downes, 1997).

Korsakoff’s syndrome is caused by chronic alcohol abuse combined with an insufficient diet. As a consequence, patients have suffered from a chronic thiamine (vitamin B1) shortage which results in cortical atrophy, especially affecting the frontal and temporal cortex (Joyce, 1994; Harper, 1998; Kril et al., 1997; Oscar-Berman et al., 1990) and damage to the limbic structures, such as thalamic nuclei, mammillary bodies and the hippocampus (Joyce and Robbins, 1991; Langlais and Savage, 1995; Mayes et al., 1988; Sziklas and Petrides, 1998). Several studies report evidence for affective changes in Korsakoff’s syndrome. Generally, these patients show little spontaneous affective behaviour and demonstrate emotional blandness and passivity, which is often mistaken for a clinical depression (Oscar-Berman et al., 1990). In turn, Johnson et al. (1985) found in their study that Korsakoff patients were still able to develop affective reactions. In line with this latter finding, Douglas and Wilkinson (1993) concluded that Korsakoff’s syndrome does not necessarily involve flattening of affect or other emotional abnormalities. Snitz et al. (2002) looked in detail at the processing of affective prosody in Korsakoff’s syndrome. They found that both linguistic (question or statement intonation) and affective (angry, sad, happy, fearful and neutral intonation) prosody discrimination were intact when semantic content and affective prosody were congruent. However, affective prosody identification was impaired when the semantic content was either neutral or incongruent with prosody. These findings suggest that Korsakoff patients may have problems interpreting affective prosody in the absence of semantic cues.

In the present study, we focus on a specific aspect of affective behaviour, that is, the recognition of facial expressions. Interpersonal behaviour depends to a large extent on the correct understanding of emotional facial expressions of others. It has been shown that the recognition of emotional facial expressions may be severely impaired in chronic alcoholics, and it has been argued that the social problems these patients experience are partly due to the deficit in the recognition of facial expressions. Kornreich et al. (2001), for example, demonstrated that detoxified alcoholics showed deficits in the recognition of emotional facial expressions and showed more
interpersonal dysfunctioning compared to a healthy control group. The social behaviour problems were highly correlated with the deficits in the recognition of emotional facial expressions. Oscar-Berman et al. (1990) observed that chronic alcoholic subjects attribute more emotional intensity to facial expressions compared to controls. This was later confirmed by Philippot et al. (1999), who also found that alcoholics showed a bias in their decoding of emotional facial expressions, especially for the emotions “anger” and “contempt”. In a recent study by Frigerio et al. (2002), a newly developed test was used to assess the labelling of facial emotional expressions (i.e., anger, sadness, happiness and disgust). It was found that alcoholics made more errors than control subjects in recognising expressions overall and specifically mislabelled sad expressions as being hostile (i.e., angry or disgusted). Furthermore, alcoholics needed a more emotionally expressive face to correctly recognise the facial expressions.

There are relatively few studies that have looked at the recognition of facial expressions in Korsakoff patients compared to the number of studies with alcoholic patients. This is surprising, as the former group has been shown to suffer from more severe brain damage compared to the later group, and demonstrably suffers from more profound cognitive deficits in general. In their study, Oscar-Berman et al. (1990) also investigated Korsakoff patients and observed that they, like the alcoholics, attributed more emotional intensity to facial expressions compared to controls.

The slight evidence for the existence of deficits in the recognition of facial emotional expressions in Korsakoff patients might be understood as a consequence of the neuropathology underlying Korsakoff’s syndrome, such as the atrophy in the limbic system (Harper, 1998; Jernigan et al., 1991; Paller et al., 1997). In the present study, the perception and recognition of emotional expressions is investigated in Korsakoff patients using a newly developed experimental paradigm in which we gauge the ‘amount of expression information’ on the face that is needed for correct identification. We employ video clips of varying length in which a neutral facial expression gradually changes into an emotional expression. In the first instance, subjects only see a change from neutral to 20% full-blown expression, followed by video clips that incrementally increase the expression by 10% steps. The level from which point onwards the subjects correctly identify an expression is assumed to represent the minimal amount of information that is required to identify that expression. Our hypotheses are that Korsakoff patients show impaired recognition of facial expressions, and that certain types of emotional expressions are more severely affected than others. The pattern of the expected results is hypothesised to mirror that which has been observed in chronic alcoholics, perhaps to an even more severe extent.

**METHODS**

**Research Participants**

Twenty-three patients (16 males, 7 females) diagnosed with Korsakoff’s syndrome according to DSM-IV (American Psychiatric Association, 1994), were recruited from the Korsakoff Clinic of the Vincent van Gogh Institute, Venray, The Netherlands. Intelligence test results were available for 15 patients. Mean intelligence as measured with the Wechsler Adult Intelligence Scale (WAIS) or the shortened Groningen Intelligence Test (GIT) was in the normal range for the neuropsychologically tested patients (mean = 91.67, SEM = 3.6). The educational levels of all participants were recorded using 7 categories, 1 being the lowest education, 7 the highest. Twenty-three healthy participants were matched on age and educational level (16 males, 7 females) and included as a control group. No significant group differences in age or educational level were found (see Table I).

**Materials and Tests**

**Emotion Recognition Task**

Stimuli for this test were based on colour pictures from actors mimicking emotional expressions and a neutral face. There were four actors (two male, two female) who each posed six emotions (anger, disgust, fear, happiness, sadness and surprise). A computer-generated programme, developed from algorithms designed by Benson and Perrett (1991), was used to develop the stimuli. This programme was devised to create intermediate morphed images (see Figure 1) between a neutral face (0% emotion) and a full-blown expression (100% emotion). The original colour pictures of the

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<td>Mean age, education level and Benton Face Recognition Test Score (+ SD) for the Korsakoff patients (N = 23) and the control group (N = 23)</td>
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<td>Korsakoff patients</td>
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<td>Mean</td>
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Faces were manually delineated by 179 feature-points that define the shape of the important facial features (Rowland and Perrett, 1995). For each expression, two photographs were available, the neutral expression and the full-blown expression. The intermediate expressions were simulated. This was done by generating 19 morphed images by calculating the difference in spatial position between two corresponding feature points in the neutral and full-blown expressions. In the morphed images, both the dimension of shape and texture underwent gradual transitions. These images were used to construct video clips that incrementally increase the degree of expression by 10% steps. Consequently, each complete video clip entailed 21 images and 20 image steps and was used to make the individual video clips for the different trials. The first image is the neutral face, which has 0% emotion on the face, the second image has an increment of 5% emotion on the face, and each next image has an additional 5% more emotion on the face, ending with the last image, which is a 100% emotional face. Thus, the video clip from 0-20% contains the first 5 images, and for each subsequent video clip increments of 10% were used, which consists of two images (e.g., the sequence from 0-30% are the first 7 images, from 0-40% are the first 9 images and so on). For each actor, nine video clips (0-20, 0-30, 0-40, 0-50, 0-60, 0-70, 0-80, 0-90, 0-100%) were constructed for each of the six emotions by increasing the number of morphed images presented in succession. In total there were 216 trials, i.e. the number of emotions used (6) \times the number of actors (4) \times the number of sequences (9). The presentation procedure was as follows. First, the subjects saw, in a random order, the first 24 trials (6 emotions \times 4 actors), which were video clips from 0 to 20% (there was no set of 24 trials with video clips from 0-10%). The second set of 24 trials were video clips from 0 to 30% and then for each set of 24 trials the video clip goes up with increments of 10%, until they reached the final sequence of clips in which the neutral face changed into a full-blown expression. In each trial, the subject was required to make a forced choice between one of six emotional expression labels that were displayed on the screen after the presentation of the video clip. There was no time restriction.

Benton Facial Recognition Test (Short Form)

The Benton and Van Allen Test of Facial Recognition (Benton et al., 1994) comprises a series of sheets containing a single photographed target face to be matched to a set of six face photographs. In the first six trials, an identical face has to be selected from among five decoys. In the remaining 7 trials, three different views (changed in orientation or lighting conditions compared to the target photograph) have to be distinguished from three incorrect alternatives. The faces are physically similar, without spectacles or facial hair. The maximum number of correctly identified faces is 27.

Procedure

Patients were asked to participate by the resident psychologist (AW). They subsequently received a letter providing information and, if they agreed to participate in the study, signed an informed consent. The tests were presented to each subject in a fixed order. The first task was the Emotional Recognition Task, followed by the Benton Facial Recognition Task (short version). The whole procedure took about 30 minutes. The procedure for the control subjects was identical.
RESULTS

Emotion Recognition Task

Sensitivity was conceptualised as the minimal amount of expression information required for systematic correct identification. As a measure we used the percentage of emotion from which point onwards the expression was consistently recognised in subsequent trials (i.e., a sensitivity of 40% for a given emotion indicates that the participant correctly identified the emotion at all percentages between 40% and 100%). A 6 × 2 MANOVA was carried out with Expression (six levels: anger, fear, disgust, happiness, sadness, surprise) as within-subject factor and Group (two levels: patient, control) as between-subject variable. This MANOVA revealed a main effect of Expression \[ F(5, 13) = 14.52, p < .005 \] and an interaction effect of Expression × Group \[ F(5, 85) = 2.43, p < .05 \]. Bonferroni-corrected post-hoc tests (alpha set at .008) showed a significant difference in sensitivity between the two groups for the emotions anger \[ t(40) = 3.25, p < .002 \], fear \[ t(24) = 2.51, p < .02 \] and surprise \[ t(38) = 3.23, p < .003 \], with the Korsakoff patients being less sensitive compared to the control subjects (see Figure 2).

To compare the performance on the six emotions directly, standardised effect sizes (Cohen’s \( d \)) were calculated (Cohen, 1988). Generally, an effect size less then .2 is considered to be “smal”, between .2 and .8 is considered to be “moderate” and above .8 is regarded as “large”. The effect sizes for happy \( d = .32 \) and disgust \( d = .33 \) were moderate, effect sizes for the emotions anger \( d = 1.04 \), fear \( d = 1.16 \), sadness \( d = .80 \) and surprise \( d = 1.04 \) can be regarded as large, with the largest effect size for fear.

Benton Face Recognition Task

Scores were analysed using an independent sample T-test, which revealed no significant difference between the patient group and the control group \[ t(44) = 1.67 \]. Mean score for the patients was 20.52 (SEM = .45), mean score for the controls 21.57 (SEM = .43). These performances are within the normal range according to the normative data of the test.

DISCUSSION

The present study examined whether patients suffering from Korsakoff’s syndrome are impaired in the processing of facial emotional expressions. The results demonstrate that Korsakoff patients are significantly less sensitive to the facial expressions of anger, fear and surprise compared to controls, but not to the facial expressions of happiness, disgust or sadness. This deficit in facial expression recognition is not the result of an overall face recognition deficit, since both groups performed equally well on the Benton Face Recognition Task. The effect-size analysis showed that the deficits in recognizing fear, anger and surprise could be regarded as large effects, with fear showing the largest effect size. With respect to the non-
significant findings, it should be mentioned that happiness is a relatively easy emotion to recognize and often shows a ceiling effect. In addition, sadness shows a large, but non-significant effect size, possibly due to limited power. Thus, it might be that Korsakoff patients suffer from a more generalized emotion perception deficit. Overall, the current findings are interpreted as evidence for a general expression recognition deficit with a suggestion that certain emotions are more affected than others. Future studies in larger patient groups should be performed in order to replicate this finding, especially focusing on differences between types of emotional expressions.

Our findings are in line with previous studies in chronic alcoholics (e.g., Philippot et al., 1999) in which a deficit in the decoding of the emotions was also observed. Frigerio et al. (2002) found that alcoholics have a tendency to give hostile attributions to faces looking towards them. These impairments can be related to the changes in behaviour, such as apathy and emotional flattening (Egger et al., 2002). This is the first study looking at the recognition of emotional expressions in patients suffering from Korsakoff’s syndrome to include the expression of fear. With respect to chronic alcoholics, Philippot et al. (1999) showed a negative bias for emotions of disgust and contempt but not for fear, while other studies did not include the emotion fear (Oscar-Berman et al., 1990). This finding of impaired recognition of fear in Korsakoff patients is in line with a hypothesised dysfunction of the amygdala (Harper, 1998; Kril et al., 1997; Oscar-Berman et al., 1990), since several studies (Adolphs et al., 1995; Phillips et al., 1999) showed a clear relation between amygda damage and impaired recognition of the emotion fear. The emotion surprise has been even less studied in these populations. However, the currently found impairment in recognizing this emotion might be associated with dysfunction of the frontal and temporal lobes (Best et al., 2002; Coupland et al., 2003).

Finally, several studies have shown effects of depressed mood on emotion recognition (Cooley and Nowicki, 1989; George et al., 1998; Halse et al., 1998). It could be suggested that depression plays an important role in the development of facial emotional recognition deficits in Korsakoff patients as well. However, it is not possible to obtain reliable and valid depression scores using psychological questionnaires in these patients because Korsakoff patients typically show a severe lack of insight and profound amnesia (Butters, 1985; Egger et al., 2002). Also, Davidson (1995) found that depression and alcohol dependence coexist in the majority (67%) of chronic alcoholics, but not in detoxified alcoholics, which is the case in our Korsakoff patients. In addition, the pattern of results found within the patient group is different from the pattern usually found in depressed patients. Several studies have showed a negative bias in the latter group (e.g., Gur et al., 1992; Mandal and Bhattacharya, 1985). Depressed patients tended to see happy faces as neutral and neutral faces as sad. Thus, the present results are most likely not due to a depressed mood.

In sum, the results of this study show that Korsakoff patients suffer from problems in the recognition of multiple facial emotional expressions. The effects were largest with the emotions fear, anger, and surprise. It is suggested that these deficits might be in part related to subcortical (amygdala) and cortical (frontal lobe) dysfunction, but further research is needed to scrutinise this hypothesis.

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