Ipsilateral Pushing in Stroke: Incidence, Relation to Neuropsychological Symptoms, and Impact on Rehabilitation. The Copenhagen Stroke Study

Palle M. Pedersen, MA, Anette Wandel, PT, Henrik S. Jørgensen, MD, Hans O. Raaschou, MD, Tom S. Olsen, MD, PhD


Objectives: A “pusher syndrome” encompassing postural imbalance and hemineglect is believed to aggravate the prognosis of stroke patients. Our aim was to determine the incidence, associated neuropsychological symptoms, and the consequences for rehabilitation of ipsilateral pushing.

Design: Consecutive and community-based.

Setting: A stroke unit receiving all acute stroke patients from a well-defined catchment area. All stages of rehabilitation were completed within the unit.

Patients: 647 acute stroke patients admitted during a 1-year period. Excluded were 320 patients who did not receive physiotherapy because they did not have parases of the leg, had a fast remission, or died.

Main Outcome Measures: Gain in activities of daily living (ADL) function (Barthel Index), time course of functional remission, and discharge rate to nursing home. The independent impact of ipsilateral pushing was analyzed with multiple linear and logistic regression analyses.

Results: Ipsilateral pushing was found in 10% of the included patients. No significant differences were found in the incidence of hemineglect and anosognosia between patients with and without ipsilateral pushing. No association with side of stroke lesion was found. Ipsilateral pushing had no independent influence on gain in ADL function or discharge rate to nursing home, but patients with ipsilateral pushing used 3.6 weeks (p < .0001) more to reach the same final outcome level than did patients without ipsilateral pushing.

Conclusions: The existence of a “pusher syndrome” was not confirmed. Ipsilateral pushing did not affect functional outcome, but slowed the process of recovery considerably.

© 1996 by the American Congress of Rehabilitation Medicine and the American Academy of Physical Medicine and Rehabilitation

SOME HEMISPHERIC stroke patients display a peculiar behavior pattern: a tendency to push away from the unaffected side of the body, which impairs their postural balance. This presents difficulties for retraining because of the imbalance of the body. It has been suggested that there is a “pusher syndrome,” which includes both the physical symptom of pushing away from the unaffected side of the body and neuropsychological right hemisphere symptoms, prominently hemineglect.1 No systematic study has been conducted of any aspect of this proposed syndrome, but despite the lack of a scientific basis, this term is often used in the rehabilitation of stroke patients. The aim of this study was to investigate (1) the incidence of ipsilateral pushing, (2) whether ipsilateral pushing is part of a pusher syndrome, and (3) whether ipsilateral pushing has an adverse impact on functional recovery or the time course of recovery. The behavior of ipsilateral pushing, if connected to right hemisphere neuropsychological symptoms, should emerge from mainly right hemisphere lesions, especially from the right parietal lobe.

METHODS

Patients. This study is part of the Copenhagen Stroke Study described in detail elsewhere,2 and was approved by the Ethics Committee of Copenhagen (approval number V. 100.2263/91). The setting is community-based and includes all admitted stroke patients from a well-defined catchment area, regardless of the age of the patient, the severity of the stroke, and the condition of the patient before the stroke. All stages of acute care, workup, and rehabilitation take place within a stroke unit.

Included were all acute stroke patients admitted in a 1-year period, from September 1, 1992, to August 31, 1993: a total of 647 (mean age 74.6 years [SD 10.5], 45.3% men). Excluded were 213 patients without lower extremity paresis on admission, and 107 patients who were not assessed by a physiotherapist because of early death (59) or early full recovery (48). Thus, a total of 327 patients (mean age 76.3 years [SD 9.0], 41.0% men) were investigated.

Ipsilateral pushing was defined according to Davies1: The patient leans towards the hemiplegic side in any posture, and resists any attempt at passive correction of posture that would move his or her weight towards or across the midline of the body toward the unaffected side. The presence of ipsilateral pushing was assessed by the treating physiotherapist, who would score the patient as being a “pusher” if pushing was found in any posture.

Stroke severity. The severity of the stroke was assessed by the Scandinavian Stroke Scale (SSS)3-4 on admission, weekly during the hospital stay, and at discharge. The SSS evaluates level of consciousness; eye movement; power in arm, hand and leg; orientation; aphasia; facial paresis; and gait. The total score ranges from 0 to 58 points.

Neuropsychological symptoms. Anosognosia and neglect were assessed on admission using the test procedures described by Bisiac and coworkers.5 According to these procedures, visual...
neglect is assessed by a paper and pencil cancellation test, and body neglect is assessed by asking the patient to reach out for the upper limb on the affected side. Anosognosia is rated by questioning the patient about limb weakness and visual field defects. Aphasia was assessed using the aphasia subscale of the SSS. To assess upper limb motor apraxia, the patient was asked to point, wave, and salute; to assess oral apraxia, the patient was asked to put out the tongue, to blow up the cheeks, and to blow at something. All neuropsychological scores were dichotomized to symptoms present or absent for the purpose of the statistical analyses.

Functional severity and rehabilitation outcome. Activities of daily living (ADL) function was assessed by the Barthel Index (BI), which evaluates 10 different abilities and ranges in total score from 0 to 100 points. It was assessed the first time during the first week of the hospital stay and then subsequently every week during the hospital stay by the nursing and training staff. To compensate for the ceiling effect on the BI, "the percentage gain" was also computed as the obtained gain in BI multiplied by 100 and then divided by the possible gain in BI (the maximal score of 100 minus initial score). Patients with an initial BI of 100 were allocated a percentage gain score of 100. To appraise the effective recovery period, time in weeks from admission to achievement of the highest Barthel score during hospital stay was calculated.

Length of Rehabilitation was calculated as the length of hospital stay minus days spent in hospital for nonmedical reasons after completed rehabilitation, i.e., waiting time for nursing home. Thus, time spent in hospital only because of a shortage of places at nursing homes was not included in the length of rehabilitation.

Type, size, and localization of stroke lesion were determined by computed tomography (CT). CT was done in the very acute stage of stroke if needed for diagnostic purposes or therapeutic planning (e.g., to exclude a hemorrhage if anticoagulant therapy was being considered). In all other cases, the scan was performed according to the accessibility of the scanner, which varied during the study period. All scans were evaluated by the same radiologist, who was blinded to patient data. CT scans were obtained by a Siemens Somatom DR scanner. Description included type, size, and localization of stroke lesion. The size of stroke lesion was measured as the largest diameter. The side of the stroke lesion was determined by combining clinical data and CT data.

Rehabilitation based on the Bobath technique was given daily by the nursing staff, the physiotherapists, and the occupational therapists. Rehabilitation was completed within the department. Patients were discharged when further improvement in function was considered unlikely.

Statistics. Comparisons for continuous data were carried out with Student's t test for single comparisons. Categorical tables were analyzed with the χ² test. Univariate Pearson correlation coefficients were computed to inspect the structure of the data set. To evaluate the relative importance of multiple influences on outcome, multiple linear regression and logistic linear regression analyses were performed. Backward stepwise linear regression was followed by forward stepwise regression for all covariates with a probability <.02. The explanatory power of the resulting equations was determined by the adjusted R² statistic. For logistic regression, backward and forward Wald analysis was carried out. The required two-tailed significance level for all tests was set to .05, except for univariate correlations, for which the required one-tailed significance level was set to .001 because of the large number of significance tests. All analyses were performed with the SPSS for Windows 6.0 statistical package.

RESULTS

Incidence and Patient Characteristics

Patient characteristics are listed in table 1. The incidence of ipsilateral pushing in the included patients was 10.4%. This corresponds to 5.3% in all patients admitted in the study period. No differences were found between patients with and without ipsilateral pushing for age, sex, handedness, lesion side, or mortality. The patients with ipsilateral pushing had more severe strokes as expressed by lower neurological score (SSS) on admission and lower initial ADL function (BI score).

Table 1: Basic Characteristics for Patients Without and With Ipsilateral Pushing

<table>
<thead>
<tr>
<th></th>
<th>Without Ipsilateral Pushing</th>
<th>With Ipsilateral Pushing</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (incidence)</td>
<td>293 (89.6%)</td>
<td>34 (10.4%)</td>
<td></td>
</tr>
<tr>
<td>Age, years (SD)</td>
<td>76.4 (9.2)</td>
<td>75.0 (7.8)</td>
<td>NS</td>
</tr>
<tr>
<td>Sex, male %</td>
<td>40.3%</td>
<td>47.1%</td>
<td>NS</td>
</tr>
<tr>
<td>Handedness, right %</td>
<td>92.5%</td>
<td>85.2%</td>
<td>NS</td>
</tr>
<tr>
<td>Lesion side, left %</td>
<td>56.8%</td>
<td>47.1%</td>
<td>NS</td>
</tr>
<tr>
<td>Mortality %</td>
<td>17.1%</td>
<td>11.8%</td>
<td>NS</td>
</tr>
<tr>
<td>SSS on admission (SD)</td>
<td>32.6 (15.5)</td>
<td>24.3 (11.7)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Initial BI score (SD)</td>
<td>39.4 (37.5)</td>
<td>22.1 (15.4)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Abbreviations: SSS, Scandinavian Stroke Scale; BI, Barthel Index.

Associated Neuropsychological Symptoms

Not all patients were able to cooperate for the assessments of neglect, anosognosia, or apraxia on acute admission. Neglect could be assessed in 210 (64.2% of the included patients), anosognosia in 205 (62.7%), and apraxia in 222 (67.9%). There were no significant differences in the presence of any of the assessed neuropsychological symptoms on admission between the two groups (table 2).

Table 2: Neuropsychological Symptoms in Relation to Ipsilateral Pushing

<table>
<thead>
<tr>
<th></th>
<th>Without Ipsilateral Pushing</th>
<th>With Ipsilateral Pushing</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neglect %</td>
<td>29.5%</td>
<td>40.0%</td>
<td>NS</td>
</tr>
<tr>
<td>Anosognosia %</td>
<td>25.1%</td>
<td>27.3%</td>
<td>NS</td>
</tr>
<tr>
<td>Aphasia %</td>
<td>37.7%</td>
<td>47.1%</td>
<td>NS</td>
</tr>
<tr>
<td>Apraxia %</td>
<td>10.4%</td>
<td>4.8%</td>
<td>NS</td>
</tr>
</tbody>
</table>

Type, Size, and Localization of Stroke Lesions

CT was performed on 87% of the patients. Median duration from stroke onset to CT was 11 days. There was a different distribution of CT scans showing no visible lesion, infarct, and hemorrhage between the two groups, but no difference in lesion size in patients with visible lesions, and no difference in percentage of patients with cortical involvement (table 3). CT scans showing visible lesions were available for 29 of the 34 patients.
with ipsilateral pushing and for 173 of the 293 without ipsilateral pushing. Comparisons of involved regions for patients with and without ipsilateral pushing are listed in table 4. A Bonferroni correction for multiple comparisons was performed and only p values <.006 were taken into account. A significant difference is found for the crus posterior of the internal capsule, and the p value for crus anterior is close to the required level. However, it is not possible from inspection of the individual localization charts to point out any single lesion localization either necessary or sufficient for the presence of ipsilateral pushing.

Influence of Ipsilateral Pushing on Rehabilitation

Outcome

The results of univariate analyses of outcome in survivors are listed in table 5. All outcome parameters are significantly different for patients with and without ipsilateral pushing. Figure 1 shows the time course of functional recovery for patients with and without pushing expressed as the cumulated percentages of patients with stationary ADL function as a function of time poststroke. Stationary function is obtained in 80% of the patients without ipsilateral pushing within 6 weeks and in 80% of the patients with ipsilateral pushing within 13 weeks. The 95% level is obtained within 13 weeks for the patients without ipsilateral pushing and within 19 weeks for the patients with ipsilateral pushing.

A significant relationship between ipsilateral pushing and the severity of stroke was evident in the univariate analyses of correlations among potential predictor variables. Significant negative correlations were found between ipsilateral pushing and SSS on admission (r = -.22, p < .001) and BI on admission (r = -.29, p < .001). There were no significant correlations between ipsilateral pushing and age, prior stroke, comorbidity, or side of stroke lesion. Univariate analyses of correlations of outcome variables with potential predictor variables showed that the univariate correlation between ipsilateral pushing and percentage BI gain was low (r = -.13, p = .02). Ipsilateral pushing however, was, highly correlated with recovery period (r = .35, p < .001) and with length of rehabilitation (r = .44, p < .001).

In stepwise backward regression analysis of percentage BI gain, dependent variables were removed in the following order: (1) prior stroke, (2) sex, (3) ipsilateral pushing, and (4) side of stroke lesion. In the subsequent forward regression analysis, dependent variables were entered in the following order: (1) BI on admission, (2) SSS on admission, and (3) age on admission. The resulting equation explained 40% of the total variance: 61.92 (constant) + .54 · BI on admission + .59 · SSS on admission − .68 · age.

In stepwise backward regression analysis of recovery period, predictor variables were removed in the following order: (1) sex, (2) SSS on admission, and (3) age. In subsequent stepwise backward regression, variables were entered in the following order: (1) BI on admission, (2) ipsilateral pushing, and (4) side of stroke lesion. The resulting equation explained 22% of the total variance: 6.25 (constant) − .43 · BI on admission + 3.64 · ipsilateral pushing. To verify that this result was not an artifact of the BI percentage computation, similar analyses were carried out of BI score on discharge and raw BI gain. In no case was the b coefficient significant for ipsilateral pushing.

In stepwise backward linear regression analyses of length of rehabilitation, predictor variables were removed in the following order: (1) SSS on admission, (2) sex, (3) prior stroke, and (4) age. In subsequent forward stepwise regression, variables were entered in the following order: (1) BI on admission, (2) ipsilateral pushing, (3) comorbidity, and (4) side of stroke lesion. The resulting equation explained 35% of the total variance: 55.21 (constant) − .35 · BI on admission + 29.11 · ipsilateral pushing − 10.17 · comorbidity − 6.86 · side of stroke lesion.

Ipsilateral pushing thus had no independent influence on percentage gain in ADL function. It did, however, independently influence the recovery period and length of rehabilitation. In backward stepwise regression of independence in living after discharge from hospital, predictor variables were removed in the following order: (1) side of stroke lesion, (2) sex, (3) prior stroke, (4) ipsilateral pushing, (5) marital status, and (6) comorbidity. In subsequent forward stepwise regression variables were entered in the following order: (1) BI on admission and (2) age. The resulting equation correctly classified 82.6%
of all cases: 7.13 (constant) + .07 • BI on admission – .11 • age. Ipsilateral pushing thus had no independent significant influence on independent living.

**DISCUSSION**

This study was carried out in a unique community-based population of acute stroke patients. Uniform rehabilitation was given to all patients who needed it and was completed within the department. This is the first time the incidence of ipsilateral pushing has been reported. Ipsilateral pushing appeared in 10% of the included patients, corresponding to 5% of the total number of stroke patients admitted in the area.

**Syndrome Status**

Neglect was found in 40% of the patients with ipsilateral pushing and in 30% of the patients without ipsilateral pushing; this difference was not significant. Furthermore, there was no difference in another typical right hemisphere symptom, anosognosia (25% and 27%). Thus, no support was found for a "pusher syndrome" in the sense of a syndrome encompassing both physical and neuropsychological symptoms. It should be noted that this result runs contrary to the probable expectation bias in the observing physiotherapists who based their observations on the description by Davies.

**Localization**

The expected association with the right hemisphere neuropsychological symptoms of neglect and anosognosia was not found. An inverse relationship to left hemisphere neuropsychological symptoms of aphasia and apraxia also was not found. Thus, a right hemisphere localization is not likely, and correspondingly, no significant difference was found for side of stroke lesion between patients with and without ipsilateral pushing. The percentage of patients with cortical involvement in stroke lesions was not different between the two groups. In comparisons of single areas involved in stroke lesions between patients with and without ipsilateral pushing, a significant difference was found for the crus posterior of the internal capsule, suggesting that damage of sensory pathways may be involved in producing the symptom. However, examination of the individual localization charts found no obvious pattern; thus, it was not possible to single out lesions of one or more areas as either sufficient or necessary for producing ipsilateral pushing. However, it is surprising that no special association with parietal lobe involvement was seen, considering the importance for the body scheme usually ascribed to the parietal lobes.

**The Nature of the Symptom**

The lack of association with neuropsychological symptoms and the lack of a clear pattern of lesions in affected patients unfortunately leaves us with only few hints at possible mechanisms causing the symptom. Of particular interest for future research is the role of subcortical sensory pathways and relay stations. It could be speculated that exaggerated sensory feedback from the affected side6 (or defective gating of short-latency feedback pathways10) leads the patient to reflexively compensate a false feeling of leaning toward the unaffected side.

It is our clinical impression that pushing in different postures represents the degree of severity, so that during remission pushing will first disappear in the supine position, then in the sitting position, and finally in the standing position. A more detailed study of the pusher symptom is thus needed to ascertain whether this clinical impression is correct or whether pushing in different postures is in fact dissociable as an independent symptom.

**Impact on Functional Outcome**

Functional outcome was not influenced by ipsilateral pushing. In the multiple linear and logistic regression analyses, ipsilateral pushing had no independent influence on percentage gain in ADL function or on the rate of independent living after discharge. Ipsilateral pushing however, had, a very significant impact on the time course of recovery measured by the time it took to obtain the best ADL function score. Controlling for difference in initial stroke severity, ipsilateral pushing per se increased the recovery period by 3.6 weeks, or twofold the time needed by patients without ipsilateral pushing. Ipsilateral pushing also prolonged the stay in hospital to 29 days, which is an increase of 63% over the stay of patients without ipsilateral pushing.

The observation of Davies7 that patients with ipsilateral pushing often are transferred too early to nursing homes or other long-term institutions after unsuccessful rehabilitation attempts in hospitals may be valid for situations in which treatment planning does not take the symptom into account or the time needed for rehabilitation of these patients is not allowed.

**CONCLUSIONS**

The existence of a symptom of postural imbalance due to ipsilateral pushing among hemiplegic stroke patients was confirmed. The final functional outcome is not—as formerly believed—worsened by ipsilateral pushing per se, at least not in a specialized stroke unit designed to take care of all stages of rehabilitation. It does, however, adversely affect the speed of ADL recovery and, thus, prolong the length of hospital stay. The existence of a pusher syndrome including neuropsychological hemineglect symptoms was not confirmed, and ipsilateral pushing was not found to be associated with right hemisphere stroke lesions. Further research on ipsilateral pushing is needed to explore the nature of the symptom so that more efficient rehabilitation strategies can be developed.

**References**


**Supplier**