

Emotion processing in Alzheimer's disease

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Abstract

Emotion processing deficits may have an important effect on the quality of life of Alzheimer's disease (AD) patients and their families, yet there are few studies in this area and little is known about the cause of such deficits in AD. This study sought to determine if some AD patients have a disruption in a specific right hemisphere emotion processing system, and to determine if the processing of emotional facial expression is more vulnerable to the pathology of AD than is the perception of emotional prosody. It was specifically hypothesized that patients with greater right hemisphere dysfunction (low spatial AD patients) would be impaired on emotion processing tasks relative to those with predominantly left hemisphere dysfunction (low verbal AD patients). Both groups showed impairment on emotion processing tasks but for different reasons. The low verbal patients performed poorly on the affect processing measures because they had difficulty comprehending and/or remembering the task instructions. In contrast, low spatial AD patients have emotion processing deficits that are independent of language and/or memory and may be due to a more general visuo-perceptual deficit that affects the perception of static but not dynamic affective stimuli. (*JINS*, 1997, 3, 411–419.)

Keywords: Alzheimer's disease, Emotion processing, Lateralization of function

INTRODUCTION

Loss of the ability to express feelings and to comprehend the affective expression of others is potentially one of the most important factors leading to the degradation of quality of life in demented patients, contributing to their alienation from family members, and producing caregiver stress. (Nadeau, 1990)

In fact, Greve et al. (1994) found that the quality of the marital relationship in Alzheimer's disease (AD) was significantly associated with the patient's ability to accurately perceive the emotional state of their spouse, even when the effect of cognitive impairment and problem behavior was removed. Nonetheless, there are few studies in this area, and little is known about the emotion processing defects in the AD population. The purpose of the present study is to better understand the cognitive mechanism underlying the emotion processing deficits reported in AD.

The term *emotion processing* refers to the cognitive processes involved in the ability to (1) comprehend the emotional state of others using cues provided in facial expression, or the intonation of speech (prosody); and/or (2) commu-

nicate one's own internal emotional state via the same mechanisms (facial expression, vocal prosody). While many neural systems are involved in emotion processing, considerable evidence has accumulated over the past 10 to 15 years indicating that an independent mechanism residing in the parietotemporal area of the nondominant (usually right) hemisphere is responsible for the comprehension and expression of facial-prosodic emotional cues (for detailed reviews of lateralization of emotion processing in brain damage and normals, see Heilman et al., 1993, and Ley & Strauss, 1986, respectively).¹ Important evidence comes from patients with unilateral focal lesions.

Emotion Processing with Unilateral Focal Lesions

In one of the earliest studies of the perception of emotional facial expressions in unilateral stroke, DeKosky et al. (1980)

¹The finding of right hemisphere dominance for the processing of all emotional stimuli is remarkably consistent across both normal and patient samples, though some studies of emotion processing (e.g., Mandel et al., 1991) suggest that the right hemisphere may be particularly important for the processing of negative emotion.

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found that right hemisphere damaged (RHD) patients were consistently impaired relative to left hemisphere (LHD) patients on facial emotion processing tasks. Further, using tasks similar to those of DeKosky et al. (1980), Bowers et al. (1985) determined that the emotion processing deficit seen in RHD patients could not be attributed to a general deficit in visuoperceptual processes. Bowers et al. (1984) proposed a “right hemisphere iconic field” that may contain the schema or prototypes for affective facial expressions. Similarly, Rapcsak et al. (1989) suggested that impairments in affective expression identification may result from a “disconnection between visual semantic and verbal semantic representations for facial emotions,” supporting Bowers’ and Heilman’s hypothesis. These studies clearly indicate that RHD patients are differentially impaired on tasks assessing their ability to comprehend emotional facial expression.

Similar findings have been reported for the ability to accurately perceive emotional prosody. Heilman et al. (1975) presented RHD and LHD patients with semantically neutral tape recorded sentences (e.g., “The boy went to the store”) which were read in four different emotional intonations (happy, sad, angry, indifferent) to patients with right temporoparietal infarctions and to aphasic patients with left temporoparietal infarctions. The patients’ task was to identify the emotional tone of the speaker. Patients with right hemisphere lesions performed more poorly on this task than those with left hemisphere lesions, suggesting that the right hemisphere is more critically involved in processing affective intonations of speech.

Heilman et al. (1984) provide additional evidence that the right hemisphere “is behaviorally dominant for comprehending emotional prosody” (p. 920). They instructed RHD and LHD patients to listen to “speech-filtered” sentences (which render the semantic message unintelligible) and to indicate their comprehension of emotional (happy, sad, angry) or non-emotional (questions, commands, statements) prosody. They found that both the RHD and the LHD groups were equally impaired on the nonemotional prosody, relative to normal controls. However, on the emotional prosody task, the RHD patients performed significantly more poorly than the LHD patients. These results suggest that, although both hemispheres may be important in comprehending propositional prosody, the right hemisphere is dominant for comprehending affective intonations.

Bowers et al. (1987) used a similar paradigm, making semantic content congruent with emotional content in one-half of the sentences (e.g., “The couple beamed at their brand new grandson” said in a happy tone of voice), and incongruent with emotional content in the other half of the sentences (e.g., “The couple beamed at their brand new grandson” said in an angry tone of voice). They found that RHD patients were more impaired than LHD patients on overall comprehension of emotional prosody conveyed by semantically meaningful sentences, and that comprehension of emotional prosody was worse when it was incongruent with semantic content than when it was congruent. Thus, the findings concerning hemispheric dominance for

comprehension of emotional prosody are consistent with studies of emotional face perception in implicating a right-hemisphere-specific mechanism underlying the perception of nonverbal emotional communications.

Emotion Processing in Alzheimer’s Disease

Early AD pathology predominantly affects the hippocampus and posterior association cortex (Kemper, 1984). It seems likely that such pathology would damage the same right hemisphere areas implicated in the emotion processing deficits in focal lesion cases. Unfortunately, the literature concerning the emotion processing abilities of AD patients is sparse, and the results are mixed. Three experimental studies (Brosigole et al., 1981; Allender & Kaszniak, 1989; Albert et al., 1991) have demonstrated impaired emotional processing abilities in AD patients. However, since AD patients almost always perform more poorly than normal controls on most cognitive tasks, it is difficult to determine the source of their impairment on emotion processing tasks. Albert et al. (1991) did attempt to disconfound the factors that might contribute to the observed deficits by statistically covarying performance on a variety of nonemotional cognitive tasks. When this was done, differences between normal controls and AD patients disappeared, leading to the conclusion that the emotion processing impairments in AD were secondary to general cognitive decline. Nonetheless, this study did not take into account the diversity of deficits seen among individual patients with AD nor the possibility that different patients may perform poorly on emotion tasks for different reasons. Thus the question remains as to whether disruption of a specific emotion processing mechanism may contribute to the emotion processing deficits seen in AD patients.

Interestingly, clinical lore suggests that in real world settings affective processing abilities remain intact even after other cognitive functions, such as language and memory, have become significantly impaired. For example, Bartol (1979) states that “the person with Alzheimer’s disease is capable, probably to an even greater extent than prior to illness, of perceiving the emotional climate of his environment” (p. 22). She suggests that nurses use body language (e.g., posture, and eye contact) and vocalizations (e.g., rate, level, loudness, tone, and pitch) to communicate with the patients. Moreover, caregiver guides often advocate non-verbal communication strategies with AD patients. For example, *The Clinical Management of Alzheimer’s Disease* (Brown et al., 1988) recommends that the “family can be helped to learn that tone of voice is often more important than words, that a low soothing voice may accomplish far more than a voice of authority” (p. 123). Thus, the implication is that, at the very least, sensitivity to the prosodic characteristics of vocalizations is retained in AD.

The discrepancy between the experimental literature and clinical lore concerning emotion processing in AD patients needs to be reconciled. General cognitive decline may indeed cause the observed deficits on emotion processing tasks

in AD, but in the published studies the roles of general cognitive impairment and specific emotion processing abilities (either facial expression, prosody, or both) have not been satisfactorily examined. The purpose of the present study is to unconfound emotion processing ability and cognitive impairment experimentally and determine the role of each in the performance of AD patients on tasks designed to tap emotion processing ability. Further, since the clinical lore emphasizes the use of prosody over facial expression in non-verbal communication with AD patients, this study will compare the perception of emotion in faces to that of prosody to determine if one is more affected by AD.

Lateralization of Dysfunction in AD

The sticky methodological point is the proper control for cognitive impairment. Recent data concerning the lateralization of dysfunction that occurs in some AD patients provide a means of designing a study that is methodologically analogous to the studies of emotion processing in focal lesion patients described above, and in which general cognitive ability is controlled. Several functional imaging studies (Katzman et al., 1978; Foster et al., 1983; Martin et al., 1986) have recorded lateralized patterns of hypometabolism in early AD patients that were associated with consistent patterns of cognitive impairment. For example, Martin et al. (1986) found that AD patients with prominent language deficits (low verbal AD) showed decreased metabolism on PET primarily in the left hemisphere, whereas those with prominent spatial deficits (low spatial AD) showed decreased metabolism in their right hemisphere. These same patterns of functional asymmetry have also been observed on neuropsychological evaluation (Becker et al., 1988). In fact, Delis et al. (1992) have devised a method for dividing AD patients into groups based on the nature of their cognitive impairment (see below). This scheme has been validated using measures of global and local processing that are sensitive to the hemispheric laterality of lesion in focal cases (Delis et al., 1988).

In summary, low spatial AD patients exhibit cognitive impairments characteristic of right hemisphere pathology and show abnormal physiological activity primarily in the right hemisphere; similarly, low verbal AD patients exhibit impairment that is more characteristic of left hemisphere pathology, and have left hemisphere abnormalities on PET scans. Both the behavioral and physiological findings are consistent with those of unilateral focal lesion patients, with the exception that AD patients are more likely to show additional general cognitive impairment; especially memory problems. It follows logically, then, that low spatial AD patients should be impaired on measures of emotion processing relative to the low verbal AD patients. Careful matching of the sample should effectively rule out general cognitive impairment as an explanation of the observed results. If one wishes to argue that the relative deficit in low spatial AD patients is due to dysfunction of a specific emotion processing mechanism, one must demonstrate the independence of visuo-perceptual deficits from emotion processing ability.

METHOD

Research Participants

AD patients were recruited through local support groups, and fliers distributed by local physicians in the greater New Orleans area. The normal control (NC) participants were recruited via community senior citizen groups. All participants were right-handed and free of a history of alcoholism, psychiatric illness, serious cardiovascular disease, head trauma, multi-infarct dementia, or other severe neurological conditions. Thirty-four AD patients met diagnostic criteria developed by NINCDS/ADRDA (McKhann et al., 1984) for probable AD. AD participants were excluded if they scored less than 110 on the Dementia Rating Scale (DRS; Mattis, 1988), a measure of general cognitive function.

The AD patients were assigned to one of three groups based on their performance on the Boston Naming Test (BNT; Kaplan et al., 1983) and Wechsler Intelligence Scale for Children–Revised Block Design subtest (BD; Wechsler, 1974). Following the procedure of Delis et al. (1992), BNT and BD raw scores were converted to standard scores and a difference score was calculated ($z_{\text{BNT}} - z_{\text{BD}}$). Participants with difference scores of 1.00 or more (BNT > BD) were assigned to the low spatial (LS) AD group. Participants with difference scores of -1.00 or less (BD > BNT) were assigned to the low verbal (LV) AD group. Any AD participants with both test scores in the normal range ($1.96 > z > -1.96$) were excluded. NC subjects with either test score in the impaired range ($z \leq -1.96$) were excluded.

The final sample consisted of 8 LS–AD patients (1 male, 8 female), 10 LV–AD patients (2 male, 8 female), and 15 (1 male, 14 female) NC participants. Four participants in each AD group scored in the normal range on one grouping test and the impaired range on the other. The remaining AD participants were impaired on both grouping tasks, though performance on one was significantly more impaired than on the other. Excluded from this study were 2 AD patients with normal BD and BNT scores, 2 NC participants with one impaired score (1 BD, 1 BNT), and 5 AD patients with a bilateral presentation (difference score $< \pm 1.00$). Nine AD participants did not meet the minimum DRS cutoff.

Florida Affect Battery

The Florida Affect Battery (FAB; Bowers et al., 1991) was used to measure emotion processing. Variations of this task have been used in several studies of emotion processing after unilateral stroke (e.g., Bowers et al., 1985; Blonder et al., 1992). The FAB consists of 10 subtests (see below), each designed to evaluate either visual (facial expression), auditory (prosody), or visual/auditory cross-modal emotion processing. Two tasks are designed to evaluate the discrimination of nonemotional faces or prosody for control purposes. Unless otherwise stated the score for each subtest is the percent correct responses. A total of 13 FAB scores were analyzed.

1. *Facial Identity Discrimination* consists of vertically arranged pairs of female faces with neutral expressions and covered hair. Half of the pairs consist of identical photographs of the same person and half consist of photographs of two different persons. The task is to indicate whether the photographs are of the same or different persons. This is a control task.
2. *Facial Affect Discrimination* measures the ability to discriminate emotional facial expressions across different persons. Twenty pairs of vertically arranged faces are presented. The two faces in each pair are never the same person, but for half the pairs the two people have the same expression and for the other half they have different expressions. The task is to indicate whether the facial expressions are the same or different.
3. *Facial Affect Naming* consists of 20 individual faces with happy, sad, angry, frightened, or neutral expressions. The subject must name the emotional expression on each face. The choice of affect names is repeated for each item if necessary.
4. *Facial Affect Selection* requires the participant to select from a vertically arranged set of five faces the one face which bears the expression named by the examiner. All photographs are of different people and each has a different expression (happy, sad, angry, frightened, neutral).
5. *Facial Affect Matching* requires the participant to select from among a vertically arranged set of five faces the one face that bears the same expression as the stimulus face. The persons making the stimulus and target expressions are never the same and there is always one *identity foil*. The identity foil is a photograph of the same person as the stimulus but with a different expression. The number of times the identity foil was selected is the identity error score.
6. *Nonemotional Prosody Discrimination* requires the participant to indicate whether the prosody of 16 sentence pairs is the same or different. Both sentences in each pair contain the same words (e.g., the chair is made of wood) but may differ in terms of propositional (as opposed to emotional) prosody (i.e., one may be read as a declarative sentence and the other may be read as interrogatory). Half of the pairs have the same prosody, and the other half are different. This is a control task.
7. *Emotional Prosody Discrimination* is exactly like Subtest 6 except that it requires the participant to indicate whether semantically neutral sentence pairs are spoken in the same or different emotional tone. This subtest contains 20 trials.
- 8a. *Name the Emotional Prosody* requires the participant to name the emotional tone (happy, sad, angry, frightened, neutral) in which each sentence is spoken. The semantic content of the sentence is neutral (e.g., the shoes are in the closet) so the affect can only be determined by prosody.
- 8b. *Conflicting Emotional Prosody* requires the participant to choose the affect of each of 32 sentences based on their emotional tone while ignoring the emotional content of the sentence. In incongruent sentences (8b–I) the affect expressed by prosody differs from the affect suggested by the semantic content (e.g., “All the puppies are dead” said in a happy tone). In congruent sentences (8b–C) the prosody and content indicate the same affect.
9. *Match Emotional Prosody to Emotional Face* requires the participant to judge the affect expressed by the prosody of a sentence and then pick the photograph expressing the same affect from a set of three photographs.
10. *Match Emotional Face to Emotional Prosody* requires the participant to judge which affect is expressed by an emotional face and indicate which of these three sentences has emotional prosody that matches that expression.

RESULTS

All analyses were conducted using SPSS, Release 6.0 (Norusis, 1993). Unless otherwise stated, all variables were analyzed with one-way ANOVAs and followed up with Scheffé tests.

Sample Characteristics

The NC participants were significantly younger [$F(2,30) = 8.35, p < .01$] and less impaired on the DRS [$F(2,30) = 57.85, p < .01$]; however, there was no difference for education [$F(2,30) = 4.89, p > .01$]. All three groups differed on BD standard score [$F(2,30) = 77.94, p < .01$], with the NC group scoring highest and the LS group lowest. Similar performance was seen for the BNT standard scores [$F(2,30) = 77.94, p < .01$], with the LV–AD participants scoring lowest. All three groups differed on the difference score [$F(2,30) = 48.53, p < .01$], with the LSs scoring highest and the LVs scoring lowest. Thus, the two AD groups were matched on age and total DRS score, and differed in the appropriate direction on the classification variables. Please refer to Table 1 for descriptive statistics. Demographically and cognitively, the five excluded bilateral AD patients were comparable to the lateralized patients except that their BNT and BD performances did not differ.

Florida Affect Battery

The FAB scores analyzed were the percentage of correct responses on each subtest. Because of the large number of variables, the critical alpha level was adjusted to .01 to reduce the risk of Type I error associated with multiple analyses. Four clusters of outcomes were observed and are described below. Details of these analyses are presented in Table 2.

Table 1. Sample descriptive statistics

Test	Group		
	Normal control <i>M</i> (SD)	Low verbal <i>M</i> (SD)	Low spatial <i>M</i> (SD)
Age	69.1 (4.9) ^a	77.6 (5.5) ^b	75.9 (6.5) ^b
Education	12.5 (1.5) ^a	15.9 (3.8) ^a	14.0 (1.6) ^a
DRS total score	141.2 (2.7) ^a	121.3 (7.8) ^b	123.6 (3.8) ^b
Block Design (std)	.88 (.95) ^a	-1.35 (.59) ^b	-3.34 (1.04) ^c
Boston Naming Test (std)	.11 (1.02) ^a	-4.76 (.49) ^b	-.42 (.86) ^c
Difference score	-.77 (1.01) ^a	-3.41 (.89) ^b	2.92 (1.87) ^c

^{abc}Row means with the same letter are not significantly different at $\alpha < .01$

1. No group effects were found for Facial Identity Discrimination, Emotional Prosody Discrimination, Conflicting Emotional Prosody–Congruent or Conflicting Emotional Prosody–Incongruent.
2. The NCs and LS–AD patients performed significantly better than the LV–AD patients on Facial Affect Naming, Facial Affect Matching, Identity Errors, Emotional Prosody Naming, and Prosody to Face Cross Modal Matching.
3. The NCs performed significantly better than both AD groups on Facial Affect Discrimination and Facial Affect Selection.
4. The NCs performed better than the low verbal group, while the low spatial group did not differ from either on Nonemotional Prosody Discrimination or Face to Prosody Cross Modal Matching.

Single Subject Analysis

Because group analyses can obscure important findings the individual performances of the 8 AD patients who performed normally on one grouping task and in the impaired range on the other were analyzed. These individuals were selected because they showed classical dissociations (Shallice, 1988) between verbal and spatial abilities and were thus the most likely to reveal differences in affect processing if they are present. *Z* scores for each subtest were calculated based on the distribution of the NC subjects; *z* scores at or beyond -2.0 indicated impairment (the impaired direction was positive for identity errors). Examination of these data in Table 3 indicates that the individual performances were consistent with the group results. The most consistent finding was that all low verbal participants performed in the impaired range for identity errors on Subtest 5, while the

Table 2. Florida Affect Battery Subtest means

Test	Group			<i>F</i>
	Normal control <i>M</i> (SD)	Low verbal <i>M</i> (SD)	Low spatial <i>M</i> (SD)	
Facial tasks				
1. Identity Discrimination	97.3 (7.8) ^a	88.5 (10.6) ^a	93.8 (6.4) ^a	3.30
2. Affect Discrimination	89.0 (6.9) ^a	78.5 (4.1) ^b	76.9 (2.6) ^b	18.19
3. Affect Naming	89.3 (3.7) ^a	81.0 (8.1) ^b	89.4 (6.2) ^a	6.90
4. Affect Selection	99.3 (1.8) ^a	89.0 (9.9) ^b	90.0 (4.6) ^b	11.14
5. Affect Matching	89.3 (6.5) ^a	72.5 (8.9) ^b	82.5 (8.0) ^a	14.53
5. Identity Errors	.47 (.64) ^a	2.20 (.63) ^b	.75 (.71) ^a	22.26
Prosody tasks				
6. Nonemot. Prosody Discrim.	98.0 (2.9) ^a	77.5 (20.3) ^b	89.4 (6.5) ^{ab}	9.19
7. Emotional Prosody Discrim.	97.7 (4.6) ^a	91.5 (11.6) ^a	95.0 (5.3) ^a	2.02
8a. Emotional Prosody Naming	88.0 (9.2) ^a	65.5 (17.4) ^b	83.8 (8.8) ^a	10.70
8b. Conf. Emot. Prosody (C)	90.3 (7.7) ^a	71.9 (25.8) ^a	90.0 (7.5) ^a	4.84
8b. Conf. Emot. Prosody (I)	76.1 (10.0) ^a	52.1 (31.1) ^a	63.4 (28.0) ^a	3.39
Crossmodal tasks				
9. Emot. Pros. to Emot. Face	93.7 (5.2) ^a	72.0 (14.2) ^b	87.5 (9.3) ^a	15.38
10. Emot. Face to Emot. Prosody	97.0 (3.2) ^a	72.0 (16.2) ^{ab}	84.4 (16.8) ^a	12.72

^{abc} Row means with the same letter are not significantly different at $\alpha = .01$

Table 3. Single subject analysis of FAB performance for each AD participant showing a classical dissociation on the grouping tasks

Task	Participant							
	Low verbal				Low spatial			
	A16	A10	A57	A05	A60	A51	A55	A58
Facial tasks								
1. Identity Discrimination	V	+	+	+	+	+	+	+
2. Affect Discrimination	V	+	+	+	V	V	+	+
3. Affect Naming	V	+	V	V	+	+	+	+
4. Affect Selection	V	+	V	V	V	V	V	+
5. Affect Matching	V	V	+	V	+	V	V	+
5. Identity Errors	V	V	V	V	+	+	+	+
Prosody tasks								
6. Nonemot. Pros. Discrim.	V	+	V	V	+	+	V	V
7. Emotional Pros. Discrim.	+	+	+	+	+	+	+	+
8a. Name the Emot. Prosody	V	+	V	V	+	+	+	+
8b. Conf. Emot. Pros. (C)	V	+	+	+	+	+	+	+
8b. Conf. Emot. Pros. (I)	+	+	+	V	V	+	+	+
Crossmodal tasks								
9. E. Pros. to E. Face	V	V	V	V	V	+	+	+
10. E. Face to E. Pros.	V	V	V	V	+	V	+	V

Note: 'V' indicates that the observed score was at least 2 standard deviations from the NC mean in the impaired direction, while '+' indicates the performance was within normal limits.

performance of all low spatial participants was within normal limits (Fisher's Exact Test, $p = .014$). In fact, when all AD participants were examined, only one low verbal individual had a performance within normal limits, and only one low spatial individual was impaired.

Analyses of Covariance

Contrary to hypothesis, when the AD groups differed, the LV participants perform worse than the low spatial participants; this implies the possibility that language dysfunction seriously impacted the LV patients' performance on the affect tasks. The LV participants also committed significantly more identity errors. Identity errors were significantly correlated ($p < .01$) with BNT ($r = -.78$), but not BD ($r = -.34$, $p > .05$). Further, identity errors were significantly correlated with scores on all FAB subtests on which the LV participants scored significantly below the low spatial participants. These findings suggest that some of the group differences observed may be the result of a task comprehension problems rather than a deficit in emotion perception *per se*.

Thus, all analyses for which there were group effects were rerun while covarying the identity error score. Only Facial Affect Discrimination [$F(2,29) = 13.23$, $p < .01$] and Facial Affect Selection [$F(2,29) = 5.47$, $p = .01$] showed significant group effects when identity errors were covaried. Examination of the adjusted means showed that the low spatial group performed significantly worse than the NC group on both tasks. The LV-AD group did not differ from either

the NC or the low spatial AD groups. When BNT score was covaried the results were identical. Thus, when the effect of language dysfunction is removed, the low spatial AD patients show emotion processing deficits relative to the low verbal AD patients.

DISCUSSION

Summary

Using an analogue of the methods used to study emotion processing in focal lesion cases, the present study sought to determine if some AD patients have a disruption in a specific right hemisphere emotion processing system or whether the deficits reported in earlier studies are the result of more general cognitive impairment, or disruption of a specific emotion processing system. Further, it attempted to determine if the processing of emotional facial expression was more vulnerable to the pathology of AD than the perception of emotional prosody. This study took advantage of recent research indicating that many mildly impaired AD patients show significantly lateralized patterns of brain and behavioral pathology. It was specifically hypothesized that patients with greater right hemisphere dysfunction (low spatial AD) would be impaired on emotion processing tasks relative to those with predominantly left hemisphere dysfunction (low verbal AD).

Two sets of findings are striking. First, neither AD group differed from the NC group on measures of emotional pros-

ody comprehension (the only exception was Name the Emotional Prosody). Further, the low spatial AD group never differed from the NCs on any task that had a prosodic component. Differences did occur, however, between NC and at least one AD group on all emotion processing tasks that involved faces. This suggests that the processing of emotional prosody is relatively spared in AD. Second, contrary to hypothesis, when differences between AD groups occurred, the low verbal AD patients were consistently more impaired. With some tests this result can be explained by the fact the low spatial AD patients were impaired in their general naming ability. The other tasks tend to have more complex instructions that may be hard to comprehend or remember. Thus the difficulty experienced by the low verbal patients may be due to what Shallice (1988) calls "task-demand artefacts" that "can result from . . . a comprehension disorder arising from aphasia or dementia" (p. 229). Task-demand artefacts may also explain why the low verbal patients were strongly influenced by the identity foil in the Facial Affect Matching task. The low spatial participants rarely made identity errors. Their clearer understanding of the task demands may have been the reason.

A Specific Emotion Processing Deficit in AD?

While task-demand artefacts may explain the performance of the low verbal patients on a number of tasks, they cannot explain the performance of both sets of patients on Facial Affect Discrimination and Facial Affect Selection. The two tests on which both AD groups differed from the NC participants share the characteristics of having extremely simple instructions and being easy to execute. Facial Affect Discrimination simply asks the participant to determine if the two pictured people are feeling the same or different. The two people are always different, so there is no risk of being confused by identity. In Facial Affect Selection, the participant merely selects the face with the expression named by the examiner. An important question arises from the results of these two tests. If the other tasks on which group effects occurred were performed poorly by the low verbal group because of their general comprehension deficit, and the low spatial patients, lacking significant language impairment, were unimpaired, why should the low spatial participants suddenly show significant impairment, both as a group and individually, on two relatively simple emotion processing tasks?

This question was addressed by covarying the identity error score, and thus statistically controlling for any comprehension problems that might have influenced performance. When this was done, group effects were eliminated on all tasks except the two the low spatial participants were impaired on. Further, while the low spatial patients remained impaired, the performance of the low verbal participants no longer differed from the NC group. In other words, removal of the effects due to comprehension and/or mem-

ory impairment helped the low verbal participants, but had no effect on the performance of the low spatial participants. These findings have several implications.

First, the poor performance of the low verbal group cannot be attributed to general cognitive decline, since both AD groups were matched in terms of general cognitive ability (DRS total score). It appears that the low verbal participants performed poorly on the affect processing measures because of specific language-related deficits. Thus, the emotion processing defects observed in the low verbal group are an experimental artefact and do not indicate their actual ability to comprehend emotional facial and vocal expressions.

Second, the low spatial group was specifically impaired on facial emotion tasks. This deficit cannot be attributed to a deficit in general face perception, because the low spatial patients, individually and as a group, were unimpaired on the identity discrimination task. Further, their difficulty cannot be attributed to either general cognitive decline, nor to more specific comprehension difficulties. This implies that the low spatial AD patients, like their counterparts with focal right hemisphere lesions, have a disruption in a cognitive system specific to the processing of emotional facial expression. This is, however, not the only explanation for these findings. An alternative will be discussed below.

Resiliency of Emotional Prosody

Preliminary examination of the pattern of results, with and without covarying identity errors and for group and individual findings, suggests that perception of emotional prosody is spared in AD, compared to the perception of facial emotion. There are two alternative explanations. First, an examination of group data reveals that the performance of the AD participants, particularly the low verbal group, on the prosody tasks was extremely variable compared with that of the other participants. Thus, the failure to find statistical differences for prosody may be a function of low statistical power due to small sample size in the face of relatively large within-group variability. Two pieces of data, however, suggest otherwise. First, reanalyzing the group data (with and without the covariate) excluding the highly variable low verbal group produces the same nonsignificant results. Second, the individual performances of the low spatial group on the emotional prosody tasks were uniformly unimpaired relative to the normals. Thus, while the AD samples were relatively small and variable, the unimpaired performance of the low spatial group on the prosody tasks cannot be attributed to statistical artefact.

The second explanation concerns the specific nature of the tasks themselves. In order to infer that processing of emotional prosody is spared, the face and prosody tasks must be comparable. However, the face and prosody tasks used in this study differ in an important way. The facial stimuli are still photographs, and thus present emotions as static visual patterns. In the natural environment, however, facial expressions evolve; they are dynamic events (Gibson & Spelke, 1983; Hofsten, 1983). In contrast, and by necessity, the pro-

sodic stimuli are audiotaped utterances. In these utterances, the emotional expression is dynamic and the task is much more similar to the kind of emotion prosody task an individual will encounter in the natural environment. Thus, the face and prosody tasks are qualitatively different. It is possible that, because of their static nature, the facial photographs lack essential information that would allow the AD patients to more accurately interpret the portrayed emotion.

This hypothesis is supported by research on the development of emotion perception in infants. Caron et al. (1985) have argued that “because photographs provide none of this dynamic information, they undoubtedly pose a much more formidable discriminative problem for the infant” (p. 1558). Infants as young as 5 months appear sensitive to dynamic affective facial expression and vocal prosody (Kreutzer & Charlesworth, 1973; Walker, 1981) but may be insensitive to emotion in static faces beyond even 9 months or age (Caron et al., 1985). Further, dissociations between facial and prosodic emotional expressions like that reported here have also been observed in infants. Seven-month-old infants could discriminate videotaped facial expressions when they were accompanied by appropriate vocal prosody but not when they were presented silently (Caron et al., 1985). Similar infants could, however, distinguish prosody alone (Walker-Andrews & Grolnick, 1983). Thus, developmental research indicates that static facial photographs are not as rich emotionally as either dynamic presentations of facial expression or vocal prosody.

It is possible that the impaired performance of the low spatial AD patients is actually the result of a subtle visuo-perceptual deficit. Visuo-perceptual function (specifically face perception) may be inferred from performance on the Identity Discrimination subtest (Bowers et al., 1985). However, this task, too, may be qualitatively different from the facial emotion tasks. Whereas a facial expression is a dynamic event, identity can be discerned easily from static faces. Further, the ability to discriminate between static faces based on emotion develops much later than the ability to differentiate static faces based on such features as age and sex (Fagan & Singer, 1979). The Identity Discrimination task requires only a template matching strategy (i.e., the stimuli for a *same* discrimination are identical photographs, thus a single discrepancy between a pair of photographs is enough to mark them as *different*) so normal performance may be achieved even in the presence of a subtle visuo-perceptual disturbance that is sufficient to disrupt perception of emotion in static faces. Thus, the visuo-perceptual ability of the low spatial AD patients may be adequate for template matching but inadequate to make discriminations based on emotional facial expression.

If the deficit observed in the low spatial participants reflects a subtle visuo-perceptual disturbance, then the dynamic evolution of an emotional facial expression, as on videotape (or in the natural environment), may provide more cues that an AD patient can utilize in making their judgments of facial expression. These judgments may then be as accurate as those with prosody. If, however, it is a conse-

quence of a damaged emotion processing system, then one would expect continued difficulty regardless of whether the facial stimuli are static or dynamic.

Conclusions

Cognitive impairments; specifically, language-related deficits, negatively influence performance on experimental emotion processing tasks because they affect comprehension of task demands. It is not clear that these same factors negatively impact emotion processing in the natural environment. Clinical lore would suggest otherwise (Bartol, 1979; Brown et al., 1988). Further, some AD patients (i.e., those who have functional impairments similar to persons with focal right hemisphere lesions) have deficits in the perception of emotion that are independent of language comprehension and/or memory. These deficits appear, at first glance, to be specific to the perception of emotional facial expression with perception of emotional prosody spared. However, the fact that the static facial photographs may not be as rich emotionally as the audio recordings of emotional prosody raises the possibility that the observed emotional face perception impairment in low spatial AD patients is, in fact, the result of a more general visuo-perceptual deficit rather than dysfunction of a specific emotion processing mechanism.

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