Fear recognition impairment in early-stage Alzheimer's disease: When focusing on the eyes region improves performance

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ABSTRACT

A decline in the ability to identify fearful expression has been frequently reported in patients with Alzheimer's disease (AD). In patients with severe destruction of the bilateral amygdala, similar difficulties have been reduced by using an explicit visual exploration strategy focusing on gaze. The current study assessed the possibility of applying a similar strategy in AD patients to improve fear recognition. It also assessed the possibility of improving fear recognition when a visual exploration strategy induced AD patients to process the eyes region. Seventeen patients with mild AD and 34 healthy subjects (17 young adults and 17 older adults) performed a classical task of emotional identification of faces expressing happiness, anger, and fear in two conditions: The face appeared progressively from the eyes region to the periphery (eyes region condition) or it appeared as a whole (global condition). Specific impairment in identifying a fearful expression was shown in AD patients compared with older adult controls during the global condition. Fear expression recognition was significantly improved in AD patients during the eyes region condition, in which they performed similarly to older adult controls. Our results suggest that using a different strategy of face exploration, starting first with processing of the eyes region, may compensate for a fear recognition deficit in AD patients. Findings suggest that a part of this deficit could be related to visuo-perceptual impairments. Additionally, these findings suggest that the decline of fearful face recognition reported in both normal aging and in AD may result from impairment of non-amygdalar processing in both groups and impairment of amygdalar-dependent processing in AD.

1. Introduction

Cumulative evidence suggests that, parallel to classical episodic memory deficit, several "nonmemory" declines could occur in the early stages of Alzheimer's disease (AD). Specifically, a growing number of studies have recently focused on emotional processing in AD patients (Chaby & Narme, 2009; McLellan, Johnston, Dalrymple Alford, & Porter, 2008), showing that a decline could occur in the earliest stages of the disease (Bediou et al., 2009; Kohler et al., 2005), including mild cognitive impairment (i.e., MCI; Spoletini et al., 2008). Previous studies of emotional processing in AD have suggested that methodological factors could modulate the deficit in the context of facial emotion recognition (McLellan et al., 2008). In particular, performance could be varied across the type of emotions (Table 1), suggesting that the ability to decode positive emotions is preserved in both normal aging and AD, whereas the ability to decode negative emotions is impaired (Guaita et al., 2009; Phillips, Scott, Henry, Mowat, & Bell, 2009; Rosen et al., 2006).

In the context of negative emotions, it was proposed that the deficit occurs for specific emotions. A decline in the ability to identify fear emotions has mainly been demonstrated (Burnham & Hogervorst, 2004; Drapeau, Gosselin, Gagnon, Peretz, & Lorrain, 2009; Hargrave, Maddock, & Stone, 2002; Henry et al., 2008; Lavenu, Pasquier, Lebert, Petit, & Van der Linden, 1999; Phillips et al., 2009; Weiss et al., 2008; Wiechetek Ostos, Schenk, Baenziger, & von Gunten, 2011). Recent neuroimaging studies confirm that the "emotional brain" is touched early in AD, with histopathological and atrophic damage reported in the amygdale (Horinek et al., 2006; Poulin, Dautoff, Morris, Barrett, & Dickerson, 2011). Functional roles identified for the amygdala (Whalen & Phelps, 2009; Whalen et al., 1998) support the suggestion that deficits in the processing of facial emotion recognition should be greater for fear faces in AD patients.

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Surprisingly, considerable variations in this ability have been found across AD studies (see Table 1 and Klein-Koerkamp, Beaudoin, Baciu, & Hot, 2012). One explanation for this discrepancy has been that deficits in the identification of emotion increase with AD severity (Bediou et al., 2009; Spoletini et al., 2008; Weiss et al., 2008). Most of these studies considered only one AD sample of mildly affected patients (see Table 1, Mini-Mental State Examination ⩾18); diminished abilities were, however, shown to further increase with AD progression. Patients with moderate cognitive impairments have reduced emotional naming abilities compared with mildly affected patients (Spoletini et al., 2008; Weiss et al., 2008) further demonstrated that an early impairment in emotion decoding abilities of a low-intensity fearful face was already present in the setting of mild cognitive impairment (Spoletini et al., 2008), again emphasizing a selective deficit in the identification of fear emotions in the initial stage of AD.

A possible explanation for the selectivity of emotion recognition impairments in AD can be found in face-processing models. Numerous studies have proposed that modulating specific features among emotional faces could contribute to a change in reaction time in the labeling of emotion in healthy subjects (Adams & Kleck, 2003, 2005; Sander, Grandjean, Kaiser, Wehrle, & Scherer, 2007). For example, a threatening stimulus that displays faces with a direct gaze enhances the perception of anger, whereas displaying faces with an averted gaze facilitates the perception of fear (Adams & Kleck, 2003). The eyes thus carry information relevant to perceiving emotion, and this was demonstrated particularly with fear emotions. As used in the "bubbles" paradigm, authors have demonstrated that the eyes are particularly relevant for discriminating fear from other emotions (Schyns, Petro, & Smith, 2007; Smith, Gosselin, & Schyns, 2007). Interestingly, AD patients show difficulties in extracting this crucial emotional information from a visual stimulus. By measuring eye movements, Ogrocki, Hills, and Strauss (2000) reported that AD patients spent less time scanning the emotional face and, in particular, fixated less on the eyes area than healthy older adults (HOAs) did. Although AD patients "see" the emotional stimuli, they (1) possibly explore their environment less, or (2) may not cognitively process visual information in the same way that HOAs do (Daffner, Mesulam, Cohen, & Scinto, 1999; Daffner, Scinto, Weintraub, Guinessey, & Mesulam, 1992; Scinto et al., 1994). Similar disturbances in identifying facial features have been observed in autistic disorders (Dawson, Webb, & McPartland, 2005) with particular difficulties in processing the eyes region (Klin, Jones, Schultz, Volkmar, & Cohen, 2002). As in AD, oculomotor exploration revealed that autistic patients spent less time on the eyes region than healthy subjects did (Hernandez et al., 2009). These AD deficits translated to apparent impairments in facial emotion identification, which were therefore related to impaired visual exploration strategies (Ogrocki et al., 2000). Crucially, AD patients may allocate attention differently to the most salient aspects of the face (Ogrocki et al., 2000; Scinto et al., 1994).

Moreover, numerous AD researchers have examined visual and perceptual abilities as a potential cognitive deficit suspected to interfere with emotional decoding scores (Albert, Cohen, & Koff, 1991; Bucks & Radford, 2004; Burnham & Hogervorst, 2004; Cadieux & Greve, 1997; Guaita et al., 2009; Koff, Zaitchik, Montepare, & Albert, 1999; Ogrocki et al., 2000; Roudier et al., 1998). They made the assumption that the emotional deficits are related to a deficit in visuo-spatial processing or impairment in the ability to visually extract relevant facial traits. To control visual processing, numerous researchers have assessed the ability of AD patients to process facial cues through their ability to process facial identity (Table 1). Among these control tasks, the Benton Facial Recognition Test (Benton, 1983), the facial gender discrimination task, and the identity discrimination tasks of the Florida Affect Battery (Bowers, Blonder, & Heilman, 1998) are commonly used (Table 1). When AD patients' cognitive and perceptual abilities were controlled for, the deficit in emotional recognition was significantly decreased using morphing (Maki, Yoshida, Yamaguchi, & Yamaguchi, 2012) or statistical procedures (see Table 1; Albert et al., 1991; Cadieux & Greve, 1997; Freedman, Binns, Black, Murphy, & Stuss, 2012; Hargrave et al., 2002; Henry et al., 2008; Klein-Koerkamp et al., 2012). This result means, therefore, that AD patients' emotional performances could be improved when perceptual impairments are taken into account. Nevertheless, none of the previous AD studies has investigated whether emotional performance could be improved by modulating a patient's visual exploration strategy. This assumption is supported by findings from a well-designed study performed by Adolphs et al. (2005) on a patient (SM) who presented massive and bilateral damage of the amygdala. SM had a selective impairment in fear recognition, which disappeared when she was instructed to judge the facial expression by focusing on the eyes region. The fact that SM was able to identify fearful faces by orienting attention on the eyes region highlighted the key role of the amygdala. In addition to being a structure of fear processing (Breiter et al., 1996; Fusar-Poli et al., 2009; Morris et al., 1996; Vuilleumier & Pourtois, 2007; Whalen et al., 2004), the amygdala, when atrophied, could induce impairment of a visual exploration strategy (Adolphs et al., 2005), leading to difficulties in the extraction of salient features of the emotional stimulus.

Congruent with the idea that patients with AD have difficulties in processing emotions, in particular of fear, we aim to test the hypothesis that these deficits could be remediated. Knowing the contribution of visuo-perceptual deficits to emotional performance in AD (Klein-Koerkamp et al., 2012), we predict that fear recognition can be improved by modulating the visual exploration strategy, which suggests a deficit in the spontaneous direction of visual attention to this face region, instead of a loss in the ability to process emotional information. In the protocol, we presented faces in two conditions: (a) global, which presents the whole face; and (b) eyes region-to-face, which presents first the eyes followed by the other facial regions. Emotion recognition was tested in mild AD patients as compared with healthy participants (older and younger adults). We predicted that (a) AD patients would show impairment of fear recognition in the global condition, and (b) the deficit would be corrected during the eyes region condition. To assess the specificity of the eyes region condition, we also assessed the ability of AD patients to recognize angry faces. Most studies have reported that the recognition of anger was reduced in HOAs compared with young controls (Calder et al., 2003; Henry et al., 2008; Maki et al., 2012), but that there was no significant difference between AD and HOA performance (Table 1; Bucks & Radford, 2004; Burnham & Hogervorst, 2004; Drapeau et al., 2009; Fernandez-Duque & Black, 2005; Hargrave et al., 2002; Lavenu et al., 1999; Weiss et al., 2008; Wiechetek Ostos et al., 2011). Anger is, however, sometimes more impaired in patients for specific emotional tasks (Bediou et al., 2009; Granato, Godefroy, Van Gansberghe, & Bruyer, 2009; Henry et al., 2008; Maki et al., 2012; Phillips et al., 2009; Spoletini et al., 2008). On the whole, we expect that the assessment of anger identification will provide a useful index of changes in emotion processing during normal aging.

2. Method

2.1. Participants

Fifty-one subjects participated in the study: 17 were patients with probable mild AD (mean age ± SD: 74.1 ± 4.2 years), 17 were healthy young adults (mean age ± SD: 21 ± 2.4 years), and 17 were HOAs (mean age ± SD: 74.1 ± 4.1 years). There were 10 males and seven females in each group. All subjects were native French speakers and were recruited in the Neuropsychology Unit at the