Prosody impairment and associated affective and behavioral disturbances in Alzheimer's disease

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Article abstract—We examined the ability to produce, repeat, and comprehend emotional prosody in 20 patients with Alzheimer's disease (AD) and in 11 elderly normal control subjects. In addition, caregivers of AD patients completed affective and behavioral measures with reference to the patient. Relative to control subjects, comprehension of emotional prosody was marginally impaired in mildly demented AD patients, whereas production, comprehension, and repetition of emotional prosody were significantly impaired in moderately demented AD patients. The moderately demented patients performed significantly poorer than the mildly demented patients on the production and repetition tasks. In contrast, there was no significance difference between the two groups on the prosody comprehension task. Additional analyses revealed an inverse relationship between the ability to correctly produce and repeat emotional prosody and the frequency of agitated behaviors and depressive symptomatology in moderately demented patients. This latter finding suggests that the inability to communicate emotional messages is associated with disturbances in mood and behavior in AD patients. Implications for the management of disruptive behavior in agitated and aprosodic AD patients include the development of caregiver sensitivity to unexpressed emotion and caregiver assistance with emotional expression.

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Little is known about the integrity of systems that underlie communication of affective information in patients with Alzheimer's disease (AD). Most studies that have examined emotional prosody, or the nonverbal affective components of language, have used normal control subjects and patients with unilateral brain damage. These studies suggest that the integrity of the right hemisphere is important for both expression and comprehension of emotional prosody.[1-3] Ross[2] proposed that the right hemisphere is organized for affective language expression and comprehension in a manner that is comparable to the organization of propositional language in the left hemisphere. However, in a recent review, Borod[4] concluded that prosody may be more diffuse and nonlocalized within the right hemisphere. The integrity of the right hemisphere is also implicated in the processing of affective facial information[5-8] and in sensitivity to both pictorial and linguistic versions of emotional situations.[5] Bowers et al.[9] suggested that the right hemisphere contains a nonverbal affect lexicon that is modular in organization, with independent lexicons for prosody and faces. They speculated that this system can be dissociated from other components of the emotion system, such as experience and expression.

In AD, damage to right hemisphere structures occurs as part of the widespread progressive degeneration, suggesting that emotional prosody and other areas of affective communication may be compromised. Indeed, Allender and Kasniak[10] demonstrated that AD patients were impaired relative to age-matched control subjects in discriminating and identifying affective facial cues and prosody. In another study,[11] AD patients performed significantly worse on tasks involving assessment of recognition of facial emotion, labeling of facial emotion, and recognition of emotion in both drawings and verbal descriptions of emotional situations. Although these studies provide some support for impairment in the discrimination or comprehension of affective communication in AD patients, the ability to produce or express emotionally intoned speech has not been investigated.

One purpose of the present study was to examine emotional prosody expression, repetition, and comprehension in AD patients. We hypothesized that affective language deficits, like other higher cognitive functions, would become progressively impaired as a function of dementia severity. In a study of linguistic language functioning in AD patients, Appell et al.[12] reported that transcortical sensory and sensory aphasias were prominent in the early stages of AD, with global aphasia appearing at the end stage. If affective language deficits mirror linguistic language deficits in AD, then transcortical sensory and sensory aprosodias may predominate in the ear-

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Table 1 Mean age, education, and MMSE score for each group

	Mild AD (n = 10)	Moderate AD (n = 10)	Control subjects (n = 11)
Age	71.1 (5.6)	74.0 (9.7)	70.8 (8.8)
Education	15.8 (3.2)	15.5 (3.0)	15.3 (2.7)
MMSE*	23.6 (1.1)	19.7 (1.3)	28.5 (1.6)

* All groups significantly different, $p < 0.05$.

Standard deviations are given in parentheses.

lier stages of AD, with global aprosodias appearing in the latter stages. To assess the possible parallel between linguistic and affective language impairment, we included both mildly and moderately demented AD patients and examined performance in the various components (i.e., expression, repetition, comprehension) of prosodic communication both between and within the groups.

A second purpose of this study was to determine whether relationships exist between prosody impairment and disturbances in affect and behavior in AD patients. Behavioral and emotional disturbances are common concomitants of AD. Although the frequency of particular behavioral problems varies by report, symptoms of depression and agitation are common.[13] There are few studies that examined the relationships between behavioral disturbances and neurocognitive deficits. In the current investigation, we hypothesized that impairment in the ability to express or comprehend emotional prosody would be associated with agitation, disturbances in affect, or both.

Methods. *Subjects.* Twenty patients with AD were studied. All patients were diagnosed by neurologists at the Emory University School of Medicine using National Institute of Neurological and Communicative Disorders and Stroke-Alzheimer's Disease and Related Disorders Association criteria[14] for probable AD. Patients received the prosody battery during their diagnostic evaluation or after enrollment in research projects. Patients were divided into two groups based on a median split of their Mini-Mental State Examination (MMSE) scores, resulting in assignment of patients with relatively less cognitive impairment to the "mildly demented" group (MMSE, 22 to 25) and those with relatively more cognitive impairment to the "moderately demented" group (MMSE, 17 to 21). Community volunteers and spouses of AD patients were recruited to form a group of 11 elderly control subjects (MMSE, 26 to 30). None of the AD patients or elderly control subjects had other conditions known to cause cognitive deficits, such as significant head trauma, stroke, alcoholism, or psychiatric illness. None of the participants were taking psychoactive medications at the time of the evaluation or in the months immediately preceding the evaluation. Table 1 shows the mean age, education, and MMSE for each group. Analyses of variance (ANOVA) revealed that the three groups were comparable in age and education ($p > 0.05$) and significantly different with respect to MMSE ($p < 0.05$). The research protocol was approved by the university's institutional review board. Informed consent was obtained before data collection.

Prosody measures. Elicitation of prosody. This measure was used to assess the subject's ability to accurately produce emotional prosody in response to command. Twelve recorded sentences, with accompanying text, were presented to the subjects. The vocal intonation of the speaker and the content of each sentence were emotionally neutral. Before the presentation of each sentence, the subject was asked to reproduce the sentence in either a surprised, an angry, or a sad tone of voice. A cartoon face with the appropriate facial expression was also provided to decrease memory demands. A predetermined, random order of emotions was followed.

The subjects' responses were recorded on audiotape for later analysis. Each sentence was classified as either correct or abnormal. Abnormal responses were further classified as either aprosodic or inappropriately prosodic (paraprosodic). One reviewer rated all of the audiotapes, and a second reviewer independently rated 50% of the tapes. The reviewers were unaware of the grouping status of the subjects. Interrater agreement was high (82%); consequently, only the ratings from the primary reviewer were used for statistical purposes. Ratings were unavailable for one subject from the mildly demented AD group and for one subject from the moderately demented AD group because of poor tape quality.

Repetition of prosody. This measure was used to test the subject's ability to accurately reproduce or imitate emotional prosody. Twelve recorded sentences, with accompanying text, were presented to the subjects. Although the content of the sentences was emotionally neutral, sentences were conveyed with surprise, sadness, or anger. The three emotions were randomly interspersed throughout the 12 sentences. Subjects were asked to repeat the sentence using the same emotional tone as the model on the tape.

As before, subjects' responses were recorded on audiotape and rated by two independent reviewers who were unaware of the grouping status of the subjects. Responses were classified as described above. Interrater agreement was high (84%). As before, only the ratings of the primary reviewer were used for statistical purposes. Ratings were unavailable for one subject from each group because of poor tape quality and tape recorder malfunction.

Comprehension of prosody. This measure was used to assess the subject's ability to correctly identify emotional prosody. Twelve recorded sentences with emotionally neutral content were presented to the subject. Each sentence was conveyed with either surprise, sadness, or anger. The subject was asked to identify the emotional intonation of the speaker as either surprised, sad, angry, or neutral. Before the beginning of the subtest, four cartoon pictures with verbal labels representing each emotion were placed in front of the subjects. This was done to decrease the load on memory and to rule out anomia as a possible explanation for poor performance. Subjects were allowed to respond verbally or to point to the appropriate cartoon face.

All responses were scored as either correct or incorrect according to an answer key. Pilot testing indicated that healthy young control subjects were able to correctly classify all sentences without difficulty.

Affective and behavioral measures. Family members of AD patients were asked to complete the Cohen-Mansfield Agitation Inventory[15] and the Cornell Depression Invento-

ry[16] with reference to the patient. The Cohen-Mansfield Agitation Inventory is a questionnaire consisting of 29 agitated behaviors (e.g., restlessness, spitting, hitting, pacing) that are rated on a seven-point Likert scale for frequency over the previous 2-week period. Higher scores reflect greater frequencies of agitated behaviors. The Cornell Depression Inventory is a 19-item questionnaire that assesses symptoms of depression (e.g., weight loss, agitation, difficulty sleeping, suicidal ideation, irritability) in demented patients. Symptoms are rated as absent, mild/intermittent, or severe (0 to 2 points). A category reflecting uncertainty on the part of the rater is also provided. Higher scores reflect more depressive symptomatology. Family members' ratings on these inventories were used to assess whether impairments in prosody were related to behavioral or mood disturbances in the AD patients.

Procedure. Subjects were administered the prosody tasks as part of a comprehensive neuropsychological evaluation. The elicitation task was always administered first; the repetition and comprehension subtests were then administered and counterbalanced across subjects to control for order effects. Before the beginning of the study, three sets of 12 sentences devoid of emotional content were recorded twice, with each set recorded in both an emotionally neutral and a predetermined emotionally prosodic manner (e.g., sad, surprised, and angry were randomly interspersed). During administration of the tasks, a different set of sentences was used during each prosody subtest. In addition, the two versions (neutral or emotionally intoned) of the three sets were counterbalanced across prosody subtests and subjects to control for any possible differences in difficulty among the three sets of sentences.

Results. *Between-group differences in prosody performance.* Between-group differences in prosody performance were examined via three separate (e.g., elicitation, repetition, and comprehension) 3 (group) × 3 (affect type) mixed-model ANOVAs based on the percentage of correct responses in each condition. "Group" was the between-subjects factor, and "affect type" was the within-subject factor.

Prosody elicitation. There was a significant main effect of group [$F(2,26) = 10.61$, $p < 0.001$]. Post-hoc Newman-Keuls analysis indicated that control subjects (mean, 75%; SD, 38%) and mildly demented AD patients (mean, 68%; SD, 36%) performed significantly better than moderately demented AD patients (mean, 26%; SD, 36%). There was no significant difference between the control subjects and mildly demented AD patients. There was no significant main effect of affect type [$F(2,52) = 2.26$, $p > 0.05$]. Finally, the interaction between group and affect type was not significant [$F(4,52) = 0.81$, $p > 0.05$].

Prosody repetition. There was a significant main effect of group [$F(2,25) = 6.5$, $p < 0.01$]. Post-hoc Newman-Keuls analysis indicated that control subjects (mean, 83%; SD, 27%) and mildly demented AD patients (mean, 70%; SD, 36%) performed significantly better than moderately demented AD patients (mean, 41%; SD, 36%). There was no significant difference between the control subjects and mildly demented AD patients. As in the elicitation condition, there was no significant main effect of affect type [$F(2,50) = 2.27$, $p > 0.05$], or an interaction between group and affect type [$F(4,50) = 2.37$, $p < 0.05$].

Prosody comprehension. There was a significant main effect of group [$F(2,28) = 4.25$, $p < 0.05$]. Post-hoc Newman-Keuls analysis indicated that the mildly (mean, 67%; SD, 30%) and moderately demented (mean, 57%; SD, 35%) AD patients performed comparably ($p = 0.30$). In contrast, the performance of the control subjects (mean, 83%; SD, 25%) was significantly better than that of the moderately demented AD patients ($p < 0.01$), and marginally better than that of the mildly demented AD patients ($p = 0.08$). There also was a significant main effect of affect type [$F(2,54) = 16.74$, $p < 0.001$]. Angry prosody was significantly more difficult to comprehend than either sad prosody ($t = -3.5$, $p < 0.001$) or surprise prosody ($t = -5.7$, $p < 0.001$). In addition, sad prosody was more difficult to comprehend than surprise prosody ($t = -2.2$, $p < 0.04$). The interaction between group and affect type was not significant [$F(4,54) = 0.65$, $p > 0.05$].

Within-group differences in prosody performance. Within-group differences in prosody performance were examined via separate repeated measures ANOVAs that examined the percentage of correct task responses on each prosody task (elicitation, repetition, and comprehension) for each group. No significant effects of task emerged for either the control subjects [$F(2,18) = 2.3$, $p > 0.05$] or the mildly demented AD patients [$F(2,16) = 0.05$, $p > 0.05$]. In contrast, a significant main effect of task [$F(2,16) = 5.8$, $p < 0.02$] emerged for the moderately demented AD patients. Their performance on the elicitation task was significantly poorer than their performance on the comprehension task ($t = -3.4$, $p < 0.01$). This suggests that performance in the various components of affective communication differentially declines as AD dementia progresses.

Analysis of aprosodic and paraprosodic errors. Error type on elicitation and repetition tasks was examined by calculating the proportion of a particular error type (aprosodic or paraprosodic) relative to the total number of errors on a given task. For example, the proportion of aprosodic errors on the elicitation task was calculated by dividing the number of aprosodic errors by the total number of errors in this condition. Calculation of error types in this manner minimized the difficulty of comparing groups with differential prosody performance. Individual subjects with perfect performances were not included in the subsequent analysis. A one-way ANOVA that examined the proportion of aprosodic errors revealed a main effect for group on the elicitation task [$F(2,22) = 7.96$, $p < 0.01$]. Post-hoc Newman-Keuls analyses revealed that the moderately demented AD patients made a significantly higher proportion of aprosodic errors (mean, 62.2; SD, 43.97) than either the mildly demented AD patients (mean, 8.57; SD, 14.25) or the control subjects (mean, 13.19; SD, 24.48). There was no significant difference between the latter two groups. Although the moderately demented AD patients also made a higher proportion of aprosodic errors on the repetition task (mean, 41.32; SD, 31.97) than either the mildly demented AD patients (mean, 14.29; SD, 20.25) or the control subjects (mean, 24.4; SD, 38.1), the results of a one-way ANOVA did not reveal a significant main effect of group [$F(2,19) = 1.59$, $p = 0.23$].

Given the lack of independence between the two types of possible errors (e.g., errors were classified as either aprosodic or paraprosodic), the mean proportion of paraprosodic errors for the groups on each task would naturally

Table 2 *Percentage of subjects in each group who performed within normal limits (e.g., within 1 SD from the control group's mean), within the mildly impaired range (e.g., within 1 to 2 SDs from the control group's mean), and within the severely impaired range (e.g., below 2 SDs from the control group's mean) on each prosody subtest*

	Elicitation			Repetition			Comprehension		
	Normal	Mild	Severe	Normal	Mild	Severe	Normal	Mild	Severe
Control subjects	82%	18%		80%	20%		82%	18%	
Mildly demented AD patients	67%	33%		67%	11%	22%	70%	20%	10%
Moderately demented AD patients	22%	44%	33%	22%	33%	44%	60%	10%	30%

be a mirror image of the mean proportion of aprosodic errors. This indicates that the moderately demented AD patients made fewer paraprosodic errors on the elicitation and repetition tasks than either the mildly demented AD patients or the control subjects. Although the mildly demented AD patients made a slightly higher proportion of paraprosodic errors than the control subjects, the difference was not significant.

Relationship between prosody and behavioral disturbances. Correlational analyses were performed to examine the possible relationships between prosody task measures (e.g., number correct on each prosody task combined across affect type; number correct for each affect type combined across prosody tasks) and behavioral and mood disturbances (e.g., scores on the Cohen-Mansfield Agitation Inventory and scores on the Cornell Depression Inventory). Separate analyses were conducted for the mildly and moderately demented AD groups because MMSE combined across the groups was positively correlated with prosody impairment and any significant correlations that may have emerged could have been attributed to the severity of dementia. Furthermore, no significant correlations emerged between MMSE and any of the prosody performance measures when the groups were examined separately ($p > 0.05$).

As table 2 indicates, there was considerable within-group variability in performance, with some individuals in both the mildly and moderately demented AD groups still performing within normal limits on each prosody task. There also was considerable variability in scores on the Cornell (mildly demented: mean, 7.4; SD, 5.3; range, 2 to 19; moderately demented: mean, 9.3; SD, 8.5; range, 2 to 30) and Cohen-Mansfield (mildly demented: mean, 46.3; SD, 18.7; range, 33 to 88; moderately demented: mean, 44.5; SD, 14.4; range, 30 to 76) inventories.

In the mildly demented AD patients, a significant positive correlation was observed between Cornell scores and the accuracy of performance on tasks that required processing of sad prosody ($r = 0.69$, $p < 0.04$). Thus, as depressive symptomatology increased, mildly demented AD patients comprehended, repeated, and produced sad prosody more accurately. No other significant correlations emerged.

In the moderately demented AD patients, significant negative correlations were observed between Cohen-Mansfield scores and performance on the prosody elicitation task ($r = -0.67$, $p < 0.05$) and the prosody repetition task ($r = -0.81$, $p < 0.01$) as well as on tasks that required processing of angry prosody ($r = -0.75$, $p < 0.02$) and surprise prosody ($r = -0.72$, $p < 0.03$). A significant negative correlation also emerged between Cornell scores and accuracy on tasks that used angry prosody ($r = -0.68$, $p < 0.05$). Thus, as agitation and depressive symptomatology increased, the patients' ability to correctly produce and repeat prosody decreased.

Discussion. The results of the present study suggest that AD patients have deficits in production, repetition, and comprehension of emotional prosody. Specifically, we found that comprehension of emotional prosody was significantly impaired in moderately demented AD patients and marginally impaired in mildly demented AD patients compared with normal elderly control subjects. There was no significant difference in emotional prosodic comprehension between the mildly and moderately demented AD patients. This suggests that deficits in comprehension of emotional prosody appear relatively early in the course of AD and are relatively resistant to further decline throughout the mild and moderate stages of the disease. The moderately demented AD patients were also significantly impaired in their ability to produce and repeat emotional prosody relative to control subjects and mildly demented AD patients. However, the mildly demented AD patients did not differ from the control subjects in these abilities. These observations suggest that repetition and production of emotional prosody are relatively preserved early in the course of the illness but that significant impairment emerges by the time the AD patient reaches the middle stages of the disease.

Although longitudinal studies are needed to confirm this impression, our results suggest that the pattern of progression in prosody impairment throughout the earlier stages of AD may differ as a function of component of affective communication. In both the control subjects and the mildly demented AD patients, there was no difference in relative performance on emotional prosody production, repetition, and comprehension tasks, suggesting equivalent abilities across the various components of prosodic communication. However, the moderately demented AD patients' performance on the production task was significantly inferior to their performance on the comprehension task. Thus, moderately demented AD patients exhibit a relative strength in emotional prosody comprehension and a relative weakness in emotional prosody production.

Finally, the moderately demented AD patients made significantly more aprosodic errors and signifi-

cantly fewer paraprosodic errors than either the mildly demented AD patients or the control subjects. These observations suggest that the difficulties in production of emotional prosody displayed by moderately demented AD patients are primarily associated with emotional aprosody or the absence of prosody rather than with the presence of inappropriate emotional prosody.

Previous research[12] with AD patients suggests that transcortical sensory and sensory aphasias are frequent in earlier stages, that linguistic comprehension declines at a relatively greater rate with the passage of time, and that global aphasia appears at the end stage of the disease. Similar to the reported linguistic language comprehension deficits in AD, we found a more rapid early decline in comprehension of emotional prosody relative to other modes of prosodic communication. The mildly demented AD patients in the present study exhibited very mild deficits in comprehension of emotional prosody with relatively intact repetition and production (transcortical sensory aprosodia) compared with normal elderly control subjects. The more advanced AD patients exhibited deficits in all areas of prosodic communication (global aprosodia) but had relatively greater deficits in production. Overall, the results of the present study suggest that impairment in the various components of prosodic communication approximately mirrors impairment in the various components of linguistic communication, at least during the early stages of AD. Our results also suggest that the rate of decline in affective expression may be greater at an earlier stage of the disease. Unfortunately, we were unable to examine severely demented AD patients because our pilot studies indicated that they had significant difficulty with comprehension of the prosody task instructions.

Although impairment in emotional prosodic communication skills increases as a function of severity of AD, there is considerable individual variability, at least at the mild and moderate stages of AD. Consequently, associations between measures of affective and behavioral functioning and impairment in emotional prosody are of considerable interest. In mildly demented AD patients, overall severity of prosody impairment was unrelated to severity of depressive or agitated symptomatology. However, a positive relationship did emerge between scores on the Cornell Depression Inventory and accuracy on tasks requiring mildly demented AD patients to process sad prosody. Thus, as depressive symptomatology increased, mildly demented AD patients comprehended, produced, and repeated sad prosody more accurately. This may simply reflect mood congruent performance. Alternatively, the positive relationship between the processing of sad prosody and depressive symptomatology could be related to a third variable, such as awareness of deficit. Like prosody performance, awareness of deficit may be dependent on the integrity of the right hemisphere.[17] Auchus et al.[18] found that AD patients who were unaware of their deficits performed more poorly on neuropsychological measures sensitive to right hemisphere functioning than did a group of aware AD patients. Furthermore, depression is more common in more mildly demented patients,[19] who also maintain preserved awareness of deficit.[20] Thus, the positive association between depressive symptomatology and the processing of sad affective information may also be related to the integrity of a common underlying neurologic substrate in the right hemisphere, which also mediates awareness of deficit. This possibility is supported by a study with stroke patients[21] that found a positive relationship between aprosody and anosognosia. Finally, there were few relationships between prosody impairment and measures of emotional and behavioral disturbances in mildly demented AD patients, whose overall performance on prosody tasks differed minimally from that of elderly control subjects.

In contrast to the mildly demented AD patients, several significant relationships emerged between impairment in prosody performance and severity of affective and behavioral disturbances in moderately demented AD patients. There were negative associations between Cohen-Mansfield scores and the accuracy of performance on both prosody repetition and production tasks. Thus, as impairment in production and repetition of emotional prosody increased, agitated behaviors increased. Furthermore, increased frequency of agitated behaviors and depressive symptomatology were also associated with greater impairment on tasks that assessed angry prosody. Finally, increased frequency of agitated behaviors was associated with greater impairment on tasks that assessed surprise prosody. One possible explanation for the association between impairment in emotional prosody and increased agitation and affective disturbances is that decreased ability to communicate emotional messages may contribute to disturbances in behavior and mood.

There is considerable support for a relationship between inadequate nonverbal emotional communication and behavioral disturbance. Failure to express emotion may relate to development of a number of psychological and medical disorders.[22-28] Decreased nonverbal emotional expression in type A individuals is associated with higher scores on personality measures of anger and aggression.[24] In contrast, family support of emotional expressiveness has been associated with exhibition of positive social and interpersonal behaviors. In a study examining parents' support of their children's emotional expressiveness, nonhostile expression of emotion was associated with greater peer popularity, more positive self-concept, and more positive social behavior.[29]

The relationship between expression of emotional messages and behavior has implications for management of AD patients, particularly those with disturbances in affect and behavior. Caregivers and significant others should be encouraged to assist AD patients with emotional communication. For exam-

ple, communication may be enhanced by frequently asking the patient how a given situation or event makes them feel. Empathic statement or restatement of the patient's feelings may also be helpful. Probably of greatest importance is the development of caretaker awareness that the patient may be unable to express their emotions adequately, the development of caretaker sensitivity to detect unexpressed emotion, and the development of caretaker ability to assist the patient in expression of their emotions. The results of a study by Vitaliano et al.[30] demonstrated that caretakers who are high in expressed emotion and also demonstrate greater levels of depression and suppressed anger and lower levels of life satisfaction report more behavioral disturbances in their family members with AD. This observation suggests that emotional expressiveness in caregivers is associated with behavioral disturbances in AD patients. Caregivers with greater levels of depression and suppressed anger and with lower levels of life satisfaction may be less sensitive to and have more difficulty identifying unexpressed emotion in their care recipients. This may be especially devastating for patients with an impaired ability to express emotions. A complex relationship among the AD patient's ability to communicate emotional messages, caregiver sensitivity to unexpressed emotion and ability to assist with emotional expression, and behavioral disturbance is suggested. Further research on the relationship between these variables may be of considerable value.

In conclusion, the results of the present study demonstrate that affective components of language, like other higher cognitive functions, are impaired in AD patients. Although the progression of impairment observed in affective components of communication approximately mirrors the progression of impairment observed in linguistic components of communication during the early stages of AD, the rate of progression of affective language impairment may be more rapid. It is unclear whether this more rapid decline may also apply to propositional prosody because such studies are not available. Our results also demonstrate positive relationships between impairment in communication of emotional prosody and disturbances in affect and behavior. This relationship and its possible association with caregiver behavior, particularly in ecologically valid settings (e.g., home), merit further examination because assistance in expression of emotional messages may decrease disruptive behaviors, which frequently lead to institutionalization in AD patients. Research is also needed to examine the integrity of multimodal and higher-level affective communication skills (e.g., sensitivity to various emotions in emotional intoned situations and events) in AD patients. Findings from such research may have significant implications for management issues and treatment planning.

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Post-traumatic movement disorders in survivors of severe head injury

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Article abstract—The present study investigates the occurrence of post-traumatic movement disorders in survivors of severe head injury. We studied a series of 398 consecutive patients who were admitted to the hospital with a Glasgow Coma Score of 8 or less after they sustained a head trauma. One hundred thirty-four out of 398 patients (34%) died after they were admitted to the hospital or in the further course. A recent follow-up was obtained in 221 of the 264 remaining patients (84%). Follow-up consisted of a three-level assessment, including questionnaires, telephone interviews, and personal examinations. Fifty out of 221 patients (22.6%) had developed movement disorders secondary to the head trauma, which were transient in 23 patients (10.4%) and persistent in 27 patients (12.2%). Forty-two patients (19%) had tremors, nine (4.1%) had dystonia, and seven (3.2%) had other movement disorders. Twelve patients (5.4%) had disabling low-frequency kinetic tremors (2.5 to 4 Hz) or dystonia, or both. Low-frequency kinetic tremors developed with a latency from 2 weeks to 6 months after trauma, and dystonia with a latency from 2 months to 2 years. When compared with patients without movement disorders, this subgroup was characterized by a different distribution profile of Glasgow Coma Scores with a higher proportion of lower scores on admission ($p < 0.05$). When we compared the initial CT findings, there were highly significant associations between generalized brain edema and the occurrence of any movement disorders, between generalized brain edema and the occurrence of persistent movement disorders, and between generalized brain edema and the occurrence of kinetic tremors and dystonia. We detected similar associations for focal cerebral lesions, but not for subdural and epidural hematomas. In conclusion, transient or persistent movement disorders are common sequelae in survivors of severe head injury. Disabling movement disorders such as kinetic tremors and dystonia, however, occur only in a small group of patients.

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There is a causal relationship between head trauma and several different movement disorders.[1-8] Possible etiologic links between head injury and neurodegenerative movement disorders such as Parkinson's disease are, however, controversial.[9-12] Since movement disorders after static brain lesions may be apparent only after a variable latency,[13,14] their occurrence after head trauma may be underestimated. Recent reviews have redrawn attention to head trauma as being an important etiologic factor in a variety of movement disorders.[15-17] Particularly, tremors and dystonia are recognized sequelae of severe head trauma.[2-8,18-20]

There is no previous epidemiologic study on post-traumatic movement disorders after severe head trauma. Some studies evaluated the frequency of particular movement disorders in restricted, mainly pediatric populations.[21-24] These studies reported a wide variability ranging from 13 to 66%. There was no significant increase in standardized morbidity ratios for parkinsonism or Parkinson's disease in adults with head trauma.[25]

We analyzed the occurrence of post-traumatic movement disorders in surviving patients of a series of 398 consecutively admitted patients who sustained a severe head trauma.

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Prosody impairment and associated affective and behavioral disturbances in Alzheimer's disease

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