The Prevalence and Associated Factors of Poststroke Depression

A thesis
submitted in partial fulfilment
of the requirements for the Degree
of
Doctor of Philosophy
at the
University of Waikato
by
Shirley Grace Hosking

University of Waikato
1998

Abstract

A group of 103 people over the age of 60 were assessed on three occasions in the first year following their stroke. The stroke patients were assessed to determine the prevalence and associated factors of poststroke depression (PSD) within the first 12 months poststroke. Assessments were conducted at the time of discharge from the hospital (the acute poststroke stage), at 3 months poststroke, and at 12 months poststroke. Thirty-four caregivers of the stroke patients were also assessed at 3 and 12 months poststroke to examine psychological distress in the caregiver. Approximately one third of the stroke patients were depressed at all three stages of the poststroke period. The strongest predictors of PSD at the acute poststroke stage were left hemisphere stroke, and a history of stroke. At 3 months poststroke, the strongest predictors of PSD were a history of stroke, physical impairment, and impairment of simple attention abilities. The strongest predictors of PSD at 12 months poststroke were a history of stroke, and physical impairment. The prevalence of clinically significant symptoms of psychological disorder did not change significantly between 3 and 12 months poststroke. Although changes were evident in the physical and cognitive functioning of individual patients, on average the group demonstrated no significant recovery of physical functioning. With regard to cognitive functioning, on average the stroke patients demonstrated no significant recovery of general intelligence, simple attention abilities, immediate verbal memory, and verbal fluency. However, by 12 months poststroke, as a group the stroke patients demonstrated significant improvement in basic cognitive functioning and delayed verbal memory, although there was significant decline in complex attention abilities. Depressed patients were more physically impaired at 1 month poststroke than non-

depressed patients, although no difference between the groups was evident at 3 and 12 months poststroke. Depressed patients did not demonstrate more cognitive impairment, nor did their levels of physical or cognitive functioning improve less, than non-depressed stroke patients. Similarly, patients who had experienced a previous stroke did not demonstrate more physical or cognitive impairment, nor did their levels of functioning improve less, than patients for whom this was their first stroke. PSD at the acute poststroke stage was not related to mortality within the first 12 months poststroke. At 3 months poststroke, 25% of the caregivers reported clinically significant symptoms of psychological disorder. This prevalence decreased to 19% at 12 months poststroke. Patient depression was related to caregiver psychological distress at 3 months poststroke, although this relationship was not evident at 12 months poststroke. The implications of these findings are discussed.

"Don't wait for the light to appear at the end of the tunnel,

go down there and light the thing yourself."

Author unknown

Acknowledgments

I would like to thank the patients and caregivers involved in this study for their time and effort, and for welcoming me into their homes and lives. Obviously, this study would not have been possible without their input.

Many thanks go to my supervisor Dr Nigel Marsh, whose constant enthusiasm for my research and neuropsychology inspired me at times when I lacked motivation, and whose career advice should hold me in great stead when I finally step out into the big world of neuropsychology.

I would like to thank Dr. Paul Friedman, Director of the Rehabilitation Unit at Waikato Hospital, for his interest in the study, and for being the vital link between the hospital and myself.

Thanks also to the staff at Waikato Hospital, particularly Ward 58, for all their time and help.

Thanks to all those who agreed to the laborious task of proofreading this work. Your courage and tactfulness in delivering feedback is extremely appreciated. Thanks also to those in the Psychology Department (University of Waikato) who provided assistance and encouragement whenever I needed it most.

Many thanks to my family and friends for their support and interest, even when I wasn't the nicest person to be around. Your understanding and encouragement at times of stress was invaluable.

Last, but definitely not least, I would like to thank my husband Rob for his support, encouragement, understanding, and patience, for his ability to say the right things at the right time, and for knowing when it's best not to say anything. This thesis is also a product of his efforts.

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CHAPTER 1

General Introduction

Stroke (or cerebrovascular accident) is a common medical condition, with approximately 200 new cases occurring each year for every 100,000 individuals in the Western population (Anderson, 1992). Unfortunately, the rate of survival from stroke is not extremely high; approximately one third of stroke patients die as a result of stroke, and the majority of deaths occur early in the poststroke period (Anderson, 1992). The incidence of stroke is 30% higher in males than in females, with this gender difference being greatest in people under 65 years of age (Toole, 1989). Older people are more at risk of stroke (Anderson, 1992; Toole, 1989), although generally the relationship between age and incidence of stroke differs according to the type of stroke (Toole, 1989).

There are two main types of stroke, cerebral haemorrhage and cerebral infarct. Cerebral haemorrhage refers to bleeding into the brain, which occurs as a result of the rupture of a blood vessel (Rose, 1990). Cerebral haemorrhage is often caused by high blood pressure, toxins, blood disorders, or defects in cerebral arteries (Kolb & Whishaw, 1990; Toole, 1989). Cerebral infarct is caused by the blockage of an artery (thrombosis), or by embolism. Embolism is the movement of a small semi-blockage, which then blocks smaller blood vessels (Kolb & Whishaw, 1990; Rose, 1990). Although stroke can occur in individuals of any age, cerebral haemorrhage is most common in individuals aged between 60 and 80 years, while cerebral infarct is more common in individuals aged between 40 and 60 years (Toole, 1989).

There are a number of risk factors associated with stroke. The main medical

conditions which can increase the probability of stroke include hypertension, diabetes (Woo, Lau, & Kay, 1992), and cardiovascular disease (Toole, 1989). In addition to these medical conditions, lifestyle choices such as smoking, alcohol abuse, drug abuse, and chronic stress are also associated with an increased risk of stroke (Toole, 1989).

Stroke often results in a number of physical, cognitive, and affective changes in the patient. Of these changes, physical impairment is the most frequently investigated. However, cognitive impairment is also common, and can result in substantial distress for the patient, and the family, particularly when communication abilities are affected (Anderson, 1992).

Emotional changes are also a frequent consequence of stroke (Allman, Hope, & Fairburn, 1992), and poststroke depression (PSD) is considered to be the most common emotional outcome of stroke (Nelson, Cicchetti, Satz, Sowa, & Mitrushina, 1994; Price, 1990). PSD is a phenomenon that is distinct from other affective disorders that are also known to be common sequelae of stroke, such as emotional lability (otherwise known as pathological laughing and crying), apathy, and anxiety (Morris, Robinson, & Raphael, 1993).

The Behaviours of Depression

Although it has been suggested that PSD may differ from psychiatric depression, particularly as the impairments that occur as a result of the brain injury may mask or alter the symptoms of depression (Johnson, 1991), recent research has indicated that PSD is not a unique form of depression (Barry, Phillips, Williams, & Dinan, 1990; Clark & Smith, 1998). The signs of depression remain the same,

regardless of whether they are attributable to some form of impairment, or actual depression. It falls on the examiner to determine whether the signs are a result of impairment, or depression.

Psychologists consider depression to be a syndrome which is composed of many different, and often seemingly unrelated, behaviours. While many of its symptoms may be difficult to define, depression can affect many aspects of functioning. Rehm (1988) outlined four basic categories of symptoms, including: (a) verbal-cognitive symptoms (sad affect, cognitive distortions, changes in cognitive functioning); (b) overt-motor behavioural symptoms (behavioural excesses and/or behavioural deficits); (c) somatic symptoms (physical complaints and disturbances); and (d) interpersonal symptoms (person with depression may be dependent, demanding, manipulative, negative, withdrawn).

Particular behavioural disturbances within certain areas of functioning may be considered necessary symptoms in order to reach a clear diagnosis of depression (Rehm, 1988). These may include suicidal behaviour, and disturbances in the areas of sleeping, eating, working, and sexual behaviour. While some researchers have found that elderly individuals are thought to present with essentially the same symptoms of depression as other age groups (Blazer, 1997), other researchers report that elderly people are less likely to experience lack of self-esteem, but are more likely to present with symptoms of distress and unhappiness (Bieliauskas, 1993). Depressed elderly individuals are also less likely to experience feelings of guilt, and are more likely than younger depressed individuals to report somatic complaints (Davison & Neale, 1990). In addition, seasonal fluctuations in depression and other mood disorders are not common in older persons; in fact, seasonal affective disorder is most common in

women of reproductive age (Eagles, McLeod, & Douglas, 1997).

Early Research on PSD

In one of the first major studies of PSD, Robinson and Price (1982) examined poststroke depressive disorders in 103 stroke patients (mean age = 63 years, *SD* = 11). Not all of these patients had experienced their strokes immediately prior to the initial assessment, as some patients had experienced their strokes up to 15 years earlier. In addition to the initial interviews, 83 of the 103 patients were reassessed at varying stages over a 12 month period. Robinson and Price reported that approximately 30% of the 103 stroke patients were depressed at the initial interview, with 66% of that initial 30% remaining depressed for 7 to 8 months. Only 16% of the 30% were still depressed 9 months after the initial assessment, with no depression being found in the patients re-evaluated 12 months after the initial assessment. They concluded that damage to the left hemisphere resulted in a higher frequency and severity of PSD, and that the severity of depression was most prevalent between 6 months and 2 years poststroke.

In addition to this study, one of the most extensive and widely cited longitudinal studies of poststroke mood disorders was also completed by Robinson and colleagues (Parikh, Lipsey, Robinson, & Price, 1987; Robinson, Bolduc, & Price, 1987; Robinson, Starr, Kubos, & Price, 1983; Robinson, Starr, Lipsey, Rao, & Price, 1984; Robinson, Starr, & Price, 1984) using a further group of 103 stroke patients, with an average age of 59 years (*SD* = 13). The initial evaluation (Robinson et al., 1983) was conducted within 2 weeks following the stroke, and indicated that 47% of the patients were depressed. A total of 27% of the patients presented with symptoms

of major depression. The presence of physical and cognitive impairment were significantly correlated with severity of depression. No significant overall hemispheric differences were found for the prevalence of depression, although patients with lesions in the left frontal lobes were significantly more depressed than patients with lesions in other locations.

In the same study, 61 patients were assessed within 6 months following their stroke, and the results indicated that 60% of the patients had been depressed at some time during the 6 month poststroke period (Robinson, Starr, Lipsey, et al., 1984; Robinson, Starr, & Price, 1984). These researchers also reported a strong relationship between functional physical impairment and severity of depression.

Follow-up assessments were also completed at 12 and 24 months poststroke (Parikh et al., 1987; Robinson et al., 1987). Of the 37 patients seen at 12 months poststroke, 14% had symptoms of major depression, and 19% had symptoms of dysthymic (minor) depression. However, of the patients with major depressive symptoms, only 1 patient had also had symptoms of major depression at the initial evaluation. Therefore, although prevalence rates remained stable across the poststroke intervals, the actual individuals experiencing depression changed.

At 24 months poststroke, 48 patients were reassessed, and the researchers reported no significant changes in the prevalence of depression (Parikh et al., 1987; Robinson et al., 1987). However, as was evident at the 12 month evaluation, the composition of the depressed group had altered, and none of the 10 patients presenting with major depressive disorder at 24 months poststroke had been depressed at the initial evaluation. Although a significant relationship was found between severity of depression and physical impairment at 12 and 24 months

poststroke, the patients with dysthymic depression showed no significant improvement in physical functioning (Robinson et al., 1987). Overall, the researchers reported that the prognosis was least favourable for the patients presenting with dysthymic symptoms, as they showed the least improvement in both depression severity, and physical functioning.

Prevalence of PSD

In addition to, and in discussion of, the Robinson studies, reports have indicated that PSD occurs in 20 - 50% of stroke patients in the first year following the stroke (Andersen, Vestergaard, & Lauritzen, 1994; Andersen, Vestergaard, Riis, & Lauritzen, 1994; Price, 1990; Shima, Kitagawa, Kitamura, Fujinawa, & Watanabe, 1994). Studies have also indicated a possible fluctuation of the prevalence of PSD throughout the poststroke period (e.g., Astrom, Adolfsson, & Asplund, 1993). These fluctuations in the prevalence of PSD are discussed below.

The wide range of reported prevalence rates may occur as a result of a number of methodological differences, such as the involvement of differing age groups in the studies, and/or researchers using different criteria to define the presence, and severity of, the disorder. Stroke patients are often considered depressed as a result of their ratings on various depression measures (Price, 1990; Shima et al., 1994). Many researchers use the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*; American Psychiatric Association) criteria to diagnose PSD (e.g., Astrom, Adolfsson, et al. [1993] use the *DSM-III* [1980]). In the *DSM*, PSD is defined as either major or minor depression (Morris, Robinson, Raphael, Samuels, & Molloy, 1992). However, it is important to use a depression measure suited to the

population being assessed, and it has been argued that the *DSM* may not be the best diagnostic tool to use for assessing PSD, for reasons that will be discussed later (Gainotti, 1992).

The distinction between major and minor depression (whether made on the basis of the *DSM* criteria, or by fulfilling the criteria of other measures) may be responsible for some discrepancy in the reported PSD prevalence rates. For example, Fedoroff, Starkstein, Parikh, Price, and Robinson (1991) reported a 41% prevalence of PSD, and suggested that 10 of their 205 patients were not diagnosed with major depression as the patients did not acknowledge their depressed mood. Inclusion of these 10 additional patients would have increased the reported prevalence of PSD. In addition, House et al. (1991) found that mood disorders in general were evident in their stroke patients during the first 12 months poststroke, although only 2 of the 78 patients were diagnosed with major depression for the entire year. House et al. concluded that major depression is over-emphasised in the literature, and a wider consideration of poststroke mood disorders is needed.

Part of the discrepancy in reported prevalence rates of PSD may also be due to other methodological differences, such as assessments being conducted at different stages of the poststroke period, and whether the patients are assessed more than once during the poststroke period. These methodological differences are noteworthy, as the presence of PSD may fluctuate throughout the poststroke period (Astrom, Adolfsson, et al., 1993; Price, 1990). Therefore, care must be taken in comparing prevalence rates reported in studies, as the time of assessment may vary between studies.

The Course of PSD

Robinson et al. (1987) reported that the prevalence of PSD did not change during the first 2 years poststroke. However, other research (including some reports of the Robinson studies) have indicated that the rate of PSD may not remain constant throughout the poststroke period (Andersen, Vestergaard, Riis, et al., 1994; Astrom, Adolfsson, et al., 1993; Robinson & Price, 1982; Robinson, Starr, & Price, 1984). Andersen, Vestergaard, Riis, et al. (1994) assessed PSD in patients within 7 days of stroke, and proceeded with follow-up assessments at 1, 6, and 12 months poststroke. These authors reported that most cases of depression were evident within the first few months of the poststroke period, with a prevalence of 21% at 1 month poststroke. The assessments completed during the year showed that 25% of those patients previously assessed as being non-depressed displayed symptoms of depression. The rate of new cases of depression occurring at 12 months poststroke was comparable to those cases appearing in a community-based control group of individuals with no history of cerebral disease.

Astrom, Adolfsson, et al. (1993) completed a 3-year longitudinal study of PSD in which 80 stroke patients were initially assessed at the time of discharge from hospital, and again at 3, 12, 24, and 36 months poststroke. Eighty percent of the population involved were presenting with their first stroke. Major depression was diagnosed on the basis of *DSM-III* (1980) criteria. Astrom, Adolfsson, et al. found that the prevalence of major depression at 3 months poststroke was 25%, decreasing to 16% by 12 months poststroke, and increasing to 19% by 2 years poststroke. By 3 years poststroke, the prevalence had increased further, stabilising at around 30%. In contrast to the earlier findings of changes in the composition of the depressed group

within the first 2 years poststroke (e.g., Parikh et al., 1987; Robinson et al., 1987), Astrom, Adolfsson, et al. reported that 7 of the 14 stroke patients who had presented with depressive symptoms were still reporting symptoms of depression at the 3-year follow-up.

Nelson et al. (1994) assessed the emotional changes of 19 male stroke patients (mean age = 64 years, *SD* = 7) at 2 weeks, 2 months, and 6 months poststroke. They reported that at 2 months poststroke, depressed stroke patients with right hemisphere lesions appeared to experience a more rapid decrease of depressive symptoms than depressed stroke patients with left hemisphere lesions. However, at 6 months poststroke patients with right hemispheric stroke exhibited a resurgence of depressive symptoms, while patients with left hemisphere strokes showed steady improvement with no relapse. At 6 months poststroke patients with left hemisphere lesions had lower depression scores than patients with right hemisphere lesions. Despite the small sample size, had the research by Nelson and colleagues continued for a longer duration, their results may have come closer to mimicking those of Astrom, Adolfsson, et al. (1993). Further research using a longitudinal design with repeated assessments is required to provide a clear description of the course of PSD.

Aetiology of PSD

The overall causes of PSD are not easily identified. When considering the aetiological involvement of impairment, the question arises as to whether a stroke patient becomes depressed due to their new physical incapabilities or whether the depression aggravates their disabilities, thereby reducing the effects of the natural recovery process. Although not conclusive, it has been suggested by some researchers

that the latter is the more probable case, as depression is likely to affect not only the natural recovery process, but is also likely to inhibit the process and the benefits of rehabilitation (e.g., Agrell & Dehlin, 1989).

Contrary to this assumption, Stern and Bachman (1991) assessed depressive symptoms in 52 stroke patients (mean age = 66 years, *SD* = 8) who had experienced their strokes between 1 and 4 years earlier, and found that there was no association between PSD and functional impairment, where functional impairment was measured in terms of activities of daily living (ADL), physical strength, and degree of aphasia. Instead, they reported that specific lesion location was more central to PSD, as left parietal/occipital, left inferior frontal, right superior frontal, and right temporal lesions were significantly correlated with depressive symptoms. These researchers emphasised that the relationship involving lesion location is more complex and specific than merely right or left hemisphere lesions being associated with PSD. They suggested that "the ultimate etiology of poststroke mood disorders will reflect a complex interaction of neurochemical, neuroendocrine, metabolic, and cognitive alterations following stroke, as well as differences in both inter- and intrahemispheric cerebral specialization of emotional processing" (p. 355). It follows that at least part of the cause of PSD may be found in the many factors associated with the depression (Price, 1990).

Factors Associated With PSD

As outlined in recent review articles (e.g., Gordon & Hibbard, 1997; Hosking, Marsh, & Friedman, 1996), there are many factors which have been shown to be associated with, and possibly contributing to, the presence of PSD. These include

lesion location, lesion size (volume), demographic characteristics of the patient, the level of social support received by the patient, and the degree of cognitive and physical impairment experienced by the patient. Although these factors are assessed frequently in PSD studies, discrepancies clearly exist with regard to their importance to PSD.

Lesion Location

Brain lesions located in the left hemisphere regions are often associated with the presence of PSD (Astrom, Adolfsson, et al., 1993; Fedoroff et al., 1991; Starkstein et al., 1990). In particular, severe depression has been associated with left anterior lesions (Astrom, Adolfsson, et al., 1993; Heilman, Bowers, & Valenstein, 1993; Price, 1990). Indeed Astrom, Adolfsson, et al. reported that lesion location was the major determinant of PSD, as PSD was three times more prevalent in patients with lesions in the left anterior hemisphere, than in patients with left posterior lesions. In addition, PSD was 10 times more prevalent in patients with left anterior lesions, than in patients with right hemisphere lesions. However, this relationship was significant only in the acute poststroke period, and was not found at or beyond 3 months poststroke.

Fedoroff et al. (1991) examined 205 stroke patients (mean age = 59 years, *SD* = 13) within 2 weeks of hospital admission, and found that left hemisphere stroke was significantly correlated with depressed mood. Starkstein et al. (1990) examined anxiety and depression in 98 stroke patients, comparing four groups of patients; those with major depression only, those with generalised anxiety disorder only, those who presented with both major depression and generalised anxiety disorder, and patients who presented with neither major depression nor generalised anxiety disorder. These

researchers also reported a correlation between left hemisphere lesions and PSD (both with and without anxiety). More specifically, they found that the depression-only group was significantly more likely to have subcortical lesions, while the depressed-anxious group had significantly more cortical lesions.

In a study of 47 stroke patients (aged 24 - 79 years) assessed between 1 week and 2.5 months poststroke, Herrmann, Bartels, Schumacher, and Wallesch (1995) failed to find a relationship between severity of depression (major or minor depression) and hemispheric location of the lesion. However, hemispheric differences were evident as, while nine of the patients with left hemisphere strokes were diagnosed as having major depression, none of the patients with right hemisphere lesions presented with major depression.

Further questions regarding the exclusive role of the left hemisphere in PSD have arisen in other recent studies. For example, Yamaguchi, Kobayashi, Koide, and Tsunematsu (1992) examined regional cerebral blood flow changes in 60 stroke patients (mean age = 64 years, *SD* = 9) at varying poststroke stages (between 2 weeks and 553 days), with a follow-up assessment approximately 14 months after the initial assessment. These researchers found that lesion location was related to PSD at the initial assessment, as patients with left frontal or right parieto-occipital lesions showed a significantly higher frequency of depression than patients with lesions in the opposite locations (i.e., right frontal or left parieto-occipital). However, these differences were not significant at the follow-up evaluation. Regional cerebral blood flow was low in those patients with left frontal or right parieto-occipital lesions, indicating that PSD was related to low regional cerebral blood flow. However, the poststroke assessment intervals used in this study varied. As the prevalence rates of

PSD may vary depending on the stage of the poststroke period being assessed (Astrom, Adolfsson, et al., 1993), all patients involved should have been assessed at similar stages of the poststroke period. Therefore, Yamaguchi et al. (1992) did not take into consideration the possible fluctuations in the prevalence of PSD. In addition, despite the associations found between lesions in the parieto-occipital regions of the right hemisphere and PSD (Yamaguchi et al., 1992), other studies have found no significant relationship between the affected hemisphere and PSD (Grasso et al., 1994; Morris, Raphael, & Robinson, 1992).

Similar to the study by Yamaguchi et al. (1993), Grasso et al. (1994) examined cerebral blood flow in 15 patients with subcortical strokes (mean age = 68 years, *SD* = 10), at an average time poststroke of 2.5 months (*SD* = 2). Although failing to find a significant relationship between lesion location and PSD, Grasso et al. also concluded that low regional cerebral blood flow is related to PSD, but only with regard to blood flow in the mesial temporal cortex of the affected hemisphere. Similarly, lesion location (or volume) was not found to be a significant factor in PSD in a study by Morris, Raphael, et al. (1992), in which they assessed 49 stroke patients (mean age = 67 years, *SD* = 11) between 4 and 8 weeks poststroke, with a follow-up assessment approximately 14 months later. However, Gass and Lawhorn (1991) examined 98 stroke patients (mean age = 55 years, *SD* = 11; mean time poststroke = 5 months, *SD* = 7), and found that significantly more patients with right hemisphere lesions were depressed, although they concluded that any study which finds hemispheric effects are founding their conclusions on small differences, and that PSD may be a common sequelae to stroke regardless of the location of the lesion. In addition, the exclusive importance of the left hemisphere (as opposed to the right

hemisphere) in PSD has been questioned by some researchers (e.g., Heilman et al., 1993; Nelson et al., 1994). It is possible that damage to either hemisphere is related to PSD, but lesions in the left hemisphere produce a different form of depression than lesions in the right hemisphere. To be more precise, lesions in the left hemisphere may result in more overt symptoms of depression than those in the right hemisphere (Gainotti, 1992).

Lesion Volume

Lesion volume is also often considered when researching the presence of PSD. While some studies have found no link between lesion volume and the presence, or severity, of depression (Astrom, Adolfsson, et al., 1993; Grasso et al., 1994; Herrmann et al., 1995; Morris, Raphael, et al., 1992), other studies have reported that patients with larger lesions are more likely to present with PSD (Morris, Robinson, Andrzejewski, Samuels, & Price, 1993; Schwartz et al., 1990; Sharpe et al., 1994). Morris, Robinson, Andrzejewski, et al. examined PSD in 91 stroke patients (mean age = 50 years, *SD* = 13). These researchers reported that the lesion volume of the depressed patients was three times that of non-depressed patients. A relationship between higher rates of PSD and increased lesion volume was also found by Schwartz et al. who used single photon emission computed tomography to measure lesion volume in 14 depressed stroke patients.

Sharpe et al. (1994) examined PSD in a sample of 60 community-based stroke patients, who had experienced their first stroke 3 to 5 years earlier. Although they also reported that large lesion volume was related to higher levels of PSD, they suggested that this relationship may be due to the fact that larger lesions generally result in greater functional impairment, and it is the greater functional impairment

which increases the probability of PSD.

Previous Strokes

Despite the fact that many patients experience more than one stroke (Jorgensen, Nakayama, Reith, Raaschou, & Olsen, 1997), few studies examine the relationship between PSD and a history of previous strokes (hereafter referred to as "previous strokes"). Eastwood, Rifat, Nobbs, and Ruderman (1989) found that, compared with patients who had experienced one stroke only, depression at 3 months poststroke was likely to be higher in patients who had experienced a previous stroke. Similarly, Andersen, Vestergaard, Ingemann-Nielsen, and Lauritzen (1995) found that patients who had experienced a previous stroke reported a higher rate of symptoms of PSD at 12 months poststroke. Although some researchers have failed to find this relationship (e.g., Morris, Raphael, et al., 1992; Morris, Robinson, et al., 1992; Parikh et al., 1990), it does not seem illogical for patients who have had a previous stroke to experience depression. That is, the depression may be a reaction to the subsequent stroke, in that the patient is likely to be more aware of what has happened, and what will happen, to them. They are also likely to be aware that their chances of having an additional stroke is further increased with each subsequent stroke. Andersen et al. (1995) suggested that patients with a history of stroke should be excluded from PSD research, as it may be the inclusion of these patients that leads to the discrepancies in reported results. However, as many patients experience more than one stroke, excluding these patients from studies of PSD results in a sample of stroke patients unrepresentative of the true stroke population (Eastwood et al., 1989).

Demographic Characteristics

Factors such as the age and gender of patients are usually considered during

research into PSD, as are psychosocial factors. As with the factors already discussed, there are obvious discrepancies regarding the findings on the relationships between these factors and the presence of PSD. However, as with the differences between reported prevalence rates of PSD, these discrepancies may be at least in part due to methodological limitations within, and differences between, the studies.

Age and PSD. When considering the contribution of age to PSD, Sharpe et al. (1994) concluded that PSD was more common among patients aged 75 years and over. In a study examining possible risk factors for PSD, Morris, Robinson, et al. (1992) assessed 99 in-hospital stroke patients (mean age = 71 years, *SD* = 10). Amongst other factors, older age was found to be significantly associated with major depression. However, in comparison with the findings of Morris, Robinson, et al., Fedoroff and colleagues (1991) found that the depressed stroke patients were younger on average (mean age = 57 years, *SD* = 12) than those who were not depressed (mean age = 60 years, *SD* = 14).

However, many researchers have failed to find a significant relationship between age and PSD (Andersen, Vestergaard, Riis, et al., 1994; Grasso et al., 1994; Morris, Raphael, et al., 1992; Parikh et al., 1990; Shima et al., 1994). In a study of 285 stroke patients, Andersen, Vestergaard, Riis, et al. (1994) examined the course of PSD for the first year of the poststroke period. Many factors were examined, but age was not found to be significantly related to PSD. In a study of 63 stroke patients which examined the effect of PSD on physical recovery, Parikh et al. (1990) failed to find a significant relationship between age and PSD. In addition, Shima et al. (1994) assessed 68 stroke patients (aged 38 - 94 years) who had experienced their stroke between 3 months and 10 years earlier, in order to examine many possible factors

involved in PSD. These researchers also failed to find a significant relationship between age and PSD. Therefore, although many studies have failed to find a significant relationship between the age of the stroke patient and PSD, the presence of a few significant relationships establishes doubt as to the overall conclusion. Reasons for these discrepancies are not clear, although as with all correlational research, it is possible that the presence (or absence) of a relationship between variables may be more than two-fold, and additional factors may be involved in the relationship.

Gender and PSD. With regard to the relationship between gender and PSD, Morris, Robinson, et al. (1992) reported that minor depression was most common in males. In contrast to these findings, Angeleri, Angeleri, Foschi, Giaquinto, and Nolfe (1993) examined the influence of PSD on poststroke quality of life in 180 stroke patients, and found that women had a significantly higher incidence of PSD. However, as with age, other studies have also reported that gender is unrelated to PSD (e.g., Fedoroff et al., 1991; Grasso et al., 1994; Morris, Raphael, et al., 1992; Parikh et al., 1990; Shima et al., 1994), and, again, the precise reason for these discrepancies is not clear.

Psychosocial Functioning

Psychosocial functioning is often affected following stroke (Astrom, Asplund, & Astrom, 1992), and the effects may be further aggravated by depression (Starkstein et al., 1990). In order to determine the role of social factors, Astrom et al. (1992) examined the course of psychosocial functioning for 3 years following stroke in 50 stroke patients (mean age = 71 years, *SD* = 11). These researchers reported that between 3 and 12 months poststroke, psychosocial functioning improved in most patients (as major depression decreased), but remained lower than that of the general

elderly population. This study also highlighted correlations between poor life satisfaction and a higher frequency of PSD.

Variables such as the living situations of the patients during the studies, (e.g., hospitalised, out-patients, or living in their own home), may influence results. A low prevalence of PSD has been found in patients who are not hospitalised following stroke, while studies reporting high prevalence rates have involved patients who were hospitalised, or in rehabilitation (Johnson, 1991). Further support for the impact of the patient's living situation on the presence of PSD was provided by Sharpe et al. (1994). These researchers found that institutionalisation was closely correlated with greater severity of PSD, as was a lack of personal relationships.

Living alone has been shown to be a predictor of PSD in the early stages following stroke (Astrom, Adolfsson, et al., 1993), although the isolation does not necessarily lead to depression of a greater severity than that found in other stroke patients (Angeleri et al., 1993). Marital status, however, does not appear to be related to PSD (Fedoroff et al., 1991; Greveson, Gray, French, & James, 1991; Morris, Robinson, et al., 1992; Parikh et al., 1990). The study by Greveson et al. differed from most, as in order to assess the long-term outcome for 82 surviving stroke patients, their first evaluation was completed at 3 years poststroke. Although failing to find a significant relationship between marital status and PSD, they did report that PSD was associated with greater physical dependency, and that patients with PSD were most likely to request access to a stroke support club.

In contrast to Greveson et al.'s (1991) findings of desire for social contact (via access to a stroke club), decreased social activities and contact with others following stroke has been associated with PSD (Gainotti, 1992; Price, 1990),

although the precise relationship is not clear. A lack of social contact has been shown to be related to the presence of depression at 12 months poststroke (Astrom, Adolfsson, et al., 1993), in accordance with the theory that depression results in a decrease in social functioning (Price, 1990). In addition, Angeleri et al. (1993) found that female stroke patients tended to demonstrate a greater reduction in social activities and a higher incidence of depression than their male counterparts. Although this does not necessarily indicate a relationship between the two factors, as it is common for women to isolate themselves socially after stroke (Sandin, Cifu, & Noll, 1994).

Physical Functioning

It is usually considered that physical impairment following stroke is related to PSD (Kelly-Hayes & Paige, 1995; Price, 1990; Sharpe et al., 1994). The suggestion that, in many cases, depression occurs as a reaction to the realisation that some ADL are no longer as simple to perform as they were prior to the stroke, is consistent with psychological theories of depression (e.g., Lewinsohn, 1974). However, as mentioned previously, it is not clear whether the depression leads to decreased physical functioning, or whether the impairment leads to, or aggravates, the depression.

While examining the possibility of a causal relationship between physical impairment and PSD, Astrom, Adolfsson, et al. (1993) found that rather than one condition causing the other, it was the interaction of the two conditions which increased the effects of both. However, after finding an association between depression and decreased physical functioning following stroke, Schubert, Taylor, Lee, Mentari, and Tamaklo (1992) suggested a causal relationship, whereby depression results in the deterioration of physical functioning. They reached this

conclusion after completing a study involving 21 stroke patients aged 47 - 72 years. High scores on the Beck Depression Inventory (a self-report depression measure) were associated with lower physical functioning scores and, although the relationship did not reach statistical significance, patients who were categorised as depressed had a slower rate of physical recovery. These researchers suggested that depression may decrease the functional abilities of the stroke patient as it increases fatigue, promotes feelings of hopelessness, and decreases the patient's motivation. While the finding that physical recovery from stroke tends to be slower in depressed patients than in non-depressed stroke patients (Parikh et al., 1990; Schubert, Taylor, et al., 1992) may suggest fruitful avenues for further research, there is currently insufficient evidence to confirm any causal link between physical impairment and PSD.

Activities of daily living (ADL). One particular form of physical functioning affected by stroke is the ability to perform everyday activities, as is understandable considering the likelihood of functional impairment following stroke (Angeleri et al., 1993). When examining the involvement of physical functioning in PSD, some studies examine ADL functioning specifically, or at least refer to the physical functioning as ADL. A variety of results have been reported by researchers examining the association between depression and ADL functioning in stroke patients.

Although Stern and Bachman (1991) failed to find a significant relationship between PSD and ADL, a strong association between these two variables has been reported by other researchers (e.g., Shima et al., 1994; Starkstein et al., 1990). However, both Shima et al. and Starkstein et al. reported that the clinical strength of the relationship between the two variables was questionably low.

In a longitudinal assessment of recovery from stroke, Parikh et al. (1990)

found that, compared with non-depressed patients, depressed stroke patients were more impaired in ADL at 2 years poststroke, although there had been no significant differences in ADL functioning between the two groups while hospitalised in the acute poststroke stage. This is not an infrequent research finding and may be due to the patient's inability to perform daily tasks becoming a reality in the later stages of the poststroke period, thereby demonstrating the interaction between PSD and physical impairment where each factor exacerbates the effects of the other.

Neuropsychological Functioning

Cognitive impairment, as with physical impairment, is a common outcome of stroke, the latter often exacerbating the former (Tatemichi et al., 1994). Tatemichi et al. compared cognitive function in 227 stroke patients at 3 months poststroke, with the cognitive function of 240 control participants. Cognitive function was determined by assessing memory, orientation, verbal abilities, attentional skills, visuospatial competence, and abstract reasoning, with failure on any four of these six items indicating cognitive impairment. These researchers reported that cognitive impairment was evident in 35% of the stroke patients, compared with 4% of the control group. Although establishing a relationship between cognitive impairment and decreased physical functioning following stroke, Tatemichi et al. concluded that cognitive impairment has immense implications with regard to poststroke functioning, independent of any consequences of physical impairment.

Cognitive impairment is also known to occur simultaneously with PSD (Starkstein et al., 1990), and may be more common in stroke patients reporting symptoms of depression (Sharpe et al., 1994). Interestingly, although Starkstein et al. found a relationship between the presence of depression and increased cognitive

impairment (and increased physical impairment), they failed to find a significant relationship between anxiety and cognitive functioning.

Bieliauskas (1993) addressed the relationship between depression and cognitive impairment in the elderly. Bieliauskas stated that cognitive impairment does not occur as a result of depression, but that it is possible for depression to occur as a result of physical impairment and/or a medical condition (such as stroke). These conclusions were reached after studying this relationship in patients with Parkinson's Disease. Bieliauskas found that amongst patients with Parkinson's Disease, it was the patients without cognitive impairment that were likely to be depressed. Findings that link depression with cognitively intact individuals raise another question: is it possible that stroke patients with no cognitive impairment become depressed as they are more aware of what is happening to them as a result of the stroke?

The interaction between PSD and cognitive deficits is frequently not addressed by researchers, though some studies have failed to find any statistically significant relationship between the two factors (e.g., Shima et al., 1994). Although Parikh et al. (1990) reported no significant differences in cognitive functioning between depressed and non-depressed stroke patients, the study did reveal increased language difficulties in depressed patients.

Assessment of PSD

There are a number of evaluation formats used to diagnose and assess the severity of PSD; some are completed by the patient (self-rating), while others are examiner-rated. It has been noted that a fundamental element to successful self-rating scales is that they are kept short, and are able to be completed quickly (Agrell &

Dehlin, 1989). This may in fact be true for all assessment involving self-report procedures.

Neuropsychological Procedures

There are many psychometric tests available for the assessment of depression. Unfortunately, psychometric methods specifically designed to assess depression in the elderly population are limited in number. Such specific tests are necessary, as tests which over-emphasise somatic complaints tend to result in the elderly being misdiagnosed as depressed. To date, the Geriatric Depression Scale (GDS; Yesavage et al., 1983) is the only depression rating scale designed for use specifically with the elderly (Shaver & Brennan, 1991). However, there are many tests designed for use with the adult population which are also deemed useful for the elderly.

In a study of 40 elderly stroke patients (mean age = 80 years), Agrell and Dehlin (1989) found that of six depression rating scales used for assessing PSD, the GDS was one of the more suitable self-rating scales, along with the Zung scale. The most suitable examiner-rating scale was the Comprehensive Psychopathological Rating Scale - Depression. These three scales produced acceptable levels of external and concurrent validity, with high internal consistency, sensitivity, and predictive values.

Agrell and Dehlin (1989) reported that an examination of the internal consistency of the GDS showed that four items did not correlate with the final score, though they suggested that this was due to the population they assessed. Although the use of the Zung scale produced seemingly satisfactory results, it must be noted that item 6 (I still enjoy sex) was replaced with the average score of the remaining questions, as most patients did not answer the question satisfactorily. This may be an

example of the questionable use of general adult depression scales on elderly people, though this was not stated by the researchers, and it cannot be assumed that the problems with item 6 were due to the age of the stroke patients.

Burke, Nitcher, Roccaforte, and Wengel (1992) evaluated the usefulness of the GDS with cognitively impaired elderly people. They assessed 194 elderly patients with regard to their cognitive functioning, and their performance on the GDS as compared to an independent clinical assessment of their depressive state. They reported acceptable correlations between the clinical assessment and the GDS in both cognitively impaired patients and cognitively intact patients.

Gompertz, Pound, and Ebrahim (1993) examined the test-retest reliability of the GDS (amongst other measures) by postal survey of 191 stroke patients at 6 months poststroke. Twenty-one of these patients (mean age = 69 years) completed the GDS again 2 weeks after the initial assessment. Gompertz et al. reported that the GDS had little reliability when re-tested, and suggested that the differences may be due to responses to the more subjective items. However, it is possible that the low test-retest reliability may be due to actual changes in the depression of the stroke patients.

The Zung scale is utilised by many PSD researchers (e.g., Grasso et al., 1994; Parikh et al., 1990; Starkstein et al., 1990) and, in addition to the previously stated evidence of its acceptability for use with stroke patients, is known to have acceptable internal consistency and a split-half reliability of .73 when assessing the general adult population (Fischer & Corcoran, 1994). Also in common usage is the Hamilton Depression Scale (Andersen, Vestergaard, & Lauritzen, 1994; Andersen, Vestergaard, & Riis, 1993; Andersen, Vestergaard, Riis, et al., 1994; Barry et al.,

1990; Fedoroff et al., 1991; Grasso et al., 1994; Starkstein et al., 1990; Stern & Bachman, 1991), and the Beck Depression Inventory (Angeleri et al., 1993; Grasso et al., 1994; House et al., 1991; Nelson et al., 1994), although the reliability and validity of these measures when assessing elderly stroke patients has not been established. At times a combination of these tests is used for assessment (e.g., Grasso et al., 1994; Parikh et al., 1990; Starkstein et al., 1990), as many researchers choose to employ more than one form of assessment to screen for, or diagnose, PSD.

There are other depression instruments that are available, and have been used, to assess depression in the elderly. One of these is the General Health Questionnaire (GHQ; Goldberg, 1972), which does not focus exclusively on depression, and is often used as a measure of symptoms of psychological distress (Goldberg & Hillier, 1979; Lykouras et al., 1996), although it has been described as a suitable measure of depression in stroke patients (Robinson & Price, 1982). The GHQ is available as a full 60-item questionnaire, and also in abbreviated versions of 12-, 28-, and 30-item questionnaires. The GHQ has excellent psychometric properties, and is widely used (McDowell & Newell, 1987). As with many of the other measures of depression, its reliability and validity when used to assess elderly stroke patients are unknown.

Psychiatric Procedures

There are a variety of psychiatric procedures used to assess PSD. This section contains only a brief outline as the present study does not utilise these procedures.

The dexamethasone suppression test is a biological test which has been used to diagnose PSD. Dexamethasone normally suppresses cortisol secretion, and non-suppression has been detected in depressed stroke patients (Astrom, Olsson, & Asplund, 1993). However, it is not always considered an ideal form of assessment,

due to the imprecise nature of the test (Mullen, Linsell, & Parker, 1986; Price, 1990).

Various editions of the *DSM* have also been used in the assessment of PSD, at times as the sole diagnostic tool (e.g., Astrom, Adolfsson, et al., 1993; Morris, Robinson, & Samuels, 1993). As with the psychological forms of assessment, *DSM* has also been used in conjunction with other methods (e.g., Andersen, Vestergaard, Riis, et al., 1994; Morris, Robinson, Andrzejewski, et al., 1993). However, the use of the *DSM* as an evaluative tool for assessing PSD has been questioned due to the other consequences of stroke resulting in symptoms similar to those required to make a *DSM* diagnosis of depression (Gainotti, 1992).

Issues in the Assessment of PSD

There are many variables that can interfere with the evaluation of PSD, which in turn, make conducting a valid assessment difficult. Cognitive impairment can be a barrier to accurate assessment, although as mentioned previously, Burke et al. (1992) evaluated the usefulness of the GDS with cognitively impaired elderly people, and concluded that the GDS was suitable for use with this population. In contrast to this research, McGivney, Mulvihill, and Taylor (1994) assessed the cognitive abilities of 66 nursing home residents (mean age = 83 years, *SD* = 4). These participants also underwent psychiatric assessment and completed the GDS. McGivney et al. reported that the GDS was valid when used on patients with Mini-Mental State Examination (a cognitive screening measure) scores of at least 15, suggesting that cognitive function is an issue in the assessment of PSD. Other researchers have excluded demented patients from their PSD studies due to the difficulty in obtaining accurate assessment using examiner-rated forms of evaluation (Agrell & Dehlin, 1989).

The lack of a clear definition of PSD is also a barrier to adequate assessment.

As mentioned previously, some researchers believe that PSD essentially closely resembles depression as outlined by the *DSM* (e.g.; Barry et al., 1990). However, others believe that depression following stroke may differ from other forms of depression, as the additional impairments produced as a result of the brain injury may mask or alter the symptoms of depression, thus affecting diagnosis (Johnson, 1991).

The effect of aphasia on assessment. Aphasia is also considered to be a factor which interferes with diagnosis of PSD (Gainotti, 1992). Aphasic patients are often excluded from studies of PSD due to the fact that their language impairment can interfere with the accurate assessment of depression, particularly when they are unable to communicate their responses clearly (Andersen, Vestergaard, & Lauritzen, 1994). In accordance with this, researchers of PSD may exclude only those aphasic stroke patients who are unable to either clearly communicate their responses, or to comprehend the questions or instructions given by the assessor (e.g., Andersen, Vestergaard, & Lauritzen, 1994; Andersen, Vestergaard, Riis, et al., 1994).

Despite these difficulties, the relationship between depression and aphasia in stroke patients has been the subject of some research. While it has been suggested that there may not be a relationship between the presence of aphasia and PSD (Stern & Bachman, 1991), other researchers have reported that dysphasic stroke patients have a significantly higher probability of being diagnosed with depression (Astrom, Adolfsson, et al., 1993). In addition, Herrmann, Bartels, and Wallesch (1993) analysed the course of depression in 21 acute, and 21 chronic, aphasic stroke patients. Both groups of participants had single left sided strokes. They concluded that although there is no significant difference in depression ratings between the stages of aphasia, depression was evident, and the group with acute aphasia presented with a

significantly higher frequency of physical symptoms of depression. Herrmann et al. concluded that aphasia is an important outcome of stroke, and therefore stroke patients with aphasia should not be excluded from PSD studies. However, to date, difficulties with the assessment of aphasic stroke patients continue.

The finding that aphasia may produce difficulties in the assessment of PSD needs to be balanced against the fact that aphasia remains a frequent outcome of stroke. Individuals should not be immediately excluded from participation in PSD research because of their language difficulties. It is a matter of clinical judgement in determining whether the communication impairments of the participant are so great that clear assessment is impossible, and that their inclusion may lead to distorted results.

Consequences of PSD

Depression is known to have many consequences for the stroke patient. Leaving aside the debate as to whether physical impairment causes PSD (or the converse), it is clear that general recovery is likely to be affected by PSD (Kelly-Hayes & Paige, 1995; Morris, Raphael, et al., 1992). Due to the possible consequences of these effects, the relationship between PSD and mortality has been the focus of recent research.

Mortality and PSD

In an examination of the relationship between PSD and mortality within 10 years poststroke, Morris, Robinson, Andrzejewski, et al. (1993) found that patients who were depressed at 2 weeks poststroke, were over three times more likely to have died over the following 10 years. Increased mortality in depressed stroke patients has

also been demonstrated over the shorter time period of 17 months poststroke (Morris, Robinson, & Samuels, 1993). In their study, Morris, Robinson, and Samuels assessed 94 stroke patients at 2 months poststroke, and collected information on 84 of these patients 15 months later. Depressed patients were found to be eight times more likely to die prior to follow-up than non-depressed patients. Interestingly, the 7 patients who had died had been more cognitively impaired at the initial evaluation.

Although there may be a relationship between PSD and mortality, it is not necessarily a causal relationship. Other factors such as the side-effects of anti-depressants, tendencies of depressed patients to demonstrate certain behaviours (for example, alcoholism, drug abuse), or their irresponsiveness to instructions regarding treatment, may all have an effect on mortality (Morris, Robinson, & Samuels, 1993). The natural aging process may also be a factor, particularly when considering the 10 year study by Morris, Robinson, Andrzejewski, et al. (1993). Alternatively, if PSD is associated with greater stroke severity, then the stroke severity rather than the presence of PSD may account for the greater mortality of PSD patients.

Caregiver Issues

The overall effects of stroke and their impact on the primary caregiver are often examined, particularly with regard to the psychological reactions of the caregiver (Anderson, 1992; Bethoux, Calmels, Gautheron, & Minaire, 1996; Carnwath & Johnson, 1987; Hodgeson, Wood, & Langton-Hewer, 1996; Tyman, 1994; Wade, Legh-Smith, & Langton-Hewer, 1986). Although a range of prevalence rates are reported, some form of psychological distress is experienced by 10 - 50% of people who care for stroke patients (Bethoux et al., 1996; Carnwath & Johnson,

1987; Hodgeson et al., 1996; Wade et al., 1986).

Although studies have indicated that poststroke emotional problems in the patient do have an impact on caregiver functioning (Draper, Poulos, Cole, Poulos, & Ehrlich, 1992; Greveson et al., 1991; Wade et al., 1986), it has also been suggested that the two factors are unrelated. For example, Thompson, Bundek, and Sobolew-Shubin (1990) interviewed 40 caregivers of stroke patients at approximately 9 months poststroke. The stroke patients were assessed with regard to their cognitive and physical functioning, and depression status. Caregiver burden and depression were also assessed, along with caregiver control, meaning, and hope. The nature of the patient-caregiver relationship was also considered. Thompson et al. reported that the stroke patient's degree of physical functioning had the main psychological effect on the caregiver, as caregivers were more depressed if the patient was severely physically impaired. Caregiver depression was also increased if the caregiver was caring for a family member who had experienced more than one stroke. Caregivers were also more likely to be depressed if they had less hope and/or felt more burdened by caregiving. However, caregiver depression was not found to be significantly correlated with the depression status of the patient.

Despite some studies reporting findings to the contrary (e.g., Anderson, Linto, & Stewart-Wynne, 1995), the patient's level of physical functioning may affect the psychological wellbeing of the caregiver (Carnwath & Johnson, 1987; Thompson et al., 1990; Wade et al., 1986). Wade et al. examined the emotional state of 302 caregivers of stroke patients for the first 2 years poststroke. Physical disability in the patient, including a low level of improvement and a lack of activity, were associated with caregiver depression within the first year poststroke. During the same time

period, depression was also higher in caregivers who were caring for depressed stroke patients. However, none of these factors were associated with caregiver depression at 2 years poststroke, indicating that other factors (such as length of time in the caregiving role) may be associated with, and possibly responsible for, caregiver depression later in the poststroke period.

Carnwath and Johnson (1987) examined the psychological wellbeing of 103 caregivers of stroke patients (aged < 75 years) between 1 and 3 years poststroke. The rate of depression was higher in caregivers of patients who were more impaired as a result of their stroke. In addition, caregiver depression increased with the length of time in the caregiving role.

The role of PSD in affecting caregiver burden following stroke has not been the subject of extensive research. Kausar and Powell (1996) examined the caregivers of neurological patients (of which the majority were stroke patients) to assess the impact of the post-injury difficulties (objective burden) on the subjective burden of the caregivers. These researchers found that subjective burden was related to objective burden, a relationship that increased with time. In addition, psychological problems in the patient were more likely than physical problems to result in caregiver feelings of burden.

The psychological and physical effects of stroke clearly lead to psychological distress in the caregivers of the patients. As the emotional wellbeing of the caregiver has an effect on the general wellbeing of the stroke patient, further work is needed in this area to identify those caregivers at risk, and to acknowledge the difficulties associated with the caregiving role.

Summary and Conclusions

Studies examining PSD have reported a range of results regarding the prevalence and associated factors of PSD. Despite some studies indicating that the prevalence of PSD remains stable during the poststroke period (e.g., Parikh et al., 1987; Robinson et al., 1987), the results of other studies indicate that the prevalence changes throughout the poststroke period (e.g., Astrom, Adolfsson, et al., 1993). Andersen, Vestergaard, Riis, et al. (1994) found that the majority of new cases of depression were evident within the first few months of the poststroke period, with a prevalence of 21% at 1 month poststroke. The assessments completed during the first year after the stroke showed that 25% of those previously assessed as being non-depressed displayed symptoms of depression. Other recent studies have also reported a substantial presence of PSD in the early stages poststroke (e.g., Nelson et al., 1994; Shima et al., 1994). However, the necessity for longitudinal studies of PSD is demonstrated by the results reported by Astrom, Adolfsson, et al. (1993). Astrom, Adolfsson, et al. examined major depression in stroke patients to 3 years poststroke, concluding that the prevalence of major depression fluctuated throughout the poststroke period, reaching as high as 31% at 3 months poststroke. Although the rates of PSD reported by Astrom, Adolfsson, et al. may be considered high as they included measures of major depression only, the prevalence of PSD (regardless of severity) has been reported to be as high as 50% in the acute poststroke stage, and 30% in the more chronic stages (Gainotti, 1992).

In addition to the need for longitudinal studies examining PSD, the issue of an appropriate definition of PSD has been extended by recent claims that mood disorders in general should be examined in the study of stroke patients. It was after examining

these overall mood disorders for the first year of the poststroke period that House et al. (1991) concluded that excessive importance has been given to major depression as a specific sequelae of stroke. These researchers found that while mood disorders did exist in the poststroke period, major depression was only evident in 2 of their 78 stroke patients for the entire year. In reply to concerns regarding PSD being either over-emphasised (in particular, major depression) or under-diagnosed (due to the presence of other disorders), Fedoroff et al. (1991) assessed patients for depression in the absence or presence of mood disturbance. They found that only 5% of their patients could be diagnosed with major depression as some patients failed to acknowledge their depressed mood. Given these findings, and the limitations of psychiatric diagnosis in adequately assessing PSD, future researchers may make a significant contribution to this area by assessing the affect of stroke patients from a broader perspective than that offered by the diagnosis of major depression.

Several studies have attempted to describe the changes and the factors associated with these changes. The relationship between PSD and hemispheric location of lesion is frequently examined in PSD research. Astrom, Adolfsson, et al. (1993) reported a relationship between PSD at 1 month poststroke and left anterior lesions, although this relationship was not evident at or beyond 3 months poststroke. However, Shima et al. (1994) failed to find a significant relationship between PSD and lesion location. In addition, Herrmann et al. (1995) found that, during the acute poststroke stage, the hemisphere damaged by the stroke was not related to a diagnosis of either major or minor depression. Herrmann et al. also noted that while nine of the left hemisphere patients were diagnosed as having major depression, there was no evidence of major depression in any of the right hemisphere patients. While

Andersen et al. (1995) failed to find a significant relationship between lesion location and PSD at 12 months poststroke, they did identify several other factors which correlated with the occurrence of PSD. Andersen et al. concluded "that (the) etiology to PSD is a complex mixture of prestroke personal and social factors, and stroke induced social, emotional and intellectual handicap" (p. 193). Overall, there are obvious inconsistencies in the results of studies which have examined the relationship between depression and the hemispheric location of the brain lesion. While these conflicting results may be due to the time since stroke at which the patient was assessed, it is also possible that the type and/or severity of PSD differs depending on the location and size of the lesion.

The results of many studies indicate that PSD is related to physical impairment (Astrom, Adolfsson, et al., 1993; Kelly-Hayes & Paige, 1995; Price, 1990; Sharpe et al., 1994). While research to date has failed to conclusively determine a causal relationship between PSD and physical impairment, it is possible that these two outcomes of stroke each exacerbate the effects of the other (Astrom, Adolfsson, et al.). Cognitive impairment is also a frequent outcome of stroke (Tatemichi et al., 1994), and has been associated with PSD (Sharpe et al., 1994; Starkstein et al., 1990). However, some studies have failed to find a significant relationship between PSD and cognitive impairment (e.g., Parikh et al., 1990; Shima et al., 1994). In addition, the association between PSD and cognitive impairment is frequently not examined by researchers, although the reason for this omission is not clear.

Research has also indicated that depression occurs in the caregivers of stroke patients, although not always when the patient is depressed (Thompson et al., 1990). Thompson et al. concluded that caregiver depression was most severe if the stroke

patient was physically impaired, or if the caregiver felt burdened. Caregiver depression was not found to be significantly correlated with patient depression. However, some studies have found that patient depression is significantly related to psychological distress in the caregiver (e.g., Wade et al., 1986). In addition, although they did not specifically assess for caregiver depression, Draper et al. (1992) found that mood disturbances in both stroke and dementia patients were significantly correlated with the degree of burden felt by caregivers. There is ample evidence that caregivers of stroke patients experience difficulties as a result of their caregiver role (e.g., Greveson et al., 1991). Despite this, very little research has been done on the effects that PSD can have on the psychological wellbeing of the caregiver of the stroke patient.

Despite the large amount of research that has been undertaken on the many aspects of PSD, a number of key issues on the cause and course of this disorder remain unresolved. One consistent finding appears to be that the occurrence of PSD has many additional negative consequences for the stroke patient, further complicating their chances of recovery from what can be a devastating event. Johnson (1991) reviewed PSD research to date and outlined the methodological deficiencies in much of the previous research. Fortunately, more recent research (e.g., Andersen et al., 1995) is methodologically suited to providing findings which are of practical benefit to those clinical neuropsychologists who work with stroke patients and their families.

While many studies examine PSD in stroke patients of all ages, the present study will examine the prevalence, nature, and associated factors of PSD, in patients aged at least 60 years (for the purposes of this study, these patients will be referred to

as "elderly"). Regardless of whether the overall prevalence of PSD remains stable or fluctuates throughout the poststroke period, previous research has indicated that very few individual stroke patients report symptoms of PSD consistently throughout the poststroke period. In addition, previous research has indicated that many factors may be associated with PSD. Therefore, the present study has the following aims:

1. To further investigate the prevalence of PSD in stroke patients at the time of discharge from hospital, with follow-up assessments at 3 and 12 months poststroke.
2. To examine the hypothesis that relationships between PSD and demographic, medical, physical and/or cognitive factors change throughout the poststroke period. In particular, this study will examine the hypothesis that PSD is organic in nature early in the poststroke period, but that depression occurs as a reaction to impairment later in the poststroke period.
3. Longitudinal comparison will determine any change in the level of affective, physical, and cognitive functioning. Comparisons will also be made between depressed and non-depressed patients, to examine the hypothesis that depression at the acute poststroke stage has a negative effect on physical and cognitive recovery.
4. Analysis of the data will examine the hypothesis that PSD at the acute poststroke stage is related to mortality within the first 12 months poststroke.
5. Assessments of the psychological distress in the caregivers will be completed to determine the impact of patient PSD on the caregiver, and the overall prevalence of caregiver psychological distress.

The implications of these findings regarding the outcome for both patients and caregivers will be discussed.

CHAPTER 2

General Method

Participants

Stroke Population

The stroke population was selected from consecutive admissions to Waikato Hospital for ischaemic stroke or intracerebral haematoma, over a 12 month period (1 October 1995 to 30 September 1996). Inclusion criteria required the patients to be: (a) at least 60 years of age at the time of the stroke, and (b) willing and able to provide consent. The residing physician approached 186 patients for inclusion in the study over the 12 month period. All of these patients agreed to see the researcher. Therefore the names of 186 possible participants were referred to the researcher over the course of the 12 months.

Of these 186 patients, further investigation and CT scans determined that the symptoms of 3 patients were due to an event other than a stroke. In addition, 7 patients were not able to understand the information provided concerning the study, 6 patients had left the hospital prior to obtaining the information and were unable to be contacted, and a further 6 patients died prior to the initial assessment. Sixty-one patients refused to participate. Following the method described by Andersen, Vestergaard, Riis, et al. (1994), patients were excluded if they had been treated for a psychological or psychiatric condition within 12 months prior to their stroke. None of the patients were excluded on this basis, as only 2 patients had a history of psychiatric treatment, and both patients had received the treatment approximately 20 to 30 years prior to their stroke. Therefore 103 patients constituted the final stroke population.

Demographic information regarding these patients is presented in Table 1. The

Table 1

Demographic Information of Stroke Population ($N = 103$)

Age:	
M	75
SD	7
Range	60 - 94
Gender:	
Female	57 (55%)
Male	46 (45%)
Handedness:	
Right handed	97 (94%)
Left handed	6 (6%)
Marital status:	
Married	60 (58%)
Widowed	37 (36%)
Divorced/Separated	5 (5%)
Never Married	1 (1%)
Ethnicity: *	
New Zealand European	76 (75%)
New Zealand Maori	4 (4%)
British	17 17%)
Pacific Islander	1 (1%)
Other	4 (4%)

Table 1 (continued)

Demographic Information of Stroke Population ($N = 103$)

Occupation: *	
Retired	87 (85%)
Paid employment	7 (7%)
Homemaker	8 (8%)
Living situation (prestroke / poststroke):	
Living at home with partner	49 (48%) / 41 (40%)
Living at home alone	30 (29%) / 18 (18%)
Living with partner and/or children	15 (15%) / 16 (16%)
Living with friend	1 (1%) / 0 (0%)
Resthome care	7 (7%) / 10 (10%)
District hospital	0 (0%) / 17 (17%)

* $N = 102$

average age of the 103 patients at the time of the stroke was 75 years ($SD = 7$, range = 60 - 94). Fifty-seven (55%) patients were female and 46 (45%) patients were male. Ninety-seven (94%) patients were right-handed and 6 (6%) patients were left-handed. Sixty (58%) patients were married, 37 (36%) patients were widowed, 5 (5%) patients were separated or divorced, and 1 (1%) patient had never married. One patient did not provide information regarding their ethnicity. Of the remaining 102 patients, 76

(75%) patients were of New Zealand European ethnicity, 17 (17%) patients were British, 4 (4%) patients were New Zealand Maori, 1 (1%) patient was a Pacific Islander, and 4 (4%) patients were of "other" ethnicities. One patient did not state their occupation. Of the remaining 102 patients, 87 (85%) patients were retired, 7 (7%) were in paid employment, and 8 (8%) patients gave their occupation as "homemaker".

One patient did not provide information regarding their living situation prior to their stroke. Of the remaining 102 patients, 49 (48%) patients had been living at home with their partner prior to their strokes, 30 (29%) patients had been living alone in their own homes, 15 (15%) patients had been living with their partner and children, or living alone with their children, 7 (7%) patients had been living in resthomes, and 1 (1%) patient had been living with a friend.

The information regarding living situation after discharge from Waikato Hospital was available for 102 patients, as 1 patient died immediately prior to discharge from the hospital. Of the 102 patients, 41 (40%) patients lived with their partners in their own homes after discharge. Eighteen (18%) patients returned to living alone in their own homes, 16 (16%) patients lived with their partner and children, or alone with their children, 17 (17%) patients were discharged to a district hospital, and 10 (10%) patients were discharged to resthome care.

Lesion Location and CT Scan Analysis

Medical information regarding these patients is presented in Table 2. Lesion location and type of stroke were determined using CT scan analysis. CT scans were performed as part of the patient's medical treatment, and not specifically for use in this study. Therefore scan information was not available for all patients. In the

Table 2

Medical Information of Stroke Population ($N = 103$)

Previous strokes:	
None	75 (73%)
One previous stroke	23 (22%)
Two previous strokes	5 (5%)
Lesion hemisphere:	
Right hemisphere	54 (52%)
Left hemisphere	44 (43%)
Cerebellar	2 (2%)
Bilateral	2 (2%)
Brainstem	1 (1%)
Lesion type:	
Infarct	45 (44%)
Haematomas	6 (6%)
Lacunar infarcts	4 (4%)
Haemorrhagic infarcts	4 (4%)
Multi-infarcts	13 (13%)
Atrophied brain	2 (2%)
Unknown	29 (28%)

absence of CT scan information, hemispheric location of lesion was determined by medical examination. Information regarding lesion hemisphere and type was obtained from Dr. Paul Friedman (Director of the Rehabilitation Unit, Waikato Hospital).

Ninety-eight (95%) patients had CT scans; for the remaining 5 (5%) patients the hemispheric location of stroke was determined by clinical examination. Fifty-four (52%) patients had right hemisphere strokes, 44 (43%) patients had strokes in the left hemisphere, 2 (2%) patients had cerebellar strokes, 2 (2%) patients had bilateral strokes, and 1 (1%) patient had a brainstem stroke. Forty-five (44%) patients had strokes of the infarct type, 6 (6%) patients had haematomas, 4 (4%) patients had lacunar infarcts, 4 (4%) patients had haemorrhagic infarcts, 13 (13%) patients had multi-infarcts, and the scans of 2 (2%) patients indicated an atrophied brain. Including the 5 patients who did not have a CT scan, information regarding the type of stroke was unavailable for 29 (28%) patients. For 75 (73%) patients this was their first stroke, 28 (27%) had experienced at least one previous stroke, with 5 of these latter 28 patients having had two previous strokes.

Caregiver Population

The caregiver population was defined as someone who knew the patient prior to their stroke, and was caring for the patient full-time. Staff employed to care for patients residing in hospitals and residential care facilities were excluded, as in these situations the caregiver often did not know the patient prior to the stroke, and more than one person was usually responsible for the care of the patient. Therefore it was considered unlikely that the wellbeing of the patient, and the caregiving situation, would have a direct effect on the psychological wellbeing of the caregiver.

Of the 103 patients initially included in the study, 10 patients had either died

or were not assessable at 3 months poststroke. Of the remaining 93 patients, 73 patients had caregivers at 3 months poststroke. As 27 of these patients were residing in hospitals or residential care facilitates their caregivers were not included in the study. Of the remaining 46 caregivers, 12 refused to participate in the study. The remaining 34 caregivers were assessed at 3 months poststroke, and 1 additional caregiver was introduced to the study at 12 months poststroke. Therefore, 35 caregivers were assessed during this study.

The average age of the 35 caregivers at the time of their first assessment was 67 years (*SD* = 10, range = 28 - 82). Twenty-eight (80%) caregivers were female, and 7 (20%) caregivers were male. Thirty-three (94%) caregivers were married to the patient, and 2 (6%) caregivers were the daughter of the patient. The caregivers had been caregiving for their patients, at the time of their first assessment, for an average of 14 months (*SD* = 29, range = 3 - 120). This average was higher than may be expected at 3 months poststroke, as many caregivers had been caring for the patients prior to their current stroke.

Measures

All patients completed an interview, in addition to completing psychological tests and questionnaires.

Stroke Population

The principal researcher administered all the interviews and psychological measures. Necessary information for the demographic questionnaire and for the ADL measures was obtained from the patient (if possible and reliable), from hospital records, from personal observation by the researcher, from the spouse and/or family

members, or, for those patients in hospitals or resthomes, from nursing staff.

Interview. All stroke patients were interviewed by the principal researcher. The purpose and nature of the study were explained, and written consent for participation in the study was obtained (Appendix A). A demographic questionnaire concerning the living situation, and medical and psychological history of the stroke patient was completed (Appendix B).

The following measures were administered to the stroke patients (Appendix C):

Barthel Activities of Daily Living Index. The Barthel Activities of Daily Living Index (Barthel ADL Index; Wade & Langton-Hewer, 1987) determined the level of physical functioning for each patient. This is a descriptive measure that is considered to be the preferred ADL scale (Wade, 1992), and has been established as acceptable for use with stroke patients (Wade & Langton-Hewer). It is a measure of physical independence in ten basic self-care activities including: bowel control, bladder control, grooming, toilet use, feeding, transfer (from bed to chair and back), walking mobility, dressing, bathing, and walking up and down 3 - 4 stairs. The independence level of each activity within the previous 24 hours was recorded. The range of possible scores for each activity varied from 0 - 1 (dependent or independent, respectively) to 0 - 3 (totally dependent, needs much help, needs some help, to fully independent, respectively). The sum of scores for each item gave a final score from 0 (completely dependent) to 20 (fully independent).

The categorical level of physical functioning for each patient was determined from the final Barthel ADL score, according to the categories established by Wade and Langton-Hewer (1987). Patients were classified as being either very severely

impaired (a final score of 0 - 4), severely impaired (scores 5 - 9), moderately impaired (scores 10 - 14), mildly impaired (scores 15 - 19) or independent (a score of 20).

Nottingham Extended Activities of Daily Living Index. The Nottingham Extended Activities of Daily Living Index (Nottingham Extended ADL Index; Nouri & Lincoln, 1987) is a measure of common activities that do not directly involve self-care tasks, and was designed specifically for use with stroke patients. Patients were asked questions concerning what activities they performed in each of four areas of daily activities. The first section consisted of six questions regarding mobility, including walking outside, climbing stairs, getting in and out of cars, crossing roads, and using public transport. The second section consisted of five questions regarding kitchen activities, including feeding abilities, making hot drinks and snacks, carrying hot drinks from one room to another, and doing the dishes. The third section consisted of five questions regarding domestic tasks, including washing clothes, going shopping and managing money, and doing housework. The fourth and final section consisted of six questions regarding leisure activities, including reading, writing letters, using the telephone, going out socially, gardening, and driving a car. Patients were recorded as either performing the task on their own, on their own with difficulty, performing the task with help, or not performing the task.

Two methods of scoring were used, as two forms of analysis were completed. Using the scoring method recommended by Nouri and Lincoln (1987), for use as a descriptive measure, scoring was completed as follows: performing the task on their own, or on their own with difficulty received a score of 1, performing the task with help, or not performing the task received a score of 0. The total score for each of the four categories was recorded separately. The average score of each of these four

categories was used to compare performance across time. In addition, within each category each patient was recorded as completing all, some, or none of the tasks represented. Using the scoring method recommended by Wade (1992), for use in the correlation and regression analysis, scoring was completed as follows: performing the task on their own received a score of 3, performing the task on their own with difficulty received a score of 2, performing the task with help received a score of 1, and not performing the task received a score of 0. This latter method of scoring was used in the correlations and regressions to provide scoring comparative to the other measure of physical functioning used in the present study, the Barthel ADL Index.

Geriatric Depression Scale. The Geriatric Depression Scale (GDS; Yesavage et al., 1983) is an instrument designed specifically to screen for depression in the elderly population. It consists of 30 questions that require yes/no responses based on how the person has felt in the past week. Twenty of the 30 questions indicate a tendency towards depression when answered positively, while the remaining ten questions indicate a tendency towards depression when answered negatively; therefore 10 questions are reverse scored. One point is scored for each answer indicating depression, resulting in a total score out of a possible 30. For the purposes of this study, the researcher administered the GDS orally. This was due largely to the substantial proportion of the stroke patients involved in the study who had visual and/or writing impairments.

The GDS was developed in a study of 100 elderly participants, and is suitable for use with both healthy elderly and elderly with physical and/or cognitive impairments. Patients were classified as having either no depression, mild depression, or severe depression based on the cutoff scores recommended by Spreen and Strauss

(1991). A score of 0 - 9 indicated no symptoms of depression, a score of 10 - 19 indicated symptoms of mild depression, and a score of 20 - 30 indicated symptoms of severe depression.

General Health Questionnaire-28. The General Health Questionnaire (GHQ; Goldberg, 1972) was designed to detect the presence of psychological disorder occurring within the last three weeks approximately. Varying lengths of the GHQ have been developed; the present study used the GHQ-28 (i.e., this version of the GHQ contains 28 questions). The GHQ-28 has four scales, each containing seven questions. The four scales inquire about the patients wellbeing in the following categories: somatic symptoms (scale A), anxiety and insomnia (scale B), social dysfunction (scale C), and severe depression (scale D).

Prior to being asked each question by the researcher, the patients were instructed as follows: "I would like to know if you have had any medical complaints and how your health has been in general, over the past few weeks. These questions are just about present and recent complaints, not those that you had in the past." Each question had four similar response categories. Two methods of scoring were used to record patients' responses. To obtain a score for each of the four scales, and therefore a score for each of the four disorders, the Likert method was used. Using the Likert scoring method, responses were scored from 0 (low dysfunction) to 3 (high dysfunction). The total score for each of the four sections was recorded. These totals provided a measure of each of the four emotional disorders represented by the scales. To obtain a total score of psychological disorder, the GHQ scoring method of 0-0-1-1 was used (therefore scoring from low dysfunction to high dysfunction, respectively). The total score for all questions was recorded. A cutoff of 5/6 was used

to determine the presence of psychological disorder, where a score of 0 - 5 is considered to be within the normal range, and a score of 6 or greater is indicative of dysfunction. This cutoff has been established as most suitable for use with neurological patients (Lykouras et al., 1996).

Minnesota Differential Diagnosis of Aphasia Test - short form. The short form of the Minnesota Differential Diagnosis of Aphasia Test (Powell, Bailey, & Clark, 1980) was developed from the full Minnesota Differential Diagnosis of Aphasia Test (Schuell, 1965). While the full Minnesota Differential Diagnosis of Aphasia Test determines the type of aphasia presented by the patient, the short form of the Minnesota Differential Diagnosis of Aphasia Test is a screening instrument for detecting the presence or absence of aphasia. The short form of the Minnesota Differential Diagnosis of Aphasia Test was standardised in a study of 86 stroke patients (mean age = 61 years, *SD* = 13). The test correlated .90 with the full Minnesota Differential Diagnosis of Aphasia Test. For the purposes of this study, a screening instrument was sufficient as it was necessary only to determine whether the patient presented with aphasia, as such a condition affects performance on many of the measures used.

The short Minnesota Differential Diagnosis of Aphasia Test consists of four subtests: (a) identifying names serially, (b) oral reading of words, (c) naming pictures, and (d) written spelling. For subtest A the patient was presented with a picture card and asked to point out specific items in the picture. Subtest B required the patient to read 15 words presented on a card. Subtest C required the patient to name 20 items presented in picture form. Subtest D required the patient to write 10 specific words given by the researcher. The number of errors for each section was recorded, with an

overall total of more than two errors indicating aphasia. However, as many of the patients participating in this study had a limited formal education, more than two errors in the written spelling section (subtest D) was not considered to be indicative of aphasia, unless they were: (a) simple words (e.g., girl, went), or (b) accompanied by errors in other sections. In addition to this exception, lack of recognition of picture 19 (a sled) in subtest C was not considered to be an error, as sleds are not a common item in New Zealand.

Cognitive Assessment Scale. The Cognitive Assessment Scale (CAS) is a subtest of the Clifton Assessment Procedures for the Elderly (Pattie & Gilleard, 1979). The CAS contains three sections: (a) Information/Orientation, (b) Mental abilities, and (c) Psychomotor abilities. The Information/Orientation section requires the patient to answer 12 questions concerning their age, birth date, and chronological and geographical whereabouts. The total number of correct answers was recorded, resulting in a final score out of 12.

The Mental Ability section has four subsections, requiring the patient to: (a) count from 1 to 20, (b) say the alphabet, (c) write their full name, and (d) read a list of 14 words. Both the counting and alphabet subsections were timed and, with the reading subsection, were each scored out of 3, while the name writing subsection was scored out of 2, giving a total score out of 11.

The Psychomotor section consists of the Gibson Spiral Maze which is a spiral drawn on a piece of card that measures 247 x 350 mm. The spiral contains 56 circles (which measure 6 mm in diameter) that lie between the lines of the spiral as obstacles. The patient was required to draw around the spiral. Drawing on or through the lines or the circles that lie between the lines was recorded as an error. The time taken to

complete the Gibson Spiral Maze was noted, and the total number of errors recorded. The time taken and number of errors made were then converted to a final score of psychomotor abilities using the table provided on the report form.

The total of all three sections determined the level of basic cognitive impairment, using the cutoff scores provided by Pattie and Gilleard (1979). A score of 30 - 34 indicated no impairment, a score of 24 - 29 indicated mild impairment, a score of 16 - 23 indicated moderate impairment, a score of 9 - 15 indicated marked impairment, and a score of 0 - 8 indicated severe impairment of basic cognitive functioning.

The CAS was standardised against several groups of elderly individuals, including a community "well" group, a community "care" group (receive regular medical attention), an acute "psychiatric" group (recently admitted, non-organic psychiatric illness), an acute "medical" group (recently admitted for physical illness), a social services group (rest home residents), a social services "elderly mental infirm" group, an acute "psychogeriatric" group (organic psychiatric illness, likely to remain in care), a chronic "geriatric" group (geriatric hospital patients), and a chronic "psychogeriatric" group (psychogeriatric hospital patients). The normative data indicates that a healthy elderly person living in their own home is likely to score 11, 10, and 10 on the Information/Orientation, Mental Ability, and Psychomotor sections, respectively (Pattie & Gilleard, 1979).

Controlled Oral Word Association Test. The Controlled Oral Word Association Test (COWA; Spreen & Strauss, 1991) is a test of verbal fluency. Patients were instructed as follows: "I will say a letter of the alphabet. Then I want you to give me as many words that you can think of that begin with that letter, as

quickly as you can. You may say any words at all, except proper names such as the names of people or places (like Rotorua, or Robert). Also, do not use the same word again with a different ending (like eat, and eating). For example, if I say 'B', you could say 'bad', 'battle', or 'bed'. Can you think of other words that begin with the letter 'B'? (pause). That is fine, now I am going to give you another letter, and again you say all the words beginning with that letter that you can think of. Remember, no names of people or places, just ordinary words. Also, if you should draw a blank, I want you to keep on trying until the time limit is up. You will have a minute for each one. The first letter is 'F'."

On completion of the first minute, the patients were asked for words beginning with "A", then "S". Each word was recorded, and the total number of correct words given by the patient provided the final score. The total number of correct words was converted into a scaled score ($M = 10$, $SD = 3$) using the normative data for individuals aged over 55 years produced by Ivnik, Malec, Smith, Tangalos, and Petersen (1996).

The age-corrected norms developed by Ivnik et al. (1996) were produced from a study involving 743 people. These norms are necessary because, although there is some indication that age and gender do not significantly affect performance on the COWA (Lezak, 1995), additional studies have indicated that performance can deteriorate with increasing age (Spreen & Strauss, 1991).

Verbal Paired Associates. The Verbal Paired Associates (VPA) is a verbal memory test from the Wechsler Memory Scale-Revised (WMS-R; Wechsler, 1987). The WMS-R was originally standardised in a study of 300 people aged between 16 and 75 years. Norms for an older population have been developed in a study (Mayo's

Older Americans Normative Studies [MOANS]) of 441 cognitively normal individuals aged between 56 and 94 years (Ivnik et al., 1992b).

The VPA includes an immediate verbal recall subtest, and a delayed verbal recall subtest. The VPA consists of eight pairs of words; four "easy" pairs, and four "hard" pairs. At the commencement of the immediate verbal recall subtest, the patients were instructed as follows: "I am going to read you a list of words, two at a time. Listen carefully because after I am through I will ask you which words go together. For example, if the words are East - West, Gold - Walk, then when I say the word East, you would answer (pause) West. And when I say the word Gold, you would answer (pause) Walk. Do you understand? Now listen carefully to the list as I read it." The patients were also informed that any errors would be corrected, and the list would be read more than once to allow for any improvement. One point was given for each correct response. The total number of correct answers was recorded for both easy and hard pairs.

The immediate verbal recall subtest was administered for three consecutive trials. Alternatively, the researcher stopped testing if the patient remembered all words correctly before the completion of the three trials. At the completion of the trials the patients were reassured of their performance, and given the following instruction: "Good. Later on I will ask you the words again, so try to remember them now."

The delayed verbal recall subtest was administered at the completion of the remainder of the assessment (approximately 30 minutes later). At this stage the patients were instructed as follows: "A while ago I read you a list of words, two at a time. Then I read you the first word in each pair, and you were to tell me the word

that went with it. For example, if the words were East - West, and I said the word East, you would answer (pause) West. Do you remember? Now I want to see how well you remember the word pairs. I'm going to read you one of the words, and you are to tell me the word that goes with it. The first word is Rose. What word went with it?" Again, each correct answer was worth one point, and the total number of correct answers was recorded for both easy and hard pairs.

Total scores for both the immediate and delayed verbal recall subtests were generated by combining the totals for the hard and easy scores, giving two total scores: a score out of 24 for the immediate trials, and a score out of 8 for the delayed trial. Standard scores ($M = 10$, $SD = 3$) were obtained for these two overall totals from MOANS (Ivnik et al., 1992b) norms for the WMS-R. Use of the standard scores for the older population was necessary as increasing age may have an effect on WMS-R performance (Lezak, 1995).

General intelligence. Premorbid IQ was estimated using the National Adult Reading Test (NART; Nelson, 1982). The NART was standardised in a study of 120 patients, aged 20 to 70 years, with extra-cerebral nervous diseases. The NART consists of a reading list of 50 words that are irregular to normal phonetic rules, and are presented in increasing difficulty. The list was presented to the patients on a card measuring 210 x 296 mm. The patients were asked to read the list of words aloud to the best of their ability. The researcher reassured the patients that they were not expected to recognise and know all 50 words, but told the patients that it would be preferable if they attempted all words. Words that were mispronounced or not attempted were recorded as an error. The range of possible errors is 0 to 50. The number of errors was converted into an estimated premorbid Full Scale IQ according

to the table provided in the manual. Possible estimated premorbid Full Scale IQs ranged from 131, (no errors), to 69 (50 errors). Although the conversion table provided in the NART manual was developed for use with individuals aged 20 to 70 years, this table was also used in the present study to estimate the premorbid IQ of patients over the age of 70 years, as a more suitable alternative was not available. The scoring of the NART was based on the correct New Zealand pronunciation of the words, as established by a recording of the reading of the NART pronunciation guide by a professional linguist, providing a pronunciation guide for the New Zealand context (Fisher, 1996). Clinical judgement was used when assessing patients with speech difficulties as a result of their stroke, and when assessing the 2 patients for whom English was not their first language.

The Vocabulary test, together with the Block Design test, was used to assess the current level of intellectual ability of each patient. The Vocabulary test is a subtest of the Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler, 1981). The Vocabulary test consisted of 35 words, and the participant was required to correctly define each of the words. In addition to the researcher saying each word, the words were displayed on a card measuring 210 x 296 mm, which was given to the patient. The patient was then able to read the words if necessary. The first 20 words were presented on one side of the card, the remaining 15 on the reverse side. The patient's answer was written verbatim on the scoring sheet by the researcher. The test was discontinued after five consecutive incorrect responses. Answers were scored according to the criteria provided in the WAIS-R manual; that is, correct answers were given a score of 2, while close approximations were given a score of 1. The total score was recorded and converted to an age-scaled standard score using data

from the norms provided by part of the MOANS project (Ivnik et al., 1992a). This normative data for older individuals was developed in a study of 512 people, aged from 56 to 97.

The Block Design test is a subtest of the WAIS-R (Wechsler, 1981). It uses nine cubic blocks, coloured red on two sides, white on two sides, and half red/half white on two sides. A bound book of pictures of nine designs (trials) was presented to the patient. Each card measures 71 x 96 mm. The picture cards were placed directly in front of the patient, allowing room for manoeuvring the blocks. Trials 1 to 5 use four blocks; trials 6 to 9 use nine blocks. The blocks were given to the patient, and the patient was instructed as follows: "You see these blocks? They are all alike. On some sides they are all red; on some, all white; and on some, half red and half white. I am going to put them together to make a design. Watch me." The blocks were arranged as pictured in Card 1. The patient was then told: "Now make one just like this". The researcher then administered the remainder of the Block Design test in strict accordance with the instructions provided in the WAIS-R manual. The researcher timed each trial, and scored as was appropriate on the WAIS-R record form. The test was discontinued after three consecutive failures, or at the completion of all nine designs. A total score for the Block Design test was calculated as determined in the WAIS-R manual, and converted to an age-scaled standard score using the normative data provided by the MOANS project (Ivnik et al., 1992a).

For each patient, the age-scaled scores of the Vocabulary test and the Block Design test were combined and converted to a Full Scale IQ (FSIQ), using the tables provided by Brooker and Cyr (1986). Although these tables were developed for use with individuals aged up to 74 years of age, these tables were also used to provide a

FSIQ for the older patients in the present study, as more precise tables for this age group were not available.

Digit Span. The Digit Span test is also from the WAIS-R (Wechsler, 1981), and is a measure of simple attention abilities. It consists of two sections: Digits Forward, and Digits Backward. Each section consists of seven pairs of lists of numbers presented in random sequence. The lists increase in length, with each pair (2 trials) being of the same length.

Of the two Digit Span tests, the Digits Forward test was administered first. The patients were instructed as follows: "I am going to say some numbers. Listen carefully, and when I am through say them right after me." The researcher read the digits at a rate of one digit per second. Each correct response received a score of 1. The number of correct responses was recorded. Testing was discontinued when both trials of the same length were incorrect.

On completion of the Digits Forward test, the researcher administered the Digits Backward test. For the Digits Backward test the patients were instructed as follows: "Now I am going to say some more numbers, but this time when I stop I want you to say them backwards. For example, if I say 7 - 1 - 9, what would you say?" If the patient did not respond correctly they were instructed as follows: "No, you would say 9 - 1 - 7. I said 7 - 1 - 9, so to say it backwards you would say 9 - 1 - 7. Now try these numbers. Remember, you are to say them backwards. 3 - 4 - 8." After the patient responded, either correctly or incorrectly, the researcher administered the first list of the Digits Backward test. Scoring was recorded as with the Digits Forward subtest. Scores for each section were recorded separately, and then totalled. The total Digit Span score was converted to a standard score ($M = 10$,

SD = 3) using the normative data from the MOANS project (Ivnik et al., 1992a).

Digit Symbol. Complex attention ability was measured using the Digit Symbol test, which is also from the WAIS-R (Wechsler, 1981). The Digit Symbol test requires the participant to fill in blank squares, numbered in random sequence, with a symbol denoted by the key above the blank squares. The key consists of the numbers 1 to 9, each represented by a different symbol. There are 100 blank squares, with the first seven used as sample exercises for the instructional demonstration. The researcher explained the nature of the test and began demonstrating, as described in the WAIS-R manual. On the completion of the sample exercises, the patient was instructed as follows: "When I tell you to start, you do the rest of them. (Pointing to the first test item.) Begin here and fill in as many squares as you can, one after the other, without skipping any. Keep working until I tell you to stop. Work as quickly as you can without making mistakes. When you finish the first line (pointing) go on to the next one. Now go ahead and begin." The researcher timed the test, and the patient was asked to stop at the completion of 90 seconds. The number of completed squares was recorded, excluding any errors, any squares completed out of sequence, and any squares that were filled in after the researcher had asked the patient to stop. This total was converted to a standard score (*M* = 10, *SD* = 3) using the normative data from the MOANS project (Ivnik et al., 1992a).

Caregiver Population

Interview. All caregivers were interviewed by the principal researcher. The purpose and nature of the study were explained, and written consent for participation in the study was obtained. A demographic questionnaire detailing the background of the caregiver was completed (Appendix D).

The caregiver was given the following questionnaires to complete (Appendix E):

General Health Questionnaire-28 (GHQ-28). The GHQ-28 was completed by the caregivers, with reference to their own recent medical and psychological wellbeing. The GHQ scoring method of 0-0-1-1 was used, with a cutoff of 4/5, where a score of 0 - 4 is within the normal range, and a score of 5 or greater is indicative of psychological disorder. This cutoff has been established as suitable for this version of the GHQ (Goldberg & Hillier, 1979).

Katz Adjustment Scales. The Katz Adjustment Scales-Relatives forms (KAS-R; Katz & Lyerly, 1963) consist of five sections. The KAS-R was validated in a study involving 19 relatives of discharged patients.

The caregivers completed sections R2 (Level of Performance of Socially-expected Activities) and R3 (Level of Expectations for Performance of Social Activities) of the relative forms. Together, these two sections indicated how satisfied the caregiver was at the level of the patient's functioning. Form R2 consisted of 16 questions inquiring about the involvement of the patient in social and self-care activities. Involvement of the patient in activities was categorised into three responses: (a) is not doing (1 point), (b) is doing some (2 points), and (c) is doing regularly (3 points). Section R3 consisted of the same 16 items used in subscale R2, but asks the caregiver what they expected the patient's level of activity to be. Responses for section R3 were recorded in a similar manner to that of section R2, but the responses were categorised with reference to the caregiver's expectations rather than the actual involvement of the patient. The caregivers' level of satisfaction with the patients' performance of social activities was determined by the discrepancy

between the totals of section R2 and section R3 (R3 - R2). Possible scores of satisfaction ranged from -32 (very satisfied, patient doing more than expected), to 32 (very dissatisfied, patient doing less than expected). A score of 0 indicated that the caregiver considered the patient to be doing everything that they expected of them.

Objective and subjective burden. Level of objective and subjective burden was determined using the questionnaires of objective and subjective burden developed by Montgomery, Gonyea, and Hooyman (1985). The Measurement of Objective Burden consists of nine items that require the caregiver to state the extent to which nine aspects of their life have changed as a result of caregiving. Five categories of responses were available: (a) a lot more/better (1 point), (b) a little more/better (2 points), (c) the same (3 points), (d) a little less/worse (4 points), and (e) a lot less/worse (5 points). A total score based on all nine items was obtained. The possible scoring range for objective burden was 9 - 45, where a score of 9 - 26 indicated no burden, a score of 27 indicated no change since the stroke, and a score or 28 - 45 indicated burden.

The Measurement of Subjective Burden consists of 13 items relating to attitude and feelings experienced as a caregiver. As with objective burden, the frequency of each feeling or attitude was categorised into one of five responses: (a) rarely or never (1 point), (b) a little of the time (2 points), (c) sometimes (3 points), (d) often (4 points), and (e) most of the time (5 points). A total score based on all 13 items was obtained. The possible scoring range for subjective burden was 13 - 65; scores increase with the extent of the subjective burden.

The measures of Objective and Subjective Burden were developed in a study of 80 individuals who were involved, to varying degrees, in caring for an elderly

relative.

Social Activities Questionnaire. The Social Activities Questionnaire (Donald, Ware, Brook, & Davies-Avery, 1978), was developed from a study involving patients aged 14 to 61 years. It provides an indication of social wellbeing by inquiring about the amount and frequency of social contact. While the original scale consisted of 11 items, one item (writing letters) was discontinued as it was considered invalid (McDowell & Newell, 1987). Therefore, the questionnaire used in this study contained 10 items only. Each item was answered and scored separately, according to the schedule accompanying the Social Activities Questionnaire. The final score from each item was recorded separately. Responses to questions 3, 4, and 5 provided a score of social contact. Responses to questions 9 and 10 provided a group participation score. Overall social support was derived from the total scores for social contacts and group participation, together with the responses to questions 1, 2, 6, and 8. Question 7 was excluded from the final analysis as it has been found to be unreliable (Wilkin, Hallam, & Doggett, 1992).

Functional Social Support Questionnaire. The Duke-UNC Functional Social Support Questionnaire (DUFSS; Broadhead, Gehlbach, de Gruy, & Kaplan, 1988) was developed and standardised in a study of 401 women aged 18 to 44 years. Originally consisting of 14 questions, it was reduced to eight questions following standardisation. The DUFSS provides information about the quality and usefulness of the caregiver's social network. It consists of eight items that are scored on a scale from 1 (as much as I would like) to 5 (much less than I would like). Scores from questions 1, 3, 4, 5, and 7 were combined to form a score of confidant support, while the remaining three questions provided an score of affective support. These two

scores were recorded and analysed separately.

Procedure

This study was approved by the Waikato Ethics Committee (Appendix F), and the Ethics Committee of the Department of Psychology, University of Waikato. All patients and caregivers were given written and oral information about the study (Appendix G), and all participants gave written consent before entering the study.

Patients were approached initially by Dr. Friedman, who briefly explained the purpose of the study. The names of patients interested in participating were passed to the principal researcher, who then approached each patient and explained the study in more detail, providing written information for the patient (and, in many cases, their family) to read and consider. The researcher contacted each patient at a later date for their response. Those agreeing to participate signed the consent form. At times the patient was discharged without the researcher's knowledge, prior to consenting (or otherwise) to participating in the study. The researcher contacted these patients at their place of residence for their response.

The stroke patients were assessed on three occasions. Assessments were conducted at a time suitable for the patient, primarily either late morning or early afternoon. The initial assessment occurred as close to the date of discharge as possible. Most initial assessments took place at Waikato Hospital. In situations where the patient was discharged without the researcher's knowledge the researcher contacted the patient and made an appointment for an initial assessment to be completed at the patient's place of residence.

The initial assessment took approximately 30 minutes to complete. In addition

to the interview and collation of the demographic information, the initial assessment consisted of four measures, administered in the following order: the Barthel ADL Index, the GDS, Minnesota Differential Diagnosis of Aphasia test-short form, and the CAS.

Follow-up assessments took place at the patient's place of residence. At 3 and 12 months poststroke, the follow-up assessments each took approximately 1.5 hours to complete. They included the four tests completed in the initial assessment, in addition to the following tests, administered in this order: COWA, Daily Living Activities Questionnaire, VPA (immediate recall), GHQ-28, NART (given at the 3-month follow-up only), Vocabulary test, Digit Span, Block Design, Digit Symbol, and the VPA (delayed recall). The patient had a rest between tests if necessary. In three cases the patient became fatigued during the assessment to the point of affecting performance. In response to this fatigue the assessment was discontinued and the remainder completed within the following few days.

The researcher approached the caregiver at the time of the 3-month follow-up, or in one case where the living situation of the patient had altered between the 3- and 12-month follow-up assessments, the new caregiver was approached at the 12-month follow-up. Therefore, only 1 caregiver was introduced to the study at the 12-month follow-up. The caregivers were provided with information regarding the study and, if in agreement, signed the consent form.

The caregivers were assessed at the time of their patient's 3- and/or 12-month follow-up assessment. Where possible the caregivers were assessed at both 3 and 12 months poststroke. Demographic information was obtained from consenting caregivers during the interview of their first assessment. The researcher explained the

questionnaires to the caregiver, following which the caregiver completed their questionnaires while the researcher worked with the patient. The caregiver questionnaires were presented in the following order: KAS-R, GHQ-28, measures of Objective and Subjective Burden, Social Activities Questionnaire, and the DUFSS.

Data Analysis

All data analysis was completed using Statistica (Statsoft, Incorporated, 1994). An alpha level of .05 was used for all statistical tests. The exact alpha is reported where a significant result is obtained. Where a significant result is not obtained, alpha is reported as $p > .05$.

Following the data screening considerations recommended by Tabachnick and Fidell (1989), data were checked to ensure accurate input, and missing data were identified. Cases with missing data were excluded from correlational and regression analyses.

Tabachnick and Fidell (1989) recommend checking data for normality of distribution, and outliers. Previous research has indicated that the concept of normal distribution in clinical populations is unrealistic (Scheffe, 1959; Tiku, Tan, & Balakrishnan, 1986), and that it is acceptable to use the robust parametric tests to analyse data that is not normally distributed (Kraemer, 1981; Scheffe, 1959; Tiku et al., 1986). Therefore, the present study used parametric testing where necessary, regardless of the normality of the data under analysis.

Tabachnick and Fidell (1989) outline four possible explanations for outliers. As the data was checked for errors, missing data were accounted for, and all data belonged to the intended population of interest, the only remaining explanation for

any outliers was that they were extreme values, and did not belong to a normal distribution. As explained above, it was considered unnecessary to normalise the clinical data obtained in the present study. Therefore any outliers were not altered or deleted to conform to normality of distribution. In addition, it has been suggested that outliers are an important source of information (Wilcox, 1998).

It has been suggested that, regardless of normality, parametric test are not always robust against unequal variances (Kraemer, 1981; Scheffe, 1959; Tiku et al., 1986). Therefore, the present study has addressed this issue where possible. Where *t* tests were required, and variances were unequal, *t* tests for separate variance estimates were used. Parametric tests were also used where a non-parametric alternative was not available (e.g., hierarchical regression, MANOVA), as recommended by Blalock (1972).

Tabachnick and Fidell (1989) recommend checking for multicollinearity during data screening. Therefore, following the suggestion of Tabachnick and Fidell, variables with a correlation greater than .70 were combined prior to regression analysis.

Clinical interpretation of obtained scores was undertaken using the cutoff scores or outcome categories provided by the normative data. For those measures that did not provide an interpretative system, obtained scores were interpreted in terms of standard deviation from the mean of the normative sample. Those scores lying at, or greater than, one standard deviation from the mean were considered to reflect "mild" impairment and those scores equal to, or greater than, two standard deviations from the mean of the normative sample were considered to reflect "severe" impairment.

Data was described using the following procedures: mean (*M*), standard deviation (*SD*), and range. Data was analysed using the following statistical procedures: two-tailed independent *t* test, *t* test for separate variance estimates, two-tailed dependent *t* test, Pearson product-moment correlation, point-biserial correlation (r_{pb}; used where one variable was dichotomous), hierarchical regressions, one-way repeated measures ANOVA, MANOVA, Pearson's chi-square, and McNemar chi-square. Post hoc analysis was completed where required, using the Fishers LSD test, or the Scheffes test.

Regression analysis was completed only when the analysis included at least 5 cases for every independent variable, as recommended by Tabachnick and Fidell (1989). Hierarchical regressions were performed with demographic variables added in step 1, medical variables added in step 2, physical variables added in step 3, and the cognitive variables added in step 4. Although physical and cognitive variables are both measures of outcomes of stroke, cognitive variables were analysed in the final step as cognitive functioning following brain insult is generally of greater interest to neuropsychologists than physical functioning.

CHAPTER 3

PSD and Associated Factors at the Acute Poststroke Stage

During the acute poststroke stage (up to 1 month poststroke), approximately 25% of all stroke patients experience depression (Astrom, Adolfsson, et al., 1993; House, 1987; Robinson et al., 1983). However, some researchers have reported the prevalence of PSD to be much higher at this stage (Morris, Raphael, et al., 1992), with some reports as high as 55% (Ng, Chan, & Straughan, 1995), while others have reported a lower prevalence of 10% (Andersen, Vestergaard, Riis, et al., 1994).

Previous research has reported varying associations between PSD and demographic, medical, physical, and cognitive factors throughout the poststroke period. These differences are clearly evident in studies of the acute poststroke stage. In particular, no specific relationship emerges in the reported associations between the demographic characteristics and the psychological wellbeing of the stroke patient. While some researchers have failed to find a significant relationship between the age of the patient and depression during the acute poststroke stage (e.g., Herrmann et al., 1995; Morris, Raphael, et al., 1992; Ng et al., 1995; Parikh et al., 1990), others have reported that such a relationship does exist. Fedoroff et al. (1991) found that major depression was more prevalent in slightly younger stroke patients assessed within 2 weeks of admission for acute stroke. Robinson et al. (1983) found that younger stroke patients reported more depression than older stroke patients within the first month following the stroke. Contrary to these results, Morris, Robinson, et al. (1992) found an association between older age and major depression at 2 months poststroke. However, any discrepancies in reports of a relationship between age and PSD may be

due to a difference in the average age of the stroke patients in each study as, although the prevalence of stroke is higher in the elderly population (Anderson, 1992; Toole, 1989), few researchers have limited their analysis of PSD to this group of patients.

Conflicting findings are also reported concerning the role of gender in PSD during the acute poststroke stage. Research has indicated that female stroke patients are more likely to report symptoms of PSD than males (e.g., Ng et al., 1995). Conversely, other researchers suggest that males are more likely to experience PSD (e.g., Morris, Robinson, et al., 1992), while various studies report that no relationship exists between PSD and gender (e.g., Fedoroff et al., 1991; Morris, Raphael, et al., 1992; Parikh et al., 1990).

Research on the role of previous strokes in PSD has also produced inconsistent results. However, many researchers fail to address the impact that previous strokes may have on depression, instead limiting the mention of previous strokes to the description of the participants of the study (e.g., Robinson et al., 1983). Given the reactive origins of some forms of depression (Davison & Neale, 1990), it is reasonable to suggest that having a medical history of stroke is likely to increase the chances of experiencing PSD; after experiencing a second stroke the patient is more aware of what may happen to them, and aware that their chances of having a third stroke are even greater. However, this hypothesis is not proven, and alternative reasons may exist, perhaps based on organic factors. Furthermore, many studies of the acute poststroke stage fail to find a relationship between the occurrence of previous strokes and PSD (Morris, Robinson, et al., 1992; Parikh et al., 1990).

Researchers have debated the role of lesion location in PSD for some time. In fact, the extent that lesion location affects PSD is one of the main controversies in this

area of research, even when considering the basic comparison of the left versus the right hemisphere. A relationship between PSD and lesion location would indicate that PSD may have organic origins. Astrom, Adolfsson, et al. (1993) completed a 3-year longitudinal study of major depression following stroke, and reported that while the hemispheric location of the stroke lesion was significantly related to major PSD in the acute poststroke stage, the relationship was no longer significant at 3 months poststroke, nor at any of the later poststroke stages. These researchers concluded that this time factor may account for many of the discrepancies reported previously in the literature.

These results indicate that while PSD may be of an organic nature during the acute poststroke stage, PSD experienced by stroke patients later in the poststroke period may be a result of other factors, such as level of either physical or cognitive impairment. However, Ng et al. (1995) found no significant relationship between hemispheric location of lesion and PSD within 1 week of admission for acute stroke, and Herrmann et al. (1995) also failed to find hemispheric differences in levels of depression within 2 months poststroke. While Robinson et al. (1983) failed to find a relationship between mood disorders and lesion hemisphere at 10 days poststroke, they determined that the introduction of more specific lesion location information resulted in the emergence of a distinct relationship; patients with left frontal lesions were more depressed than patients with lesions located in other areas of the brain. However, Stern and Bachman (1991) questioned the usefulness of examining any relationship between PSD and the hemispheric location of the lesion, suggesting that such analysis "may inadequately characterize the profile of lesion location factors influencing poststroke mood disorders" (p. 354).

The potential relationship between PSD and physical impairment is also debated frequently in the literature. Many researchers have reported that depression and physical impairment are related during the acute stage following stroke (e.g., Fedoroff et al., 1991; Morris, Raphael, et al., 1992; Ng et al., 1995; Robinson et al., 1983) and some studies have indicated that this relationship may continue throughout the poststroke period (Parikh et al., 1990). After finding a relationship between physical impairment and level of depression, Schubert, Taylor, et al. (1992) concluded that depression, together with the overall effects of the stroke, decreases the level of physical functioning in the stroke patient, and in turn, the physical impairment intensifies the depression. However, the association between these two variables has not been determined conclusively, as other researchers have failed to find a relationship between the level of physical impairment and depression during the acute poststroke stage (e.g., Astrom, Adolfsson, et al., 1993; Stern & Bachman, 1991).

Research examining the involvement of cognitive functioning in PSD has also produced a range of results. Irrespective of its relationship with PSD, cognitive impairment is indeed a frequent outcome of stroke (Tatemichi et al., 1994). In accordance with this, Parikh et al. (1990) found that cognitive impairment was evident in many of their stroke patients, although there were no significant differences in cognitive functioning between the depressed and non-depressed groups. Ng et al. (1995) also reported that cognitive impairment was not related to depression during the acute poststroke stage, although a relationship may exist if the depression is severe (Robinson, Bolla-Wilson, Kaplan, Lipsey, & Price, 1986). However, research has indicated that depressed stroke patients will exhibit less improvement in their cognitive functioning than non-depressed stroke patients (Morris, Raphael, et al.,

1992). In addition, Fedoroff et al. (1991) found that depressed stroke patients had more cognitive impairment than the non-depressed stroke group during the acute poststroke stage, and similar findings of such a relationship have also been reported by other researchers (Downhill & Robinson, 1994; Robinson et al., 1983).

Clearly a more comprehensive understanding of the determinants of depression during the acute poststroke stage is needed. If there is a relationship between the hemispheric location of the lesion and PSD, previous research has indicated that the relationship is more evident within the first few weeks following the stroke (Astrom, Adolfsson, et al., 1993). In addition, it has been suggested that if PSD is reactive in nature, any relationship between PSD and physical or cognitive functioning should not become evident until later in the poststroke period, and therefore no relationship between the level of either cognitive or physical functioning and PSD would be found during the acute poststroke stage. The aims of this initial assessment were to determine the prevalence of depression in the acute stage following stroke in patients aged 60 years and older, and also to examine the involvement of demographic characteristics, medical factors, and levels of physical and cognitive functioning in PSD during the acute poststroke stage.

Method

Participants

Of the original 103 patients selected for inclusion in the study, 2 patients were excluded from the initial assessment as they were currently receiving medication as treatment for depression, and an additional 2 patients were not able to be assessed due to the severity of their impairments. Of the remaining 99 patients, 13 patients

were excluded as their results on the short form of the Minnesota Differential Diagnosis of Aphasia Test (Powell et al., 1980) indicated that they were too aphasic to reliably take part in the assessment. The remaining 86 patients formed the sample for the initial assessment of this study.

The 86 patients were assessed, on average, 27 days (*SD* = 16, range = 3 - 73 days) poststroke. The time of this initial assessment will be referred to as 1 month poststroke for the remainder of this study. The average age of the patients at the time of the stroke was 75 years (*SD* = 7, range = 60 - 94 years). Forty-eight (56%) patients were female and 38 (44%) patients were male. Eighty-two (95%) patients were right-handed and 4 (5%) patients were left-handed. Seventy-one (83%) patients were retired, 7 (8%) patients were in paid employment, and 8 (9%) patients gave their occupation as "homemaker".

Eighty-one (94%) patients had CT scans. The CT scans of these 81 patients, and clinical examination of the remaining 5 patients, determined that 50 (58%) patients had a right hemisphere stroke, 31 (36%) patients had left hemisphere strokes, 2 (2%) patients had bilateral strokes, an additional 2 (2%) patients had cerebellum strokes, and 1 (1%) patient had a brainstem stroke. For 64 (74%) patients this was their first stroke, 22 (26%) patients had experienced at least one previous stroke, with 4 of these latter 22 patients having had two previous strokes.

Measures

Demographic information was obtained from hospital records, relatives, and the patient themselves during the course of the clinical interview. The Cognitive Assessment Scale (CAS) from the Clifton Assessment Procedures for the Elderly (Pattie & Gilleard, 1979) was administered to provide an overall assessment of

cognitive functioning. Physical functioning was assessed by administration of the Barthel ADL Index (Wade & Langton-Hewer, 1987). Oral administration of the Geriatric Depression Scale (GDS; Yesavage et al., 1983) assessed affective functioning.

Procedure and Data Analysis

The patients were assessed at the time of discharge from hospital, either at the hospital, or shortly after returning home. The assessment took approximately 30 minutes to complete.

Data was described using the following descriptive procedures: mean (*M*), standard deviation (*SD*), and range. Statistical analysis was completed using the following procedures: independent *t* test, Pearson product-moment correlation, point-biserial correlation (r_{pb}; used where one variable was dichotomous), and hierarchical regression.

Results

Physical Functioning

The Barthel ADL Index was completed for all patients. Responses indicated that 34 (40%) patients were physically independent, while 29 (34%) patients had mild impairment, 15 (17%) had moderate impairment, 6 (7%) had severe impairment, and 2 (2%) patients had very severe physical impairment.

Affective Functioning

All of the patients completed the GDS. Fifty-five (64%) patients had no clinically significant symptoms of depression. Thirty (35%) patients reported symptoms of mild depression, and 1 (1%) patient reported symptoms of severe

depression.

Cognitive Functioning

Six patients were unable to complete the CAS due to sight and/or writing impairments. Of the remaining 80 patients, 46 (58%) patients had no cognitive impairment, while 21 (26%) patients had mild impairment, 8 (10%) patients had moderate impairment, and 5 (6%) patients had marked impairment. No patients had severe cognitive impairment.

Correlational Analysis

The 6 patients who were unable to complete the CAS were excluded from the correlational and regression analyses as information regarding their cognitive functioning was not complete. A further 5 patients were excluded as their lesions were not located in either the left or right hemisphere (i.e., cerebellum, bilateral, or brainstem strokes). Excluding these patients was necessary as the analysis included determining whether there was a relationship between either left or right hemisphere strokes, and PSD. Therefore 75 stroke patients were included in the correlational and regression analyses.

Following the method recommended by Tabachnick and Fidell (1989), statistical analysis determined whether the 11 patients excluded from the correlational and regression analyses were significantly different from the 75 patients included in the analyses regarding depression status. *T* tests determined that the 11 excluded patients were not significantly more depressed than the 75 patients included in the analyses, $t\,(84) = -1.30, p > .05$.

Table 3 displays the results of the correlational analysis. There was no significant correlation between PSD and the age of the patient at the time of the

Table 3

Correlational Analysis of Individual Variables at the Acute Poststroke Stage ($N = 75$)

Variable	1	2	3	4	5	6	7
1. GDS: Depression	--						
2. Gender	.02	--					
3. Age	.19	-.15	--				
4. Previous stroke	.40*	.10	.15	--			
5. Hemisphere	.24*	.14	-.10	.08	--		
6. ADL (Barthel)	-.23	.31*	-.31*	-.14	.21	--	
7. Basic cognition	-.05	.02	-.32*	.01	.07	.52*	--

Note. ADL = Activities of daily living; GDS = Geriatric Depression Scale.

* $p < .05$

stroke ($p > .05$), nor between PSD and the gender of the patient ($p > .05$). However, patients who had experienced a previous stroke had significantly higher depression scores than patients for whom this was their first stroke ($r_{pb} = .40, p < .001$). Left hemisphere strokes, as opposed to right hemisphere strokes, also resulted in significantly higher depression scores ($r_{pb} = .24, p = .042$). There was no significant correlation between PSD and either physical or cognitive functioning ($p > .05$).

Regression Analysis

Hierarchical regression was used to determine whether demographic variables (age and gender), medical status (hemispheric location and previous stroke), physical impairment, and/or cognitive impairment, predicted PSD at the acute poststroke stage.

Table 4 displays the unstandardised (*B*) and standardised (Beta) regression coefficients, R^2 for step 1, and the change in R^2 after entry of each of the additional blocks of independent variables.

At step 1, the demographic variables of gender and age explained only 4% of the variance in the GDS, $F(2, 72) = 1.42$, $p > .05$. At step 2, the demographic and medical variables explained a total of 23% of the variance in the GDS, $F(4, 70) = 5.20$, $p = .000996$, and the medical variables alone accounted for 19% of the variance at this step. Both medical variables contributed significantly to the amount of variance accounted for at this step (previous stroke, $p = .001111$; hemispheric location of the lesion, $p = .037267$). The addition of the medical variables resulted in a significant increment in R^2 ($p = .000427$).

At step 3, the physical variable alone accounted for 4% of the variance at this step, and the demographic, medical, and physical variables explained a total of 27% of the variance in the GDS, $F(5, 69) = 4.98$, $p = .000591$. At step 4, the cognitive variable alone accounted for 1% of the variance in the GDS. With all of the independent variables in the equation, the regression model explained 27% of the variance in the GDS, $F(6, 68) = 4.26$, $p = .001056$.

Therefore, in total, the 6 independent variables explained 27% of the variance in the GDS. The variables included in the regression equation did significantly predict

Table 4

Summary of Hierarchical Regression Analysis for Variables Predicting PSD at the Acute Poststroke Stage, as Measured by the GDS ($N = 75$)

	Variable	*B*	*SE B*	Beta
Step 1	Gender	0.45	1.19	.04
	Age	0.15	0.10	.20
Step 2	Previous stroke	4.20	1.24	.37 *
	Hemisphere	2.37	1.12	.23 *
Step 3	Physical functioning	-0.25	0.14	-.21
Step 4	Basic cognition	0.11	0.13	.11

Note. $R^2 = .04$ for Step 1 ($p > .05$),

change in $R^2 = .19$ for Step 2 ($p = .000427$),

change in $R^2 = .04$ for Step 3 ($p > .05$),

change in $R^2 = .01$ for Step 4 ($p > .05$).

*$p < .05$.

PSD. However, these results indicate that, at the acute poststroke stage, the strongest predictors of PSD were previous stroke and hemispheric location of the lesion.

Discussion

The initial assessment of this study demonstrated that 36% of the present group of stroke patients were depressed at the acute poststroke stage. Patients who had left hemisphere strokes and/or had experienced a previous stroke were more likely to report symptoms of depression. This study failed to find a significant relationship between PSD and the levels of physical or cognitive impairment, nor was a significant relationship evident between PSD and either age or gender.

Approximately one third of the present group of stroke patients exhibited clinically significant symptoms of depression at the acute poststroke stage, although symptoms of severe depression were evident in only one of these patients. The prevalence of depression reported in this study is slightly higher, although not altogether inconsistent, with the findings of previous studies. That is, while House (1987) reported that 25% of stroke patients reported symptoms of PSD at the acute poststroke stage, several studies have reported higher prevalence rates (e.g., Morris, Raphael, et al., 1992; Ng et al., 1995). Reasons for these differences in reported prevalence rates are numerous. The use of different diagnostic criteria is one possible explanation, as few studies use the GDS to determine the presence of PSD as they assess stroke patients of all ages. However, this study investigated elderly stroke patients only, and the GDS is an appropriate measure to use when assessing depression in this population (Agrell & Dehlin, 1989).

The prevalence of physical and cognitive impairment among the present group

of stroke patients was higher than that of depression. Some degree of physical impairment was evident in 60% of these patients. However, approximately half of the patients with physical impairment were only mildly impaired. Although cognitive impairment was less prevalent than physical impairment (40% of the patients demonstrated cognitive impairment), again approximately half of the patients with cognitive impairment had only mild impairment. Furthermore, no patients demonstrated severe cognitive impairment. Therefore, physical impairment was more prevalent than cognitive impairment in this group of stroke patients at the acute poststroke stage.

The results of this initial assessment indicated that having either a left hemisphere stroke and/or a medical history that includes the occurrence of a previous stroke are both strong predictors of PSD. No relationship was evident between depression and any of the other predictor variables. While previous reports of the involvement of gender in PSD varied (Morris, Robinson, et al., 1992; Ng et al., 1995), other studies have found that younger stroke patients were significantly more depressed than older patients (Fedoroff et al., 1991; Robinson et al., 1983). The fact that this study examined elderly stroke patients only may have lessened the probability of finding a significant relationship between age and level of depression.

This initial assessment found that patients with strokes in the left hemisphere were more likely than patients with right hemisphere strokes to report symptoms of depression at the acute poststroke stage. As a relationship was evident between hemispheric site of stroke and depression, it is possible that depression during this stage of the poststroke period may be of an organic nature. Other studies have also reported a relationship between hemispheric location of the lesion and PSD during the

acute poststroke stage (Astrom, Adolfsson, et al., 1993; Robinson et al., 1983), although research indicates that this relationship may not be evident later in the poststroke period (Astrom, Adolfsson, et al., 1993). Therefore, although depression in the acute stage following stroke may have organic origins, depression later in the poststroke period may be due to other factors; PSD later in the poststroke period may occur as a reaction to the level of impairment. Clearly this hypothesis requires further examination, and will be included in the analysis of these stroke patients at 3 months poststroke.

This initial assessment also found that patients who had experienced a previous stroke were more likely to report symptoms of depression. This is an understandable relationship, as it is possible that a patient who has experienced a previous stroke is more aware of what may happen to them, and subsequently may be aware that their chances of having a third (or fourth) stroke are greatly increased. Little information regarding this hypothesis is available in the literature as few researchers examine this relationship, particularly at the acute poststroke stage. Longitudinal analysis of the stroke patients at 3 and 12 months poststroke will determine whether a history of stroke continues to have a negative impact on the psychological wellbeing of patients during the first year following stroke.

No predictive relationship was evident between PSD and physical impairment during the acute poststroke stage. This is consistent with the findings of Astrom, Adolfsson, et al. (1993). However, other studies have reported finding this relationship (Fedoroff et al., 1991; Ng et al., 1995; Robinson et al., 1983). As the present study examined stroke patients at the time that they were leaving, or had just left, the hospital, it is possible that they were still receiving considerable assistance in

their self-care activities, hence many patients would not yet be aware of their physical limitations. If PSD is a reaction to physical impairment, the timing of this assessment may be an explanation for finding no relationship between the two factors. Assessing these patients at a later stage of the poststroke period may uncover a relationship between physical functioning and PSD, which could indicate that PSD is, at least in part, reactive in nature.

This initial assessment failed to find a significant relationship between PSD and cognitive impairment at the acute poststroke stage, although other researchers have determined that this relationship may occur (Fedoroff et al., 1991; Robinson et al., 1983). It is still possible that PSD may be a reaction to cognitive impairment, but while these patients were still coming to terms with their physical impairment in their everyday care at the time of their discharge from hospital, they may also have yet to realise the extent of their cognitive impairment. Therefore it is possible that a relationship may exist between PSD and physical and/or cognitive impairment later in the poststroke period. Further analysis of this group of stroke patients at 3 and 12 months poststroke will address this issue.

In summary, clinically significant symptoms of depression were evident in approximately one third of these stroke patients at the acute poststroke stage. The strongest predictors of PSD at 1 month poststroke were left hemisphere lesion, and a history of stroke. While no relationship was evident between PSD and the levels of physical and cognitive impairment at this stage, further assessment of these patients at 3 and 12 months poststroke may uncover such a relationship as the patients realise the degree of their impairment. Previous studies have found that left hemisphere stroke is no longer related to PSD at 3 months poststroke (Astrom, Adolfsson, et al., 1993),

while physical impairment has an effect on, or is affected by, PSD throughout the poststroke period (Parikh et al., 1990). Further assessment of these stroke patients will ascertain whether these relationships are evident later in the poststroke period, as well as determine the prevalence of PSD during the first 12 months following stroke.

CHAPTER 4

PSD and Associated Factors at 3 Months Poststroke

The majority of new cases of PSD develop in the first few months following a stroke (Andersen, Vestergaard, Riis, et al., 1994). Astrom, Adolfsson, et al. (1993) reported a slight increase in rates of depression from 25% at the acute poststroke stage (1 month poststroke), to 31% at 3 months poststroke. Although this is only a slight increase, clearly PSD is still a concern at 3 months poststroke.

There are changes in the relationships between PSD and associated factors within the first few months following stroke. Generally, most studies fail to find a significant relationship between PSD and demographic factors such as age and gender at 3 and 6 months poststroke (Astrom, Adolfsson, et al., 1993; Robinson, Starr, Lipsey, et al., 1984, Shima et al., 1994). Medical factors, such as a history of stroke, may also be related to PSD. Eastwood et al. (1989) found that patients who had experienced a previous stroke were more likely to report symptoms of PSD at approximately 3 months poststroke. However, many studies fail to address the involvement of previous strokes in PSD during this stage of the poststroke period, instead choosing to focus on the role of lesion location in PSD.

Although much of the research to date indicates evidence of a significant relationship between PSD and left hemisphere strokes, this association has not been proven conclusively (House, 1987). Interestingly, although finding a significant relationship between PSD and left hemisphere strokes during the acute poststroke stage, Astrom, Adolfsson, et al. (1993) reported that the relationship was not evident at or beyond 3 months poststroke. Consistent with these findings, Yamaguchi et al.

(1992) found that while significant relationships were found between PSD and left frontal or right parieto-occipital lesions during the acute poststroke stage, these relationships were not evident later in the poststroke period. This failure to find a relationship between lesion location and PSD at these later stages of the poststroke period suggests that while the depression may be due to organic factors at the acute poststroke stage, at later stages of the poststroke period other factors may be responsible for, and associated with, PSD. House, Dennis, Warlow, Hawton, and Molyneux (1990) also failed to find a relationship between the hemispheric location of the lesion and PSD at 6 months poststroke. However it must be noted that these researchers failed to find a significant relationship between lesion location and PSD at any stage during the first year poststroke.

Nelson et al. (1994) reported an interesting contrast to the hypothesis that left hemisphere strokes are related to PSD. These researchers found that, at 6 months poststroke, emotional problems (including depression) increased in patients with right hemisphere strokes. During the same poststroke stage, the emotional functioning of patients with left hemisphere strokes stabilised. This pattern of emotional change may account, at least in part, for the findings that there is no relationship between depression and hemispheric location of lesion at 3 or 6 months poststroke. That is, as the patients with the left hemisphere strokes stabilised emotionally (although remained depressed) and the patients with right hemisphere strokes experienced increased emotional dysfunction, it is possible that by 3 months poststroke there is little difference in the level of depression between patients with left hemisphere strokes and those with right hemisphere strokes. In accordance with the finding by Nelson et al., Astrom, Adolfsson, et al. (1993) reported that while hemispheric location of the

lesion was not related to PSD at 3 months poststroke, the prevalence of depression among patients with right hemisphere strokes clearly increased. Therefore a relationship between PSD and the hemispheric location of the lesion, and the concept of PSD being organic in nature, is still a possibility within the first 6 months following stroke. However, the results of these studies suggest that it is right hemisphere lesions that have an impact on depression at this stage of the poststroke period.

By 3 months poststroke, most stroke patients will have left acute hospital care, and will have settled into their daily routine to some extent. As the majority of stroke patients recover most of their ability to perform activities of daily living by 3 months poststroke (Jorgensen et al., 1995b), many will be increasingly aware of their remaining physical and cognitive limitations. If PSD occurs as a reaction to impairment, the impact of physical and cognitive impairment on PSD during this time may therefore be greater than was evident earlier in the poststroke period. In accordance with this hypothesis, Robinson, Starr, Lipsey, et al. (1984) reported a steady increase in the relationship between depression and physical impairment during the first 6 months after stroke. Astrom, Adolfsson, et al. (1993) also reported finding a significant relationship between depression and degree of physical impairment at 3 months poststroke.

In addition to physical impairment, cognitive impairment also occurs frequently following stroke (Tatemichi et al., 1994). However, the existence of a relationship between cognitive impairment and PSD at various stages of the poststroke period continues to be debated. Robinson, Starr, Lipsey, et al. (1984) reported that while they failed to find a significant relationship between depression and degree of cognitive impairment was evident at 3 months poststroke, there was a

clear relationship between the two factors at 6 months poststroke. Downhill and Robinson (1994) found that while the relationship between depression and cognitive impairment was strongest in the acute poststroke stage, some form of this relationship was evident for the entire first year following the stroke. However, this relationship was only found in patients with left hemisphere strokes. Furthermore, the bi-directional interaction between depression and cognitive impairment was such that the patients experiencing both depressive symptoms and cognitive impairment remained depressed for a longer duration than the depressed patients who had no cognitive impairment. Robinson et al. (1986) reported similar results; in their examination of patients with left hemisphere strokes they found that all depressed stroke patients were cognitively impaired during the acute poststroke stage, and only the non-depressed stroke patients had improved in their cognitive functioning by 6 months poststroke.

Within the months following stroke, the nature and prevalence of PSD appears to change from its initial presentation in the acute poststroke stage. While the depression experienced in the acute poststroke stage may have organic origins, it is possible that PSD takes on a reactive form later in the poststroke period, as indicated by the development of a relationship between depression and physical and/or cognitive impairment. The aim of this follow-up assessment was to examine the prevalence and associated factors of PSD at 3 months poststroke, in turn providing possible hypotheses towards a better understanding of this emotional disorder in stroke patients.

Method

Participants

At the 3-month follow-up, 7 of the 103 stroke patients originally selected for inclusion in the study were deceased, 5 were either unable or unwilling to take part in the follow-up assessment, and 3 were currently receiving medication as treatment for depression. One additional patient was excluded due to their admission to hospital as the result of a subsequent stroke since the initial assessment. Screening for the presence of aphasia was undertaken by administration of the short-form of the Minnesota Differential Diagnosis of Aphasia Test (Powell et al., 1980). Results of the aphasia test indicated that 8 patients were too aphasic to provide meaningful responses to the assessment procedures. The remaining 79 patients constituted the final sample for the 3-month follow-up assessment.

The 79 stroke patients were assessed, on average, at 94 days poststroke (*SD* = 5, range = 71 - 114). The average age of the patients at the time of the stroke was 74 years (*SD* = 7, range = 60 - 94). Forty-three (54%) patients were female, and 36 (46%) patients were male. Seventy-six (96%) patients were right-handed, and 3 (4%) patients were left-handed. Sixty-seven (85%) patients were retired, 6 (8%) patients were in paid employment, and 6 (8%) patients gave their occupation as "homemaker".

Seventy-five (94%) of the 79 stroke patients had CT scans. The CT scans of these 75 patients, and clinical examination of the remaining 4 patients, determined that 44 (56%) patients had right hemisphere strokes, 31 (39%) patients had strokes in the left hemisphere, 2 (3%) patients had cerebellar strokes, 1 (1%) patient had a brainstem stroke, and 1 (1%) patient had a bilateral stroke. Sixty (76%) patients did

not have a personal history of previous stroke. Nineteen (24%) patients had experienced at least one previous stroke, with 2 of these latter 19 patients having had two previous strokes.

Measures

In comparison with the assessment completed at 1 month poststroke, a more extensive assessment of poststroke functioning was conducted at 3 months poststroke. The measures used at the 3-month follow-up were as follows:

Cognitive measures. The Cognitive Assessment Scale (CAS) from the Clifton Assessment Procedures for the Elderly (Pattie & Gilleard, 1979) was administered to provide an overall assessment of cognitive functioning. Four subtests from the Wechsler Adult Intelligence Scale-Revised (WAIS-R) were also administered (Wechsler, 1981). Scores on the Vocabulary and Block Design subtests of the WAIS-R were combined to provide a current level of intellectual functioning (Full Scale IQ; FSIQ) using the tables provided by Brooker and Cyr (1986). The Digit Span subtest of the WAIS-R was used to assess simple attention abilities. The Digit Symbol subtest of the WAIS-R was used to measure complex attention abilities. Premorbid level of intelligence was assessed by administration of the National Adult Reading Test (NART; Nelson, 1982). Verbal fluency was assessed by administration of the Controlled Oral Word Association Test (COWA; Spreen & Strauss, 1991). Immediate and delayed verbal memory were assessed by administration of the Verbal Paired Associates (VPA)-immediate recall, and VPA-delayed recall subtests from the Wechsler Memory Scale-Revised (Wechsler, 1987).

Self-report questionnaires. Physical functioning was assessed by administration of the Barthel ADL Index (Wade & Langton-Hewer, 1987). The

Nottingham Extended ADL Index (Nouri & Lincoln, 1987) was administered as a measure of the patient's independence within the home. Oral administration of the Geriatric Depression Scale (GDS; Yesavage et al., 1983) assessed level of depression, and the 28-item version of the General Health Questionnaire (GHQ-28; Goldberg, 1972) was administered to determine the presence of psychological disorder.

Procedure and Data Analysis

Each 3-month follow-up assessment took approximately 1.5 hours to complete. Assessments took place at the patient's place of residence.

Data was described using the following procedures: mean (*M*), standard deviation (*SD*), and range. Statistical analysis was completed using the following procedures: independent *t* test, *t* test for unequal variance estimates, Pearson product-moment correlation, point-biserial correlation (r_{pb}; used where one variable was dichotomous), and hierarchical regression.

Results

Physical Functioning

The Barthel ADL Index was completed for all patients. Responses to the Barthel ADL Index indicated that 35 (44%) patients were physically independent, 27 (34%) had mild physical impairment, 9 (11%) had moderate impairment, and 8 (10%) patients had severe impairment. No patients had very severe physical impairment.

The Nottingham Extended ADL Index was completed for all patients. Responses to the Nottingham Extended ADL Index indicated that 6 (8%) patients performed all of the mobility tasks on their own, 54 (68%) patients performed some of the mobility tasks on their own, and 19 (24%) patients did not perform any of the

mobility tasks on their own. All of the patients performed at least some of the kitchen tasks on their own, with 24 (30%) performing all of the kitchen tasks on their own, and 55 (70%) performing some of the kitchen tasks on their own. Six (8%) patients performed all of the domestic tasks on their own, 46 (58%) patients performed some of the domestic tasks on their own, and 27 (34%) patients did not perform any of the domestic tasks on their own. Seven (9%) patients performed all of the leisure activities on their own, 70 (89%) performed some of the leisure activities on their own, and 2 (3%) patients did not perform any of the leisure activities on their own.

Affective Functioning

All of the patients completed the GDS. Forty-eight (61%) patients had no clinically significant symptoms of depression. Twenty-seven (34%) patients reported symptoms of mild depression, and 4 (5%) patients reported symptoms of severe depression.

All patients completed the GHQ-28. Responses to the GHQ-28 indicated that 13 (16%) patients reported clinically significant symptoms of psychological disorder. The remaining 66 (84%) patients had no clinically significant symptoms of psychological disorder.

Cognitive Functioning

Eight patients were unable to complete the CAS due to sight and/or writing impairments. Performance on the CAS indicated that 49 (69%) of the remaining 71 patients had no impairment of basic cognitive functioning, while 14 (20%) patients had mild impairment, and 8 (11%) patients had moderate impairment. No patients had marked or severe cognitive impairment.

Due to sight impairments, 5 patients were unable to complete the NART.

Three of these 5 patients were also unable to complete the Block Design subtest. One additional patient refused to complete both the Block Design and Vocabulary subtests. The remaining 73 patients were assessed with regard to their estimated premorbid and current FSIQ. The average estimated premorbid IQ for the patients was 99 (SD = 11, range = 69 - 121), which was significantly higher than the obtained FSIQ of 78 (SD = 15, range = 55 - 111), t (72) = -12.93, $p < .000001$.

Table 5 shows the degree of impairment in tests of attention, memory, and verbal fluency. All of the patients completed the Digit Span subtest. Due to sight and writing impairments, it was not possible to administer the Digit Symbol subtest to 12 patients, and 1 additional patient refused to complete the test. Therefore it was possible to assess the simple attention abilities of all 79 patients, and the complex attention abilities of 66 patients. Scaled scores on the Digit Span subtest ranged from 2 - 17, with an average score of 8 (SD = 3). Results of the Digit Span subtest indicated that 23 (30%) patients had mild impairment of simple attention, and 9 (11%) patients had severe impairment. Forty-seven (59%) of the 79 patients had no impairment of simple attention. Scaled scores on the Digit Symbol subtest ranged from 2 - 11, with an average score of 4 (SD = 2). Results of the Digit Symbol subtest indicated that 20 (30%) patients had mild impairment, and 39 (59%) patients had severe impairment of complex attention. Only 7 (11%) patients presented with no impairment of complex attention.

Due to the high level of difficulty of the VPA subtests, 3 patients were unable to complete the measures of immediate and delayed verbal memory. One additional patient refused to complete the delayed verbal recall subtest. Therefore the immediate verbal memory of 76 patients was assessed, and the delayed verbal memory of 75

Table 5

Degree of Clinically Significant Cognitive Impairment at 3 Months Poststroke

Aspects of Cognitive Function	*N*	Mild *n* (%)	Severe *n* (%)	Total *n* (%)
Simple attention	79	23 (30%)	9 (11%)	32 (41%)
Complex attention	66	20 (30%)	39 (59%)	59 (89%)
Immediate verbal memory	76	30 (40%)	14 (18%)	44 (58%)
Delayed verbal memory	75	17 (23%)	17 (23%)	34 (45%)
Verbal fluency	79	34 (43%)	18 (23%)	52 (66%)

patients was assessed. Scaled scores on the immediate verbal recall subtest ranged from 2 - 14, with an average score of 7 (SD = 3). Results indicated that 30 (40%) patients had mild impairment, and 14 (18%) patients had severe impairment of immediate verbal memory. Thirty-two (42%) patients had no impairment of immediate verbal memory. Scaled scores on the delayed verbal recall subtest ranged from 2 - 14, with an average score of 8 (SD = 3). Results indicated that 17 (23%) patients had mild impairment, and 17 (23%) patients had severe impairment of delayed verbal memory. Forty-one (55%) patients had no impairment of delayed verbal memory. Therefore, more patients had impairment of immediate verbal memory (58%) than impairment of delayed verbal memory (45%). However, while

the level of impairment on the delayed verbal recall task was distributed evenly between mild and severe impairment (23% each), a higher proportion of the patients with impairment of immediate verbal memory demonstrated mild impairment (40%) as opposed to severe impairment (18%).

All of the patients completed the COWA. Scaled scores on the COWA ranged from 2 - 12, with an average score of 6 ($SD = 3$). As indicated in Table 5, results from the COWA indicated that 34 (43%) patients had mild impairment, and 18 (23%) patients had severe impairment of verbal fluency. Twenty-seven (34%) patients had no impairment of verbal fluency.

In total, 15 of the 79 stroke patients were unable to complete all measures of cognitive functioning due to sight or writing impairments, and 1 additional patient refused to complete all tests. These 16 patients were therefore excluded from the correlational and regression analyses, as information regarding their performance on all tests was not available.

Correlational Analysis

In addition to the 16 patients excluded from the correlational and regression analyses as they were either unable or unwilling to complete all cognitive measures, 4 patients were excluded on the basis that their lesions were not located in either the left or right hemisphere (i.e., cerebellum, bilateral, or brainstem strokes). Excluding these patients was necessary as the analysis included determining whether there was a relationship between either left or right hemisphere strokes, and PSD. Therefore 59 stroke patients were included in the correlational and regression analyses. Following the method described by Tabachnick and Fidell (1989), *t* tests were used to determine whether the 20 patients who were excluded from further analysis were significantly

more depressed than the 59 patients included in the analyses. The excluded patients did not differ from those included in the analyses on any of the measures of affective functioning, GDS: t (77) = -0.05, p >.05; GHQ-28, somatic symptoms: t (77) = -0.07, p > .05; GHQ-28, anxiety/insomnia: t (77) = 1.06, p > .05; GHQ-28, social dysfunction: t (71.17) = 1.10, p > .05; GHQ-28, severe depression: t (77) = 0.63, p > .05.

Correlational analysis determined the degree of relationships between the variables. These results are displayed in Table 6.

Patients who had experienced at least one previous stroke were more likely to report symptoms of PSD, as measured by the GDS (r_{pb} = .53, p < .001). PSD was significantly correlated with physical impairment as measured by the Nottingham Extended ADL Index (r = -.33, p = .011), but no significant relationship was evident between PSD and the Barthel ADL Index (p > .05). Symptoms of PSD were also more likely to be reported by patients demonstrating impairment of both simple attention abilities (Digit Span subtest; r = -.26, p = .044), and complex attention abilities (Digit Symbol subtest; r = -.30, p = .021). Therefore, patients with higher depression scores were more likely to have impaired functioning of extended ADL tasks, and impaired simple and complex attention abilities.

Somatic symptoms, as measured by the GHQ-28, were significantly correlated with older age (r = .29, p = .026). Somatic symptoms were also significantly correlated with physical functioning as measured by the Barthel ADL Index (r = -.33, p =.010), but there was no significant correlation between somatic symptoms and the Nottingham Extended ADL Index (p > .05). Somatic symptoms were not significantly correlated with any of the measures of cognitive functioning.

Table 6

Correlational Analysis of Individual Variables At 3 Months Poststroke ($N = 59$)

Variable	1	2	3	4	5	6	7	8	9
1. GDS: Depression	--								
2. GHQ: Somatic	.41*	--							
3. GHQ: Anxiety	.49*	.51*	--						
4. GHQ: Social	.44*	.53*	.46*	--					
5. GHQ: Depression	.63*	.46*	.52*	.52*	--				
6. Gender	-.01	-.18	-.03	.03	-.06	--			
7. Age	.24	.29*	-.02	.29*	.18	-.17	--		
8. Previous stroke	.53*	.07	.09	.17	.18	.01	.27*	--	
9. Hemisphere	.15	-.22	-.005	.01	.13	.19	-.16	.09	--
10. ADL (Barthel)	-.25	-.33*	-.29*	-.24	-.13	.27*	-.35*	-.10	.28*
11. ADL (Nottingham)	-.33*	-.23	-.26*	-.17	-.17	.16	-.30*	-.11	.11
12. Basic cognition	-.13	-.21	-.07	-.22	-.06	.10	-.35*	-.02	.03
13. FSIQ	-.01	-.01	-.18	.12	.19	-.05	.14	.07	.07
14. Simple attention	-.26*	-.06	-.17	-.16	-.15	-.07	-.0003	.13	.05
15. Complex attention	-.30*	-.21	-.16	-.10	-.01	-.11	-.45*	-.15	.14
16. Immediate memory	-.23	-.06	-.31*	-.22	-.26*	-.29*	-.15	.06	-.09
17. Delayed memory	-.08	.11	-.22	-.16	-.09	-.25	-.03	.04	-.06
18. Verbal fluency	-.17	-.01	-.12	-.19	-.07	-.13	-.16	-.05	-.18

Table 6 (continued)

Correlational Analysis of Individual Variables at 3 Months Poststroke ($N = 59$)

Variable	10	11	12	13	14	15	16	17	18
10. ADL (Barthel)	--								
11. ADL (Nottingham)	.82*	--							
12. Basic cognition	.48*	.49*	--						
13. FSIQ	.27*	.25	.28*	--					
14. Simple attention	.11	.15	.35*	.32*	--				
15. Complex attention	.53*	.55*	.33*	.45*	.22	--			
16. Immediate memory	.03	.14	.25	.24	.54*	.26	--		
17. Delayed memory	-.09	.04	.13	.20	.38*	.16	.71*	--	
18. Verbal fluency	.21	.38*	.40*	.25	.50*	.26*	.59*	.50*	--

Note. ADL = Activities of daily living; FSIQ = Full Scale IQ; GDS = Geriatric Depression Scale; GHQ = 28-item version of the General Health Questionnaire.

* $p < .05$.

Anxiety/insomnia, as measured by the GHQ-28, was significantly correlated with physical impairment as measured by the Barthel ADL Index (r = -.29, p = .027), and the Nottingham Extended ADL Index (r = -.26, p = .045). Symptoms of anxiety/insomnia were also significantly correlated with impairment of immediate verbal memory (r = -.31, p = .016).

Social dysfunction, as measured by the GHQ-28, was significantly correlated with older age (r = .29, p = .027), but not with any other predictor variables. Severe depression, as measured by the GHQ-28, was significantly correlated with impairment of immediate verbal memory (r = -.26, p = .044).

Due to the high correlation between the two measures of physical functioning (Barthel ADL Index, and Nottingham Extended ADL Index; r = .82, p < .001), the scores for the two measures were combined to form one total score of physical functioning for use in the regression analyses, as is recommended by Tabachnick and Fidell (1989). In addition, the scores for the immediate and delayed verbal recall subtests were also highly correlated with each other (r = .71, p < .001), and were combined to form one score of Verbal Memory for use in the regression analyses.

Regression Analyses

Hierarchical regressions were performed on the outcome variables of PSD (as measured by the GDS), and the four psychological disorders represented by the scales of the GHQ-28. The regression analyses were used to determine if demographic variables (gender and age), medical variables (previous stroke and hemispheric location of the lesion), level of physical functioning (as measured by the combined scores of the Barthel ADL Index and the Nottingham Extended ADL), and/or cognitive impairment, were related to PSD and/or the four psychological disorders, at

3 months poststroke. Aspects of cognitive functioning included in the regression analyses were basic cognition (CAS), FSIQ, simple attention (Digit Span), complex attention (Digit Symbol), verbal memory, and verbal fluency (COWA).

Depression. Table 7 displays the unstandardised (*B*) and standardised (Beta) regression coefficients, R^2 for step 1, and the change in R^2 after entry of each of the additional blocks of independent variables.

At step 1, the demographic variables of age and gender explained only 6% of the variance in the GDS, $F(2, 56) = 1.79, p > .05$. At step 2, the demographic and medical variables explained a total of 31% of the variance in the GDS, $F(4, 54) = 5.94, p = .000487$, and the medical variables alone accounted for 25% of this variance. The medical variable of previous stroke made a unique contribution to the amount of variance accounted for at this step ($p = .000160$), and the addition of the medical variables resulted in a significant increment in R^2 ($p = .000280$).

At step 3, the demographic, medical, and physical variables explained a total of 38% of the variance in the GDS, $F(5, 53) = 6.36, p = .000107$, and the physical variable alone accounted for 7% of the variance at this step. The addition of the physical variable resulted in a significant increment in R^2 ($p = .018756$). At step 4, the cognitive variables alone accounted for 16% of the variance in the GDS, and the addition of the cognitive variables resulted in a significant increment in R^2 ($p = .026377$). The cognitive measure of simple attention (Digit Span) made a unique contribution to the amount of variance accounted for at this step ($p = .003028$). With all the independent variables in the equation, the regression model explained 53% of the variance in the GDS, $F(11, 47) = 4.89, p = .000053$.

Therefore, in total, the 11 independent variables explained 53% of the

Table 7

Summary of Hierarchical Regression Analysis for Variables Predicting PSD at 3 Months Poststroke, as Measured by the GDS ($N = 59$)

	Variable	*B*	*SE B*	Beta
Step 1	Gender	0.29	1.38	.03
	Age	0.18	0.10	.25
Step 2	Previous stroke	5.69	1.40	.48 *
	Hemisphere	1.31	1.24	.12
Step 3	Physical functioning	-0.08	0.03	-.28 *
Step 4	Basic cognition	0.21	0.19	.14
	FSIQ	0.05	0.05	.12
	Simple attention	-0.86	0.28	-.41 *
	Complex attention	-0.05	0.10	-.09
	Verbal memory	-0.17	0.15	-.16
	Verbal fluency	0.14	0.08	.26

Note. FSIQ = Full Scale IQ; GDS = Geriatric Depression Scale.

$R^2 = .06$ for Step 1 ($p > .05$);

change in $R^2 = .25$ for Step 2 ($p = .000280$);

change in $R^2 = .07$ for Step 3 ($p = .018756$);

change in $R^2 = .16$ for Step 4 ($p = .026377$).

* $p < .05$.

variance in the GDS. The variables included in the regression model did significantly predict PSD. However, these results indicate that the strongest predictors of PSD at 3 months poststroke were previous stroke, physical impairment, and impairment of simple attention abilities.

Somatic symptoms (GHQ-28, scale A). Table 8 displays the unstandardised (*B*) and standardised (Beta) regression coefficients, R^2 for step 1, and the change in R^2 after entry of each of the additional blocks of independent variables.

At step 1, the demographic variables of age and gender explained 10% of the variance in the measure of somatic symptoms, $F(2, 56) = 3.15, p > .05$. Although the amount of variance explained by the regression equation at step 1 was not significant, the individual variable of age produced a significant result ($p = .041668$). At step 2, the demographic and medical variables explained a total of 13% of the variance in the measure of somatic symptoms, $F(4, 54) = 1.93, p > .05$, and the medical variables alone accounted for 2% of the variance at this step. At step 3, the demographic, medical, and physical variables explained a total of 15% of the variance in the measure of somatic symptoms, $F(5, 53) = 1.81, p > .05$, and the physical variable alone accounted for 2% of the variance at this step. At step 4, the cognitive variables alone accounted for 1% of the variance in the measure of somatic symptoms. With all of the independent variables in the equation, the regression model explained only 16% of the variance in the measure of somatic symptoms, $F(11, 47) = 0.81, p > .05$.

Therefore, in total, the 11 independent variables explained 16% of the variance in the measure of somatic symptoms. Although the individual variable of age was significant, the full regression model did not significantly predict somatic symptoms at 3 months poststroke.

Table 8

Summary of Hierarchical Regression Analysis for Variables Predicting Somatic Symptoms at 3 Months Poststroke, as Measured by the GHQ-28, scale A ($N = 59$)

	Variable	*B*	*SE B*	Beta
Step 1	Gender	-0.80	0.78	-.13
	Age	0.11	0.05	.27*
Step 2	Previous stroke	0.18	0.91	.03
	Hemisphere	-0.98	0.81	-.16
Step 3	Physical functioning	-0.02	0.02	-.16
Step 4	Basic cognition	-0.07	0.15	-.09
	FSIQ	0.01	0.04	.05
	Simple attention	-0.08	0.21	-.06
	Complex attention	-0.02	0.08	-.05
	Verbal memory	-0.02	0.11	-.03
	Verbal fluency	0.04	0.06	.12

Note. FSIQ = Full Scale IQ; GHQ-28 = 28-item version of the General Health Questionnaire.

$R^2 = .10$ for Step 1 ($p > .05$);

change in $R^2 = .02$ for Step 2 ($p > .05$);

change in $R^2 = .02$ for Step 3 ($p > .05$);

change in $R^2 = .01$ for Step 4 ($p > .05$).

* $p < .05$.

Anxiety/insomnia (GHQ-28, scale B). Table 9 displays the unstandardised (*B*) and standardised (Beta) regression coefficients, R^2 for step 1, and the change in R^2 after entry of each of the additional blocks of independent variables.

At step 1, the demographic variables of age and gender explained less than 1% (0.1%) of the variance in the measure of anxiety/insomnia, $F(2, 56) = 0.04, p > .05$. At step 2, the demographic and medical variables explained a total of 1% of the variance in the measure of anxiety/insomnia, $F(4, 54) = 0.17, p > .05$, and the medical variables alone accounted for 1% of the variance at this step. At step 3, the demographic, medical, and physical variables explained a total of 10% of the variance in the measure of anxiety/insomnia, $F(5, 53) = 1.15, p > .05$, and the physical variable alone accounted for 9% of the variance at this step. The addition of the physical variable resulted in a significant increment in R^2 ($p = .029807$). At step 4, the cognitive variables alone accounted for 15% of the variance in the measure of anxiety/insomnia. The increment in R^2 at step 4 was not significantly different from zero ($p > .05$), although the individual variable of verbal memory made a unique contribution to the amount of variance accounted for at this step ($p = .012507$). With all of the independent variables in the equation, the regression model explained 24% of the variance in the measure of anxiety/insomnia, $F(11, 47) = 1.38, p > .05$.

Therefore, in total, the 11 independent variables explained 24% of the variance in the measure of anxiety/insomnia. Although individual variables were significant, the full regression model did not significantly predict anxiety/insomnia at 3 months poststroke.

Social dysfunction (GHQ-28, scale C). Table 10 displays the unstandardised (*B*) and standardised (Beta) regression coefficients, R^2 for step 1, and the change in

Table 9

Summary of Hierarchical Regression Analysis for Variables Predicting Anxiety/ Insomnia at 3 Months Poststroke, as Measured by the GHQ-28, scale B ($N = 59$)

	Variable	*B*	*SE B*	Beta
Step 1	Gender	-0.17	0.76	-.03
	Age	-0.01	0.05	-.02
Step 2	Previous stroke	0.70	0.89	.11
	Hemisphere	-0.10	0.79	-.02
Step 3	Physical functioning	-0.05	0.02	-.31*
Step 4	Basic cognition	0.09	0.13	.12
	FSIQ	-0.01	0.03	-.07
	Simple attention	-0.08	0.19	-.07
	Complex attention	0.02	0.07	.07
	Verbal memory	-0.26	0.10	-.47 *
	Verbal fluency	0.08	0.06	.28

Note. FSIQ = Full Scale IQ; GHQ-28 = 28-item version of the General Health Questionnaire.

$R^2 = .001$ for Step 1 ($p > .05$);

change in $R^2 = .01$ for Step 2 ($p > .05$);

change in $R^2 = .08$ for Step 3 ($p = .029807$);

change in $R^2 = .15$ for Step 4 ($p > .05$).

* $p < .05$.

Table 10

Summary of Hierarchical Regression Analysis for Variables Predicting Social Dysfunction at 3 Months Poststroke, as Measured by the GHQ-28, scale C ($N = 59$)

	Variable	*B*	*SE B*	Beta
Step 1	Gender	0.34	0.54	.08
	Age	0.09	0.04	.30 *
Step 2	Previous stroke	0.43	0.63	.09
	Hemisphere	0.15	0.56	.04
Step 3	Physical functioning	-0.01	0.01	-.13
Step 4	Basic cognition	-0.06	0.10	-.10
	FSIQ	0.03	0.03	.18
	Simple attention	-0.11	0.14	-.13
	Complex attention	0.03	0.05	.12
	Verbal memory	-0.07	0.08	-.17
	Verbal fluency	0.01	0.04	.06

Note. FSIQ = Full Scale IQ; GHQ-28 = 28-item version of the General Health Questionnaire.

$R^2 = .09$ for Step 1 ($p > .05$);

change in $R^2 = .01$ for Step 2 ($p > .05$);

change in $R^2 = .01$ for Step 3 ($p > .05$);

change in $R^2 = .08$ for Step 4 ($p > .05$).

* $p < .05$.

R^2 after entry of each of the additional blocks of independent variables.

At step 1, the demographic variables of age and gender explained 9% of the variance in the measure of social dysfunction, F (2, 56) = 2.76, $p > .05$. Although the amount of variance explained by the regression equation at step 1 was not significant, the individual variable of age produced a significant result ($p = .023073$). At step 2, the demographic and medical variables explained a total of 10% of the variance in the measure of social dysfunction, F (4, 54) = 1.49, $p > .05$, and the medical variables alone accounted for 1% of the variance at this step. At step 3, the demographic, medical, and physical variables explained a total of 11% of the variance in the measure of social dysfunction, F (5, 53) = 1.36, $p > .05$, and the physical variable alone accounted for 1% of the variance at this step. At step 4, the cognitive variables alone accounted for 8% of the variance in the measure of social dysfunction. With all of the independent variables in the equation, the regression model explained 20% of the variance in the measure of social dysfunction, F (11, 47) = 1.04, $p > .05$.

Therefore, in total, the 11 independent variables explained 20% of the variance in the measure of social dysfunction. Although the individual variable of age was significant, the full regression model did not significantly predict social dysfunction at 3 months poststroke.

Severe depression (GHQ-28, scale D). Table 11 displays the unstandardised (B) and standardised (Beta) regression coefficients, R^2 for step 1, and the change in R^2 after entry of each of the additional blocks of independent variables.

At step 1, the demographic variables of age and gender explained 3% of the variance in the measure of severe depression, F (2, 56) = 0.96, $p > .05$. At step 2, the demographic and medical variables explained a total of 8% of the variance in the

Table 11

Summary of Hierarchical Regression Analysis for Variables Predicting Severe Depression at 3 Months Poststroke, as Measured by the GHQ-28, scale D ($N = 59$)

	Variable	*B*	*SE B*	Beta
Step 1	Gender	-0.20	0.83	-.03
	Age	0.08	0.06	.17
Step 2	Previous stroke	0.88	0.96	.13
	Hemisphere	0.99	0.85	.16
Step 3	Physical functioning	-0.02	0.02	-.13
Step 4	Basic cognition	0.08	0.14	.09
	FSIQ	0.05	0.04	.23
	Simple attention	-0.31	0.20	-.25
	Complex attention	0.06	0.07	.17
	Verbal memory	-0.22	0.11	-.37 *
	Verbal fluency	0.11	0.06	.33

Note. FSIQ = Full Scale IQ; GHQ-28 = 28-item version of the General Health Questionnaire.

$R^2 = .03$ for Step 1 ($p > .05$);

change in $R^2 = .04$ for Step 2 ($p > .05$);

change in $R^2 = .02$ for Step 3 ($p > .05$);

change in $R^2 = .19$ for Step 4 ($p > .05$).

* $p < .05$.

measure of severe depression, F (4, 54) = 1.12, $p > .05$, and the medical variables alone accounted for 4% of the variance at this step. At step 3, the demographic, medical, and physical variables explained a total of 9% of the variance in the measure of severe depression, F (5, 53) = 1.07, $p > .05$, and the physical variable alone accounted for 2% of the variance at this step. At step 4, the cognitive variables alone accounted for 19% of the variance in the measure of severe depression. The increment in R^2 at step 4 was not significantly different from zero ($p > .05$), although the individual variable of verbal memory made a unique contribution to the amount of variance accounted for at this step ($p = .042187$). With all of the independent variables in the equation, the regression model explained 28% of the variance in the measure of severe depression, F (11, 47) = 1.66, $p > .05$.

Therefore, in total, the 11 independent variables explained 28% of the variance in the measure of severe depression. Although the individual variable of verbal memory was significant, the full regression model did not significantly predict severe depression at 3 months poststroke.

Discussion

This follow-up assessment determined that 39% of these stroke patients reported clinically significant symptoms of depression at 3 months poststroke. It also confirmed that the relationship between PSD and lesion hemisphere was no longer evident, while the relationship between PSD and previous strokes continued beyond the acute poststroke stage. These results also indicate the emergence of relationships between PSD and levels of physical and cognitive impairment. This study failed to find a significant relationship between PSD and either gender or age at 3 months

poststroke.

The rate of depression among these patients was higher than that reported in other studies; for example Astrom, Adolfsson, et al. (1993) reported that 31% of their stroke patients reported symptoms of PSD at 3 months poststroke. In addition, in the present study, symptoms of mild depression were evident in 34% of the patients, while 5% reported symptoms of severe depression. In addition to this high prevalence of depression, 16% of these patients presented with clinically significant symptoms of psychological disorder. Although few patients had symptoms of severe depression, the high prevalence of PSD and clinically significant psychological disorder in these patients at 3 months poststroke is disturbing.

The results of the Nottingham Extended ADL Index show that the majority of patients performed at least some of the mobility, kitchen, domestic, and leisure activities. However, over half of the patients were physically impaired on basic activities of daily living as measured by the Barthel ADL Index. Fewer patients (31%) had impairment of basic cognitive functioning. However, further comprehensive assessment of cognitive functioning indicated that a greater number of patients (41 - 89%) had impairment of more complex tasks of cognitive functioning. The poststroke decrease in IQ of these patients further demonstrated this cognitive impairment; comparisons of the estimated premorbid IQ and the poststroke IQ of these patients indicated a substantial decrease in intellectual functioning. Therefore, cognitive impairment was clearly evident in these patients at 3 months poststroke, consistent with that reported previously in the literature (e.g., Tatemichi et al., 1994).

Depression was significantly correlated with previous stroke, simple and complex attention, and physical functioning as measured by the Nottingham Extended

ADL Index. No relationship was evident between PSD and physical functioning as measured by the Barthel ADL Index. As impairment is still evident on the Barthel ADL Index, and if PSD is a reaction to impairment, this finding indicates that at 3 months poststroke a patient's ability to perform basic self-care activities (as measured by the Barthel ADL Index) does not have as much detrimental effect on their emotional wellbeing as their ability to perform a wider range of daily tasks which contribute to their quality of life and their involvement in community and family life (as measured by the Nottingham Extended ADL Index). It is possible that these patients have adapted to their inability to perform all of their self-care activities by 3 months poststroke, but are still affected by their failure to perform tasks represented by the extended ADL measure. Alternatively, if the depression leads to the physical impairment, it is possible that depressed patients are less likely to perform the wider range of tasks of daily life that may impact on their overall quality of life, but continue to perform their basic self-care activities. Further analysis of the physical activity of these patients at 12 months poststroke may help to determine which of these explanations may be responsible for this finding. Regardless, these findings suggest that physical impairment should be assessed in a more complex manner than that provided by measures of basic ADL functioning.

Interestingly, none of the regression models predicted any of the psychological disorders measured by the GHQ-28, and the correlational analysis revealed very few relationships between any of the GHQ-28 scales and the demographic, medical, physical, or cognitive variables. Physical impairment was related to somatic symptoms and anxiety/insomnia, although the relationship between physical impairment and somatic symptoms did not reach significance in the regression analysis. The

relationship between physical impairment and somatic symptoms indicates that patients with physical impairment believe that they are not keeping in good physical health. Furthermore, as anxiety and depression are both considered to be mood disorders, and there is a relationship between depression and physical impairment, the relationship between physical impairment and anxiety/insomnia is understandable. In addition to these relationships, older age was related to both somatic symptoms and social dysfunction. The first of these two relationships is easily understood as older patients are more likely to complain of somatic symptoms simply because their age determines that they are more likely to have somatic problems. The nature of the social dysfunction subscale provides an explanation for the latter relationship as it refers to the patient's satisfaction in their ability to complete tasks. That is, it is possible that older patients are limited in their ability to complete daily activities (due to physical disabilities, or an inability to drive, for example), and as a result may feel less fulfilled by their daily life.

The only measure of cognitive functioning that was related to any of the psychological disorders measured by the GHQ-28 scales was verbal memory, which was related to the anxiety/insomnia and severe depression subscales. Although this suggests a relationship between memory and mood disorders in stroke patients, no relationship was evident between depression (as measured by the GDS), and memory.

Irrespective of these relationships between the GHQ-28 subscales and individual variables, the regression analyses indicated that there was an absence of any significant predictive relationship between the GHQ-28 subscales and groups of predictor variables. Perhaps there is a relationship between symptoms of psychological disorder and factors not assessed in this study, such as social support.

The relationships found in this study between PSD and associated factors are consistent with many of the findings reported in the literature to date. The fact that neither gender nor age were significant predictors of PSD is in agreement with much of the literature (e.g., Astrom, Adolfsson, et al., 1993; Robinson, Starr, Lipsey, et al., 1984; Shima et al., 1994), and the involvement of lesion location in PSD has produced conflicting results in the research to date. The findings of the present study clearly support the findings of Astrom, Adolfsson, et al. (1993); that is, although there was a relationship between PSD and hemispheric location of the lesion at the acute poststroke stage, no such relationship is evident by 3 months poststroke.

The continuing relationship between PSD and the occurrence of a previous stroke raises several issues. As mentioned previously, this relationship may be a normal interaction; if the individual has survived a previous stroke, they may be more likely to respond to the subsequent stroke in a depressed manner as they already know what to expect, and as the first stroke increased their chances of having a second stroke, the chances of having additional strokes increase with each subsequent stroke (Jorgensen et al., 1997). As PSD was also related to previous strokes at the acute poststroke stage, the possible impact of the longitudinal effects of this relationship should be of concern to researchers and practitioners. As the involvement of a history of stroke in PSD is rarely examined, assessment of the present group of stroke patients at 12 months poststroke will help clarify the extent of this relationship.

Consistent with the findings of previous studies (Astrom, Adolfsson, et al., 1993; Robinson, Starr, Lipsey, et al., 1984), this study found a significant relationship between depression and the level of physical functioning. It is possible that PSD may be a reaction to physical impairment. If this is the case, then it is possible that the

depression may lessen as the patient learns to cope with their impairments. Hence it is also possible that the depression will continue further into the poststroke period, particularly if the patient's physical functioning does not improve. Alternatively, if the relationship is bi-directional, the depression and physical impairment will exacerbate the effects of each other, and the patient may not recover their physical abilities due to the depression. Further examination of these patients at 12 months poststroke may provide a better understanding of this relationship.

While this study reported a relationship between the level of cognitive functioning and PSD at 3 months poststroke, the only aspect of cognitive functioning involved in this relationship was simple attention abilities. (Although correlational analysis suggested a relationship between PSD and both simple and complex attention abilities, the latter failed to reach significance in the regression analysis.) No other aspect of cognitive functioning was related to depression. These results indicate that discrepancies in the literature regarding the reported presence or absence of a relationship between cognitive impairment and PSD may be due to a failure to examine specific cognitive functions, such as attention. Accordingly, it is possible that a relationship between cognitive functioning and depression in these patients during the acute poststroke stage was not found because a basic cognitive measure was used to assess their level of cognitive functioning. However, it is more likely that no such relationship was evident due to the possible reactive nature of the depression; more simply, some patients were not depressed during the acute poststroke stage as they had not yet realised the extent of their cognitive impairment, yet some realisation of these impairments had occurred by 3 months poststroke.

In summary, symptoms of PSD were evident in 39% of the stroke patients at

3 months poststroke. There was no longer a relationship between PSD and left hemisphere strokes, while the relationship between previous stroke and depression remained. In addition, a clear relationship between PSD and physical and cognitive impairment emerged, although simple attention abilities was the only aspect of cognitive functioning evident in this relationship. These results indicate a reactive component of PSD, based on the assumption that by 3 months poststroke the patient is aware of their physical and cognitive limitations, and responds with depressive symptoms. Further examination of these patients at 12 months poststroke will determine whether these relationships exist on a longitudinal basis.

CHAPTER 5

PSD and Associated Factors at 12 Months Poststroke

The reported prevalence rates of PSD at 12 months poststroke range from approximately 16% (Astrom, Adolfsson, et al., 1993) to as high as 33% (Robinson et al., 1987). Irrespective of these discrepancies, the results of these studies indicate that PSD is still a concern 12 months after stroke.

At 12 months poststroke, research has indicated that some relationships are still evident between PSD and previously associated factors. Researchers examining depression at this stage of the poststroke period rarely mention specific relationships between depression and demographic factors, possibly indicating that no such relationships are evident. Consistent with this assumption, Astrom, Adolfsson, et al. (1993) failed to find a significant relationship between PSD and demographic characteristics at 12 months poststroke. However, Andersen et al. (1995) found that female stroke patients were more likely than their male counterparts to experience depression 12 months after their stroke.

Andersen et al. (1995) reported that PSD at 12 months poststroke was higher in patients who had experienced a previous stroke. These researchers also failed to find a significant relationship between lesion location and PSD at 12 months poststroke, consistent with the findings of Astrom, Adolfsson, et al. (1993) who reported that there was no relationship between PSD and hemispheric location of the lesion at or beyond 3 months poststroke. Contrary to these findings, Robinson and colleagues (Parikh et al., 1987; Robinson & Price, 1982) reported that a significant relationship between left hemisphere lesions and PSD was evident for the first 2 years

following stroke. However, these researchers emphasised that this finding was based on information from very few stroke patients. Therefore, although inconclusive, research to date indicates that PSD is not likely to be related to the hemispheric location of the lesion at 12 months poststroke.

Throughout the poststroke period the possible relationships between physical and/or cognitive functioning and PSD continue to be a main point of interest in much of the literature. Parikh et al. (1987) reported findings of a significant relationship between level of physical impairment and PSD at 12 months poststroke, although this relationship was somewhat weaker than had been reported at 6 months poststroke. Other researchers have failed to find a significant relationship between physical impairment and PSD at 12 months poststroke (Andersen et al., 1995; Astrom, Adolfsson, et al., 1993). As mentioned in the previous chapter, it is thought that the majority of patients reach the full potential of their physical recovery by 3 months poststroke (Jorgensen et al., 1995b), though less than half will fully recover their ADL abilities (Jorgensen et al., 1995a). Therefore the weakening of the relationship between PSD and physical impairment at 12 months poststroke is unlikely to be due to the patients having fully recovered to their prestroke level of physical functioning. Alternatively, it is possible that by 12 months poststroke many of the stroke patients have learnt to cope with their physical impairment, and/or have developed methods of compensating for their disability.

As was reported with physical functioning, the relationship between cognitive impairment and PSD also weakens by 12 months poststroke (Downhill & Robinson, 1994; Parikh et al., 1987). Downhill and Robinson found that the relationship between PSD and cognitive impairment was still evident in patients with left

hemisphere strokes at 12 months poststroke, although the relationship had been strongest in the acute poststroke stage. Other researchers have reported that no relationship exists between PSD and cognitive impairment at 12 months poststroke (Andersen et al., 1995; Parikh et al., 1987). As was suggested as an explanation of the weakening of the relationship between physical functioning and depression at 12 months poststroke, it is entirely possible that by 12 months poststroke the patients have learnt to cope with, and compensate for, their cognitive impairments. However, many cognitive impairments would be difficult to compensate for, as the patient would need to possess a reasonable level of cognitive abilities to learn how to compensate. Another possible explanation for the decrease in the strength of this relationship is that the cognitive functioning of the patients has improved, lessening the depression. Clearly further research is needed to fully explain the weakening relationships between PSD and both physical and cognitive impairment at 12 months poststroke.

This follow-up assessment aimed to determine the prevalence of depression at 12 months poststroke, and examine the relationships between PSD and associated factors. Previous research has indicated that levels of physical and cognitive impairment may not be related to depression at 12 months poststroke, despite evidence of these relationships earlier in the poststroke period. This assessment will examine this issue, together with determining if demographic and/or medical factors are involved in PSD at 12 months poststroke.

Method

Participants

At the 12-month follow-up, 20 of the 103 stroke patients originally selected for inclusion in the study were deceased, 7 patients were either unable or unwilling to take part in the follow-up assessment, and 2 patients were currently receiving medication as treatment for depression. Two patients were excluded as they had been admitted to hospital as a result of a subsequent stroke since the initial assessment. Screening for the presence of aphasia was undertaken by administration of the short-form of the Minnesota Differential Diagnosis of Aphasia Test (Powell et al., 1980). Results of the aphasia test indicated that 5 patients were too aphasic to provide meaningful responses to the assessment procedures. The remaining 67 patients constituted the final sample for the 12-month follow-up.

The 67 stroke patients were assessed, on average, at 367 days poststroke (*SD* = 5, range = 357 - 384). The average age of the patients at the time of the stroke was 74 years (*SD* = 7, range = 60 - 87). Thirty-five (52%) patients were female and 32 (48%) patients were male. Sixty-four (96%) patients were right-handed, and 3 (5%) patients were left-handed. Fifty-six (84%) patients were retired, 6 (9%) patients were in paid employment, and 5 (7%) patients gave their occupation as "homemaker".

Sixty-three (94%) of the 67 stroke patients had CT scans. The CT scans of these 63 patients, and clinical examination of the remaining 4 patients, determined that 35 (52%) patients had right hemisphere strokes, 28 (42%) patients had strokes in the left hemisphere, 2 (3%) patients had cerebellar strokes, 1 (1%) patient had a brainstem stroke, and 1 (1%) patient had a bilateral stroke. For 52 (78%) patients this was their first stroke, 15 (22%) patients had experienced at least one previous stroke,

with 2 of these latter 15 patients having had two previous strokes.

Measures

The measures included in the 12 month assessment were similar to those used in the 3-month follow-up. However, the National Adult Reading Test (NART; Nelson, 1982) was not administered at 12 months poststroke. Therefore, the measures used at the 12-month follow-up were as follows:

Cognitive measures. The Cognitive Assessment Scale (CAS) from the Clifton Assessment Procedures for the Elderly (Pattie & Gilleard, 1979) was administered to provide an overall assessment of cognitive functioning. Four subtests from the Wechsler Adult Intelligence Scale-Revised (WAIS-R) were also administered (Wechsler, 1981). Scores on the Vocabulary and Block Design subtests of the WAIS-R were combined to provide an estimate of the current level of intellectual functioning (Full Scale IQ; FSIQ) using the tables provided by Brooker and Cyr (1986). Scores from the administration of the NART (Nelson, 1982) at 3 months poststroke were used to estimate premorbid level of intelligence. The Digit Span subtest of the WAIS-R was used to measure simple attention abilities. The Digit Symbol subtest of the WAIS-R was used to measure complex attention abilities. Verbal fluency was assessed by administration of the Controlled Oral Word Association test (COWA; Spreen & Strauss, 1991). Immediate and delayed verbal memory were assessed by administration of the VPA-immediate recall, and VPA-delayed recall subtests from the Wechsler Memory Scale-Revised (Wechsler, 1987).

Self-report questionnaires. Physical functioning was assessed by administration of the Barthel ADL Index (Wade & Langton-Hewer, 1987). The Nottingham Extended ADL Index (Nouri & Lincoln, 1987) was administered as a

measure of the patients' independence within the home. Oral administration of the Geriatric Depression Scale (GDS; Yesavage et al., 1983) assessed level of depression, and the 28-item version of the General Health Questionnaire (GHQ-28; Goldberg, 1972) was administered to determine the presence of psychological disorder.

Procedure and Data Analysis

The 12-month follow-up assessments each took approximately 1.5 hours to complete. Each patient was assessed at their place of residence.

Data was described using the following procedures: mean (*M*), standard deviation (*SD*), and range. Statistical analyses were completed using the independent *t* test, *t* test for separate variance estimates, Pearson product-moment correlation, point-biserial correlation (r_{pb}; used where one variable was dichotomous), and hierarchical regression.

Results

Physical Functioning

The Barthel ADL Index was completed for all patients. Responses to the Barthel ADL Index indicated that 33 (49%) patients were physically independent, 23 (34%) patients had mild impairment, 5 (7%) patients had moderate impairment, 5 (7%) had severe impairment, and 1 (1%) patient had very severe physical impairment.

The Nottingham Extended ADL Index was completed for all patients. Responses to the Nottingham Extended ADL Index indicated that 11 (16%) patients performed all of the mobility tasks on their own, 44 (66%) performed some of the mobility tasks on their own, and 12 (18%) patients did not perform any of the mobility tasks on their own. Thirty (45%) patients performed all of the kitchen tasks

on their own, 36 (54%) performed some of the kitchen tasks on their own, and 1 (1%) patient did not perform any of the kitchen tasks on their own. Ten (15%) patients performed all of the domestic tasks on their own, while 34 (51%) performed only some of the domestic tasks on their own, and 23 (34%) patients did not perform any of the domestic tasks on their own. Nine (13%) patients performed all of the leisure activities on their own, while 55 (82%) performed only some of the leisure activities on their own, and 3 (4%) patients did not perform any of the leisure activities on their own.

Affective Functioning

All of the patients completed the GDS. Forty-five (67%) patients had no clinically significant symptoms of depression. Nineteen (28%) patients reported symptoms of mild depression and 3 (4%) patients reported symptoms of severe depression.

All of the patients completed the GHQ-28. Responses to the GHQ-28 indicated that 64 (96%) patients had no clinically significant symptoms of psychological disorder. The remaining 3 (4%) patients reported clinically significant symptoms of psychological disorder. Analysis of the responses of individual patients determined that the 3 patients reporting symptoms of severe depression on the GDS were not the same people as the 3 patients reporting symptoms of psychological disorder.

Cognitive Functioning

Six patients were unable to complete the CAS due to sight and/or writing impairments. Performance on the CAS indicated that 44 (72%) of the remaining 61 patients had no impairment of basic cognitive functioning. Eleven (18%) patients had

mild impairment, and 6 (10%) patients had moderate impairment of basic cognitive functioning. No patients had marked or severe impairment of basic cognitive functioning.

All of the patients completed the Vocabulary subtest. Due to sight impairments, 5 patients were unable to complete the Block Design subtest. Three of these 5 patients had been unable to complete the NART at 3 months poststroke due to sight impairments, and 1 patient had been unable to be assessed at the 3-month follow-up. The remaining 61 patients were assessed with regard to their estimated premorbid and current FSIQ. The average estimated premorbid IQ for the patients was 99 (SD = 12, range = 69 - 121), which was significantly higher than the obtained FSIQ of 79 (SD = 15, range = 55 - 114), t (60) = -12.08, $p < .000001$.

Table 12 shows the degree of impairment in tests of attention, memory, and verbal fluency. All 67 patients completed the Digit Span subtest. Due to sight and writing impairments, it was not possible to administer the Digit Symbol subtest to 14 patients. It was therefore possible to assess simple attention abilities of 67 patients, and the complex attention abilities of 53 patients. Scaled scores on the Digit Span subtest ranged from 2 - 18, with an average score of 8 (SD = 3). Results of the Digit Span subtest indicate that 16 (24%) patients had mild impairment, and 8 (12%) patients had severe impairment of simple attention abilities. Forty-three (64%) patients had no impairment of simple attention abilities. Scaled scores on the Digit Symbol subtest ranged from 2 - 13, with an average score of 5 (SD = 2). Results of the Digit Symbol subtest indicate that 15 (29%) patients had mild impairment, and 31 (58%) patients had severe impairment of complex attention abilities. Only 7 (13%) patients had no impairment of complex attention abilities.

Table 12

Degree of Clinically Significant Cognitive Impairment at 12 Months Poststroke

Aspect of Cognitive Function	*N*	Mild *n* (%)	Severe *n* (%)	Total *n* (%)
Simple attention	67	16 (24%)	8 (12%)	24 (36%)
Complex attention	53	15 (29%)	31 (58%)	46 (87%)
Immediate verbal memory	66	16 (24%)	13 (20%)	29 (44%)
Delayed verbal memory	65	17 (26%)	6 (9%)	23 (35%)
Verbal fluency	67	23 (35%)	15 (22%)	38 (57%)

Due to the high level of difficulty of the VPA subtests, it was not possible to administer this test to 1 of the 67 patients. One additional patient refused to complete the delayed verbal recall subtest. Therefore it was possible to assess the immediate verbal memory of 66 patients, and the delayed verbal memory of 65 patients. Scaled scores on the immediate verbal recall subtest ranged from 2 - 14, with an average score of 8 (SD = 3). Results of the immediate verbal recall subtest indicated that 16 (24%) patients had mild impairment, and 13 (20%) patients had severe impairment of immediate verbal memory. Thirty-seven (56%) patients had no impairment of immediate verbal memory. Scaled scores on the delayed verbal recall subtest ranged from 2 - 14, with an average score of 9 (SD = 3). Results of the delayed verbal recall

subtest indicated that 17 (26%) patients had mild impairment, and 6 (9%) patients had severe impairment of delayed verbal memory. Forty-two (65%) patients had no impairment of delayed verbal memory.

All of the 67 patients were able to complete the COWA. Scaled scores on the COWA ranged from 2 - 13, with an average score of 7 (*SD* = 3). As indicated in Table 12, 23 (35%) patients had mild impairment, and 15 (22%) patients had severe impairment of verbal fluency. Twenty-nine (43%) patients had no impairment of verbal fluency.

In total, 15 of the 67 stroke patients were unable to complete all measures of cognitive functioning due to sight or writing impairments, and one of these patients also refused to complete one of the cognitive measures. These 15 patients were therefore excluded from the correlational and regression analyses, as information was not available regarding their performance on all tests.

Correlational Analysis

In addition to the 15 patients excluded from the correlational and regression analyses as they were either unable or unwilling to complete all cognitive measures, 4 patients were excluded on the basis that their lesions were not located in either the left or right hemisphere (i.e., cerebellum, bilateral, or brainstem strokes). Excluding these patients was necessary as the analysis included determining whether there was a relationship between either left or right hemisphere strokes, and PSD. Therefore 48 stroke patients were included in the correlational and regression analyses. Following the method described by Tabachnick and Fidell (1989), *t* tests determined whether the 19 patients who were excluded from further analysis were significantly different from the 48 patients included in the analyses, with regard to affective functioning. The

excluded patients did not differ from those included in the analyses on any of the measures of affective functioning, GDS: t (65) = -0.89, p > .05; GHQ-28, somatic symptoms: t (23.55) = -0.65, p > .05; GHQ-28, anxiety/insomnia: t (21.60) = 0.19, p > .05; GHQ-28, social dysfunction: t (21.19) = -0.80, p > .05; GHQ-28, severe depression: t (20.45) = -0.65, p > .05.

Correlational analysis determined the degree of relationships between the variables. These results are displayed in Table 13.

Patients who had experienced a previous stroke were more likely to report symptoms of PSD, as measured by the GDS (r_{pb} = .46, p = .001). Scores on the GDS were also significantly correlated with physical impairment as measured by the Nottingham Extended ADL Index (r = -.32, p = .027), but no significant relationship was evident between PSD and scores on the Barthel ADL Index (p > .05). PSD was also significantly correlated with complex attention abilities (Digit Symbol subtest; r = -.40, p = .005).

Somatic symptoms, as measured by the GHQ-28, were significantly correlated with older age (r = .34, p = .018), and with impairment of complex attention (Digit Symbol subtest; r = -.32, p = .027).

Patients who had experienced a previous stroke were likely to report symptoms of anxiety/insomnia, as measured by the GHQ-28 (r_{pb} = .39, p = .006). Anxiety/insomnia was not significantly correlated with any of the measures of physical or cognitive functioning (p > .05).

Social dysfunction, as measured by the GHQ-28, was significantly correlated with impairment of complex attention abilities (Digit Symbol subtest; r = -.38, p = .008). Severe depression, as measured by the GHQ-28, was significantly correlated

Table 13

Correlational Analysis of Individual Variables at 12 Months Poststroke ($N = 48$)

Variable	1	2	3	4	5	6	7	8	9
1. GDS: Depression	--								
2. GHQ: Somatic	.38*	--							
3. GHQ: Anxiety	.48*	.23	--						
4. GHQ: Social	.24	.31*	-.05	--					
5. GHQ: Depression	.55*	.23	.10	.18	--				
6. Gender	.09	-.18	.04	.14	-.07	--			
7. Age	.18	.34*	-.17	.22	.09	-.10	--		
8. Previous stroke	.46*	.13	.39*	.03	.06	.13	.13	--	
9. Hemisphere	.20	.02	.14	-.10	.16	.13	-.03	.05	--
10. ADL (Barthel)	-.18	-.17	-.05	-.11	-.05	.38*	-.27	-.06	.28
11. ADL (Nottingham)	-.32*	.04	-.06	-.15	-.08	.15	-.35*	-.17	.15
12. Basic cognition	-.15	-.01	-.13	-.17	-.34*	-.01	-.24	.09	-.11
13. FSIQ	-.04	.02	-.03	-.07	-.10	.04	.08	.03	.03
14. Simple attention	-.14	-.16	-.09	-.02	-.24	-.12	.09	.08	-.17
15. Complex attention	-.40*	-.32*	-.14	-.38*	-.15	-.12	-.41*	-.20	-.10
16. Immediate memory	.13	.10	.12	-.21	-.06	-.13	-.18	.04	-.06
17. Delayed memory	-.07	-.12	.10	-.28	-.07	-.11	-.20	-.07	-.11
18. Verbal fluency	-.09	.03	-.04	-.22	-.09	-.07	-.14	-.10	-.14

Table 13 (continued)

Correlational Analysis of Individual Variables at 12 Months Poststroke ($N = 48$)

Variable	10	11	12	13	14	15	16	17	18
10. ADL (Barthel)	--								
11. ADL (Nottingham)	.76*	--							
12. Basic cognition	.31*	.41*	--						
13. FSIQ	.19	.26	.38*	--					
14. Simple attention	-.12	.02	.42*	.51*	--				
15. Complex attention	.27	.43*	.42*	.56*	.37*	--			
16. Immediate memory	-.24	-.01	.39*	.41*	.37*	.27	--		
17. Delayed memory	-.17	.08	.33*	.36*	.27	.34*	.65*	--	
18. Verbal fluency	-.02	.25	.41*	.32*	.47*	.30*	.61*	.36*	--

Note. FSIQ = Full Scale IQ; GDS = Geriatric Depression Scale; GHQ = 28-item version of the General Health Questionnaire.

* $p < .05$.

with impairment of basic cognitive functioning (CAS; $r = -.34, p > .05$).

Due to the high correlation between the two measures of physical functioning (Barthel ADL Index and Nottingham Extended ADL Index; $r = .76, p < .001$), the scores for these two measures were combined to form one total score of physical functioning for use in the regression analyses, as recommended by Tabachnick and Fidell (1989).

Regression Analyses

Hierarchical regressions were performed on the outcome variables of PSD (as measured by the GDS), and the four psychological disorders represented by the scales of the GHQ-28. As there were only 48 cases included in the regression analyses, care was taken to limit the number of independent variables to a ratio of five cases for every independent variable, as recommended by Tabachnick and Fidell (1989). Therefore, nine independent variables could be included in the present regression analyses. In order to comply with this restriction, variables were combined or excluded, as recommended by Tabachnick and Fidell. The variables that were combined or excluded were all cognitive measures, as there were more measures included in the cognitive step of the hierarchical regression than any other step. As the scores on the immediate and delayed verbal recall subtests were highly correlated, and as they measure similar constructs, these scores were combined to form one score of Verbal Memory. In addition, the measures of verbal fluency (COWA) and general intelligence were excluded from the regression analyses, as the correlations between these measures and the outcome variables were the lowest of all available cognitive measures (and were statistically insignificant).

The regression analyses were used to determine if demographic variables

(gender and age), medical factors (previous stroke and hemispheric location of the lesion), level of physical functioning (as measured by the combined scores of the Barthel ADL Index and the Nottingham Extended ADL Index), and/or cognitive functioning, were related to PSD and/or the four psychological disorders at 12 months poststroke. Aspects of cognitive functioning included in the analyses were basic cognition (CAS), simple attention (Digit Span), complex attention (Digit Symbol), and verbal memory.

Depression. Table 14 displays the unstandardised (B) and standardised (Beta) regression coefficients, R^2 for step 1, and the change in R^2 after entry of each of the additional blocks of independent variables.

At step 1, the demographic variables of gender and age explained only 4% of the variance in the GDS, F (2, 45) = 0.98, $p > .05$. At step 2, the demographic and medical variables explained a total of 26% of the variance in the GDS, F (4, 43) = 3.79, $p = .010052$, and the medical variables alone accounted for 22% of the variance at this step. The medical variable of previous stroke made a unique contribution to the amount of variance accounted for at this step ($p = .002328$), and the addition of the medical variables resulted in a significant increment in R^2 ($p = .003797$).

At step 3, the demographic, medical, and physical variables explained a total of 33% of the variance in the GDS, F (5, 42) = 4.07, $p = .004189$, and the physical variable alone accounted for 7% of the variance at this step. The addition of the physical variable resulted in a significant increment in R^2 ($p = .048705$). At step 4, the cognitive variables alone accounted for 8% of the variance in the GDS, although none of the individual cognitive variables made a unique contribution to the amount of variance accounted for at this step ($p > .05$). With all the independent variables in

Table 14

Summary of Hierarchical Regression Analysis for Variables Predicting PSD at 12 Months Poststroke, as Measured by the GDS ($N = 48$)

	Variable	*B*	*SE B*	Beta
Step 1	Gender	1.15	1.63	.10
	Age	0.15	0.12	.19
Step 2	Previous stroke	5.72	1.77	.43 *
	Hemisphere	2.03	1.47	.18
Step 3	Physical functioning	-0.08	0.04	-.29 *
Step 4	Basic cognition	-0.06	0.33	-.03
	Simple attention	-0.26	0.32	-.13
	Complex attention	-0.14	0.10	-.23
	Verbal memory	0.26	0.17	.23

Note. GDS = Geriatric Depression Scale.

$R^2 = .04$ for Step 1 ($p > .05$);

change in $R^2 = .22$ for Step 2 ($p = .003797$);

change in $R^2 = .07$ for Step 3 ($p = .048705$);

change in $R^2 = .08$ for Step 4 ($p > .05$).

* $p < .05$.

the equation, the regression model explained 40% of the variance in the GDS, F (9, 38) = 2.84, p = .011344.

Therefore, in total, the 9 independent variables explained 40% of the variance in the GDS. The variables included in the regression equation did significantly predict PSD. However, these results indicate that the strongest predictors of PSD at 12 months poststroke were previous stroke and physical impairment.

Somatic symptoms (GHQ-28, scale A). Table 15 displays the unstandardised (B) and standardised (Beta) regression coefficients, R^2 for step 1, and the change in R^2 after entry of each of the additional blocks of independent variables.

At step 1, the demographic variables of gender and age explained 14% of the variance in the measure of somatic symptoms, F (2, 45) = 3.55, p = .037037, and the demographic variable of age made a unique contribution to the amount of variance accounted for at this step (p = .024068). At step 2, the demographic and medical variables explained a total of 15% of the variance in the measure of somatic symptoms, F (4, 43) = 1.88, $p > .05$, and the medical variables alone accounted for 1% of the variance at this step. At step 3, the demographic, medical, and physical variables explained a total of 18% of the variance in the measure of somatic symptoms, F (5, 42) = 1.84, $p > .05$, and the physical variable alone accounted for 3% of the variance at this step. At step 4, the cognitive variables alone accounted for 16% of the variance in the measure of somatic symptoms. With all of the independent variables in the equation, the regression model explained 34% of the variance in the measure of somatic symptoms, F (9, 38) = 2.13, p = .050837.

Therefore, in total, the 9 independent variables explained 34% of the variance in the measure of somatic symptoms. Although the demographic variable of age was

Table 15

Summary of Hierarchical Regression Analysis for Variables Predicting Somatic Symptoms at 12 Months Poststroke, as Measured by the GHQ-28, scale A ($N = 48$)

	Variable	*B*	*SE B*	Beta
Step 1	Gender	-0.68	0.65	-.15
	Age	0.11	0.05	.33*
Step 2	Previous stroke	0.58	0.79	.11
	Hemisphere	0.20	0.66	.04
Step 3	Physical functioning	0.02	0.02	.19
Step 4	Basic cognition	0.06	0.15	.07
	Simple attention	-0.19	0.14	-.23
	Complex attention	-0.09	0.05	-.35
	Verbal memory	0.11	0.07	.24

Note. GHQ-28 = 28-item version of the General Health Questionnaire.

$R^2 = .14$ for Step 1 ($p = .037037$);

change in $R^2 = .01$ for Step 2 ($p > .05$);

change in $R^2 = .03$ for Step 3 ($p > .05$);

change in $R^2 = .16$ for Step 4 ($p > .05$).

* $p < .05$.

significant, and the regression model did approach significance, the full regression model did not significantly predict somatic symptoms at 12 months poststroke.

Anxiety/insomnia (GHQ-28, scale B). Table 16 displays the unstandardised (B) and standardised (Beta) regression coefficients, R^2 for step 1, and the change in R^2 after entry of each of the additional blocks of independent variables.

At step 1, the demographic variables of gender and age explained 3% of the variance in the measure of anxiety/insomnia, F (2, 45) = 0.69, $p > .05$. At step 2, the demographic and medical variables explained a total of 22% of the variance in the measure of anxiety/insomnia, F (4, 43) = 3.00, $p = .028534$, and the medical variables alone accounted for 19% of the variance at this step. The medical variable of previous stroke made a unique contribution to the amount of variance accounted for at this step ($p = .003762$), and the addition of the medical variables resulted in a significant increment in R^2 ($p = .009561$).

At step 3, the demographic, medical, and physical variables explained a total of 23% of the variance in the measure of anxiety/insomnia, F (5, 42) = 2.48, $p = .046715$, and the physical variable alone accounted for 1% of the variance at this step. However, neither the increment in R^2 at step 2, nor the physical variable, were significant ($p > .05$). At step 4, the cognitive variables alone accounted for 8% of the variance in the measure of anxiety/insomnia. With all of the independent variables in the equation, the regression model explained 31% of the variance in the measure of anxiety/insomnia, F (9, 38) = 1.89, $p > .05$.

Therefore, in total, the 9 independent variables explained 31% of the variance in the measure of anxiety/insomnia. Although individual variables were significant, the full regression model did not significantly predict anxiety/insomnia at 12 months poststroke.

Table 16

Summary of Hierarchical Regression Analysis for Variables Predicting Anxiety/Insomnia at 12 Months Poststroke, as Measured by the GHQ-28, scale B (N = 48)

	Variable	B	$SE\ B$	Beta
Step 1	Gender	0.06	0.44	.02
	Age	-0.04	0.03	-.17
Step 2	Previous stroke	1.48	0.48	.42 *
	Hemisphere	0.35	0.40	.12
Step 3	Physical functioning	-0.008	0.01	-.11 *
Step 4	Basic cognition	-0.15	0.10	-.29
	Simple attention	0.004	0.09	.007
	Complex attention	-0.03	0.03	-.16
	Verbal memory	0.07	0.05	.22

Note. GHQ-28 = 28-item version of the General Health Questionnaire.

R^2 = .03 for Step 1 ($p > .05$);

change in R^2 = .19 for Step 2 ($p = .009561$);

change in R^2 = .01 for Step 3 ($p > .05$);

change in R^2 = .08 for Step 4 ($p > .05$).

* $p < .05$.

Social dysfunction (GHQ-28, scale C). Table 17 displays the unstandardised (*B*) and standardised (Beta) regression coefficients, R^2 for step 1, and the change in R^2 after entry of each of the additional blocks of independent variables.

At step 1, the demographic variables of gender and age explained 7% of the variance in the measure of social dysfunction, $F(2, 45) = 1.82, p > .05$. At step 2, the demographic and medical variables explained a total of 9% of the variance in the measure of social dysfunction, $F(4, 43) = 1.03, p > .05$, and the medical variables alone accounted for 1% of the variance at this step. At step 3, the demographic, medical, and physical variables explained a total of 10% of the variance in the measure of social dysfunction, $F(5, 42) = 0.88, p > .05$, and the physical variable alone accounted for 1% of the variance at this step. At step 4, the cognitive variables alone accounted for 13% of the variance in the measure of social dysfunction. The increment in R^2 was not significantly different from zero, although the individual measure of complex attention abilities (Digit Symbol subtest) made a unique contribution to the variance accounted for at this step ($p = .049371$). With all of the independent variables in the equation, the regression model explained 22% of the variance in the measure of social dysfunction, $F(9, 38) = 1.21, p > .05$.

Therefore, in total, the 9 independent variables explained 22% of the variance in the measure of social dysfunction. Although the cognitive measure of complex attention was significant, the full regression model did not significantly predict social dysfunction at 12 months poststroke.

Severe depression (GHQ-28, scale D). Table 18 displays the unstandardised (*B*) and standardised (Beta) regression coefficients, R^2 for step 1, and the change in R^2 after entry of each of the additional blocks of independent variables.

Table 17

Summary of Hierarchical Regression Analysis for Variables Predicting Social Dysfunction at 12 Months Poststroke, as Measured by the GHQ-28, scale C ($N = 48$)

	Variable	*B*	*SE B*	Beta
Step 1	Gender	0.51	0.44	.17
	Age	0.05	0.03	.23
Step 2	Previous stroke	-0.05	0.53	-.01
	Hemisphere	-0.34	0.44	-.11
Step 3	Physical functioning	-0.008	0.01	-.10
Step 4	Basic cognition	-0.02	0.10	-.03
	Simple attention	0.11	0.10	.20
	Complex attention	-0.07	0.03	-.40 *
	Verbal memory	-0.05	0.05	-.17

Note. GHQ-28 = 28-item version of the General Health Questionnaire.

$R^2 = .07$ for Step 1 ($p > .05$);

change in $R^2 = .01$ for Step 2 ($p > .05$);

change in $R^2 = .01$ for Step 3 ($p > .05$);

change in $R^2 = .13$ for Step 4 ($p > .05$).

* $p < .05$.

Table 18

Summary of Hierarchical Regression Analysis for Variables Predicting Severe Depression at 12 Months Poststroke, as Measured by the GHQ-28, scale D ($N = 48$)

	Variable	*B*	*SE B*	Beta
Step 1	Gender	-0.26	0.59	-.06
	Age	0.02	0.04	.08
Step 2	Previous stroke	0.23	0.72	.05
	Hemisphere	0.67	0.60	.17
Step 3	Physical functioning	-0.007	0.02	-.07
Step 4	Basic cognition	-0.25	0.14	-.36
	Simple attention	-0.11	0.13	-.16
	Complex attention	0.004	0.04	.02
	Verbal memory	0.05	0.07	.13

Note. GHQ-28 = 28-item version of the General Health Questionnaire.

$R^2 = .01$ for Step 1 ($p > .05$);

change in $R^2 = .03$ for Step 2 ($p > .05$);

change in $R^2 = .004$ for Step 3 ($p > .05$);

change in $R^2 = .13$ for Step 4 ($p > .05$).

$^{*}p < .05$.

At step 1, the demographic variables of gender and age explained 1% of the variance in the measure of severe depression, F (2, 45) = 2.77, $p > .05$. At step 2, the demographic and medical variables explained a total of 4% of the variance in the measure of severe depression, F (4, 43) = 0.49, $p > .05$, and the medical variables alone accounted for 3% of the variance at this step. At step 3, the demographic, medical, and physical variables explained a total of 5% of the variance in the measure of severe depression, F (5, 42) = 0.42, $p > .05$, and the physical variable alone accounted for less than 1% (0.4%) of the variance at this step. At step 4, the cognitive variables alone accounted for 13% of the variance in the measure of severe depression. With all of the independent variables in the equation, the regression model explained 18% of the variance in the measure of severe depression, F (9, 38) = 0.91, $p > .05$.

Therefore, in total, the 9 independent variables explained 18% of the variance in the measure of severe depression. The full regression model did not significantly predict severe depression at 12 months poststroke.

Discussion

These results indicate that 32% of the stroke patients reported symptoms of depression at 12 months poststroke. Regression analysis indicated that PSD was related to previous stroke and physical impairment. The demographic factors of age or gender were not significantly related to depression at this stage of the poststroke period, nor was hemispheric location of the lesion. Level of cognitive functioning was not predictive of depression.

The prevalence of depression at 12 months poststroke reported in this study

was similar to that reported by Robinson et al. (1987), although other researchers have reported lower prevalence rates (e.g., Astrom, Adolfsson, et al., 1993). Although only 4% of the patients reported clinically significant symptoms of psychological disorder as measured by the GHQ-28, the results of this study confirm that PSD and other emotional disorders are still a concern at 12 months poststroke.

Results of the Nottingham Extended ADL Index indicated that the majority of patients performed at least some of the mobility, kitchen, domestic, and leisure activities, although half of the patients indicated impairment on self-care activities as measured by the Barthel ADL Index. Approximately one quarter of the patients demonstrated impairment on basic cognitive functioning tasks. As was evident at 3 months poststroke, the rate of cognitive impairment was greater on more difficult tasks of cognitive functioning, with the rates of impairment on the more complex tasks ranging from 35 - 87%. The average IQ of these patients was 79, therefore remaining similar to that found at 3 months poststroke, and still substantially less than the level of intellectual functioning prior to the stroke.

PSD was significantly correlated with previous stroke, complex attention (although this relationship did not reach significance in the regression analysis), and physical functioning as measured by the Nottingham Extended ADL Index. Consistent with the finding at the 3-month follow-up, PSD was not significantly correlated with performance of self-care activities as measured by the Barthel ADL Index at 12 months poststroke. As was mentioned previously, there are two possible explanations for this relationship. First, failure to perform, or inability to perform, the extended ADL activities may have a negative impact on the patients' emotional wellbeing to a greater extent than failure, or inability, to perform the basic self-care

activities. Alternatively, the patients who are depressed may be less likely to perform the extended ADL activities, but continue to perform their self-care activities. Given that PSD is likely to be a reaction to impairment beyond the acute poststroke stage, the former alternative is the more likely explanation.

Very few relationships were found between any of the GHQ-28 subscales and the demographic, medical, physical, or cognitive variables in the correlational analysis. Somatic symptoms were related to older age and impairment of complex attention abilities, although this latter relationship was not evident in the regression analysis. Impairment of complex attention was also related to social dysfunction. Anxiety/insomnia was more evident in patients who had experienced at least one previous stroke. Reasons for this latter relationship may be similar to that suggested for the relationship between PSD and previous stroke; the anxiety occurs as the patient is more aware of the possible outcome of this subsequent stroke. Patients with impairment of basic cognitive function were more likely to report symptoms of severe depression, as measured by the GHQ-28, scale D. As impairment of basic cognitive function was not demonstrated by patients with symptoms of severe depression as measured by the GDS, clearly the GDS measures an alternative aspect of severe depression than that measured by the GHQ-28.

None of the regression equations for the GHQ-28 were significant. Reasons for the paucity of predictive relationships are unclear, although as mentioned previously, the aspects of affective functioning measured by the GHQ-28 may best be predicted by factors not assessed in the present study.

Regression analysis provided a more defined pattern of relationships between depression and associated factors. As was evident at the 3-month follow-up, neither

the demographic variables nor hemispheric location of the lesion were related to depression. Depression was still higher in patients who had experienced a previous stroke. Although other researchers have also reported this relationship at this stage of the poststroke period (Andersen et al., 1995), reasons for the continuation of this relationship for the entire first year poststroke are unclear. The relationship itself may be reactive in nature; if the patient has experienced a previous stroke, they will be more aware of what may happen to them, and that their chances of having further strokes are increased, therefore the patient becomes depressed. However, the duration of the relationship is a concern as it suggests that regardless of the rate and extent of the patient's recovery, depression is still a likely outcome if the patient has had a previous stroke. Perhaps a third factor, such as the rate of recovery, is responsible for this relationship; the patient who has had a previous stroke may have more physical impairment, or improve less, than the patient who has had their first stroke. This hypothesis will be examined in the next chapter.

The results of this study indicated that physical impairment was still related to depression at 12 months poststroke. As mentioned previously, it is possible that the depression and the impairment each exacerbate the effects of the other. However, if the depression is, at least in part, a reaction to the disability, it is possible that the patient may be reacting to a different aspect of the impairment than earlier in the poststroke period. That is, earlier in the poststroke period the patient may have become depressed due to the *extent* of their impairment. However, by 12 months poststroke, it is possible that the patient is depressed due to the *duration* of their impairment.

There was no predictive relationship between cognitive impairment and PSD

at 12 months poststroke. Other researchers have also failed to find a relationship between PSD and cognitive functioning at this stage of the poststroke period (Andersen et al., 1995; Parikh et al., 1987). If the depression is a reaction to the impairment, it is possible that the patients' overall level of cognitive functioning had improved since earlier in the poststroke period, resulting in a decrease in the relationship between cognitive impairment and depression. It is also possible that the patients may have come to terms with their impairments, and learnt to cope with their decreased cognitive functioning. Whether the level of cognitive functioning at 12 months poststroke had improved or deteriorated in comparison with earlier in the poststroke period will be examined in the next chapter.

In summary, at 12 months poststroke symptoms of depression were evident in approximately one third of these stroke patients. Both previous stroke and physical impairment were significantly related to PSD, although there was no significant relationship between PSD and cognitive impairment. As had been evident at 3 months poststroke, PSD was not related to the hemispheric location of the lesion at 12 months poststroke, and neither of the demographic variables of gender or age were predictive of PSD. Longitudinal comparisons of the assessments will provide further understanding of these patterns of behaviour. Regardless of the possible explanations for these relationships, PSD clearly continues to be a concern at 12 months poststroke.

CHAPTER 6

Longitudinal Changes in PSD and Associated Factors

As the previous chapters have indicated, many changes are evident in the relationships between PSD and its associated factors. There are a number of possible explanations for these changes, some of which have been discussed previously. Further understanding of the course of cognitive and physical recovery may also help to explain the changes in these relationships. This longitudinal assessment will examine the recovery of physical and cognitive functioning, and the relationships between PSD and recovery. This chapter will also address the possibility that the duration of the relationship between PSD and previous strokes may be due to the level of physical or cognitive impairment in, and recovery of, those patients who have had a previous stroke. As previous research has indicated that there is a relationship between depression and mortality in stroke patients (Morris, Robinson, & Samuels, 1993), the impact of depression on mortality is also an issue that will be addressed.

Clearly with time, there are changes in the extent of physical and cognitive impairment experienced by stroke patients. Jorgensen et al. (1995b) reported that the majority of stroke patients receiving rehabilitation recover most of their basic ADL functioning within 3 months following the stroke, with all patients reaching the full poststroke potential of their ADL functioning by 20 weeks poststroke. Despite this finding, all stroke patients do not recover to their full prestroke level of functioning. In addition, the recovery reported by Jorgensen et al. is not generalisable beyond self-care activities as many activities involve more complex motor and cognitive abilities than are necessary to complete basic activities of daily living. Physical impairment is

likely to remain an issue for many stroke patients for some time after the stroke, possibly permanently. Further research is needed to assesses the physical recovery of stroke patients.

Research has shown that improvement in cognitive functioning does occur in stroke patients (Desmond, Moroney, Sano, & Stern, 1996). Bowler, Hadar, and Wade (1994) reported that the cognitive functioning of stroke patients had improved by 3 months poststroke, particularly in tasks thought to involve subcortical and right hemisphere processing. This included tasks of verbal fluency, information processing, and arithmetic. There was little indication of memory impairment in the patients examined by these researchers, although previous research has indicated that memory impairment is common in stroke patients (Tatemichi et al., 1994). Bokura and Robinson (1997) also found that during the first 2 years poststroke, cognitive functioning improved in patients with subcortical strokes, although patients with caudate strokes demonstrated a decrease in cognitive functioning. As mentioned in the previous chapter, if depression is a reaction to impairment, a decrease in cognitive impairment may provide an explanation for the lack of relationship between PSD and cognitive impairment at 12 months poststroke. The present chapter will examine this hypothesis.

Despite research indicating a general trend towards improved physical and cognitive functioning within the first year following stroke, the improvement may be considerably less in depressed stroke patients. That is, studies have shown that depressed stroke patients recover significantly fewer of their physical abilities than non-depressed patients within the first 2 years poststroke (Astrom, Adolfsson, et al., 1993; Morris, Raphael, et al., 1992; Parikh et al., 1990). A similar relationship has

been reported between cognitive impairment and PSD. Morris, Raphael, et al. (1992) reported that, while all stroke patients had similar levels of cognitive functioning at 2 months poststroke, by 16 months poststroke non-depressed patients experienced greater cognitive recovery than the depressed patients. Examining the relationship from a different perspective, Downhill and Robinson (1994) found those stroke patients presenting with both cognitive impairment and depression were depressed for a longer duration than the depressed patients without cognitive impairment. These findings of the impact of depression on physical and cognitive recovery have important implications for the effectiveness of rehabilitation for depressed stroke patients. This chapter will examine whether these patterns of recovery are evident in the present group of stroke patients.

Another issue raised in the earlier chapters was the long-term impact of previous strokes on recovery. The lengthy duration of the relationship between PSD and previous strokes may be, at least in part, due to an additional factor, such as the rate of recovery. That is, a patient who has experienced a previous stroke may have more impairment (either cognitive or physical), and/or recover less of their prestroke physical or cognitive functions, than a patient who has had one stroke only. As the relationship between physical impairment and depression in this study was still evident at 12 months poststroke it seems more likely that physical impairment, as opposed to cognitive impairment, would be exacerbating the relationship between PSD and previous strokes. The relationship between PSD and physical impairment is often described as a bi-directional relationship. It is possible that this extends to a three-way relationship, where depression, physical impairment, and the effects of a previous stroke all interact with each other. This chapter will look at the extent of impairment

in those patients who have experienced a previous stroke, examining the hypothesis that the physical and/or cognitive impairment of the patients who have had a previous stroke is involved in the duration of the relationship between depression and previous strokes, or alternatively, that a history of a previous stroke affects physical and/or cognitive recovery.

An important finding regarding the long-term impact of depression on recovery from stroke is the relationship between PSD and mortality. Although Astrom, Adolfsson, et al. (1993) found no relationship between mortality and depression, Morris, Robinson, and Samuels (1993) found that patients who had either minor or major depression at 2 months poststroke were eight times more likely than non-depressed patients to have died by 15 months poststroke. The rate of mortality increased with the severity of depression. In a 10-year follow-up of another group of stroke patients, these researchers reported that patients with depression at 2 weeks poststroke were approximately three times more likely than the non-depressed patients to have died by 10 years poststroke (Morris, Robinson, Andrzejewski, et al., 1993). The rate of mortality did not increase with the severity of depression in the group of patients examined by Morris, Robinson, Andrzejewski, et al. Although the rate of co-existing medical conditions was higher amongst the patients who had died within 10 years of the stroke, a relationship between depression and mortality was still evident after controlling for any effect of these medical states. Due to the serious implications of this relationship, further research is clearly needed in this area.

The extent of physical and cognitive impairment appears to lessen within the first year following stroke, and research has indicated that depression affects the degree of improvement. This longitudinal examination aims to further analyse the

rates of recovery in physical and cognitive functioning within the first 12 months poststroke, together with addressing the role of depression in this recovery. The possible impact of impairment on the duration of the relationship between previous strokes and depression will be examined, as will the relationship between depression and mortality.

Method

Participants

Only those patients who were able to be assessed at 1, 3, and 12 months poststroke were included in the longitudinal comparison of functioning. Of the 103 stroke patients originally selected for inclusion in the study, 20 had died during the first year poststroke. Of the remaining 83 patients, 8 patients were either unable or unwilling to take part in at least one of the three assessments, and 2 were receiving medication as treatment for depression. Two patients were excluded as they had been admitted to hospital due to a subsequent stroke. Screening for the presence of aphasia was undertaken by administration of the short-form of the Minnesota Differential Diagnosis of Aphasia Test (Powell et al., 1980). Results of the aphasia test indicated that 8 patients were too aphasic to provide meaningful responses to the assessment procedures for at least one of the three assessments. The remaining 63 patients constituted the final sample for the longitudinal comparison.

The initial assessment was conducted, on average, 27 days poststroke (*SD* = 15, range = 3 - 73). As mentioned previously, the time of this initial assessment will be referred to as 1 month poststroke. The 3-month follow-up was conducted, on average, 94 days poststroke (*SD* = 5, range = 71 - 114), and the 12-month follow-up

was conducted, on average, 367 days poststroke (*SD* = 5, range = 357 - 384). The average age of the patients at the time of the stroke was 74 years (*SD* = 7, range = 60 - 87). Thirty-four (54%) patients were female and 29 (46%) patients were male. Sixty (95%) patients were right-handed, and 3 (5%) patients were left-handed. Fifty-two (83%) patients were retired, 6 (10%) patients were in paid employment, and 5 (8%) patients gave their occupation as "homemaker".

Sixty (95%) of the 63 stroke patients had CT scans. The CT scans of these 60 patients, and clinical examination of the remaining 3 patients, determined that 34 (54%) patients had right hemisphere strokes, 25 (40%) patients had strokes in the left hemisphere, 2 (3%) patients had cerebellar strokes, 1 (2%) patient had a brainstem stroke, and 1 (2%) patient had a bilateral stroke. For 49 (78%) patients this was their first stroke, 14 (22%) patients had experienced at least one previous stroke, with 1 of these latter 14 patients having had two previous strokes.

Measures

The measures used in the longitudinal comparison of poststroke functioning were the same measures as those used at 3 and 12 months poststroke. These measures were as follows:

Cognitive measures. The Cognitive Assessment Scale (CAS) from the Clifton Assessment Procedures for the Elderly (Pattie & Gilleard, 1979) was administered to provide an overall assessment of cognitive functioning. Four subtests from the Wechsler Adult Intelligence Scale-Revised (WAIS-R) were also administered (Wechsler, 1981). Scores on the Vocabulary and Block Design subtests of the WAIS-R were combined to provide an estimate of current level of intellectual functioning (Full Scale IQ; FSIQ) using the tables provided by Brooker and Cyr (1986). The

Digit Span subtest of the WAIS-R (1981) was used to measure simple attention abilities. The Digit Symbol subtest of the WAIS-R was used to measure complex attention abilities. Verbal fluency was assessed by administration of the Controlled Oral Word Association test (COWA; Spreen & Strauss, 1991). Immediate and delayed verbal memory were assessed by administration of the VPA-immediate recall, and VPA-delayed recall subtests from the Wechsler Memory Scale-Revised (Wechsler, 1987).

Self-report questionnaires. Physical functioning was assessed by administration of the Barthel ADL Index (Wade & Langton-Hewer, 1987). The Nottingham Extended ADL Index (Nouri & Lincoln, 1987) was administered as a measure of the patient's independence within the home. Scoring of the Nottingham Extended ADL Index was completed using the scoring method recommended by Nouri and Lincoln, where performing the task on their own, or on their own with difficulty received a score of 1, and performing the task with help, or not performing the task received a score of 0. Oral administration of the Geriatric Depression Scale (GDS; Yesavage et al., 1983) was used to assess PSD, and the 28-item version of the General Health Questionnaire (GHQ-28; Goldberg, 1972) was used to determine the presence of psychological disorder.

As only the Barthel ADL Index, the GDS, and the CAS were used in the assessment at 1 month poststroke, these were the only measures compared across the three stages of assessment. Comparisons of the patients' performance at 3 and 12 months poststroke included the remainder of the measures.

Procedure and Data Analysis

The initial assessment took approximately 30 minutes to complete, and each 3

and 12-month follow-up assessment took approximately 1.5 hours to complete. Each patient was assessed at their place of residence.

One-way repeated measures ANOVAs (for those measures used at all three stages of assessment) and *t* tests (for those measures used at only 3 and 12 months poststroke) were performed to determine whether there was any significant improvement in functioning between 1, 3, and 12 months poststroke. McNemar chi-square was used to determine whether there was a significant change in the prevalence of psychological disorder between 3 and 12 months poststroke.

MANOVAs were used to determine whether depressed patients demonstrated more physical or cognitive impairment, or whether they demonstrated less recovery, than non-depressed patients. MANOVAs were also used to determine whether patients who had experienced a previous stroke demonstrated more physical or cognitive impairment, or less recovery, than patients who had not experienced a previous stroke. Pearson chi-square analysis (X^2) was used to determine whether PSD at 1 month poststroke was related to mortality within the first 12 months poststroke.

Results

Changes in Variables Across Time

A description of the clinically significant changes in the levels of physical, affective, and cognitive functioning is presented in Appendix H.

Physical functioning. The Barthel ADL Index was completed for all patients. Table 19 shows the results of the Barthel ADL Index at 1, 3, and 12 months poststroke in terms of the level of physical functioning. Thirty (48%) patients were physically independent at 1 month poststroke, 20 (32%) patients had mild

Table 19

Levels of Physical Functioning in Patients at 1, 3, and 12 Months Poststroke, as Measured by the Barthel ADL Index ($N = 63$)

	Time Poststroke		
	1 month	3 months	12 months
Level of functioning	*n* (%)	*n* (%)	*n* (%)
Independent	30 (48%)	32 (51%)	32 (51%)
Mild impairment	20 (32%)	19 (30%)	21 (33%)
Moderate impairment	8 (13%)	7 (11%)	4 (6%)
Severe impairment	4 (6%)	5 (8%)	5 (8%)
Very severe impairment	1 (2%)	0 (0%)	1 (2%)

impairment, 8 (13%) patients had moderate impairment, 4 (6%) patients had severe impairment, and 1 (2%) patient had very severe physical impairment. At 3 months poststroke, 32 (51%) patients were physically independent, 19 (30%) patients had mild physical impairment, 7 (11%) patients had moderate impairment, and 5 (8%) patients had severe physical impairment. No patients presented with very severe physical impairment at 3 months poststroke. At 12 months poststroke, 32 (51%) patients were physically independent, 21 (33%) patients had mild impairment, 4 (6%)

patients had moderate impairment, 5 (8%) patients had severe impairment, and 1 (2%) patient had very severe physical impairment.

Thirty-eight (60%) patients did not demonstrate a clinically significant change in their physical functioning during the first 12 months poststroke. Two (3%) patients improved progressively during the first 12 months poststroke. Four (6%) patients improved between 1 and 3 months poststroke, then deteriorated between 3 and 12 months poststroke. Six (10%) patients deteriorated between 1 and 3 months poststroke, then improved between 3 and 12 months poststroke. Six (10%) patients improved between 1 and 3 months poststroke only, and 1 (2%) patient improved between 3 and 12 months poststroke only. Three (5%) patients deteriorated between 1 and 3 months poststroke only, and an additional 3 (5%) patients deteriorated between 3 and 12 months poststroke only.

Table 20 shows the average scores on the Barthel ADL Index. At 1 month poststroke, the average Barthel ADL Index score was 17.38 (SD = 4.09, range = 3 - 20). At 3 months poststroke, the average Barthel ADL Index score was 17.49 (SD = 3.94, range = 5 - 20). At 12 months poststroke, the average Barthel ADL Index score was 17.27 (SD = 4.36, range = 3 - 20). Repeated measures ANOVA determined that there was no statistically significant difference between the average Barthel ADL Index scores at 1, 3, and 12 months poststroke, F (2, 124) = 0.32, $p > .05$.

The Nottingham Extended ADL Index was completed for all patients. Table 20 shows the results of the Nottingham Extended ADL Index. The average number of activities performed by the patients on their own increased slightly between 3 and 12 months poststroke. The average number of mobility activities performed by the patients at 3 months poststroke was 3.60 (SD = 2.09, range = 0 - 6), which increased very slightly to 3.68 (SD = 2.15, range = 0 - 6) at 12 months poststroke. The average

Table 20

Mean Scores and Standard Deviations of Measures at 1, 3, and 12 Months Poststroke

		Time Poststroke		
		1 month	3 months	12 months
Measure	*N*	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Barthel ADL Index	63	17.38 (4.09)	17.49 (3.94)	17.27 (4.36)
Mobility activities	63		3.60 (2.09)	3.68 (2.15)
Kitchen activities	63		3.30 (1.61)	3.40 (1.78)
Domestic activities	63		1.87 (1.71)	1.89 (1.87)
Leisure activities	63		3.13 (1.69)	3.35 (1.85)
GDS	63	7.92 (5.27)	8.24 (5.24)	8.02 (5.60)
GHQ-28 total score	63		2.49 (3.56)	1.43 (3.29)*
CAS	57	29.39 (4.89)	29.89 (4.50)	30.39 (4.00)
FSIQ	57		79.49 (15.56)	79.58 (14.96)
Digit Span	63		11.22 (2.89)	11.35 (3.28)
Digit Symbol	51		22.18 (8.23)	21.00 (9.41)*
VPA, immediate	59		12.81 (3.63)	13.44 (3.64)
VPA, delayed	57		4.33 (1.96)	4.82 (1.73)*
COWA	63		22.44 (9.62)	23.56 (9.70)

Note. CAS = Cognitive Assessment Scale; COWA = Controlled Oral Word Association Test; FSIQ = Full Scale IQ; GDS = Geriatric Depression Scale; GHQ-28 = 28-item version of the General Health Questionnaire; VPA = Verbal Paired Associates, immediate and delayed verbal recall subtests of the Wechsler Memory Scale.

* $p < .05$.

number of kitchen tasks performed by the patients at 3 months poststroke was 3.30 (*SD* = 1.61, range = 1 - 5), which increased very slightly to 3.40 (*SD* = 1.78, range = 0 - 5) at 12 months poststroke. The average number of domestic tasks performed by the patients at 3 months poststroke was 1.87 (*SD* = 1.71, range = 0 - 5), which increased very slightly to 1.89 (*SD* = 1.87, range = 0 - 5) at 12 months poststroke. The average number of leisure activities performed by the patients at 3 months poststroke was 3.13 (*SD* = 1.69, range = 0 - 6), which increased slightly to 3.35 (*SD* = 1.85, range = 0 - 6) at 12 months poststroke. As expected, none of these four changes were statistically significant ($p > .05$).

Affective functioning. All of the patients completed the GDS. Table 21 displays the results of the GDS, in terms of clinically significant symptoms of depression. Forty-two (67%) patients reported no clinically significant symptoms of depression at 1 month poststroke, 20 (32%) patients reported symptoms of mild depression, and 1 (2%) patient reported symptoms of severe depression. At 3 months poststroke, 40 (63%) patients reported no clinically significant symptoms of depression, 21 (33%) patients reported symptoms of mild depression, and 2 (3%) patients reported symptoms of severe depression. At 12 months poststroke, 43 (68%) patients reported no clinically significant symptoms of depression, 17 (27%) patients reported symptoms of mild depression, and 3 (5%) patients reported symptoms of severe depression.

Thirty-six (57%) patients did not demonstrate a clinically significant change in the level of depression during the first 12 months poststroke. Four (6%) patients experienced decreasing levels of depression between 1 and 3 months poststroke, then increasing levels of depression between 3 and 12 months poststroke. Five (8%)

Table 21

Prevalence of Clinically Significant Symptoms of Depression in Patients at 1, 3, and 12 Months Poststroke, as Measured by the GDS ($N = 63$)

	Time Poststroke		
	1 month	3 months	12 months
Level of depression	*n* (%)	*n* (%)	*n* (%)
No depression	42 (67%)	40 (63%)	43 (68%)
Mild depression	20 (32%)	21 (33%)	17 (27%)
Severe depression	1 (2%)	2 (3%)	3 (5%)

patients experienced increasing levels of depression between 1 and 3 months poststroke, then decreasing levels of depression between 3 and 12 months poststroke. Four (6%) patients experienced decreasing levels of depression between 1 and 3 months poststroke only, and the level of depression of 5 (8%) patients decreased between 3 and 12 months poststroke only. One (2%) patient had depression of increasing severity through all three assessments. Four (6%) patients experienced increasing levels of depression between 1 and 3 months poststroke only, and 4 (6%) patients experienced increasing levels of depression between 3 and 12 months poststroke only.

Table 20 also shows the average scores on the GDS. The average GDS scores at each of the three stages of assessment were as follows: 7.92 (SD = 5.27, range = 0 - 24) at 1 month poststroke, 8.24 (SD = 5.24, range = 1 - 21) at 3 months poststroke, and 8.02 (SD = 5.60, range = 0 - 25) at 12 months poststroke. Repeated measures ANOVA determined that there was no statistically significant difference in the average GDS scores between 1, 3, and 12 months poststroke, F (2, 124) = 0.16, $p >$ .05.

All of the patients completed the GHQ-28. The changes in clinically significant symptoms of psychological disorder are presented in Table 22. At 3 months poststroke, 54 (86%) patients did not report clinically significant symptoms of psychological disorder, while the remaining 9 (14%) patients did report clinically significant symptoms. At 12 months poststroke, the number of patients reporting clinically significant symptoms of psychological disorder had decreased to 3 (5%); the remaining 60 (95%) patients did not report clinically significant symptoms of psychological disorder.

Although this indicates a general trend of decreasing reports of symptoms of psychological disorder, 55 (87%) patients demonstrated no clinically significant change in the presence of psychological disorder, 1 (2%) patient who had not had clinically significant symptoms of psychological disorder at 3 months poststroke did report symptoms at 12 months poststroke, while 7 (11%) patients with clinically significant symptoms of psychological disorder at 3 months poststroke did not report clinically significant symptoms at 12 months poststroke. The change in the prevalence of clinically significant symptoms of psychological disorder between 3 and 12 months poststroke was not significant, McNemar Chi-square, X^2 (4, N = 63) = 3.13, $p >$ .05.

Table 22

Presence of Psychological Disorder at 3 and 12 Months Poststroke, as Measured By the GHQ-28 ($N = 63$)

	Time Poststroke	
Presence of Psychological Disorder	3 months *n* (%)	12 months *n* (%)
Absent	54 (86%)	60 (95%)
Present	9 (14%)	3 (5%)

scores at 3 and 12 months poststroke were as follows: 2.49 ($SD = 3.56$, range = 0 - 15) at 3 months poststroke, and 1.43 ($SD = 3.29$, range = 0 - 23) at 12 months poststroke. The difference between these GHQ-28 scores was statistically significant, $t(62) = 2.26$, $p = .027242$. Therefore, the average GHQ-28 total scores decreased significantly between 3 and 12 months poststroke, although the change in the prevalence of clinically significant symptoms of psychological disorder was not significant.

Cognitive functioning: Basic cognitive skills. Table 20 shows the average scores on the cognitive measures, while the clinically significant levels of cognitive functioning are presented in Table 23. Six patients were unable to complete the CAS

Table 23

Clinically Significant Levels of Cognitive Impairment in Patients at 1, 3, and 12 Months Poststroke

Aspect of Functioning	*N*	Level of Impairment	Time Poststroke: 1 month *n* (%)	3 months *n* (%)	12 months *n* (%)
Basic cognition	57	None	36 (63%)	40 (70%)	42 (74%)
		Mild	13 (23%)	9 (16%)	10 (18%)
		Moderate	6 (11%)	8 (14%)	5 (9%)
		Marked	2 (4%)	0 (0%)	0 (0%)
		Severe	0 (0%)	0 (0%)	0 (0%)
Simple attention	63	None		37 (59%)	42 (67%)
		Mild		20 (32%)	15 (24%)
		Severe		6 (10%)	6 (10%)
Complex attention	51	None		7 (14%)	7 (14%)
		Mild		17 (33%)	13 (25%)
		Severe		27 (53%)	31 (61%)
Immediate memory	59	None		25 (42%)	34 (58%)
		Mild		26 (44%)	16 (27%)
		Severe		8 (14%)	9 (15%)
Delayed memory	57	None		32 (56%)	37 (65%)
		Mild		14 (25%)	16 (28%)
		Severe		11 (19%)	4 (7%)
Verbal fluency	63	None		24 (38%)	29 (46%)
		Mild		27 (43%)	21 (33%)
		Severe		12 (19%)	13 (21%)

Table 20 shows the average GHQ-28 total scores. The average GHQ-28 total at all three stages of assessment due to sight and/or writing impairments. Therefore it was possible to assess the basic cognitive functioning of 57 patients at 1, 3, and 12 months poststroke. At 1 month poststroke, 36 (63%) patients had no basic cognitive impairment, 13 (23%) patients had mild impairment, 6 (11%) patients had moderate impairment, and 2 (4%) patients had marked basic cognitive impairment. No patients had severe impairment of basic cognitive functioning. At 3 months poststroke, 40 (70%) patients had no basic cognitive impairment, 9 (16%) patients had mild impairment, and 8 (14%) patients had moderate basic cognitive impairment. No patients had marked or severe impairment of basic cognitive functioning. At 12 months poststroke, 42 (74%) patients had no basic cognitive impairment, 10 (18%) patients had mild impairment, and 5 (9%) patients had moderate basic cognitive impairment. No patients had marked or severe impairment of basic cognitive functioning.

Although these results indicate a general trend of improved cognitive functioning, individual comparisons of each assessment stage indicate that while some patients did improve their level of cognitive functioning, the cognitive functioning of others deteriorated. Thirty-four (60%) patients demonstrated no clinically significant change in their level of basic cognitive functioning during the first 12 months poststroke. Two (4%) patients improved between 1 and 3 months poststroke, then deteriorated between 3 and 12 months poststroke. Four (7%) patients deteriorated between 1 and 3 months poststroke, then improved between 3 and 12 months poststroke. Ten (18%) patients improved between 1 and 3 months poststroke only, and 3 (5%) patients improved between 3 and 12 months poststroke only. Two (4%)

patients deteriorated between 1 and 3 months poststroke only, and 2 (4%) patients deteriorated between 3 and 12 months poststroke only.

The average CAS scores at each of the three stages of assessment were as follows: 29.39 (*SD* = 4.89, range = 14 - 34) at 1 month poststroke, 29.89 (*SD* = 4.50, range = 16 - 34) at 3 months poststroke, and 30.39 (*SD* = 4.00, range = 19 - 34) at 12 months poststroke. The difference between the average CAS scores at 1, 3, and 12 months poststroke was not statistically significant, $F(2, 112) = 2.51$, $p > .05$.

Cognitive functioning: General intelligence. One patient refused to complete the Vocabulary and Block Design subtests at 3 months poststroke and, due to sight impairments, 5 patients were unable to complete the Block Design subtest at 3 and/or 12 months poststroke. Three of these patients were also unable to complete the NART at 3 months poststroke due to sight impairments. Therefore 57 patients were assessed with regard to their obtained FSIQ and estimated premorbid IQ. The average FSIQ at 3 months poststroke was 79.49 (*SD* = 15.56, range = 55 - 111), and the average FSIQ at 12 months poststroke was 79.58 (*SD* = 14.96, range = 55 - 114). The average estimated premorbid IQ at 3 months poststroke was 98.79 (*SD* = 11.64, range = 69 - 121).

Repeated measures ANOVA determined that there was a statistically significant difference between these IQ scores, $F(2, 112) = 104.83$, $p < .000001$. Post hoc analysis failed to find a significant difference between the two obtained FSIQs (Fishers LSD; $p > .05$). However, there was a significant difference between the estimated premorbid IQ and FSIQ at 3 months poststroke (Fishers LSD; $p < .000001$) and the estimated premorbid IQ and FSIQ at 12 months poststroke (Fishers LSD; $p < .000001$).

Cognitive functioning: Simple attention. All of the patients completed the Digit Span subtest at 3 and 12 months poststroke. The average scores of simple attention abilities are presented in Table 20, and the clinically significant levels of simple attention abilities are presented in Table 23.

At 3 months poststroke, 37 (59%) patients had no impairment of simple attention, 20 (32%) patients had mild impairment, and 6 (10%) patients had severe impairment of simple attention. At 12 months poststroke, 42 (67%) patients had no impairment of simple attention, 15 (24%) patients had mild impairment, and 6 (10%) patients had severe impairment of simple attention.

Although 6 patients had severe simple attention impairment at 3 and 12 months poststroke, they were not the same patients. Forty (63%) patients demonstrated no clinically significant change in their level of simple attention abilities during the first 12 months poststroke. Thirteen (21%) patients experienced clinically significant improvement of simple attention abilities between 3 and 12 months poststroke. Ten (16%) patients experienced clinically significant deterioration of simple attention abilities between 3 and 12 months poststroke.

The average raw scores on the Digit Span subtest were as follows: 11.22 (*SD* = 2.89, range = 6 - 19) at 3 months poststroke, and 11.35 (*SD* = 3.28, range = 4 - 23) at 12 months poststroke. As expected, the difference between the average Digit Span scores at 3 and 12 months poststroke was not statistically significant, t (62) = -0.47, p > .05.

Cognitive functioning: Complex attention. Twelve patients were unable to complete the Digit Symbol subtest at 3 and 12 months poststroke due to sight and/or writing impairments. Therefore it was possible to assess the complex attention

abilities of 51 patients at both 3 and 12 months poststroke. The average scores of complex attention abilities are presented in Table 20, and the clinically significant levels of simple complex abilities are presented in Table 23.

At 3 months poststroke, 7 (14%) patients had no impairment of complex attention, 17 (33%) patients had mild impairment, and 27 (53%) patients had severely impaired complex attention abilities. At 12 months poststroke, 7 (14%) patients had no impairment of complex attention, 13 (25%) patients had mild impairment, and 31 (61%) patients had severely impaired complex attention abilities.

Although 7 patients had no impairment of complex attention abilities at 3 and 12 months poststroke, they were not the same patients. Thirty-nine (76%) patients demonstrated no clinically significant change in their level of complex attention abilities during the first 12 months poststroke. Four (8%) patients experienced clinically significant improvement of complex attention functioning between 3 and 12 months poststroke. Eight (16%) patients experienced clinically significant deterioration of complex attention functioning between 3 and 12 months poststroke.

The averages of the raw scores on the Digit Symbol subtest were as follows: 22.18 (SD = 8.23, range = 7 - 44) at 3 months poststroke, and 21.00 (SD = 9.41, range = 3 - 51) at 12 months poststroke. The difference between the average Digit Symbol scores at 3 and 12 months poststroke was statistically significant, t (50) = 2.17, p = .035004. Therefore, on average, these patients demonstrated deteriorating complex attention abilities between 3 and 12 months poststroke.

The levels of physical, affective, and cognitive functioning for each individual patient were recorded to establish whether the group of patients demonstrating this decline in complex attention also demonstrated a decline in any other area of

functioning. No clear pattern of functioning was evident. This record of individual performance is presented in Appendix I.

Cognitive functioning: Immediate verbal memory. Four patients were unable to complete the immediate verbal recall subtest at 3 and 12 months poststroke due to the difficulty of the test. Therefore it was possible to assess the immediate verbal memory of 59 patients at 3 and 12 months poststroke. The average scores of immediate verbal memory are presented in Table 20, and the clinically significant levels of immediate verbal memory are presented in Table 23.

At 3 months poststroke, 25 (42%) patients had no impairment of immediate verbal memory, 26 (44%) patients had mild impairment, and 8 (14%) patients had severely impaired immediate verbal memory. At 12 months poststroke, 34 (58%) patients had no impairment of immediate verbal memory, 16 (27%) patients had mild impairment, and 9 (15%) patients had severely impaired immediate verbal memory.

Thirty (51%) patients demonstrated no clinically significant change in their level of immediate verbal memory during the first 12 months poststroke. Eighteen (31%) patients experienced clinically significant improvement of immediate verbal memory between 3 and 12 months poststroke. Eleven (19%) patients experienced clinically significant deterioration of immediate verbal memory between 3 and 12 months poststroke.

The average raw scores on the immediate verbal recall subtest of the VPA were as follows: 12.81 (SD = 3.63, range = 5 - 21) at 3 months poststroke, and 13.44 (SD = 3.64, range = 7 - 22) at 12 months poststroke. The difference between the average immediate verbal recall scores at 3 and 12 months poststroke was not statistically significant, t (58) = -1.49, $p > .05$.

Cognitive functioning: Delayed verbal memory. Due to the high level of difficulty of the delayed verbal recall subtest, it was not possible to administer this test to 4 patients. Two additional patients refused to complete the subtest. Therefore it was possible to assess the delayed verbal memory of 57 patients at 3 and 12 months poststroke. The average scores of delayed verbal memory are presented in Table 20, and the clinically significant levels of delayed verbal memory are presented in Table 23.

At 3 months poststroke, 32 (56%) patients had no impairment of delayed verbal memory, 14 (25%) patients had mild impairment, and 11 (19%) patients had severe impairment of delayed verbal memory. At 12 months poststroke, 37 (65%) patients had no impairment of delayed verbal memory, 16 (28%) patients had mild impairment, and 4 (7%) patients had severe impairment of delayed verbal memory.

Thirty-nine (68%) patients demonstrated no clinically significant change in their level of delayed verbal memory during the first 12 months poststroke. Fourteen (25%) patients experienced clinically significant improvement of delayed verbal memory between 3 and 12 months poststroke. Four (7%) patients experienced clinically significant deterioration of delayed verbal memory between 3 and 12 months poststroke.

The average raw scores on the delayed verbal recall subtest of the VPA were as follows: 4.33 (SD = 1.96, range = 0 - 8) at 3 months poststroke, and 4.82 (SD = 1.73, range = 0 - 8) at 12 months poststroke. The difference between the average delayed verbal recall subtest scores at 3 and 12 months poststroke was statistically significant, t (56) = -2.60, p = .012006. Therefore, on average, these patients demonstrated improvement in their delayed verbal memory between 3 and 12 months

poststroke.

Cognitive functioning: Verbal fluency. All of the patients completed the COWA at both 3 and 12 months poststroke. The average scores of verbal fluency are presented in Table 20, and the clinically significant levels of verbal fluency are presented in Table 23.

At 3 months poststroke, 24 (38%) patients had no impairment of verbal fluency, 27 (43%) patients had mild impairment, and 12 (19%) patients had severely impaired verbal fluency. At 12 months poststroke, 29 (46%) patients had no impairment of verbal fluency, 21 (33%) patients had mild impairment, and 13 (21%) patients had severely impaired verbal fluency.

Forty-one (65%) patients demonstrated no clinically significant change in their level of verbal fluency during the first 12 months poststroke. Twelve (19%) patients experienced clinically significant improvement of verbal fluency between 3 and 12 months poststroke. Ten (16%) patients experienced clinically significant deterioration of verbal fluency between 3 and 12 months poststroke.

The average COWA scores at 3 and 12 months poststroke were 22.44 (SD = 9.62, range = 3 - 42) and 23.56 (SD = 9.70, range = 8 - 44), respectively. The difference between the average COWA scores at 3 and 12 months poststroke was not statistically significant, t (62) = -1.34, $p > .05$.

Depression and Physical Impairment

MANOVA was used to determine whether the patients with symptoms of depression at 1 month poststroke had more physical impairment (as measured by the Barthel ADL Index) throughout the first 12 months poststroke, and if the depressed patients experienced less improvement in physical functioning than the non-depressed

patients.

The Barthel ADL Index was completed for all of the patients at all three stages of assessment, and all of the patients completed the GDS at 1 month poststroke. The patients were placed into one of two groups according to their GDS scores. The first group included all patients with clinically significant symptoms of depression (either mild or severe), while the second group included all patients with no clinically significant symptoms of depression. Twenty-one patients had clinically significant symptoms of depression at 1 month poststroke, and the remaining 42 patients had no clinically significant symptoms of depression. As expected, the depressed group did have significantly higher GDS scores than the non-depressed group, t (61) = -12.46, $p <$ .000001.

Table 24 shows the Barthel ADL Index scores according to depression status. The results of the MANOVA are presented in Figure 1. There was no significant difference in the average of all the Barthel ADL Index scores between the two groups of patients, F (1, 61) = 0.37, $p > .05$. There was no significant difference in the average Barthel ADL Index scores across time, F (2, 122) = 0.35, $p > .05$. There was a significant interaction between the depressed and non-depressed patients and the Barthel ADL Index scores at 1, 3, and 12 months poststroke, F (2, 122) = 3.86, p = .023766. Post hoc analysis determined that depressed stroke patients had significantly lower Barthel ADL Index scores at 1 month poststroke than the patients with no depression (Scheffe test; p = .035919). There was no significant difference between depressed and non-depressed patients with regard to their Barthel ADL scores at 3 and 12 months poststroke ($p > .05$).

Therefore, there was a significant interaction between depression and the Barthel ADL Index scores; the non-depressed stroke patients on average were less impaired than the depressed stroke patients. However, the depressed stroke patients had significantly

Table 24

Comparison of Depressed and Non-Depressed Patients, and of Patients Who Have Had a Previous Stroke, and Those With No Previous Stroke, With Regard to Scores on the Barthel ADL Index and the CAS

		Time Poststroke		
		1 month	3 months	12 months
Group	*n*	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Physical Impairment				
Depressed	21	16.43 (3.67)	17.05 (3.64)	17.38 (3.19)
Non-depressed	42	17.86 (4.24)	17.71 (4.10)	17.21 (4.88)
Cognitive Impairment				
Depressed	20	28.45 (4.84)	29.30 (4.45)	30.35 (3.87)
Non-depressed	37	29.89 (4.90)	30.22 (4.55)	30.41 (4.12)
Physical Impairment				
Previous stroke	14	16.43 (5.02)	17.07 (4.53)	17.00 (4.66)
No previous stroke	49	17.65 (3.79)	17.61 (3.80)	17.35 (4.32)
Cognitive Impairment				
Previous stroke	12	28.75 (4.20)	30.00 (3.95)	30.58 (4.25)
No previous stroke	45	29.56 (5.08)	29.87 (4.67)	30.33 (3.98)

Note. CAS = Cognitive Assessment Scale.

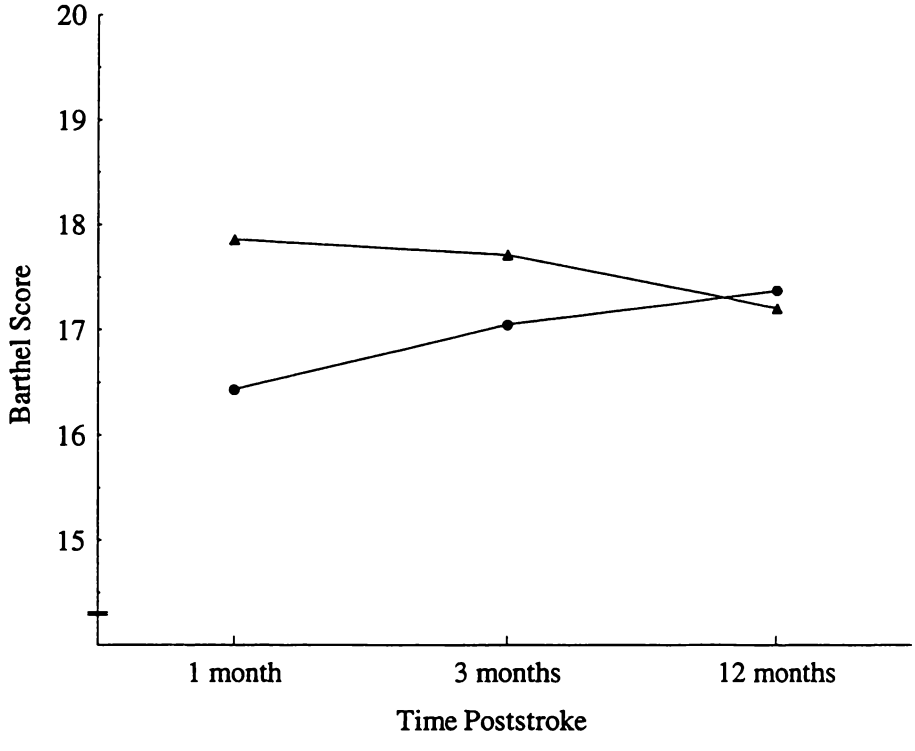

Figure 1. Comparison of the performance of the depressed (circles) and non-depressed (triangles) stroke patients on the Barthel ADL Index at 1, 3, and 12 months poststroke.

lower Barthel ADL scores at 1 month poststroke than the non-depressed patients. This difference between the groups decreased over time, and by 3 and 12 months poststroke there was no statistically significant difference between the two groups of patients.

Depression and Cognitive Impairment

MANOVA was used to determine whether the patients with symptoms of depression at 1 month poststroke had more basic cognitive impairment (as measured by the CAS) throughout the first 12 months poststroke, and if the depressed patients demonstrated less improvement in cognitive functioning, than the non-depressed stroke patients.

All of the stroke patients completed the GDS at 1 month poststroke, and 57 patients completed the CAS at all three stages of assessment. Therefore, 57 patients were included in this analysis. As with the analysis of physical impairment and PSD, the patients were placed into one of two groups according to their GDS scores. The first group included all patients with clinically significant symptoms of depression (either mild or severe), while the second group included all patients with no clinically significant symptoms of depression at 1 month poststroke. Twenty patients had clinically significant symptoms of depression at 1 month poststroke, and the remaining 37 patients had no clinically significant symptoms of depression. As expected, the depressed group did have significantly higher GDS scores than the non-depressed group, $t\,(55) = -11.99, p < .000001$.

Table 24 shows the CAS scores according to depression status. The results of the MANOVA are presented in Figure 2. There was no significant difference in the average of all the CAS scores between the two groups of patients, $F\,(1, 55) = 0.51, p$

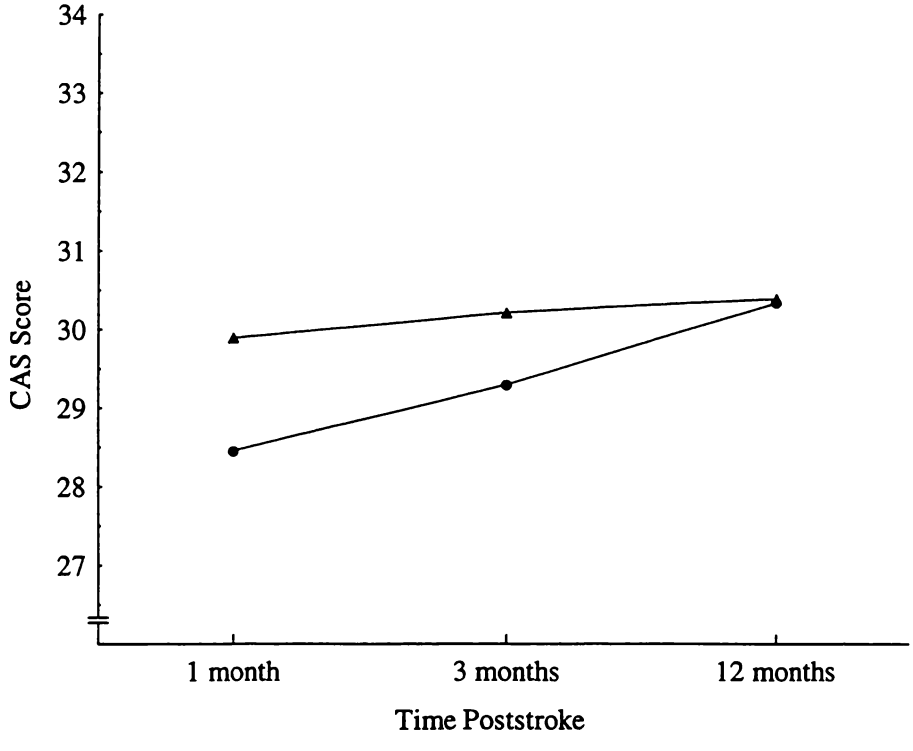

Figure 2. Comparison of the performance of the depressed (circles) and non-depressed (triangles) stroke patients on the Cognitive Assessment Scale (CAS) at 1, 3, and 12 months poststroke.

> .05. However, there was a significant difference in average CAS scores across time, regardless of depression status, F (2, 110) = 3.34, p = .039061. The average CAS scores of the all the patients (regardless of depression status) increased, indicating an overall improvement in cognitive functioning. Post hoc analysis determined that there was a significant difference between the average CAS scores at 1 and 12 months poststroke (Fishers LSD; p = .028771). This result differs from the earlier comparison of CAS scores, where depression status was not a factor in the ANOVA equation. As this present MANOVA is a more rigorous statistical procedure, these results indicate that overall the patients demonstrated an improvement in basic cognitive functioning.

There was no significant interaction between the groups and the CAS scores at 1, 3, and 12 months poststroke, F (2, 110) = 1.12, p > .05. Therefore, although there was a significant improvement in cognitive functioning overall, the depressed stroke patients did not demonstrate either more impairment or less improvement in cognitive functioning than the non-depressed patients.

Previous Stroke and Physical Impairment

It was mentioned in the previous chapter that the continuing relationship between depression and previous stroke may be due, at least in part, to the patients who have had a previous stroke having either more physical and/or cognitive impairment, or demonstrating less improvement over time than patients who have not had a previous stroke.

MANOVA was used to determine whether the patients who had experienced a previous stroke had more physical impairment (as measured by the Barthel ADL Index) throughout the first 12 months poststroke, and if the patients who have had a previous stroke demonstrated less improvement in physical functioning, than the

patients who had not had a previous stroke.

The Barthel ADL Index was completed for all of the patients at each of the three stages of assessment. Fourteen patients had experienced a previous stroke; the remaining 49 patients had not experienced a previous stroke.

Table 24 shows the Barthel ADL scores according to history of stroke. There was no significant difference in the average of all Barthel ADL Index scores between the stroke patients who had experienced a previous stroke, and those for whom this was their first stroke, $F(1, 61) = 0.35, p > .05$. There was no significant difference in average Barthel ADL scores across time, $F(2, 122) = 0.41, p > .05$. There was no significant interaction between the groups and the Barthel ADL Index scores at 1, 3, and 12 months poststroke, $F(2, 122) = .96, p > .05$. Therefore, there was no significant difference in physical functioning between the two groups of patients, nor did the patients who had experienced a previous stroke demonstrate significantly less improvement in physical functioning than the patients for whom this was their first stroke.

Previous Stroke and Cognitive Impairment

MANOVA was used to determine whether the patients who had experienced a previous stroke had more cognitive impairment (as measured by the CAS) throughout the first 12 months poststroke, and if the patients who had experienced a previous stroke demonstrated less improvement in cognitive functioning, than the patients for whom this was their first stroke.

Of the 63 patients included in the longitudinal analysis, 57 patients completed the CAS at all three stages of assessment. Therefore, 57 patients were included in this analysis. Of these 57 patients, 12 had experienced a previous stroke; this was the first

stroke for the remaining 45 patients.

Table 24 shows the CAS scores according to history of stroke. There was no significant difference in the average of all CAS scores between the stroke patients who had experienced a previous stroke, and those for whom this was their first stroke, $F(1, 55) = .01, p > .05$. There was no statistically significant difference in average CAS scores across time, $F(2, 110) = 2.86, p > .05$. There was no significant interaction between previous stroke and the CAS scores at 1, 3, and 12 months poststroke, $F(2, 110) = .55, p > .05$. Therefore, there was no statistically significant difference in cognitive functioning between the two groups of patients, nor did the patients who had experienced a previous stroke demonstrate significantly less improvement in cognitive functioning than the patients for whom this was their first stroke.

Depression and Mortality

Statistical analysis was used to determine whether depression at 1 month poststroke was related to mortality within 12 months poststroke. Of the 103 stroke patients originally included in the study, 13 patients were excluded as they were aphasic at 1 month poststroke, and 2 patients were too impaired at 1 month poststroke to provide reliable responses to the GDS. Therefore it was possible to assess the relationship between depression and mortality using the remaining 88 patients.

Depression status was determined from oral administration of the GDS at 1 month poststroke, which all remaining 88 patients completed. Each patient was categorised as either depressed or not depressed; patients with symptoms of either mild or severe depression were classified as depressed. Demographic information was

used to determine the mortality status of each patient at 12 months poststroke.

Thirty-one (35%) patients scored as depressed on the GDS; the remaining 57 (65%) patients did not have clinically significant symptoms of depression. As expected, the depressed group did have significantly higher GDS scores than the non-depressed group, t (86) = -14.28, $p < .000001$.

By 12 months poststroke 15 (17%) of the 88 patients had died. Of the 15 patients who died, 7 patients had been depressed at 1 month poststroke; the remaining 8 patients had not been depressed. Of the 73 patients who were still alive, 24 patients had been depressed at 1 month poststroke; the remaining 49 patients had not been depressed.

Although a higher rate of depression was evident amongst those patients who died, statistical analysis determined that depression status at 1 month poststroke was not related to mortality within the first 12 months poststroke, Pearson Chi-square, X^2 (1, N = 88) = 1.04, $p > .05$. Therefore depressed stroke patients were not more likely than non-depressed patients to have died by 12 months poststroke.

Discussion

No clear pattern of physical or cognitive improvement was evident in the present group of stroke patients during the first 12 months poststroke. However, there were many individual changes in the levels of depression, and physical and cognitive impairment.

On average, performance on the Barthel ADL Index did not indicate a clear pattern of physical recovery, although some individual patients demonstrated recovery of physical functioning. That is, while 18 patients improved their level of physical

functioning, 16 patients experienced a decline in the physical abilities examined by this measure. However, there was slight improvement in the four types of activities examined in the Nottingham Extended ADL Index. Jorgensen et al. (1995b) found that those stroke patients receiving rehabilitation recovered most of their ADL functioning within the first 3 months poststroke. This clearly was not the case in the present study, as there was very little improvement in ADL abilities between 1 and 3 months poststroke. It is possible that these results are not consistent with those of Jorgensen et al. as the present group of stroke patients were initially assessed at the time of discharge from hospital. For many of these patients, leaving the hospital marked the end of their rehabilitation; therefore, without continuing therapy, there was limited improvement in their physical functioning. It is also possible that a clear improvement in ADL functioning was not evident in all of the patients as much of the improvement in ADL functioning may have occurred in many patients prior to the first assessment. If the examiner had assessed the physical functioning of these patients within the first few days after the stroke, improvement may have been more evident.

Timing of assessment may also explain the improvement, however small, in the performance of the extended activities. The patient is unlikely to become aware of their inability to complete these types of activities until a later stage of the poststroke period, and is therefore more likely to attempt to improve their functioning in these areas at a later stage. This improvement may not have been evident earlier in the poststroke period even if performance of these activities had been assessed at the acute poststroke stage. If this hypothesis is correct, improvement in these extended activities may continue beyond 12 months poststroke.

As with physical improvement, the present group of patients did not display a

clear pattern of decreasing depression in the first 12 months poststroke. At each of the stages of assessment, approximately one third of the stroke patients experienced some form of depression. Nineteen patients reported increasing symptoms of depression within the first 12 months poststroke, and 18 patients reported decreasing symptoms of depression. These results are consistent with the findings of Robinson et al. (1987), who reported a similar prevalence of depression that was constant for the first 2 years poststroke, with individuals changing depression status within that time. However, other researchers have reported that the prevalence of PSD varies throughout the poststroke period (e.g., Astrom, Adolfsson, et al., 1993). These differences in reported prevalence rates may be due to differing diagnostic criteria; to diagnose depression Astrom, Adolfsson, et al. used the *DSM-III* (1980), whereas Robinson et al. used three depression rating scales. Gainotti (1992) stated that the *DSM* may not be suitable for diagnosing PSD. Research indicating fluctuations in the prevalence of PSD may be based on unsuitable diagnoses. Therefore, the prevalence of PSD throughout the poststroke period may not vary as much as previously suggested by some researchers (e.g., Astrom, Adolfsson, et al., 1993).

Although the rates of depression did not improve substantially, some improvement was evident in the reported symptoms of psychological disorder, as measured by the GHQ-28. Only 1 patient experienced a clinically significant increase in symptoms of psychological disorder within the first 12 months poststroke. In accordance with this finding, there was a significant decrease in GHQ-28 scores, although the change in the prevalence of clinically significant symptoms of psychological disorder was not significant. The GHQ-28 has been described as a "valid measure of depression" (Robinson & Price, 1982, p. 636). However, the results of this

study indicate that it may measure depression of a greater severity, or an altogether different construct, than the GDS, as the prevalence rates indicated by the GDS and the GHQ-28 differ substantially.

Although rarely statistically significant, on average slight improvement was evident during the first 12 months poststroke in some of the aspects of cognitive functioning. Basic cognitive functioning as measured by the CAS increased on average; only 10 patients experienced declining basic cognitive functioning, while 19 patients experienced improvement in this area. Results from the MANOVA indicated that there was significant improvement in basic cognitive functioning, regardless of the depression status of the patients. Therefore, on average, the basic cognitive functioning of the patients did improve during the first 12 months poststroke. Simple attention abilities also improved slightly over the 12 month period, with 10 patients experiencing a decline in simple attention abilities, while the simple attention abilities of 13 patients improved. Interestingly, complex attention abilities *decreased* significantly during the first 12 months poststroke. Eight patients experienced declining abilities in this area, while only 4 patients demonstrated improvement of their complex attention abilities. The patients that demonstrated decreasing complex attention abilities did not all deteriorate, or improve, in any other aspect of cognitive, physical, or affective functioning assessed. There appears to be no extraneous factor responsible for the decrease evident in this study, it may simply be that this aspect of cognitive functioning decreases with time following a stroke.

On average, performance on tasks of general intelligence and verbal fluency barely changed during the 12 month period. Ten patients demonstrated decreasing verbal fluency, while 12 patients demonstrated improving fluency. Similarly, no clear

improvement or deterioration was evident for immediate verbal memory. Eleven patients experienced declining immediate verbal memory, while 18 patients improved in this area. However, statistically significant improvement was evident in delayed verbal memory during the first 12 months poststroke. While declining delayed verbal memory was demonstrated by only 4 patients, 14 patients demonstrated improvement on this task.

Therefore, on average, scores of the patients' performances on most of the aspects of cognitive functioning examined indicate little cognitive improvement during the first 12 months poststroke, despite a large number of individual changes in cognitive functioning. The previous chapter suggested that the failure to find a relationship between cognitive impairment and depression at 12 months poststroke may be due to improvement of cognitive functioning if the depression is a reaction to cognitive impairment. The present results indicate that this is not the case, as, on average, little cognitive improvement was demonstrated within the first 12 months poststroke (although individual patients did demonstrate improvement of cognitive functioning). The present group of stroke patients demonstrated significant improvement in delayed verbal memory, and basic cognitive functioning, while significant deterioration of complex attention abilities was evident. Reasons for the paucity of overall improvement are not clear. However, when considering the cognitive improvement of individual patients, approximately one third of patients demonstrated improvement of basic cognitive functioning within the first 12 months poststroke. With the exception of complex attention abilities (in which only 8% of patients improved), 19 - 31% of patients demonstrated improvement on the remainder of the cognitive measures. Previous studies have reported similar results. Desmond et al. (1996)

reported long-term cognitive improvement in 19 of 151 stroke patients. As cognitive impairment was not evident in all 151 of their patients at the initial evaluation, they determined that approximately one third of patients who had demonstrated impairment of cognitive functioning at the initial evaluation did improve in the long-term. Therefore, although it appears that, on average, improvement of cognitive functioning in the present group of stroke patients was scarce, the rate of improvement demonstrated in the present study is not uncommon.

Previous research has indicated that, in comparison with non-depressed stroke patients, depressed stroke patients recover considerably less of their physical and cognitive functioning (Astrom, Adolfsson, et al., 1993; Downhill & Robinson, 1994; Morris, Raphael, et al., 1992; Parikh et al., 1990). This was not the case in the present group of stroke patients, as physical and cognitive recovery were not affected by depression. However, compared with the non-depressed stroke patients, the depressed patients demonstrated more physical impairment at 1 month poststroke.

In the assessment of the patients at 12 months poststroke it was suggested that the duration of the relationship between previous stroke and PSD may be, at least in part, due to a third factor such as physical and/or cognitive impairment. That is, patients who have experienced a previous stroke may have more cognitive and/or physical impairment, or demonstrate less improvement in these areas, than patients who are experiencing their first stroke. This impairment or lack of improvement then impacts on the relationship between previous stroke and PSD. However, this was not the case in the present study; a history of previous stroke did not result in less improvement, or more impairment of physical and/or cognitive functioning in this group of stroke patients.

The final hypothesis examined in this longitudinal assessment was the relationship between PSD at 1 month poststroke, and mortality within the first 12 months poststroke. Previous research has indicated that depressed stroke patients are likely to die earlier than non-depressed patients (Morris, Robinson, & Samuels, 1993). However, this study found no evidence of a relationship between PSD and mortality. This finding was not unprecedented; Astrom, Adolfsson, et al. (1993) also found no relationship between PSD and mortality in their 3-year follow-up of stroke patients. As Morris, Robinson, and Samuels (1993) followed-up their patients for 10 years, it is possible that the relationship between depression and mortality is only evident later in the poststroke period. It is also possible that deaths later in the poststroke period may simply be due to the natural aging process of the stroke patients.

In summary, while no distinct pattern of overall improvement in levels of depression, or physical and cognitive functioning was evident, there were many individual changes. Although many patients demonstrated changes in the levels of depression, and in the degree of physical and/or cognitive functioning, no clear pattern of overall improvement or deterioration was evident. Approximately one third of the patients improved their level of cognitive functioning. Significant improvement of cognitive functioning was evident in delayed verbal memory, and in basic cognitive functioning. Significant deterioration was evident in complex attention abilities. No substantial improvement was evident in either depression or the physical functioning of the group as a whole. In addition, neither depression nor a history of previous stroke affected improvement in physical or cognitive functioning, although the depressed patients did have more physical impairment at 1 month poststroke than the non-depressed patients. Finally, there was no relationship between depression at the acute

poststroke stage, and mortality within the first 12 months poststroke. The paucity of overall improvement in these stroke patients is alarming, despite the improvement demonstrated by individual patients. Clearly more work is needed in this area, both in research, and in developing better access to rehabilitation for all stroke patients.

CHAPTER 7

Psychological Distress in Caregivers of Stroke Patients

Previous research has indicated that caring for a stroke patient can cause the caregiver substantial psychological distress (Anderson et al., 1995; Bethoux et al., 1996; Carnwath & Johnson, 1987; Wade et al., 1986). In addition, difficulties occurring as a result of caring for the patient often lead to the caregiver feeling burdened by their role. These difficulties are often referred to as objective burden, while the feelings experienced by the caregiver are referred to as subjective burden (Kausar & Powell, 1996). This subjective burden is a form of psychological distress. Caregiver psychological distress may be increased by caring for a depressed patient (Wade et al., 1986). Other factors may also be responsible for caregiver psychological distress, including caring for a severely impaired patient, length of time in the caregiving role, degree of objective burden, lack of social contact, lack of social support, and dissatisfaction at the patient's level of functioning. This assessment of caregiver functioning will examine whether patient PSD and the other aforementioned factors are associated with psychological distress in the caregivers of the patients involved in this study.

Knight (1992) discussed a model of caregiver stress that was based on the work by Lazarus and Folkman (1984, cited in Knight). This three-level model can be easily applied to the stress experienced by caregivers of stroke patients. The first level of the model (the "stressors") includes the level of patient functioning, and objective burden (the difficulties that arise as a result of the impairments). The second level comprises the value the caregiver places on these stressors, the caregiver's ability to

cope, and the support they receive. These factors lead to the final level of the model: the stress experienced by the caregiver (psychological distress). The model is useful as it does not assume that caregiver stress is a result of the *severity* of the stressors, but suggests that caregiver stress may also be a result of the individual caregiver's interpretation of, and response to, the stressors. That is, caregiver stress may be mediated by those factors presented on the second level of the model. This chapter will examine the extent of psychological distress experienced by the caregivers of patients involved in this study, and attempt to analyse that distress in terms of this model.

Although reported prevalence rates vary, depression and psychological distress clearly occur in caregivers of stroke patients. Anderson et al. (1995) and Wade et al. (1986) assessed caregivers at 12 months poststroke. The prevalence rates of caregiver depression and psychological distress reported in these studies were vastly different. While Wade et al. found that approximately 10% of caregivers were emotionally distressed, Anderson et al. reported a much higher prevalence of 79%. In studies examining the wellbeing of caregivers of stroke patients beyond 1 year poststroke, Carnwath and Johnson (1987) reported that the prevalence of psychological distress in their caregiver participants (between 1 and 3 years poststroke) was 40%, while Bethoux et al. (1996) reported that, at an average of 18 months poststroke, 20% of the caregivers in their study presented with symptoms of depression. Regardless of these large differences, caring for stroke patients clearly causes substantial distress and is worthy of further attention.

The likelihood of a caregiver experiencing some form of psychological distress has been found to increase with the length of time in the caregiving role (Carnwath &

Johnson, 1987). Similarly, caregiver satisfaction has also been associated with the duration of caregiving (Segal & Schall, 1996). As the length of time in the caregiving role, and perhaps the intensity of the caregiving, increases with the number of strokes had by the patient, it is understandable that caregivers of patients who have experienced more than one stroke are also more likely to report dissatisfaction (Segal & Schall, 1996). However, these relationships may also be confounded by other factors, such as social support. That is, although the stroke patient and caregiver may initially receive an abundance of support from friends and family, this support may gradually diminish with time.

The overall wellbeing of the patient clearly has an impact on the caregiver. As mentioned previously, Wade et al. (1986) reported that depression in the caregiver within the first 12 months poststroke was associated with caring for a depressed patient. In addition to PSD, the patient's level of physical and cognitive functioning can also affect the psychological wellbeing of the caregiver. While some researchers have failed to find a significant relationship between the level of physical functioning in the patient and caregiver psychological distress (Anderson et al., 1995), other studies have reported evidence of this relationship (Carnwath & Johnson, 1987; Thompson et al., 1990; Wade et al., 1986). At 12 months poststroke, Wade et al. found that caregiver depression was associated with the patient's level of physical functioning, including the patient's level of activity and degree of recovery. Carnwath and Johnson also found this relationship in stroke patients and caregivers assessed between 1 and 3 years poststroke. Despite failing to find a relationship between caregiver depression and patient disability, Bethoux et al. (1996) found that the extent of the physical disability of the patient was associated with the caregivers' reported

quality of life.

In contrast to these findings, improvement in the physical functioning of stroke patients has been associated with increased burden and dissatisfaction in caregivers (Elmstahl, Malmberg, & Annerstedt, 1996). Reasons for this relationship are not clear, although the researchers suggested that the caregiver may fear losing their social support as the patient recovers their physical functioning.

Limited research has been conducted looking at the effect of the patient's cognitive impairment on the psychological wellbeing of the caregiver. However, Anderson et al. (1995) reported that caregivers were more likely to experience emotional distress if they were caring for a patient with signs of dementia. An inability to communicate effectively is likely to be the causal factor in this relationship.

In addition, there is no doubt that both the level and quality of social support have an impact on the caregiver's wellbeing, as caregivers receiving social support tend to report fewer symptoms of psychological distress (Carnwath & Johnson, 1987; Tompkins, Schulz, & Rau, 1988). However, this relationship may be bi-directional; just as the caregiver without sufficient social support may experience psychological distress, they may have less social support *because* of that distress, as people may be less inclined to want to spend time with a distressed person (Tompkins et al.). Regardless of the possibility that a lower degree of social support may be a response to caregiver psychological distress, social support and social contact are obviously fundamental to the psychological wellbeing of caregivers.

All of the difficulties that arise as a result of caregiving surely increase the burden felt by the caregiver. In a study of neurological patients, of which the majority were stroke patients, Kausar and Powell (1996) found a relationship between

caregiver objective and subjective burden. This association strengthened with time. As the overall psychological wellbeing of the caregiver is clearly affected by the length of time in the caregiving role, it appears that longitudinal analysis is necessary when examining the effects of caregiving on caregivers of stroke patients.

In summary, many caregivers of stroke patients clearly experience psychological distress. In addition to caring for a depressed patient, there are a number of factors associated with this distress. Many of these factors can be applied to the model of caregiver stress described by Knight (1992). This chapter will determine the prevalence of psychological distress experienced by the caregivers of the stroke patients assessed in this study, and identify factors associated with their distress, in turn determining whether the caregiver distress reported by the present group of caregivers is consistent with the model of caregiver stress. Patient PSD, patient physical and cognitive impairment, previous stroke, length of time as a caregiver, and objective burden are aspects of the present study that constitute the first level of the model of caregiver stress. Caregiver satisfaction, and support measures constitute the second level of the model. Measures of psychological distress constitute the third level of the model of caregiver stress, where psychological distress is assessed in terms of psychological disorder, and subjective burden.

Method

Participants

Three months poststroke. Thirty-four caregivers were assessed at the 3-month follow-up. Of these 34 caregivers, the assessment of 1 caregiver was excluded from analysis as the patient they were caring for was receiving medication as treatment for

depression, and the assessments of 5 caregivers were excluded as the patients they were caring for were aphasic, as determined by the administration of the short-form of the Minnesota Differential Diagnosis of Aphasia Test (Powell et al., 1980). The remaining 28 caregivers and patients were included in the assessment of caregiver functioning at 3 months poststroke.

The average age of these caregivers was 69 years (*SD* = 8, range = 53 - 82 years). Twenty-two (79%) caregivers were female, and 6 (21%) caregivers were male. Twenty-seven (96%) of the caregivers were caring for their partner, and 1 (4%) caregiver was caring for their parent. These 28 caregivers had been caring for the patients, on average, for 15 months (*SD* = 31, range = 3 - 120). For 20 (71%) of these patients this was their first stroke. The remaining 8 (29%) patients had experienced at least one previous stroke, with 2 of these latter 8 patients having had two previous strokes.

Twelve months poststroke. Of the 34 caregivers and patients initially assessed at 3 months poststroke, by 12 months poststroke 3 patients were deceased, 1 patient was not able to be assessed, and 1 caregiver refused to participate. In addition, the assessment of 1 caregiver was excluded from analysis as the patient they were caring for was receiving medication as treatment for depression, and the assessments of 3 caregivers were excluded as the patients they were caring for were aphasic, as determined by the administration of the short-form of the Minnesota Differential Diagnosis of Aphasia Test (Powell et al., 1980). One additional caregiver was introduced at the 12-month follow-up. Therefore 26 caregivers and patients were included in the 12-month follow-up of caregiver functioning.

The average age of these caregivers was 69 years (*SD* = 8, range = 53 - 82

years). Nineteen (73%) caregivers were female, and 7 (27%) caregivers were male. Twenty-five (96%) of the caregivers were caring for their partner, and 1 (4%) caregiver was caring for their parent. These 26 caregivers had been caring for the patients, on average, for 20 months (*SD* = 24, range = 9 - 117). For 19 (73%) of these patients this was their first stroke. The remaining 7 (27%) patients had experienced at least one previous stroke, with 1 of these latter 7 patients having had two previous strokes.

Longitudinal comparison. Of the 28 caregivers and patients assessed at 3 months poststroke, and the 26 caregivers and patients assessed at 12 months poststroke, 24 caregivers and patients were assessed at both stages. Therefore 24 caregivers and patients were included in the longitudinal comparison of caregiver functioning between 3 and 12 months poststroke.

The average age of these caregivers was 69 years (*SD* = 8, range = 53 - 82 years). Eighteen (75%) caregivers were female, and 6 (25%) caregivers were male. Twenty-three (96%) of the caregivers were caring for their partner, and 1 (4%) caregiver was caring for their parent. At 3 months poststroke, these 24 caregivers had been caring for the patients, on average, for 12 months (*SD* = 25, range = 3 - 108). For 17 (71%) of these patients this was their first stroke. The remaining 7 (29%) patients had experienced at least one previous stroke, with 1 of these latter 7 patients having had two previous strokes.

Measures

All caregivers were interviewed in order to provide demographic information, in addition to completing questionnaires.

Caregiver functioning. The 28-item version of the General Health

Questionnaire (GHQ-28; Goldberg, 1972) was administered to determine the presence of psychological disorder. The GHQ scoring method of 0-0-1-1 was used. To determine the presence of psychological disorder, a cutoff of 4/5 was used, where a score of 0 - 4 is within the normal range, and a score of 5 or greater is indicative of psychological disorder. This cutoff has been established as suitable for this version of the GHQ (Goldberg & Hillier, 1979).

The Katz Adjustment Scales-Relatives forms (KAS-R; Katz & Lyerly, 1963) provided information regarding the patient's level of performance (R2) and the caregiver's level of expectations of the patient's performance (R3). The difference between these two KAS-R forms provided a measure of caregiver satisfaction. Possible scores of satisfaction ranged from -32 (very satisfied, patient doing more than expected), to 32 (very dissatisfied, patient doing less than expected). A score of 0 indicated that the caregiver considered the patient to be doing everything expected of them.

Objective and subjective burden were measured using the questionnaires of objective and subjective burden developed by Montgomery et al. (1985). The possible scoring range for objective burden was 9 - 45, where a score of 9 - 26 indicated no burden, a score of 27 indicated no change since the stroke, and a score or 28 - 45 indicated burden. The possible scoring range for subjective burden was 13 - 65; scores increased with the extent of the subjective burden.

The frequency of social contact was measured using the Social Activities Questionnaire (Donald et al., 1978), which provided a measure of social contact, group participation, and overall social support. Scores on the Social Activities Questionnaire increased with the level of social support.

The quality and usefulness of the caregivers' social network was measured using the Duke-UNC Functional Social Support Questionnaire (DUFSS; Broadhead et al., 1988), which is a measure of confidant and affective support. The possible scoring range for confidant support was 5 - 25, and the possible scoring range for affective support was 3 - 15. For both confidant and affective support, a low score indicated a high level of support.

Patient functioning. The Cognitive Assessment Scale (CAS) from the Clifton Assessment Procedures for the Elderly (Pattie & Gilleard, 1979) was administered to provide an overall assessment of the patient's cognitive functioning. The level of physical functioning of the patient was assessed by administration of the Barthel ADL Index (Wade & Langton-Hewer, 1987). Oral administration of the Geriatric Depression Scale (GDS; Yesavage et al., 1983) assessed the patient's level of depression.

Procedure

Each caregiver was interviewed at their first assessment to obtain written consent and demographic information. The interview took approximately 5 minutes to complete. The questionnaires were then explained to the caregiver, following which the caregiver was given the questionnaires to complete while the researcher worked with the patient. The caregiver's questionnaires were presented in the following order: KAS-R, GHQ-28, Measurements of Objective and Subjective Burden, Social Activities Questionnaire, and the DUFSS. The questionnaires took approximately 30 minutes for the caregiver to complete.

Data Analysis

At both 3 and 12 months poststroke, performance on each of the measures

was analysed separately, with results described in terms of the mean (*M*), standard deviation (*SD*), and range of scores. Correlational analysis was also completed at 3 and 12 months poststroke, using Pearson product-moment correlation and point-biserial correlation (r_{pb}; used where one variable was dichotomous). As recommended by Tabachnick and Fidell (1989), regression analysis was not performed due to the small number of caregivers assessed. A longitudinal comparison of the 24 caregivers assessed at both 3 and 12 months poststroke was also completed using dependent *t* tests.

Results

Three Months Poststroke

Patient functioning. The Barthel ADL Index was completed for all patients. Results of the Barthel ADL Index showed that 18 (64%) patients were physically independent, 9 (32%) patients had mild impairment, and 1 (4%) patient had moderate physical impairment. No patients had severe or very severe impairment of physical functioning.

All patients completed the GDS. Twenty (71%) patients had no clinically significant symptoms of depression, 7 (25%) patients reported symptoms of mild depression and 1 (4%) patient reported symptoms of severe depression.

Due to sight and/or writing impairments 2 patients were unable to complete the CAS. Therefore it was possible to assess the basic cognitive functioning of the remaining 26 patients. Twenty-two (85%) patients had no clinically significant impairment of basic cognitive functioning, 2 (8%) patients had mild cognitive impairment, and 2 (8%) patients had moderate cognitive impairment. No patients had

marked or severe impairment of basic cognitive functioning.

Caregiver functioning. All caregivers completed the measures of Objective and Subjective Burden. Out of a possible scoring range of 9 - 45 (with a score of 28 - 45 indicating some level of objective burden), the average objective burden score was 31 (*SD* = 4, range = 25 - 38). Out of a possible scoring range of 13 - 65, the average subjective burden score was 33 (*SD* = 7, range = 25 - 51). Therefore, these caregivers reported a relatively higher degree of subjective burden than objective burden.

All caregivers completed the KAS-R. The average score of satisfaction was 2 (*SD* = 5, range = -5 - 15). Therefore, the caregivers generally indicated some dissatisfaction.

All caregivers completed the Social Activities Questionnaire. The average score of social contact was 8 (*SD* = 2, range = 3 - 10). The average group participation score was 5 (*SD* = 3, range = 1 - 9). The average overall social support score was 28 (*SD* = 6, range = 17 - 40).

All caregivers completed the DUFSS. Out of a possible scoring range of 5 - 25, the average score of confidant support was 9 (*SD* = 4, range = 5 - 21). Out of a possible scoring range of 3 - 15, the average score of affective support was 5 (*SD* = 2, range = 3 - 13). Therefore, although some caregivers reported receiving little confidant and/or affective support, on average they received a relatively high level of support.

All caregivers completed the GHQ-28. Twenty-one (75%) caregivers had no clinically significant symptoms of psychological disorder, while 7 (25%) caregivers did report clinically significant symptoms of psychological disorder.

Correlational analysis. The 2 patients who were unable to complete the CAS

were excluded from the correlational analysis, as information regarding their performance was not complete. Hence their respective caregivers were also excluded from further analysis. Therefore the correlational analysis of the 3-month follow-up included the assessments of 26 patients and their caregivers.

Table 25 shows the results of the correlational analysis at 3 months poststroke. Patient depression was significantly correlated with caregiver symptoms of psychological disorder ($r = .62$, $p = .001$), and subjective burden ($r = .46$, $p = .019$). Therefore, caregivers were more likely to report symptoms of psychological disorder and subjective burden if they were caring for a depressed patient. Patient depression was also significantly correlated with less confidant support ($r = .63$, $p = .001$), and less affective support ($r = .44$, $p = .026$).

In addition to the relationship between caregiver symptoms of psychological disorder and patient depression, symptoms of psychological disorder were more likely to be reported by caregivers of patients who had experienced a previous stroke ($r_{pb} = .62$, $p = .001$), and by those caregivers who had been caring for their patient for a longer period of time ($r = .59$, $p = .002$). Symptoms of psychological disorder were also expressed by caregivers who had less confidant support ($r = .72$, $p < .001$), and less affective support ($r = .79$, $p < .001$). There was no significant relationship between the extent of the patient's physical or cognitive impairment and caregiver symptoms of psychological disorder ($p > .05$).

As with caregiver symptoms of psychological disorder, subjective burden was higher in caregivers of patients who had experienced a previous stroke ($r_{pb} = .43$, $p = .030$), and in caregivers that had been caring for the patient for a longer time ($r = .41$, $p = .039$). Caregivers reporting feelings of subjective burden also believed they had

Table 25

Correlational Analysis of Individual Variables Included in the Analysis of Caregiver Functioning at 3 Months Poststroke ($N = 26$)

Variable	1	2	3	4	5	6	7	8
1. Psychological disorder	--							
2. Subjective burden	.44*	--						
3. Previous stroke	.62*	.43*	--					
4. ADL (Barthel)	-.26	-.08	-.11	--				
5. Patient depression	.62*	.46*	.65*	-.38	--			
6. Basic cognition	.15	-.27	.21	.19	-.04	--		
7. Caregiver length	.59*	.41*	.27	-.16	.29	.07	--	
8. Objective burden	.06	-.18	.16	-.35	.36	-.01	.26	--
9. Social contact	-.13	.15	-.11	.46*	-.28	-.10	.13	-.30
10. Group participation	.19	.24	.25	.25	.11	.29	-.10	-.48*
11. Overall social support	-.13	.13	-.04	.48*	-.27	.22	-.02	-.46*
12. Satisfaction	.13	.32	.34	.19	.10	.30	.20	-.26
13. Confidant support	.72*	.55*	.44*	-.28	.63*	-.14	.33	-.03
14. Affective support	.79*	.51*	.47*	-.26	.44*	.20	.44*	-.13

Table 25 (continued)

Correlational Analysis of Individual Variables Included in the Analysis of Caregiver Functioning at 3 Months Poststroke ($N = 26$)

Variable	9	10	11	12	13	14
9. Social contact	--					
10. Group participation	.19	--				
11. Overall social support	.61*	.70*	--			
12. Satisfaction	.29	.51*	.52*	--		
13. Confidant support	-.23	.21	-.13	-.05	--	
14. Affective support	-.30	.19	-.09	.12	.78*	--

Note. ADL = Activities of daily living.

* $p < .05$.

less confidant support ($r = .55$, $p = .004$), and less affective support ($r = .51$, $p = .008$). There was no significant correlation between subjective burden and extent of the patients' physical or cognitive impairment ($p > .05$). However, the caregivers received more overall social support if the patient was physically impaired ($r = .48$, $p = .014$).

Therefore, in terms of the model of caregiver stress (Knight, 1992), the stressors measured in this study that were related to caregiver psychological disorder

at 3 months poststroke were patient depression, previous stroke, and length of time as caregiver (r = .62, r_{pb} = .62, r = .59, respectively). However, the relationships between caregiver symptoms of psychological disorder and both confidant and affective support (r = .72, and r = .79, respectively) were stronger than the relationship between caregiver symptoms of psychological disorder and the related stressors.

Twelve Months Poststroke

Patient functioning. The Barthel ADL Index was completed for all patients. Results of the Barthel ADL Index showed that 17 (65%) patients were physically independent, and 9 (35%) patients had mild impairment. No patients had moderate, severe, or very severe impairment of physical functioning.

All patients completed the GDS. Twenty-one (81%) patients had no clinically significant symptoms of depression, 4 (15%) patients reported symptoms of mild depression and 1 (4%) patient reported symptoms of severe depression.

Due to sight and/or writing impairments 2 patients were unable to complete the CAS. Therefore it was possible to assess the basic cognitive functioning of the remaining 24 patients. Twenty (83%) patients had no impairment of basic cognitive functioning, 2 (8%) patients had mild cognitive impairment, and 2 (8%) patients had moderate cognitive impairment. No patients had marked or severe impairment of basic cognitive functioning.

Caregiver functioning. All caregivers completed the measures of Objective and Subjective Burden. Out of a possible scoring range of 9 - 45 (with a score of 28 - 45 indicating some level of objective burden), the average objective burden score was 30 (SD = 6, range = 13 - 40). Out of a possible scoring range of 13 - 65, the average

subjective burden score was 33 (*SD* = 6, range = 25 - 49). Therefore, these caregivers reported a higher degree of subjective burden than objective burden.

All caregivers completed the KAS-R. The average score of satisfaction was 4 (*SD* = 6, range = -2 - 18). Therefore, the caregivers generally indicated some dissatisfaction.

All caregivers completed the Social Activities Questionnaire. The average social contact score was 8 (*SD* = 2, range = 3 - 10). The average group participation score was 5 (*SD* = 2, range = 1 - 9). The average overall social support score was 27 (*SD* = 5, range = 15 - 36).

All caregivers completed the DUFSS. Out of a possible scoring range of 5 - 25, the average confidant support score was 9 (*SD* = 5, range = 5 - 23). Out of a possible scoring range of 3 - 15, the average affective support score was 5 (*SD* = 3, range = 3 - 12). Therefore, although some caregivers reported receiving little confidant and/or affective support, on average they received a relatively high level of support.

All caregivers completed the GHQ-28. Twenty-one (81%) caregivers had no clinically significant symptoms of psychological disorder, while 5 (19%) caregivers did have clinically significant symptoms of psychological disorder.

Correlational analysis. The 2 patients who were unable to complete the CAS were excluded from the correlational analysis, as information regarding their performance was not complete. Hence their respective caregivers were also excluded from further analysis. Therefore the correlational analysis of the 12-month follow-up included the assessments of 24 patients and their caregivers.

Table 26 shows the results of the correlational analysis at 12 months

Table 26

Correlational Analysis of Individual Variables Included in the Analysis of Caregiver Functioning at 12 Months Poststroke ($N = 24$)

Variable	1	2	3	4	5	6	7	8
1. Psychological disorder	--							
2. Subjective burden	.48*	--						
3. Previous stroke	-.07	.37	--					
4. ADL (Barthel)	-.13	-.21	.12	--				
5. Patient depression	.11	.25	.53*	-.25	--			
6. Basic cognition	-.06	-.13	.29	.04	.06	--		
7. Caregiver length	.40	.08	.004	.16	-.09	-.07	--	
8. Objective burden	.44*	.27	-.01	-.51*	.55*	-.21	-.06	--
9. Social contact	-.20	-.03	-.21	.54*	-.50*	-.12	.19	-.50*
10. Group participation	-.29	.04	.20	.16	-.17	.43*	-.31	-.26
11. Overall social support	-.45*	-.15	-.16	.38	-.56*	.23	-.04	-.57*
12. Satisfaction	.12	-.13	.14	-.32	.19	.13	-.16	.37
13. Confidant support	.62*	-.54*	.12	-.15	.51*	-.12	-.02	.45*
14. Affective support	.41*	.38	.14	-.17	.39	-.18	.37	.27

Table 26 (continued)

Correlational Analysis of Individual Variables Included in the Analysis of Caregiver Functioning at 12 Months Poststroke ($N = 24$)

Variable	9	10	11	12	13	14
9. Social contact	--					
10. Group participation	.27	--				
11. Overall social support	.72*	.68*	--			
12. Satisfaction	-.52*	.002	-.24	--		
13. Confidant support	-.35	-.27	-.52*	.09	--	
14. Affective support	-.15	-.28	-.37	-.12	.64*	--

Note. ADL = Activities of daily living.

* $p < .05$.

poststroke. Patient depression was not significantly correlated with caregiver symptoms of psychological disorder ($p > .05$). However, caregivers of depressed stroke patients reported less social contact ($r = -.50$, $p = .013$), less overall social support ($r = -.56$, $p = .004$), and less confidant support ($r = .51$, $p = .011$).

Caregiver symptoms of psychological disorder were significant correlated with objective burden ($r = .44$, $p = .033$), less confidant support ($r = .62$, $p = .001$), and less affective support ($r = .41$, $p = .046$). Caregivers reporting symptoms of

psychological disorder also reported receiving less overall social support (r = -.45, p = .026). Subjective burden was significantly correlated with less confidant support (r = .54, p = .007).

Therefore, in terms of the model of caregiver stress (Knight, 1992), the only stressor measured in this study that was related to caregiver symptoms of psychological disorder at 12 months poststroke was objective burden (r = .44). This relationship between caregiver symptoms of psychological disorder and objective burden was similar in strength to the relationship between caregiver psychological disorder and overall social support (r = -.45), but was not as strong as the relationship between caregiver symptoms of psychological disorder and confidant support (r = .62). However, the relationship between caregiver symptoms of psychological disorder and affective support (r = .41) was slightly weaker than the relationship between objective burden and caregiver symptoms of psychological disorder.

Longitudinal Comparison of Caregiver Functioning

Twenty-four caregivers were included in the longitudinal comparison of caregiver functioning as they completed all the caregiver measures at both 3 and 12 months poststroke. Table 27 shows the changes between 3 and 12 months poststroke in the average scores of the caregiver measures.

Objective and Subjective Burden. The average objective and subjective burden scores are presented in Table 27. The average objective burden score at 3 months poststroke was 30.92 (SD = 3.90, range = 25 - 38), and the average objective burden score at 12 months poststroke was 29.38 (SD = 5.48, range = 13 - 39). The difference between these objective burden scores was not significant, t (23) = 1.59, p > .05. The average subjective burden score at 3 months poststroke was 32.04 (SD =

Table 27

Longitudinal Comparison of the Average Scores of the Measures of Caregiver Functioning at 3 and 12 Months Poststroke ($N = 24$)

	Time Poststroke		
	3 months	12 months	
Measure	*M* (*SD*)	*M* (*SD*)	*t*
GHQ-28 total score	2.25 (3.23)	2.42 (4.36)	-0.22
Subjective burden	32.04 (5.53)	33.08 (6.14)	-0.95
Objective burden	30.92 (3.90)	29.38 (5.48)	1.59
Social contacts	8.25 (2.09)	7.75 (2.19)	1.81
Group participation	4.75 (2.66)	4.96 (2.54)	-0.54
Overall social support	27.50 (5.70)	27.38 (5.54)	0.16
Confidant support	8.04 (3.26)	8.83 (4.98)	-0.96
Affective support	4.54 (1.96)	4.75 (2.61)	-0.34
Satisfaction	1.71 (5.03)	3.42 (5.75)	-1.33

Note. GHQ-28 = 28 item version of the General Health Questionnaire.

5.53, range = 25 - 43), and the average subjective burden score at 12 months poststroke was 33.08 (*SD* = 6.14, range = 25 - 49). The difference between these subjective burden scores was not significant, t (23) = -0.95, $p > .05$.

Caregiver satisfaction. The average caregiver satisfaction scores are presented in Table 27. The average satisfaction score at 3 months poststroke was 1.71 (*SD* = 5.03, range = -5 - 15), and the average satisfaction score at 12 months poststroke was 3.42 (*SD* = 5.75, range = -2 - 18). The difference between these caregiver satisfaction scores was not significant, t (23) = -1.33, $p > .05$.

Social support. The average social support scores are presented in Table 27. The average social contact score at 3 months poststroke was 8.25 (*SD* = 2.09, range = 3 - 10), and the average social contact score at 12 months poststroke was 7.75 (*SD* = 2.19, range = 3 - 10). The difference between these average scores of social contact was not significant, t (23) = 1.81, $p > .05$. The average group participation score at 3 months poststroke was 4.75 (*SD* = 2.66, range = 1 - 8), and the average group participation score at 12 months poststroke was 4.96 (*SD* = 2.54, range = 1 - 9). The difference between these average scores of group participation was not significant, t (23) = -0.54, $p > .05$. The average overall social support score at 3 months poststroke was 27.50 (*SD* = 5.70, range = 17 - 37), and the average social support score at 12 months poststroke was 27.38 (*SD* = 5.54, range = 15 - 36). The difference between these overall social support scores was not significant, t (23) = 0.16, $p > .05$.

Confidant and affective support. The average confidant and affective support scores are presented in Table 27. The average confidant support score at 3 months poststroke was 8.04 (*SD* = 3.26, range = 5 - 16), and the average confidant support score at 12 months poststroke was 8.83 (*SD* = 4.98, range = 5 - 23). The difference between these confidant

support scores was not significant, t (23) = -0.96, $p > .05$. The average affective support score at 3 months poststroke was 4.54 (SD = 1.96, range = 3 - 9), and the average affective support score at 12 months poststroke was 4.75 (SD = 2.61, range = 3 - 12). The difference between these average scores of affective support was not significant, t (23) = -0.34, $p > .05$.

Symptoms of psychological disorder. The presence of clinically significant symptoms of psychological disorder at 3 and 12 months poststroke is presented in Table 28. At 3 months poststroke, 19 (79%) caregivers reported no clinically significant symptoms of psychological disorder, while the remaining 5 (21%) caregivers did report these symptoms. At 12 months poststroke, the number of caregivers reporting clinically significant symptoms of psychological disorder had decreased to 4 (17%); the remaining 20 (83%) caregivers reported no such symptoms.

Three caregivers with clinically significant symptoms of psychological disorder at 3 months poststroke still had those symptoms at 12 months poststroke. Two of the caregivers with symptoms at 3 months poststroke did not report those symptoms at 12 months poststroke, and 1 caregiver who had not reported symptoms at 3 months poststroke did have clinically significant symptoms of psychological disorder at 12 months poststroke.

The change in the prevalence of clinically significant symptoms of psychological disorder between 3 and 12 months poststroke was not significant, McNemar Chi-square, X^2 (4, N = 24) = 0.00, $p > .05$.

The average GHQ-28 total scores are presented in Table 27. The average GHQ-28 total score at 3 months poststroke was 2.25 (SD = 3.23, range = 0 - 13), increasing slightly to 2.42 (SD = 4.36, range = 0 - 16) at 12 months poststroke. As

Table 28

Presence of Psychological Disorder in the Caregivers at 3 and 12 Months Poststroke ($N = 24$)

	Time Poststroke	
Presence of Psychological Disorder	3 months *n* (%)	12 months *n* (%)
Absent	19 (79%)	20 (83%)
Present	5 (21%)	4 (17%)

was expected, the difference between these average GHQ-28 total scores was not statistically significant, $t(23) = -0.22$, $p > .05$.

Discussion

This chapter examined the prevalence of psychological distress in the caregivers of the stroke patients involved in this study, where psychological distress was measured in terms of psychological disorder and subjective burden. Psychological distress was evident in this group of caregivers of stroke patients, as psychological disorder was present in 25% of these caregivers at 3 months poststroke, decreasing to 19% by 12 months poststroke. A number of variables were related to caregiver symptoms of

psychological disorder at 3 and 12 months poststroke. These relationships were examined in terms of their consistency with the model of caregiver stress outlined in Knight (1992).

It must be noted that the patients involved in this analysis of caregiver functioning had a relatively high level of both physical and cognitive functioning. However, the prevalence of patient depression at 3 and 12 months poststroke was approximately 30% and 20% respectively. Therefore, analysis of the caregiver assessments should be considered carefully, as while these caregivers were caring for patients with overall rates of depression similar to that of the entire sample examined in this study, they were caring for patients with disproportionately less physical and cognitive impairment. This situation is expected as patients with higher levels of impairment are likely to be in residential homes or hospitals, in which case their caregivers are not included in this study. However, considering the relatively low rate of physical and cognitive impairment evident in these patients, the high rate of caregiver psychological distress is not only a concern, but also indicates that it is not the severity of the patient's impairments that has the most detrimental effect on the psychological wellbeing of the caregiver. This finding is consistent with the model of caregiver stress discussed by Knight (1992).

Longitudinal comparisons of caregiver functioning indicated that between 3 and 12 months poststroke there was very little change in objective burden, subjective burden, overall social support, and confidant support. Very little change was evident in the level of caregiver satisfaction also, although generally these caregivers reported very little dissatisfaction at both 3 and 12 months poststroke. No change was evident in the average amount of social contact, group participation, or affective support. The

number of caregivers with clinically significant symptoms of psychological disorder decreased only slightly. However, a number of changes were evident in the relationships between the measures of caregiver functioning.

At 3 months poststroke, patient depression was related to caregiver symptoms of psychological disorder, subjective burden, less confidant support, and less affective support. Therefore it appears that caregivers of depressed stroke patients do not receive, or believe that they receive, adequate support. In turn, lack of both confidant and affective support were related to symptoms of psychological disorder, and subjective burden. This cluster of relationships indicates that caregivers of depressed patients report receiving inadequate support, which increases their own feelings of psychological distress. This finding is consistent with previous findings (e.g., Carnwath & Johnson, 1987; Tompkins et al., 1988).

The findings of the assessment at 3 months poststroke were also consistent with the model of caregiver stress discussed by Knight (1992), although caregiver coping skills (which form part of the second level of the model) were not assessed in the present study. Stressors related to symptoms of psychological disorder and subjective burden included caring for a depressed patient, caring for a patient who had experienced a previous stroke, and length of time in the caregiving role. However, stronger relationships were evident between these two measures of psychological distress and both confidant and affective support. This could indicate that support had more of an effect than the severity of the stressors on the psychological wellbeing of the caregiver. Alternatively, these results could indicate that caregivers who were distressed reported receiving less support. Regardless, these results are consistent with the model of caregiver stress as they indicate that the severity of the stressors do not

necessarily have the greatest impact on the psychological wellbeing of the caregiver.

It must be noted that these results deviated from the model of caregiving stress as caregiver satisfaction with the patient's level of social activity was not related to either the stressors or the psychological wellbeing of the caregiver. Also, while the frequency of social support and social contact were related to objective burden and patient physical impairment, these social support measures were not related to either of the measures of caregiver psychological distress. Further work is needed in this area to determine precisely what can be done to raise awareness of the need for adequate support for caregivers of stroke patients.

Patient depression was not related to caregiver symptoms of psychological disorder at 12 months poststroke. However, caregivers of depressed patients generally had less social contact with others, and received less confidant support. As mentioned by Tompkins and colleagues (1988), it is possible that these caregivers receive less support because they are distressed, rather than experiencing distress because they receive less support. Further work is needed to reach a better understanding of this relationship. Clearly the involvement of both the quality and quantity of social support in the psychological wellbeing of stroke caregivers continues at 12 months poststroke.

There were fewer relationships between stressors and caregiver psychological distress at 12 months poststroke than had been evident at 3 months poststroke. At 12 months poststroke, objective burden was the only stressor related to caregiver psychological disorder. Subjective burden at 12 months poststroke was not related to any of the stressors assessed in this study. However, both of the measures of caregiver psychological distress were related to confidant support. In addition, caregiver symptoms of psychological disorder were more likely to be experienced by caregivers

who reported receiving less overall social support, and less affective support. The relationship between both measures of caregiver psychological distress and confidant support, and the relationship between caregiver symptoms of psychological disorder and overall social support were stronger than the relationship between objective burden and caregiver symptoms of psychological disorder. However, the relationship between caregiver symptoms of psychological disorder and affective support was the weaker of these relationships. Therefore, it appears likely that the majority of forms of support the caregivers reported they received has had more of an effect than the severity of the stressors on the psychological wellbeing of the caregiver. However, this was not the case concerning affective support. That is, objective burden and affective support seem to have had a similar impact on caregiver psychological distress. The model of caregiver stress (Knight, 1992) suggests that, rather than caregiver stress being a direct response to the severity of stressors, individual differences in caregivers and caregiving situations determine the level of stress experienced by the caregiver. Therefore, these results do not deviate from this model, but rather indicate that, at 12 months poststroke, a stressor (objective burden) had a similar impact to a mediator (affective support), on the psychological wellbeing of the caregiver. However, the effect of the severity of objective burden on the caregiver was mediated by overall social support, and confidant support.

Apart from a small mention in the measure of objective burden, the financial burden of caregiving was not directly examined in this study. As it is likely that financial strain would increase the distress experienced by the caregiver, further research in this area should examine this issue.

In summary, psychological distress was evident in this group of caregivers.

Although there was a relationship between patient depression and caregiver symptoms of psychological disorder at 3 months poststroke, this relationship failed to reach significance at 12 months poststroke. The relationships found in this study at 3 months poststroke were consistent with the model of caregiver stress discussed by Knight (1992), as the relationships between support and caregiver psychological distress were stronger than that between the stressors and caregiver psychological distress. This indicates that it is not necessarily the severity of the patient's impairment and other stressors that has the greatest impact on the caregiver. The relationships evident at 12 months poststroke were also consistent with the model of caregiver stress, although these results differed from those reported at 3 months poststroke as the severity of one stressor (objective burden) did have a similar effect to affective support on the psychological wellbeing of the caregiver. However, the effects of this stressor were mediated by overall social support, and confidant support, which both had stronger relationships with caregiver symptoms of psychological disorder, in comparison with the relationship between objective burden and caregiver symptoms of psychological disorder.

Therefore, although there was very little significant change in the caregiver measures between 3 and 12 months poststroke, the relationships between variables did change. The extent of the patient's physical and cognitive impairment appears to have little association with caregiver psychological distress in this group of caregivers. This may be at least in part due to the relatively high level of functioning of the patients examined in this assessment. However, despite the low level of impairment in the patients, psychological distress is clearly evident in some caregivers.

CHAPTER 8

General Discussion

The objective of this study was to examine the prevalence of PSD, and the relationships between PSD and associated factors. This study assessed PSD during the first 12 months poststroke, in stroke patients aged 60 years and older. The patients were assessed at 1, 3, and 12 months poststroke to determine the prevalence of PSD at these stages of the poststroke period, and to examine the relationships between PSD and a number of factors. Previous research has indicated that changes occur throughout the poststroke period, both in the prevalence of PSD, and in the relationships between PSD and associated factors (e.g., Astrom, Adolfsson, et al., 1993). Changes may also occur in the degree of physical and cognitive impairment experienced by stroke patients (Desmond et al., 1996; Jorgensen et al., 1995a, 1995b). The results of the present study demonstrated changes in the relationships between PSD and associated factors; however, despite changes in the affective, physical, and cognitive functioning of individual patients, few other changes in the group as a whole were evident.

At each of the three stages of the poststroke period, approximately one third of the present group of stroke patients were depressed. However, individual longitudinal analysis determined that the composition of the depressed group changed. For example, patients who were depressed at 1 month poststroke were not necessarily depressed at 3 and/or 12 months poststroke. These results are consistent with the findings of Robinson et al. (1987), who also found that one third of their stroke patients were depressed at various stages during the first 2 years of the

poststroke period. Therefore, it is possible that the prevalence of PSD may not fluctuate to the extent that has been previously reported (e.g., Astrom, Adolfsson, et al., 1993). In addition, other studies have also shown that PSD may continue beyond 12 months poststroke (e.g., Astrom, Adolfsson, et al., 1993). Clearly PSD is a major concern in the first year following a stroke, and continuing analysis of these patients beyond 12 months poststroke would determine whether this prevalence rate persists further into the poststroke period.

This group of stroke patients, on average, demonstrated very little improvement in affective, physical, or cognitive functioning within the first 12 months poststroke. However, many individual patients demonstrated improvement in at least one area of functioning, although no patient demonstrated a clear pattern of recovery across all aspects of functioning. With regard to physical functioning, it is possible that little improvement was evident in basic ADL functioning as the majority of improvement may have occurred prior to the initial assessment at 1 month poststroke. If the patients had been assessed within a few days of their stroke, perhaps a greater range of improvement may have been apparent. Therefore, it is possible that the actual improvement in basic ADL functioning experienced by these stroke patients following their stroke was greater than that found in this study. In addition, there was only a slight increase in the number of extended activities performed by these patients within the first 12 months poststroke. This suggests that once the patients had settled into their daily poststroke routines, few patients increased the number of daily activities they performed. As there was no assessment of their prestroke involvement in such activities, it is not possible to make any assumptions about whether or not these patients returned to their prestroke level of physical functioning.

Within the first 12 months poststroke, this group of stroke patients demonstrated more improvement in cognitive functioning than was evident in physical functioning. However, general intelligence remained impaired, and the improvement in most other aspects of cognitive functioning was still unsubstantial. Simple attention abilities improved slightly, as did immediate verbal memory, and verbal fluency. The only aspects of cognitive functioning that improved substantially were delayed verbal memory, and basic cognitive functioning. Interestingly, complex attention abilities declined significantly. Reasons for the paucity of general improvement are not clear. Perhaps a more extensive assessment of the aspects of cognitive functioning examined in the present study would reveal an area of substantial improvement. However, this study assessed cognitive abilities using measures that were both brief and valid. Given the medical condition of many elderly stroke patients, a more extensive, lengthy assessment may not be appropriate.

The only factors predictive of PSD at 1 month poststroke were previous stroke and left hemisphere stroke. At 3 months poststroke, the hemispheric location of the lesion was no longer related to PSD, while the relationship continued between previous stroke and PSD. A predictive relationship between PSD and both physical impairment and one of the cognitive measures, simple attention, also emerged at 3 months poststroke. At 12 months poststroke the relationship between PSD and both previous stroke and physical impairment continued, although no other predictive relationships were evident.

The finding of a relationship between left hemisphere strokes and PSD at 1 month poststroke, but not at or beyond 3 months poststroke, is consistent with the finding by Astrom, Adolfsson, et al. (1993). This result indicates that PSD may have

organic origins in the acute poststroke stage, but that later in the poststroke period the depression occurs as a result of an interaction with other factors. Alternatively, the relationship between PSD and hemispheric location of lesion may become more specific beyond the acute poststroke stage; that is PSD may be more prevalent in patients with left anterior frontal strokes, as opposed to simply left hemisphere strokes. Unfortunately, the facilities and resources needed to accurately assess precise lesion location and lesion volume were not available for the present study. However, there was a significant relationship between PSD and physical and cognitive impairment at 3 months poststroke, but not at 1 month poststroke; this suggests that PSD is reactive in nature beyond the acute poststroke stage. This reactive nature of PSD may not be evident at the acute poststroke stage as the patient has yet to return home and/or is still receiving substantial assistance in their daily lives, and as a result is yet to realise the full extent of their impairments.

The present study found that patients with physical impairment were more likely than others to experience symptoms of PSD at 3 and 12 months poststroke. Other studies have also determined that there is a relationship between these factors, although at differing stages of the poststroke period. Astrom, Adolfsson, et al. (1993) found a relationship between PSD and physical impairment at 3 months poststroke, but not at 12 months poststroke. Parikh et al. (1987) determined that physical impairment was related to PSD at 12 months poststroke, although the relationship was not as strong as that found at 6 months poststroke. The findings of Astrom, Adolfsson, et al. and Parikh et al. suggest that the relationship between physical impairment and PSD may decrease with time. Hence, this relationship may not be evident in the present group of patients at 18 months poststroke. If PSD is a reaction

to physical impairment, the absence of this relationship at later stages of the poststroke period may be a result of the stroke patients having come to terms with their impairments, and/or having learnt to compensate for their disabilities. If this hypothesis is true, the results of this study suggest that these patients have not adjusted to their physical limitations by 12 months poststroke.

A weakening of the relationship between PSD and cognitive impairment was clearly evident between 3 and 12 months poststroke. At 3 months poststroke, PSD was related to only one aspect of cognitive functioning (simple attention abilities), and this study failed to find a significant predictive relationship between any aspect of cognitive functioning and PSD at 12 months poststroke. Other studies have also reported a weakening relationship between cognitive functioning and PSD throughout the poststroke period (Downhill & Robinson, 1994). Generally, the overall cognitive functioning of these patients did not improve substantially, therefore a decrease in the extent of the impairment cannot be responsible for the absence of this relationship at 12 months poststroke, as was hypothesised. As with physical functioning, if the depression is a reaction to the cognitive impairment, it is likely that the weakening of this relationship is due to the patients' learning to cope with, and perhaps compensate for, their decreased cognitive functioning.

The predictive relationship found in the present study between PSD and cognitive impairment at 3 months poststroke, differs from the findings of Bieliauskas (1993). While examining individuals with Parkinson's Disease, Bieliauskas found that symptoms of depression were more likely to be reported by cognitively-intact individuals. In the main introduction of this thesis it was hypothesised that this may have occurred as the cognitively-intact individuals with Parkinson's Disease were

more aware of their physical condition than those with cognitive impairment. However, the results of the present study indicate that the cognitively-intact stroke patients did not report more symptoms of depression than the patients with cognitive impairment during the first 12 months of the poststroke period.

This study found a relationship between PSD and previous stroke at each of the three stages of assessment. This finding is not altogether uncommon; Eastwood et al. (1989) also reported a relationship between these factors at 3 months poststroke, and Andersen et al (1995) found that previous stroke was predictive of PSD at 12 months poststroke. This relationship is probably best described as a reaction; a patient who has experienced a previous stroke is likely to be more aware of what may happen to them, and subsequently they may be aware that their chances of having a third (or fourth) stroke are greatly increased. Some researchers have suggested that patients with a history of stroke should be excluded from PSD research (Andersen et al., 1995). Reasons for excluding these patients are unclear, although Andersen et al. suggested that it may be the inclusion of these patients in studies that are the cause of discrepancies in reported results. However, excluding these patients may also lead to a sample unrepresentative of the true stroke population (Eastwood et al., 1989), as many patients experience more than one stroke. As has previously been discussed, discrepancies in the results reported in different studies are more likely to be due to differing diagnostic criteria, and the varying ages of the stroke patients involved in the studies. Therefore, there does not appear to be any good reason for studies of PSD to exclude patients who have had more than one stroke.

The ongoing relationship between PSD and previous stroke is a concern. As mentioned in Chapter 5, the duration of this relationship indicates that, regardless of

the rate and extent of the patient's recovery, depression is still a likely outcome if the patient has had a previous stroke. It was suggested that a third factor, such as the rate of recovery, is responsible for this relationship; that is, the patient who has had a previous stroke may have more impairment, or improve less, than the patient who has had their first stroke. However, this study found that patients who had experienced a previous stroke were not more impaired, either physically or cognitively, than patients for whom this was their first stroke. In addition, patients who had experienced a previous stroke did not demonstrate less improvement in physical and cognitive functioning. Examination of the relationship between PSD and previous stroke beyond 12 months poststroke may help to establish the duration, and lead to a better understanding, of this relationship.

None of the regression models indicated that any of the factors assessed in the present study were significantly predictive of psychological disorders, as measured by the GHQ-28. However, psychological disorder was present in this group of stroke patients, with 16% of the patients assessed at 3 months poststroke presenting with clinically significant symptoms of psychological disorder, although these symptoms were evident in only 4% of the patients assessed at 12 months poststroke. This change in the prevalence of clinically significant symptoms of psychological disorder was not significant, although there was a significant decrease in the average GHQ-28 scores. Therefore, regardless of the somewhat stable prevalence of depression (as measured by the GDS), clearly the prevalence of clinically significant symptoms of psychological disorder decreased by 12 months poststroke.

Previous research has indicated that, compared with nondepressed stroke patients, depressed patients recover less of their physical and cognitive functioning

(Astrom, Adolfsson, et al., 1993; Downhill & Robinson, 1994; Morris, Raphael, et al., 1992; Parikh et al., 1990). The results of the present study indicate that the depressed stroke patients were more physically impaired than the nondepressed patients at 1 month poststroke. However, by 3 and 12 months poststroke the physical functioning of the depressed patients improved, such that no significant difference was evident in the physical functioning of the depressed and non-depressed patients. No other differences in physical or cognitive impairment were evident, nor did the depressed stroke patients demonstrate less improvement than the patients with no clinically significant symptoms of depression. It is likely that the general lack of physical and cognitive improvement in the stroke patients contributes to this unexpected result.

In the present study, 20 patients had died by 12 months poststroke. Other researchers have reported similar mortality rates (e.g., Parikh et al., 1987). The present study failed to find a significant relationship between PSD at the acute poststroke stage, and mortality within the first 12 months poststroke. This finding is consistent with that of Astrom, Adolfsson, et al. (1993). However, other researchers have reported that depressed stroke patients were more likely than non-depressed patients to have died by 15 months poststroke (Morris, Robinson, & Samuels, 1993). Examining the present group of stroke patients further into the poststroke period may also result in a significant relationship between early PSD and eventual mortality. However, if this relationship is evident later in the poststroke period, mortality may be due to the natural aging process of the patients, rather than either PSD, or the stage of the poststroke period.

Previous research has also indicated that there is a relationship between

patient PSD and psychological distress in the caregiver (e.g., Wade et al., 1986). As PSD was evident in the present group of stroke patients, the caregivers of the patients who were not in residential care or hospital were assessed to determine the prevalence of caregiver psychological distress, and the relationship between this distress and patient depression. Other factors of patient and caregiver functioning were also assessed. Psychological distress was assessed in terms of psychological disorder, and subjective burden. The results of this study show that psychological distress was evident in this group of caregivers. Clinically significant symptoms of psychological disorder were reported by 25% of the caregivers at 3 months poststroke. A slight reduction in the prevalence of symptoms to 19% was evident at 12 months poststroke. These prevalence rates are alarmingly high considering that these caregivers were caring for patients with a relatively high level of physical and cognitive functioning, as the patients with lower levels of functioning were in residential care or hospital.

A number of factors were associated with caregiver psychological distress. Patient depression was related to caregiver symptoms of psychological disorder at 3 months poststroke, but not at 12 months poststroke. Other stressors related to caregiver symptoms of psychological disorder at 3 months poststroke were caring for a patient who had experienced a previous stroke, and increasing length of time in the caregiving role. Interestingly, very few of the stressors involved in the examination of caregiver functioning were related to caregiver psychological distress at 12 months poststroke, as objective burden was the only stressor related to caregiver psychological disorder at this stage of the poststroke period.

While the effects of the severity of the stressors on caregiver psychological

distress were mediated by confidant and affective support at 3 months poststroke, a different pattern of relationships was evident at 12 months poststroke. That is, while overall social support and confidant support mediated the effects of the severity of objective burden on caregiver psychological distress at 12 months poststroke, affective support did not appear to mediate these effects, as the relationship between objective burden and caregiver symptoms of psychological disorder was stronger than the relationship between affective support and caregiver symptoms of psychological disorder. This is consistent with the model of caregiver stress discussed by Knight (1992), in that caregiver stress is not necessarily related to the severity of the stressors (such as objective burden), but rather that individual differences in the lives of caregivers determine the distress felt by the caregiver. Therefore, these results clearly indicate that these individual differences (support) mediated the relationship between the stressors and caregiver distress at 3 months poststroke. However, although by 12 months poststroke fewer stressors were related to caregiver distress, the relationship between the remaining related stressor (objective burden) and caregiver distress was slightly stronger than that between one of the mediating variables (affective support) and caregiver distress. Further work is clearly needed to identify caregivers at risk of distress, and to provide the necessary assistance and support throughout the poststroke period to improve the psychological wellbeing of caregivers.

This study examined stroke patients of at least 60 years of age who were admitted to hospital subsequent to having a stroke. One major limitation of this study, and indeed of most studies, is that there is no way of determining the level of depression in the stroke patients who did not participate in this study. This includes

patients who chose not to participate, patients who were too impaired to participate, and patients who were not informed of the study as they were not admitted to hospital following their stroke. This is an important issue, as previous research has indicated that a higher rate of depression is found in stroke patients who are hospitalised, or in rehabilitation, compared with patients who do not receive such care (Johnson, 1991). It is entirely possible that the levels of depression in those patients who did not participate in the present study would average out to a similar level as that demonstrated by the participating stroke patients, particularly if depression is a reaction to the level of poststroke impairment. That is, if the patients who either chose not to participate or were too impaired to participate, have a relatively high level of depression, this may be counteracted by the potentially lower level of depression experienced by the patients who were only mildly affected and therefore not admitted to hospital following their stroke.

An additional limitation of the present study was that the present group of stroke patients were not assessed with regard to their prestroke level of depression. It is possible, and indeed likely, that the prestroke affective state of the patients has an impact on their poststroke condition. Unfortunately, there does not appear to be a practical and reliable way to assess prestroke affective state.

The methodological weaknesses of many studies of PSD have been outlined in a recent review by Spencer, Tompkins, and Schulz (1997). These authors discussed four areas of research methodology that require consideration in order to conduct a reliable study of PSD, namely: (1) define the population of interest, (2) screen participants for language and cognitive impairments occurring as a result of a neurological condition, (3) select suitable depression measures, and (4) determine the

presence of factors which may act as covariates in participants (e.g., psychiatric history).

These issues have been addressed in the present study, as the population of interest was clearly defined (stroke patients, 60 years and over, admitted to Waikato Hospital), patients were screened for conditions that may have affected assessment (aphasia), the assessment tools used were appropriate for the population being studied, and participants were excluded if they were currently receiving medication as treatment for depression, and if, within the 12 months prior to their stroke, they had been diagnosed with a psychological or psychiatric condition. Therefore, according to the guidelines outlined by Spencer et al. (1997), many of the methodological weaknesses often found in PSD research are not apparent in the present study.

Regardless of the methodological considerations taken into account in the present study, further research is needed in the area of poststroke affective disorders. Fortunately, more methodologically rigorous research into poststroke disorders continues.

Recent Developments

Since the commencement of this study, Gainotti and colleagues (1997) have developed the Post-stroke Depression Rating Scale (PSDS). The PSDS is a clinician rating scale, based on a range of symptoms often found in depressed stroke patients. These symptoms are divided into 10 sections: (1) depressed mood, (2) feelings of guilt, (3) thoughts of death and/or suicide, (4) vegetative disorders, (5) apathy and loss of interest, (6) anxiety, (7) catastrophic reactions, (8) hyper-emotionalism, (9) anhedonia, and (10) diurnal mood variations. Reliability and validity evaluations of

the PSDS determined that the PSDS has high interrater reliability on most sections (.80 - 1.00). However, the Diurnal mood variations and Hyper-emotionalism sections had less reliability (.60 - .80). Although a clear comparison was not possible between the PSDS and the Hamilton Depression Rating Scale, validity evaluations determined that the two scales were highly correlated. Although the PSDS is not for use specifically with the elderly population, the development of a scale specifically designed for screening for PSD is useful and promising.

Future Directions

Rehabilitation Outcome

The idea that PSD influences the effectiveness of rehabilitation has arisen due to the possible connection between depression and physical disability following stroke. In response to the ongoing debate as to whether depression causes physical impairment, or the converse, Gainotti et al. (1997) reported that PSD was likely to be a reaction to the poststroke impairments. Research also suggests that PSD prevents the rehabilitation procedures from having maximum beneficial effects on the functioning of the patient (Price, 1990).

It is also possible that the effects of rehabilitation differ for the elderly stroke patients due to their needs being different from those of a younger group (Gladman, Lincoln, & Barer, 1993). Therefore the reported age difference in the effectiveness of rehabilitation may not be due to depression, but due to the inability of rehabilitation systems to cater for the specific needs of the older stroke patient.

While examining the interaction of depression and rehabilitation outcome for 14 stroke patients and 17 amputee patients, Schubert, Burns, Paras, and Sioson

(1992) found that both groups of patients showed a decrease in depression (as assessed by the GDS) during the rehabilitation period. However, they suggested that the decrease in depression may not be only due to rehabilitation, as extraneous variables were present, such as the effect of brief interactions with the psychologist during the assessments of depression.

Therefore it is likely that there is a relationship of some form between PSD and rehabilitation outcome. In addition, research has suggested that there is a relationship between PSD and physical functioning at some stage of the poststroke period. Clearly further work is needed to determine whether rehabilitation is only affected by PSD at the time that the relationship between physical impairment and PSD is evident, or whether rehabilitation is affected by PSD regardless of physical impairment.

Treatment of PSD

Previous research has suggested that stroke patients experiencing depression tend to respond to antidepressant medication in a manner similar to that of depressed psychiatric patients (Andersen, Vestergaard, Riis, et al., 1994). However, stroke patients may be more susceptible to adverse side-effects (Andersen, Vestergaard, & Lauritzen, 1994).

Citalopram, a selective serotonin reuptake inhibitor, has been effective in decreasing PSD (Andersen, Vestergaard, & Lauritzen, 1994), and poststroke pathological crying (Andersen et al., 1993). Andersen, Vestergaard, and Lauritzen completed a double-blind trial on 66 stroke patients. No serious side-effects of citalopram were detected, although the drug was most effective in decreasing PSD in patients who became depressed no earlier than 7 weeks poststroke.

Fluoxetine, also a selective serotonin reuptake inhibitor, has been effective in treating clinical depression, although PSD was not specifically studied (Norman, Judd, & Burrows, 1992). Fluoxetine has also been effective in improving the emotional lability of 5 stroke patients, and 1 patient with traumatic brain injury (Sloan, Brown, & Pentland, 1992). As these patients were diagnosed with emotional lability, but not clinical depression, it is not clear how effectively fluoxetine would treat PSD. However, selective serotonin reuptake inhibitors are generally considered to be a good treatment for PSD, as are drugs such as methylphenidate (Sandin et al., 1994).

Methylphenidate, a psychostimulant, and nortriptyline, a tricyclic antidepressant, were compared with regard to their respective effectiveness and accompanying side-effects in treating PSD. The researchers (Lazarus, Moberg, Langsley, & Lingam, 1994), found that there was no difference in the responding rates between the two groups, but patients in the methylphenidate group ($N = 28$) responded more quickly (2.4 days) than the 30 patients who were given nortriptyline (27 days). Only mild side-effects were detected, although tricyclic antidepressants (such as nortriptyline) have many side-effects to which elderly people are often more susceptible (Sloan et al., 1992).

Psychologically-based treatments for PSD include rehabilitative techniques, such as behaviour modification and therapy, along with social support. Negative reinforcement is discouraged as it has proven to be ineffective (Sandin et al., 1994). It is unfortunate that, given the questionable efficacy and obvious negative side-effects of pharmacological treatments for PSD, so little research has been undertaken on the psychological treatment of PSD (Antonuccio, Danton, & DeNelsky, 1995; Jacobson

& Hollon, 1996; Larner & Leeming, 1984).

As the frequency and efficacy of the patients' social support were not assessed in the present study, it is not clear whether sufficient social support was a variable affecting the presence of PSD in this group of stroke patients. Regardless, very few of the original group of 103 stroke patients were receiving medication as treatment for PSD. It is possible that symptoms of depression were not clearly evident in the majority of patients, and as a result they did not receive medical treatment. In addition, many of the patients developed symptoms of depression after discharge from hospital. These patients may not have received medical treatment for their depressive symptoms as the depression had not been recognised by their general medical practitioner. Clearly further work is needed in raising awareness of PSD, and in developing an acceptable and effective form of treatment.

Conclusions

In summary, this study found that approximately one third of the stroke patients were depressed at 1 month poststroke. Similar prevalence rates of PSD were found at 3 and 12 months poststroke; however, the composition of the depressed group changed. There was a significant decrease in the average GHQ-28 scores between 3 and 12 months poststroke, although the change in the prevalence of clinically significant symptoms of psychological disorder was not significant. In addition, analysis of the level of functioning of these patients indicated that, although individual patients demonstrated improvement in their level of physical and/or cognitive functioning, the functioning of the group as a whole did not change substantially. Significant improvement was evident in basic cognitive functioning, and

delayed verbal memory, while complex attention abilities deteriorated significantly.

Relationships between PSD and associated factors changed within the first 12 months poststroke, although PSD was related to previous stroke at each of the three assessments. A predictive relationship was evident between PSD and left hemisphere stroke at 1 month poststroke, but this relationship was not evident at or beyond 3 months poststroke. Physical impairment was predictive of depression at 3 and 12 months poststroke. However, cognitive impairment was a predictor of depression at 3 months poststroke only.

Comparison of the depressed and the non-depressed stroke patients indicated that the depressed patients had more physical impairment at 1 month poststroke. However, no other differences in physical or cognitive impairment were evident. Similarly, examination of the relationship between PSD and recovery indicated that, compared with nondepressed stroke patients, depressed patients did not demonstrate less recovery within the first 12 months poststroke. In addition, there was no significant relationship between patient depression at 1 month poststroke, and mortality within the first 12 months poststroke. Despite the absence of these relationships, the high prevalence rate of depression amongst these stroke patients is alarming.

Assessment of the functioning of caregivers of the stroke patients who were living at home indicated that patient depression was related to caregiver psychological distress at 3 months poststroke, but not at 12 months poststroke. Regardless of the absence of this relationship at 12 months poststroke, caregiver psychological distress was still evident at this stage of the poststroke period as psychological disorder was present in 19% of the caregivers. However, this was a decrease from 25% at 3

months poststroke. Further examination of these caregivers beyond 12 months poststroke may indicate a further decrease in this prevalence of psychological disorder; however, it is likely that some evidence of caregiver psychological distress would remain.

Clearly further, methodologically rigorous research is needed to assess PSD and the functioning of stroke patients beyond 12 months poststroke. In addition, further work is needed in the rehabilitation of stroke patients, and in recognising the need for treatment to overcome depression in the patients, and psychological distress in their caregivers.

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APPENDICES

Appendix A

3.3 CONSENT FORM: PART 3

RESEARCHER'S COPY: **CONSENT**

Name of Research Project: Recovery following stroke

Name of Principal Researcher: Shirley Hosking

I have received a copy of the information sheet. The researcher has explained it to me and I have had a chance to discuss it with other people. Any questions have been answered to my satisfaction. I have read and understood all the information, including the information about compensation should injury be caused to me as a result of participating in this research project.

I agree to participate in this research project. I understand that I may withdraw at any time.

Signature: _____

Printed Name: _____

Date: _____

..... Detach .....

PARTICIPANT'S COPY: **CONSENT**

Name of Research Project: Recovery following stroke

Name of Principal Researcher: Shirley Hosking

I have received a copy of the information sheet. The researcher has explained it to me and I have had a chance to discuss it with other people. Any questions have been answered to my satisfaction. I have read and understood all the information, including the information about compensation should injury be caused to me as a result of participating in this research project.

I agree to participate in this research project. I understand that I may withdraw at any time.

Signature: _____

Printed Name: _____

Appendix B

STROKE RESEARCH GROUP
(University of Waikato & Waikato Hospital)

Recovery Following Stroke

Demographic Information - *Patient*

Participant ID : _____

Name : _____

Address : _____

Telephone : _____

Age (at time of Stroke) : _____

Date of Birth : _____

Gender : 1. Female 2. Male ( )

Dominant Hand : 1. Right 2. Left ( )

Date of Stroke : _____

Previous Strokes : 1. None 2. One ( )

3. Other - Specify : _____

Time since first stroke : _____

Time since last stroke : _____

Strokes occurring during study (excl only suspected) ( )

1. None 2. One

3. Other - Specify : _____

Dates of additional strokes : _____

Date of discharge : _____

Date (Discharge Assessment) : _____

Date (3 month Follow-up) : _____

Date (12 month Follow-up) : _____

Lesion Hemisphere : 1. Right 2. Left ( )

Lesion Location : ( )

1. Frontal
2. Parietal
3. Occipital
4. Temporal
5. Combination - Specify :_____

Marital Status : ( )

1. Never Married
2. Married/De facto
3. Separated/Divorced
4. Widowed
5. Other - Specify : _____

Ethnicity : ( )

1. NZ European/Pakeha
2. NZ Maori
3. Pacific Islander
4. Other - Specify : _____

Education :

Years of secondary education : _____

Years of tertiary education : _____

Age when left school : _____

Years of education : _____

Occupational Status : ( )

1. Retired
2. Unemployed
3. Homemaker
4. Sickness Beneficiary
5. Student
6. Paid employment - Specify : _____

Living Arrangements : ( )

(A)
1. Alone at own home
2. At immediate family's home
3. At relations' home
4. With partner at home
5. With partner &/or children at home
6. Friend(s)/Flatmate(s) at home
7. With caregiver at home
8. Resthome
9. Hospital
10. Other - Specify : _____

(B) Number of other people at residence _____

Number of dependents _____

Daily Life Functioning Prior to Stroke: ( )

1. Fully self-sufficient
2. Only needed assistance with some more manual duties (eg:vacuuming)
3. Needed assistance with some basic care activities
4. Needed full-time care
5. Other - Specify : _____

Medical Status Prior to Stroke : ( )

1. Physically fit
2. Needed walking aids only
3. Disabling arthritis or other injury
4. Diabetes
5. Emphysema
6. Heart disease
7. Other - Specify : _____

Previous Psychiatric History : ( )

1. None
2. Family history
3. Previous psychiatric admittance
4. Previous psychiatric medication
5. Previous psychological counselling

Diagnosis : _____

Time since last psychiatric discharge/medication: _____

Time since last psychological counselling : _____

Current Psychiatric Treatment : ( )

1. None
2. Current psychiatric medication
3. Current psychological counselling

Diagnosis : _____

Treatment in relation to depression : ( )

1. None
2. Anti-depressants for depression
3. Counselling for depression
4. Anti-depressants and counselling for depression
5. Anti-depressants for sleeping
6. Other medication which may affect depression
7. Other - Specify : _____

Name of anti-depressant prescribed : _____

Current Medication : ( )

1. No
2. Yes (Type : _____)

Previous Neurological Complaints : ( )

(Prior to your stroke, have you frequently suffered from headaches, dizziness, fainting spells, or insomnia?)

1. No
2. Yes (How often? _____)

Caregiver Status : ( )

1. No caregiver
2. Caregiver participating in study
3. Caregiver not participating in study -

(fill in details below)

Details if non-participating caregiver :

Caregiver details at 3 months poststroke :

(A) 1. Full-time caregiver - Live-in 2. Full-time caregiver - Live away ( )

3. Part-time caregiver 4. No caregiver

Name of Caregiver : _____

(B) Relationship of caregiver to patient : ( )

1. Spouse/Partner 2. Son/Daughter

3. Grandchild 4. Brother/Sister

5. Other family member 6. Friend

7. Paid employee 8. Volunteer

9. Other - Specify : _____

Caregiver details at 12 months poststroke :

(A) 1. Same caregiver as above ( )

2. Change in caregiver situation -

(fill in details below)

(B) 1. Full-time caregiver - Live-in ( )

2. Full-time caregiver - Live away

3. Part-time caregiver 4. No caregiver

Name of Caregiver : _____

(C) Relationship of caregiver to patient : ( )

1. Spouse/Partner 2. Son/Daughter

3. Grandchild 4. Brother/Sister

5. Other family member 6. Friend

7. Paid employee 8. Volunteer

9. Other - Specify : _____

Appendix C

The following measures of the patients functioning are not included in this appendix due to copyright regulations:

1. Cognitive Assessment Scale (CAS) of the Clifton Assessment Procedures for the Elderly (CAPE; Pattie & Gilleard, 1979).
2. Minnesota Differential Diagnosis of Aphasia Test - short form (Powell et al., 1980).
3. Verbal Paired Associates (VPA) subtest of the Wechsler Memory Scale-Revised (Wechsler, 1987).
4. 28-item version of the General Health Questionnaire (GHQ-28; Goldberg, 1972).
5. Vocabulary, Block Design, Digit Span, and Digit Symbol subtests of the Wechsler Adult Intelligence Scale-Revised (Wechsler, 1981).
6. National Adult Reading Test (NART; Nelson, 1982).

Therefore, this appendix includes the following measures (title on measure is presented here in parenthesis):

1. Barthel ADL Index (Barthel Scale).
2. Geriatric Depression Scale (GDS).
3. Controlled Oral Word Association Test (COWA scale).
4. Nottingham Extended ADL Index (ADL scale).

STROKE RESEARCH GROUP
(University of Waikato & Waikato Hospital)

Recovery Following Stroke

Barthel Scale

Participant ID :_____

Bowels	0 = incontinent 1 = occasional accident 2 = continent	( )
Bladder	0 = incontinent / catheterised & unable to manage 1 = occasional accident 2 = continent	( )
Grooming	0 = needs help 1 = independent for face/hair/teeth/shaving	( )
Toilet use	0 = dependent 1 = needs some help 2 = independent	( )
Feeding	0 = dependent 1 = needs help eg: cutting, spreading butter 2 = independent in all actions	( )
Transfer (bed-chair)	0 = unable 1 = major help, can sit 2 = minor help (verbal or physical) 3 = independent	( )
Walking	0 = unable 1 = independent in wheelchair 2 = walks with help of person (verbal/physical) 3 = independent (may use aid)	( )
Dressing	0 = dependent 1 = needs help, but does half 2 = independent (including buttons/zips/laces)	( )
Stairs	0 = unable 1 = needs help (verbal/physical) 2 = independent	( )
Bathing	0 = dependent 1 = independent	( )

STROKE RESEARCH GROUP
(University of Waikato & Waikato Hospital)

Recovery Following Stroke

GDS

Participant ID : _____

1. Are you basically satisfied with your life?.....yes / no
2. Have you dropped many of your activities and interests?.....yes / no
3. Do you feel that your life is empty?.....yes / no
4. Do you often get bored?.....yes / no
5. Are you hopeful about the future?.....yes / no
6. Are you bothered by thoughts you can't get out of your head?.....yes / no
7. Are you in good spirits most of the time?.....yes / no
8. Are you afraid that something bad is going to happen to you?.....yes / no
9. Do you feel happy most of the time?.....yes / no
10. Do you often feel helpless?.....yes / no
11. Do you often get restless and fidgety?.....yes / no
12. Do you prefer to stay at home, rather than going out and doing new things?.....yes / no
13. Do you frequently worry about the future?.....yes / no
14. Do you feel you have more problems with memory than most?.....yes / no
15. Do you think it is wonderful to be alive now?.....yes / no
16. Do you often feel downhearted and blue?.....yes / no
17. Do you feel pretty worthless the way you are now?.....yes / no
18. Do you worry a lot about the past?.....yes / no
19. Do you find life very exciting?.....yes / no
20. Is it hard for you to get started on new projects?.....yes / no
21. Do you feel full of energy?.....yes / no
22. Do you feel that your situation is hopeless?.....yes / no
23. Do you think that most people are better off than you are?.....yes / no
24. Do you frequently get upset over little things?.....yes / no
25. Do you frequently feel like crying?.....yes / no
26. Do you have trouble concentrating?.....yes / no
27. Do you enjoy getting up in the morning?.....yes / no
28. Do you prefer to avoid social gatherings?.....yes / no
29. Is it easy for you to make decisions?.....yes / no
30. Is your mind as clear as it used to be?.....yes / no

STROKE RESEARCH GROUP
(University of Waikato & Waikato Hospital)

Recovery Following Stroke

COWA Scale

Participant ID : _____

F **A** **S**

STROKE RESEARCH GROUP
(University of Waikato & Waikato Hospital)

Recovery Following Stroke

ADL Scale

Participant ID : _____

	on my own	on my own with difficulty	with help	no
Mobility.				
1. Do you walk around outside?	[ ]	[ ]	[ ]	[ ]
2. Do you climb stairs?	[ ]	[ ]	[ ]	[ ]
3. Do you get in and out of the car?	[ ]	[ ]	[ ]	[ ]
4. Do you walk over uneven ground?	[ ]	[ ]	[ ]	[ ]
5. Do you cross roads?	[ ]	[ ]	[ ]	[ ]
6. Do you travel on public transport?	[ ]	[ ]	[ ]	[ ]
In the Kitchen.				
1. Do you manage to feed yourself?	[ ]	[ ]	[ ]	[ ]
2. Do you manage to make yourself a hot drink?	[ ]	[ ]	[ ]	[ ]
3. Do you take hot drinks from one room to another?	[ ]	[ ]	[ ]	[ ]
4. Do you do the washing up?	[ ]	[ ]	[ ]	[ ]
5. Do you make yourself a hot snack?	[ ]	[ ]	[ ]	[ ]
Domestic Tasks.				
1. Do you manage your own money when you are out?	[ ]	[ ]	[ ]	[ ]
2. Do you wash small items of clothing?	[ ]	[ ]	[ ]	[ ]
3. Do you do your own housework?	[ ]	[ ]	[ ]	[ ]
4. Do you do your own shopping?	[ ]	[ ]	[ ]	[ ]
5. Do you do a full clothes wash?	[ ]	[ ]	[ ]	[ ]
Leisure Activities.				
1. Do you read newspapers or books?	[ ]	[ ]	[ ]	[ ]
2. Do you use the telephone?	[ ]	[ ]	[ ]	[ ]
3. Do you write letters?	[ ]	[ ]	[ ]	[ ]
4. Do you go out socially?	[ ]	[ ]	[ ]	[ ]
5. Do you manage your own garden?	[ ]	[ ]	[ ]	[ ]
6. Do you drive a car?	[ ]	[ ]	[ ]	[ ]

Appendix D

STROKE RESEARCH GROUP
(University of Waikato & Waikato Hospital)

Recovery Following Stroke

Demographic Information - *Caregiver*

Participant ID : _____

Name : _____

Address : _____

Telephone : _____

Age : _____

Date of Birth : _____

Gender : 1. Female 2. Male ( )

Length of time as caregiver (for this patient): _____

Date (3 month Follow-up) : _____

Date (12 month Follow-up) : _____

Caregiver Status :

(A) 1. Full-time caregiver - Live-in 2. Full-time caregiver - Live away ( )
3. Part-time caregiver
Name of Patient : _____

(B) Number of other people at residence _____
Number of other dependents _____

(C) Relationship of caregiver to patient : ( )
1. Spouse/Partner 2. Son/Daughter
3. Grandchild 4. Brother/Sister
5. Other family member 6. Friend
7. Paid employee 8. Volunteer
9. Other - Specify : _____

Marital Status : ( )

1. Never Married
2. Married/De facto
3. Separated/Divorced
4. Widowed
5. Other - Specify : _____

Ethnicity : ( )

1. NZ European/Pakeha
2. NZ Maori
3. Pacific Islander
4. Other - Specify : _____

Medical Conditions : ( )

1. Physically fit
2. Mobility : Needs some assistance
3. Disabling arthritis or other injury
4. Diabetes
5. Emphysema
6. Heart disease
7. Other - Specify : _____

Appendix E

The 28-item version of the General Health Questionnaire (GHQ-28; Goldberg, 1972) is not included in the appendix of caregiver measures due to copyright regulations.

Therefore, this appendix includes the following measures of caregiver functioning (title on measure is presented here in parenthesis):

1. Katz Adjustment Scale-Relatives Forms (KAS-R).
2. Measure of Objective and Subjective Burden (Caregiver Inventory [Part A and Part B]).
3. Social Activities Questionnaire (Social Questionnaire).
4. Duke-UNC Functional Social Support Questionnaire (FSS).

STROKE RESEARCH GROUP
(University of Waikato & Waikato Hospital)

Recovery following stroke

KAS - R

Subject ID:
Date:

R2

Instructions: *People differ in what they are able to do. We would like you to go through this list and tell us which of these the person with the stroke is doing, or has done in the past few weeks.*

For example, if they are not helping with household chores you would place a check in column (1). If they help some, then you would check column (2). If they are doing this regularly, then place a check in column (3).

	is not doing	is doing some	is doing regularly	does not apply
1. Helps with household chores	☐	☐	☐	☐
2. Visits his/her friends	☐	☐	☐	☐
3. Visits his/her relatives	☐	☐	☐	☐
4. Entertains friends at home	☐	☐	☐	☐
5. Dresses and takes care of themself	☐	☐	☐	☐
6. Helps with the family budgeting	☐	☐	☐	☐
7. Remembers to do important things on time	☐	☐	☐	☐

	is not doing	is doing some	is doing regularly	does not apply
8. Gets along with family members	☐	☐	☐	☐
9. Goes to parties and other social activities	☐	☐	☐	☐
10. Gets along with neighbours	☐	☐	☐	☐
11. Helps with family shopping	☐	☐	☐	☐
12. Helps in the care and training of children	☐	☐	☐	☐
13. Goes to church	☐	☐	☐	☐
14. Takes up hobbies	☐	☐	☐	☐
15. Works	☐	☐	☐	☐
16. Supports the family	☐	☐	☐	☐

R3

Instructions: *Families differ in what they think their relatives should do. We want you to tell us which of these things you would have expected the person with the stroke to be doing, if they had not had the stroke.*

For, example, if you would have expected them to be regularly helping with household chores, then place a check in column (3). If you would not have expected them to be doing any of this, place a check in column (1).

	did not expect them to be doing	expected them to be doing some	expected them to be doing regularly	does not apply
1. Helps with household chores	☐	☐	☐	☐
2. Visits his/her friends	☐	☐	☐	☐
3. Visits his/her relatives	☐	☐	☐	☐
4. Entertains friends at home	☐	☐	☐	☐
5. Dresses and takes care of themself	☐	☐	☐	☐
6. Helps with the family budgeting	☐	☐	☐	☐
7. Remembers to do important things on time	☐	☐	☐	☐
8. Gets along with family members	☐	☐	☐	☐
9. Goes to parties and other social activities	☐	☐	☐	☐
10. Gets along with neighbours	☐	☐	☐	☐
11. Helps with family shopping	☐	☐	☐	☐

	did not expect them to be doing	expected them to be doing some	expected them to be doing regularly	does not apply
12. Helps in the care and training of children	☐	☐	☐	☐
13. Goes to church	☐	☐	☐	☐
14. Takes up hobbies	☐	☐	☐	☐
15. Works	☐	☐	☐	☐
16. Supports the family	☐	☐	☐	☐

STROKE RESEARCH GROUP
(University of Waikato & Waikato Hospital)

Recovery Following Stroke

Caregiver Inventory

Participant ID : _____

Instructions : Caregiving can have a variety of effects on your life. Please circle the option you feel best describes your situation. Please circle only one option for each situation.

Part A.

1. Amount of time you have to yourself.

   A lot more a little more the same a little less a lot less

2. Amount of privacy you have.

   A lot more a little more the same a little less a lot less

3. Amount of money you have available to meet expenses.

   A lot more a little more the same a little less a lot less

4. Amount of personal freedom you have.

   A lot more a little more the same a little less a lot less

5. Amount of energy you have.

   A lot more a little more the same a little less a lot less

6. Amount of time you spend in recreational and/or social activities.

   A lot more a little more the same a little less a lot less

7. Amount of vacation activities and trips you have.

   A lot more a little more the same a little less a lot less

8. Your relationships with other family members.

A lot better	a little better	the same	a little worse	a lot worse

9. Your health.

A lot better	a little better	the same	a little worse	a lot worse

Part B.

1. I feel it is painful to watch my (relative) age.

rarely or never	a little of the time	sometimes	often	most of the time

2. I feel useful in my relationship with my (relative).

rarely or never	a little of the time	sometimes	often	most of the time

3. I feel afraid for what the future holds for my (relative).

rarely or never	a little of the time	sometimes	often	most of the time

4. I feel strained in my relationship with my (relative).

rarely or never	a little of the time	sometimes	often	most of the time

5. I feel that I am contributing to the well-being of my (relative).

rarely or never	a little of the time	sometimes	often	most of the time

6. I feel that my (relative) tries to manipulate me.

rarely or never	a little of the time	sometimes	often	most of the time

7. I feel pleased with my relationship with my (relative).

rarely or never	a little of the time	sometimes	often	most of the time

8. I feel that my (relative) doesn't appreciate what I do for him/her as I would like.

rarely or never	a little of the time	sometimes	often	most of the time

9. I feel nervous and depressed about my relationship with my (relative).

rarely or never	a little of the time	sometimes	often	most of the time

10. I feel that my (relative) makes requests which are over and above what he/she needs.

rarely or never	a little of the time	sometimes	often	most of the time

11. I feel that I don't do as much for my (relative) as I could or should.

rarely or never	a little of the time	sometimes	often	most of the time

12. I feel that my (relative) seems to expect me to take care of him/her as if I were the only one she/he could depend on.

rarely or never	a little of the time	sometimes	often	most of the time

13. I feel guilty over my relationship with my (relative).

rarely or never	a little of the time	sometimes	often	most of the time

STROKE RESEARCH GROUP
(University of Waikato & Waikato Hospital)

Recovery Following Stroke

Social Questionnaire

Participant ID : _____

1. About how many families in your neighbourhood are you well enough acquainted with, that you visit each other in your homes?
_____ families.

2. About how many *close* friends do you have - people you feel at ease with and can talk with about what is on your mind? You may include relatives.
_____ close friends.

3. Over a year's time, about how often do you get together with friends or relatives, like going out together or visiting in each other's homes? (Tick the appropriate box).

Every day	[ ]
Several days a week	[ ]
About once a week	[ ]
2 or 3 times a month	[ ]
About once a month	[ ]
5 to 10 times a year	[ ]
Less than 5 times a year	[ ]

4. During the *past month,* about how often have you had friends over to your home? Do *not* count relatives.

Every day	[ ]
Several days a week	[ ]
About once a week	[ ]
2 or 3 times in past month	[ ]
Once in past month	[ ]
Not at all in past month	[ ]

5. About how often have you visited with friends at *their* homes during the *past month?* Do not count relatives.

Every day	[ ]
Several days a week	[ ]
About once a week	[ ]
2 or 3 times in past month	[ ]
Once in past month	[ ]
Not at all in past month	[ ]

PLEASE TURN OVER

6. About how often were you on the telephone with close friends or relatives during the *past month?*

Every day	[ ]
Several times a week	[ ]
About once a week	[ ]
2 or 3 times	[ ]
Once	[ ]
Not at all	[ ]

7. In general, how well are you getting along with other people these days - would you say better than usual, about the same, or not as well as usual?

Better than usual	[ ]
About the same	[ ]
Not as well as usual	[ ]

8. How often have you attended a religious service during the *past month?*

Every day	[ ]
More than once a week	[ ]
Once a week	[ ]
2 or 3 times in past month	[ ]
Once in past month	[ ]
Not at all in past month	[ ]

9. About how many voluntary groups or organisations do you belong to - like church groups, clubs or lodges, parent groups, etc. ("Voluntary" refers to "because you want to".)

_____ groups or organisations.

10. How active are you in the affairs of these groups or clubs you belong to? (If you belong to a great many, just count those you feel closest to.)

Very active, attend most meetings	[ ]
Fairly active, attend fairly often	[ ]
Not active, belong but hardly ever go	[ ]
Do not belong to any groups or clubs	[ ]

STROKE RESEARCH GROUP
(University of Waikato & Waikato Hospital)

Recovery Following Stroke

FSS

Participant ID : _____

Instructions : Here is a list of some things that other people do for us or give us that may be helpful or supportive. Please read each statement carefully and circle the number that is closest to your situation.

Here is an example: I get...	As much as I would like				Much less than I would like
enough vacation time	1	2	3	4	5

If you put a circle where we have, it means that you get almost as much vacation time as you would like, but not quite as much as you would like.

Answer each item as best you can. There are no right or wrong answers.

I get ...	As much as I would like				Much less than I would like
1. invitations to go out and do things with other people	1	2	3	4	5
2. love and affection	1	2	3	4	5
3. chances to talk to someone about my problems at work or with my housework	1	2	3	4	5
4. chances to talk to someone I trust about my personal and family problems	1	2	3	4	5
5. chances to talk about money matters	1	2	3	4	5
6. people who care what happens to me	1	2	3	4	5
7. useful advice about important things in life	1	2	3	4	5
8. help when I'm sick in bed	1	2	3	4	5

Appendix F

WAIKATO ETHICS COMMITTEE

8 September 1995

Shirley G Hosking
107 Tramway Road
HAMILTON

Dear Ms Hosking

PREVALENCE AND ASSOCIATED FACTORS OF POSTSTROKE DEPRESSION (No. 027/95:269)

Thank you for your letter of 4 September 1995 and for the revised Information Sheet. You have addressed the Committee's concerns and may now proceed with your study.

The Committee would like to view copies of correspondence with Age Concern when you are in a position to start collecting control group data which you anticipate being about mid 1997.

Please note that any proposed amendments to protocol must be submitted for ethical review and a final report on completion of the research is required.

Best wishes for the success of your study.

Yours sincerely

Rosemary J De Luca
Chairperson

P O Box 322 HAMILTON Telephone: 07 846 1539 Fax: 07 846 1496

Appendix G

INFORMATION SHEET: PART 1

Title of project: Recovery following stroke.

Principal Investigator : Shirley Hosking BSocSc (Hons)
Post-graduate student
Department of Psychology
University of Waikato

Telephone : (07) 855 3438

Co-investigators: Dr Nigel V. Marsh (University of Waikato)
Dr Paul J. Friedman (Waikato Hospital)

This study is concerned with finding out about any difficulties that people have in the first year following stroke. We are also interested in finding out about any problems that may occur for other members of the family.

The reason for this research is so that we can get a better idea of any problems stroke patients and their families may have once they leave hospital. In this way we may be better able to assist future stroke patients and their families.

If you agree to take part in this study the person who has had the stroke and another person who knows them well (and preferably lives with them) will be seen on three occasions. The first time will be just before the person with the stroke is discharged from Hospital, then at 3 months and 12 months following the stroke. The second and third time you are seen can be at the Hospital, the University of Waikato, or your own home, depending on which is most convenient for you.

These visits will last about 2 hours. During these visits the person who has had the stroke will be asked a number of questions about how they are spending their time and will be asked to complete a number of puzzle/game-type assessments. Most people find these fun and stimulating. The person who has not had the stroke will be asked a number of questions about how they see the stroke person as progressing and about any effects the stroke has had on their life. The information we receive during these visits will remain completely confidential, and you will not be identified in any future use of this information.

Dr Friedman will be checking the medical records to ensure that we have relevant information about the stroke. All information from your medical records will remain strictly confidential.

INFORMATION SHEET: PART 2

You have been asked to take part in a research project which has been reviewed and approved by the Waikato Ethics Committee.

It is important that you know exactly what your participation in this project means. Therefore, if you are unsure about any part of this research or about what it means for you, please ask the person explaining it to you, or the person in charge of the research project, about it. You may wish to discuss it with you own doctor, a family member, or friend. Before you sign the consent form, the person who gave it to you will ask you to tell him or her what you understand your involvement in the research project to be, and how it will affect you.

DO NOT sign the consent form until you understand all the information given.

If you do not want to take part in this research project, or if you do sign the form but then change your mind and do not want to continue at any stage, tell the person in charge of the project. In either case, you will still be given the best possible health treatment.

The Health Consumer Trust (Telephone: 07 846 1991) is available to all patients in the Midland Regional Health area. Any patient in a research project who has a concern about treatment may contact one of the people listed in the Health Consumer Service Pamphlet.

Appendix H

Clinically Significant Changes in Patients' Functioning

Physical functioning: Barthel ADL Index. The level of physical functioning of 2 patients improved progressively during the first 12 months poststroke, with 1 patient improving from severe impairment at 1 month poststroke, to moderate impairment at 3 months poststroke, to mild impairment at 12 months poststroke. One patient improved from moderate impairment at 1 month poststroke, to mild impairment at 3 months poststroke, to independence of physical functioning at 12 months poststroke.

Ten patients improved their level of physical functioning between 1 and 3 months poststroke only; 1 patient improved from very severe to severe impairment, 2 patients improved from moderate to mild impairment, 1 patient improved from moderate impairment to independence of physical functioning, and 6 patients improved from mild impairment to independence of physical functioning.

Seven patients experienced clinically significant improvement in physical functioning between 3 and 12 months poststroke only; 1 patient improved from severe to moderate impairment, 3 patients improved from moderate to mild impairment, and 3 patients improved from mild impairment to independence of physical functioning.

Nine patients experienced clinically significant deterioration of physical functioning between 1 and 3 months poststroke; 5 patients deteriorated from independent physical functioning to mild impairment, 3 patients deteriorated from mild to moderate impairment, and 1 patient deteriorated from moderate to severe impairment of physical functioning.

Seven patients experienced clinically significant deterioration of physical functioning between 3 and 12 months poststroke; 4 patients deteriorated from independent physical functioning to mild impairment, 2 patients deteriorated from mild to severe impairment, and 1 patient deteriorated from severe to very severe physical impairment.

Affective functioning: GDS. Eight patients experienced decreasing symptoms of depression between 1 and 3 months poststroke; 7 patients who reported symptoms of mild depression at 1 month poststroke reported no clinically significant symptoms of depression at 3 months poststroke, and 1 patient who reported symptoms of severe depression at 1 month poststroke reported symptoms of mild depression at 3 months poststroke.

The depression status of 10 patients improved between 3 and 12 months poststroke; 8 patients who reported symptoms of mild depression at 3 months poststroke reported no clinically significant symptoms of depression at 12 months poststroke, 1 patient with symptoms of severe depression at 3 months poststroke reported no clinically significant symptoms of depression at 12 months poststroke, and 1 additional patient with symptoms of severe depression at 3 months poststroke reported symptoms of mild depression at 12 months poststroke.

One patient reported depressive symptoms of increasing severity through all three assessments, from no clinically significant symptoms of depression at 1 month poststroke, to symptoms of mild depression at 3 months, to symptoms of severe depression at 12 months poststroke.

Nine patients experienced clinically significant increases in the level of depression between 1 and 3 months poststroke only; 7 patients with no clinically

significant symptoms of depression at 1 month poststroke reported symptoms of mild depression at 3 months poststroke, and 1 patient who was not depressed at 1 month poststroke reported symptoms of severe depression at 3 months poststroke. In addition, the level of depression for 1 patient increased from mild at 1 month poststroke to severe at 3 months poststroke.

Eight patients experienced clinically significant increases in symptoms of depression between 3 and 12 months poststroke only; 6 patients who were not depressed at 3 months poststroke reported symptoms of mild depression at 12 months poststroke, and 2 patients with mild depression at 3 months poststroke were severely depressed at 12 months poststroke.

Cognitive functioning: Basic cognitive functioning. Twelve patients experienced clinically significant improvement of basic cognitive functioning between 1 and 3 months poststroke; 1 patient improved from marked to moderate impairment, 1 patient improved from marked to mild impairment, 1 patient improved from moderate to mild impairment, 1 patient improved from moderate to no impairment, and 8 patients improved from mild to no impairment of basic cognitive functioning.

Seven patients improved between 3 and 12 months poststroke; 2 patients improved from moderate to mild impairment, 2 patients improved from moderate to no impairment, and 3 patients improved from mild to no impairment of basic cognitive functioning.

Six patients showed clinically significant decreases in their basic cognitive functioning between 1 and 3 months poststroke; 3 patients deteriorated from no impairment to mild impairment, 2 patients deteriorated from no impairment to moderate impairment, and 1 patient deteriorated from mild to moderate impairment.

Four patients demonstrated decreasing levels of basic cognitive functioning between 3 and 12 months poststroke; 3 patients deteriorated from no impairment to mild impairment, and 1 patient deteriorated from mild to moderate impairment of basic cognitive functioning.

Cognitive functioning: Simple attention. Thirteen patients experienced clinically significant improvement of simple attention abilities between 3 and 12 months poststroke; 2 patients improved from severe to mild impairment, 2 patients improved from severe impairment to no impairment, and 9 patients improved from mild impairment to no impairment of simple attention abilities.

Ten patients experienced clinically significant deterioration of simple attention abilities between 3 and 12 months poststroke; 6 patients deteriorated from no impairment to mild impairment, and 4 patients deteriorated from mild to severe impairment of simple attention abilities.

Cognitive functioning: Complex attention. Four patients experienced clinically significant improvement of complex attention abilities between 3 and 12 months poststroke; 1 patient improved from severe to mild impairment, and 3 patients improved from mild impairment to no impairment of complex attention abilities.

Eight patients demonstrated clinically significant deterioration of complex attention abilities between 3 and 12 months poststroke; 3 patients deteriorated from no impairment to mild impairment, and 5 patients deteriorated from mild to severe impairment of complex attention abilities.

Cognitive functioning: Immediate verbal memory. Eighteen patients experienced clinically significant improvement of immediate verbal memory between 3 and 12 months poststroke; 4 patients improved from severe to mild impairment, 1

patients improved from severe impairment to no impairment, and 13 patients improved from mild impairment to no impairment of immediate verbal memory.

Eleven patients experienced clinically significant deterioration of immediate verbal memory between 3 and 12 months poststroke; 5 patients deteriorated from no impairment to mild impairment, and 6 patients deteriorated from mild to severe impairment of immediate verbal memory.

Cognitive functioning: Delayed verbal memory. Fourteen patients experienced clinically significant improvement of delayed verbal memory between 3 and 12 months poststroke; 5 patients improved from severe to mild impairment, 2 patients improved from severe impairment to no impairment, and 7 patients improved from mild impairment to no impairment of delayed verbal memory.

Four patients experienced clinically significant deterioration of delayed verbal memory between 3 and 12 months poststroke; all 4 patients deteriorated from no impairment to mild impairment of delayed verbal memory.

Cognitive functioning: Verbal fluency. Twelve patients experienced clinically significant improvement of verbal fluency between 3 and 12 months poststroke; 3 patients improved from severe to mild impairment, 2 patients improved from severe to no impairment, and 7 patients improved from mild to no impairment of verbal fluency.

Ten patients experienced clinically significant deterioration of verbal fluency between 3 and 12 months poststroke; 4 patients deteriorated from no impairment to mild impairment, and 6 patients deteriorated from mild to severe impairment of verbal fluency.

Appendix I

Individual Analysis of Clinically Significant Changes in Patients' Functioning ($N = 63$)

Type of Change	*n*	Patient Identification Number
Barthel ADL Index		
Improved (1 - 3 months)	12	004, 010, 013, 015, 023, 026, 032, 041, 046, 062, 096, 102.
Deteriorated (1 - 3 months)	9	008, 009, 028, 029, 033, 061, 070, 072, 084.
Improved (3 - 12 months)	9	008, 009, 028, 029, 046, 070, 084, 102, 103.
Deteriorated (3 - 12 months)	7	007, 013, 015, 023, 041, 063, 079.
GDS		
Improved (1 - 3 months)	8	004, 013, 035, 042, 046, 070, 082, 103.
Deteriorated (1 - 3 months)	10	010, 026, 027, 033, 061, 062, 075, 079, 083, 091.
Improved (3 - 12 months)	10	016, 019, 027, 033, 041, 066, 079, 081, 083, 091.
Deteriorated (3 - 12 months)	9	004, 013, 026, 045, 070, 077, 082, 087, 097.

Type of Change	*n*	Patient Identification Numbers
CAS		
Improved (1 - 3 months)	12	004, 033, 035, 037, 044, 062, 063, 067, 070, 093, 097, 102.
Deteriorated (1 - 3 months)	6	009, 016, 029, 054, 077, 103.
Improved (3 - 12 months)	7	009, 029, 046, 053, 054, 084, 103.
Deteriorated (3 - 12 months)	4	007, 032, 044, 070.
Digit Span		
Improved	13	004, 009, 016, 026, 029, 031, 053, 062, 066, 070, 081, 084, 099.
Deteriorated	10	007, 008, 012, 032, 033, 035, 077, 080, 082, 102.
Digit Symbol		
Improved	4	031, 052, 063, 071.
Deteriorated	8	007, 008, 010, 016, 028, 054, 091, 103.
VPA, immediate memory		
Improved	18	007, 012, 019, 026, 035, 037, 044, 045, 061, 062, 067, 069, 072, 081, 089, 096, 102, 103.
Deteriorated	11	016, 027, 039, 041, 046, 053, 064, 080, 082, 087, 093.

Type of Change	*n*	Patient Identification Numbers
VPA, delayed memory		
Improved	14	009, 031, 041, 044, 062, 064, 067, 081, 084, 089, 091, 093, 097, 099.
Deteriorated	4	016, 026, 102, 103.
COWA		
Improved	12	001, 010, 019, 061, 066, 077, 081, 087, 093, 096, 097, 103.
Deteriorated	10	007, 016, 027, 028, 031, 039, 044, 054, 064, 102.

Note. CAS = Cognitive Assessment Scale; COWA = Controlled Oral Word Association Test; GDS = Geriatric Depression Scale; VPA = Verbal Recall subtest of the Wechsler Memory Scale.

ADDENDUM

The following issues are raised briefly in this thesis. However, this addendum contains further discussion of these issues.

Factors which may contribute to the Stress Level of Poststroke Depression Sufferers, such as Financial Burden and Pre-injury Quality of Life

The present study examined the involvement of many factors in PSD. However, there are a number of additional factors which may also impact on the psychological wellbeing of stroke patients.

The financial situation of the patient is one such factor. Understandably, if the patient feels that they are in a situation of financial stress, this may add to their feelings of depression. In addition, a poor financial position may result in the patient being unable to receive the care they desire (e.g., resthome care).

The quality of life of the patient prior to the stroke may also impact to some extent on PSD. Patients who perceive themselves to have a reduced quality of life as a result of their stroke may feel depressed due to their poststroke condition. Alternatively, patients for whom the strokes had little affect on their quality of life, but feel that their prestroke quality of life was poor, may feel that the stroke was just one more burden dealt to them.

Similarly, the occurrence of a major life event around the time of the stroke may also add to the PSD, whether before or after the stroke. Again, this may seem like just another situation/event the patient has to deal with.

The level of caregiver functioning, both cognitive and physical, has the potential to affect patient PSD. In many cases, particularly in terms of older stroke patients, the person who has had the stroke may have been caring to some extent for their partner prior to the stroke. However, as a result of the stroke, the roles

have been reversed.

It must be noted that the above factors may not only impact on the stroke patient, but are just as likely to have an affect on the psychological wellbeing of the caregiver.

Implications of the Current Findings for the Treatment of PSD at Various Points since the Stroke

The present study highlighted relationships between PSD and associated factors at the various stages of the poststroke period. These relationships have certain implications for treatment, particularly in terms of the changes in the relationships across the first year poststroke.

At 1 month poststroke the strongest predictors of PSD were left hemisphere stroke, and the occurrence of a previous stroke. The significant involvement of the left hemisphere suggests that PSD at the acute poststroke stage may be of an organic nature, therefore perhaps best treated using pharmacological methods. In addition, psychotherapy of some nature may be useful to assist the patient with any issues regarding the impact of a repeated stroke.

At 3 months poststroke the strongest predictors of PSD in the patient were previous stroke, physical impairment, and cognitive impairment (particularly impairment of simple attention abilities). At 12 months poststroke, the strongest predictors were previous stroke and physical impairment. Therefore, at these stages of the poststroke period, assuming that depression occurs as a result of the impairment, clinical interventions may include helping the patient deal with their impairment through therapy, such as working through grief issues.

In addition to the above aspects of functioning that may provide focus issues for treatment, the pattern of recovery reported in the present study may also

have implications for treatment. That is, although individual patients indicated changes in the degree of physical and cognitive functioning, the group on average did not demonstrate clinically significant improvement in physical functioning. Cognitively, clinically significant improvement in the group as a whole was demonstrated in the areas of basic cognitive functioning, and delayed verbal memory. In addition, a significant decline in complex attention abilities was evident in the group by 12 months poststroke. These changes (or lack thereof) may have implications for treatment, particularly when considering treating someone who may experience a decline in attention abilities.

In addition to the clinical implications of the findings of the present study, there are also implications in terms of social/emotional support. People supporting stroke patients need to be aware that depression after stroke is a common occurrence, and that patients should be watched for signs of developing PSD. It is also important for friends, family, GPs and others involved to also recognise and act on the importance of support for the caregiver.

The Merits and Demerits of Cut-off Points in Scales, and Issues relating to 'Clinically Significant' scores

Cut-off points are often used by researchers and clinicians to identify whether the relevant aspect of a person's functioning indicate impairment. The cut-off scores for various measures are determined by identifying the score that indicates a substantial shift from the mean, or a significance difference from the score of the general population. This is similar to the manner in which scores are considered to be clinically significant. If a person obtains a score that differs from the mean by one standard deviation, then they are considered mildly impaired. A score two standard deviations from the mean is an indication of severe

impairment. Therefore, 'symptoms' become 'clinically significant symptoms' when they are at least one standard deviation from the mean, or when they are on the side of the cut-off score that indicates impairment, or abnormal functioning.

There may be some drawbacks of this method as, using the Geriatric Depression Scale (GDS) for example, a person with a score of 9 is not considered to be depressed, whereas a person who scores 10 is considered to be depressed. Therefore, important information is lost as each score is translated into a category. However, what the cut-off score has determined is that while the person with a score of 9 may indicate some symptoms of depression, the symptoms are not clinically significant, and the person with a score of 10 does present with clinically significant depression. The argument in favour of the cut-off scores is that a score of 10 differs significantly from the normal population, and therefore warrants further attention. While in some research the continuous scale of the GDS may be more useful in calculations, particularly in order to examine relationships, many forms of clinical research (such as the present study) prefer to examine prevalence rates, therefore cut-off points are of more use.

Further Research

The present study brings up interesting questions for further research, involving both the stroke patient, and the caregiver.

Firstly, and obviously, it would be interesting to continue the study further into the poststroke period. It would be particularly interesting to establish whether the relationship between PSD and previous strokes is still evident, for example at 24 and 36 months poststroke, in addition to the relationship between PSD and physical impairment. The present study found that, although a very slight decrease in depression was evident, approximately one third of stroke patients were

depressed at 1, 3, and 12 months poststroke. An extension of the study would also examine the prevalence of PSD later in the poststroke period, determining whether the slight decrease in prevalence continues, and/or decreases further with time. It would also be interesting to examine the relationship between PSD and mortality later in the poststroke period, particularly examining the possibility that any relationship between PSD and mortality may be more due to the increasing age of the patients, as opposed to PSD.

An additional matter for further research would be to complete a similar study on a younger stroke population. As stroke is more common in the older population (Toole, 1989) and the lifestyles of younger people are likely to differ from that of the older population, it is possible that the impact of stroke on a person's life also differs. Therefore, the potential for PSD may be greater, and/or PSD in younger patients may be best predicted by factors other than that found in the present study.

As the present study found that approximately one third of the stroke patients were depressed during the first year poststroke, it would be interesting to complete an intervention study, where patients received some form of treatment for their depression. It may be possible to treat some patients with anti-depressants, and some with psychotherapy. As the majority of depressed patients in the present study were not depressed for the entire first year poststroke, it would be difficult, but interesting, to determine what changes were due to the treatment?

Further research could also be completed concerning the caregivers of the stroke patients. For example, it would be interesting to examine the impact on caregiver psychological wellbeing of other aspects of caregiver functioning not included in the present study. These aspects could include the financial situation

of the patient and caregiver, and caregiver physical functioning. It would also be interesting to determine whether any aspects of caregiver functioning have a predictive relationship with patient PSD.