ABSTRACT. The four-year prognosis of patients with visuospatial inattention in a stroke register (altogether 255 patients) was studied. Sixty-six surviving patients under the age of 66 were examined neurologically and neuropsychologically after 3 months and 1 year from stroke. Fifty-two of these 66 patients were still reassessed after 4 years from onset. Twelve patients with ischaemic brain infarction had visuospatial inattention 7 had a clear-cut and contralateral neglect and 5 had milder and less lateralized inattention. The recovery of these 12 patients was poorer in ADL than the other 54 patients. Even when hemiparesis was taken into account, the difference still existed in ADL. The recovery of the 7 neglect patients was poorer than that of the 5 inattention patients. During the follow-up the visuospatial neglect persisted in all 7 cases and the visuospatial inattention disappeared in only one case.

Keywords: stroke, ischaemic brain infarction, contralateral neglect, visuospatial inattention, recovery, activities of daily living

Several studies concerning the recovery of stroke patients have shown that visuospatial deficits are correlated with poor outcome (4, 5, 14). Visuospatial inattention especially inattention of one half of visual field has been the focus of many studies while the milder forms of inattention have received much less interest. In addition, the reported recovery patterns are mostly based on shorter follow-ups lasting up to 6–7 months (1, 7, 11). The aim of this stroke register based follow-up study was to analyze the long-term prognosis of patients with visuospatial inattention of different levels of severity.

RESULTS

In neuropsychological examinations the performance of 12 patients indicated visuospatial inattention. Seven of these patients had a clear-cut and contralateral neglect and 5 patients had milder and less lateralized visuospatial inattention. We called the first group neglect and the latter inattention.

The mean age of the 12 patients was 55 years (range 36–64 years). All had ischaemic brain infarction (IBI). Ten patients (7 neglect, 3 inattention) had right hemispheric lesion and 2 (both inattention) left hemispheric lesion. The location of lesion was verified by brain scan in 6 cases. Only one
patient had restroke during the follow-up. At the acute stage 6 neglect patients and 2 inattention patients had hemiplegia, 1 neglect patient and 2 inattention patients had severe hemiparesis and 1 inattention patient had mild hemiparesis. All 12 patients received physiotherapy during the first 3 months following illness. No one received neuropsychological rehabilitation. Four of these 12 patients (3 neglect, 1 inattention) died between 1 and 4 years from onset of stroke.

During the follow-up period none of the 7 neglect patients gained independence in ADL. All but one of the 5 inattention patients were already independent at the 3-month examination.

Three neglect and 3 inattention patients were gainfully employed prior to stroke. Only 1 inattention patient returned to work (after 3 months examination).

During the follow-up the visuospatial neglect persisted in neuropsychological tests in all 7 cases and visuospatial inattention disappeared in only one case. All 12 patients had constructional apraxia in addition to other neuropsychological deficits. The recovery from these deficits varied among the patients.

Hemiparesis of the neglect patients persisted during the follow-up. Only one inattention patient still had hemiparesis at the 4-year examination.

At the all follow-ups the 12 patients with neglect or inattention were more dependent in ADL than the other patients in the series (8/12 vs. 10/54, \(p < 0.01\) at 3 months; 7/12 vs. 3/25, \(p < 0.001\) at 1 year; 4/8 vs. 3/44, \(p < 0.01\) at 4 years). However, the 12 patients had hemiparesis at the acute stage significantly more often than the other patients (12/12 vs. 27/54, \(p < 0.001\)). When only those patients having initial hemiparesis were compared, the difference was still seen in ADL: the 12 patients were more dependent in ADL than the other 27 patients with initial hemiparesis (7/12 vs. 7/27, \(p < 0.05\) at 3 months and 7/12 vs. 3/27, \(p < 0.01\) at 1 year).

When diagnosis and initial hemiparesis were considered together the difference still existed on ADL: the 12 patients were more dependent in ADL than the other 21 patients with hemiparesis (8/12 vs. 4/21, \(p < 0.01\) at 3 months and 7/12 vs. 1/21, \(p < 0.01\) at 1 year). However, initial hemiparesis of the 12 patients was more severe than that of the other patients (11/12 had hemiplegia or severe hemiparesis vs. 11/21, \(p < 0.05\)).

**DISCUSSION**

In this follow-up study concerning patients under the age of 65 in a stroke registry there were 12/66 (18%) patients who had visuospatial inattention: 7 had a clear-cut and contralateral neglect and 5 had milder and less lateralized inattention. They all had ischemic brain infarction. The lesion was in the right hemisphere in 10 of the cases; this was half of all the cases with right hemispheric lesion in the series.

The differentiation of milder forms of visuospatial inattention and the more severe and clear-cut neglect has seldom been considered in clinical studies. In our series the incidence of visuospatial inattention was 8% of the left hemisphere patients and 15% of the right hemisphere patients, which is slightly more than Diller & Weinberg (4) have reported. The clinical features of the visuospatial neglect syndrome in general are well known. The neglect is mostly associated with cerebrovascular disease of the right hemisphere (6, 8, 9). The incidence of neglect in the present series (35% of the right hemispheric patients) is in accordance with previous studies (4, 10, 15).

The 12 patients with visuospatial neglect or inattention remained more dependent in ADL than the other patients of the series. However, these 12 patients had more severe and persisting initial hemiparesis than the other patients. The same kind of results have also been reported earlier (3, 11).

Visuospatial inattention disappeared in only one case and visuospatial neglect persisted. The persist- ence of neglect has also been noted in other studies (2, 9, 16), although contradictory results have been reported (7). This might partly be due to differences in methods and in criteria used for neglect. Also the etiologies behind neglect vary in different studies. It is interesting that in our study the neglect did not change from the contralateral form to the milder and more generalized inattention. In the present series inattention also persisted during the whole follow-up.

In this study patients with milder forms of inattention recovered better than those with neglect. Further studies are needed to search the possible neuropsychological differences between milder forms of inattention and severe neglect.

**REFERENCES**


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Three neglect and 3 inattention patients were gainfully employed prior to stroke. Only 1 inattention patient returned to work (after 3 months examination).

During the follow-up the visuospatial neglect persisted in neuropsychological tests in all 7 cases and visuospatial inattention disappeared in only one case. All 12 patients had constructional apraxia in addition to other neuropsychological deficits. The recovery from these deficits varied among the patients.

Hemiparesis of the neglect patients persisted during the follow-up. Only one inattention patient still had hemiparesis at the 4-year examination. At the all follow-ups the 12 patients with neglect or inattention were more dependent in ADL than the other patients in the series (8/12 vs. 10/54, p<0.01 at 3 months; 7/12 vs. 32/54, p<0.001 at 1 year; 48 vs. 3/44, p<0.01 at 4 years). However, the 12 patients had hemiparesis at the acute stage significantly more often than the other patients (12/12 vs. 27/54, p<0.001). When only those patients having initial hemiparesis were compared, the difference was still seen in ADL: the 12 patients were more dependent in ADL than the other 27 patients with initial hemiparesis (7/12 vs. 7/27, p<0.05 at 3 months and 7/12 vs. 3/27, p<0.01 at 1 year).

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**REFERENCES**


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**Table II. The diagnostic distribution and location of lesion**

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