NEUROPSYCHOLOGICAL SEQUELAE OF ANEURYSMAL SUB-ARACHNOID HAEMORRHAGE IN PATIENTS WITHOUT NEUROLOGICAL DEFICITS 6 TO 8 MONTHS POST OPERATIVELY - AN EXPLORATORY STUDY [1]

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[1] This paper was written in partial fulfilment of the requirements of the MA (Clinical Psychology) degree.

Dedicated to Sandy and Ann -
my editorial team who stayed
up until all hours!

THE CONTRIBUTION OF THE FOLLOWING PEOPLE IS GRATEFULLY ACKNOWLEDGED:

* Dr G Marus, Department of Neurosurgery, Groote Schuur Hospital, who made this study possible.

* My supervisors, Ann Jordan and Andy Dawes.

* The many people who provided advice and support - unfortunately space does not allow me to mention you all here. I am particularly grateful to Andrew Elliot, Beverley Moss-Morris, Cora de Villiers, Mike Saling, and Sandy Burbach for their particular contributions. In addition the support of my class - Jonathan, Pamela, Beverley, Marianne and Desiree was greatly appreciated.

* Charmaine Robertson, who produced a beautiful manuscript under great pressure.

ABSTRACT

Relatively little is known about the neuropsychological sequelae of sub-arachnoid haemorrhage (SAH) and to date, there has only been one study (Ljunggren et al, 1985) focussing on patients without neurological deficits after aneurysm surgery for major SAH. A detailed exploratory study was designed to investigate the physical, cognitive, emotional, personality and behavioural sequelae of SAH in this group of patients. Neuropsychological sequelae were investigated in terms of a wide range of variables which could affect prognosis, viz location of aneurysm, timing of surgery, severity of the initial bleed, age, gender, educational level and estimated premorbid intelligence. An opportunity sample (N = 18) was drawn from patients treated at the Neurosurgery Department, Groote Schuur Hospital, Cape Town from May to November 1986. Patients were assessed 6.1 to 8.0 months post-operatively (mean 7.0 months) by means of an extensive neuropsychological test battery. A detailed structured interview was also conducted with the patient and somebody who was familiar with the patient's everyday functioning. Subjects ranged in age from 17 to 67 years (mean 38.8 years) and had a mean educational level of Standard 6 (range 2-10). Results were analysed in terms of the total sample and then according to successive subdivisions of subjects, on the basis of prognostic variables. The incidence and severity of symptoms ascribed to SAH are reported. Test data was interpreted in terms of two complementary psychometric theories (cognitive fall-off, and specific cognitive ability models). Results of the various aspects of the assessment were integrated in terms of ratings of general functional outcome. A diffuse SAH-induced encephalopathy was detected, which confirmed the findings of Ljunggren et al (1985). Impairment in functional ability was present in 61% of the sample. Similarities were noted with the Ljunggren et al (1985) study in terms of the cognitive abilities that were impaired, as well as the physical, emotional, personality and behavioural symptoms that were reported. Differences in interpretation between the two studies are critically discussed and implications for rehabilitation based on integrated functional assessment are examined. Further, tentative findings on the effects of prognostic variables are reported. The implications of this study with regard to neuropsychological assessment and further research are discussed.

SECTION 1 : INTRODUCTION

Recently many studies on subarachnoid haemorrhage (SAH), concerning the outcome of surgery, have appeared in the literature (eg. Saveland et al, 1986; Brismar and Sundbarg, 1985; Ljunggren et al, 1985, 1982; Ropper & Zervas, 1984; Kassel and Torner, 1984; Shephard, 1983). These studies assess outcome primarily in broad medical terms (eg. mortality rate; incidence of neurological deficits). Some of these studies refer to cognitive and emotional/personality changes and Saveland et al (1986) suggest that "persistent cognitive and psychosocial disturbances" be taken into account for outcome evaluation. This aspect has been particularly emphasised in studies concentraing on patients with a good physical recovery. However, the cognitive, personality and emotional sequence of SAH have received relatively little in-depth attention. The few studies (eg. Steinman and Biggler, 1986; Sengupta et al, 1975; Storey 1972, 1970, 1967; Logue et al 1968) which have examined these factors hav done so in a broad spectrum of aneurysmal SAH patients. There appears to be only one study (Ljunggren et al, 1985) which has focussed specifically on the neurologically intact subgroup.

The relative paucity of studies focussing on patients with good neurological recovery after SAH is not surprising when the overall statistics for this disorder are considered. Eight percent of all cerebrovascular catastrophes are SAH (Lishman, 1978). Studies worldwide have shown that this amounts to between 4 and 23.9/100.000 per year (Ljunggren et al, 1987).

Studies have consistently shown that 60 to 70% of patients will eventually die from the disease; and approximately 20% will be disabled (Walton, 1971, in Lishman 1978; Ask-Upmark & and Ingvar, 1950, in Ljunggren et al, 1985). Ljunggren et al (1985) estimate that only 20% of patients will have the chance to become "functional survivors", while Walton (1971, in Lishman, 1978) found that approximately one-third of survivors (ie 10-15% of all patients) do not have any neurological symptoms. A recent estimate of the natural course of aneurysmal SAH is illustrated in Figure 1.

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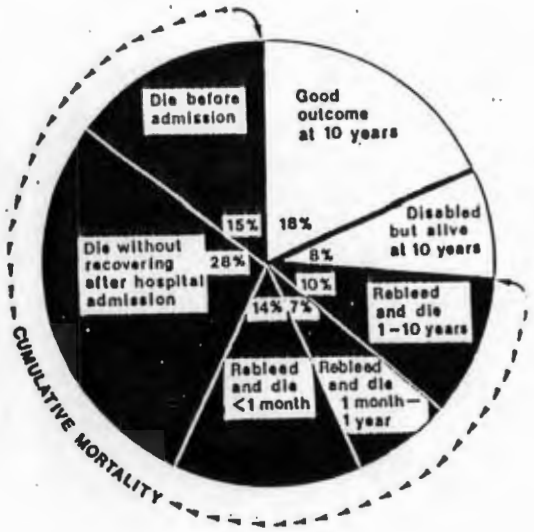

Fig 1 : NATURAL COURSE OF ANEURYSMAL SAH
(JENNETT & GALBRAITH, 1983; IN LJUNGGREN ET AL, 1987)

ANATOMY AND NEUROPATHOLOGY

Subarachnoid haemorhage is caused by the rupture of an intracranial aneurysm in 60% of cases and the greatest incidence is in the 40 to 60 years age group (Lishman, 1978). Most aneurysms arise close to the circle of Willis at the base of the brain. Common sites inclue the internal carotid artery, the middle cerebral artery and the anterior communicating artery. Storey (1970) summarised the anatomy and neuropathology of SAH in the following manner:

Internal Carotid Artery (ICA) Aneurysms

These form where the posterior communicating artery (PCoA) arises from the upper end of the internal carotid artery, and lie medial to the uncus of the temporal lobe. Haemorrhage may result in the formation of haematomas (mainly in the temporal lobe), and sometimes widespread and severe infarction (frequently in the territory supplied by the middle cerebral artery). Interference with the fine perforating vessels to the basal ganglia and hypothalamus also occurs.

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Middle Cerebral Artery (MCA) Aneurysms

These are found in the Sylvian fissure or lie embedded in the frontal or temporal lobes nearby. Haematomas commonly form in the Sylvian fissure, in the temporal lobe, or in the external capsular regions. Infarction is largely confined to the area supplied by the MCA.

Anterior Communicating Artery (ACoA) Aneurysms

These arteries are situated in the midline between the frontal lobes, near the anterior hypothalamus. Rupture may cause damage to nearby structures, resulting in haematomas in the frontal lobes and corpus callosum, or may cause infarction (mainly in the teritory supplied by the anterior cerebral artery (ACA)).

The literature provides support for both diffuse effects of SAH, and for localised effects at these three sites.

Localised Effects

The ICA and MCA aneurysms may both cause infarction in the teritory of the MCA and the sequelae may thus be similar. Such aneurysms are likely to produce a "hippocampal amnesia" ('pure forgetting' where cues do not improve recall) due to the destruction of the medial temporal areas (Walsh, 1978). Haemorrhage of anterior communicating artery aneurysms is however, likely to produce a 'frontal syndrome' with symptoms of adynamia and expressive aphasia. "Thalamic amnesia" (impaired retrieval which can be improved by means of cues) may result from ACoA aneurysms, since it is hypothesised that this form of amnesia results when temporal lobe circuits remain intact (Simpson, 1969, in Walsh, 1985). Walsh (1985) cites a recent study by Gade (1982) and argues strongly that, during surgery, entrapment of branches of the ACoA which supply midline structures is the probable basis for this amnestic disorder.

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Unlike more common forms of stroke (eg. emboli, thrombosis, or hypertensive haemorrhages), SAH does not necessarily result in strictly localised damage. Due to the fact that haemorrhaging may exert pressure on blood vessels in the subarachnoid space which are often not anatomically related to the aneurysm, it may not be possible to accurately predict the probable area of damage acording to the site of haemorrhage.

Localisation effects may also be observed by diffuse brain damage since SAH often results in raised intracranial pressure for hours or days. It has also been suggested that spasm of the aneurysm-bearing vessel, as well as possible propagated spasm of other branches of the circle of Willis, is responsible for lesions throughout the cerebral hemispheres, and that pressure from oedema of white matter may perpetuate the cortical ischaemia after the vasospasm has worn off (Smith, 1963).

NEUROPSYCHOLOGICAL SEQUELAE OF SAH

The term "neuropsychological" is defined in a broad sense to include emotional, personality and behavioural, as well as cognitive, features (Lezak, 1983).

Lishman (1978) noted that Walton (1952, 1956) was one of the first to emphasise the importance of the neuropsychological sequelae of SAH. There have been a number of further studies concerned with such sequelae. Most noteworthy are two major studies on a large number of patients. Storey (1972, 1970, 1967) followed up 261 patients six months to six years after SAH (81 ACoA; 71 MCA; 72 PCoA ie ICA) and Logue et al (1968) studied 79 patients six months to $8^{1}/2$ years after anterior cerebral aneurysms.

With regard to the cognitive sequelae, Storey (1967) judged 40% of his patients to be cognitively impaired (10% of which were rated moderate to severe) and noted that MCA aneurysms resulted in relatively greater impairment.

Logue et al (1968) found that 10% of his anterior cerebral (AC) aneurysm patients had global dementia, 40% made dysphasic errors, and that memory was more impaired than intelligence, overall. Amnestic syndromes are commonly reported after rupture of anterior communicating aneurysms (Steinman and Biggler, 1986; Okawa et al, 1980). These amnestic syndromes appear to be the result of focal damage, but may also be due to diffuse brain damage, for example as a result of hydrocephalus (Theander and Granholm, 1967, in Lishman, 1978).

In his review of the literature Lishman (1978) notes that the most common personality changes after SAH include loss of drive and vitality, decreased interest and initiative, withdrawal, irritability, emotional lability, anxiety and catastrophic reactions. Storey (1970) found that 46% of his subjects underwent personality changes (22% mild impairment, 15% moderate impairment, 4% severe impairment, 5% improvement) and that personality impairment was most common in patients with MCA aneurysms. Storey (1970) reported chronic shallow depression, boredom, loss of interest and fatigue, which he termed "organic moodiness".
Storey (1970) and Logue et al (1968) noted that anterior cerebral patients were described as uninhibited, less easily worried, and less irritable. This classic "frontal lobe syndrome" was also found by Steinman and Biggler (1986) who noted apathy in addition to the above symptoms. It has been consistently found that anterior cerebral artery SAH more often results in personality change without measurable intellectual impairment (Steinman and Biggler, 1986; Sengupta et al, 1975; Storey, 1970; Logue et al, 1968).
For all other sites of haemorrhage (besides ACoA) personality change is largely proportional to the incidence and severity of intellectual impairment and neurological disability, as indicated by central nervous system signs (Storey, 1970).

Symptons of anxiety and depression are also common. Storey (1970) found such symptoms in 25% of his patients (rated moderate and severe in 14% of these cases). Anxiety and

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depression generally correlated with the severity of brain damage, but in premorbidly neurotic personalities, was found to be disproportionately severe.
Depression was more persistent, severe, and more common in patients with PCoA (ICA) aneurysms, which Storey (1970) hypothesised to be due to haemorrhaging interfering with the fine perforating vessels to the hypothalamus.

Many studies (Brismar and Sundbarg, 1985; Esksen et al, 1984; Theander and Granholm, 1967, in Lishman, 1978) have also noted other symptoms such as fatigue, frequent headaches, vertigo and sensitivity to noise ("neurasthenic" symptoms). Such symptoms are often reported after cerebral trauma (Levin et al, 1979, Lishman, 1973) and may reflect diffuse brain damage.

The review of SAH sequelae may be summarised as follows:

1. In general there is a correlation between the severity of neurological disability, and the extent of intellectual impairment, personality change, and symptoms of anxiety and depression.
2. Most studies suggest that MCA aneurysms result in relatively more severe cognitive and personality impairment.
3. There is generally no clear relationship between site of haemorrhage and intellectual, personality or psychiatric sequelae.

4. Exceptions to the above general findings are:
   (a) Anterior (ACoA) aneurysms may result in personality change without intellectual impairment.
   (b) Anterior cerebral artery aneurysms also tend to cause greater impairment of memory than intelligence.
   (c) Depression may be more common, severe and persistent with ICA (PCoA) aneurysms.

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It should be noted that the above findings are not necessarily applicable to the neurologically intact sub-group as they are based ons tudies of patients with varying degrees of neurological deficit.

The first comprehensive study, focussing exclusively on patients who had made a good neurological recovery after aneurysmal SAH (and early operation), was conducted by Ljunggren et al (1985). They point out that neurosurgeons are familiar with the fact that some patients with a good neurological recovery ("functional survivors") demonstrate a diffuse encephalopathy with emotional or psychological disturbances, which may interfere with their reintegration in social and occupational environments.

Ljunggren et al (1985) randomly selected 40 patients without post-operative neurological deficits form a series of 160 patients who were classified preoperatively as neurological grades I, II and III (Hunt and Hess, 1968). The sample consisted of 15 patients with ACoA aneurysms, 13 with ICA aneurysms and 12 with MCA aneurysms. Patients were assessed 14 months to 7 years after SAH by means of a questionnaire, an interview and neuropsychological testing. The test battery consisted of the SRB : 1 (Swedish standard verbal intelligence scale), paired associates (immediate and 3 hour delay), Benton visual retention test, Graham Kendall memory for designs, block design test, Bourdon-Wiersma dot test, trail making test, and the Wisconsin card sorting test.

The results of the study were as follows:

1. Cognitive disturbances appeared to be permanent.
2. The degree of impairment appeared to correlate with the patient's age.
3. There was no obvious relationship between aneurysm location and quality of cognitive dysfunction.
4. Substantial post-haemorrhagic malajustment with regard to vitality, social management, self assertion, emotional

control, temperament, mood and cognitive abilities was found. They noted that due to psychological defense mechanisms these are easily overlooked.

5. Cognitive impairment : Only 1 patient had no cognitive impairment; 6 patients (15% of sample) were classified as mildly impaired (predominantly verbal learning and memory difficulties of a short-term or intermediate nature); 12 patients (30%) were classified moderately impaired (disturbed spatial organisation and visuo-constructive impairment in addition to the above impairments. There were also deficits in higher mental processes, eg abstraction and concept formation); and 21 patients (53%) had marked cognitive impairment. In this group, learning and memory difficulties (verbal and non-verbal), visuo-spatial disorganisation and defective conceptualization, reduced perceptual speed and accuracy, fluctuation in attention and task orientation, and lack of cognitive flexibility were common.

There are a number of important issues for future research which arise out of the Ljunggren et al (1985) paper. In their conclusion they note that the timing of surgery requires further study as early surgery may have enhanced the encephalopathy shown by patients in their sample. They also suggest that cognitive impairment is probably related to the severity of the initial bleed, but have not investigated this variable. Similarly, emotional 'adjustment' and personality changes were apparently not analysed in relation to the site of aneurysm. In addition, the methodology of the study has two potential weaknesses; the choice of tests in the neurospychological assessment battery does not appear to have a theoretical basis, which may obscure possible relationships between cognitive dysfunction and aneurysm location. Furthermore the wide variation in time between the SAH and assessment of sequelae (14 months to 7 years) has introduced an unnecessary confounding variable, since recovery from head injury may continue for 2 to 3 years or even longer

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(Trabaddor et al, 1984; Bond, 1976, Bond and Brooks, 1976; Lishman, 1973). Finally, this study does not consider the differential effects on outcome of other variables such as gender and educational level (Lezak, 1983).

The present study is aimed at furthering the knowledge about the neuropsychological sequelae of SAH in cases where there are no neurological deficits on neurological examination. This is particularly important since such patients are often given the option of returning to work relatively soon after the haemorrhage. Subtle cognitive, personality and/or emotional changes may, however, interfere with their reintegration at work and socially, and unrealistic expectations (based on the absence of neurological deficits) may result in unnecessary psychological distress (Boll, 1985). The greatest amount of recovery is experienced in the first sixt months after cerebral trauma (Bond, 1976) and patients are thus often assessed at this time with regard to prognosis and recommendations concerning return to work. The present patients were therefore assessed 6 to 8 months post-operatively, although improvement may theoretically still occur over the next 18 months, or beyond.

This study is a preliminary investigation into the cognitive, personality and emotional sequelae of SAH, in patients who have made a good neurological recovery. In the light of the studies reviewed above, the following factors will be investigated in terms of their possible effect on the prognosis of such patients:

1. location of aneurysm.
2. timing of surgery (acute versus delayed).
3. severity of the initial bleed (grade of aneurysm).
4. age of the patient.
5. gender.
6. educational level.

7. In addition, it is of particular importance in the South African context to consider premorbid cognitive functioning in order to interpret test scores not standardised on this population (Jacobs, 1985; Crawford-Nutt, 1977). Furthermore, Boll (1985) has pointed out that individuals from a poor socio-economic background have more marginal coping capacities and suffer significantly greater deficit from a similar injury.

SECTION 2 : METHOD

SUBJECTS

All patients treated for SAH at the neurosurgery department, Groote Schuur Hospital, Cape Town, from May 1986 to November 1986 and who were judged to be neurologically intact by the neurosurgeon at the routine medical follow-ups (2 weeks, 1 month, 2 months, 3 months and 6 months post-operatively), were selected for a 6-8 month post-operative neuropsychological follow-up assessment (mean 7.02 months; range 6.1 - 8.0 months). The opportunity sample consisted of 24 possible subjects, of which 6 could not be assessed because they did not return for follow up. Data from one subject is incomplete after she requested that the assessment process be spread over two days (due to fatigue), but later refused further assessment. The study is therefore based on 17 full protocols and one incomplete protocol.

Subjects ranged in age from 17 to 67 years (mean age 38.8 years) and had a mean education level of Standard 6 (range Standard 2-10, one subject with additional nursing diplomas).

APPARATUS AND PROCEDURE

(i) Structured Interview

An extensive (1-1^{1}/2 hour) interview consisting of open ended and closed questions was conducted with the

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patient and somebody who was familiar with the patient's everday functioning (eg. spouse). Numerous factors associated with cerebral insult were rated by the clinician (0 : does not apply; 1 : applies somewhat; 2 : definitely applies) once consensus between the patient and family member had been achieved. Ratings were applied to the present situation and to the premorbid circumstances as reported by the patient and significant other.

The factors which were tapped by the interview were drawn from a variety of sources (in particular Lezak, 1983; Walsh, 1985, 1978; Lishman, 1978) and are listed in Table 1.

TABLE I : CONTENT OF STRUCTURED INTERVIEW/QUESTIONNAIRE

1. Physical/neurological symtoms (including "neurasthenic" symptoms viz. headache, fatigue, dizziness, sensitivity to noise)	11. Elation 12. Anxiety 13. Obsessionality 14. Social withdrawal 15. Disinhibition 16. Diminished sensitivity & concern
2. Speech and language	17. Aggression
3. Memory	18. Rigidity
4. Attention & concentration	19. Paranoid/schizoid/ histrionic/dependent personality traits
5. Anergia	
6. Diminished performance	
7. Restlessness	
8. Emotional lability	20. Assertiveness
9. Lowered frustration tolerance	21. Childishness 22. Substance use
10. Depression	23. Sexual performance

(ii) Neuropsychological Testing

An extensive test battery was administered to the patient (duration $2^1/2$ to $3^1/2$ hours). Refreshments were provided during regular rest periods to prevent undue fatigue.

The researcher was blind as to the aneurysm site and grade, as well as the timing of surgery, until all the individual subjects' results had been calculated.

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The test battery was selected to enable analysis of results according to two complementary models:

(A) Cognitive Fall-Off Model

The first model is based on the analysis of the full SAWAIS (South African standardisation of the Wechsler Adult Intelligence Scale) to determine whether there has been a fall-off in cognitive functioning according to the deficit measurement method of Lezak (1983).

Cognitive deterioration was diagnosed by comparing present functioning to individual comparison standards derived from estimates of premorbid ability. Subtest scatter was analysed to determine whether significant deterioration in functioning had taken place. A discrepancy of 4 or more scaled score points was used to determine whether or not subtests were significantly depressed.

The best premorbid performance level was estimated from the highest subtest score; with the exception of digit span, Object assembly and Digit symbol, which are unreliable indicators of premorbid functioning. In addition, the estimate of premorbid functioning was moderated by data from the educational and occupation history.

(B) Specific Cognitive Ability Model

The second model falls within the neuropsychological approach which focusses on specific psychological content, such as attention, memory, language processes etc. (Boll, 1985). This is also known as the process or ability model (Wiig and Semel, 1980) which is based on the

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assumption that neurological dysfunction in one or more cognitive process will result in identifiable syndromes or deficit clusters. Thus this method of analysis requires the assessment of the efficacy of various components of cognitive functioning. Impairment in any one area of cognitive functioning is hypothesised to have a potentially disabling effect on the whole cognitive system despite all other abilities remaining intact (Saling, M; 1987 - personal communication).
The areas of cognitive fucntioning assessed in this study and the neuropsychological tests used, are illustrated in Table II.

Because patients were selected on the basis of having no neurological deficits, specific neurological deficits which are routinely screened in a neuropsychological examination (Saling, M.; 1987 - personal communication), were briefly assessed, in order to increase the stringency of the inclusion procedure. This was necessary in order to identify any neurological deficits that may have developed in the interim between the neurosurgeon's assessment and this one. In addition, impairment on this assessment could also indicate localised brain dysfunction.
The assessment covered the following (based on Luria's neuropsychological investigation (Christensen, 1984, 1983; and Lezak, 1983) : dysgraphia, dyslexia, dyscalculia, perseveration, conflict reaction, neglect, agnosia, aphasia, impaired tactile sensation, impaired right/left discrimination.

Both models of cognitive functioning, in terms of which the neuropsychological assessment was structured, fall

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TABLE II : AREAS OF COGNITIVE ABILITY ASSESSED AND NEUROPSYCHOLOGICAL TESTS USED

AREA OF COGNITIVE ABILITY	NEUROPSYCHOLOGICAL TESTS
I. General Cerebral Efficiency (TMT) (DS)	(a) Trail Making Test (Reitan, 1956) : test of visual-conceptual and visuomotor tracking involving motor speed and attention functions. Highly vunerable to brain injury (Lezak, 1983). (b) WAIS Digit Symbol Test (HSRC, 1983) : a symbol-substitution task involving motor persistence, sustained attention, response speed and visuomotor co-ordination. Sensitive to minimal, nonspecific brain damage (Lezak, 1983).
II. Planning ability and problem solving (CFT) (Mazes)	(a) Complex Figure Test (Rey, 1941) : a test of perceptual organisation sensitive to planning/programming errors in copying the figure (Lezak, 1983). Copying strategy (Osterreith, 1944, in Lezak, 1983) is a particularly important consideration. (b) WISC Mazes (Wechsler, 1974) : a test involving planning and foresight which is a clinically satisfactory substitute for the lengthier Porteus test (Lezak, 1983).
III. Immediate memory/ apprehension span (Digits) (Corsi)	(a) WAIS Digit Span Test (HSRC, 1983) : a test of short term verbal memory and passive apprehension span (Lezak, 1983). (b) Corsi Block Board Test (Milner, 1971) : a test of figural short term memory.
IV. Recent memory (Ass L) (Log M) (Vis R)	I. <u>Immediate Recall</u>: Wechsler Memory Scale (WMS) - Form 1 Wechsler, 1945) : (a) WMS Associate Learning test : a test of verbal retention where the patient has 3 trials to learn 10 pairs of words presented auditorily. (b) WMS Logical Memory test : a test of immediate memory of simple stories presented auditorily. (c) WMS Visual Reproduction test : a test requiring reproduction of geometric patterns immediately after a 10 second fixation period. II. <u>Delayed Recall</u> Tests (a) (b) (c) were repeated after 30 minutes without warning the patient beforehand (Russel, 1975; Brooks, 1972). (Ass LD; Log MD; Vis RD)
V. New Learning (Dss) (Css) (Hard Assns)	(a) Digit supraspan : this test allows a maximum of 12 trials to learn a series of digits one item longer than the patient's maximum WAIS digit span. (b) Corsi block supraspan : this test is a figural equivalent to Dss - the patient is required to learn a sequence of blocks which is one item longer than his natural span. (c) Ass L Hard Associations : A subsection of the Ass L. Test which tests retention of unfamiliar verbal material, indicating the process of learning over 3 trials.
VI. Manual dexterity (Purdue)	(a) Purdue Pegboard Test (Lezak, 1983) : a timed test of motor dexterity.

within the psychometric approach as opposed to the neurological approach (as exemplified by Luria, 1973). The advantage of the psychometric approach is its use of interval scales of measurement rather than the nominal types of tests of the neurological approach (ie the psychometric approach measures degree of impairment as opposed to pass/fail criteria of the neurological approach). As association area functions are impaired in a graded manner, interval scales are more suitable, because they are able to measure degrees of brain dysfunction (Russell, 1981).

(iii) Analysis of Results

1. Results of the Structured Interview

1.1 Total Sample

The total sample was analysed, initially, in terms of the amount of symptomatic change due to SAH. Patients were rated on the degree of emotional, behavioural and physical symptoms (see Table I) present pre-operatively, and 6-8 months post-operatively, on the basis of consensus established between the clinician, patient and significant other, in the interview. From this the amount of change due to SAH was then determined for each individual on the following scale : 0 : no change; 1 : some/slight change; 2 : definite/clear change.

Thereafter the total sample was analysed in terms of the severity of symptom clusters which resulted from SAH. This was necessary because the severity of symptoms elicited in the questionnaire is not reflected in the above analysis. For example, someone with premorbid depression which was excacerbated by the SAH would have been rated as having a slight increase

(rated 1). This rating would not reflect the fact that depression may now be seriously incapacitating in terms of post-operative functional level. Severity of symptoms was therefore judged by the clinician and rated on a 3 point scale (1 slight; 2 moderate; 3 severe). Questionnaire data was then grouped into clusters of symptoms which tended to occur simultaneously, and whose precise constribution to functional impairment was difficult to quantify. The incidence of these symptom-clusters was then calculated for the total sample.

Occupation status 6-8 months post-operatively was examined separately to allow a more detailed analysis of this important aspect of recovery.

1.2 Subgroup Analysis

Incidence of symptoms and mean ratings of impairment on each symptom, were examined in terms of the subgroup into which the sample was successively subdivided (Table III, below).

TABLE III : VARIABLES INVESTIGATED WITH REGARD TO PROGNOSIS

VARIABLE	DISTRIBUTION
(1) ANEURYSM SITE	ICA (N = 12); MCA (N = 3); ACoA (N = 3)
(2) TIMING OF SURGERY	acute (mean 2.8 days; range 2-4 days) (N=6) delayed select (x 8.4 days; range 7-10 days) (N=5) delayed (poor grade) (x 17.0 days; range 10-26 days) (N=6).
(3) ANEURYSM GRADE	grade 1 (N = 6); grade 2 (N = 8); grade 3 (N = 1); grade 4 (N = 2).
(4) AGE	(i) 3 groups : 17-35 years (x 25) (N = 7); 36-45 years (x 39.2) (N = 6); 46 years or more (x 54.8) (N = 5). (ii) 2 groups : 35 years (x 26) (N = 7); 35 years (x 47) (N = 11).
(5) EDUCATION	Standards 1-5 (N = 7); Std 6 & 7 (N = 6); Std 8-10 (N = 5).
(6) GENDER	Males (N = 6); Females (N = 12).
(7) PREMORBID COGNITIVE FUNCTIONING	IQ estimate high average (110+) (N = 4); average (90+) (N=10); low average (80+) (N=3).

1.3 Individual Analysis

Because group analysis may obscure the presence of specific syndromes which could be anticipated from the literature, an individual analysis was carried out to determine to what extent the sequelae manifested were commensurate with the symptoms expected due to aneurysm location.

2. Results of the Neuropsychological Evaluation

Individual test results were analysed according to the most applicable neuropsychological norms, and the data was transformed into standard (2) scores to facilitate comparison between disparate test scores.

2.1 Total Sample

The total sample was analysed in terms of the Cognitive Deterioration and the Functional Impairment models outlined above.

2.2 Subgroup Analysis

Neuropsychological test scores were analysed in terms of the factors which may affect prognosis after SAH (see Table III). Due to the small sample size (17 full protocols, 1 incomplete protocol) and the large number of predictive variables (7), neither parametric nor non-parametric statistical tests could be used. Descriptive techniques were therefore utilised in order to provide preliminary hypotheses to inform future research.

Subgroup means were calculated and illustrated graphically on a computer graphics package (Lotus,

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1985). Tests on which the mean scores of the subgroups differed by more than 1 z score were analysed more closely. The range of subgroup scores, the incidence of impaired scores within subgroups, and the "percentage forgetting" (Brooks, 1972) scores on the Wechsler Memory Scale tests, were computed. Trends reported in this study are therefore more reliable than if based on subgroup mean scores only.

3. General Functional Outcome

The results of the interview and the neuropsychological evaluation were integrated in terms of a measure of general functional impairment.

The concept of functional outcome or recovery refers to the extent to which a patient has been restored to his/her premorbid physical, cognitive, social and occupational status (Artiola et al, 1981, in Ljunggren et al, 1987; Bond, 1976). A rating of functional impairment may thus be made on the grounds of any combination of cognitive, emotional, personality or behavioural sequelae.

Subjects were rated by the clinician as having (i) Minimal impairment (impairment detected on testing only, normal functioning), (ii) Slight impairment (impairment or change noticed by the patient or significant others, but little change in functional ability), (iii) Moderate impairment (definite impairment of functional ability, but not incapacitating), and (iv) Severe impairment (serious functional incapacity). The symptoms of patients within each impairment group were tabulated and comparative descriptive profiles of each category of impairment were developed.

SECTION 3 : RESULTS

1. RESULTS OF THE STRUCTURED INTERVIEW

1.1 Total Sample

1.1.1 Amount of Change due to SAH

The amount of change and most common areas of impairment which emerged from an analysis of the total sample (N = 18), are displayed in Table IV. Symptoms that appeared in less than 20% of the sample are not included in the table.

TABLE IV DEGREE OF SYMPTOMATIC CHANGE DUE TO SAH, RANKED IN ORDER OF INCIDENCE. (THE FIGURES IN PARENTHESIS INDICATE THE NUMBER OF SUBJECTS RANKING THE SYMPTOM AS 1 : SLIGHT CHANGE; AND AS 2 : CLEAR CHANGE, RESPECTIVELY

SYMPTOM	NUMBER OF CASES	% OF TOTAL SAMPLE	SYMPTOM	NUMBER OF CASES	% OF TOTAL SAMPLE
1. Fatigue	14(5;9)	78%	13. Impaired hearing	6(2;4)	33%
2. Lowered frustration tolerance	12(6;6)	67%	14. Incoordination/ poor balance	6(3;3)	33%
3. Depression	12(11;1)	67%	15. Headache	6(4;2)	33%
4. Diminished performance	10(4;6)	56%	16. Somatic symptoms	6(6;0)	33%
5. Attention and concentration	10(7;3)	56%	17. Sensitivity to noise	5(2;3)	28%
6. Anxiety	10(8;2)	56%	18. Sensitivity to bright light	5(3;2)	28%
7. Impaired memory	9(4;5)	50%	Dizziness	5(3;2)	28%
8. Impaired sight	9(6;3)	50%	Hallucinations	5(3;2)	28%
9. Anergia	8(3;5)	44%	Impaired speech and language	5(3;2)	28%
Emotional lability	8(3;5)	44%	Aggression	5(3;2)	28%
11. Social withdrawal	8(4;4)	44%	23. Weakness of limbs	4(1;3)	22%
12. Impaired sexual performance	6(2;4)	43%*	24. Increased substance use	4(3;1)	22%

* 4 subjects were excluded since they were celibate 6/14 = 43%

1.1.2 Severity of Symptoms due to SAH

The severity of functional impairment was related to clusters of symptoms which occurred together in various combinations.

The incidence of these symptom clusters is displayed in Table V.

TABLE V SEVERITY OF IMPAIRMENT RATINGS, RANKED IN ORDER OF INCIDENCE (THE FIGURES IN PARENTHESIS INDICATE THE NUMBER OF CASES RATED AS HAVING SLIGHT, MODERATE AND SEVERE IMPAIRMENT, RESPECTIVELY).

SYMPTOMS	NUMBER OF CASES	PERCENTAGE TOTAL SAMPLE
1. Physicial, and/or neurological, and/or 'neurasthenic' symptoms	15(5;9;1)	83%
2. Lowered frustration tolerance, and/or aggression	12(3;9;0)	67%
3. Anxiety	12(5;5;2)	67%
4. Depression	12(9;2;1)	67%
5. Memory and/or attention and concentration impairment	11(5;4;2)	61%
6. Emotional lability	10(2;8;0)	56%
7. Restlessness and/or frenetic activity, and/or overtalkative, and/or elation	9(6;3;0)	50%
8. Anergia	8(3;3;2)	44%
9. Social withdrawal	8(4;4;0)	44%
10. Obsessionality	7(3;4;0)	39%
11. Speech and language impairment	5(2;2;1)	28%
12. Disinhibition and/or childishness	4(1;3;0)	22%

1.1.3 Occupational Status 6-8 Months Post-Operatively

Fourteen subjects (78%) returned to work, three subjects (17%) changed their occupations and one subject remained unemployed. Table VI indicates the occupations and impairment ratings of the total sample, 6-8 months post-operatively.

(i) Four subjects returnted to their former jobs, and continued to function on their premorbid level.

(ii) Ten subjects returned to their former occupations, but all evidence some impairment in functioning (5 slight eg. due to fatigue, and 5 moderate).

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(iii) Three subjects were unable to cope with former job requirements and are now "housewives". All 3, however, have difficulty in performing domestic tasks in comparison to premorbidly (2 slightly, 1 moderately).

(iv) One patient was unemployed for 1 year prior to the SAH and remains so. He is judged to be moderately impaired, however, and would therefore have difficulty in coping with previous occupational demands (bricklaying).

TABLE VI : OCCUPATIONAL STATUS 6-8 MONTHS POST-OPERATIVELY

	SUBJECT	GENDER	AGE	PREVIOUS OCCUPATION	PRESENT OCCUPATION	IMPAIRMENT RATING
(i)	C	F	19	Data Operator	Data Operator	0
	F	F	54	Housewife	Housewife	0
	M	M	46	Electrical Faultsman	Electrical Faultsman	0
	Q	M	43	Clerk	Clerk	0
(ii)	A	F	39	Nursing Sister	Nursing Sister	1
	D	M	17	Welder	Welder	1
	G	F	29	Housewife	Housewife	1
	I	F	26	Clerk/Internal Auditor	Clerk/Internal Auditor	1
	R	F	41	Housewife	Housewife	1
	E	F	47	Housewife	Housewife	2
	J	M	29	Semi-skilled Electrician/ Driver	Semi-skilled Electrician/ Driver	2
	K	M	32	Labourer	Labourer	2
	L	F	39	Housewife	Housewife	2
	N	F	39	Domestic Servant	Domestic Servant	2
(iii)	B	F	30	Shop Assistant	Housewife	1
	O	F	67	Domestic Servant	Housewife	1
	H	F	42	Shop Assistant	Housewife	2
(iv)	P	M	60	Unemployed Bricklayer	Unemployed Bricklayer	2

1.2 Subgroup Analysis

There were no significant differences between the subgroups in terms of the prognostic variables by which the sample was successively subdivided.

1.3 Individual Analysis

Clear differences were not found between ACA aneurysm patients and ICA or MCA aneurysm patients and the hypothesis that ACA aneurysm patients would present a classical "frontal lobe syndrome" was not supported. Anterior cerebral artery aneurysm patients exhibited a variety of combinations of the expected symptoms (eg. disinhibition, impulsivity, anergia, poor error evaluation), but many were also emotionally labile, had lowered frustration tolerance and were more aggressive - features which were anticipated for the ICA and MCA aneurysm patients. Similarly, many of the ICA and MCA aneurysm patients exhibited anergia, poor planning, poor error evaluation and perseveration - symptoms which were expected in the ACA aneurysm patients.

2. RESULTS OF THE NEUROPYSCHOLOGICAL EXAMINATION

2.1 Total Sample

2.1.1 Cognitive Deterioration Model

The mean cognitive fall-off of the total sample was 19.7 IQ points (mean premorbid IQ estimate 98.8, mean IQ on testing 79.1). the estimates of cognitive fall-off ranged from 5.5 to 28 IQ points, with 4 subjects having a fall-off of less than 15 points, 4 a fall-off of between 15 and 20 points, 7 between 20 and 25 and 3 between 25 and 30 points (see Table VII).

TABLE VII : COGNITIVE DETERIORATION

SUBJECT	ESTIMATED COGNITIVE DETERIORATION (IQ POINTS)	SUBJECT	ESTIMATED COGTNITIVE DETERIORATION (IQ POINTS)
K	28	B	21
L	28	J	17
F	28	A	17
R	24.5	G	17
N	24	D	15.5
I	23.5	H	14
O	21.5	C	13.5
P	21	Q	13.5
E	21	M	5.5
		MEAN	19.7

The results of the analysis of WAIS subtest scatter is illustrated in Table VIII. The number of subtests that were indicative of dysfunction (3 point discrepancy), but which did not reach statistical significance, is reported in parenthesis.

TABLE VIII : INCIDENCE OF SIGNIFICANTLY IMPAIRED SUBTESTS

	NUMBER OF SUBJECTS SIGNIFI-CANTLY IMPAIRED 4+ DIFFERENCE	INDICATIVE 3+ DIFFERENCE		NUMBER OF SUBJECTS SIFNIFI-CANTLY IMPAIRED 4+ DIFFERENCE	INDICATIVE 3+ DIFFERENCE
Information	6	(1)	Picture Comp	0	(2)
Comprehension	2	(1)	Object Ass	5	(4)
Arithmetic	3	(2)	Block Design	2	(3)
Digit span	0		Digit Symbol	2	(3)
Similarities	1	(4)	Picture Arr	0	

2.1.2 Specific Cognitive Ability Model

General cerebral efficiency was impaired in 8 subjects, planning ability and problem solving in 7 subjects, immediate memory in 4 subjects, recent

memory in 12 subjects (10 impaired on immediate recall and 12 on delayed recall), 8 impaired on new learning, and manual dexterity in 4 patients.

Deficits on the various tests as conceived within the model of cognitive abilities are illustrated in Table IX, which lists the areas of impaired ability in order of severity. (Tests are also ranked within each area to indicate relative sensitivity to impairment).

Process analysis of the complex figure test revealed that 8 subjects used a type I strategy, 1 subject used a type II strategy, 1 a type III strategy, 5 type IV, and 2 type V strategies. Type IV and V strategies were correlated with impaired accuracy scores in 2 cases.

Screening for neurological deficits revealed 2 subjects with slight impairment in tactile sensation, while perseveration was noted on certain cognitive tests, in 7 cases.

TABLE IX : IMPAIRMENT OF TESTS WITHIN THE SPECIFIC COGNITIVE ABILITY MODEL

COGNITIVE ABILITY	NUMBER OF SUBJECTS IMPAIRED		DISTRIBUTION OF IMPAIRMENT ON TESTS
(1) Recent Memory	12	(71%)*	
(a) 30' delayed recall	12		
Visual reproduction delayed	11		1 impaired on all 3 tests; 3 on vis RD and Log MD; 1 on Vis RD and Ass LD; 6 on Vis RD only; 1 on Log MD only
Logical memory delayed	5		
Associate learning delayed	2		
(b) Immediate Recall	10		2 impaired on all 3 tests; 1 on Vis R & Ass L; 1 on Vis R & Log M; 4 on Vis R only; 2 on Ass L only
Visual reproduction	8		
Associate learning	5		
Logical memory	3		
(2) General Cerebral Efficiency	8	(44%)	
Digit symbol	6		6 impaired on both; 1 on DS only; 1 on TMT only
Trail making test	6*		
(2) New Learning	8	(44%)	
WMS hard associations	4*		2 impaired on hard assns & Corsi ss; 2 on hard assns only; 2 on Corsi ss only; 2 on Digit ss only
Corsi supra-span	4*		
Digit supra-span	2		
(4) Planning ability & problem solving	7	(41%)*	
Complete figure test (Copy)	7		4 impaired on both; 3 on CFT only
WISC Mazes	4		
(5) Immediate memory	4	(22%)	
Digit span	3		1 impaired on both; 2 on digit span only; 1 on block span only
Corsi block span	2		
(6) Manual dexterity	4	(24%)*	
Purdue pegboard	4		

* N = 17:1 protocol incomplete

2.2 Subgroup Analysis

Analysis of neuropsychological tests scors in terms of (Table III), revealed that certain tests were sensitive to particular prognostic variables. Some of

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the findings, however, may not necessarily be explained in terms of one parameter only, but appear to be the result of interaction between several variables.

This interaction may be summarised as follows:

1. The Standard 8-10 education group is correlated with the high average premorbid IQ group, and both are correlated with a better preoperative grade and a younger age.
2. The 45+ age group is correlated with the lower grade (3 + 4) group, and both are correlated with a lower education level and a lower estimate of premorbid IQ.

The results of the subgroup analysis are displayed in Table X:

TABLE X : RESULTS OF SUBGROUP ANALYSIS

PROGNOSTIC VARIABLE	TEST RESULTS
1) Aneurysm site	1) ICA aneurysm group of patients less impaired on Ass L, Log MD.
2) Timing of surgery	1) Delayed (select) patients superior on Ass LD.
3) Aneurysm grade	1) Grade 3/4 patients are more impaired on Ass L, Log MD.
4) Age	1) Younger subjects (age 35) are superior to older subjects on DS, Mazes, CFT. 2) Older of 3 groups (age 46+) are more impaired on Ass L, Ass LD, Log M, Log MD.
5) Education	1) Std 8-10 patients superior on DS, TMT(B), Ass L, Ass LD, Corsi ss. 2) Std 8-10 group substantially higher than the Std 1-5 group on Vis RD, Log M. 3) Std 6-7 group score substantially lower on Mazes.
6) Gender	1) Males' performance is significantly superior on Mazes.
7) Premorbid IQ	1) Low average premorbid IQ patients significantly more impaired on CFT; Ass L; Ass LD; Log M; Log MD; D. 2) High average substantially higher on Ds; Mazes; Ass L. 3) Low average substantially worse than high average on TMT(B); Vis R; Vis RD.

Taking the interaction between variables into consideration, the main findings may be summarised as follows:

(1) Aneurysm Site: ICA patients apeear to be less impaired on Ass L and Log Md. It must be noted that the educational levels of this group (mean education Standard 6.5 are higher than the MCA and ACoA groups (both have a mean education of Standard 5.3) and thus this finding must be viewed with caution.

(2) Timing of Surgery: Delayed (select) patients are superior to acute and delayed (poor grade) patients on Ass LD. This finding, too, must be viewed with caution due to the possible effect of educational differences (delayed selected mean education Standard 7.2 as opposed to the other groups Standard 6.0).

(3) Gender: Males are superior to females on Mazes.

(4) Younger subjects are superior on DS, Mazes, CFT.

(5) The older age group (46+ years) which has a worse grade, lower education and lower premorbid IQ is particularly impaired on Ass L and Log MD, but also on Ass LD and Log M.

(6) The higher education group (Standard 8-10) achieved superior scores on DS, TMT (B), Ass L, Ass LD, Vis RD, Log M, Corsi ss. It should be noted, however, that this group is younger and has a higher premorbid IQ.

(7) The low average premorbid IQ group has particularly low scores on CFT, Ass L, Ass LD, Log M, Log MD.

3. INTERACTION OF RESULTS IN TERMS OF GENERAL FUNCTIONAL OUTCOME

The incidence and symptomatology profiles of impaired general functioning (see Table XI) were as follows:

(a) Minimal impairment was defined as impairment detected on testing only, but with normal functioning. Two subjects (11%), with a mean estimated fall-off in IQ of 9.5 points, were classified in this category. One subject reported slight fatigue, and some impulsivity was noted on testing.

(b) Slight impairment was defined as impairment or change noticed by the patient or others (eg. family), but with little change in functional ability. All 5 patients in this group reported 'neurasthenic'/physical problems (fatigue, headache, problems with hearing and sight), anxiety and slight depression. There is also a high incidence of lowered frustration tolerance, social withdrawal and obsessionality. Recent memory was impaired in over half of the subjects, and was particularly impaired on the 30 minute delayed recall adminstration (4 subjects (80%) were impaired).

(c) Moderate impairment was defined as definite impairment of functional ability, but not incapacitating (7 patients). Symptoms noted in the previous subgroup are more severe, in particular the neurasthenic/physical problems (fatigue, headache and dizziness, mental and physical slowing) and lowered frustration tolerance (in 86% of subjects). In addition, restlessness was reported by 5 patients (71%) and memory and/or attention and concentration problems and emotional lability were each reported by 4 subjects (57%). On testing recent memory (immediate and delayed) was impaired in four patients, as was information processing and new learning.

(d) Severe impairment, which was defined as serious incapacitation of functioning, occurred in 4 patients. Previously mentioned symptoms were generally more severe. However, the most seriously incapacitating symptoms for this subgroup were anergia (1 slight, 1 moderate, 2 severely impaired), memory and/or attention and concentration problems (1 slight, 1 moderate, 2 severe), and neurasthenic/physical problems. The neurasthenic/physical problems of these subjects more frequently included incoordination, weakness, somatic symptoms, and sensitivity to noise and bright light.
On testing recent memory (immediate and delayed), planning and new learning were the most impaired ($^3/_4$ cases). Information processing, immediate memory and manual dexterity were also impaired in half the subjects.

TABLE XI : INTEGRATION OF RESULTS IN TERMS OF FUNCTIONAL IMPAIRMENT

INTERVIEW AND OBSERVATIONAL DATA: Ranked in order of incidence (The figures in parenthesis indicate the number of cases rated as having slight, moderate and severe impairment, respectively).	NEUROPSYCHOLOGICAL TEST BATTERY DATA: Impaired cognitive functions ranked in order of incidence.
1. MINIMAL IMPAIRMENT (N=2;11%) Fatigue & impulsivity (N=1)	+ 10 IQ points fall-off
2. SLIGHT IMPAIRMENT (N=5;28%) NUMBER OF CASES (1) Anxiety 5(2;2;1) (2) Phys/neurol/ neurasthenic (fatigue, headache, hearing sight) 5(2;3;0) (3) Depression 5(5;0;0) (4) Lowered frustration tolerance/ aggression 4(0;4;0) (5) Social withdrawal 4(2;2;0) (6) Obsessionality 4(3;1;0) (Symptons occurring only once are not reported)	Mean IQ fall-off : 21.3 points COGNITIVE FUNCTION — NUMBER OF SUBJECTS IMPAIRED (1) Recent Memory : 4 (a) Delayed recall : 4 (1 impaired on Vis RD & Log MD 2 on Vis RD only, 1 on Log MD only) (b) Immediate recall : 3 (2 on Ass L only; 1 on Vis R only) (2) General Cerebral Efficiency : 2 (3) Planning and Problem Solving : 1 Immediate memory : 1 Manual dexterity : 1

3. MODERATE IMPAIRMENT (N=7;39%)	NUMBER OF CASES
(1) Phys/neurol./ neurasthenic (fatigue, headache, dizzy, sight)	6(3;3;0)
(2) Lowered frustration tolerance/ aggression	6(2;4;0)
(3) Restlessness/ overtalkative/ elation	5(4;1;0)
(4) Depression	4(2;1;0)
(5) Anxiety	4(2;2;0)
Memory/attention & conc.	4(2;2;0)
Emotional lability/oversensitive	4(2;2;0)
(8) Anergia (Symptons in 2 or less subjects are not reported)	3(1;2;0)

Mean IQ fall-off : 21.1 points

COGNITIVE FUNCTION		NUMBER OF SUBJECTS IMPAIRED
(1) Recent Memory	: 4	
(a) Immediate Recall	: 4	(1 impaired on Vis R & Log M; 1 on Vis R & Ass L; 2 on Vis R only)
(b) Delayed Recall	: 4	(1 on Vis RD & Log MD; 3 on Vis RD only)°
General Cerebral Efficiency	: 4	
New Learning	: 4	
(4) Planning and Problem Solving	: 3	
Immediate memory	: 1	
Manual dexterity	: 1	

4. SEVERE IMPAIRMENT (N=4;22)	NUMBER OF CASES
(1) Anergia	4(1;1;2)
Memory/attention & conc.	4(1;1;2)
(3) Phys/neurol./ neurosthenic (fatigue, incoordination, weakness, somatic sympt, sensitive to noise & bright light, hearing, sight)	4(3;1;0)
(4) Anxiety	3(1;1;1)
(5) Emotional lability/oversensitive	3(1;2;0)
Social withdrawal	3(1;2 0)
(7) Restlessness/ overtalkative/ elation	3(2;1;0)
Depression	3(2;1;0)
(9) Obsessionality	2(0;2;0)
Lowered frustration tolerance/ aggression	2(0;2;0)
10)Speech and language	2(1;1;0)
(11)Disinhibition/ Childishness	2(1;1;0)

Mean IQ fall-off : 20 points

COGNITIVE FUNCTION		NUMBER OF SUBJECTS IMPAIRED
(1) Recent Memory	: 3	
(a) Immediate Recall	: 3	(2 impaired on Ass L, Vis R, Log M; 1 on Vis R only)
(b) Delayed Recall	: 3	(1 on Ass LD, Vis RD, Log MD; 1 on Ass LD, Vis RD; 1 on Vis RD, Log MD)
Planning & Problem Solving	: 3	
New Learning	: 3	
(4) General Cerebral Efficiency	: 2	
Immediate memory	: 2	
Manual dexterity	: 2	

SECTION 4 : DISCUSSION

This study supports the contention of Ljunggren et al (1985) that the sequelae of SAH in patients without neurological deficits are in danger of being underestimated and overlooked during routine medical follow-up. The results indicate that all 18 patients were impaired in one or more neuropsychological aspects (cognitive, emotional, personality or behavioural) despite being judged as neurologically intact. At the 6-8 month post-operative assessment 14 patients (78% of the sample) were found to be impaired in occupational functioning, and 67% of patients reported symptoms of emotional distress. In addition, 11 patients (61%) reported personality changes - 7 patients were now more difficult to live with (personality deterioration) and 4 reported less anxiety and restlessness, increased tolerance and decreased sensitivity to criticism, all of which were considered to be "improved" personality features.

All patients were rated as having some cognitive impairment and 14 (78%) had a fall-off of 15 IQ points or more (mean cognitive deterioration 19.7 points) from estimated premorbid functioning. Recent memory was the most impaired cognitive function on neuropsychological assessment (71% of patients) and had been reported as impaired by 50% of the subjects during the interview. General cerebral efficiency and new learning were impaired in 44%, and planning and problem solving was impaired in 41%, of the sample. An overall functional assessment, which takes into account physical, cognitive, social and occupational impairment, 16 patients (89%) had noticed changes, ascribed to SAH, and 11 (61%) reported definite impairment of functional ability.

Investigation of the nature of sequelae following aneurysms at particular locations revealed that, despite a tentative finding which will be discussed later, subjects generally exhibited a homogeneous set of sequelae. There were no clearcut localised effects on the group- nor the detailed

individual- analyses, and the nature of the cognitive, emotional, personality and behavioural sequelae appear to reflect a diffuse SAH-induced encephalopathy. The neuropsychological sequelae of diffuse brain dysfunction have been studied largely in relation to closed head injury (Lezak, 1983). Sequelae of such diffuse damage usually include impairment of memory, attention and concentration, higher level functions such as complex reasoning, and general response slowing (Lezak, 1983). In the present study 61% of the sample reported impairment of memory and/or attention and concentration, and on testing 71% of the sample was found to be impaired on recent memory. Higher intellectual functions such as planning ability and problem solving, as well as tests which detect impaired motor speed and attention (especially well known for their sensitivity to nonspecific brain damage), were impaired in over 40% of patients.

Lezak (1983) also points out that diffuse brain damage is often not only reflected in the patient's inability to concentrate or perform complex mental operations, but also in complaints of irritability, fatigue, confusion and perplexity, distractibility and an inability to do things as well as premorbidly. In this study a number of patients complained of the above symptoms, but an analysis of the reasons for work impairment revealed that only 3 patients (17%) were definitely impaired due to deficits in concentration or complex mental process (eg. an electrician who had difficulty in positioning lights in a room so as to ensure maximum illumination, a skill that he previously performed "without thinking"). Most subjects (N = 10; 56%) were impaired in work performance largely because of physical/neurasthenic problems (especially fatigue). This finding may, in part, be ascribed to the manual nature of many patients' work (physical rather than mental fatigue would be experienced). It is also possible that the high incidence of fatigue and other physical/neurasthenic symptoms

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reflects the relatively short convalescence time (6-8 months).

Although it is possible that physical/neurasthenic symptoms of this nature could decrease substantially over time, the deficits found in the present study could also reflect chronic brain damage as fatigue, temper outbursts and poor memory are characteristic features of chronic brain damage (Lezak, 1983). The extent to which further improvement may be expected beyond the 6 to 8 month post-operative period may be tentatively inferred from a comparison of the present results with those of Ljunggren et al (1985) who also assessed neurologically intact SAH patients, but at an average time of $3^1/2$ years post-operatively (range 14 months to 7 years). Fifty-eight percent of Ljunggren et al's (1985) sample reported fatigue while 50% reported a decline in activity level and energy investment (lack of initiative, loss of interest in former activities, etc.). Comparative figures in this study (78% reported fatigue, 44% anergia) suggest that, although there may be a slight improvement in time, these complaints are probably chronic. Other comparative statistics (see Table XII) also indicate that most symptoms reported in the present sample (6-8 months post-op) were not strikingly more severe than those reported by Ljunggren et al (1985) ($3^1/2$ years post-op), which suggests that there may be a <u>limited</u> improvement over time and thus implies a substantial proportion of chronic symptoms. It is likely that the chronic symptoms are the direct effect of the diffuse SAH-induced encephalopathy.

In contrast the symptoms which appear to improve substantially over time, viz. depression, lowered frustration tolerance/aggression, emotional lability/oversensitive (see table XII), may reflect a large contribution of indirect (psychological) effects (ie. secondary reactions to impairment and loss, Lezak, 1983). Bond and Brooks (1976) argue that the greater part of recovery of physical and mental functions occurs within 6 months of severe brain

injury, and that later recovery primarily consists of adaptation to fixed mental and physical deficits. Walsh (1985) also believes that symptoms such as depression, anxiety and irritability are to a large degree reactive rather than a product of specific neural damage.

TABLE XII : INCIDENCE OF SYMPTOMS AS REPORTED BY LJUNGGREN ET AL (1985) AND IN THE PRESENT STUDY

LJUNGGREN ET AL (1985) DESCRIPTION OF SYMPTOMS	INCIDENCE % OF SAMPLE	BURBACH (1987) DESCRIPTION OF SYMPTOMS	INCIDENCE % OF SAMPLE	DIFFERENCE
1. Irritability/ hostility/weakened emotional control	35%	Lowered frustration tolerance/aggression	67%	32
2. Anxiety/tension/ undue concern and apprehension	50%	Anxiety	56%	6
3. Vulnerability and disproportionate sensitivity	28%	Emotional lability/ oversensitive	56%	28
4. Recurrent dysphoria and depressive mood swings	30%	Depression	67%	37
5. Emotional adjustment (symptoms 1-4 above)	75%	Symtoms 1-4	78%	3
"Fundamental changes reported" in 1-4	45%	"Definite changes (rating 2) reported"	39%	6
6. Fatigue	58%	Fatigue	78%	20
7. Decline in activity level and energy investment	50%	Anergia	44%	6
8. Decreased libido	20%	Sexual dysfunction	43%	23
9. "Changes in social demeanor"	50%	Social withdrawal	44%	6

It is interesting to note that despite differences in the samples (size and demographic variables) and in the test batteries used, results of the cognitive assessments, when

compared in terms of dimensions of intellectual ability, were remarkably similar in both studies (see Table XIII). However, in the Ljunggren et al (1985) study cognitive sequelae are viewed in isolation, which results in a somewhat restricted reflection of the degree of disability found in neurologically-intact patients after SAH. The present study is based on a wholistic approach to prognosis in that overall functional outcome in terms of physical, social, occupational and emotional status, in addition to cognition, was used to classify patients. The consequent impairment profiles encompass a greater diversity of neuropsychological sequelae and are thus clinically more useful.

TABLE XIII : INCIDENCE OF COGNITIVE IMPAIRMENT REPORTED BY LJUNGGREN ET AL (1985) AND IN THE PRESENT STUDY

LJUNGGREN ET AL (1985) COGNITIVE ABILITIES IMPAIRED	INCIDENCE % OF SAMPLE IMPAIRED	BURBACH (1987) COGNITIVE ABILITIES IMPAIRED	INCIDENCE % OF SAMPLE IMPAIRED
1. Learning and memory	80%+*	Recent memory or new Learning	82%
		Recent memory	71%
		New Learning	44%
2. Both verbal and non-verbal/spatial memory	38%	Both verbal and non-verbal memory	35%
Verbal memory only (CMI, CMII)	20%*	Verbal memory only (Log M & Log MD)	6%
Nonverbal memory only (BVR; MFD	28%	Nonverbal memory only (Vis R & Vis RD)	29%
Immediate verbal recall (CM1)	43%	Immediate verbal recall (Log M)	59%
3Hour delayed verbal recall (CMII)	30%	30 Min delayed verbal recall (Log MD)	71%
3. Perceptual speed and accuracy *	28% text 58% table	General cerebral efficiency	44%
4. Disturbed concept formation	48%	Planning and problem solving	41%

* The Ljunggren et al (1985) paper contains a number of inconsistencies between the text, and the results as summarized in Table III. Some figures in the above table are thus extrapolated from both reported figures.

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The cognitive profiles in this study were derived from this wide-angle focus on general functional outcome, whereas Ljunggren et al's (1985) purely cognitive system of classification appears to consist of an arbitrary division of patients according to the number of tests on which the patient was impaired. In spite of this, there is a remarkable similarity between the two sets of profiles. For example, those classified as mildly or slightly impaired are impaired primarily in terms of recent memory, which is frequently associated with impairment of new learning.
The two sets of impairment profiles differed in terms of the severity-rankings of specific cognitive abilities in two ways:
In the present study decreased general cerebral efficiency (perceptual speed and accuracy) was found in patients categorised as moderately functionally impaired whereas Lyunggren et al (1985) considered patients with impairment of this function as being markedly cognitively impaired. This study also found that the higher mental processes such as planning and problem solving were impaired in subjects with severe functional impairment, whereas patients with similar cognitive impairment were part of Ljunggren et al's (1985) moderately cognitively impaired group.

The above differences appear to be due to the different classification systems used in the two studies. It is suggested, however, that the degrees of severity reflected in the Ljunggren et al (1985) study are problematic because they are based on a questionable summation of cognitive deficits, and thus do not necessarily reflect on functional ability, ie. the additional effects of physical, emotional and personality factors. Thus Ljunggren et al's (1985) categories appear to be limited in terms of recommendations for rehabilitation.

Prognostic Variables

Although there were no statistically significant findings due to small sample size, various tentative trends emerged from the subgroup analysis.

The results of the present study tentatively lend support to Ljunggren et al's (1985) finding that the degree of cognitive impairment is correlated with the patient's age. This finding is commensurate with the general evidence that stroke patients over the age of 45 have a worse prognosis (Lezak, 1983). The results also suggest that the severity of the internal bleed may be related to cognitive outcome. It seems that patients with the most severe cognitive impairment were generally older (age 45+), had a poorer grade on admission, and a lower level of education. However, it is not possible to determine the proportional contribution to outcome of the abovementioned factors due to the small sample size of this study.

It appears that ICA aneurysm patients may be somewhat less cognitively impaired than patients with aneurysms at other sites. The study also provides tentative support for the practice of delayed surgery (delayed select patients were less impaired on Ass LD). It is suggested that the extent of surgical dissection required to treat ACoA and MCA aneurysms, in contrast to the relatively easily accessible ICA aneurysm, may explain the above finding. Theoretical arguements have also been put forward by Hubschmann and Krieger (1985) and Kassell (1983) to delay surgery. It is argued that early (acute) surgery may result in relatively greater diffuse brain damage because the brain is more prone to contusion and laceration the first few days after haemorrhage, and that it may less able to tolerate the focally reduced perfusion pressure resulting from brain retraction during surgery.

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Analysis of the sample in terms of estimated premorbid functioning revealed that the low average IQ group had disproportionately low scores on the Complex figure test, Associate learning test and Logical memory test.

It is possible that patients with premorbidly marginal intellectual capacities have suffered relatively greater cognitive impairment as measured on these particular tests, however, it is considered more likely that these results are a reflection of inappropriate norms. The norms used in the present study have been established on a variety of small normative populations in the United States of America and variables such as socio-economic status and educational level do not appear to have been taken into account. The importance of such variables has been demonstrated in the South African context by Dugmore (1987) and Jacobs (1985). Dugmore (1987) found that skilled workers with an education of Standard 10, or above, had a superior performance on a verbal fluency test when compared to semi-skilled and unskilled groups with an eduation of less than Standard 8. The three tests with disproportionately low scores all have a verbal component (including the CFT - Walsh, 1985). Furthermore, Jacobs (1985) found that subjects with an education of Standard 5 or below performed significantly less well on the Complex figure test than did subjects with a Standard 10 education.

It should be noted, however, that the present sample included only four patients with an estimated low-average premorbid IQ and thus the lack of SA norms does not invalidate the results of this study.

The results of the WAIS subtest scatter analysis are surprising from a neuropsychological perspective (for example, the Information subtest which is expected to hold (Lezak, 1983), was found to be the most frequently impaired of all subtests), but can also be explained by a number of subjects having had less contact with the dominant culture, knowledge of which is tested on this subtest. Relative cultural deprivation is probably also reflected in the low

Comprehension and Similarities scores. The deterioration noted on the Object assembly, Block design, Digit symbol and Arithmetic subtests is commensurate with effects of SAH-induced diffuse encephalopathy. Fall-off in functioning on these subtests is similarly found in other organic brain syndromes involving diffuse brain dysfunction, such as Alyheimer's dementia (Lezak, 1983).

No significant differences or trends in terms of prognostic variables, could be identified on the structured interview reports of physical, emotional, personality or behavioural sequelae. This aspect deserves further study because the lack of findings may be due to the small sample size. Brismar and Sundbarg (1985) followed up 127 patients with SAH of unknown origin at an averge of 5.4 years post operatively and found that patients classified as Grade 1 on admission were significantly less impaired in terms of subjective complaints (headache, vertigo, irritability, fatigue) than patients originally classified as Grade 2.

Finally, although there were no significant differences on any other tests, males' perfomance on the WISC mazes test was far superior to that of the females. This finding is in accordance with the well known fact that males are generally superior to females on visuospatial processing, while females are generally superior on verbal tasks (Lezak, 1983).

Evaluation of the Study

In evaluating this study two limitations should be noted, the small sample size and the problems involved in applying unsuitable norms to four patients, judged to have a low-average premorbid IQ. However, the findings in this study closely approximate those found in Ljunggren et al's (1985) larger sample (N = 40). Thus these limitations do not appear to invalidate the results of this study and certain noteworthy implications may be derived from it.

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The stengths of this study lie in certain aspects of its methodology:

(i) The methodology has been structured to allow a detailed, thorough study of the area through extensive assessment on a smaller sample, as opposed to a more limited assessment on a larger number of subjects.

(ii) The time period in which follow-up assessment was done was limited to 6-8 months post-operatively, as opposed to the 14 months to 7 years period of Ljunggren et al (1985). This enabled the researcher to establish the status of the patient at a time when the most important recovery is expected to have taken place and recommendations regarding rehabilitation are customarily made.

(iii) Assessment was made on the basis of integrated functional outcome, in which all variables are accorded equal importance in rehabilitation planning. This is not possible when the incidence of behavioural dimensions are reported separately, as in Ljunggren et al's (1985) study.

(iv) This study is the first to specifically examine whether the timing of surgery has implications for prognosis of patients with good neurological recovery after SAH.

(v) This study considered a far greater range of prognostic variables than the other study on SAH with neurologically-intact patients: aneurysm site, age, gender, education, grade of haemorrhage, premorbid IQ and timing of surgery.

(vi) Because the use of an ad-hoc selection of tests places severe limitations on the interpretation of findings, the cognitive assessment procedure was metatheortically grounded and given coherence in two cognitive assessment models.

Despite the exploratory nature of the study, and small sample size, the fact that the results correlate well with Ljunggren et al's (1985) general findings, suggests that it has

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supplied cross-validation for both studies, while increasing and affirming the general body of knowledge about this particular group of SAH patients.

General Implications of the Study

It must be stressed that the following implications relate only to patients who are neurologically-intact and exhibit a diffuse SAH-induced encephalopathy 6-8 months post operatively. They cannot be generalised to the broad spectrum of SAH patients, as they have varying degrees of neurological deficit.

It is apparent from the results of this study that every patient who is neurologically intact after SAH should be referred for a neuropsychological assessment. Ideally, neuropsychological assessment should take place before the patient returns to work (generally 6-8 months post-operatively). The neuropsychologist has a vital role to play in making a realistic assessment of the patients functional status in order to determine effective rehabilitation strategies. The disasterous psycho-social consequences of premature return to work (Bond and Brooks, 1976), illustrates the vital role for the neuropsychologist who is trained to assess subtle cognitive, personality and emotional impairment, and is able to counsel the patient, his family and others in order to maximise adaptation to the deficits directly resulting from cerebral impairment. In this way the indirect, psychological components of SAH sequelae such as depression and lowered frustration tolerance may be minimised.

Tentative findings suggest that delayed surgery (7 or more days after SAH) results in a better prognosis than acute surgery (2 to 4 days after SAH). The potential implications of this finding with regard to surgical practice are important, but other large-scale studies are required to confirm or disprove this finding.

Implications with regard to Neuropsychological Assessment

(i) This study highlights the fact that overall functional outcome, taking into account physical, cognitive, emotional, personality and behavioural factors, is necessary to inform realistic rehabilitation plans.
There is a danger that classification systems based solely on cognitive impairment (eg. Ljunggren et al, 1985) will result in relative neglect of the other aspects of functional outcome. All aspects have equal importance in determining future performance in the social and occupational spheres. This approach is commensurate with that of Lezak (1983) who notes that brain damage usually affects all three "functional systems" - intellect, emotionality and control. Furthermore, it is important to bear in mind that cognitive deterioration is not necessarily the most important effect of brain damage (cf. Lezak's (1983) findings that impairment of "executive functions" may be incapacitating despite "intellect" remaining intact).

(ii) This study directs attention to the fact that certain emotional, personality and behavioural sequelae evident 6-8 months post-operatively are more likely to be direct consequences of brain damage (eg. anergia) while others result from a combination of direct and indirect effects.
Although Lezak (1983) points out that personality changes, emotional distress and behaviour problems are the "product of extremely complex interactions involving (the patient's) neurlogical disabilities, present social demands, previously established behaviour patterns, and his ongoing reactions to all of these "(p36) it is important to be aware that certain symptoms are more likely to be primary, as opposed to secondary, consequences because of the implications for the neuropsychologist's therapeutic

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intervention programme. For instance, the neuropsychologist may decide to take the patient into therapy in order to facilitate his/her acceptance of, and adaptation to, the direct effects of brain damage. He/she may also counsel the family as to the true nature of the patient's irreversible disabilities to minimise family conflict (eg. by misinterpreting anergia as "laziness"); or effect environmental changes (eg. recommending that the patient change occupations to minimise failure and resultant feelings of despair). Alternatively, it is important to be aware of the indirect effects because the symptoms themselves are amenable to therapeutic intervention.
If the specific nature of the sequelae are not recognised and dealt with in an appropriate manner, it is likely that rehabilitation will be slower, and the secondary reactions to brain damage may be excacerbated.

(iii) This study has also highlighted the need for the development of South African norms, and for caution in interpreting test scores in the SA context - particularly with regard to patients who are judged to have a low-average premorbid IQ as these patients are the least likely to be similar to the population on which the test norms are based.
However, there is no reason for overreaction to the general lack of South African norms as the practice of neuropsychologoy does not depend solely on a comparison of the individual's performance against a population norm but also on the use of the deficit measurement method (comparison of a patient's performance against his/her own norm). Furthermore, the syndrome analysis approach reflects patterns of expected deficit, which can be confirmed independently of norms. In addition, the existing (USA) norms are probably not as inapplicable to South Africans with a

higher level of education. Support for this is suggested by the similarity in patterns and incidence of congitive deficits noted between the present study and Ljunggren et al (1985).

(iv) This study has clearly illustrated the utility of the psychometric approach of Walsh (1985, 1978), Lezak (1983), and others. Russell (1981) argues that the utility of the approach lies in its acurate detection of the subtle effects of diffuse brain damage. In contrast, the nominal qualitative nature of the neurological approach (Luria, 1973) is suited primarily to the assessment of more severe localised cerebral pathology. In the present study the nature of the haemorrhage (spreading into the subarachnoid space) resulted in a diffuse encephalopathy which was not detectable on the brief assessment of neurological/localising signs, but was clearly demonstrated on the psychometric test battery.

Implications for future research

Longitudinal studies (for example at 6 months, 1 year, 2 years and 5 years post-operatively) should be conducted to determine the natural course of recovery in this group of patients as this study only focussed on the six-month-post-SAH sequelae. This will enable the clinician to make more accurate predictions and thus optimise rehabilitation.

As this study very tentatively indicates that timing of surgery may affect prognosis, a most urgent need is large-scale research to evaluate the sequelae of acute versus delayed surgery. Ideally this research would be conducted independently at a number of institutions worldwide to control for the effects of different surgical procedures. Such studies should include other variables such as neurlogically intact versus neurlogically impaired patients. The presence or absence of neurlogical deficits after SAH

could, in itself, be an important aspect for research. It is suggested that patients with neurlogical deficits may exhibit localised signs of brain dysfunction (see literature review-Introduction) overlayed on a similar picture of diffuse encephalopathy as found in the present study.

A final suggestion is that, because of the close correlation between the findings of the present study and that of Ljunggren et al (1985), future research need not consider as broad a range of variables in as much depth. A more focussed research methodology concerning the specific variables highlighted in the above two studies, and larger numbers of subjects would appear to be appropriate.

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ERRATA

P.1 Line 12: "concentraining" should read "concentrating"
14: "sequence" should read "sequelae"
18: "hav" should read "have"

P.4 First Line: subheading omitted: "Diffuse effects"
Second paragraph, first line: "observed" should read "obscured"

P.5 Last paragraph, first line: "symptons" should read "symptoms"

P.7 Line 3: "ons tudies" should read "on studies"
Paragraph 3, line 2: "form" should read "from"

P.8 Paragraph 3, line 12 "neurospychological" should read "neuropsychological"

P.15 Last paragraph, line 2: "severity" should have been underlined.

P.16 Paragraph 2, line 1: "Occupation" should read "Occupational"

P.17 Paragraph 2, line 3: "(2) scores" should read "(Z) scores"

P.19 Table IV No. 5: should read "Impaired attention and concentration"

P.20 1.1.3. (1): "returnted" should read "returned"

P.25 Last paragraph, line 1: "scors" should read "scores"
Last paragraph, line 1/2 should read "...in terms of prognostic variables Table 111),..."

P.26 Table x 4) 1): < sign omitted," (< age 35)"

P.27 Line 7: parenthesis omitted, "(mean education Standard 6.5)"

P.29/30 Table x 1 2.(6) and 3.(8) "Symptons" should read "Symptoms"

P.39 1st paragraph, last line: "Alyheimers" should read "Alzheimers"