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Mixed emotions: alcoholics' impairments in the recognition of specific emotional facial expressions

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Abstract

Facial expression recognition is a central feature of emotional and social behaviour and previous studies have found that alcoholics are impaired in this skill when presented with single emotions of differing intensities. The aim of this study was to explore biases in alcoholics' recognition of emotions when they were a mixture of two closely related emotions. The amygdala is intimately involved in encoding of emotions, especially those related to fear. In animals an increased number of withdrawals from alcohol leads to increased seizure sensitivity associated with facilitated transmission in the amygdala and related circuits. A further objective therefore was to explore the effect of previous alcohol detoxifications on the recognition of emotional facial expressions.

Fourteen alcoholic inpatients were compared with 14 age and sex matched social drinking controls. They were asked to rate how much of each of six emotions (happiness, surprise, fear, sadness, disgust and anger) were present in morphed pictures portraying a mix of two of those emotions.

The alcoholic group showed enhanced fear responses to all of the pictures compared to the controls and showed a different pattern of responding on anger and disgust. There were no differences between groups on decoding of sad, happy and surprised expressions. In addition the enhanced fear recognition found in the alcoholic group was related to the number of previous detoxifications.

These results provide further evidence for impairment in facial expression recognition present in alcoholic patients. In addition, since the amygdala has been associated with the processing of facial expressions of emotion, particularly those of fear, the present data furthermore suggest that previous detoxifications may be related to changes within the amygdala.

Keywords: Alcohol withdrawal; Anxiety; CANTAB; Fear; Anger; Disgust

1. Introduction

Facial expression recognition is a central feature of emotional and social behaviour. There is now increasing evidence that the processing of different emotional facial expressions are controlled by separate neural circuits [2,6]. Neuropsychological studies and functional imaging studies have highlighted the role of the amygdala in the recognition of facial expressions of fear and possibly sadness [6,7,29]. The recognition of disgust and anger expressions do not appear to be mediated by the amygdala, more probable areas are the basal ganglia and anterior insular. Evidence for the involvement of these areas comes from patients with Huntingdon's disease who were found to be impaired in their recognition of disgust [32], and from an fMRI study that found increased activation of the anterior insular in response to mild and strong facial expressions of disgust; strong disgust also activated structures linked to a limbic cortico–striatal–thalamic circuit [29]. The orbitofrontal cortex has also been put forward as a candidate for the neural substrate of both disgust [32] and anger [6]. Furthermore, the existence of separable neuro-cognitive circuits for facial expression recognition has been demonstrated pharmacologically. Healthy volunteers who were given diazepam were found to be selectively impaired in the recognition of angry expressions [5], while propanolol an adrenergic beta-blocker increased reaction time in identifying the facial expression of sadness [13].

A number of previous studies have shown that alcoholics are impaired on the recognition of emotional facial expressions [16,17,28]. Using different intensities of morphed facial expressions Kornreich and co-workers found that alcoholics overestimated the intensity of all emotional expressions. A decoding deficit for anger and contempt

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was also reported. The work of Kornreich and co-workers has used morphed stimuli (morphing refers to the process of creating computer images along a continuum between two prototypes) depicting different intensities of separate emotions from neutral to 100% intensity. However, in many situations emotions are not entirely straightforward, surprise will often have an element of fear and disgust and anger are extremely difficult to tease apart. The purpose of this current study was to measure decoding deficits and overestimation of intensity using 'mixed emotions'.

The deleterious effects of chronic alcohol use on cognitive functions have previously been demonstrated [14,25]. An impairment in cognitive function may interact with the ability of the patients to decode facial emotional expressions. We intended therefore, in the present study to evaluate higher cognitive functions using a pattern and spatial recognition task and an extradimensional/intradimentional shift and reversal task. In both tasks recognition of visual patterns represents an important element.

A number of clinical and experimental reports suggest that previous experience of withdrawal from alcohol increases the severity of subsequent withdrawal episodes. The increased intensity of withdrawal symptoms does not simply reflect a longer period of exposure to the drug, since in animal experiments that controlled for total exposure time and dose [4,33] withdrawal sensitivity was increased in the animals that had had prior withdrawal experience. Ballenger and his co-workers [1,3] have suggested that repeated withdrawal from alcohol results in a sensitisation of brain mechanisms, similar to that occurring during epileptic kindling (increase of seizure response with repeated stimulation).

Duka et al. [11] have recently found that alcoholics with more than two previous detoxifications differed from those with two or less on 'anger' as measured by the Profile of Mood States (POMS, [20]) and in the number of errors on a modified emotional Stroop task for negative words [35]. The group with a higher number of previous detoxifications had higher anger scores and made more errors than their counterparts with fewer previous detoxifications.

Repeated withdrawal from alcohol may be associated with facilitated transmission in the amygdala as shown in amygdala kindling experiments [8,30], while also enhanced metabolic activity in limbic and cortical brain areas has been found in animals previously exposed to multiple withdrawals from alcohol [9]. In addition, Stephens et al. [33] have recently demonstrated in animals that the experience of a number of previous detoxifications from alcohol impairs the acquisition of a conditioned emotional fear response, when compared with only one previous withdrawal experience. The involvement of amygdala in the acquisition and expression of conditioned fear responses has also been previously acknowledged [15,19]. Thus, a second aim of this study was to investigate the effects of previous withdrawals on emotional facial expression recognition.

2. Methods

2.1. Study population

A total of 28 volunteers participated in the study, 14 alcohol-dependent inpatients (six women and eight men) and 14 normal controls matched for age and gender. The alcohol-dependent subjects were recruited from diagnosed alcoholics seeking treatment as inpatients at Farm Place clinic (Westminster Health Care). All of the patients presented with alcohol as their main problem but one of the patients also had concurrent illicit drug use. The normal control group consisted of 14 social drinkers with no previous history of alcohol-related problems. The control group were recruited from among the postgraduate student and staff populations at the University of Sussex via announcements on information boards. Farm Place is a private clinic and the socio-economic background of the patients attending the clinic and the recruited controls was very similar. IQ score measured by the National Adult Reading Test (NART: [24]) was also matched between groups. Volunteers were paid for their participation with an amount approximately equivalent to £5 per hour, plus any travel expenses incurred. The present study is one of a series of studies set out to investigate the effects of previous detoxifications on cognitive performance and emotional reactivity of alcohol dependent individuals (e.g. [11]).

2.2. Inclusion criteria

Alcoholic patients who participated in the trial were aged 27–63 years (mean 47.9, S.D. 10.3) and were in generally good physical condition, as documented by their medical history. Alcohol dependence was diagnosed by independent psychiatrists according to Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association 1994) or the ICD 10 classification of mental and behavioural disorders (World Health Organisation, 1992). All subjects were in alcohol abstinence at the time of the study and at least two weeks had passed since admission. Patients had been medically supported during withdrawal, with standard detoxification treatments, including administration of a non-benzodiazepine sedative (chlormethiazole; Heminevrin™), an anticonvulsant (phenytoin; Epanutin™) and a Vitamin B1 preparation (thiamine). In addition two patients had received benzodiazepines for a short period during withdrawal. At the time of testing all patients had ceased any pharmacological treatment for withdrawal. As part of their detoxification, patients underwent counselling, and intensive group, individual and family psychotherapy, based on the 12-step treatment of Alcoholics Anonymous (Minnesota model). Volunteers who constituted the group of social drinkers were aged 25 to 60 years (mean 42, S.D. 9.9). All patients and controls were of Caucasian origin.