Major Depression in Stroke Patients
A 3-Year Longitudinal Study

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Background and Purpose: This prospective study was designed to examine the contributions of neurobiological, functional, and psychosocial factors to major depression after stroke. In addition, the prevalence and longitudinal course of major depression were studied.

Methods: Major depression, functional ability, and social network were assessed repeatedly for a period of 3 years in a population-based cohort of 80 patients with acute stroke (mean age, 73 years). Cerebral atrophy and brain lesion parameters were determined from computed tomographic scans performed acutely and after 3 years.

Results: The prevalence of major depression was 25% at the acute stage and approximately the same at 3 months (31%). It decreased to 16% at 12 months, was 19% at 2 years, and increased to 29% at 3 years. The most important predictors of immediate major depression were left anterior brain lesion, dysphasia, and living alone. Dependence in activities of daily living was the most important predictor at 3 months. From 12 months on, the patient's having few social contacts outside the immediate family contributed most to depression, and at 3 years cerebral atrophy also contributed. At 1 year, 60% of the patients with early depression (0 to 3 months) had recovered; those not recovered at this follow-up had a high risk of development of chronic depression.

Conclusions: The study has provided evidence of a differentiation of factors likely to be implicated in the development of depression after stroke based on the period of time since the stroke event. (Stroke 1993;24:976-982)

KEY WORDS • activities of daily living • depression • social support

Depressive disorders often follow a stroke. The reported prevalences vary widely from less than 25%1 to more than 60%2 depending on the selection of patients, the time elapsed since the stroke, and different diagnostic criteria. Numerous studies, recently summarized,3,4 have given conflicting information not only on prevalence but also on pathogenesis and course of the depressive disorders after stroke. The disorder has been explained as an understandable response of patients to their losses.5 From a neurobiological viewpoint, the importance of lesion location has been emphasized.2,6-10 The contributions of physical impairment and psychosocial factors to depression after stroke are less well known and probably change over time.11,12

For a greater understanding of the contributions of cerebral, functional, and psychosocial parameters to depressive disorder after stroke, we have thoroughly followed a population-based cohort of stroke patients with repeated assessments of major depression, functional ability, and social network at regular time intervals for a period of 3 years. The aim of this study was to (1) determine the prevalence of major depression during the immediate poststroke period and after 3 months, 1 year, 2 years, and 3 years; (2) identify neurobiological, functional, and psychosocial factors related to major depression at the various time points and examine their relative importance over time; and (3) examine the longitudinal course of major depression after stroke.

Subjects and Methods

All patients admitted to the stroke unit of the Department of Medicine, Umeå University Hospital (Sweden), during 1 year were considered for the study (n=98). This unit admits a population-based sample of patients who have had an acute stroke of no more than 1 week's duration.13,14 In the consecutive series of 98 patients, 16 died early after admission, 1 patient was excluded because of congenital mental handicap, and 1 refused to participate. The remaining 80 patients are the subjects of the study. Mean age was 73 years (range, 44 to 100 years); 49 were men and 31 women. The majority (80%) suffered from their first stroke. The diagnoses were cerebral infarction in 79%, intracerebral hemorrhage in 5%, and transient ischemic attack in 16%.

After 3 months, 3 patients had died. One patient refused to participate; all others were seen (n=76). After 1 year, another 3 patients had died. Two patients were excluded due to recurrent stroke. One patient refused to participate; all others were seen (n=70). After 2 years, 11 more patients had died. All survivors were seen except 1 patient who had moved out of the region (n=58). At the 3-year follow-up, another 4 patients had died. Two patients had a recurrent stroke and were excluded. Two patients refused to participate,
and one partly refused and was excluded. All other survivors were seen (n=49).

Computed tomographic (CT) scan of the brain was performed within the first 2 days after admission and also after 3 years. Activities of daily living were recorded according to Katz et al. Three years after the stroke, cognitive ability was screened by the Mini-Mental State Examination. The psychiatric interviews were performed by the same psychiatrist (M.A.) without knowledge of the radiological/neurological assessments. The diagnosis of a major depression was made on the basis of the Diagnostic and Statistical Manual of Mental Disorders, edition 3 (DSM-III) symptom criteria with the physical disorder (stroke) on axis III (see “Discussion”). Patients were interviewed 4 to 5 days after admission, at 10 days, and at discharge as a control for stability of symptoms. The interviews were performed at the same time of the day (early afternoon). When appropriate, eg, in dysphasic patients, informants’ accounts and information from the staff were used to supplement patient interviews. In the acute stage, 4 patients were not assessable regarding depression in that they could not reliably answer questions with affirmative or negative answers (76 patients were assessed); at 3 months 3 patients were not assessable (73 patients assessed), at 1 year 2 patients were not assessable (68 patients assessed), and at 2 years 1 patient was not assessable (57 patients assessed).

A questionnaire on living conditions and social network was administered at the same time points. Interviews on conditions before the stroke were performed 4 to 5 days after admission to the hospital, and assessments were then done after 3 months, 1 year, 2 years, and 3 years. We have reported in detail elsewhere our findings on this group of patients and compared them with a general population of elderly people. Concerning the social network, most of the patients had children (85%), and contacts with children did not change after the stroke. Contact with other relatives, neighbors, and friends was more likely to change and was used in this study as an indicator of the social network as well as the factor “living together” with someone or not (spouse or adult child). This person was defined by all patients as someone in whom they could confide.

All CT scans were analyzed by the same neuroradiologist, who did not have access to the clinical assessments. The following structural brain measurements were made: (1) brain volume was expressed as the volume of brain substance on three consecutive slices beginning with the first slice passing through the lateral ventricles; (2) lesion volume was measured by a computerized calculation procedure, summing the lesion volumes in each slice where the lesion was visible. The lesion volume was divided by the overall brain volume, giving a relative lesion volume; (3) the minimum distance of the anterior border of the lesion to the frontal pole was determined. By dividing this distance by the overall anterior-posterior (A-P) length of the cerebral hemisphere, the relative lesion distance was constructed according to criteria described by Starkstein et al and used by others. Lesions were classified as anterior if their A-P position was less than 40%; otherwise they were classified as posterior. Cortical brain atrophy was dichotomized as evident or not. Ventricular size was measured by the anterior horn index, defined as the maximum distance between the tips of the anterior horns divided by the maximum transverse inner diameter of the skull.

During the 3-year follow-up period 3 patients were treated with antidepressants in low doses (1 patient had nortriptyline 25 mg/d, and 2 patients had clomipramine 10 to 20 mg/d). A few more patients had been treated with antidepressants for short periods (less than 3 weeks) but canceled the treatment because of side effects or poor compliance.

Informed consent was obtained from all subjects and/or their relatives, and the study was approved by the Ethics Committee of Umeå University.

Statistical analyses were performed using SYSTAT. In the case of continuous measures, comparisons between groups were made by means of Student’s t test. For categorical data, χ² was used as follows. For two-way square tables, in which data represent paired comparisons, McNemar symmetry χ² was used; otherwise Pearson χ² or, when appropriate, Fisher’s Exact Test was used. All tests were two-tailed. A value of P<.05 was considered significant. For factor analysis, the principal component analysis model in SYSTAT was used. The discriminant function analysis was performed with variables from the factor analysis including the use of dummy variables (0/1 corresponding to no/yes). Age was dichotomized as younger than or equal to the median of 74 years versus older than 74 years.

Results

Correlates of Depression at Different Time Points After Stroke

Prestroke conditions. The majority of the group lived at home (91%); the remainder (9%) lived in homes for the aged. Of the 73 patients living at home, 45 (62%) lived with someone else (spouse and/or adult child). Fifty-one percent of the patients were married, 13% were single or divorced, and 36% were widowed. By the Katz criteria, 88% of the prestroke patients were independent of others in their activities of daily living. We have previously shown that these characteristics as well as highest educational level, economic resources, social network, and prestroke psychiatric morbidity did not differ from those of the general elderly population. On the basis of retrospective assessments through interviews with the patient and informants who knew the patient well, none of the subjects fulfilled the criteria for major depression at the time of the index stroke. Six percent reported the symptom “sadness,” which is the same frequency as in the general elderly population. Thirteen patients (16%) had a personal history of psychiatric disorder; of these, 8 had been treated in a psychiatric hospital.

In-hospital evaluations. At discharge, 39% of the patients were dependent on others in their activities of daily living. The majority were discharged to their own homes (66%), 8% to homes for the aged, and 26% to a geriatric hospital. Of the 76 patients who could be assessed, 19 (25%) fulfilled the criteria for major depression (ie, at all three assessments in the acute stage). Patients who lived alone before their stroke were significantly more likely to be depressed than patients
living with another person (12/30 vs 7/46; \(P=.028\); Fisher’s Exact Test).

Ten patients (12%) had dysphasia, and another 12% had mild dysphasic problems, 8% of which were transient during the first days after the stroke. In the following analyses only the first-mentioned 10 patients were classified as dysphasic. Of those, 7 could be assessed regarding depression (they could reliably answer questions with affirmative or negative answers). The dysphasic patients were significantly more likely to be depressed (\(P=.001\); Fisher’s Exact Test). No association with other social network variables, personal history of psychiatric disorder, age, sex, or capacity in activities of daily living was found.

Recent lesions were visualized on the CT scan in 51 patients (64%). Forty-seven patients had a cerebral hemispheric lesion, 24 localized in the left hemisphere and 23 in the right hemisphere (Table 1). Three of the patients with left hemispheric lesions were not assessed regarding major depression because of severe comprehension difficulties. Major depression was significantly more frequent in patients with left hemispheric lesions than in those with right hemispheric lesions (14/21 vs 2/23; \(P<.001\); Fisher’s Exact Test). There were 27 anterior and 20 posterior lesions (Table 1). Patients with anterior left hemispheric lesions were significantly more often depressed than patients with posterior left hemispheric lesions (12/14 vs 2/7; \(P=.017\); Fisher’s Exact Test). Only 2 patients with right hemispheric lesions were depressed, and therefore no association was found in the right hemisphere. There was no association with lesion volume, subcortical versus cortical lesion, or cerebral atrophy.

Three months after stroke. Of the 73 patients assessable at 3 months, 23 were depressed (31%). Significantly more patients dependent on others in their primary activities of daily living were depressed compared with those who were independent (11/21 vs 12/52; \(P=.020\); Fisher’s Exact Test). The prior association with dysphasia remained (\(P<.001\); Fisher’s Exact Test). In regard to social network, the variable “living alone” was no longer significant, whereas “few social contacts” outside the immediate family now was associated with depression. Of the 23 depressed patients, 5 (22%) had met a friend or relative other than spouse or children the last week, as opposed to 27 (54%) of the 50 nondepressed patients (\(P=0.012\); Fisher’s Exact Test). No associations were found with age, sex, personal history of psychiatric disorder, living in a geriatric hospital or not, or stroke diagnosis or the CT scan parameters. The prior associations with hemispheric side and A-P position were not significant. However, breaking down by side of lesion showed a significantly increased depression frequency among patients with right hemispheric lesions between the acute stage and 3 months (2/23 vs 8/22; \(P=.014\); McNemar symmetry \(\chi^2\)) and no significant change for patients with left hemispheric lesions.

One year after stroke. Between 3 and 12 months (Fig 1), the prevalence of major depression decreased to 16% (23/13 vs 11/68; \(P=0.004\); McNemar symmetry \(\chi^2\)). The depressed patients had significantly fewer contacts with friends and relatives except their children (\(P=0.017\); Fisher’s Exact Test). No significant associations were found with other social network variables, age, sex, personal history of psychiatric disorder, dysphasia, stroke diagnosis, or the CT scan parameters. Capacity in activities of daily living also was not associated with depression at 1 year. It should be noted, however, that patients who were depressed in the acute stage did not recover in functional ability, whereas patients who were not depressed recovered significantly during this first poststroke year (\(P=.25\); McNemar symmetry \(\chi^2\)). Actually, all 5 patients who had changed from dependence on others to independence were not depressed in the acute poststroke period.

Two years after stroke. Approximately the same proportion of patients (11/57; 19%) were depressed, and the association with fewer social contacts remained (\(P=0.016\); Fisher’s Exact Test). No other significant associations were found.

Three years after stroke. Between 2 and 3 years after stroke, the prevalence of major depression increased to 29% (14/49), but the change did not reach statistical significance (\(P=.102\); McNemar symmetry \(\chi^2\)). However, compared with 1 year after stroke, significantly more patients were depressed (\(P=.030\); McNemar symmetry \(\chi^2\)). The depressed patients had fewer social contacts. Of the 14 depressed patients, only 1 had met a friend or relative other than their children during the last week, as opposed to 23 of the 35 nondepressed patients (7% vs 66%; \(P<.001\); Fisher’s Exact Test). For the nondepressed patients, this is not significantly different from the prestroke frequency, whereas for the depressed patients these frequencies differed significantly (at 3 years 7% had met a friend or relative other than their children at least once a week compared with 64% prestroke; \(P=.005\); McNemar symmetry \(\chi^2\)). No associations were found with other social network vari-
variables, capacity in activities of daily living, age, sex, personal history of psychiatric disorder, dysphasia, stroke diagnosis, or the initial lesion characteristics: volume, hemispheric side, or A-P position. The depressed patients tended to have more cognitive impairment as measured by Mini-Mental State Examination, but the difference was not significant (mean±SD, 24.3±5.5 vs 21.5±8.0; P=.255; two-tailed t test). Analysis of the CT scan performed at 3 years (n=47; in 2 patients CT scan was not obtained) showed no associations between major depression and the characteristics of the index lesion, whereas cortical atrophy at 3 years significantly differed between depressed and nondepressed patients (11/12 vs 16/35; P=.007; Fisher’s Exact Test). Also, subcortical atrophy showed a significant difference between the depressed and nondepressed group (mean±SD, 30.3±4.0 vs 25.5±4.8; P=.004; two-tailed t test).

During the 3 years since the stroke, 21 patients in our cohort (n=80) had died. Mortality was not associated with depression at any of the follow-ups. Patients who died were older (mean±SD, 78±8 vs 72±11 years; P=.003; two-tailed t test), were more likely to be disoriented in-hospital, were more dependent on others in activities of daily living before the stroke, and more often had cortical atrophy on CT scan (data not shown). To control for the possibility that mortality could explain the results concerning changes in associated factors, a subsequent analysis was limited to the group of patients who had survived up to 3 years. The results did not change; the same factors as in the cross-sectional analyses were associated with depression at the different time points.

Predictors of Depression: Multivariate Analysis

In an attempt to study the relative importance of factors associated with depression, discriminant function analysis was undertaken (both independent and dependent variables were discrete). The variables that were associated with depression in the univariate analysis in the acute stage (Table 2) together with age and sex were used as predictor variables. Because the predictors showed intercorrelations, data were first transferred to orthogonal components by a factor analysis. These factors were then entered in the discriminant function analysis with major depression as the grouping variable. As shown in Table 3, left-sided hemispheric lesion was the most important predictor of immediate depression. To take into account relations between variables that could be overlooked, another discriminant function analysis was undertaken, which also included relative lesion volume, cortical atrophy, social contacts, capacity in activities of daily living, and personal history of psychiatric disorder as predictor variables. The result was the same; the most important predictors of immediate depression after stroke were left-sided hemispheric lesion, anterior lesion, dysphasia, and living alone. All other variables had very low canonical loadings (data not shown). The original model (Table 3) gave an 82% total correct classification rate (36/44). Sixteen patients who had a CT-verified hemispheric lesion were depressed; of these, 13 were correctly classified (81%).

The same model applied at the later follow-ups gave insignificant discriminant functions, indicating that depression at these time points could have other predictors. To get a significant model at 3 months, it had to include impairment in activities of daily living, which had the highest loading (0.51). At 1, 2, and 3 years, “few social contacts” had the highest canonical loading. At 3 years, cerebral atrophy was also an important predictor of depression (results were the same for cortical atrophy and anterior horn index). Results are shown in Table 4. The four predictors correctly grouped 11 of the 12 depressed patients (92%); total correct classification rate was 77% (36/47).

The results from the multivariate analyses were in agreement with the univariate correlates of depression presented in Table 2.

The Longitudinal Course of Depression

Of the 27 patients who developed depression soon after their stroke (0 to 3 months), 25 were reassessed at 1 year. Of these patients, 15 (60%) had recovered. At 3 years, 14 of these early depressed patients were alive and assessed. Restricting the analysis to these survivors, 8 of 14 (57%) had recovered at 1 year. At 3 years, only 1 more patient had recovered. Hence, of the depressed patients 36% (5/14) had such a long-lasting depression that they did not recover during the 3 years. Two patients relapsed by 3 years. Thus, half (7/14) of the

<table>
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<td>Sex</td>
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<tr>
<td>Side of lesion</td>
<td>0.66</td>
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<tr>
<td>Anterior/posterior position</td>
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<td>Dysphasia</td>
<td>0.27</td>
</tr>
<tr>
<td>Living with someone</td>
<td>0.26</td>
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Λ =.51; P < .001; canonical correlation =.70.
In a recent community-based study of mood disorders after stroke, House et al\(^1\) found major depression in 11% of the patients 1 month after stroke compared with 25% in our study. The 95% confidence intervals (4% to 18% vs 15% to 35%) are overlapping. Social characteristics, mean age, and independence in activities of daily living did not differ between the studies. In another community-based study, 22% of the patients were “definitely depressed” at 3 weeks after stroke.\(^9\) Robinson et al\(^1\) reported a prevalence of major depression of 27% within 2 weeks after the stroke, although patients were younger (mean age, 59 years) and mainly from lower socioeconomic groups.

At 1 year, we found a significant decrease in prevalence of depression (from 31% at 3 months to 16%), which differs from Robinson et al\(^26\) who in a subsample of their original group found a stable prevalence of major depression up to 2 years. Our results conform to those of House et al\(^1\) and Morris and Robinson,\(^32\) who reported approximately halved prevalence of major depression at 12 and 17 months after stroke, respectively. Our study is the only prospective longitudinal study extended to 3 years after stroke. At this later stage after stroke, there was an increased prevalence of major depression, so that after 3 years the prevalence was as high as in the acute stage (29%).

Because we have a very low rate of missing subjects, our longitudinal data allow individual patients to be followed through the 3 years. At 1 year, 60% of the patients with early depression after stroke (0 to 3 months) had recovered. House et al\(^1\) reported a better prognosis; depression persisted throughout the year in only 2 of the 10 patients with early major depression (80% 1-year outcome). In the follow-up by Robinson et al\(^26\) 80% of patients with major depression had recovered after 1 year. The corresponding proportion in a study by Morris and Robinson\(^32\) was 71% and by Wade et al\(^5\) 50%. Robinson et al\(^1\) reported all patients with major depression in-hospital to be recovered by 2 years, but their results are uncertain because of the high attrition rate. In our study, patients who recovered did so within 1 year, and only 1 more patient recovered between 1 and 3 years. We therefore conclude that if the patient with early depression has not recovered by 1 year, there is a high risk of chronic depression.

In the acute poststroke period, the single most important determinant of major depression was the location of lesion in the left anterior hemisphere, thus confirming results by Robinson et al.\(^33\) In our study, when the lesion was located in the left anterior hemisphere, the rate of major depression was 3 times higher than with a left posterior lesion and as much as 10 times higher than with a lesion in the right hemisphere (Fig 2). It should be noted that our results showed a change over time not demonstrated previously. From 3 months, these intrahemispheric and interhemispheric differences were no longer significant. An interesting finding was that depression occurred more often in the right than in the left hemisphere group after 3 years. However, the difference was not significant. This time dependency explains many of the contradictory results from other studies.\(^6,10,34,35\) Other lesion characteristics such as volume and subcortical or cortical location were not associated with depression, a result in accord with other reports.\(^21,33\)

### Discussion

This study confirms the high prevalence of depression in the acute stage after stroke. Our prospective data also provide information on changes over time in prevalence and correlates of depression, which has not been reported in previous studies. Before further discussion, some methodological issues should be pointed out.

The method for patient selection ascertains a representative sample of patients admitted to the hospital for stroke.\(^1\) The problem of spurious associations in studies of hospital inpatients\(^10\) may be less severe in the Scandinavian countries than in other medical settings because more than 90% of stroke patients (excluding transient ischemic attacks) are admitted to the hospital.\(^25\) We have avoided sampling bias by studying a consecutive series of patients during 1 year. This population-based cohort was almost completely followed up at the different time intervals up to 3 years. This represents an improvement over the only other extended longitudinal study of depression after stroke.\(^26\) Thus, our study group should permit the results to be generalized to other stroke patients.

The special problems in connection with diagnosis of depressive disorder in this patient group have been discussed by House\(^3\) and Robinson,\(^27\) among others. In our study, a careful clinical interview performed by the same psychiatrist at all seven time points was considered the best tool for assessing, with minimal exclusions, patients with dysphasia, denial of affective symptoms, intellectual impairment, pathological emotionalism,\(^1\) and general fragility. As discussed in “Subjects and Methods,” we exclusively applied the DSM-III criteria of major depression. We did not use the category of dysthymia because of its 2-year time criterion, nor the modified construction of “minor depression.” Price\(^28\) has pointed out that irrespective of an eventual etiological organic factor, patients can be described as fulfilling the symptom criteria for major depression. In accordance with others,\(^1,12,27\) all psychiatric symptoms were evaluated without assumptions of their cause, so as not to prejudice this issue. For that reason, we did not apply the DSM-III categories of “organic mood disorder” or “adjustment disorder with depression”; these syndromic clusters are also too vaguely defined for research purposes. There is no evidence that depression following organic illness is phenomenologically different from “functional depression,” another reason for keeping strictly to the criteria of major depression.\(^29,30\)

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<th>Predictor variable</th>
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<td>Age</td>
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<tr>
<td>Sex</td>
<td>0.21</td>
</tr>
<tr>
<td>Social contacts*</td>
<td>0.73</td>
</tr>
<tr>
<td>Cortical atrophy†</td>
<td>0.44</td>
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\(\Delta=0.71; \ P<.01; \) canonical correlation=0.54.

*Contact with friends or relatives other than spouse and/or children.
†Computed tomographic scan performed 3 years after the index stroke.
Multivariate statistics showed that dysphasia was an independent predictor of depression. Dysphasia is a frustrating condition with considerable psychological and psychosocial consequences which, according to our results, contribute to depression. One previous study has reported a high depression rate in patients with dysphasia. Often dysphasic patients have been excluded from studies, and this association has accordingly been concealed.

Together with lesion location and dysphasia, the social support parameter “living alone” was a determinant of immediate depression. Hence, under the stressful condition of an acute stroke, being without the social support of a family seems to promote the development of depression. We therefore recognize contributions from both biological and psychosocial factors interacting in the development of depression in the immediate poststroke period.

Dependence in activities of daily living was not associated with immediate depression, but at 3 months this factor was the most important predictor of depression. Our results indicate that functional impairment does not determine the onset of depression but interacts with depression, resulting in a poorer long-term functional recovery. These results confirm a few previous reports on more selected samples.

We have previously shown that the social network before the stroke was as good as for the general elderly population. After stroke, contacts outside the immediate family declined and remained lower than in the general elderly population. In this study, we have shown that after the stroke and throughout the following 3 years, the depressed patients had fewer social contacts than the nondepressed patients who actually had an intact social network. At 1 year and later, “few social contacts” was the most important predictor of depression. Reduced social contacts can be a cause as well as a result of depression; dependent and independent variables in this system of continuous interaction can hardly be isolated. More important is the clinical implication: this vicious circle has to be broken by early and active treatment at all levels, including drug therapy or electroconvulsive treatment, psychotherapy and family work, support and education, and psychosocial interventions. Otherwise, there is a risk that these elderly patients, when recovered, cannot reestablish social ties.

There is evidence that degenerative changes in the brain are associated with depression in old age. In one investigation of stroke patients, subcortical atrophy was associated with depression. We have shown that early after stroke, depression had other determinants. But at 3 years after stroke, cortical as well as subcortical atrophy contributed to depression. It should be observed that the total group of depressed patients at 3 years consisted partly of patients with depression occurring late after stroke (1 to 3 years) and partly of early depressed patients with a chronic course. Cerebral atrophy was equally frequent in both subgroups (data not shown), suggesting influence on both maintenance and onset of depression late after stroke. This linkage between depression after stroke and cerebral atrophy raises important questions about common underlying neurobiological changes.

A greater understanding of these multifactorial interactions from biological, psychological, and social levels contributing to depression after stroke would enable targeted preventive strategies and more active and comprehensive treatment programs.

**Acknowledgments**

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