

行政院國家科學委員會專題研究計畫成果報告

腦幹與大腦半球梗塞對情緒狀態之影響：長期性追蹤研究

計畫編號： NSC 88-2413-H-002-022

執行期間： 87年 8月 1日至 88年 7月 31日

計畫主持人：花茂琴

共同主持人：陳獻宗、徐文俊

執行單位：台灣大學

中華民國 88年 11月 22日

Effects of Brainstem and Hemispheric Infarction on Emotional Status: A Longitudinal Study (I)

Mau-Sun Hua, Sien-Tsong Chen and Wen-Chuin Hsu

中文摘要：

第一年研究主要目的是檢驗腦幹中風病人的情緒狀態。總共五組中風病人分別為左側大腦與右側大腦皮質中風病人、左側與右側皮質下腦部中風病人以及腦幹中風病人，以及一組正常受試者參與本研究。每位受試者皆接受一組情緒功能的測驗，這六組受試者在年齡教育程度與性別相互配對。資料分析結果顯示：腦幹病人有輕微程度情緒功能的改變；憂鬱是主要的症狀。除此以外情緒功能的問題上包括極輕微到輕微體化型、強迫性意念與行為以及焦慮等症狀，由於目前這些結果來自有限病人之樣本，因此進一步繼續探討這些問題是否真正存在是有其必要性的。至於原計畫在第一年欲探討部分有關這些腦幹中風病人情緒功能改變的現象是否為短暫性質或持久性質的問題，同樣的，在相當有限的追蹤資料情況下，僅能留待第二年的研究計畫來探討。

關鍵字：憂鬱症、情緒功能狀態、腦幹中風

ABSTRACT

The aim of the first-year study was to examine the emotional status of the patients with brainstem stroke. An emotional function test battery was administered to five groups of the adult patients with cerebral hemispheric, subcortical, and brainstem strokes, and one group of normal control adults. Members of these six groups were matched in terms of age, educational level, and sex. Comparing the test results revealed that the patients with brainstem had a mild impairment of emotional function, mainly depression. In addition, otherwise psychopathological manifestations including slight to mild somatization, obsessive-compulsive symptom, and anxiety were also noticed. Since our findings were based on the limited data, further investigation is necessary. Likewise, only a few patients have received the 6-month follow-up evaluation of their emotional function in this first-year study. The issue regarding whether or not emotional changes evident in the patients with brainstem stroke are transient or persistent, thus, awaits the second-year investigation.

Key words: Depression, Emotional Status, Brainstem Stroke

Stroke is a common disease of central nervous system. In Taiwan this disease has been the second leading cause of death for all ages (Huang, Chiang & Lee, 1997). Physical disability, and a variety of cognitive impairments, such as aphasia, amnesia, apraxia, agnosia, aprosodia, dysarthria, attentional problem, neglect, and visuospatial disorientation are frequent consequences of cerebral hemispheric stroke (Heilman & Valenstein, 1993; Lezak, 1995; Walsh, 1994). In addition to these deficits, emotional changes are also a common sequela (Hosking, Marsh & Friedman, 1996). The disturbances include depression, emotional lability, apathy, irritability, agoraphobia, social withdrawal, self-neglect, and anxiety (House, Dennis, Mogridge, Warlow, Hawton & Jones, 1991; Morris, Robinson, & Raphael, 1993; Nelson, Cicchetti, Satz, Sowa & Mitrushina, 1994).

The implied clinical and economic impacts of these emotional disturbances on the stroke patients are multifold (Spencer, Tompkins & Schultz, 1997). First, the patients with emotional problems undertake more negative thinking and stress negative results (Ingram, Kendall, Smith, Donnell & Ronan, 1987). Consequently, sundry cognitive function changes in these patients might be exaggerated though cognitive impairments tend to co-occur with emotional problems (Seibert & Ellis, 1991; Speedie et al., 1990). Secondly, sundry cognitive function changes, such as depression can be a vital hindrance to rehabilitation (Reynolds, 1992). Finally, the quality of life of the patients

and their caregivers might be changed tensely by these emotional sequelae (Spencer et al.,1997). Therefore, in order to offer the patients the supreme opportunity for treatment profits, a great care for their emotional disturbances is merited.

Depression has generally been considered to be the most common emotional consequence of cerebral hemispheric stroke (Nelson, 1994; Price, 1990). The prevalence rate of poststroke depression, however, varies extensively from less than 25% up to more than 60% (Astrom, Adolfsson & Asplund, 1993). Methodological discrepancies mainly including the selection of patients, the time of evaluation since the stroke and the duration of the assessment period, as well as diverse diagnostic criteria and psychopathological measures have a significant contribution to this variation (Astrom et al., 1993; Hosking et al., 1996). Furthermore, the proportion of poststroke depression does not keep steady throughout the poststroke period (Astrom et al., 1993). The manifestations of otherwise emotional problems, such as agoraphobia, social withdrawal, anxiety, mania, catastrophic reactions, and apathy recently have also been observed in the patients following cerebral hemispheric stroke (e.g., House et al., 1991; Morris et al., 1993; Robinson & Starkstein, 1997). The issue regarding whether or not depression is the most frequent emotional result of cerebral hemispheric stroke, thus, becomes controversial and awaits further investigation.

Emotional abnormalities are also evident in the patients with subcortical stroke

though the literature in this area is less extensive than that of cerebral hemispheric stroke. Generally, depression is significantly related to lesions of left basal ganglia (Starkstein, Robinson, Berthier & Price, 1988). However, mania, bipolar affective disorders, and psychosis are highly associated with right subcortical (basal ganglia or thalamus) lesions (Robinson et al., 1988; Starkstein et al., 1991; Starkstein, Robinson & Berthier, 1992; Trimble & Cummings, 1981). Anxiety and apathy have a high frequency of lesions involving the posterior limb of the internal capsule (Robinson & Starkstein, 1997; Starkstein et al., 1993a) while catastrophic reactions are notably related to anterior subcortical lesions (Starkstein et al., 1993b).

The dysfunction of the biogenic amine system has been hypothesized to be one of the mechanisms that may play an important role in the cause of depression (Beatty, 1995; Conn, 1995). Both noradrenergic and serotonergic cell bodies are located in the locus coeruleus and raphe nucleus of the brainstem, and send ascending projections to the frontal cortex through the median forebrain bundle. The ascending pathways then arc posteriorly and travel longitudinally through the deep layers of the cortex, arborizing and sending terminal projections into the superficial cortical layer (Morrison, Molliver, & Grzanna, 1979). On the basis of neuroanatomical evidences and clinical findings, Robinson and his colleagues (1984) proposed that poststroke depression might be the result of severe depletions of norepinephrine and/or serotonin

due to lesions in either the frontal cortex or basal ganglia. If this is the case, depressive symptoms should also be evident in the patients with brainstem stroke involving the rostral pons and caudal mesencephalon which are sites for the serotonergic raphe nucleus and the noradrenergic locus coeruleus (Lang, 1991).

The literature of emotional disturbances in the patients following brainstem stroke, however, is very limited. Trimble and Cumminings (1981) reported that manifestations of suicidal ideation, unstable mood, euphoria, depressive feelings, and emotional lability were noted in two patients with brainstem stroke. However, the definite lesioned areas of both patients determined by the CT scan were located in the thalamus rather than the brainstem. Starkstein, Robinson, Berthier and Price (1988) found that patients with posterior circulation lesions involving the brainstem and/or cerebellum had a remarkably low frequency of depression, and a significantly short duration of depressive symptoms. Unfortunately, since no otherwise psychopathological measures, with the exception of depression and anxiety tests, were used in their study, the issue of whether or not other emotional sequelae might also be manifested in patients following brainstem stroke remains unknown.

The report of emotional disturbances in poststroke patients in Taiwan is lacking. In order to explore the above unclear issue, and to document the literature in Taiwan, we design this two-year longitudinal study. In our study, 6 groups of the participants,

that is, 5 patient cohorts and 1 group of normal controls will be included. 5 patient groups consist of 1 group of patients following brainstem stroke, 1 with left and 1 with right cortical stroke, and 1 with left and 1 with right basal ganglion stroke. The first-year study goal is to complete 1- and 6-monthss poststroke assessment of emotional function; the second-year study goal is to complete 1-year poststroke evaluation of emotional status. The specific aims of the study, thus, are to examine the following questions: 1) Is there an impairment of emotional status in patients with brainstem stroke? 2) If so, does the deficit only involve depression, as Starkstein et al's observation (1988), or also include otherwise emotional function tested? 3) If depression or/and otherwise emotional changes does/or occur in the patients with brainstem stroke, is/are the deficit(s) transient or persistent?

METHOD

Participants. Because of a very high dropout rate of the participants, 6 groups with only 55 volunteer adults were included in the first-year study. There were 37 stroke patients including 12 with brainstem (Group 1), 3 with left cortical (Group 2), 5 with left subcoritcal (Group 3), 8 with right cortical (Group 4), and 9 with right subcortical (Group 5) lesions, and 18 normal control subjects (Group 6). The lesion locations were verified by MRI, and all of these patients were victims of the first stroke in the absence of any other CNS, and systemic disease (e.g., diabetes and hypertension with

drug treatment), and psychiatric history. The patients were also free of dementia, confusional state, anosognosia, and moderate or severe comprehension deficits.

The mean differences in age were not significantly different, with the exception of Groups 4 and 5 and Groups 4 and 6. That is, the mean age of the patients with right cortical stroke was significantly younger than that of the patients with right subcortical stroke and of the normal controls (Table 1). The mean differences in education were also not significantly different. All participants were right-handed in which hand dominance was ascertained by the history that the participant has always used his/her right hand preferentially for doing skillful activities, such as writing and holding chopsticks.

Tests and procedure. After giving informed consent, each participant will receive a series of neuropsychological tests. They included cognitive function and emotional status measures. The Temporal Orientation Test (Benton, Hamsher, Varney & Spreen, 1983), the Orientation to Personal Information and Place (Hamsher, 1983), Object Naming Test (Spreen & Benton, 1969), and Aural Comprehension (Benton & Hamsher, 1978) were used to rule out the participants susceptible for dementia, moderately or severely receptive aphasia, confusional state, and anosognosia. The test battery of emotional status consisted of Symptom Checklist-90-R (Derogatis, 1977), Beck Depression Inventory (Beck, 1987), Neurobehavioral Rating Scale (Levin et al.,

1987), and a semi-structured Standard Neurobehavioral Interview Inventory (Hamsher, 1983). In addition, Canadian Neurological Scale and Barthel Index (Mahoney & Barthel, 1965) were also used to measure the severity of the patient's physical disability, and evaluate the degree of his/her independence in basic activities of daily living respectively. The assessment was administered in an examing room between 11 AM and 2PM to minimize any possible effects of diurnal mood variation on interview responses, as Starkstein, Cohen, Fedoroff, Parikh, Price, and Robinson (1990). All measures were Chinese versions.

In order to obtain reliable and valid ratings of the patient's emotional status, we also asked the participant's significant others, particularly family caregivers of the patient, to rate the emotional status of the participant. All of the significant informants of the patients will be free of emotional disturbances, dementia, and psychiatric history. For the present, our available patients only received the first examination, that is, they were examined at the poststroke periord of 1 month while a few (2 patients) received the second examination, that is, 6 months after the onset of stroke.

RESULTS

Since the measures of emotional status are ordinal scales, Kruskal-Wallis one-way analysis of variance by Ranks (Marascuili and McSweeney, 1977) was used to determine whether the performance of the patient groups on the Beck Depression

Inventory and SCL-90-R was different from that of the normal control participants. On the Beck Depression Inventory, the patients with brainstem stroke had the highest mean rank followed by the patients with left subcortical stroke, the patients with right subcortical stroke, the patients with right cortical stroke, the patients with left cortical stroke, and the normal controls. The mean rank differences among the participant groups did reach a statistical difference (Chi square=17.92, df = 5, $p < .05$). Nemenyi's post-hoc pairwise contrasts with Bonferroni corrections for a control of Type I error were subsequently used to analyze the data. The results, however, did not reveal any significant differences between the participant groups (Tables 2 and 3).

Likewise, on the SCL-90-R, the highest mean rank to the lowest on the Somatization subscale was the patients with brainstem stroke, the patients with left subcortical stroke, the patients with right cortical stroke, the patients with right subcortical stroke, the normal controls, and the patients with left cortical stroke. On the Obsessive-Compulsive subscale, the patients with brainstem stroke had the highest mean rank followed by the patients with right subcortical and cortical stroke, the patients with left subcortical and cortical stroke, and the normal controls. On the Interpersonal Sensitivity subscale, the highest mean rank to the lowest was the patients with brainstem stroke, with left subcortical stroke, with right subcortical stroke, the normal controls, and the patients with left cortical stroke. The patients with

brainstem stroke had the highest mean rank on the Depression subscale, followed by the patients with right and left subcortical stroke, with right cortical stroke, the normal controls, and left cortical stroke. On the Anxiety subscale, the patients with brainstem stroke had the highest mean rank followed by the patients with right cortical stroke, with right and left subcortical stroke, the normal controls, and with left cortical stroke. The highest mean rank on the Hostility subscale was the patients with brainstem stroke, followed by the patients with left and right subcortical stroke, the normal controls, and the patients with right and left cortical stroke. On the Phobia subscale, the highest mean rank to the lowest was the patients with brainstem stroke, with right cortical stroke, with left and right subcortical stroke, the normal controls, and the patients with left cortical stroke. The highest mean rank to the lowest on the Paranoid Ideation subscale was the patients with right cortical stroke followed by the patients with brainstem stroke, the normal controls, the patients with left and right subcortical stroke, and with left cortical stroke. On the psychoticism subscale, the highest mean rank score was the patients with brainstem stroke, followed by the patients with right cortical stroke, with left subcortical stroke, the normal controls, the patients with right subcortical stroke, and the patients with left cortical stroke. The differences of the mean rank over these subscales, however, only involved Somatization (Chi square=12.06, df = 5, p<.04), Psychoticism (Chi square=14.49, df =5, p<.02), and

Global (Chi square=12.15, df =5, $p<.04$) reached a statistically significant level. No significant differences were found when Nemenyi's post-hoc pairwise contrasts with Bonferroni corrections for controlling Type I error were subsequently performed.

The highest mean rank to the lowest of the significant informants of the participants on the BDI was the patients with brainstem stroke, followed by the patients with left and right subcortical stroke, the patients with right and left cortical stroke, and the normal controls. The mean rank differences, analyzed by Kruskal-Wallis one-way ANOVA revealed, did reach a statistical significant level (Chi square=3.74, df=5, $p<.007$). Nemenyi's post-hoc pairwise comparisons with Bonferroni corrections for controlling Type I error were subsequently performed. However, none reached a statistically significant level.

The rating of the significant informants of the participants on the subscales of the SCL-90-R was as followed. On the Somatization subscale, the patients with brainstem stroke had the highest mean rank, followed by the patients with left subcortical stroke, with right cortical and subcortical stroke, the normal controls, and the patients with left cortical stroke. The highest mean rank to the lowest on the Obsessive-Compulsive subscale was the patients with brainstem stroke, followed by the patients with right subcortical and cortical stroke, with right coritcal and subcortical stroke, with left subcortical and cortical stroke, and the normal controls. On the Interpersonal

Sensitivity subscale, the patients with brainstem stroke had the highest mean rank, followed by the patients with left subcortical stroke, with right cortical and subcortical stroke, the normal controls, and the patients with left cortical stroke. The highest mean rank to the lowest on the Depression subscale was the patients with the patients with brainstem, followed by the patients with right subcortical and cortical stroke, with left subcortical and cortical stroke, and the normal controls. On the Anxiety subscale, the patients with brainstem stroke had the highest mean rank, followed by the patients with right subcortical stroke, with left cortical stroke, with right cortical and left subcortical stroke, and the normal controls. On the Hostility subscale, the patients with brainstem stroke had the highest mean rank, followed by the patients with right subcortical and cortical stroke, with left subcortical stroke, the normal controls, and the patients with left cortical stroke. The highest mean rank to the lowest on the Phobia subcale was the patients with right subcortical and cortical stroke, with left subcortical stroke, the normal controls, and the patients with left cortical stroke. On the Paranoid Ideation subscale, the patients with brainstem stroke had the highest mean rank, followed by the patients with right cortical and left subcortical stroke, with right subcortical stroke, the normal controls, and the patients with left cortical stroke. The highest mean rank on the Psychoticism subscale was the patients with brainstem stroke, followed by the patients with right subcortical and cortical stroke,

with left subcortical stroke, the normal controls, and the patients with left cortical stroke.

The data analysis by Kruskal-Wallis one-way ANOVA revealed that the mean rank differences of Somatization (Chi square= 11.28, df=5, $p<.05$), Obsessive-Compulsive (Chi square=12.04, df=5, $p<.04$), Depression (Chi square=11.80, df=5, $p<.04$), Anxiety (Chi square=11.24, df=5, $p<.05$), and Psychoticism (Chi square=12.13, df=5, $p<.04$) subscales among these subject groups reached a statistically significant level. Subsequent Nemenyi's post-hoc pairwise contrasts with Bonferroni corrections for controlling Type I error, however, did not reach a statistically significant level.

In order to determine whether or not the ratings of the participants and their significant informants on the BDI and SCL-90-R were different. On the BDI, the discrepant score between the participant and his/her significant informant for each subject group was calculated, and then a match-paired t test was used to analyze the data. The result revealed that the differences did not reach a statistically significant level. Likewise, for each subject group the ranking differences between the participant and his/her significant informant on each of the subscales of the SCL-90-R were not statistically significant.

DISCUSSION

Is there an impairment of emotional status in the patients with brainstem stroke?

Because of the high dropout of the patients, our results based on the limited data did not revealed that the patients with brainstem stroke had remarkably emotional impairments compared with their normal counterparts. For the time being the answer for our first question, thus, seems to be negative. Meanwhile, our preliminary results also appear to further support Starkstein and his co-workers' findings (1988) in which they noted the patients with brainstem stroke having a significant low frequency of emotional changes. However, our results also revealed some degree of subtle to mild emotional changes in these patients with brainstem stroke. The definite conclusion, thus, awaits the second-year investigation.

If so, does the deficit only involve depression, as Starkstein and his colleagues' observation (1988), or also include otherwise emotional function tested? Accordingly, the definite answer for the first question is pending. Thus, it appears to be appropriate to make some comments on this issue. Our patients with brainstem stroke not only had mild depression, but also had other emotional changes. These otherwise emotional problems consisted of somatization, obsessive-compulsive symptoms, anxiety, hostility, paranoid ideation, and psychoticism.

Most of the literature showed that depression was major emotional changes evident in the patients with cerebral hemispheric lesions (e.g., Nelson, 1994; Price,

1990). Our findings based on the patients with either the left or right cerebral hemispheric stroke, however, were not consistent with these earlier observations. Instead, somatization was mainly evident in our patients with right cerebral hemispheric stroke. In addition, obsessive-compulsive symptom and anxiety were also evident in the patients with right cerebral hemispheric stroke. These results seemed to be consistent with the researchers' observations (e.g., House et al., 1991; Morris et al., 1993; Robinson & Starkstein, 1997).

Depression has often been seen in the patients with lesions of the subcortical regions, particularly the areas of left basal ganglia (Starkstein Robinson, Berthier & Price, 1988). Generally, our results were compatible with these observations. Nevertheless, we did not note our patients having any remarkably asymmetric manifestation between the two subcortical hemispheric strokes in terms of this specific aspect of emotional changes. Robinson and Starkstein (1997), and Starkstein and his co-workers (1993) reported that the patients with lesions involving the posterior limb of the internal capsule were often evident of anxiety. We did also notice our patients with the subcortical stroke having the symptom of anxiety. However, the lesion area in our patients not only involved the subcortical white matters, but also the adjacent subcortical gray regions. The findings of mania, bipolar affective disorders, psychosis, apathy, and catastrophic reactions have been reported

in the patients with subcortical lesions (e.g., Robinson et al., 1988; Starkstein et al., 1991; Starkstein, Robinson, & Berthier, 1992; Trimble & Cummings, 1981; Starkstein et al., 1993). We have not yet observed these emotional changes in our patients with the subcortical stroke. However, the manifestation of somatization and obsessive-compulsive symptom was also evident in our patients.

The dysfunction of the biogenic amine system has been hypothesized to be one of the mechanisms that may play an essential role in the cause of depression (Beatty, 1995; Conn, 1995). Since both noradrenergic and serotonergic cell bodies are located in the locus coeruleus and raphe nucleus of the brainstem, depressive symptoms should be evident in the patients with brainstem stroke involving the rostral pons and caudal mesencephalon which are sites for these two nuclei. In fact, depression was the cardinal psychopathological symptom in our patients with brainstem stroke. Accordingly, our results seemed to be accordance with the hypothesis of the dysfunction of the biogenic amine system. In addition, somatization, obsessive-compulsive symptoms, and anxiety were also evident in these patients.

In line with this hypothesis and on the basis of neuroanatomical evidences and clinical findings, Robinson and his co-workers (1984) also claimed that poststroke depression might be as a result of depletion of norepinephrine and/or serotonin due to lesions in either the frontal cortex or basal ganglia. Our findings of depression also

evident in the patients with subcortical stroke seemed to substantiate these researchers' proposition.

In summary, our preliminary results seemed to implicate that a slight to mild impairment of emotional status was evident in the patients with brainstem stroke, and depression was the cardinal psychopathological symptom though these were not on the statistical basis. In addition, otherwise slightly to mildly emotional changes including somatization, obsessive-compulsive symptom, and anxiety were also noticed in these patients. Since the available data were limited, the proposed questions require further investigation.

REFERENCES

- Astrom, M., Adolfsson, R., & Asplund, K. (1993). Major depression in stroke patients: A 3-year longitudinal study. *Stroke*, 24, 976-982.
- Beatty, J. (1995). Principles of behavioral neuroscience (pp. 492-495). Chicago, ILL: Brown & Benchmark Publishers.
- Beck, A. T. (1987). Beck Depression Inventory. San Antonio, TX: The Psychological Corporation.
- Benton, A. L., & Hamsher, K. deS. (1978). Multilingual Aphasia Examination. Iowa City: University of Iowa Hospital, Department of Neurology.
- Benton, A. L., Hamsher, K. deS., Varney, N. R., & Spreen, O. (1983). Contributions to neuropsychological assessment: A clinical manual. New York: Oxford University Press.
- Conn, P. M. (1995). Neuroscience in Medicine (pp. 409-417). Philadelphia, PA: J. B. Lippincott Company.
- Derogatis, L. R. (1977). SCL.90-R (revised) version manual-I. Baltimore: Clinical Psychometrics Research Unit, Johns Hopkins University School of Medicine.
- Hamsher, K. deS. (1983). Standard Neurobehavioral Interview Inventory. Milwaukee: University of Wisconsin Medical School, Department of Neurology.
- Hamsher, K. deS. (1983). Orientation to Personal Information and Place. Milwaukee: University of Wisconsin Medical School, Department of Neurology.
- Heilman, K. M., & Valenstein, E. (Eds.). (1995) Clinical neuropsychology (3rd ed.). New York: Oxford University Press.
- Hosking, S. G., Marsh, N. V., & Friedman, P. J. (1996). Poststroke depression: Prevalence, course and associated factors. *Neuropsychology Review*, 6, 107-133.

- House, A., Dennis, M., Mogridge, L., Warlow, C., Hawton., & Jones, L. (1991). Mood disorders in the year after first stroke. *British Journal of Psychiatry*, 158,83-92.
- Huang, Z.-S., Chiang, T.-L., & Lee, T.-K. (1997). Stroke prevalence in Taiwan: Finding from the 1994 National Health Interview Survey. *Stroke*, 28, 1579-1584.
- Ingram, R. E., Kendall, R. C., Smith, T. W., Donnell, C., & Ronan, K. (1987). Cognitive specificity in emotional distress. *Journal of Personality and Social Psychology*, 53,734-742.
- Lang, J. (1991). *Clinical anatomy of the posterior cranial fossa and its foramina*. New York: Thieme Medical.
- Levin, H. S., High, W. M. Goethe, K. E., et al. (1987). The Neurobehavioral Rating Scale assessment of the behavioral sequelae of head injury by the clinician. *Journal of Neurology, Neurosurgery, and Psychiatry*, 50, 183-193.
- Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.
- Mahoney, F., & Barthel, D. (1965). Functional evaluation: The Barthel Index. *Mareland State Medical Journal*, 14, 61-65.
- Morris, P. L. P., Robinson, R. G., & Raphael, B. (1993). Emotional lability after stroke. *Australian and New Zealand Journal of Psychiatry*, 27, 601-605.
- Morrison, J. H., Molliver, M. E., Grzanna, R. (1979). Noradrenergic innervation of the cerebral cortex: Widespread effects of local cortical lesions. *Science*, 205, 313-316.
- Nelson, L. D., Cicchetti, D., Satz, P., Sowa, M., & Mitrushina, M. (1994). Emotional sequelae of stroke: A longitudinal perspective. *Journal of Clinical and Experimental Neuropsychology*, 16, 796-806.
- Price, T. R. (1990). Affective disorders after stroke. *Stroke*, 21 (Supple 2), -12- -

13.

Reynolds, C. F. (1992). Treatment of depression in special populations. *Journal of Clinical Psychiatry*, 53,45-53.

Robinson, R. G., Boston, J. D., Starkstein, S. E., et al.(1988). Comparison of mania with depression following brain injury: Causal factors. *American Journal of Psychiatry*, 145, 172-178.

Robinson, R. G., Kunbos, K. L., Starr, L. B., et al. (1984). Mood disorders in stroke patients: Importance of location of lesion. *Brain*, 107, 81-93.

Robinson, R. G., & Starkstein, S. E. (1997). Neuropsychiatric aspects of cerebrovascular disorders. In S. C. Yudofsky & R. E. Hales (Eds.), *The American Psychiatric Press Textbook of Neuropsychiatry* (3rd ed.). Washington, DC: American Psychiatric Press, Inc.

Seibert, P. S., & Ellis, H. C. (1991). Irrelevant thoughts, emotional mood states, and cognitive task performance. *Memory and Cognition*, 19, 507-513.

Speedie, L., O'onnell, W., Rabins, P. Pearlson, G., Poggi, M., & Gonzalez Rothi, L. (1990). Language preformance deficits in elderly drpressed patients. *Aphasiology*, 4, 197-205.

Spencer, K. A., Tompkins, C.A., & Schulz, R. (1997). Assessment of depression in patients with brain pathology: The case of stroke. *Psychological Bulletin* , 122, 132-152.

Spreen, O., & Benton, A. L. (1969). *Neurosensory Center Comprehensive Examination for Aphasia*. Victoria, BC: Neuropsychological Laboratory, Department of Psychology, University of Victoria.

Starkstein, S. E., Cohen, B.S., Fedoroff, P., Parikh, R. M., Price T. R., & Robinson, R.G. (1990). Relationship between anxiety disorders and depressive disorders in patients with cerebrovascular injury. *Archives of General Psychiatry* , 47, 246-251.

Starkstein, S. E., Fedoroff, J. P., Berthier, M. L., et al. (1991). Manic depressive and pure manic states after brain lesions. *Biological Psychiatry*, 29, 149-158.

- Starkstein, S. E., Fedoroff, J. P., Price, T. R., et al (1993a). Apathy following cerebrovascular lesions. *Stroke*, 24, 1625-1630.
- Starkstein, S. E., Fedoroff, J. P., Price, T. R., et al. (1993b). Catastrophic reaction after cerebrovascular lesion: Frequency, correlates, and validation of a scale. *Journal of Neurology, Neurosurgery and Psychiatry*, 57, 189-194.
- Starkstein, S. E., Robinson, R. G., Berthier, M. L., & Price, T. R. (1988). Depressive disorders following posterior circulation as compared with middle cerebral artery infarcts. *Brain*, 111, 375-387.
- Trimble, M. R., & Cummings, J. L. (1981). Neuropsychiatric disturbances following brainstem lesions. *British Journal of Psychiatry*, 138,56-69.
- Walsh, K. (1994). *Neuropsychology: A clinical approach*. New York: Chrchill Livingstone.

Table 1. Demographic Characteristics of Subject Groups

	Sex		Age			Edu			TO			OPIP			ON			AC		
	F	M	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range
Brain stem stroke	6	6	62.08	9.57	42~77	3.75	4.88	0~12	1.08	1.62	0~5	11.50	1.24	8~12	15.58	1.44	11~16	14.83	3.74	8~18
R cortical	5	3	55.63	11.93	40~65	6.88	3.83	0~12	1.25	1.28	0~3	10.25	2.25	6~12	15.75	0.71	14~16	14.75	3.11	10~18
R subcortical	4	5	70.11	5.86	61~80	4.67	5.72	0~14	1.56	1.67	0~4	10.56	1.24	9~12	15.44	1.13	13~16	15.44	3.81	6~18
L cortical	0	3	68.67	15.63	52~83	6	6	0~12	2	2	0~4	6.67	6.11	0~12	16	0	16	12.33	6.03	6~18
L subcortical	2	3	67.60	6.19	61~76	2	2.83	0~6	2.40	1.95	0~5	11	0.71	10~12	15.60	0.89	14~16	14.20	3.11	11~18
健診	7	11	68.78	5.95	60~82	4.33	4.24	0~15	0.22	0.55	0~2	12	0	12	16	0	16	18	0	18

Table 2. Performance Scores of the BDI

BDI	P			F		
	Mean	SD	Range	Mean	SD	Range
Brain stem stroke	16.30	16.1	1~45	11.8	11.4	0~43
R cortical	5.75	5.65	1~16	6.29	2.50	4~11
R subcortical	7.33	7.62	1~24	7.67	6.58	1~21
L cortical	2.33	2.08	0~4	2.67	2.52	0~5
L subcortical	9.80	6.98	1~20	7.00	7.04	2~19
健診	2	2.74	0~11	2.5	4	0~13

Table 3. Patient's and Patient's Significant Informant's Rating Scores on the SCL-90-R

SCL		Brain stem stroke		R cortical		R subcortical		L cortical		L subcortical		健診	
		P	F	P	F	P	F	P	F	P	F	P	F
Somatization	Mean	0.94	0.87	0.65	0.49	0.56	0.41	0.23	0.37	0.90	0.86	0.42	0.37
	SD	0.68	0.64	0.27	0.25	0.44	0.26	0.12	0.31	0.32	0.56	0.52	0.46
	Range	0.2~2.8	0.3~2.3	0.3~1.1	0.1~0.8	0.1~1.3	0~0.8	0.1~0.3	0.1~0.7	0.6~1.4	0.3~1.8	0~2.1	0~1.9
Obsessive-Compulsive	Mean	0.66	0.54	0.44	0.45	0.59	0.46	0.27	0.43	0.40	0.48	0.21	0.16
	SD	0.63	0.49	0.36	0.27	0.56	0.51	0.25	0.23	0.29	0.51	0.32	0.26
	Range	0.1~2	0~1.3	0.1~1.1	0.2~0.9	0~1.6	0~1.5	0~0.5	0.3~0.7	0~0.7	0~1.3	0~1.1	0~1
Interpersonal Sensitivity	Mean	0.33	0.23	0.10	0.09	0.07	0.13	0	0.07	0.12	0.36	0.06	0.05
	SD	0.66	0.26	0.24	0.10	0.11	0.21	0	0.12	0.08	0.48	0.19	0.11
	Range	0~2	0~0.6	0~0.7	0~0.3	0~0.3	0~0.6	0	0~0.2	0~0.2	0~1.2	0~0.8	0~0.4
Depression	Mean	0.70	0.60	0.41	0.26	0.52	0.54	0.13	0.13	0.48	0.58	0.18	0.12
	SD	0.93	0.52	0.59	0.15	0.63	0.54	0.06	0.06	0.58	0.73	0.26	0.24
	Range	0.1~2.9	0~1.5	0~1.8	0~0.5	0~1.9	0~1.6	0.1~0.2	0.1~0.2	0~1.4	0~1.8	0~0.8	0~1
Anxiety	Mean	0.58	0.53	0.39	0.16	0.29	0.23	0.03	0.17	0.28	0.32	0.16	0.12
	SD	0.95	0.51	0.74	0.17	0.45	0.28	0.06	0.06	0.48	0.45	0.21	0.20
	Range	0~3	0~1.5	0~2.2	0~0.5	0~1.3	0~0.9	0~0.1	0.1~0.2	0~1.1	0~1.1	0~0.6	0~0.7
Hostility	Mean	0.33	0.50	0.10	0.28	0.16	0.34	0.07	0.10	0.28	0.54	0.15	0.17
	SD	0.36	0.43	0.19	0.24	0.15	0.44	0.12	0.17	0.31	0.68	0.27	0.25
	Range	0~1	0~1.2	0~0.5	0~0.7	0~0.3	0~1.3	0~0.2	0~0.3	0~0.7	0~1.5	0~1	0~0.7
Phobic Anxiety	Mean	0.38	0.18	0.24	0.11	0.17	0.27	0	0	0.22	0.18	0.01	0.02
	SD	0.86	0.29	0.56	0.20	0.36	0.45	0	0	0.44	0.40	0.03	0.07
	Range	0~2.7	0~0.9	0~1.6	0~0.6	0~1.1	0~1.3	0	0	0~1	0~0.9	0~0.1	0~0.3
Paranoid Ideation	Mean	0.19	0.28	0.21	0.25	0.02	0.19	0	0.17	0.04	0.42	0.06	0.05
	SD	0.37	0.28	0.36	0.29	0.07	0.26	0	0.29	0.09	0.52	0.18	0.10
	Range	0~1.3	0~0.8	0~1	0~0.7	0~0.2	0~0.7	0	0~0.5	0~0.2	0~1.2	0~0.7	0~0.3
Psychoticism	Mean	0.33	0.23	0.04	0.08	0.01	0.21	0	0	0.04	0.24	0.02	0.03
	SD	0.53	0.26	0.07	0.09	0.03	0.31	0	0	0.05	0.38	0.04	0.08
	Range	0~1.6	0~0.7	0~0.2	0~0.2	0~0.1	0~0.9	0	0	0~0.1	0~0.9	0~0.1	0~0.3
Global Severity Index	Mean	0.55	0.47	0.34	0.25	0.30	0.32	0.10	0.20	0.38	0.46	0.15	0.13
	SD	0.64	0.37	0.33	0.11	0.28	0.32	0.10	0.10	0.30	0.48	0.21	0.20
	Range	0.1~1.9	0.1~1.1	0.1~1.1	0.1~0.4	0~0.9	0~1	0~0.2	0.1~0.3	0.2~0.9	0.1~1.3	0~0.7	0~0.8
Positive Symptom Distress Index	Mean	1.65	1.47	1.56	1.19	1.71	1.70	1.10	1.30	1.72	1.58	1.38	1.39
	SD	0.50	0.27	0.40	0.20	0.34	0.90	0.10	0.44	0.31	0.33	0.37	0.46
	Range	1.1~2.7	1~1.8	1.2~2.4	1~1.6	1.3~2.2	1.1~4	1~1.2	1~1.8	1.4~2.1	1.2~1.9	1~2.2	1~2.2
Total	Mean	25.91	28.25	17.60	19.40	14.90	19.20	9.33	13.00	18.60	24.60	9.12	8.61
	SD	21.82	18.86	12.06	7.65	11.60	16.40	6.03	5.00	12.28	22.88	9.27	11.4
	Range	6~67	8~59	9~43	9~31	3~38	1~47	3~15	8~18	7~38	5~64	1~34	1~41

