Effective Treatment of Poststroke Depression With the Selective Serotonin Reuptake Inhibitor Citalopram

Grethe Andersen, MD; Karsten Vestergaard, MD; Lise Lauritzen, MD

Background and Purpose The aim of the study was to investigate the efficacy and safety of the selective serotonin reuptake inhibitor citalopram in treating poststroke depression, since available treatments are usually poorly tolerated.

Methods A 6-week double-blind, placebo-controlled trial was undertaken. Diagnosis and outcome were determined using the Hamilton Depression Scale, and unwanted effects were measured using the UKU side effect rating scale. Sixty-six consecutive depressed patients from an unselected population of 285 stroke patients aged 25 to 80 years entered the trial 2 to 52 weeks after stroke. They were assigned to equally sized treatment and placebo groups. The initial level of depression was comparable in the two groups (mean baseline Hamilton Depression scores, 19.4 and 18.9, respectively). Demographic parameters were also comparable in the two groups.

Results Significantly greater improvement was seen in patients treated with citalopram (10 to 40 mg/d) for 3 and 6 weeks, both when including all patients (intention-to-treat analysis, \(P<.05\)) and excluding patients who dropped out during the first 3 weeks (efficacy analysis, \(P<.005\)). Half of the 28 patients who entered the trial 2 to 6 weeks after stroke recovered within 1 month, independent of the treatment given. This indicates a high degree of spontaneous recovery in the early phase after stroke. In contrast, recovery was infrequent in placebo group patients who became depressed 7 weeks or more after stroke. No serious side effects related to the treatment were detected; those present were mild and usually transient.

Conclusions This trial demonstrates that the selective serotonin reuptake inhibitor citalopram offers an advantageous new treatment of poststroke depression that is both safe and effective. (Stroke. 1994;25:1099-1104.)

Key Words • antidepressive agents • cerebrovascular disorders • citalopram • depression

Numerous studies undertaken during approximately the last decade indicate that the incidence of poststroke depression (PSD) is 20% to 50% in the first year. However, differences in patient samples and rating scales render meaningful comparison of these studies impossible. Furthermore, it has usually been overlooked that the frequency of PSD may change with time after stroke, as may its natural history. While there is disagreement on whether PSD is predominantly organic or reactive in nature, many investigators agree on the need for clinical trials of treatments that may improve the quality of life of patients with PSD and thereby facilitate their rehabilitation.

In 1984 Lipsey et al reported significant improvement in PSD patients treated with nortriptyline. The frequency of adverse drug effects was unacceptably high, however, and this led to a warning to be extremely cautious when prescribing tricyclic antidepressants for PSD because stroke patients are particularly vulnerable to their adverse effects.

The subsequent development of second-generation antidepressants such as selective serotonin reuptake inhibitors provides an alternative means of treating PSD. These drugs have the advantage of being generally nonsedative and devoid of cardiotoxic, anticholinergic, and antihistaminergic effects. A further indication for using this approach is the recent finding that more effective treatment of PSD is obtained with the combination imipramine/mianserin than with desipramine/mianserin, with the greater efficacy of the former probably attributable to the serotonergic component of imipramine treatment. That the treatment of PSD with serotonin reuptake inhibitors is in fact effective is demonstrated by the present double-blind, placebo-controlled trial with citalopram in an unselected consecutive cohort of acute stroke patients with PSD diagnosed during the first year after stroke.

Subjects and Methods

The patient population comprised all 320 consecutive patients with acute stroke admitted to either Farso or Aalborg Hospitals or referred to the Aalborg Hospital outpatient clinic (which together serve a population of approximately 250,000) during the period February 1, 1991, through February 29, 1992 (13 months). The patients were examined within 7 days of the onset of stroke and again on follow-up at 1 month, 6 months, and 1 year after stroke. The examinations undertaken included neurological examination, test for aphasia, Barthel Index, a Danish version of Mattis Dementia Rating, Mini-Mental State Examination, Social Activities index, and psychiatric assessment with the Hamilton Depression Scale (HDS) (17 items) and the Melancholia Scale (MES). A cerebral computed tomographic scan was undertaken from days 5 through 10 and, in cases of PSD, again after a period of more than 1 month.
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Patients with subarachnoid hemorrhage orBinswanger’s disease were excluded from the study, as were patients with previous degenerative or expansive neurological diseases (such as multiple sclerosis, amyotrophic lateral sclerosis, tumors, and hydrocephalus). This reduced the patient population to 285 patients aged 25 to 80 years. Patients with a history of psychiatric illness (except depression more than 1 year earlier) were excluded from the study, as were patients with decreased consciousness, dementia, or aphasia to such a degree that they could not explain themselves or gave conflicting verbal and nonverbal signals.

The patients were visited by a study nurse 2 to 3 months after stroke (either at home or in the hospital) for the purpose of undertaking a depression self-rating using the Beck Depression Self-Rating Scale.29 Patients with suspected PSD, identified on the basis of the nurse’s report and the patients’ self-rating, were called to the outpatient clinic and assessed using the HDS. Patients who had had symptoms for at least 2 weeks and a baseline HDS score of 13 or greater were considered to have PSD in accordance with the Diagnostic and Statistical Manual of Mental Disorders, edition 3, revised (DSM-III-R).30 Categories of less than major depression (HDS, 13 to 17), major depression (HDS, 18 to 29), and more than major depression (HDS, 30 or more) Further evidence in support of using a baseline HDS score of 13 or greater as the cutoff score for participation in the trial was provided by Paykel,31 who recently showed that amitriptyline is superior to placebo in patients treated by their family doctor if the baseline HDS score is 13 or more. Patients with PSD were also identified at the 1-, 6-, and 12-month follow-up examinations, with entry into the trial possible at any time during the first year after stroke (ie, after a follow-up examination or a home visit by a study nurse), provided that assessment of HDS at the outpatient clinic confirmed PSD. Recruitment ceased on September 1, 1992, at which time 66 PSD patients had entered the trial.

The trial was designed as a randomized, double-blind, placebo-controlled study. To ensure approximately equal numbers of patients in the treatment groups, randomization was carried out in groups of 4, with 2 assigned to the citalopram group and 2 to the placebo group. The citalopram (Cipramil, Lundbeck Pharma) dose was that usually recommended, ie, 20 mg/d before bedtime in patients younger than 66 years and 10 mg/d in older patients.19 If the patient did not respond to the treatment within 3 weeks, the dose was doubled. The outcome of treatment was measured after 6 weeks20 and the patients divided into responders, in whom HDS scores fell below 13, and nonresponders, in whom HDS scores remained above 13. The former continued double-blind treatment for an additional 10 weeks; the latter were instead offered nortriptyline or mianserin (in some cases a combination of the two). Electrocardiograms (ECGs) were taken before entry and during the study. After termination of the trial all ECGs considered abnormal, ie, not fulfilling standard criteria for normality, were assessed blindly by an experienced cardiologist.

Assessment of PSD using the HDS and MES was undertaken at baseline and after 1, 3, 6, 9, 12, and 16 weeks of placebo or citalopram treatment. Unwanted drug effects were registered and evaluated at the same intervals using the UKU side effect scale.32 The Newcastle Depression Scale33 was used at baseline to determine whether the patients had endogenous or nonendogenous depression.

Table 2.

## Table 1. Demographic Characteristics of the 66 Patients Entering the Trial

<table>
<thead>
<tr>
<th>Citalopram (n=33)</th>
<th>Placebo (n=33)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y (mean±SD)</td>
<td>68.2±4.2</td>
<td>65.8±9.0</td>
</tr>
<tr>
<td>Sex (female)</td>
<td>21</td>
<td>19</td>
</tr>
<tr>
<td>Disposing illness</td>
<td>19</td>
<td>18</td>
</tr>
<tr>
<td>History of stroke</td>
<td>15</td>
<td>11</td>
</tr>
<tr>
<td>History of transient ischemic attack</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Time since stroke, wk (mean±SD)</td>
<td>10.6±9.8</td>
<td>13.2±11.0</td>
</tr>
</tbody>
</table>

Lesion on CT†
- Left-sided: 8 9
- Right-sided: 9 13 NS
- Bilateral: 4 4
- No lesion: 11 7

Barthel Index at 1 month (mean±SD): 87.2±24.2 82.2±29.5 NS

Newcastle Depression Index (endogenous): 7 11 NS

*χ² test or Mann-Whitney U test.
†Disposing illness defined as previous hypertension (diastolic pressure >95 mm Hg), diabetes, and heart disease (defined as signs of arrhythmia, cardiac decompensation, recent or former myocardial infarction, or valve disease).
‡One patient in the citalopram group died before a computed tomographic (CT) scan was performed.

Patients with subarachnoid hemorrhage orBinswanger’s disease were excluded from the study, as were patients with previous degenerative or expansive neurological diseases (such as multiple sclerosis, amyotrophic lateral sclerosis, tumors, and hydrocephalus). This reduced the patient population to 285 patients aged 25 to 80 years. Patients with a history of psychiatric illness (except depression more than 1 year earlier) were excluded from the trial, as were patients with decreased consciousness, dementia, or aphasia to such a degree that they could not explain themselves or gave conflicting verbal and nonverbal signals.

The patients were visited by a study nurse 2 to 3 months after stroke (either at home or in the hospital) for the purpose of undertaking a depression self-rating using the Beck Depression Inventory (42 items).29 Patients with suspected PSD, identified on the basis of the nurse’s report and the patients’ self-rating, were called to the outpatient clinic and assessed using the HDS. Patients who had had symptoms for at least 2 weeks and a baseline HDS score of 13 or greater were considered to have PSD in accordance with the Diagnostic and Statistical Manual of Mental Disorders, edition 3, revised (DSM-III-R).30 Categories of less than major depression (HDS, 13 to 17), major depression (HDS, 18 to 29), and more than major depression (HDS, 30 or more). Further evidence in support of using a baseline HDS score of 13 or greater as the cutoff score for participation in the trial was provided by Paykel,31 who recently showed that amitriptyline is superior to placebo in patients treated by their family doctor if the baseline HDS score is 13 or more. Patients with PSD were also identified at the 1-, 6-, and 12-month follow-up examinations, with entry into the trial possible at any time during the first year after stroke (ie, after a follow-up examination or a home visit by a study nurse), provided that assessment of HDS at the outpatient clinic confirmed PSD. Recruitment ceased on September 1, 1992, at which time 66 PSD patients had entered the trial.

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Assessment of PSD using the HDS and MES was undertaken at baseline and after 1, 3, 6, 9, 12, and 16 weeks of placebo or citalopram treatment. Unwanted drug effects were registered and evaluated at the same intervals using the UKU side effect scale.32 The Newcastle Depression Scale was used at baseline to determine whether the patients had endogenous or nonendogenous depression.

Initial psychiatric rating was undertaken by two authors (G.A. and K.V.) trained in the use of HDS, MES, UKU, and the Newcastle Scale (and members of the Danish University Antidepressant Group [1990]29) under the supervision of the third author (L.L.), an experienced psychiatrist, using video recordings of the patients. After patients entered the trial, they were rated by G.A. alone.

The study protocol was approved by the local ethics committee and the Danish Health Authority, and informed consent was obtained from each patient before entry into the study. After September 1, 1991, written consent was obtained.

### Table 2. Intention-to-Treat Analysis of Citalopram Treatment of Poststroke Depression

<table>
<thead>
<tr>
<th>Weeks of Treatment</th>
<th>Citalopram (n=33)</th>
<th>Placebo (n=33)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>19.4±3.1</td>
<td>17.7±3.1</td>
<td>18.9±2.8</td>
</tr>
<tr>
<td>1</td>
<td>15.9±3.2</td>
<td>14.8±3.2</td>
<td>15.8±3.0</td>
</tr>
<tr>
<td>3</td>
<td>12.5±4.2*</td>
<td>11.0±4.1†</td>
<td>15.0±3.7</td>
</tr>
<tr>
<td>6</td>
<td>11.4±5.1*</td>
<td>10.5±5.1</td>
<td>14.1±4.7</td>
</tr>
<tr>
<td>Decrease from week 0 to week 6</td>
<td>8.0±6.0*</td>
<td>7.2±5.8*</td>
<td>4.8±4.6</td>
</tr>
</tbody>
</table>

HDS indicates Hamilton Depression Scale; MES, Melancholia Scale. Values are mean±SD. Accumulated dropouts at 1 week: citalopram 3 and placebo 1; 3 weeks: citalopram 6 and placebo 1; 6 weeks: citalopram 7 and placebo 2.

*P<.05, †P<.01, Mann-Whitney U test.
from the patients in accordance with new instructions from the Danish Health Authority.

The data were analyzed using HDS and MES week-to-week total scores, applying both the intention-to-treat approach (ie, including all patients who entered the trial, the last observation was extended in the case of patients who did not complete the planned treatment period of 6 weeks) and efficacy analysis (ie, excluding patients who dropped out during the first 3 weeks of treatment). The latter was also conducted using a reduction of 50% or more from the baseline score as the outcome criterion. A reduction of this magnitude indicates partial or full remission and is a clinically meaningful measure.29 Two-tailed nonparametric tests were used, with significance set at 5%.

Results
The study population comprised 285 patients. Of those who survived 1 year after stroke, 95% were seen at all three follow-up examinations. Eighty-five patients were diagnosed as having PSD, with an HDS score of 13 or greater on at least one examination. Participation in the trial was offered to 81 patients; the other 4 were included in the placebo group. Thirty-three patients were randomized to each of the citalopram and placebo groups. Of these 81 patients, 4 had already started antidepressant treatment, and 11 refused entry on the grounds that they preferred to wait for possible spontaneous recovery. The remaining 66 patients were included in the trial. Mean interval since stroke was 11.9 weeks (SD 10.4), and 17 (26%) of the patients were still hospitalized at the time of inclusion. Thirty-three patients were randomly allocated to each of the citalopram and placebo groups.

There were no major differences in demographic data and computed tomographic findings when comparing trial participants with stroke patients with PSD who did not enter the trial (data not shown) or when comparing the two treatment groups (Table 1). The mean baseline HDS score of the 66 patients entering the trial was 19.1±2.9, significantly greater than that of the 15 PSD patients who did not participate (15.3±2.0, P<.001, Mann-Whitney U test) and that of the nondepressed patients (5.1±3.4, P<.001, Mann-Whitney U test). Finally, there were no differences between the citalopram and placebo groups with respect to the concomitant use of other medications (including hypnotics and anxiolytic agents) during the first 6 weeks of the trial.

Seven patients dropped out during the first 3 weeks of treatment (early dropouts): 6 from the citalopram group (1 died on day 3 of a ruptured aortic aneurysm, 1 suffered a new major stroke on day 20, 1 developed a rash on day 14, 1 violated the protocol, 1 suffered dizziness on day 1, and 1 complained of headache on day 6) and 1 from the placebo group (on day 6 because of dizziness). In addition, there were 2 late dropouts: 1 patient dropped out of the citalopram group on day 26 because of general uneasiness, and 1 patient in the placebo group died of cardiac failure on day 28.

The results of the intention-to-treat analysis, in which all patients including dropouts are included, with the last observation for the dropouts being carried forward, are given in Table 2. The outcome was better with citalopram treatment than with placebo as determined by both HDS and MES after 3 weeks and by HDS after 6 weeks (P<.05, Mann-Whitney U test). Furthermore, the decrease in HDS and MES scores (baseline minus end point) was significantly greater in the citalopram group than in the placebo group (P<.05, Mann-Whitney U test).

TABLE 4. Intention-to-Treat Analysis of Hamilton Depression Scale Score in Patients With Poststroke Depression Treated With Citalopram Starting <7 or ≥7 Weeks After Stroke

<table>
<thead>
<tr>
<th>Weeks of Treatment</th>
<th>&lt;7 Weeks (n=28)</th>
<th>≥7 Weeks (n=38)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Citalopram (n=15)</td>
<td>Placebo (n=13)</td>
</tr>
<tr>
<td>0</td>
<td>20.1±3.2</td>
<td>18.9±3.4</td>
</tr>
<tr>
<td>1</td>
<td>16.7±3.6</td>
<td>14.8±3.8</td>
</tr>
<tr>
<td>3</td>
<td>12.5±4.6</td>
<td>13.0±3.7</td>
</tr>
<tr>
<td>6</td>
<td>12.7±6.1</td>
<td>11.5±5.0</td>
</tr>
<tr>
<td>Decrease from week 0 to week 6</td>
<td>7.3±7.4</td>
<td>7.4±4.8</td>
</tr>
</tbody>
</table>

Values are mean±SD Hamilton Depression Scale score. *P<.005, Mann-Whitney U test.
The results of the efficacy analysis, in which the early dropouts are excluded, are given in Table 3. As determined by HDS and MES, outcome was better with citalopram than with placebo at both 3 and 6 weeks, and the decrease in scores during the 6-week period was almost twice that in the placebo group (P<.005, Mann-Whitney U test). When the data were analyzed using a 50% reduction from baseline scores as the outcome criterion, efficacy was significantly greater with citalopram (P<.05, Mann-Whitney U test). In contrast, outcome in the 38 patients who entered the trial 7 weeks or later after stroke was significantly better after 3 and 6 weeks of citalopram treatment (P<.005, Mann-Whitney U test; Table 4). Furthermore, while only 3 of 20 patients (15%) responded to placebo, 12 of 18 patients (67%) responded to citalopram.

The Figure shows the HDS score for all PSD patients during the 6-week trial (efficacy data) as well as for responders (HDS score <13) during the subsequent 14 weeks. The decrease in HDS was significantly greater in the citalopram group at 16 weeks, at which time the mean end point score was 6.2±2.3 in the citalopram group compared with 8.7±2.2 in the placebo group (P<.05, Mann-Whitney U test).

Computed tomographic scans revealed that of the patients in the citalopram group, 9 (27%) had right-sided lesions and 8 (24%) left-sided lesions; however, there was no overall difference in treatment response in these two rather small subgroups (Table 5).

Analysis of side effects judged by the interviewer to be related to treatment revealed a significantly greater incidence of nausea and vomiting during the first week of citalopram treatment (P<.05, Mann-Whitney U test). Correcting the severity of side effects for baseline levels revealed a transient increase in asthenia, lassitude, or fatigability during the first week of citalopram treatment (P<.05, Mann-Whitney U test). Although adverse events occurred during the 16-week treatment period, none could be related to citalopram treatment per se (Table 6). Diagnosis of abnormal ECGs showed that during the trial there were fewer recordings with left ventricular hypertrophy and a prolonged QT interval in the citalopram group but more in the placebo group. All other aspects of the ECGs remained unchanged.

**Discussion**

The present study shows that the selective serotonin reuptake inhibitor citalopram is an effective and well-tolerated treatment for PSD, especially in patients who become depressed 7 weeks or more after stroke (in which case spontaneous recovery is less likely).

A main objective of the study was to treat an unselected group of PSD patients to mimic as closely as possible the clinical situation of advising patients whether or not to undergo antidepressant therapy. Except for the exclusion of those patients unable to communicate reliably, the PSD patients entering the study were all those treated in a consecutive group of PSD patients with poststroke depression treated with citalopram or placebo. Treatment was given to all patients for the first 6 weeks but thereafter only to responders (HDS score <13 at 6 weeks).

The results of the efficacy analysis, in which the early dropouts are excluded, are given in Table 3. As determined by HDS and MES, outcome was better with citalopram than with placebo at both 3 and 6 weeks, and the decrease in scores during the 6-week period was almost twice that in the placebo group (P<.005, Mann-Whitney U test). When the data were analyzed using a 50% reduction from baseline scores as the outcome criterion, efficacy was significantly greater with citalopram (P<.05, Mann-Whitney U test).

**Table 5. Intention-to-Treat Analysis of Hamilton Depression Scale Score in Poststroke Depression Treated With Citalopram in Patients With Left-Sided or Right-Sided Lesions**

<table>
<thead>
<tr>
<th>Localization of Lesion</th>
<th>Right Hemisphere (n=9)</th>
<th>Left Hemisphere (n=8)</th>
<th>Intergroup Comparison*</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>18.6±3.8</td>
<td>21.0±3.7</td>
<td>NS</td>
</tr>
<tr>
<td>1</td>
<td>16.2±3.2</td>
<td>14.8±3.8</td>
<td>NS</td>
</tr>
<tr>
<td>3</td>
<td>11.8±3.8</td>
<td>10.0±3.6</td>
<td>NS</td>
</tr>
<tr>
<td>6</td>
<td>11.6±4.7</td>
<td>10.9±4.9</td>
<td>NS</td>
</tr>
<tr>
<td>Decrease from week 0 to week 6</td>
<td>7.0±7.1</td>
<td>10.1±3.9</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean±SD Hamilton Depression Scale score.

*Mann-Whitney U test.
as a diagnostic tool early after stroke, thereby rendering it unreliable in studies on the frequency of PSD in the acute phase of stroke. In the present study 11 of the patients diagnosed as having PSD earlier than 6 weeks after stroke refused treatment, preferring to wait for possible spontaneous recovery; this in fact occurred in most cases, but it should be remembered that their HDS scores were lower than those of the patients participating in the trial. Furthermore, half of the 28 patients who entered the trial at 6 weeks or earlier after stroke recovered within the next month whether receiving citalopram or placebo. Whether PSD is caused by the lesion per se (ie, because of direct depletion of biogenic amine neurotransmission) or is a psychological reaction to disability, one would expect a high rate of spontaneous recovery during the first 1 or 2 months after stroke, just as is the case with recovery from paresis and acute crisis reactions, respectively. However, that only 3 (15%) of the 20 patients who became depressed 7 weeks or later after stroke responded to placebo treatment indicates that spontaneous recovery in this group of PSD patients is very rare. In fact, it has previously been reported that these depressions usually last 6 months or more if left untreated.

In conclusion, we recommend that stroke patients be screened for depression after the acute phase of stroke using the HDS interview and that treatment with a selective serotonin reuptake inhibitor such as citalopram be offered if the HDS score is 13 or more. We find that citalopram treatment is effective within 3 to 6 weeks in approximately 65% of patients who become depressed more than 7 weeks after stroke (compared with recovery in the placebo group of only approximately 15%). Furthermore, the treatment seems to be safe, with side effects that are few, mild, and transient.

Acknowledgments
This study was supported by the Lundbeck Foundation (grants 109/90, 170/91, and 205/92), the Medical Research Foundation for North Jutland County, the Aalborg Diocese Research Foundation, Consultant Otorhinolaryngologist Kopp's Foundation, and Martinus Sørensen's Foundation, Aalborg. The authors wish to thank Ove Aas- koven for undertaking the statistical analyses, Dr Per Bech (Department of Psychiatry, Hillerød General Hospital, Denmark) and Dr Ib Magnussen (Department of Neurology, Aalborg Hospital, Denmark) for valuable discussions during the study and revising the manuscript. Lundbeck Pharma A/S kindly provided the citalopram and placebo tablets.

References

### Table 6. Adverse Events During the 16-Week Double-blind, Placebo-Controlled Trial of Citalopram Treatment of Poststroke Depression

<table>
<thead>
<tr>
<th>Event</th>
<th>Citalopram</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial fibrillation</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Cardiac failure (deaths)</td>
<td>0 (0)</td>
<td>3 (2)</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>New stroke</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thromboembolic</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Hemorrhagic</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Transient ischemic attack</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Epileptic seizures</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Rash</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Deaths (other causes)</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>

The effectiveness of citalopram in treating PSD seems to be as good as in primary depression, independent of whether the stroke lesion is left-sided or right-sided. This indicates that the standard diagnostic criteria used for depression in psychiatric patients, which have been used without modification in the present study, also apply to stroke patients, with the exception of patients becoming depressed shortly after stroke (<7 weeks). The HDS score thus seems to be a valid measure of depression in stroke patients, and the cut-off score applied (≥13) seems to be reasonable for PSD. Another advantage of using the HDS interview is that it is well accepted by stroke patients, and the cut-off score applied (>13) has been reported to be as good as in primary depression, independent of the regimen used.

The occurrence of somatic symptoms attributable to factors other than depression (eg, hospitalization, immobilization) probably invalidates the use of the HDS...


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