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SCIENTIFIC INVESTIGATIONS

Performance of facial expression classification tasks in patients with obstructive sleep apnea

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Study Objectives: People show a facial recognition speed advantage, termed positive classification advantage (PCA), when judging whether a facial expression is happy compared to angry or sad. This study investigated emotional face recognition by patients with obstructive sleep apnea (OSA) with impaired neurocognition.
Methods: Thirty-four patients with OSA and 26 healthy control patients who underwent 1 night of polysomnographic evaluation before recruitment were asked to complete an emotion recognition task. Accuracy rates and reaction times were recorded and analyzed using repeated-measures analysis of variance.
Results: When participants were asked to classify positive (happy) versus negative (sad) emotional expressions, the phenomenon of PCA disappeared. Importantly, however, compared with the control patients who showed PCA, patients with OSA identified sad faces faster but were similar in processing happy faces.
Conclusions: In accordance with previous studies that showed depressive emotion in patients with OSA, our results indicate that patients with OSA show negative bias in facial expression recognition, which might lead to decline in ability of social communication.
Keywords: facial expression classification, obstructive sleep apnea, positive classification advantage
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BRIEF SUMMARY
Current Knowledge/Study Rationale: It is important for people to recognize facial emotional reactivity in social interaction. Patients with obstructive sleep apnea show impaired facial expression recognition, but we have not determined whether patients with obstructive sleep apnea have the advantage of positive facial expression as in otherwise healthy individuals.
Study Impact: Our findings suggest that obstructive sleep apnea impairs recognition of emotional states and leads to rapid perception of negative emotional expressions. The D-value of response time between 2-face validity in both groups was closely related to apnea-hypopnea index and Epworth Sleepiness Scale and Montreal Cognitive Assessment scores.

INTRODUCTION

Obstructive sleep apnea (OSA) is associated with episodic sleep disruptions in airway patency, followed by reduced oxygen saturation and increased arousals throughout the night. OSA is characterized by loud snoring, excessive daytime sleepiness, and even psychological symptoms such as depression.[1] Obvious sleep quality problems confirmed by polysomnography (PSG) may cause cognitive changes that also comprise the most common daytime symptoms. In recent years, it has become clear that patients with OSA show significant cognitive dysfunction associated with deficits in attention,[2,3] executive function,[4,5] and episodic memory,[6,7] and the severity of cognitive impairment correlates closely with that of OSA.

Information processing is an important aspect of cognitive functioning and its speed is reduced by cognitive impairment[8]; thus, it is essential to observe information processing speed when assessing cognitive function. Most information gained from the surrounding environment is obtained visually. Faces are unarguably the most important stimuli in social interactions and facial expression is one of the most important types of signal. It is important for people to recognize facial emotional reactivity and modulate their emotional state. Recognition of happy faces facilitates cooperation with and influences other people in social interaction.[9]

Deficits in facial emotional information processing are a common symptom in sleep disorders. The recognition of facial expression was impaired after 1 night of sleep deprivation and resulted in a more negative response to neutral pictures[10] and an increased reaction to negative emotional pictures,[11] which was normalized after 1 night of recovery sleep.[12,13] Moreover, other findings suggest that alterations of socioemotional processing are present in patients with chronic sleep disorder.[14,15] In the aforementioned studies, participants were required to categorize the intensity using three to six emotional categories, and the results indicated that sleep is involved in regulating emotional

evaluation. Whether experimental acute deprivation[10–13] or chronic disorder,[14,15] sleep problems raise the possibility of alterations in emotional processing that has implications for many social interactions and work contexts. However, definitive evidence about a specific effect of OSA on self-reported processing of facial expressions remains sparse. Patients with sleep apnea performed worse in recognizing facial expressions, but only showed reduced accuracy for the happiness and sadness scales using the Facial Expressed Emotion Labeling (FEEL) test, and not for other emotions.[15] Unfortunately, this test failed to find the difference between happiness and sadness.

Studies on the mechanism of cognitive impairment in patients with OSA have shown that in addition to sleep rhythm disorders, chronic intermittent hypoxia is another major cause of cognitive impairment. In animal models,[16] it has been proven that chronic intermittent hypoxia causes cognitive impairment, and the mechanism is still under further study. Exposure to chronic hypoxia, such as plateau hypoxia,[17] would result in human behavioral and mood disturbances, and cognitive impairment. If there is a possibility of hypoxemia, patients with chronic obstructive pulmonary disease may have cognitive impairment, such as information processing.[18] Similarly, because of chronic hypoxia, patients with OSA have obvious alterations in brain electrical activity that are associated with emotional regulation.[19] Extrapolating from the aforementioned literature, the performance of patients with OSA in the categorization of positive and negative expression remains unclear.

Other correlative studies on facial expression processing have found some interesting phenomena in healthy volunteers that are particularly relevant to information processing speed. Early in the century, consistent evidence showed that positive facial expressions are recognized faster and more accurately compared with all of the other five basic emotional faces, including sadness, anger, and disgust.[20–23] In those studies on facial expression recognition, participants were asked to categorize pictures of facial expressions according to their emotional valence or state, and they showed a recognition speed advantage when judging whether facial expression was happy. Such happy face recognition advantage is called positive classification advantage (PCA).[24–26] PCA can always be observed regardless of task conditions and stimulus type. In 2004, Leppänen and Hietanen replicated earlier findings and showed PCA between happiness and sadness using the schematic face.[23] The empirical method can detect people's emotional preferences accurately, and the tester can determine the individual's emotional status or cognitive biases through the test results. Such a method has been widely used in research on cognitive aspects of clinical disorders (eg schizophrenia,[27,28] Parkinson disease,[29] and autism[30]). It has even been proposed that facial recognition can be used as a diagnostic indicator for the latent period or early stage of Alzheimer disease.[31] Facial expression recognition is important in clinical settings, and additional research will be needed to clarify whether there are processing differences between happiness and sadness for patients with OSA. Therefore, happy faces are chosen as a positive facial expression and sad faces as a negative facial expression in categorization tasks.

There is clinical evidence that patients with OSA show impaired facial expression recognition when compared with normal individuals. In the current study, we focused on facial expression processing speed and expression classification performance in patients with OSA. We aimed to validate: (1) whether patients had the advantage of positive facial expression, as in otherwise healthy individuals; and (2) whether patients had a longer or shorter reaction time in recognizing happy and/or sad faces compared with healthy control patients.

Participants

We recruited 34 patients with OSA (age 20–60 years) and 26 age- and education-matched healthy control patients. Those with OSA who did not undergo any treatment intervention were either inpatients or outpatients at the Center of Sleep Medicine, Shandong Provincial Hospital in Jinan, China. All patients and control patients underwent 1 night of PSG evaluation, and were required to go to bed before 10:00 PM and wake up at 6:00 AM the next day. The diagnosis and comorbidities were assessed by a trained clinician. The next day after PSG monitoring, all the volunteers who self-reported sleeping more than 6 hours a night accomplished the emotional expression recognition task at 10:00 AM and completed all the questionnaires, including Epworth Sleepiness Scale (ESS), Montreal Cognitive Assessment (MoCA), and Zung Self-Rating Depression Scale (SDS). It was confirmed by PSG that the total sleep time of all volunteers was indeed more than 6 hours.

Inclusion criteria were (1) age 20–60 years; (2) no psychiatric and/or medical disorders; and (3) for patients with OSA, apnea-hypopnea index (AHI) $\geq$ 5 events/h and for control patients, AHI < 5 events/h. All reported that they had normal or corrected-to-normal vision. Exclusion criteria were: (1) another diagnosed sleep or circadian disorder; (2) periodic limb movement index > 15 events/h based on PSG; (3) history of a medical, neurologic, or psychiatric disorder (eg, severe episode of major depression, generalized anxiety disorder, specific phobia, or panic disorder, other than OSA) that could influence excessive daytime sleepiness; (4) alcohol/substance abuse (within 12 months of study entry); (5) medication known to have an effect on brain function, sleep, and daytime vigilance (eg sedatives or hypnotic agents); (6) history of obsessive–compulsive disorder, post-traumatic stress disorder, bipolar disorder, psychotic disorder, developmental disorder, or any other physical medical condition; or (7) left-handedness. Moreover, patients in the control group were excluded if they had any current or previous history of OSA.

Participants provided written informed consent prior to participation in the study, and the project was approved by the Ethics Committee of Shandong Provincial Hospital, which acts to meet the demands of the Declaration of Helsinki.

METHODS

The study applied an emotional expression recognition task, in which different face models were used in order to avoid excessive repetition of an individual face model. Many repetitions of a single face model might have made the test monotonous and resulted in unwanted biases. Each expression category

Figure 1—Example stimuli of facial expressions.

consisted of 18 different face models by manipulating the distance among facial features and by manipulating the shape of the facial features[32] (**Figure 1**). Each of the 18 stimuli in one expression category was presented twice, making up 108 trials (18 faces × 3 facial expressions × 2) in one block in total. The task was programmed using E-Prime2.0 Psychology Software (Psychology Software Tools, Inc., Pittsburgh, Pennsylvania, USA) and the pictures were shown on a 20-inch computer screen. All participants were instructed to view at a distance from the screen from which they could read the task instructions and identify the stimuli clearly.

The entire test included the following sequence of events. First, before the practice trials, it was essential to make sure that all participants had read the test instructions and understood the procedure. After 12 practice trials, there was a hint of the official start of the experimental trials. The schematic faces were presented in random order and one face was shown for 300 ms. The face was followed by a gray rectangle in line with the size. As the neutral expression disappeared, the gray rectangle was presented for 600–800 ms, but after emotional expression, the gray rectangle was maintained on the screen until the participant made a response by pressing one of the labeled keys on a standard computer keyboard. The participants were required to identify which of the three emotions (happy, neutral, or sad) was presented on the screen and to indicate their choice by pressing a correspondingly marked response key as quickly and accurately as possible. Both hands were used to respond so that the index finger of the left hand was used to press the left-hand key "Z", and the index finger of the right hand pressed the right-hand response key "/". The markings of the response keys (happy–sad/sad–happy) were balanced between the participants. There were 2 blocks with a short break in between. The experimenter stayed in the room while the participant finished the task.

Statistical analysis

Statistical analysis was performed using SPSS 20.0 (Chicago, Illinois, USA). We used the chi-square test to compare numerical data. Differences in continuous demographic and clinical data, PSG data, and scale scores were analyzed by independent-samples *t* tests. For comparison of expression recognition data, we also used an independent-samples *t* test to analyze the effects between groups, but in one group, comparison of the time variables between different expressions was analyzed by paired-samples *t* tests. Repeated-measures analysis of variance was conducted for both indicators (group and expression), with expression (happy and sad) as the within-subjects factor. The relationship between clinical data and D value of reaction times (RTs) were assessed by Pearson correlation. A value of $P < .05$ was taken as significant.

RESULTS

The demographic and clinical characteristics of the 2 groups are summarized in **Table 1**. The participants were divided into 2 groups: healthy control patients and patients with OSA based on PSG results. There were significant differences in AHI (0.89 ± 0.87 versus 40.37 ± 31.86 events/h, $t = 7.222$, $P < .001$) and nadir of arterial oxygen saturation (SaO_2) (95.08 ± 2.02 versus 75.38 ± 16.54 %, $t = 6.876$, $P < .001$) between the 2 groups. All participants were matched for demographic data, such as sex ($P = .071$), age (37.85 ± 7.09 versus 40.15 ± 10.93 years, $t = 0.986$, $P = .328$), and years of education (15.54 ± 3.83 versus 13.79 ± 3.50, $t = 1.838$, $P = .071$), but not body mass index (BMI) (23.82 ± 2.84 versus 28.22 ± 5.88 kg/m^2, $t = 3.510$, $P = .001$). The groups were not matched for scale scores, such as ESS (2.92 ± 1.94 versus 11.09 ± 5.38, $t = 8.177$, $P < .001$) and MoCA (28.12 ± 0.95 versus 25.35 ± 2.21, $t = 6.529$, $P < .001$), but there was no significant difference in SDS scores (35.85 ± 3.43 versus 37.56 ± 3.89, $t = 1.777$, $P = .081$).

For each participant, including all patients and healthy control patients, the accuracy rate (ACC) was the percentage of correct responses, RT was the average time for correct reaction, and the difference value of RT (DiffRT) was the D-value of sad expression RT minus happy RT. Before analyzing the recognition speed data, we analyzed the mean percentage of accurate responses to 2 expressions as a function of response category (**Figure 2**). Patients with OSA had worse ACC compared with that of healthy control patients. There was no main group effect in ACC ($F = 0.595$, $P = .444$); whether for happy face ($t = 0.345$, $P = .732$) or sad face ($t = 0.930$, $P = .356$), the performance of each group was similar. There was no main expression effect ($F = 0.051$, $P = .822$), nor group-by-expression interaction ($F = 0.333$, $P = .566$) detected in ACC. No significant difference was found between the groups when recognizing different expressions.

For RT analysis, incorrect responses were excluded, and responses with RTs more than ± 2 standard deviations from the mean in each condition were removed. As the statistical model used for percentages of correct responses, the RTs were analyzed in the same way (**Table 2** and **Figure 3**). The main effect of the group was not significant ($F = 0.116$, $P = .734$), showing that RTs were similar between 2 groups (mean 654.62 ms for the patient group and mean 665.45 ms for the normal group). A significant main effect was obtained for expression ($F = 8.753$, $P = .004$), indicating that RT was significantly faster for happiness recognition (mean 651.16 ms) than that for sadness recognition (mean 667.46 ms). There was a significant interaction of group by expression ($F = 10.969$, $P = .002$) detected in the RTs. In the normal control group, the RT to happy face stimuli (mean 645.14 ms) was faster than for sad faces (mean 685.76 ms, $t = 5.705$, $P < .001$), reflecting obvious PCA. However, the difference between the 2 expressions was not significant in the OSA group (mean

Table 1—Demographic and clinical data of participants.

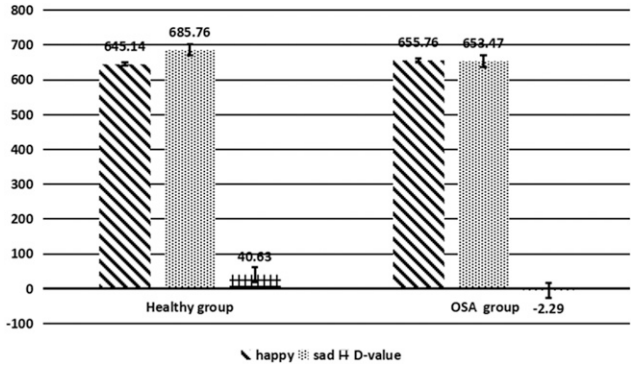
Characteristic	Control (n = 26)	OSA (n = 34)	*t*	*P*
Female, n	10	6		.071
Age, years	37.85 ± 7.09	40.15 ± 10.93	0.986	.328
Education, years	15.54 ± 3.83	13.79 ± 3.50	1.838	.071
BMI, kg/m^2	23.82 ± 2.84	28.22 ± 5.88	3.510	.001
AHI, events/h	0.89 ± 0.87	40.37 ± 31.86	7.222	< .001
SaO_2 nadir, %	95.08 ± 2.02	75.38 ± 16.54	6.876	< .001
ESS, score	2.92 ± 1.94	11.09 ± 5.38	8.177	< .001
MoCA, score	28.12 ± 0.95	25.35 ± 2.21	6.529	< .001
SDS, score	35.85 ± 3.43	37.56 ± 3.89	1.777	.081

With the exception of the proportion of women in each group, all values are mean ± standard deviation. AHI = apnea-hypopnea index, BMI = body mass index, ESS = Epworth Sleepiness Scale, MoCA = Montreal Cognitive Assessment, OSA = obstructive sleep apnea, SaO_2 = arterial oxygen saturation, SDS = Zung Self-Rating Depression Scale.

Figure 2—Accuracy rates of responses to different expressions between groups (mean, %).

OSA = obstructive sleep apnea.

Figure 3—Reaction times of responses to different expressions between groups (mean, ms).



OSA = obstructive sleep apnea.

Table 2—Reaction times of responses to different expressions between groups.

	Control	OSA
Happy	645.14 ± 102.24	655.76 ± 148.01
Sad	685.76 ± 121.34	653.47 ± 115.73
D-value	40.63 ± 36.32	−2.29 ± 57.88

Results are mean ± standard deviation. OSA = obstructive sleep apnea.

655.76 ms versus 653.47 ms, $t = 0.231$, $P = .819$). For different validity of facial expressions, the healthy control group (mean 685.76 ms) performed slower compared with patients with OSA (mean 653.47 ms, $t = 1.049$, $P = .299$) for sad face categorization, and no significant difference was found for happy face recognition (mean 645.14 ms versus 655.76 ms, $t = 0.313$, $P = .755$). However, there was a significant difference in the DiffRT (40.63 ms versus −2.29 ms, $t = 3.312$, $P = .002$) between the 2 groups.

Correlations in the whole cohort were identified, although weak, between ACC or RTs and age, years in education, BMI, AHI, nadir SaO_2, ESS score, MoCA score, and SDS score (**Table 3**). In ACC, only the sad face categorization was negatively correlated with age ($r = -.263$, $P = .042$) and AHI ($r = -.260$, $P = .045$), and positively with years in education ($r = -.370$, $P = .004$). For RTs, happy and sad expressions were both positively related to age ($r = .343$, $P = .007$, and $r = .327$, $P = .011$, respectively), and inversely to years in education ($r = -.311$, $P = .015$, and $r = -.257$, $P = .047$, respectively). Stronger positive correlations were detected between DiffRT and MoCA scores ($r = .662$, $P < .001$) (**Figure 4**). Additionally, DiffRT was found to be negatively correlated with AHI ($r = -.328$, $P = .011$) (**Figure 5**) and ESS score ($r = -.296$, $P = .022$) (**Figure 6**). ESS and MoCA scores had opposite correlations with BMI ($r = .528$, $P < .001$, and $r = -.404$, $P = .001$, respectively), AHI ($r = .578$, $P < .001$, and $r = -.682$, $P < .001$, respectively) and SaO_2 ($r = -.675$, $P < .001$, and $r = .581$, $P < .001$, respectively).

DISCUSSION

To evaluate performance of facial expression classification by patients with OSA whose AHI and SaO_2 were significantly

Table 3—Correlations between results of response and demographic and clinical characteristics.

			Age	Edu	BMI	AHI	SaO_2	ESS	MoCA	SDS
ACC	Happy	*r*	−.040	.015	−.095	−.152	.223	−.127	.147	−.082
		P	.764	.912	.468	.247	.087	.334	.264	.532
	Sad	*r*	−.263[a]	.370[b]	−.212	−.260[a]	.251	−.116	.206	−.157
		P	.042	.004	.104	.045	.053	. 376	.114	.230
RTs	Happy	*r*	.343[b]	−.311[a]	.057	.122	−.051	−.042	−.152	.081
		P	.007	.015	.666	.354	.698	.747	.246	.539
	Sad	*r*	.327[a]	−.257[a]	−.033	−.016	.053	−.181	.135	−.025
		P	.011	.047	.805	.903	.689	.167	.304	.848
	DiffRT	*r*	−.105	.183	−.208	−.328[a]	.239	−.296[a]	.662[b]	−.250
		P	.425	.161	.111	.011	.066	.022	.000	.054

[a]Correlation significant at .05 level (2-tailed). [b]Correlation significant at .01 level (2-tailed). ACC = accuracy rate, AHI = apnea-hypopnea index, BMI = body mass index, DiffRT = difference value of reaction time, Edu = education, ESS = Epworth Sleepiness Scale, MoCA = Montreal Cognitive Assessment, RT= reaction time, SaO_2 = arterial oxygen saturation, SDS = Zung Self-Rating Depression Scale.

Figure 4—Correlation between difference value of reaction time (DiffRT) and Montreal Cognitive Assessment (MoCA).

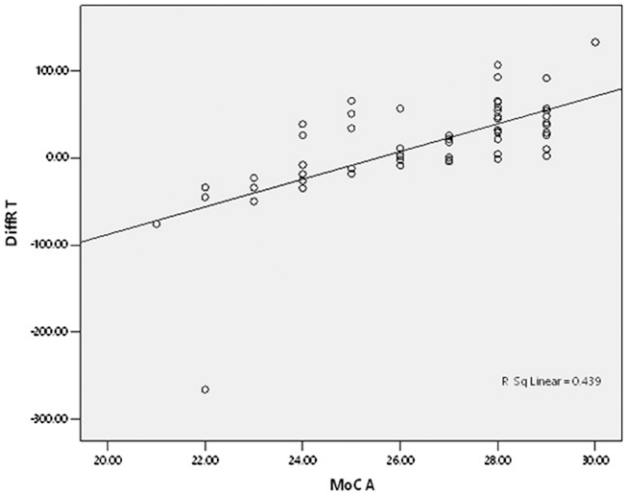
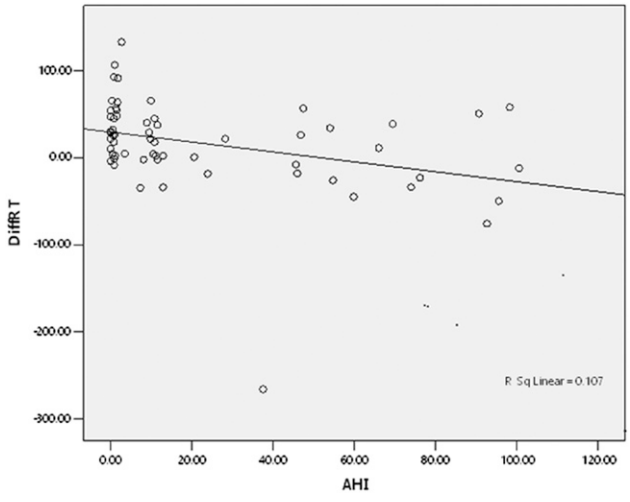


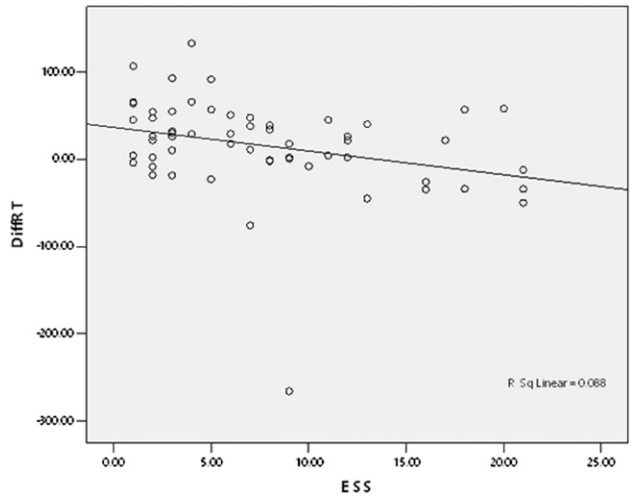
Figure 5—Correlation between difference value of reaction time (DiffRT) and apnea-hypopnea index (AHI).



abnormal based on PSG results, the phenomenon of PCA was observed in 34 patients compared with an otherwise healthy control group. Patients who identified happiness and sadness had similar accuracy rates as did the healthy control group; however, volunteers in 2 groups showed different RTs for the same task. Patients identified happiness slower than did the control group but identified sadness faster. These results suggested that the recognition of facial expression in valance was qualitatively similar but quantitatively different between the groups. In line with previous results,[24–26] the healthy control group displayed the obvious phenomenon of PCA. In agreement with our previous hypothesis, patients had impaired recognition of emotional states and showed negative emotional bias in emotion categorization. The D-value of response time between 2-face validity was different, which showed PCA in the control group and a similar time in the OSA group, and it was closely related to AHI and ESS and MoCA scores.

It has been shown that information conveyed by emotional signals is processed swiftly by the sensory systems, and depending on recognizing emotional expressions in social communication, people should understand the feelings and intentions of other people and adjust their behavior timeously to adapt to the situation.[33] Crönlein et al[15] investigated the effect of chronic sleep disturbance on emotional processing performance using a FEEL task, involving six emotional expression categories: anger, anxiety, fear, happiness, disgust, and sadness. Their results showed that patients with sleep apnea and those with psychophysiologic insomnia showed significantly worse performance in the FEEL test as compared to the control group. Differences were seen in the scales of happiness and sadness. Unlike the study of Crönlein et al, we focused on patients with OSA whose sleep disorders were identified by PSG monitoring in this study. To investigate socioemotional processing in patients with insomnia relative to healthy control patients, Kyle et al[14] found that it was less emotionally intense patients with chronic insomnia who rated facial expressions displaying sadness and fear. This finding showed for the first time that alterations of emotional processing were present in patients with

Figure 6—Correlation between difference value of reaction time (DiffRT) and Epworth Sleepiness Scale (ESS).



chronic insomnia. Although they only showed lower intensity ratings for expressions of sadness and fear on a behavioral level, this raises the possibility that chronic sleep problems may lead to alterations in emotional processing that are similar to our results. Although we demonstrated impaired facial emotion recognition in patients with chronic sleep disturbance, as reported previously,[15] our results showed further that patients with OSA performed worse in terms of facial emotional classification. Patients identified sadness faster than did the control group and showed negative emotional bias. A recent study used specific emotional expressions that were computer morphed with their confusable expression counterparts.[13] It showed that young volunteers showed reduced accuracy for identifying happiness and sadness after 1 night of total sleep deprivation, and the accuracy returned to baseline after recovery sleep. Although it is possible that different effects would be obtained if a different set of expression stimuli were tested,[13–15] the results provide additional evidence that emotional expression processing impairment was closely associated with sleep disorder. Notably, evidence from neuroimaging studies shows hypoactivation of the prefrontal cortex in patients with insomnia.[34] Similarly, abnormal amygdala activity and connectivity have been found in patients with insomnia, which is consistent with acute sleep deprivation.[35,36] It is still unclear what is the mechanism of emotional recognition impairment in patients with sleep disorder. Future work in this area will be needed to clarify this issue, especially in patients with OSA.

Our results show that the D-value of RT is closely related to AHI and not to SaO_2; however, Borges et al[37] found that executive function of patients with OSA was not related to AHI, but positively related to oxygen saturation. Hypoxia is indeed an important cause of cognitive dysfunction. In addition to structural brain abnormalities related to cognitive function, patients with chronic obstructive pulmonary disease[18] may have cognitive domains impairment, such as information processing. A experimental study suggests that chronic hypoxia would cause cognitive impairment. The researchers performed a 31-day simulated climb in a hypobaric chamber and found behavioral and mood disturbances and alterations in cognitive functions.[17] Chronic intermittent hypoxia is an important clinical feature of patients with OSA. It was supported by animal models showing chronic intermittent hypoxia resulted in cognitive impairment.[16] The results of our study showed that the expression recognition ability of patients with OSA with low SaO_2 did change. The results of correlation also showed that the response time, especially D-value of RT between different facial expressions, was related to ESS and MoCA. Considering the risk factors of cognitive function, such as chronic intermittent hypoxia and abnormal sleep rhythm, we found the higher the self-reported sleepiness score, the lower the cognitive function score. Therefore, we have reason to believe that improving the sleep quality of patients with OSA can improve their cognitive function.

Patients with OSA are accompanied by many psychological diseases, including depression. This different degree of negative emotions will affect their quality of life. Patients with depression perform abnormally in the face recognition task. They show negative emotional bias,[38,39] and even a negative interpretation of neutral mood.[40,41] In the current study, the impaired facial expression processing in patients with OSA mainly reflected negative emotional bias, showing similar response to happy face recognition but shorter time to sadness recognition compared with the control group. Patients with OSA generally have depressive emotions, which manifest as daytime sleepiness, appetite alteration, impaired concentration, and irritability.[1] Currently, it is not clear whether depressive emotions affect the performance of patients with OSA in the face recognition task. To ensure both groups in our study were matched psychologically, the SDS[42] was used to investigate whether the participants had depressive mood. Patients with OSA in the current study showed similar emotional bias as those with depression. Therefore, we were convinced that negative bias in patient performance was not caused by depressive mood.

As mentioned previously, people show a recognition speed advantage such as PCA when judging an emotional facial expression. But how do we explain the happy face advantage in expression recognition? Some different hypotheses have been proposed. Although the current data cannot explain all the possible mechanisms, these hypotheses can help us to comprehend the happy face advantage in expression recognition. Mendolia[43] holds that happy faces are the only positive expression compared with all the other expressions that share negative affective valence and cause mutual competition and interference during recognition. Other research[44] suggests that happy expressions are recognized best because they are encountered most often in daily communication. This is supported by studies in which some visual features of happy faces facilitated recognition.[45–47] Although the findings of previous studies have been summarized by meta-analysis, these conflicting views should be reconciled in future studies about the happy face recognition advantage.[33] There is mounting evidence that facial expression classification ability is impaired in patients with other diseases, such as schizophrenia,[27,28] Parkinson disease,[29] autism,[30] and Alzheimer disease,[31] as mentioned earlier. Functional magnetic resonance imaging and event-related potential[48,49] have shown that cognitive

function, including emotional face recognition processing, is impaired in patients with early-stage mild cognitive impairment. Similar to those patients, emotional face recognition of patients with OSA is abnormal, and does not show the phenomenon of PCA.

It has been shown that patients with OSA and PCA share common factors, such as sex and obesity. Women appear to be better at recognizing their own and others' emotions; therefore, we have reason to believe that our results would be affected by sex.[50–52] Unfortunately, although our data show that there is no significant sex difference between the 2 groups, it is necessary to increase the number of volunteers to observe the effect of sex on the results. Weight is an important issue regarding OSA and emotional recognition. Most of the patients with OSA are overweight and, at the same time, obesity is accompanied by sleep disorders, and even sleep apnea. It has been confirmed that individuals with obesity have impaired ability to recognize and accurately identify emotions,[53] and it is difficult to decode facial emotion even for children who are overweight or with obesity.[54] However, whether obesity has an effect on performance of facial expression recognition in our patient group should be confirmed in further studies. Although the results of this experiment suggest that RT is related to age and years in education, the further limitation of our study was that we did not consider other factors affecting the cognitive performance of patients with OSA, such as increasing age, educational level, and severity of OSA.[8] The severity of cognitive impairment correlates closely with that of OSA. It is possible that the consequences of patients' performance can better be compensated when the severity of the disease is mild. In future studies, we will focus on the effect of the severity of OSA on expression processing.

In conclusion, our findings suggest that OSA impairs cognitive function and leads to rapid perception of negative emotional expressions. This may result from the mechanisms for recognizing emotions from facial expressions. Leppänen et al[23] studied the happy face advantage with schematic faces and noted that participants decoded the faces in a holistic manner in processing happy and sad faces rather than responding to individual features alone. Miyata et al[55] clearly revealed that the perception of emotional expressions depends on the shape of the mouth. Our results confirm that patients with OSA have impaired expression classification ability. However, some questions remain. In what way do patients with OSA process emotional information in facial expression recognition tasks; is it a feature-based or holistic process? Does such negative bias lead to maintenance of their own negative emotional state or rapid detection of negative emotional messages from others? Finally, does successful treatment of OSA, such as recovery sleep after deprivation, improve performance in emotional recognition tests? There are several other factors along with OSA that are connected to impaired neurocognition. These questions will be addressed in future studies.

ABBREVIATIONS

ACC, accuracy rate
AHI, apnea-hypopnea index
BMI, body mass index
DiffRT, difference value of reaction time
ESS, Epworth Sleepiness Scale
FEEL, Facial Expressed Emotion Labeling test
MoCA, Montreal Cognitive Assessment
OSA, obstructive sleep apnea
PCA, positive classification advantage
PSG, polysomnography
RTs, reaction times
SaO_2, arterial oxygen saturation
SDS, Zung Self-Rating Depression Scale.

REFERENCES

1. McCall WV, Harding D, O'Donovan C. Correlates of depressive symptoms in patients with obstructive sleep apnea. *J Clin Sleep Med*. 2006;2(4):424–426.
2. Findley LJ, Barth JT, Powers DC, Wilhoit SC, Boyd DG, Suratt PM. Cognitive impairment in patients with obstructive sleep apnea and associated hypoxemia. *Chest*. 1986;90(5):686–690.
3. Tippin J, Sparks J, Rizzo M. Visual vigilance in drivers with obstructive sleep apnea. *J Psychosom Res*. 2009;67(2):143–151.
4. Lis S, Krieger S, Hennig D, et al. Executive functions and cognitive subprocesses in patients with obstructive sleep apnoea. *J Sleep Res*. 2008;17(3):271–280.
5. Lau EY, Eskes GA, Morrison DL, Rajda M, Spurr KF. Executive function in patients with obstructive sleep apnea treated with continuous positive airway pressure. *J Int Neuropsychol Soc*. 2010;16(6):1077–1088.
6. Bawden FC, Oliveira CA, Caramelli P. Impact of obstructive sleep apnea on cognitive performance. *Arq Neuropsiquiatr*. 2011;69(4):585–589.
7. Wallace A, Bucks RS. Memory and obstructive sleep apnea: a meta-analysis. *Sleep*. 2013;36(2):203–220.
8. Kilpinen R, Saunamaki T, Jehkonen M. Information processing speed in obstructive sleep apnea syndrome: a review. *Acta Neurol Scand*. 2014;129(4):209–218.
9. Johnston L, Miles L, Macrae CN. Why are you smiling at me? Social functions of enjoyment and non-enjoyment smiles. *Br J Soc Psychol*. 2010;49(1):107–127.
10. Tempesta D, Couyoumdjian A, Curcio G, et al. Lack of sleep affects the evaluation of emotional stimuli. *Brain Res Bull*. 2010;82(1-2):104–108.
11. Franzen PL, Buysse DJ, Dahl RE, Thompson W, Siegle GJ. Sleep deprivation alters pupillary reactivity to emotional stimuli in healthy young adults. *Biol Psychol*. 2009;80(3):300–305.
12. van der Helm E, Gujar N, Walker MP. Sleep deprivation impairs the accurate recognition of human emotions. *Sleep*. 2010;33(3):335–342.
13. Killgore WDS, Balkin TJ, Yarnell AM, Capaldi VF. Sleep deprivation impairs recognition of specific emotions. *Neurobiol Sleep Circadian Rhythms*. 2017;3:10–16.
14. Kyle SD, Beattie L, Spiegelhalder K, Rogers Z, Espie CA. Altered emotion perception in insomnia disorder. *Sleep*. 2014;37(4):775–783.
15. Crönlein T, Langguth B, Eichhammer P, Busch V. Impaired recognition of facially expressed emotions in different groups of patients with sleep disorders. *PLoS One*. 2016;11(4):e0152754.
16. Gao X, Wu S, Dong Y, et al. Role of the endogenous cannabinoid receptor 1 in brain injury induced by chronic intermittent hypoxia in rats. *Int J Neurosci*. 2018;128(9):797–804.
17. Bolmont B, Bouquet C, Thullier F. Relationships of personality traits with performance in reaction time, psychomotor ability, and mental efficiency during a 31-day simulated climb of Mount Everest in a hypobaric chamber. *Percept Mot Skills*. 2001;92(3 Pt 2):1022–1030.
18. Cleutjens FA, Janssen DJ, Ponds RW, Dijkstra JB, Wouters EF. Cognitive-pulmonary disease. *BioMed Res Int*. 2014;2014:697825.

19. Toth M, Faludi B, Wackermann J, Czopf J, Kondakor I. Characteristic changes in brain electrical activity due to chronic hypoxia in patients with obstructive sleep apnea syndrome (OSAS): a combined EEG study using LORETA and omega complexity. *Brain Topogr*. 2009;22(3):185–190.
20. Calder AJ, Young AW, Keane J, Dean M. Configural information in facial expression perception. *J Exp Psychol Hum Percept Perform*. 2000;26(2):527–551.
21. Calvo MG, Lundqvist D. Facial expressions of emotion (KDEF): identification under different display-duration conditions. *Behav Res Methods*. 2008;40(1):109–115.
22. Tottenham N, Tanaka JW, Leon AC, et al. The NimStim set of facial expressions: judgments from untrained research participants. *Psychiatry Res*. 2009;168(3):242–249.
23. Leppänen JM, Hietanen JK. Positive facial expressions are recognized faster than negative facial expressions, but why? *Psychol Res*. 2004;69(1-2):22–29.
24. Leppänen JM, Hietanen JK. Affect and face perception: odors modulate the recognition advantage of happy faces. *Emotion*. 2003;3(4):315–326.
25. Hugenberg K. Social categorization and the perception of facial affect: target race moderates the response latency advantage for happy faces. *Emotion*. 2005;5(3):267–276.
26. Hugenberg K, Sczesny S. On wonderful women and seeing smiles: social categorization moderates the happy face response latency advantage. *Soc Cogn*. 2006;24(5):516–539.
27. Kohler CG, Walker JB, Martin EA, Healey KM, Moberg PJ. Facial emotion perception in schizophrenia: a meta-analytic review. *Schizophr Bull*. 2010;36(5):1009–1019.
28. Leszczyńska A. Facial emotion perception and schizophrenia symptoms. *Psychiatr Pol*. 2015;49(6):1159–1168.
29. Clark US, Neargarder S, Cronin-Golomb A. Specific impairments in the recognition of emotional facial expressions in Parkinson's disease. *Neuropsychologia*. 2008;46(9):2300–2309.
30. Bookheimer SY, Wang AT, Scott A, Sigman M, Dapretto M. Frontal contributions to face processing differences in autism: evidence from fMRI of inverted face processing. *J Int Neuropsychol Soc*. 2008;14(6):922–932.
31. Werheid K, Clare L. Are faces special in Alzheimer's Disease? Cognitive conceptualization, neural correlates, and diagnostic relevance of impaired memory for faces and names. *Cortex*. 2007;43(7):898–906.
32. Liu XF, Liao Y, Zhou L, Sun G, Li M, Zhao L. Mapping the time course of the positive classification advantage: an ERP study. *Cogn Affect Behav Neurosci*. 2013;13(3):491–500.
33. Nummenmaa L, Calvo MG. Dissociation between recognition and detection advantage for facial expressions: a meta-analysis. *Emotion*. 2015;15(2):243–256.
34. Nofzinger EA, Buysse DJ, Germain A, Price JC, Miewald JM, Kupfer DJ. Functional neuroimaging evidence for hyperarousal in insomnia. *Am J Psychiatry*. 2004;161(11):2126–2128.
35. Beattie L, Kyle SD, Espie CA, Biello SM. Social interactions, emotion and sleep: a systematic review and research agenda. *Sleep Med Rev*. 2015;24:83–100.
36. Baglioni C, Spiegelhalder K, Regen W, et al. Insomnia disorder is associated with increased amygdala reactivity to insomnia-related stimuli. *Sleep*. 2014;37(12):1907–1917.
37. Borges JG, Ginani GE, Haehul H, et al. Executive functioning in obstructive sleep apnea syndrome patients without comorbidities: Focus on the fractionation of executive functions. *J Clin Exp Neuropsychol*. 2013;35(10):1094–1107.
38. Koster EH, De Raedt R, Goeleven E, Franck E, Crombez G. Mood-congruent attentional bias in dysphoria: maintained attention to and impaired disengagement from negative information. *Emotion*. 2005;5(4):446–455.
39. Gotlib IH, Krasnoperova E, Yue DN, Joormann J. Attentional biases for negative interpersonal stimuli in clinical depression. *J Abnorm Psychol*. 2004;113(1):121–135.
40. Milders M, Bell S, Platt J, Serrano R, Runcie O. Stable expression recognition abnormalities in unipolar depression. *Psychiatry Res*. 2010;179(1):38–42.
41. Gollan JK, Pane HT, McCloskey MS, Coccaro EF. Identifying differences in biased affective information processing in major depression. *Psychiatry Res*. 2008;159(1-2):18–24.
42. Ejaz SM, Khawaja IS, Bhatia S, Hurwitz TD. Obstructive sleep apnea and depression: a review. *Innov Clin Neurosci*. 2011;8(8):17–25.
43. Mendolia M. Explicit use of categorical and dimensional strategies to decode facial expressions of emotion. *J Nonverbal Behav*. 2007;31(1):57–75.
44. Calvo MG, Gutierrez-Garcia A, Fernandez-Martin A, Nummenmaa L. Recognition of facial expressions of emotion is related to their frequency in everyday life. *J Nonverbal Behav*. 2014;38(4):549–567.
45. Calvo MG, Fernandez-Martin A, Nummenmaa L. Perceptual, categorical, and affective processing of ambiguous smiling facial expressions. *Cognition*. 2012;125(3):373–393.
46. Calvo MG, Nummenmaa L. Detection of emotional faces: salient physical features guide effective visual search. *J Exp Psychol Gen*. 2008;137(3):471–494.
47. Calvo MG, Nummenmaa L, Avero P. Recognition advantage of happy faces in extrafoveal vision: Featural and affective processing. *Vis Cogn*. 2010;18(9):1274–1297.
48. Yamasaki T, Muranaka H, Kaseda Y, Mimori Y, Tobimatsu S. Understanding the pathophysiology of Alzheimer's Disease and mild cognitive impairment: a mini review on fMRI and ERP studies. *Neurol Res Int*. 2012;2012:1–10.
49. Yang L, Zhao X, Wang L, Yu L, Song M, Wang X. Emotional face recognition deficit in amnestic patients with mild cognitive impairment: behavioral and electrophysiological evidence. *Neuropsychiatr Dis Treat*. 2015;11:1973–1987.
50. Hall JA, Matsumoto D. Gender differences in judgments of multiple emotions from facial expressions. *Emotion*. 2004;4(2):201–206.
51. Hoffmann H, Kessler H, Eppel T, Rukavina S, Traue HC. Expression intensity, gender and facial emotion recognition: Women recognize only subtle facial emotions better than men. *Acta Psychol (Amst)*. 2010;135(3):278–283.
52. Donges US, Kersting A, Suslow T. Women's greater ability to perceive happy facial emotion automatically: gender differences in affective priming. *PLoS One*. 2012;7(7):e41745.
53. Bruch H. Hunger awareness and individuation. In: Bruch H, ed. *Eating Disorders: Obesity, Anorexia Nervosa, and the Person Within*. New York, NY: Basic Books, Inc; 1973:44-65.
54. Koch A, Pollatos O. Reduced facial emotion recognition in overweight and obese children. *J Psychosom Res*. 2015;79(6):635–639.
55. Miyata H, Nishimura R, Okanoya K, Kawai N. The mysterious noh mask: contribution of multiple facial parts to the recognition of emotional expressions. *PLoS One*. 2012;7(11):e50280.

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DISCLOSURE STATEMENT

Work for this study was performed at Center of Sleep Medicine, Shandong Provincial Hospital Affiliated to Shandong University, Jinan, Shandong, China. The authors report no conflicts of interest.