QUALITY OF LIFE AFTER STROKE
Clinical, functional, psychosocial and cognitive correlates

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Department of Neurology

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Marja-Liisa Kauhanen

Department of Neurology, University of Oulu and
Department of Rehabilitation, Oulu Deaconess Institute
To my family
Abstract

Depression is a common consequence of stroke and it is known to be associated with deterioration of quality of life. However, only limited information is available on the relationships between depression and communicative and cognitive disorders. Moreover, the present knowledge of the determinants of the domains of quality of life is limited, and little is known of e.g. the changes in sexual behaviour of stroke patients and their spouses.

This prospective study was carried out to evaluate the prevalence of post-stroke depression and aphasia and to study their interrelationships and neuropsychological and functional correlates. The particular aim of the study was to investigate the domain-specific quality of life, and to assess its clinical and sociodemographic correlates, and to study the impact of stroke on the sexual functions of stroke patients and their spouses. The study consisted of 156 first-ever stroke patients.

Depression was diagnosed in 53% of the patients at 3 months and in 42% of the patients at 12 months post-stroke according to DSM-III-R-criteria. One third of the patients were aphasic, 70% of them at 3 months and 62% at 12 months after stroke suffering from depression. Among the aphasic patients the prevalence of major depression increased from 11% to 33% during the 12 months’ follow-up. There was an association between post-stroke depression and cognitive impairment, the domains most likely to be defective being memory, non-verbal problem solving, and attention and psychomotor speed. The non-verbal neuropsychological test performance in the aphasic patients was significantly inferior to that of the patients with dominant hemisphere lesion without aphasia.

The quality of life of the patients was low at 3 months after the stroke, and it did not improve during the follow-up of a year. The test domains most often impaired were Physical functioning, Physical role limitations, Vitality and General health. Depression, although mostly minor, and being married emerged as significant independent contributors to low score value of Vitality and Physical role limitations. All the analyzed aspects of sexuality were commonly decreased as a consequence of stroke both in the patients and their spouses. Nocturnal erections were impaired in 21 (55%) of them in the male patients.

The present results demonstrate that more than half of the patients after stroke suffer from depression and the frequency of major depression seems to increase over time, especially among the aphasic patients. Both depression and aphasia increase the liability of cognitive deficits. Stroke affects various dimensions of quality of life extensively, and the most important determinants entailing low quality of life seem to be depression, and, interestingly, being married. As a part of quality of life, sexual function and satisfaction with sexual life are impaired both in stroke patients and spouses. These findings call for multidimensional evaluation of stroke patients and provide new challenges for stroke rehabilitation.

Keywords: cerebrovascular disorders, mood disorders, cognition, outcome
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Oulunsalo, October 1999

Marja-Liisa Kauhanen
### Abbreviations

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<tr>
<th>Abbreviation</th>
<th>Full Form</th>
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<tbody>
<tr>
<td>ADL</td>
<td>activities of daily living</td>
</tr>
<tr>
<td>BI</td>
<td>Barthel Index</td>
</tr>
<tr>
<td>CT</td>
<td>computed tomography</td>
</tr>
<tr>
<td>DSM</td>
<td>Diagnostic and Statistical Manual of Mental Disorders</td>
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<tr>
<td>MMSE</td>
<td>Mini Mental State Examination</td>
</tr>
<tr>
<td>MRI</td>
<td>magnetic resonance imaging</td>
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<tr>
<td>OR</td>
<td>odds ratio</td>
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<tr>
<td>PSD</td>
<td>post-stroke depression</td>
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<tr>
<td>QOL</td>
<td>quality of life</td>
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<tr>
<td>RAND 36</td>
<td>RAND 36-Item Health Survey</td>
</tr>
<tr>
<td>Rankin</td>
<td>Rankin Scale</td>
</tr>
<tr>
<td>SD</td>
<td>standard deviation</td>
</tr>
<tr>
<td>SF-36</td>
<td>MOS 36-Item Short-Form Health Survey</td>
</tr>
<tr>
<td>SPSS</td>
<td>Statistical Package for Social Sciences</td>
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<tr>
<td>SSS</td>
<td>Scandinavian Stoke Scale</td>
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<tr>
<td>WAB</td>
<td>Western Aphasia Battery</td>
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<tr>
<td>WAIS</td>
<td>Wechsler Adult Intelligence Scale</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
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<tr>
<td>WMS</td>
<td>Wechsler Memory Scale</td>
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<tr>
<td>ZDS</td>
<td>Zung Self-rating Depression Scale</td>
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List of original papers

This thesis is based on the following articles, which will be referred to in the text by their Roman numerals:


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1. Introduction

Stroke is a leading cause of long-term disability in western countries causing major individual, social and economic burdens. In spite of a declining trend in the incidence of stroke figures in Finland during the last few decades (Kotila 1984, Sarti 1994, Tuomilehto et al. 1996), an increase can be expected in the population at risk of stroke due to extended life expectancy.

Improving the quality of life (QOL) of stroke patients has received increasing attention as a part of the development of therapeutic strategies during the last decade (Hachinski 1999). Stroke patients have often been reported to have lower QOL than control subjects of similar age (Åström et al. 1992, Jonkman et al. 1998, Wyller et al. 1998), but contrary to these findings, King (1996) found stroke patients coping well with their stroke related impairments.

Mood disorders are a common but often unrecognized companion of stroke. The reported prevalence of post-stroke depression (PSD) varies from 20% to 65% (Primeau 1988, Robinson 1997). PSD is known to be related to dependence in activities of daily living (ADL) and to the severity of neurological deficits (Sinyor et al. 1986, Åström et al. 1993, Kotila et al. 1998), but the knowledge of its neuropsychological correlates is limited. Although clinical experience has shown that aphasia may markedly influence the severity and persistence of PSD, psychiatric evaluations of aphasic patients are very few (Åström et al. 1993, Sharpe et al. 1994).

PSD has been reported in some studies (Niemi et al. 1988, Åström et al. 1992, Angeleri et al. 1993) as being related to impaired QOL, but only in one of these studies was the diagnosis of depression based on psychiatric evaluation (Åström et al. 1992). Women tend to have poorer health and QOL in the general population (Jenkinson et al. 1993), but knowledge of the impact of gender and marital status on post-stroke QOL is limited. Although sexuality is a vital part of QOL, little information is available of changes in the sexuality of stroke patients and their spouses. Moreover, no measures of e.g. nocturnal penile tumescence of the male patients have been used.

The interpretation of QOL findings may be complicated by the wide variety in the methods used in evaluation of QOL (de Haan 1993, Williams 1998), by simultaneous grading of pre- and post-stroke QOL (Ahlsiö et al. 1984, Niemi et al. 1988, Viitanen et al. 1988), which may lead to overestimation of the pre-stroke condition, as well as by a
heterogeneity of time elapsed after the onset of stroke (Viitanen et al. 1988). The patients with moderate or severe aphasia and cognitive disorders have usually been excluded from the studies of PSD (Starkstein & Robinson 1988, Herrmann et al. 1993, Åström et al. 1993), although these patients are an important subpopulation in any investigation of PSD and its correlates. Very few prospective studies have been carried out using both neuropsychological tests to evaluate cognitive impairment and psychiatric examinations to diagnose depression after stroke.

The present prospective study was designed to examine the relationships between PSD and neuropsychological and functional deficits in patients with first-ever brain infarction, and to study the evolution of aphasia and its interrelationship with psychiatric, neurological and cognitive disorders. In particular, the study aimed at evaluating the domain-specific QOL, and assessing its clinical and sociodemographic correlates. As a part of QOL assessment, the study also aimed to examine the impact of stroke on the sexual behaviour of stroke patients and their spouses.
2. Review of the literature

2.1. General aspects of stroke

Stroke is a major health problem in all industrialized countries. It is the third leading cause of death and leaves many of its survivors with physical and mental disabilities, thus creating a major social and economic burden. (Kaste et al. 1998)

According to the World Health Organization (WHO) stroke is defined as “rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than vascular origin” (Aho et al. 1980). In 70-80% of cases stroke is caused by brain infarctions, in 9-15% by intracerebral hemorrhages and in about 10% by subarachnoid hemorrhages (Sivenius 1982, Rissanen 1992, Kaste et al. 1995, Numminen et al. 1996, Kaste et al. 1998).

Stroke afflicts all ages, but the incidence increases with advanced age (Kotila 1984, Dobkin 1995, Numminen et al. 1996). The incidence rate is generally greater in males aged under 65 years than females, but it equalizes with increasing age (Kotila 1984, Reunanen et al. 1986, Rissanen 1992, Numminen et al. 1995). There has been a declining trend in the stroke incidence in Finland since the 1970s (Aho 1975, Kotila 1984, Sarti et al. 1994, Numminen et al. 1996, Tuomilehto et al. 1996), but as public health, medical and social advances continue to extend life expectancy, we can expect an increase in the size of the population at risk of stroke (Special Report From the World Health Organization 1989).

Stroke is the most important cause of physical disability in people over 60 years of age (Kaste et al. 1995). Hemiparesis is the most constant neurologic finding, its frequency in the acute stage varying from 70 to 85% (Sivenius 1982, Kotila 1986, Rissanen 1992, Dobkin 1995). The neurological deficits often improve during the first months up to one year so that after a year about 22% of patients are dependent in walking (Gresham et al. 1979, Rissanen 1992). Most of the patients (53-76%) are independent in ADL after a year, 8-28% being totally dependent on the help of other people (Sivenius 1982, Kotila 1986, Aho et al. 1986, Rissanen 1992).

Disturbances of speech are quite common in the acute phase, the frequency of aphasia varying from 20% to 38% and declining to 12-28% after 6 months (Sivenius 1982, Kotila 1986, Wade et al. 1986, Dobkin 1995, Pedersen et al. 1995). Cognitive impairment has
been found in 27% to 35% of patients at 3 months after ischemic stroke, the domains most likely to be defective being memory, orientation, language, attention and visuospatial functions (Tatemichi et al. 1994, Pohjasvaara 1998). Kotila (1986) found visuoperceptual deficits in 60% of patients at 3 months and in 41% at 12 months post-stroke, and memory dysfunction in 55% and 31%, respectively.

2.2. Post-stroke depression

2.2.1. General aspects of post-stroke depression

Depression is an ever-present and often unrecognized problem after stroke having both biological and psycho-social dimensions (Hachinski 1999). The reported prevalence of PSD varies from 20% to 65% depending on the selection of patients, diagnostic criteria and the time elapsed after stroke (Primeau 1988, House et al. 1991, Åström et al. 1993, Robinson 1997, Kotila et al. 1998). Robinson et al. (1987) found in a subsample of their original group a stable prevalence of depression for up to 2 years. All the patients with major depression in-hospital had improved during the follow-up, but the prognosis of minor depression was unfavorable, only 30% of follow-up patients with minor depression being improved within 2 years. In other studies the majority of patients with major depression experienced remission within the first year (House et al. 1991, Åström et al. 1993). However, a minority of patients had not recovered by one year and were at a high risk of chronic depression (Åström et al. 1993). Table 1 presents the earlier cohorts examining PSD.

The estimation of the PSD rates must be looked at cautiously because of the different methods used for selecting the study populations and the different methods of diagnosis. Many of the earlier reports can be criticized for their small study samples (Robinson & Price 1982, Robinson et al. 1986, Morris et al. 1992) and for the exclusion of patients with aphasia and comprehensive deficits (House 1987). Moreover, some studies have not excluded patients with a previous history of depression (Åström et al. 1993, Palomäki et al. 1999). The diagnosis of PSD has often been based on self-report inventories, which may produce unreliable results due to the patients’ verbal and cognitive deficits (House 1987, Gordon & Hibbard 1997).

The etiology of PSD has aroused interest in many investigators. Some investigators claim that the etiology of PSD is a complex mixture of pre-stroke personal and social factors and it arises as a psychological reaction to stroke induced impairments and social handicap (House 1987, House et al. 1991, Andersen et al. 1995). The opposite conclusion of Lyketsos et al. (1998) is that “stroke lesions, under certain circumstances, cause depression through a direct but unknown pathophysiologic process”. This claim has been supported by many studies (Robinson et al. 1984, Herrmann et al. 1993, Åström et al. 1993) showing that left anterior brain lesion is the most important predictor of major depression in the acute phase, but there are also opposite claims (Herrmann et al. 1995, Herrmann et al. 1998, Pohjasvaara et al. 1998). In conclusion, the etiology of PSD seems to be multi-factorial including both pre-stroke personal and social factors, stroke induced psychological reactions and organic backgrounds.
Table 1. Earlier stroke cohorts examining PSD.

<table>
<thead>
<tr>
<th>Study</th>
<th>N of subjects, age</th>
<th>Time from stroke</th>
<th>Definition of PSD</th>
<th>Prevalence of PSD</th>
<th>Correlates of PSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Robinson et al. 1986</td>
<td>n=38, mean age 56 years</td>
<td>at 2 weeks and at 6 months</td>
<td>HDS, ZDS, PSE DSM-III</td>
<td>major depression 29%</td>
<td>cognitive impairment measured by MMSE</td>
</tr>
<tr>
<td>Åstööm et al. 1993</td>
<td>n=80, mean age 73 years</td>
<td>in the acute phase, at 3 months and at 1, 2 and 3 years</td>
<td>DSM-III-R, major depression</td>
<td>acute phase: 25%</td>
<td>acute phase: left anterior lesion, aphasia, living alone</td>
</tr>
<tr>
<td>Sharpe et al. 1995</td>
<td>n=60, 33 &lt;75 years 27 &gt;75 years</td>
<td>at 3 to 5 years after first stroke</td>
<td>DSM-III-R</td>
<td>18%</td>
<td>large lesion volume, female functional dependence</td>
</tr>
<tr>
<td>Heinmenn et al. 1998</td>
<td>n=150, mean age 75 years</td>
<td>at 3 and 12 months</td>
<td>MADRS, ZDS</td>
<td>3 months: ZDS 22% MADRS 27% 12 months: ZDS 21% MADRS 22%</td>
<td>neurological impairment, female, previous history of depression</td>
</tr>
<tr>
<td>Kotila et al. 1998</td>
<td>n=181, mean age 69 years in active programs n=140, mean age 68 years in control districts</td>
<td>at 3 and 12 months</td>
<td>BDI</td>
<td>3 months: active programs 42% control districts 54% 12 months: active programs 42% control districts 55%</td>
<td>age &lt;70 years, severe SSS prognostic score</td>
</tr>
<tr>
<td>Poljasvaara et al. 1998</td>
<td>n= 277, aged from 55 to 85 years</td>
<td>at 3 to 4 months</td>
<td>PSE, DSM-III-R</td>
<td>40%, major 26%, minor 14%</td>
<td>neurological impairment, female, previous history of depression</td>
</tr>
<tr>
<td>Nezu et al. 1998</td>
<td>n=65, mean age 36 years</td>
<td>at 12 to 59 months</td>
<td>MADRS, DSM-III-R</td>
<td>48%</td>
<td>localization of infarct in carotid territory, severe disability, bad general outcome</td>
</tr>
</tbody>
</table>

HDS; Hamilton Depression Scale, ZDS; Zung Self-rating Depression Scale, FSE; Present State Examination, DSM-III; Diagnostic and Statistical manual of Mental Disorders, MADRS; Montgomery Asberg Depression Rating Scale, BDI; Beck’s Depression Inventory.
Although depression is an important consequence of stroke, it is recognized and treated in only a minority of patients (Wade et al. 1987, Åström et al. 1993, Kotila et al. 1998). Selective serotonin reuptake inhibitor citalopram has been shown to be both safe and effective in the treatment of PSD (Andersen et al. 1994). On the other hand, in a recent study (Palomäki et al. 1999) early initiation of miancerin had no effect on PSD or functional outcome, but according to the investigators good medical care and comprehensive and individually planned early rehabilitation may have abolished the possibility of the antidepressant to influence PSD.

### 2.2.2. Measurement and diagnosis of post-stroke depression

One of the main problems in research of PSD is the diagnostic accuracy of standardized psychiatric assessment techniques in neurologically impaired individuals. Traditional psychiatric criteria for mood disorders rely heavily on patients’ own reports of their symptoms. This requires patients to be aware of their situation, and to be capable of providing an accurate report of it, and to be free of any medical conditions that could mimic the symptomatology of depression. (Gordon & Hibbard 1997)

Self-report inventories are widely used in research of PSD. These inventories, however, have a number of drawbacks. They tend to have relatively low specificities and predictive values because the inclusion of somatic symptoms leads to high false positive rates (Goldberg 1985). The variability in awareness of their situation can influence the accuracy of self-report inventories in stroke patients (Gordon & Hibbard 1997). Another important problem is that stroke patients may be unable to complete the investigatory forms because of aphasia and other cognitive and physical impairments (Primeau 1988, House 1991, Gordon & Hibbard 1997). Hence, a careful psychiatric interview with multiple sources of information about the patients’ mood from behavioral observations of the patients and from family members and staff is crucial for the diagnosis of PSD (House 1991, Åström et al. 1993, Sharpe et al. 1994, Gordon & Hibbard 1997).

Several studies have pointed out that the phenomenology of PSD contains the core features associated with functional depression (Lipsey et al. 1986, Fedoroff et al. 1991, House 1991). For research purposes two types of depressive disorder have been identified according to DSM-III-R criteria (American Psychiatric Association 1987): major depression, and minor depression which meets the criteria for dysthymic disorders ignoring the 2-year criterion of DSM-III-R classification (Primeau 1988). The reliability of DSM-III criteria in the diagnosis of post-stroke depression has been shown in previous reports (Fedoroff et al. 1991, Sharpe et al. 1994, Robinson 1997). Fedoroff et al. (1991) compared depressive stroke patients with non-depressive ones for the frequency of depressive symptoms, and showed that only 3% of their depressive patients may have been overdiagnosed, and only 5% of their patients underdiagnosed.
2.2.3. Cognitive impairment and post-stroke depression

Many studies have demonstrated a greater degree of cognitive impairment in depressive stroke patients than in non-depressive ones (Robinson et al. 1986, Downhill & Robinson 1994, Sharpe et al. 1994). In a 2-year prospective study of 309 stroke patients Downhill & Robinson (1994) found that the frequency and severity of cognitive deficits were significantly greater in patients with major depression following left hemisphere stroke than in patients without depression. This correlation between depression and impaired cognitive function was strongest in the acute phase, but was present for up to one year. Moreover, the duration of depression was longer in patients with both depression and cognitive impairment than in depressive patients without cognitive impairment. On the other hand, depressive patients have been shown to improve less in cognitive performance than non-depressive ones, reflecting the negative impact of depression on the recovery of cognitive functions (Morris et al. 1992). Unlike others Kase et al. (1998) found that intellectual decline was independent of the presence of PSD. These results, however, must be interpreted with caution because depression was measured with a single self-report inventory and cognitive functions with the Mini-Mental State Examination (MMSE).

There are very few prospective studies of PSD and cognitive function which have been carried out using neuropsychological tests for diagnosing cognitive impairment. Most studies have used the MMSE to detect the global deterioration of cognitive functions (Robinson et al. 1986, Downhill & Robinson 1994, Sharpe et al. 1994, Kase et al. 1998). The MMSE, however, has several limitations, including its dependence on verbal skills to communicate the test instructions and the different degree of sensitivity of its various items (Feher et al. 1992, Hill & Backman 1995).

2.2.4. Other clinical and functional correlates of post-stroke depression

PSD is known to be related to the severity of neurological deficits and to dependence in ADL (Sinyor et al. 1986, Åström et al. 1993, Kotila et al. 1998). A correlation has also been found between functional outcome and PSD in patients with relatively mild depressive disorders (Herrmann et al. 1998). In the study of Åström et al. (1993) dependency in ADL was not related to immediate major depression, but at 3 months it was an important predictor of depression. The studies indicate that functional impairment does not determine the onset of depression but interacts with depression, resulting in a poorer long-term functional recovery (Parikh et al. 1987, Morris et al. 1992, Åström et al. 1993, Shimoda & Robinson 1998).

PSD has been found to be frequent in young patients (Neau et al. 1998, Paradiso & Robinson 1998). On the other hand, elderly people may be more at risk of depression, because of functional and cognitive impairment, residence in an institution and lack of social support (Sharpe et al. 1994).
A correlation between PSD and a previous history of depression has been found in some studies (Andersen et al. 1995, Herrmann et al. 1998, Pohjasvaara et al. 1998), but not in all (Åström et al. 1993). In one study (Paradiso & Robinson 1998) the history of a psychiatric disorder was a risk factor for PSD only among women.

Epidemiologic data from around the world demonstrate that major depression is more common in women than in men (Weissman & Olfson 1995). The same relationship between depression and female gender is also found in stroke patients (Andersen et al. 1995, Herrmann et al. 1998, Kotila et al. 1998, Paradiso & Robinson 1998). In the study of Paradiso & Robinson (1998) major PSD was twice as frequent in women as in men. Among women greater severity of depression was associated with left hemisphere lesion location, a personal history of psychiatric disorders, and cognitive impairment, whereas among men, the risk factors for depression were impairment in ADL and social functioning. These findings might have important therapeutic implications for both genders (Paradiso & Robinson 1998).

An association between depression and impaired social functioning has been reported in several studies (Parikh et al. 1987, Åström et al. 1993, Andersen et al. 1995). The reduced social contacts of depressed stroke patients may be a cause as well as a result of depression.

2.3. Post-stroke aphasia

2.3.1. Occurrence of aphasia

Aphasia is a common consequence of stroke, the reported prevalence ranging up to one third of the patients in the acute phase (Kotila et al. 1984, Reinvang et al. 1984, Wade et al. 1986). Spontaneous recovery of aphasia is at its greatest during the first 3 months, but some improvement may take place even later on (Demeurisse et al. 1980, Kotila et al. 1984, Wade et al. 1986). The frequency of complete recovery varies from 21% to 50% (Kertesz & McCabe 1977, Pashek & Holland 1988, Pedersen et al. 1995). The initial severity of aphasia has been shown to be highly predictive of ultimate language function (Kertesz & McCabe 1977, Pedersen et al. 1995), and a valid prognosis of aphasia can usually be made within the first month after stroke (Pedersen et al. 1995).

According to Western Aphasia Battery (Kertesz 1982) aphasia is classified as non-fluent aphasias, including global, Broca’s, isolation and transcortical motor aphasias, and as fluent aphasias, including Wernicke’s, transcortical sensory, conduction and anomic aphasias. It has been shown that different types of aphasia have distinctive recovery patterns (Kertesz 1984, Pashek & Holland 1988), but some contrary evidence is also available (Demeurisse et al. 1980, Lendrem & Lincoln 1985). Among the aphasia types global aphasia has the poorest prognosis, Broca’s aphasics often recovering towards anomic aphasia, and Wernicke’s aphasics tending to recover towards anomic or conduction aphasias. Anomic aphasia is a common endstage in the evolutionary process of both fluent and non-fluent aphasias. Both anomic aphasia and conduction aphasia have a favorable spontaneous recovery pattern (Kertesz 1984, Pashek & Holland 1988).
2.3.2. Depression in aphasic stroke patients

Although clinical experience has shown that communicative disorders may markedly contribute to the severity and persistence of depression, psychiatric evaluations of aphasic patients are scarce. Herrmann et al. (1993) concluded that “moderately and severely aphasic patients are an important subpopulation in investigations of PSD that must not be disregarded by exclusion for methodological reasons”.

In their prospective study of PSD Åström et al. (1993) found that aphasia was an important predictor of depression in the acute phase. The correlation between aphasia and depression lasted for 3 months, but no longer. In another study (Robinson & Benson 1981) almost half of the hospitalized aphasic patients were depressive, but the sample was highly selective. Among long-term survivors of stroke no relationship between aphasia and depression could be found (Oder et al. 1991, Sharpe et al. 1994).

The non-fluency of aphasia has been shown to be related to frequency and severity of PSD by some investigators (Robinson & Benson 1981, Herrmann et al. 1993), but not all (Starkstein & Robinson 1988). This relationship is thought to be caused by the better awareness of their impairments of patients with non-fluent aphasia.

The etiology of depression in post-stroke aphasic patients seems to be multi-factorial. Psychosocial and coping factors associated with aphasia are relevant for the development of reactive depression (Herrmann et al. 1993, Åström et al. 1993). On the other hand, aphasia and depression can be viewed as coincident consequences of stroke resulting from an underlying brain lesion (Starkstein & Robinson 1988, Herrmann et al. 1993).

2.3.3. Other clinical and functional correlates of aphasia

Most studies have found no evidence of an association between prevalence and recovery of aphasia and age (Reinvang 1984, Lendren & Lincoln 1985, Pedersen et al. 1995). In one study old age appeared to predict a poor prognosis for change in global aphasia (Pashek & Holland 1988). Sex has not been found to be a determinant of aphasia in stroke (Kertesz & McCabe 1977, Kertesz 1984, Pedersen et al. 1995).

Aphasia has been shown to be related to the severity of motor deficits and impairment in ADL during the first post-stroke months (Wade et al. 1986). The association may be due to the fact that patients with aphasia may have larger lesions, causing both more severe impairment in ADL and more language disturbance (Wade et al. 1986). In the presence of motor deficits the severity of aphasia in the subacute stage does not, however, influence the long-term outcome after cerebral infarction (Oder et al. 1988).

The difficulty of defining intelligence in aphasic patients hampers research design and the interpretation of results. Verbal tests of intelligence are clearly inappropriate in aphasia but the verbalization used to perform the nonverbal tests remains undetermined. (Kertesz 1977) The findings of the correlations between non-verbal cognitive impairment and type and severity of aphasia have been contradictory. In the study of Basso et al. (1981) the presence and severity of aphasia was definitely associated with low non-verbal test scores, but the type of aphasia was not. In another study, using the same test for
measuring logical reasoning and visuospatial ability, the type of aphasia was a significant
determinant of poor performance in the non-verbal test, but the severity of aphasia was not
(Kertesz & McCabe 1975).

2.4. Quality of life after stroke

2.4.1. General aspects of quality of life

During the past two decades there has been an increasing consensus about the importance
of patients’ subjective accounts of health in monitoring medical outcomes (Ware &
gaining a better understanding of patients’ reaction to illness and for the development of
therapeutic processes, as well as in monitoring the efficacy of medical care. The data
derived from QOL measures may be used to help inform economic analyses and resource
allocation and to influence health-care policy. (de Haan et al. 1993, Guyatt et al. 1993,
Fallowfield 1996)

QOL has been defined with various modalities, for instance, health related subjective
experiences, life satisfaction or subjective well-being, the last one focusing more on
emotions. However, there is a broad consensus that the assessment of QOL should
include aspects of physical, psychological, social and general health (Ware & Sherbourne
1992, de Haan et al. 1993). QOL relates closely to the definition of health issued by the
WHO: “a state of complete physical, mental and social well-being and not merely the
absence of disease or infirmity” (World Health Organization 1952). At the patient’s level
QOL can be seen as a result of a complex process of interaction between personal traits,
medical outcome, coping behavior, social support and the quality of received health care
(Kwa et al. 1996).

2.4.2. Measurement of quality of life in stroke patients

Measuring QOL can be used to distinguish different patients, to predict patient outcomes,
and to evaluate therapeutic interventions (Fallowfield 1996, Williams 1998). QOL
instruments can be divided into generic and disease-specific scales: generic scales address
general health concepts not specific to any age, disease, or treatment, enable comparisons
of the relative burden of different diseases and the relative benefits of different treatments
(Ware & Sherbourne 1992, de Haan et al. 1993); disease-specific scales do not allow
cross-disease comparisons, but may be more sensitive to a specific population (de Haan
et al. 1993). No evaluative stroke-specific QOL instrument is currently available
(Williams 1998).

Although stroke is a major problem, the best method for measuring the outcome of
stroke is not clear, partly due to the heterogeneity of stroke signs and symptoms (Williams
1998). QOL is mostly assessed by instruments depending on self-reports. These methods
of data collection are not very suitable for patients with cognitive or communicative
disorders. (de Haan et al. 1993, Sneeuw et al. 1997) One way to avoid this methodological problem is to use so-called proxy ratings, but the evidence from caregivers’ reports is contradictory (de Haan et al. 1993, Sneeuw et al. 1997). Further research is needed to elucidate the problem concerning the measurement of QOL in non-communicative patients (Kwa et al. 1996).

The selection of the QOL measure must be based on its psychometric attributes, which include feasibility, validity, reliability, and sensitivity to change (Dorman et al. 1998). The method of collecting data requires some trade-off between costs and response rates, non-response bias and data quality (O’Mahony et al. 1998). The short form health survey (SF-36) (Ware & Sherbourne 1992) is increasingly being used to measure subjective health status in stroke clinical trials (Williams 1998). The SF-36 has eight separate dimensions: Physical functioning, Role limitations due to physical problems, Mental health, Role limitations due to emotional problems, Vitality, Social functioning, Bodily pain, and General health. Another instrument RAND-36 (Hays et al. 1993) contains the same questions as the SF-36 scale, but the scoring for the General health and Bodily pain subscales slightly differs. The validity and reliability of the SF-36 in stroke patients has been confirmed (Anderson et al. 1996, Dorman et al. 1998). The SF-36 questionnaire has also been shown to be suitable for use with elderly people when presented by investigators themselves (Anderson et al. 1996, O’Mahony et al. 1998). The characteristics of an evaluation using the SF-36 parameters soon after stroke are not known, because most of the current data are from patients almost one year post-stroke (Anderson et al. 1996, Duncan et al. 1997, Williams 1998).

2.4.3. Determinants of post-stroke quality of life

Stroke survivors often have significantly lower QOL than control subjects of similar age (Viitanen et al. 1988, Åström et al. 1992, Jonkman et al. 1998, Wyller et al. 1998) even those with only mild consequences of stroke (Duncan et al. 1997). Contrary to these findings, one study (King 1996) showed that stroke patients were coping well with stroke related impairments, and their QOL was comparable with a sample of elderly outpatients with chronic illnesses. In their prospective study Åström et al. (1992) found improvement in life satisfaction during the follow-up. Patients with restored good satisfaction retained it for up to 3 years, whereas poor life satisfaction at 1 year remained poor for the entire 3-year follow-up.

Many reports have shown the deteriorating effect of depression on the QOL of stroke patients (Niemi et al. 1988, Åström et al. 1992, Angeleri et al. 1993, King 1996). Contrary to these findings, in one study of long-term survivors of stroke, depression was correlated only with leisure time activities (Vitaten et al. 1988).

Because of the importance of communication for social contacts and integration, communicative disorders have been found to be significantly associated with poor QOL (Kwa et al. 1996, Wyller et al. 1997). The interrelationship between cognitive functions and QOL seems to be contradictory. In one study (Niemi et al. 1988) patients with restored or improved QOL had significantly better cognitive functions than those with deteriorated QOL. In another study (Kwa et al. 1996) cognitive impairment was
correlated with poorer QOL, but linear regression analysis revealed no significant impact on QOL. Jonkman et al. (1998) found no correlation between cognitive disturbances and poor QOL. However, there are methodological diversities in assessing the impact of communicative and cognitive impairment on QOL after stroke.

Several authors have reported a strong association between physical disability, dependency in ADL and QOL (Ahlsiö et al. 1984, Niemi et al. 1988, Åström et al. 1992, Anderson et al. 1996, Kwa et al. 1998). Dependency in ADL has been shown to be associated with physical functioning and the general health domains of QOL (Anderson et al. 1996, King 1996), but not to predict psychological and socioeconomic aspects of QOL (King 1996).

The correlation between age, sex and QOL has remained obscure. Anderson et al. (1996) showed that women had a better stroke outcome in terms of social functioning and mental health, but most authors report QOL either to be independent of gender (Ahlsiö et al. 1984, Kwa et al. 1998) or lower in females (Angeleri et al. 1993, Wyller et al. 1997). QOL has been shown to decline with increasing age (Åström et al. 1992, Wyller et al. 1997), but there are many studies with no differences between younger and older patients (Ahlsiö et al. 1984, Viitanen et al. 1988, Kwa et al. 1998) or with life satisfaction increasing with increasing age (Wyller et al. 1998).

Failure to maintain or reestablish social ties, except for those with family members, seems to be an important determinant of poor QOL in long-term survivors of stroke (Åström et al. 1992), whereas high levels of social support have been shown to be related with a better outcome (Glass et al. 1993, Wyller et al. 1998). On the other hand, too much support from the spouse may lead to overprotection and understimulation and lead to a less favorable outcome (Glass et al. 1993, Anderson et al. 1995). To ensure a good outcome, the support of the family is not enough: the support of society is also needed (Angeleri et al. 1993, Kotila et al. 1998) so that stroke victims feel cared for, loved, valued and esteemed and are ready to accept assistance from others if needed.

Work is also an important source of life satisfaction for those stroke patients who are capable of returning to work (Niemi et al. 1988, King 1996). Job satisfaction may be considered as an indicator of good QOL in patients returning to work (Angeleri et al. 1993).

### 2.4.4. Sexual functioning after stroke

A low quality of sexual life and a marked decline in sexuality after stroke has been reported in previous studies (Viitanen et al. 1988, Angeleri et al. 1993, Monga & Osterman 1995, King 1996, Korpelainen et al. 1999). The problems that have been identified include a decline in libido and coital frequency in both genders, a decline in vaginal lubrication and orgasmic ability in women, and poor or absent erection and ejaculation in men (Bray et al. 1981, Sjögren 1983, Monga et al. 1986, Korpelainen et al. 1999). Information regarding sexuality in aphasic patients is limited. Kinsella & Duffy (1979) reported that the marriages of aphasic patients were characterized by problems of interpersonal communication, diminished sexual satisfaction and loss of partnership.
The determinants of post-stroke sexuality have rarely been assessed. Some studies have suggested that pre-stroke sexual activity is predictive of sexual activity after stroke (Goddess et al. 1979, Bray et al. 1981, Hawton 1984). There is no general agreement about the incidence of sexual disturbance as a function of the side of the cerebral lesion. It has been suggested that lesions on the dominant cerebral hemisphere cause declined sexuality, including decreased libido, coital frequency and satisfaction with sexual activity (Kalliomäki et al. 1961, Goddes et al. 1979, Monga et al. 1986), but some reports (Sjögren et al. 1983, Boldrini et al. 1991) have not been able to find any correlation between the affected hemisphere and sexual functions. In one study (Sjögren & Fugl-Meyer 1981) the degree of cutaneous sensibility was related to the changes in coital frequency and to the level of independence in ADL, but not to the degree of motor impairment.

The etiology of post-stroke sexual dysfunction is complex and multi-factorial (Monga & Osterman 1995). Psychosocial factors seem to have a strong impact on sexual functioning and satisfaction in sexual life after stroke (Sjögren et al. 1983, Monga et al. 1986, Boldrini et al. 1991, Korpelainen et al. 1999). In addition to neurological and cognitive deficits associated with stroke, sexual satisfaction may also be impaired because of a variety of medical conditions, such as diabetes mellitus, hypertension, coronary heart disease or depression (Monga & Osterman 1995). Medication, for instance antihypertensives, antidepressives, minor tranquilizers and muscle relaxants, may also influence sexual performance (Balon et al. 1993, Monga & Osterman 1995, Korpelainen et al. 1999).

Little information is available about the sexual function of the spouses of stroke victims, although they are very much involved with the burden caused by the disease. Previous studies have reported negative changes similar to those experienced by the patients in the sexuality of the spouses (Kinsella & Duffy 1979, Sjögren 1983, Boldrini et al. 1991, Korpelainen et al. 1999).
3. Aim of the study

Although stroke may affect multiple dimensions of QOL, knowledge concerning its clinical, functional and cognitive correlates is limited. Moreover, little attention has been paid to the sociodemographic aspects of domain-specific QOL after stroke and to the sexual functioning of stroke patients. The aim of the present study was to evaluate QOL after first-ever stroke. More precisely defined, the aims were as follows:

1. To evaluate the prevalence and course of depression after stroke and to study the clinical, functional and neuropsychological correlates of PSD.
2. To study the prevalence and evolution of aphasia and to assess its psychiatric, neurological and cognitive interrelationships in stroke.
3. To evaluate the domain-specific QOL after stroke and to study its clinical and sociodemographic correlates.
4. To assess the sexual behaviour of stroke patients and their spouses.
4. Subjects and methods

4.1. Subjects

The study was carried out in the Department of Neurology, Oulu University Hospital (I-III) and the Department of Neurological Rehabilitation, Oulu Deaconess Institute (IV). The Ethics Committee of the Medical Faculty, University of Oulu, approved the protocol of the study, which was carried out in accordance the principles of the Declaration of Helsinki. Informed consent was obtained from each patient or the nearest relative.

Consecutive patients with their first-ever brain infarction admitted to the Stroke Unit of Oulu University Hospital were considered for the study (I-III). Patients with TIA, or a markedly decreased level of consciousness, as well as patients with previous psychiatric illnesses or central nervous system disorders and alcoholism were excluded. There were 159 patients, who were eligible. The first two patients of each week were randomized for the study. In this way 25 patients were excluded from the study, and 28 further patients refused to participate in the study (altogether 28 women and 25 men, mean age 66.1 years, range 40-80 years). The remaining 106 patients (46 women and 60 men, mean±SD age 65.8±11.9 years, range 19-82 years) were the subjects of studies I-III.

Two patients died before the 3-month follow-up visit and 3 further patients before the 12-month visit. Three patients refused to continue in the study at the 3-month visit, and 6 further patients at the 12-month visit. Thus, 101 of the initial 106 patients participated in the 3-month examinations and 92 patients in the 12-month examinations (I-II). Sixteen patients who were unable to answer the questionnaires because of communicative disorders were excluded from study III. Thus, 85 (36 women, 49 men, mean±SD age 65.0±12.5 years, range 19-82 years) of the initial 106 patients at 3 months and 76 at 12 months post-stroke were included in study III. Twenty-nine of the initial 106 patients had previously been healthy, 46 patients had hypertension, 38 coronary heart disease and 21 diabetes mellitus.

The series in study IV consisted of 50 consecutive patients (38 men and 12 women, mean±SD age 53.5±8.2 years, range 32 to 65 years) with first-ever stroke who were admitted to the Department of Neurological Rehabilitation, Oulu Deaconess Institute, for inpatient rehabilitation. Thirty-eight patients had a brain infarction and 12 patients an intracerebral haemorrhage. Only married patients with an active pre-stroke sexual life
were included in the study. Patients older than 65 years and patients with manifestations of other central or peripheral nervous system lesions and patients with other diseases (e.g. diabetes mellitus, alcoholism) known to affect the autonomic nervous system were excluded. Patients with severe aphasia or with major psychiatric diseases, as well as patients with other earlier diseases affecting their independence in the ADL, were also excluded. At 6 months post-stroke 7 patients and spouses refused to continue in the study.

The age and sex distribution of the patients and the localization of the lesion in different studies are presented in Table 2.

**Table 2. Description of stroke patients in the individual studies.**

<table>
<thead>
<tr>
<th>Patients</th>
<th>Study I-II</th>
<th>Study III</th>
<th>Study IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of subjects</td>
<td>106</td>
<td>85</td>
<td>50</td>
</tr>
<tr>
<td>Female/male</td>
<td>46/60</td>
<td>36/49</td>
<td>12/38</td>
</tr>
<tr>
<td>Age (mean±SD years)</td>
<td>65.8±11.9</td>
<td>65.0±12.5</td>
<td>53.5±8.2</td>
</tr>
<tr>
<td>Lesion location</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisphere</td>
<td>88</td>
<td>68</td>
<td>41</td>
</tr>
<tr>
<td>Brainstem</td>
<td>17</td>
<td>16</td>
<td>7</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Hemispheral lesion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dominant</td>
<td>53</td>
<td>34</td>
<td>21</td>
</tr>
<tr>
<td>Nondominant</td>
<td>35</td>
<td>34</td>
<td>20</td>
</tr>
</tbody>
</table>

In study III fifty-nine (69 %) of the patients were married (or cohabiting), and 26 (31%) were single, divorced or widows/widowers. In the acute phase 18 (21%) patients, at 3 months 15 (18%) patients, and at 12 months 14 (18%) patients were living alone.

**4.2. Methods**

**4.2.1. Clinical examination**

All the patients were investigated clinically in the acute phase and at 3 and 12 months after the stroke. Neurological disability was evaluated using the Scandinavian Stroke Scale (SSS) (Scandinavian Stroke Study Group 1985), and performance in the ADL using the Barthel Index (BI) (Mahoney & Barthel 1965). The degree of handicap was assessed with the Rankin Scale (Rankin 1957), and the severity of intellectual deterioration with the Mini Mental State Examination (MMSE) (Folstein et al. 1975). The characteristics of the patients by SSS, BI, and the Rankin Scale in the acute phase in individual studies are presented in Table 3. Computed tomography (CT) or magnetic resonance imaging (MRI) of the brain was performed on all the patients on admission to the hospital and visualized actual brain infarct pathology in 74 (70%) of the patients (I-III).
4.2.2. Psychiatric examination

A psychiatric examination was performed on all the patients at the 3-month and 12-month follow-up visits. Depression was evaluated according to the criteria of DSM-III-R (American Psychiatric Association 1987) and graded as absent, minor or major. The operatively defined term “minor depression” was used for dysthymic disorders ignoring the 2-year criterion of DSM-III-R classification. The interviews were performed by the same psychiatrist, experienced with psychiatric disorders in stroke and other somatic diseases, always at the same time of the day. When appropriate, e.g. in aphasic patients, additional information from family members and staff was used to supplement patients’ interviews. (I-III) In study IV the degree of depression of the patients was scored using the Zung Self-rating Depression Scale (ZDS) (Zung 1965) at 2 and 6 months post-stroke.

4.2.3. Neuropsychological examination (I-II)

The patients underwent a neuropsychological examination at the 3-month and 12-month follow-up visits using the same test battery in standardized conditions. Parallel test versions were used when available. Five patients, 2 of them aphasic, at the 3-month visit, and one patient at the 12-month visit could not participate in the neuropsychological examination due to poor general condition. The test battery included tests of intellectual functioning (5 subtests of the Wechsler Adult Intelligence Scale – Revised, the WAIS-R (Wechsler 1981)), memory (2 subtests of the Wechsler Memory Scale, WMS (Wechsler 1954) the serial learning and interference tasks (Christensen 1975) and the visual recognition memory task (Cronholm & Ottosson 1963)), attention and executive functions (the Trail-Making Test A and the verbal fluency (Lezak 1995)) and visuoconstructive functions (copy tasks and modified clock hand task (Christensen 1975)).

<table>
<thead>
<tr>
<th>Study</th>
<th>SSS (range)</th>
<th>BI (range)</th>
<th>Rankin (range)</th>
<th>MMSE (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I-II</td>
<td>45 (6-58)</td>
<td>68 (0-100)</td>
<td>4 (1-5)</td>
<td>26 (16-30)†</td>
</tr>
<tr>
<td>III</td>
<td>47 (18-58)</td>
<td>75 (5-100)</td>
<td>4 (1-5)</td>
<td>26 (16-30)‡</td>
</tr>
<tr>
<td>IV*</td>
<td>44 (19-58)</td>
<td>80 (40-100)</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

The values are median (range). *2 months post-stroke. † could not be carried out in 34 patients. ‡ Could not be carried out in 16 patients.

Table 3. Characteristics of the patients in the acute phase in individual studies.
4.2.4. Evaluation of verbal functions (I-II)

The presence of aphasia was evaluated during the first week after the onset of symptoms and at 3 and 12 months after the stroke. Aphasia was assessed using the aphasia quotient of the Western Aphasia Battery (WAB) (Kertesz 1982) and graded as severe (scores 0-39), moderate (scores 40-79), mild (scores 80-93.7) or no aphasia (scores 93.8-100), and classified as non-fluent aphasia including global, Broca’s, isolation and transcortical motor aphasias or as fluent aphasia including Wernicke’s, transcortical sensory, conduction and anomic aphasias.

4.2.5. Evaluation of quality of life (III)

The Finnish version of the RAND 36-Item Health Survey 1.0 (RAND-36) (Aalto et al.) was used as a measure of QOL. The questionnaire was administered by a personal interview when appropriate. The Rand-36 comprises 8 separate domains: Physical functioning, Role limitations due to physical problems, Mental health, Role limitations due to emotional problems, Vitality, Social functioning, Bodily pain, and General health. Scores for the 8 subscales range from 0 to 100, with higher scores indicating a better health state. The instrument contains the same questions as the SF-36 scale (Ware & Sherbourne 1992) but the scoring for the General health and Bodily pain subscales differs slightly.

4.2.6. Evaluation of sexual functions (IV)

4.2.6.1. Questionnaire

At 2 months after stroke, all the patients and spouses filled in the questionnaire which included questions concerning their pre- and post-stroke sexual functions and habits, with special reference to libido, coital frequency, erection, ejaculation, vaginal lubrication, orgasm, and satisfaction with their sexual life. Three aphasic patients needed the assistance of a nurse in filling out the questionnaire at 2 months. At 6 months after the stroke, the same questionnaire was mailed to the patients and their spouses. The patients and spouses were asked to answer the questions separately at home without their spouses being present.

4.2.6.2. Measuring nocturnal penile erection

Nocturnal penile erection of the male patients was evaluated at 2 months post-stroke using an erectiometer attached around the penile shaft at the tip just behind the glans (Karacan et al. 1978). The erectiometer is a calibrated band with a sliding collar, which determines the spontaneous increase in the penile shaft circumference during erection.
The increase of penile shaft circumference was measured during 3 consecutive nights, and the maximum value of the 3 measurements was included in the analysis. The patients were divided into 3 groups according to their erectile capacity (Karacan et al. 1978, Leyson & Powell 1982, Lamid 1985): Group 1 – patients with a penile expansion of 16 mm or more (normal); group 2 – patients with penile expansion between 2 mm and 16 mm (impaired); group 3 – patients with a penile expansion less than 2 mm (no erection).

4.2.7. Statistical methods

SPSS for Windows (Statistical Package for Social Sciences, Inc., Chicago, IL) was used for the statistical analyses. In cross-tabulations Chi-square or, when appropriate, the Fisher’s exact test were used to calculate the statistical significances between the groups (I-IV). The Kruskal-Wallis several independent samples test was used to compare the scores of the SSS, BI, Rankin scale (I-II) and MMSE (I), and RAND-36 subscales (III) in different depression (I-III), and aphasia (II) groups as well as the relationships between the sexual functions and the clinical signs of the patients (IV). The Mann-Whitney two-sample test was used to compare the scores of the non-verbal neuropsychological tests of the aphasic patients with those of the non-aphasic ones (II) and to compare the subscales of RAND-36 according to gender, age, marital status and living conditions (III). The Wilcoxon matched pairs test was used to compare the scores of the SSS, BI, Rankin Scale, and MMSE in the acute phase and at 3 and 12 months post-stroke, and to compare the scores of RAND-36 at 3 and 12 months after the stroke (III), as well as to compare the different variables of sexual function before the stroke and at 2 and 6 months post-stroke (IV).

The statistical significance of the mean values of the neuropsychological test scores between depression groups was evaluated by one-way analysis of variance. The simultaneous effects of depression and aphasia on the neuropsychological test scores were analyzed using two-way analysis of variance. (I)

Logistic regression (Hosmer & Lemeshow 1989) was used to analyze the factors associated with the RAND-36 subscales in order to discover which of these variables best describe and discriminate between the patients with low and high scores on the RAND-36 subscales. To identify the best discriminating factors, the logistic regression models were fitted with a stepwise procedure. The final models were reported using adjusted odds ratios (OR) and their 95% confidence intervals.

For the cross-tabulation and logistic regression analysis the continuous variables were dichotomized as follows. A cutoff point of ≥65 years (65 being the mean age of the study sample) was used for age. The domains of QOL were dichotomized into low and high according to the sex and age specific reference values of the Finnish population (Aalto et al.), using the mean as a cutoff point. The BI was dichotomized into patients independent (BI≥85) and dependent (BI<85) in activities of daily living (Kaste et al. 1998). The cutoff point of ≥52 was used in the SSS for mild to moderate symptoms (Kotila et al. 1998), and that of ≥24 in the MMSE for patients without cognitive impairment (Folstein et al. 1975). The Rankin Scale was dichotomized into patients with a good outcome (grades I and II) and those with moderate or poor outcome (grades III through V) (Kotila et al. 1998).
5. Results

5.1. Post-stroke depression

5.1.1. Prevalence and course of post-stroke depression

Depression was diagnosed in 53 (53%) patients at 3 months and in 39 (42%) patients at 12 months after the stroke. According to DSM-III-R criteria (American Psychiatric Association 1987) the disorder was classified as minor in 44 (44%) patients and major in 9 (9%) patients at the 3-month visit. At 12 months after the stroke 24 (26%) patients showed minor and 15 (16%) patients major depression.

Antidepressive medication was used in 19 (36%) of the 53 depressive patients at the 3-month visit, and in 14 (36%) of the 39 depressive patients, and in 4 (8%) of the 53 non-depressive patients at the 12-month visit. One patient with major depression and one patient with no depression used neuroleptic drugs at 3 months, whereas 2 depressive patients used neuroleptic drugs at 12 months. Three depressive patients used minor tranquilizers at 3 months after the stroke.

5.1.2. Correlates of post-stroke depression

The neuropsychological tests in the depressive patients showed statistically significant inferiority (p<0.05) in almost all the areas of cognitive functions in comparison with the non-depressive patients. Only the similarities subtest of verbal logical thinking, the picture completion subtest of non-verbal problem solving, the delayed visual recognition subtest of visual memory and the verbal fluency test of attention and executive functions at 3 months post-stroke did not reach statistical significance. When comparing the simultaneous effect of depression and aphasia on the neuropsychological test scores, the main effect of depression was observed on the tests reflecting non-verbal problem solving (Fig. 1a), verbal and visual memory and attention and psychomotor speed (Fig. 1b) at 12 months post-stroke (Table 4). The main effect of aphasia was statistically significant in all
the tests at 3 and 12 months post-stroke except the tests of non-verbal problem solving at 3 months. There was no statistically significant interaction between depression and aphasia except in the test of delayed visual recognition at 3 months after the stroke.

Gender was not related to the development of depression. The depressive patients were older than the non-depressive ones, the mean age of non/minor/major depressive patients being 62.4/66.3/70.9 years (p=0.046) at 12 months after the stroke. The depressive patients were more dependent in ADL-functions and had more severe impairment and handicap evaluated by the BI, SSS and the Rankin Scale than the non-depressive patients both at 3 (p<0.01) and at 12 months (p<0.001) after the stroke.

Fig. 1. Mean (SD) values of the Picture completion test scores (a) and Trail Making a test (b) the patients by depression groups.

Gender was not related to the development of depression. The depressive patients were older than the non-depressive ones, the mean age of non/minor/major depressive patients being 62.4/66.3/70.9 years (p=0.046) at 12 months after the stroke. The depressive patients were more dependent in ADL-functions and had more severe impairment and handicap evaluated by the BI, SSS and the Rankin Scale than the non-depressive patients both at 3 (p<0.01) and at 12 months (p<0.001) after the stroke.
Table 4. Results of the neuropsychological tests in non-depressive and depressive patients with and without aphasia at 12 months after the stroke.

<table>
<thead>
<tr>
<th></th>
<th>No depression</th>
<th>Minor depression</th>
<th>Major depression</th>
<th>P for main effect*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Aphasia</td>
<td>No aphasia</td>
<td>Aphasia</td>
<td>No aphasia</td>
</tr>
<tr>
<td>n</td>
<td>8</td>
<td>45</td>
<td>6</td>
<td>17</td>
</tr>
<tr>
<td>Verbal logical thinking</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Similarities† (max.34)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>10.3 (9.3)</td>
<td>22.0 (6.5)</td>
<td>12.8 (9.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No aphasia</td>
<td>5.7 (8.8)</td>
<td>19.3 (5.6)</td>
<td>9.0 (11.9)</td>
<td>0.052</td>
</tr>
<tr>
<td>Comprehension† (max.38)</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>12.8 (9.2)</td>
<td>26.8 (4.8)</td>
<td>26.8 (4.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No aphasia</td>
<td>9.0 (11.9)</td>
<td>24.6 (5.1)</td>
<td>24.6 (5.1)</td>
<td>0.052</td>
</tr>
<tr>
<td>Non-verbal problem solving</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Picture completion† (max.22)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>11.1 (4.3)</td>
<td>13.3 (4.1)</td>
<td>8.0 (5.8)</td>
<td>0.002</td>
</tr>
<tr>
<td>No aphasia</td>
<td>9.3 (4.4)</td>
<td>11.2 (4.9)</td>
<td>8.0 (9.6)</td>
<td>0.011</td>
</tr>
<tr>
<td>Block design† (max.51)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>14.8 (10.1)</td>
<td>35.0 (7.4)</td>
<td>8.0 (5.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No aphasia</td>
<td>12 (10.1)</td>
<td>13.4 (5.4)</td>
<td>10.3 (10.6)</td>
<td>0.001</td>
</tr>
<tr>
<td>Verbal memory</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Logical memory (delayed)‡ (max.23)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>3.0 (3.2)</td>
<td>8.6 (4.3)</td>
<td>3.0 (3.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No aphasia</td>
<td>1.7 (2.7)</td>
<td>7.0 (4.6)</td>
<td>1.7 (2.7)</td>
<td>0.007</td>
</tr>
<tr>
<td>Serial learning (max.50)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>14.8 (10.1)</td>
<td>35.0 (7.4)</td>
<td>19.2 (10.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No aphasia</td>
<td>12 (10.1)</td>
<td>13.4 (5.4)</td>
<td>12 (10.1)</td>
<td>0.001</td>
</tr>
<tr>
<td>Visual memory</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visual reproduction‡ (max.14)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>5.3 (3.7)</td>
<td>8.6 (3.6)</td>
<td>5.3 (3.7)</td>
<td>0.007</td>
</tr>
<tr>
<td>No aphasia</td>
<td>5.3 (2.9)</td>
<td>7.1 (4.3)</td>
<td>5.3 (2.9)</td>
<td>0.007</td>
</tr>
<tr>
<td>Visual recognition (delayed) (max.30)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>14.5 (10.6)</td>
<td>22.5 (6.1)</td>
<td>19.2 (10.1)</td>
<td>0.001</td>
</tr>
<tr>
<td>No aphasia</td>
<td>12 (10.1)</td>
<td>20.9 (5.2)</td>
<td>12 (10.1)</td>
<td>0.001</td>
</tr>
<tr>
<td>Attention and executive functions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trail Making A. s</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>156.5 (104.0)</td>
<td>81.3 (56.3)</td>
<td>156.5 (104.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No aphasia</td>
<td>182.0 (108.0)</td>
<td>111.6 (71.1)</td>
<td>81.3 (56.3)</td>
<td>0.020</td>
</tr>
<tr>
<td>Verbal fluency, words/min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>2.8 (2.4)</td>
<td>11.0 (5.3)</td>
<td>2.8 (2.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No aphasia</td>
<td>1.5 (2.0)</td>
<td>9.8 (4.0)</td>
<td>1.5 (2.0)</td>
<td>0.001</td>
</tr>
<tr>
<td>Visuoconstructive functions (max.19)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>14.4 (5.1)</td>
<td>17.7 (2.0)</td>
<td>14.4 (5.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No aphasia</td>
<td>12.7 (5.9)</td>
<td>16.6 (3.0)</td>
<td>12.7 (5.9)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Values are given as mean (SD). Max. indicates maximum score of the scale. * Statistical significance evaluated by 2-way ANOVA. † Wechsler Adult Intelligence Scale-R subtest, ‡ Wechsler Memory Scale subtest.
5.2. Post-stroke aphasia

5.2.1. Prevalence and evolution of aphasia

Aphasia was diagnosed in 36 (34%) patients in the acute phase, in 27 (27%) at 3 months and in 21 (23%) patients at 12 months after the stroke using the WAB criteria (Kertesz 1982). Eighty-four percent of the patients who were aphasic in the acute phase, remained so at 3 months post-stroke, half of them having moderate or severe aphasia. At the 12-month follow-up visit, 68% of the patients with aphasia in the acute phase were still aphasic, 62% of them having moderate or severe disorder. All the aphasic patients received speech therapy during the follow-up.

Table 5 shows the evolution and recovery of aphasia from the acute phase to 3 and 12 months post-stroke. Evolution of aphasia from one type to another or total recovery was observed in 16 (52%) of the 31 patients at 3 months post-stroke, and in 7 further patients at 12 months after the stroke.

<table>
<thead>
<tr>
<th>Acute phase</th>
<th>N</th>
<th>3 months</th>
<th>N</th>
<th>12 months</th>
<th>N</th>
<th>Recovery %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Global</td>
<td>8</td>
<td>Global</td>
<td>3</td>
<td>Global</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Wernicke's</td>
<td>3</td>
<td>Wernicke's</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Broca's</td>
<td>1</td>
<td>Broca's</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>No aphasia</td>
<td>1</td>
<td>No aphasia</td>
<td>1</td>
<td>13%</td>
</tr>
<tr>
<td>Broca's</td>
<td>4</td>
<td>Broca's</td>
<td>2</td>
<td>Broca's</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Conduction</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anomic</td>
<td>2</td>
<td></td>
<td></td>
<td>50%</td>
</tr>
<tr>
<td>Transcortical motor</td>
<td>2</td>
<td>Anomic</td>
<td>2</td>
<td>Anomic</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Wernicke's</td>
<td>9</td>
<td>Wernicke's</td>
<td>4</td>
<td>Wernicke's</td>
<td>2</td>
<td>50%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Conduction</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Transcortical Sensory</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Conduction</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anomic</td>
<td>4</td>
<td>Anomic</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>No aphasia</td>
<td>2</td>
<td></td>
<td>22%</td>
<td></td>
</tr>
<tr>
<td>Transcortical sensory</td>
<td>1</td>
<td>Anomic</td>
<td>1</td>
<td>Anomic</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Anomic</td>
<td>7</td>
<td>Anomic</td>
<td>6</td>
<td>Anomic</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>No aphasia</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>No aphasia</td>
<td>1</td>
<td>No aphasia</td>
<td>1</td>
<td>57%</td>
</tr>
<tr>
<td>Total</td>
<td>31</td>
<td></td>
<td></td>
<td></td>
<td>32%</td>
<td></td>
</tr>
</tbody>
</table>
5.2.2. Post-stroke depression in aphasic patients

At 3 months after the stroke 16 (59%) of the 27 aphasic patients had minor depression and 3 (11%) major depression as compared with the 74 non-aphasic patients of whom 28 (38%) had minor depression and 6 (8%) major depression (p=0.092, Fig. 2a). At 12 months the number of aphasic patients with minor depression was 6 (29%) and that with major depression 7 (33%) as compared with the 71 non-aphasic patients of whom 18 (25%) had minor depression and 8 (11%) major depression (p=0.033, Fig. 2b). Major depression was diagnosed in 2 (33%) of the 6 patients with non-fluent aphasia at 3 months, and in 2 (50%) of the 4 patients with non-fluent aphasia at 12 months post-stroke.

Fig. 2. The presence and severity of depression in patients with aphasia (shaded bar) and without aphasia (open bar) at 3 (a) and 12 (b) months after the stroke. p=0.092 at 3 months and p=0.033 at 12 months after the stroke using the Chi-square test.

5.2.3. Other correlates of post-stroke aphasia

In the acute phase the patients with severe aphasia were more dependent in ADL-functions (p=0.005) and had more severe impairment (p<0.001) and handicap (p=0.017) evaluated by the BI, SSS and the Rankin Scale than the patients without aphasia or with mild or moderate aphasia. The depressive aphasic patients were more dependent in ADL-functions (p=0.009) and had more severe impairment (p=0.032) and handicap (p=0.005) than the non-depressive ones at 12 months post-stroke. There were no significant differences in gender or age between the patients with and without aphasia.
Through the follow-up period the non-verbal neuropsychological test scores in the aphasic patients were lower than those of the patients who had dominant hemisphere lesion without aphasia (Table 6). When the patients with severe comprehension deficits (global, Wernicke’s, transcortical sensory aphasia) were excluded, and the non-verbal neuropsychological test scores were compared with those of the patients who had dominant hemisphere lesion without aphasia, the aphasic patients had lower scores in all the 3 tests reflecting visual memory (p<0.05) and in the test of visuoconstructive functions (p<0.05) at 3 months. There were no significant differences in the neuropsychological test scores between the aphasic patients with minor or major depression and the aphasic patients without depression.

Table 6. Non-verbal neuropsychological test scores in patients with a dominant hemisphere lesion with and without aphasia at 3 and 12 months after stroke.

<table>
<thead>
<tr>
<th>Neuropsychological test</th>
<th>3 mo Aphasia (n=25)</th>
<th>12 mo Aphasia (n=21)</th>
<th>3 mo No aphasia (n=21)</th>
<th>12 mo No aphasia (n=25)</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-verbal problem solving</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Picture completion† (max.22)</td>
<td>3 mo</td>
<td>9 (6-12)</td>
<td>12 (10-15)</td>
<td>0.004</td>
<td></td>
</tr>
<tr>
<td></td>
<td>12 mo</td>
<td>7 (6-12)</td>
<td>13 (11-16)</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td>Block design† (max.51)</td>
<td>3 mo</td>
<td>3 (1-16)</td>
<td>19 (11-23)</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>12 mo</td>
<td>3 (0-12)</td>
<td>16 (7-23)</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td>Visual memory</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visual reproduction‡ (max.14)</td>
<td>3 mo</td>
<td>3 (1-4)</td>
<td>6 (4-9)</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>12 mo</td>
<td>5 (1-8)</td>
<td>8 (6-10)</td>
<td>0.006</td>
<td></td>
</tr>
<tr>
<td>Visual recognition (immed.) (max. 30)</td>
<td>3 mo</td>
<td>20 (0-25)</td>
<td>26 (25-28)</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>12 mo</td>
<td>22 (4-26)</td>
<td>26 (24-28)</td>
<td>0.014</td>
<td></td>
</tr>
<tr>
<td>Visual recognition (delayed) (max. 30)</td>
<td>3 mo</td>
<td>15 (0-23)</td>
<td>24 (23-27)</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>12 mo</td>
<td>16 (0-24)</td>
<td>23 (20-27)</td>
<td>0.011</td>
<td></td>
</tr>
<tr>
<td>Visuoconstructive functions (max. 19)</td>
<td>3 mo</td>
<td>15 (8-18)</td>
<td>18 (17-19)</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>12 mo</td>
<td>15 (7-18)</td>
<td>18 (16-19)</td>
<td>0.009</td>
<td></td>
</tr>
</tbody>
</table>

Values are medians (quartiles). * p Values for difference between the 2 groups using the Mann-Whitney two independent samples test. † Wechsler Adult Intelligence Scale-R subtest, ‡ Wechsler Memory Scale subtest

5.3. Post-stroke quality of life

5.3.1. Domains and determinants of quality of life

All the dimensions of QOL except Mental health were found to be low in the patients with mild to moderate consequences of stroke both at 3 and 12 months post-stroke (Table 7). When compared to the age and sex specific reference values of the Finnish general population (Aalto et al.), the domains most often impaired were Physical functioning, Physical role limitations, Vitality and General health. Only the domains of Physical functioning and Physical role limitations improved between 3 and 12 months post-stroke.
Table 7. Scores for RAND-36 subscales of the patients at 3 and 12 months after stroke and those of the general Finnish population (Aalto et al.).

<table>
<thead>
<tr>
<th>Subscales</th>
<th>3 months N=85</th>
<th>12 months N=76</th>
<th>General population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical functioning</td>
<td>46.4 (31.9)</td>
<td>50.6 (34.8)*</td>
<td>84.9 (20.1)</td>
</tr>
<tr>
<td>Physical role limitations</td>
<td>25.6 (36.2)</td>
<td>37.2 (42.3)*</td>
<td>74.8 (35.5)</td>
</tr>
<tr>
<td>Mental health</td>
<td>75.6 (20.0)</td>
<td>75.3 (21.0)</td>
<td>73.7 (19.7)</td>
</tr>
<tr>
<td>Emotional role limitations</td>
<td>61.1 (43.9)</td>
<td>68.0 (39.8)</td>
<td>75.0 (36.4)</td>
</tr>
<tr>
<td>Vitality</td>
<td>57.5 (22.2)</td>
<td>55.7 (23.3)</td>
<td>64.0 (22.4)</td>
</tr>
<tr>
<td>Social functioning</td>
<td>76.3 (27.7)</td>
<td>78.1 (24.6)</td>
<td>82.1 (23.2)</td>
</tr>
<tr>
<td>Bodily pain</td>
<td>68.4 (26.9)</td>
<td>71.4 (27.6)</td>
<td>76.2 (24.0)</td>
</tr>
<tr>
<td>General health</td>
<td>51.1 (19.4)</td>
<td>55.8 (21.2)</td>
<td>65.0 (19.8)</td>
</tr>
</tbody>
</table>

Values are means (SD).* p<0.05 when comparing the scores at 3 months with those at 12 months post-stroke using the Wilcoxon matched pairs test.

Depression correlated significantly with various dimensions of QOL both at 3 and 12 months post-stroke (Fig. 3). Table 8 shows the frequency distributions at 12 months post-stroke of the main RAND-36 subscales affected by depression and sociodemographic variables. There was a significant impairment for higher age on the Mental health scale (p=0.046) and slight impairment on the Vitality scale (p=0.065) at 12 months post-stroke. There were no statistically significant differences in the scores of the subscales between women and men, but when comparisons were made with the sex and age specific reference values of the general population (Aalto et al.), men at 12 months post-stroke had a poorer outcome as measured by Physical role limitations (p=0.039) and Vitality (p=0.019). Being married correlated significantly with low scores on the Physical role limitations (p=0.043), Vitality (p=0.005) and Emotional role limitations subscales (p=0.010) one year post-stroke. When comparing the married patients with those who were not married, but were living with their family, the married patients also had a poorer outcome in the domains of Mental health (p=0.019) and General health (p=0.021). There were no differences in the domains of QOL between married men and married women, or between married and unmarried men.
Fig. 3. Median RAND-36 score profiles for patients without depression and with minor or major depression at 12 months after stroke. P-values for difference between the depression groups using the Kruskal-Wallis test for several independent samples.

The SSS correlated only with the Physical functioning (p<0.001) and Physical role limitations (p=0.002) subscales at 3 and 12 months, and the BI with the Physical functioning (p=0.016) and the Social functioning (p=0.036) scales at 3 months after the stroke. The Rankin Scale was a function of Physical functioning (p<0.001) and Physical role limitations (p=0.002) at 3 and 12 months post-stroke, and of General health (p=0.022) at 3 months post-stroke. There was no correlation between the scores of the RAND-36 subscales and lateralization of the hemispheric lesion, cognitive impairment measured by the MMSE, or living conditions.

The main domains which were affected, Physical role limitations and Vitality, were selected for stepwise logistic regression analysis. The analysis revealed that depression and being married were the most important variables when discriminating between the low scores and the high scores of Physical role limitations (Table 9). Low Vitality was related to being married, depression and old age.
Table 8. Frequency (percentage) distributions at 12 months post-stroke of the most important RAND-36 subscales affected by depression and sociodemographic variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Physical role limitations</th>
<th>General health</th>
<th>Vitality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low (N %)</td>
<td>High (N %)</td>
<td>Low (N %)</td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>26 (54)</td>
<td>25 (46)</td>
<td>18 (38)</td>
</tr>
<tr>
<td>Yes</td>
<td>25 (89)</td>
<td>3 (11)</td>
<td>22 (79)</td>
</tr>
<tr>
<td>p</td>
<td>0.002</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;65</td>
<td>18 (64)</td>
<td>10 (36)</td>
<td>16 (57)</td>
</tr>
<tr>
<td>65 or greater</td>
<td>33 (69)</td>
<td>15 (31)</td>
<td>24 (50)</td>
</tr>
<tr>
<td>p</td>
<td>0.689</td>
<td>0.547</td>
<td>0.065</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>16 (53)</td>
<td>14 (47)</td>
<td>13 (43)</td>
</tr>
<tr>
<td>Male</td>
<td>35 (76)</td>
<td>11 (24)</td>
<td>27 (59)</td>
</tr>
<tr>
<td>p</td>
<td>0.039</td>
<td>0.190</td>
<td>0.019</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unmarried</td>
<td>11 (50)</td>
<td>11 (50)</td>
<td>8 (36)</td>
</tr>
<tr>
<td>Married</td>
<td>40 (74)</td>
<td>14 (26)</td>
<td>32 (59)</td>
</tr>
<tr>
<td>p</td>
<td>=0.043</td>
<td>0.070</td>
<td>0.005</td>
</tr>
</tbody>
</table>

Significances between groups were evaluated with the Chi-square test.
Table 9. The most essential variables for probability of low Physical role limitations and Vitality subscale scores using logistic regression analysis at 12 months after the stroke. The adjusted odds ratios and the 95% confidence intervals (CI) are given.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Physical role limitations</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>Odds ratio</td>
<td>95% CI</td>
<td>p-value to remove*</td>
<td>Odds ratio</td>
<td>95% CI</td>
<td>p-value to remove*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>No</td>
<td>1</td>
<td>1</td>
<td>Yes</td>
<td>9.1</td>
<td>2.2-37.7</td>
<td>6.4</td>
<td>1.7-23.1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>&lt;65</td>
<td>1</td>
<td>0.016</td>
<td>≥65</td>
<td>4.1</td>
<td>1.2-13.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Marital status</td>
<td>Unmarried</td>
<td>0.018</td>
<td>&lt;0.001</td>
<td>Married</td>
<td>4.1</td>
<td>1.2-13.6</td>
<td>11.1</td>
<td>2.7-45.5</td>
<td></td>
<td></td>
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</tbody>
</table>

* p-value of difference of deviance test to confirm the variable as an appropriate factor affecting the low Physical role limitations or Vitality scores. The other variables considered (lateralization of lesion, MMSE, living alone) did not change or improve these models.

5.3.2. Sexual function after stroke

Thirty (60%) patients at 2 months and 21 (49%) at 6 months reported their libido unchanged after the stroke when compared to libido before stroke, whereas 19 (38%) patients at 2 months and 22 (51%) patients at 6 months considered their libido diminished or totally absent (p<0.01). Coital frequency of the stroke patients was significantly (p<0.001) decreased both at 2 and 6 months post-stroke compared with the frequency before the stroke. Fourteen (28%) patients at 2 months and 6 (14%) patients at 6 months post-stroke reported that they had ceased having sexual intercourse.

Table 10 presents the effects of stroke on the penile erection and ejaculation of the male patients and on the vaginal lubrication of the female patients. The majority of male patients at 2 and 6 months had diminished ability to achieve an erection and ejaculation. Female patients frequently had problems with vaginal lubrication and orgasmic ability. Four (30%) female patients at 2 months and 2 (20%) patients at 6 months were unable to achieve orgasm. The difference to the pre-stroke orgasmic ability was statistically significant (p<0.05 at 2 months, p<0.01 at 6 months after the stroke).

Most of the patients (90%) had been moderately or very satisfied with their pre-stroke sexual life, and none of them reported complete dissatisfaction. At 2 months post-stroke 40% of the patients and at 6 months 42% of the patients (p<0.001 compared to pre-stroke frequency) were moderately or completely dissatisfied with their sexual life. The main subjective reasons for diminished post-stroke sexual activity were hemiplegia (55% of the patients at 6 months), spasticity (29%), decreased libido (26%), fear of another stroke (24%) or of impotence (14%), sensory deficits (19%), and aphasia (14%).
In the measurements of nocturnal penile erection, 17 (45%) of the 38 male patients showed normal erectile capacity, and 21 (55%) patients showed impaired erections with penile expansions less than 16 mm, but none of the patients had no nocturnal erections. Twenty-one (57%) of the 38 male patients at 2 months, and 19 (54%) of the 33 patients at 6 months had also recognized their nocturnal erections themselves.

Post-stroke coital frequency was significantly (p=0.026) lower in patients with dominant hemisphere stroke than in patients with non-dominant hemisphere stroke. The frequency was also more decreased in the patients with hemispheric stroke than in the patients with brainstem or cerebellar stroke (p=0.033). The patients with sensory deficits had markedly more sexual dysfunction than the patients with unaffected sensation; they had significantly decreased libido (p=0.016), erectile functions (p=0.001), and orgasmic ability (p=0.011), and they were also more often dissatisfied with their sexual life (p=0.025). Changes in sexual functions were related neither to the scores of the SSS and BI, nor to cognitive deficits.

Mild depression (ZDS score between 50-57) was found in 17 (34%) of the patients, moderate depression (ZDS 60-69) in 7 (14%) patients, and severe depression (ZDS more than 70) in 2 (4%) patients at 2 months post-stroke. At 6 months after the stroke mild depression was found in 11 (26%) patients, moderate depression in 8 (19%) patients, and severe depression in 1 (3%) patient. The degree of depression was not related to any of the parameters of sexual dysfunction.

Twenty (50%) of the 40 spouses of the stroke patients reported no change in their post-stroke libido at 6 months compared with their pre-stroke libido, 11 (28%) spouses reported diminished libido, 6 (15%) markedly diminished libido, and 3 (18%) an absence of libido. The satisfaction of the spouses with their sexual life declined significantly (p<0.001 at 2 and 6 months) after their partner’s stroke, 6 (13%) of the 48 spouses having been moderately or completely dissatisfied with their sexual life before the stroke, and 12 (25%) and 13 (36%) of the 39 spouses being so at respectively 2 and 6 months post-stroke.
6. Discussion

6.1. General aspects

Improving QOL of stroke patients has received increasing interest during the last decade. QOL is difficult to measure objectively, but from the patients point of view, it is the most important indication of outcome (Kaste et al. 1998). Although there is an association between neurological deficits and QOL, they are not directly related. Therefore, the assessment of QOL should be multidimensional, including physical, social and role functioning, mental health and general health perceptions (Ware & Sherbourne 1992, de Haan et al. 1993).

6.2. Post-stroke depression

6.2.1. Prevalence and course of post-stroke depression

In the present study the prevalence of post-stroke depression was high, more than half of the ischemic stroke patients having depression at the 3-month follow-up visit and almost half of them at the 12-month visit. The results also showed that the proportion of patients suffering from major depression increased from 9% to 16% during the follow-up. In other studies using psychiatric examinations in diagnosing depression, the prevalence of PSD has varied from 24% to 41%, major depression occurring in 12-31% of patients and minor depression in 9-29% of patients, depending on the time elapsed after stroke (Åström et al. 1993, Burvill et al. 1996, Pohjasvaara et al. 1998). Robinson et al. (1987) found a stable 14% prevalence of depression up to 2 years in a subsample of their original group. In the study of Åström et al. (1993) the majority of patients with depression experienced remission within the first year, the prevalence of major depression decreasing from 31% at 3 months to 16% at 12 months after the stroke.

The occurrence of depression in the present series was even higher than in most of the previous studies, but the prevalence of major depression was lower (Morris et al. 1992, Åström et al. 1993, Herrmann et al. 1998). The differences in the occurrence of major
depression may be due to the selection of the study population. Contrary to those of the previous reports the present patients had experienced only their first-ever stroke and the patients with other central nervous system lesions, as well as with previous psychiatric illnesses were excluded. The increase in the prevalence of major depression during the follow-up may be due to the fact that patients with limited awareness of their deficits avoid depression at the acute stage. Eventually they have to face the demands of everyday life with the loss of cognitive, verbal and functional abilities, and this may contribute to the development of a depressive mood.

6.2.2. Neuropsychological and clinical correlates of post-stroke depression

A clear-cut association between the categories of depressive illness and the degree of cognitive deficits assessed by a pattern of standardized neuropsychological tests was found at 3 and 12 months post-stroke. Very few previous prospective studies have been carried out using both neuropsychological tests for diagnosing cognitive impairment and psychiatric examinations for diagnosing post-stroke depression. When comparing the simultaneous effect of depression and aphasia on cognitive impairment, depression was an independent correlate at 12 months, of the tests reflecting non-verbal problem solving, memory, and attention and psychomotor speed, but aphasia was associated with all the tests.

Stroke often causes cognitive impairment, the domains most frequently affected being memory, orientation, language and attention, and constructional and visuospatial functions (Tatemichi et al. 1994, Pohjasvaara et al. 1997, Hochstenbach et al. 1998). It is also known that depressive patients without brain damage perform poorly on cognitive tasks, especially those involving memory and concentration (Austin et al. 1992, Veiel 1997). In the study of Austin et al. (1992) the most vulnerable functions in major depression were memory and psychomotor speed. The present findings suggest that the depressive stroke patients also performed poorly in the tests of non-verbal problem solving, which has not been found in the depressive patients without brain damage (Austin et al. 1992).

The presence of PSD was associated with old age in the present study. Previously depression has been found to be frequent in young patients (Neau et al. 1998), while in some studies (Sharpe et al. 1994, Kotila et al. 1998) it has been related to old age. The lack of social support and both functional and cognitive impairment may increase the risk of depressive disorders in the elderly (Sharpe et al. 1994). As has been shown in the previous studies (Sinyor et al. 1986, Parikh et al. 1987, Åström et al. 1993, Sharpe et al. 1994, Herrmann et al. 1998, Kotila et al. 1998, Neau et al. 1998), the depressive patients of the present study were more dependent in the ADL and had more severe impairment and handicap than those without depression both at 3 and 12 months post-stroke.
6.3. Aphasia after stroke

6.3.1. Prevalence and course of post-stroke aphasia

In previous reports the incidence of post-stroke aphasia has varied from 17% to 38% depending on the diagnostic criteria, the time of first evaluation of aphasia and the selection of the study population (Kotila et al. 1984, Reinvang 1984, Wade et al. 1986, Starkstein & Robinson 1988, Parikh et al. 1990, Åström et al. 1993, Pedersen et al. 1995). In the present study one third of the stroke patients were diagnosed as aphasic, two thirds of them having a moderate to severe disorder. The greatest improvement was observed during the first 3 months after the stroke, as shown previously (Kertesz & McCabe 1977, Demeurisse et al. 1980, Kertesz 1984, Kotila et al. 1984, Wade et al. 1986, Pedersen et al. 1995).

Aphasia evolved to less severe aphasia syndromes or recovered completely in 23 (74%) of the present 31 patients, whose verbal function was initially evaluated during the first week after stroke. The frequency of recovery was higher than in previous reports using the WAB as a diagnostic method (Kertesz & McCabe 1977, Siirtola & Siirtola 1985, Pashek & Holland 1988). In one of these studies (Kertesz & McCabe 1977), however, the first evaluation of aphasia was performed during the first six weeks after the onset of stroke and some of the patients may have recovered before the first examination, and in another study (Pashek & Holland 1987) the diagnosis of aphasia in the first weeks was based on non-standardized method, which may have influenced the classification of the aphasia.

6.3.2. Depression in aphasic patients after stroke

The present aphasic patients had a significantly higher prevalence of depression than the non-aphasic ones when a psychiatric evaluation based on the DSM-III-R criteria was used. The frequency of major depression increased during the follow-up, one third of the aphasic patients having major depression at 12 months post-stroke. Another prospective study (Åström et al. 1993) also showed a similar association between the presence of aphasia and major depression, but only up to 3 months. One further study (Robinson & Benson 1981) showed almost 50% prevalence of depression in hospitalised aphasic patients, but the sample was highly selective and a self-rating depression scale was used in diagnosing depression. The present findings indicate that aphasia, being a severely disabling condition, may markedly contribute to the severity and persistence of depression in stroke, and therefore aphasic patients should not be disregarded from the studies of PSD.

Patients with non-fluent aphasia are thought to be at higher risk of suffering depression than patients with fluent aphasia (Benson 1973, Robinson & Benson 1981, Herrmann et al. 1993), caused by their better awareness of their impairments. In the present study there was a tendency towards a higher prevalence of major depression among the patients with non-fluent aphasia, but the number of patients was limited.
6.3.3. Other clinical correlates of post-stroke aphasia

The present patients with severe aphasia had more severe impairment and handicap and were more dependent in ADL-functions than the patients without aphasia or with mild or moderate aphasia. Presumably the patients with severe aphasia had more severe strokes than the patients with mild or moderate aphasia. The depressive aphasic patients had a poorer outcome as measured by the SSS, BI and Rankin Scale than the non-depressive ones. No other studies have shown such a correlation between physical deficits and depression in aphasic patients. In one study (Wade et al. 1986) aphasia was associated with severe physical disability, but the differences in physical performance between depressive and non-depressive patients were not assessed.

It is obvious that aphasic patients perform poorly in verbal neuropsychological tests, but in the present study a clear-cut impairment of the non-verbal neuropsychological tests in the aphasic patients was found compared with the patients with dominant hemisphere lesion without aphasia. Most of the patients with comprehension deficits could not perform the tests, and all of them had moderate or severe aphasia, but the patients without comprehension deficits also performed poorly in the tests of visual memory and visuoconstructive functions at 3 months post-stroke. In previous studies the correlations between non-verbal cognitive impairment and type and severity of aphasia have been controversial. In one study (Kertesz 1979) the patients with poor comprehension performed poorly in a non-verbal test, but the severity of aphasia was not a significant determinant, whereas in another study (Basso et al. 1981) the non-verbal test scores of the aphasic patients were significantly affected by the severity of the aphasia, but not by its type.

The finding of cognitive impairment cannot be explained by the presence of depression, because the non-verbal cognitive functions in the depressive aphasic patients and the non-depressive patients were the same. This is in accordance with the findings of one report (Robinson & Benson 1981) using the MMSE in diagnosing cognitive function, in which no correlation between cognitive impairment and depression in aphasic patients was established. The MMSE, however, is of limited value in aphasic patients, because of its essential dependence on verbal skills (Feher et al. 1992).

6.4. Quality of life after stroke

6.4.1. Domains and determinants of post-stroke quality of life

Assessed with the RAND-36 the domain-specific QOL was clearly impaired in the patients suffering from mild to moderate deficits resulting from stroke. The test domains most often impaired were Physical functioning, Physical role limitations, Vitality and General health, supporting the results of an earlier study (Anderson et al. 1996) using the SF-36 as a measure of QOL. In one study (Duncan et al. 1997) patients with only mild disorders of stroke had lower QOL than the control group, contrary to another report (King 1996) in which patients were coping well with their stroke-related impairments.
In the present report QOL did not improve during the one-year follow-up, although neurological impairment and functional ability did. This agrees with a study (Ahlsiö et al. 1984) using visual analogue scales to measure QOL, but contrasts with another report (Åström et al. 1992), in which QOL improved between 3 and 12 months after stroke possibly due to recovery from major depression.

In the present study depression, although mostly minor, correlated significantly with the impairment of the domains of QOL. The strongest associations were found for the Physical and Social functioning, Vitality and Mental health scales. Vitality was more strongly associated with depression than in another study also using the SF-36 (Anderson et al. 1996), which may reflect some cultural differences in health concepts (Aalto et al.). An interrelationship between depression and QOL has also been noted in some previous reports (Ahlsiö et al. 1984, Niemi et al. 1988, Åström et al. 1992, King 1996, Angeleri et al. 1997), but only one of them (Åström et al. 1992) used a psychiatric examination for diagnosing post-stroke depression. In one study (Viitanen et al. 1988) of long-term survivors of stroke, depression was associated only with low satisfaction with leisure time activities. Although the present patients had mainly mild disorders, it seems that they were unable to cope with the stressful events entailing stroke.

Reports concerning the correlation between neurological impairment, dependency in ADL and QOL seem to have been contradictory, some reports showing a strong association (Ahlsiö et al. 1984, Niemi et al. 1988, Viitanen et al. 1988, Wyller et al. 1997), but others not (King 1996, Wyller et al. 1998). The present findings showed that neurological impairment and dependency in ADL correlated only with the Physical or Social functioning scale scores, and contrast with those of another study (Anderson et al. 1996) using the SF-36. However, in the present study only a few patients had severe neurological impairment or dependency in ADL, and this may have influenced the subjective evaluations in the study. Furthermore, cognitive impairment was not associated with the domains of QOL contrary to the reports (Niemi et al. 1988, Kwa et al. 1996) in which restored cognitive ability was related to better life satisfaction.

The correlation between age and QOL has remained obscure (Ahlsiö et al. 1984, Viitanen et al. 1988, Åström et al. 1992, Wyller et al. 1998). The present study showed an association between advanced age and low scores on the Mental health and Vitality scales. In the stepwise logistic regression analysis an age of more than 65 years was an independent explanatory factor for impaired Vitality, OR being 4.1. Since Vitality reflects the psychological health dimension more than the physical one in the Finnish population (Aalto et al.), the present findings are in line with the observations of Kotila et al. (1998) who reported that the probability of depression increases with advanced age.

Women have been reported (Anderson et al. 1996) to have higher SF-36 scores than men on the Mental health and Social functioning subscales, and this contrast with the present findings with no significant gender differences in the domains of QOL. However, when comparing to the age and sex specific reference values of the general population, the men in the present study had a poorer post-stroke outcome than women as measured by the Physical role limitations and Vitality scales. Most authors have reported that QOL is either independent of gender (Ahlsiö et al. 1984, Kwa et al. 1996) or lower in females (Angeleri et al. 1993, Wyller et al. 1997).
The married stroke patients had low scores on the Physical and Emotional role limitations and Vitality subscales. When comparing the married patients and the patients who were not married but living with their family, the married patients also showed a poorer outcome in the Mental health and General health dimensions of QOL, whereas the unmarried patients were coping well with their impairments. The stepwise logistic regression analysis revealed that being married was the most essential factor of low Vitality (OR 11.1) and, together with depression, of low Physical role limitations (OR 4.1). High levels of social support have previously been associated with a better outcome (Glass et al. 1993, Kotila et al. 1998, Wyller et al. 1998). However, the present finding, although seemingly paradoxical, is understandable. Firstly, spouses may underestimate the need for support of patients with only mild disorders of stroke, as has indeed been suggested in a previous study (Glass et al. 1993). Secondly, stroke may lead to changes in the interaction between spouses and in the roles in the family (Evans et al. 1994, Anderson et al. 1995), and spouses may react by overprotection and overcaring (Anderson et al. 1995). It is also likely that unmarried patients are subjected to a more manifold support regimen than the married ones whose spouses are easily given much responsibility for the patient’s wellbeing. Finally, the willingness and ability of caregivers to support stroke patients appear to have a significant influence on a patient’s emotional reactions, and on the success of rehabilitation (Glass et al. 1993, Anderson et al. 1995). All these aspects suggest that stroke patients and their spouses need more individually tailored, continuous and coordinated information, counseling and support instead of standard approaches.

6.4.2. Sexual dysfunction in stroke patients and spouses

A significant decline in libido, sexual arousal and satisfaction with sexual life was found both in male and female stroke patients and their spouses. The present results are in agreement with previous studies carried out during the last few decades (Bray et al. 1981, Sjögren et al. 1983, Sjögren 1983, Hawton 1984, Monga et al. 1986, Boldrini et al. 1991, Monga & Osterman 1995). Kalliomäki et al. (1961) demonstrated that cerebrovascular accidents may cause diminished libido and decline in the frequency of intercourse. More recently various studies have reported a decline in coital frequency (Sjögren et al. 1983, Boldrini et al. 1991) and problems with sexual arousal (Bray et al. 1981, Sjögren & Fugl-Meyer 1981, Sjögren 1983, Sjögren et al. 1983, Hawton 1984, Monga et al. 1986), such as erection, ejaculation, vaginal lubrication and orgasm.

The frequency of patients who ceased having intercourse was lower than reported previously. Sjögren et al. (1983) reported nonexistent coital activity in 41% of male patients and in 17% of female patients, and Monga et al. (1986) in 64% and 54% of the patients respectively. The different findings of these studies may be due to some variability in the patient groups, i.e., age, previous diseases and pre-stroke sexual habits. It may also reflect different attitudes toward sexuality in different cultures.

The presence of sensory hemisyndrome was associated with decreased libido, erectile and orgasmic disability, and dissatisfaction with sexual life. This agrees with one previous study (Sjögren et al. 1983) indicating that changes in coital frequency were related to the
degree of cutaneous sensibility impairment. It is well known that tactile stimulations are extremely important in sexual arousal and orgasm, and therefore it is obvious that sensory hemisyndrome is related to sexual dysfunction.

Reports concerning the correlation between post-stroke sexual dysfunction and the side of the cerebral lesion are contradictory. Some studies (Kalliomäki et al. 1961, Goddes et al. 1979, Monga et al. 1986) have suggested that lesions of the dominant hemisphere cause more sexual dysfunction, including decreased libido, coital frequency and satisfaction with sexual activity than lesions of the non-dominant hemisphere. However, there are also reports (Sjögren et al. 1983, Boldrini et al. 1991) with no significant correlation between the side of lesion and sexual function. The present findings suggest that post-stroke coital frequency is significantly lower in patients with a dominant hemisphere stroke than in patients with a non-hemisphere stroke. This may be due to the fact that dominant hemisphere lesions cause more verbal deficits, diminished self-esteem and depression than non-dominant lesions (Åström et al. 1993), all eliciting hindrances for sexual activity.

There is general agreement that a lack of nocturnal penile erections indicates an organic etiology for impotence. In the present study 45% of the male patients showed normal nocturnal penile erections according to the previously presented criteria (Karacan et al. 1978, Leyson & Powell 1982, Lamid 1982), 55% showed impaired post-stroke nocturnal erections, and none of the patients a complete absence of nocturnal erections. Moreover, the decrease in the measured nocturnal penile erections was consistent with the decline of erections reported by the patients themselves. Thus, it seems that lesions in the autonomic and limbic nervous systems may also determine the form and quality of the sexual life of stroke patients.

The etiology of sexual dysfunction after stroke seems to be multi-factorial including a variety of physical and psychosocial factors. However, in addition to neurological and cognitive deficits, the quality of sexual life may be impaired by other diseases and medication, fear of a recurrent stroke and fear of impotence or rejection by a spouse, role changes in the family, loss of self-esteem and poor coping skills (Sjögren et al. 1983, Monga et al. 1986, Boldrini et al. 1991, Monga & Osterman 1995). The present results emphasize the importance of sexual counseling for stroke patients and their spouses.
7. Conclusions

The present findings indicate that stroke affects QOL extensively, impairing both its physical and psychosocial domains. Depression, a common companion of stroke, is interrelated with cognitive, communicative and other neurological and functional deficits. The following conclusions can be drawn from the present study:

1. More than half of patients suffer from depression after stroke. Although the disorder is mostly of a minor degree, the frequency of major depression seems to increase during the first year. In addition to neurological and functional impairments, post-stroke depression is associated with cognitive deficits in e.g., memory, non-verbal problem solving, attention and psychomotor speed.

2. One third of stroke patients have aphasia in the acute phase and two thirds of these remain aphasic during the subsequent year. Depression is a common disorder among aphasic stroke patients, more than two thirds of the patients having depression at 3 months post-stoke. The severity of depression seems to increase during the first year. The presence of aphasia increases the liability of non-verbal cognitive deficits.

3. Stroke has a clear-cut negative impact on physical and psychosocial domains of QOL. The low QOL does not improve during the first year after stroke. PSD is the most important determinant entailing low QOL even in patients with only mild to moderate stroke-related deficits. In addition to depression, it is interesting to note that being married also seems to carry a risk of low QOL in post-stroke patients and the present rehabilitation services have not been sufficiently aware of this fact.

4. Sexual dysfunction including decreased libido and sexual arousal, and dissatisfaction with sexual life, seem to be common both in male and female stroke patients and their spouses. One of the major physical factors of sexual dysfunction is stroke-related sensory impairment, but also psychosocial aspects are essential.

The results of the present study emphasize the importance of multidimensional evaluations when considering the QOL of stroke patients, especially of those having aphasia and other cognitive deficits. Such evaluations should not be limited to the acute phase alone. The findings also call for more individually tailored, multifaceted rehabilitative approaches including continuous and coordinated information, counseling and support, to improve the QOL of stroke patients.
8. References


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Original papers