PSYCHIATRIC ASPECTS OF SUBARACHNOID HAEMORRHAGE

by

P.B. Storey.

A Thesis submitted for the degree of M.D.
in the University of London.

1970
# CONTENTS

| Chapter I | General Introduction | 3 |
| Chapter II | Anatomy and Pathology | 11 |
| Chapter III | Case Material and Methods | 15 |
| Chapter IV | Overall Results with special reference to the trial of Operative and Conservative Treatment | 32 |
| Chapter V | Personality Change after Subarachnoid Haemorrhage | 51 |
| Chapter VI | Intellectual Change after Subarachnoid Haemorrhage | 90 |
| Chapter VII | Affective Change after Subarachnoid Haemorrhage | 113 |
| Chapter VIII | Emotional Aspects of the Precipitation of Subarachnoid Haemorrhage | 141 |
| Chapter IX | Subarachnoid Haemorrhage and Migraine | 155 |
| Final Discussion | | 161 |
| Final Summary | | 163 |
| References | | 169 |

_Acknowledgements_

_Reprint bound in back pocket: Lumbar Air Encephalography in Chronic Schizophrenia: a controlled experiment._
This thesis is based on a personal follow up of two series of patients who had had a proven subarachnoid haemorrhage; the first one containing 261; and a subsidiary series containing 30 patients.

The principal problem which is investigated is that of the nature and frequency of the mental changes found in patients who have suffered a subarachnoid haemorrhage; and the relationship between those changes and the following: absence or presence of a ruptured intracranial berry aneurysm; operative or conservative treatment of ruptured aneurysm; site of aneurysm and site and extent of brain damage; the patients' premorbid personality; and previous and family history of psychiatric disturbance.

A secondary problem investigated is the role of emotional stress as a precipitant of subarachnoid haemorrhage. Thirdly, the relationships between subarachnoid haemorrhage, personality and migraine are also briefly examined.

The study arose from a comparative trial of operative and conservative treatment of ruptured intracranial berry aneurysm undertaken principally at Atkinson Morley's Hospital, London, (a branch of St. George's Hospital); the organisation, methods and results of which have been published in a series of papers by McKissock, Richardson and Walsh (1960 and seq.). The measures of outcome they used were survival, physical disability and employment status; but they were impressed by the frequency and severity of mental changes, which the present author was consequently
asked to study in more detail.

The first chapter is a general introduction to the whole subject of subarachnoid haemorrhage, with a brief review of the relevant neurosurgical literature, as well as an account of the more purely psychiatric aspects, so that the latter can be seen in perspective. The second chapter deals briefly with the neuropathology of the illness and the third describes the clinical material, methods, ratings, scales and definitions used. The next five chapters describe different aspects of the results and, although they are interrelated, each can be considered separately and each, therefore, has its own supplementary review of the literature and separate discussion section.

Chapter IV and Chapter IX are only slightly modified from papers already published (Storey, 1967 and 1969), and similarly Chapter V is derived from a paper accepted for publication by the British Journal of Psychiatry.
CHAPTER I

GENERAL INTRODUCTION

1. Incidence

Subarachnoid haemorrhage is not a rare condition and, compared to other forms of cerebrovascular accident, it is relatively commoner in the young and middle aged. Crawford and Sarner (1965) made a very thorough study of a population of 1,121,000 living near Atkinson Morley's Hospital, of whom 913,000 were under 60 years old. In a two year period, 280 people under 60 died from cerebrovascular accidents, of whom 78 (28%) had ruptured intracranial aneurysms, mainly those aged between 45 and 59 years; an annual incidence of 6 per 100,000 in the population under 60 years. Giel (1965) in the Netherlands found an incidence of subarachnoid haemorrhage of 78 per million for all age groups, including cases other than those with ruptured aneurysms. As only about 45% of patients with subarachnoid haemorrhage have ruptured aneurysms (Kisscock and Paine, 1959) this is a major source of difference. In Helsinki, Pakarinen (1967) estimated the annual incidence at 16 per 100,000 for all ages, which is considerably higher than the others published and which, if it applied to the United Kingdom, would lead to about 8,000 cases per year, compared to the older estimate of 3,000 per year made by Walton (1956). According to Crawford and Sarner (op cit), about 30% of patients with aneurysms die before reaching hospital (and therefore never figure in most statistics), and only 38% of their series were alive one month later. The death rate
in the 50% (approximately) who have normal angiograms is only one tenth that of those with aneurysms (Tappura, 1962), so that at a very rough estimate, we might expect from 2,500 - 5,000 new survivors each year in this country - depending on which figures are used as a basis.

2. Natural History and Treatment

In most writings on subarachnoid haemorrhage, there is little information about mental changes. The earliest literature consists mainly of scattered accounts of ruptured aneurysm found at post mortem, although even in 1859 Gull was relating clinical and post mortem findings. Symonds (1924) and several other authors then collated clinical findings on a larger scale but it was not until after the 1939-45 war that cerebral angiography gradually became common and surgical treatment attempted. Because pre-angiographic studies do not distinguish between those with and without aneurysms, they have less relevance to present day conditions but the serious mortality and morbidity is nevertheless striking, as shown, for instance, by Hamby (1952), who found that out of 130 patients only 48 survived, of whom only 21 were completely well. In the largest pre-angiographic study, Jacobson (1954), surveying other published series, estimated 9.4% of 950 survivors as severely damaged, mentally or physically.

The literature on the historical, neurological and neurosurgical aspects of subarachnoid haemorrhage is extensively reviewed by Walton (1956), Tappura (1962) and Pakarinen (1967), among several others, but only directly relevant studies will be
Walton (1956) was the first author to give mental changes any prominence but his work was mainly preangiographic, which lessens the comparative values of his figures. Of his 312 patients, 120 survived, of whom only 30% were completely well. 73% had slight sequelae, 33% moderate or severe sequelae and 4% were completely disabled. He commented that mental changes had not had much attention in the literature but they were an important cause of disability in his series. Three patients were demented, another two nearly so and three had severe depression, panic attacks and mental slowness, respectively. Another six had less severe mental changes of various types; and four were aphasic. Altogether, 9% of his 120 patients had permanent mental deterioration; and a further 32 (27%) had significant anxiety symptoms, of whom nearly half had had neurotic previous personalities. Only five showed much improvement and 27 had persistent moderate or severe anxiety states.

One study in which angiograms were used was that by Björkesten and Troupp (1957), who followed up 101 patients, of whom 61 had no aneurysms. The mortality was 55% for those with and 5% for those without aneurysms and the latter also had a lower morbidity physically; but relatively more "functional" complaints of headache, dizziness and tiredness, despite the lower incidence of CNS signs.

An important more recent study is that by Tappura (1962), from Finland, who followed up 120 patients with aneurysms and 267 with normal angiograms. The death rate was
ten times higher in those with aneurysms than in those without and measures of morbidity showed a less marked but still significant trend in the same direction. Thus, of 60 surviving aneurysm patients, 8 (13%) were totally and 28 (47%) partially disabled. Mental changes are an important cause of morbidity under the title "diffuse cerebral symptoms", which included both 'organic' and 'functional' psychiatric states and accounts for almost half the partial disability in the aneurysm group and well over half that in the no aneurysm group. His findings are, therefore, very similar to those of Björkesten and Troupp (op cit) in the differential mortality rates, morbidity rates and the relatively higher incidence of psychiatric symptoms in the no aneurysm group, despite fewer C.N.S. signs.

In addition to papers concerned with the overall prognosis of subarachnoid haemorrhage, an increasing number of studies have appeared in which the value of surgery has been assessed. The comparative trial of operative and conservative treatment, out of which the present study arose, must be seen against a background of widespread acceptance by neurosurgeons that surgery was the treatment of choice for ruptured aneurysm, as shown, for instance, by Norlen and Ollecrona (1953), Logue (1956) and Hamilton and Falconer (1959).

In 1960 McKissock, Paine and Walsh demonstrated by a retrospective study that the better results of surgery could be explained in whole or in part by selection factors. Because the recurrence rate is at its highest in the first week after
bleeding, waiting a week or more before operating taken the patient past the danger period and surgery may seen strikingly effective. A prospective trial was, therefore, designed to allocate cases randomly to operative or conservative treatment; after stratification by site of aneurysm, blood pressure, age, clinical condition and time elapsed since the haemorrhage. McKissock, Richardson and Walsh (1960) described the design of the trial and showed that, for patients with posterior communicating (P.C.) aneurysms, operative treatment with common carotid ligation was significantly better than conservative management. Then in a series of papers by the same authors (1962, 1964 and 1965) it was shown that, for P.C. aneurysm, surgery was more effective for men but not for women; that surgery was more effective for multiple aneurysms; but that it was of no benefit for anterior communicating artery (A.C.) aneurysms.

Many other differences have been shown to exist between the different aneurysm groups, not only in prognosis but in the relevance of various other factors such as blood pressure, age, sex, size and shape of aneurysm, (e.g., Richardson, Jane and Yashan, 1966). Other important factors which affect the survival rate for all diagnostic aneurysm groups are the level of consciousness - those in clear consciousness do much better - and the general clinical condition, with degree of neurological involvement. It seems unnecessary to consider these neurosurgical details further in this thesis but they are mentioned to indicate that it is not only the presence or absence of an aneurysm that counts but also its site and numerous other clinical
factors. No psychiatric detail appears in any of these papers recently quoted but, as described earlier, the neurosurgeons involved in the trial recognised that mental changes were very important and it was considered necessary to make these the subject of a special enquiry, the results of which are described in this thesis.

3. Psychiatric Aspects

The literature on the psychiatric and psychological aspects of subarachnoid haemorrhage is very small. There are several papers on the acute disturbances which the patient commonly shows at the time of the bleed, such as that by Heidrich (1966), who found that 57% of 226 cases showed changes in consciousness, with delirious states, Korsakoff's syndrome, emotional lability, akinetic mutism and schizophrenic like psychoses. He also claimed that those with more severe mental changes in the acute phase had a worse prognosis, presumably because both reflect more severe brain damage. Pincherle (1961) also mainly described the presenting features in similar terms but mentioned briefly that headache, slowed thinking, disturbed memory and fatigue/ability were common long term sequelae. There are also several scattered case reports of patients with unusual clinical features, such as that by Silverman (1949), who described a paranoid psychotic illness during convalescence. Hoetzel (1940) and Geyer (1962) relate some of the mental changes found to post mortem findings, particularly to the deposition of pigment in the cortex and in the lining of the ventricular system.
but their information is too limited to be convincing.

Other papers have dealt with more purely psychological aspects. The most detailed of these is by Naughton (1954), who studied 51 patients in the first few weeks after the haemorrhage and whose results will be referred to in Chapter VI. Lindqvist and Norlen (1966) and Talland et al (1967) are among those who have described amnesic states resembling Korsakoff's psychoses after rupture of anterior communicating aneurysms, presumably because of direct involvement of the mammillary bodies.

Only three papers have been found which concentrate on the long term mental changes, other than that already published by the author (Storey, 1967), which is largely reproduced in Chapter IV. Unfortunately, the first two are poorly documented. Heidrich and Kambach (1967) followed up the series of patients described in the acute stages by Heidrich (op cit). Impairment of memory and concentration was frequent, with general slowing down and loss of drive. Short et al (1966) followed up 29 patients for an average of 42 months. Like Heidrich and Kambach, they did not say how many had aneurysms, nor the sites, nor did they give detail of any kind. However, two thirds had some mental impairment, mostly with increased irritability and susceptibility to anxiety and depression.

In contrast, the paper by Logue et al (1968) is a very thoroughly worked out follow up of 79 patients with anterior communicating artery aneurysms: 66 treated by operation, 13 treated
conservatively. Personality change, intellectual test results and affective symptoms are all described in detail and, as much of their study is relevant to this one, it will be mentioned later in more than one discussion section.

More surprising than the neglect of the sequelae of subarachnoid haemorrhage is the fact that there appear to have been no detailed studies of the mental changes occurring in large unselected series of patients after other cerebrovascular accidents. Most important clinical studies of personality change after cerebral lesions have been based either on the results of head injuries, surgery for cerebral tumours, or after leucotomy; and some of these will be mentioned later when appropriate.

A study of patients who have survived a subarachnoid haemorrhage and have been fully investigated in a neurosurgical unit has some advantages, when considering the relationship between site of brain damage and subsequent mental changes. Firstly, angiography can demonstrate the existence and exact site of a berry aneurysm with considerable accuracy. Secondly, unlike other stroke illnesses, subarachnoid haemorrhage is not usually part of a progressive and diffuse arteriopathy but is a clear cut episode, occurring in people who are generally younger than those who have other strokes. Thirdly, although recurrent haemorrhages are fairly common, they arise mainly from the same aneurysm as bled previously, so that localisation effects are not necessarily lost.
CHAPTER II

ANATOMY AND PATHOLOGY

This brief section is derived mainly from the work of Crompton (1962 (a) and (b), 1963 (a) and (b)), whose case material was from the same neurosurgical department as that studied here. Other papers drawn upon are those by Kibler, Couch and Crompton (1961) and Robertson (1949). Patients with subarachnoid haemorrhage from angioma, blood dyscrasias, primary intracerebral haematoma, cerebral tumour and head injury were not studied clinically and are not mentioned further. Certain less frequent aneurysm sites were also excluded from consideration and we are left with the diagnostic categories set out below. According to McKissock and Paine (1959), of the 45% in their series of 781 patients who had aneurysms, the breakdown was as follows: anterior communicating aneurysms (A.C.) 31%; posterior communicating aneurysm (P.C.) 21%; middle cerebral aneurysms (M.C.) 16%; multiple aneurysms 15%; others 15%.

Anterior Communicating Artery (A.C.) Aneurysms

These are essentially midline structures, although the blood supply may be principally from one or the other side. They lie between the frontal lobes, near the anterior hypothalamus, and rupture may cause immediate damage to nearby structures; may lead to haematoma in the frontal lobes or elsewhere; or may cause infarction, principally but not solely in the territory supplied by the anterior cerebral arteries.
Middle Cerebral Artery (M.C.) Aneurysms

These lie in the Sylvian fissure or embedded in the frontal or temporal lobes nearby. They commonly rupture to form haematomas in the Sylvian fissure itself, or in the temporal lobe or the external capsular regions. Infarction is usually, but not always, confined to the area supplied by the middle cerebral artery.

Women tend to have fusiform aneurysms, frequently embedded in the substance of the brain. In men the aneurysms are usually saccular, with a distinct neck, and more often lie free in the Sylvian fissure. Ruptured M.C. aneurysm in a woman leads to more direct brain damage, to more Sylvian fissure and intracerebral haematomata and the aneurysm is both more difficult to approach and to operate on (see Chapter IV).

Posterior Communicating Artery (P.C.) Aneurysms

These form where the posterior communicating artery arises from the upper end of the internal carotid artery and lie medial to the uncus of the temporal lobe. Haematomata form mainly in the temporal lobe but rupture into the brain is less common with these aneurysms than with the others. Infarction, however, is more widespread and severe and is mainly in the territory of the middle cerebral artery. Interference with the fine perforating vessels to the basal ganglia and hypothalamus also occurs.
Multiple Aneurysms

The pathology depends on which aneurysm has ruptured.

No Aneurysm (i.e., normal angiograms)

This group is distinguished clinically from that of patients with primary intracerebral haemorrhage in whom blood escapes into the cerebrospinal fluid, although one of the 30 patients in this group did have an intracerebral haematoma. The source of the bleeding is unknown but it may arise from microaneurysms of the leptomeningeal or subcortical vessels, (Crawford and Crompton, 1965). A small proportion of these patients are likely to have cerebral berry aneurysms which were not demonstrated.

Cerebral haematomata and areas of infarction are clearly likely to be much more common in those who have died than in survivors. However, post mortem studies of patients who have survived a subarachnoid haemorrhage, only to die later, show that both infarction and haematomata have often been survived. Haematomata also show clearly on many cerebral angiograms and there is good agreement between angiographic and post mortem findings in this regard.

In addition to damage caused by infarction, haematoma formation, oedema and that due to the direct effects of the ejected jet of blood, subarachnoid haemorrhage may also lead to
loptomeningeal fibrosis and consequent hydrocephalus - obstructive or communicating. In this series, however, no patient was considered to have hydrocephalus.

Hypertension is an important factor in the genesis of subarachnoid haemorrhage. McKissock and Paine (op cit) found that 57% of patients admitted to hospital had initial blood pressure readings of above 160/90 and Crompton (op cit) found post mortem evidence of hypertension in 52%, judged by the thickness of the wall of the left ventricle. Hypertension is also relevant to prognosis (see Chapter IV).
CASE MATERIAL AND METHODS

CASE MATERIAL

1. First Series

There were 261 patients, of whom 149 (57%) were women. Ages varied from 19-67 years; 15 (6%) were under 30; 64 (25%) were from 30-44; 131 (50%) from 45-59; and 51 (20%) were 60 years or more.

None of the patients was known to have been taking MAO inhibitor drugs. In 81 cases there was an aneurysm of the anterior communicating anterior cerebral artery complex (A.C.); in 71, there was an aneurysm of the middle cerebral artery (M.C.); in 72, there was an aneurysm of the posterior communicating artery (P.C.); in 7 there were multiple aneurysms (Mult. an.); and in 30 cases, no aneurysm was demonstrated by bilateral carotid and vertebral angiography.

The no aneurysm cases were included as a control group, in whom the mortality and morbidity was known to be much lower than with aneurysms but who had been through similar experiences both in and out of hospitals.

METHODS

The follow up period varied between six months and six years; it was more than three years in 145; between three years and eighteen months in 43; and less than eighteen months in 73. In only 11 was the period less than one year, of whom two were seen at six months. The mean follow up period was 2.0 years.
All patients were seen between December, 1963, and June, 1965. Selection of cases was based on the site (or absence) of aneurysm and, apart from excluding those who lived too far away, was consecutive within each group in order of admission to hospital. Only one patient who was approached refused to be interviewed and she is not considered further. Of the 261 cases, 260 were seen by me— all but eight in their own homes. One case is included on the basis of detailed letters from the patient and his wife and of a report from Wing Commander A. W. Black, R.A.F., who examined him and to whom I am indebted. (The patient was rated unimpaired in all respects).

The first twenty cases seen were known to have P.C. aneurysms but thereafter the groups were mixed and the name and address only provided by a secretary. As far as possible, each visit was then made without knowing of the presence or site of an aneurysm but cranial scars and statements by the patient often betrayed the facts.

Each interview lasted between one and a half to two hours and consisted of an initial informal period with the patient, followed by history taking and then by questions from a check list. Simple clinical tests of memory, grasp and comprehension were used. Lastly, the Benton Visual Retention Test and a slightly modified form of the Inglis Paired-Associate-Learning Test were given in most cases (see later). In 242 cases, another informant was also seen, usually the spouse or other close relative, who was interviewed separately. The patient was then examined physically and blood pressure was measured at the beginning and end of the examination, the lower reading being accepted.
Minor lateralizing neurological signs were carefully looked for. In addition, letters from the patient's General Practitioner were also available and a few general practitioners were seen personally - mainly in rural districts. If a patient had had inpatient or outpatient psychiatric treatment, the hospital was contacted and, in all but two cases, the notes were borrowed.

2. Second Series

The second study was of 30 women with middle cerebral (M.C.) aneurysms. Their ages varied from 28-62 years; one (3%) was under 30; 10 (33%) were from 30-44; 17 (56%) were from 45-59; and 2 (6%) were 60 years or more. The follow up period varied between 12 months and 3 years. It was between 18 months and 3 years in 26; and less than 18 months in 4. The mean follow up period was 2.1 years. These patients were all seen in 1967 and the method of study was the same as for the earlier series except that particular notice was taken of the patient's circumstances and emotional state at the time of the stroke. All but two patients were seen at home and another informant was seen for each.

RATING SCALES

At the end of the psychiatric interview and before the physical examination, ratings were made on four point scales for physical disability, personality impairment, intellectual impairment and psychiatric symptoms, as described below. The rating for abnormal neurological signs (C.N.S. signs) was made
after the physical examination. Most patients with mild C.N.S. signs were rated as having no physical disability, as they had asymptomatic reflex asymmetries, etc. (see below). As both ratings were subjective, there must be some confusion between them but there is no overlap in the uses to which they have been put in this study.

**Physical Disability**

Patients were rated subjectively as follows:

1. Mild disability indicated that the patient was able to perform most ordinary day to day tasks, although aware of difficulty; could walk enough for work and shopping purposes, etc.

2. Moderate disability fell between mild and severe and, therefore, covered a wide range of functions.

3. Severe disability indicated that the patient had a useless arm and hand and, in walking, was limited to a few steps.

**Overall Personality Impairment**

The ratings were subjective and based largely on the impact on family and other relationships, so that particular note was taken of the accounts of other informants.

1. Mild impairment was rated when change was unequivocally present but not severe enough to lead to much change in personal relationships. A good many with trivial or doubtful changes were rated 'unimpaired'.

2. Moderate impairment fell between mild and severe and, therefore,
covered a wide range.

3. Severe impairment was rated in all those frankly demented and in three others with only moderate intellectual change but in whom the personality change was of outstanding importance.

**Overall Clinical Intellectual Impairment**

In this rating, the results of the Benton Visual Retention Test and the Inglis Paired-Associate Test were ignored as far as possible but they were known at the time the rating was made, so that some bias is possible.

It is based on accounts given by the patient and other informants, the results of simple clinical tests (orientation, digit span, remembering a name and address, simple calculations, serial seven subtraction, knowledge of current affairs) and the general impression made; at the same time taking into account the patient's background and presumed previous intelligence. A rating of:

1. Mild impairment indicates some impairment of recent memory and higher intellectual abilities but is compatible with no loss of employment efficiency except in professional and executive occupations.

2. Moderate impairment falls between mild and severe and covers a wide range of functions.

3. Severe impairment is equivalent to frank dementia; with intellectual inaccessibility, with the patient unable to make meaningful attempts at psychological tests.
Psychiatric Symptoms

(a) Depression. Depression was rated as absent, mild, moderate or severe, on the basis of the history given by the patient, other informant, general practitioner and from other hospital notes. The ratings refer to the whole follow-up period and not only to the time of examination. They were made within a few minutes of the end of the interview but were changed in three cases after the later study of hospital notes, (Two rated more and one less severe).

It had been intended that "treatment offered" would be used as an operational definition but this was abandoned because so many patients had been clearly depressed but had had no treatment. It seemed that many people, including doctors, regard depression as unremarkable, and almost inevitable, after a stroke. The assessment of depression was, therefore, basically subjective, especially in those rated mildly depressed. However, the treatment offered to these patients is mentioned in Chapter VII and gives considerable objective backing to the ratings.

Depression restricted to the first three months after the stroke is ignored, because of the occasional presence of confusional features in that period.

(b) Anxiety Symptoms. These were also rated subjectively and, because of the frequent inextricable mingling of anxiety with depression, they are not considered separately in much detail.
Neurological (C.N.S.) Signs

Ocular palsies alone were not counted.

1. **Mild signs**: reflex asymmetry, cortical sensory loss, sensory inattention, mild dysphasia (not otherwise defined) and homonymous hemianopia. Except for the dysphasia, they were not obvious without neurological examination.

2. **Moderate signs**: this indicates weakness less than severe, or the presence of marked dysphasia with or without mild signs.

3. **Severe signs**: this indicates at least the presence of marked weakness and spasticity, with little or no function in an upper limb, and a limp.

**PSYCHOLOGICAL TESTS**

1. **Benton Visual Retention Test** (Benton, 1956)

   This test, form C, administration A, was given to 241 patients, and scored by the 'number correct' score after the raw scores had been corrected for age and estimated premorbid intelligence. The results are grouped as (a) Normal - not more than 2 points below the expected score, (b) Abnormal - 3 or more points below the expected score.

2. **Inglis Paired Associate Test (P.A.T.)** (Inglis, 1959)

   Form A was used. It was found in the early stages of the study that the instructions to the patient were misleading, in that illustrative examples ('East-West' and 'Gold-Silver') led patients to seek meaningful connections in the actual test pairs, where there are in fact none. Therefore, 'Flower-Cloud' and 'Oven-Grass' were substituted as examples and the earlier test
results excluded. The modified test was given to 213 patients, all of whom also completed the Benton Test. The score is the number of errors made before reaching the criterion. The results are grouped as (a) Normal - up to and including 10 errors, (b) Abnormal - more than 10 errors.

Overall Mental Disability

Unfortunately, the need for a rating of overall mental disability was not foreseen in planning the study. It later seemed desirable, however, that some overall scale should be available, comparable to that for overall physical disability, in which various forms of disability, (e.g., walking and grasping) are rated subjectively in terms of their importance to the patient. A five point rating scale of Overall Mental Disability was therefore constructed for the comparison between operative and conservative treatment, putting most weight on permanent changes and considering transient affective symptoms, even if severe, as less important than intellectual and personality damage. Each patient was rated on the scale as follows:

1. Absent or Minimal Disability - includes those with only minor memory impairment (forgetfulness) and those with transient mild anxiety or depressive states.

2. Mild Disability - includes those with intellectual impairment, which allows adequate functioning in non-intellectual occupations; those in whom personality change is not sufficient to lead to deterioration in family or other relationships; and those with depression or anxiety states responding to treatment - either as inpatients or as outpatients.
3. Moderate Disability - includes those with definite impairment of work status unless completely unskilled; deterioration in personal relationships and subjective well-being; severe and prolonged depression not responding fully to inpatient treatment even in the absence of other changes.

4. Severe Disability. In this section are included those with personality and intellectual impairment, or both, severe enough to lead to unemployment or a pronounced fall in employment status; marked deterioration in family relationships and subjective well-being, or both; and severe prolonged depression not responding fully to inpatient treatment, in conjunction with otherwise moderate intellectual and personality damage. This category contains patients who are largely incapable of independent life and need institutional care or the protection of a tolerant family.

5. Very Severe Disability - includes those who are grossly demented, inaccessible, bedfast and often grossly dysphasic. Two such cases were being nursed at home, the others were in institutions.

In the second study of 30 women with middle cerebral (H.C.) aneurysms, these ratings were completed at the end of the interview.

Employment Status

This rating is described in the text of Chapter 7.

"VERBATIM" DESCRIPTION OF PERSONALITY CHANGE

The clinical description and classification of personality change is difficult and inexact and there is a natural
tendency to place people in ready-made categories derived from the work of others, when attempting qualitative judgments. In order to supplement the simple and subjective ratings of personality impairment, already described, and to avoid the bias of preconceived ideas — especially those of the "frontal lobe syndrome" in patients with frontal craniotomy scars — the following method was used in addition.

In 242 of the 261 cases, another informant was interviewed and each was asked at the beginning of the interview and in the absence of the patient, "What changes do you notice in (the patient) since the illness?". The answers were written down verbatim and considered separately from later statements made in answer to direct questions. (The method described here will appear more clear when the results are given in Chapter V).

The verbatim statements were later studied and grouped into 17 categories by the apparent meaning; the categories themselves being derived from the statements and not preconceived. For example, two patients described as follows:

(a) "He is much more easily worried and more bad tempered"; and
(b) "He seems to worry now over the least little thing and gets irritable whenever we interrupt him". Both appear in the verbatim description categories as "more easily worried" and "more irritable".

A patient described by, "She is so untidy now and never seems to care about doing a job properly", appears as "less obsessive".

"Verbatin" descriptions indicating changes in personality are available for 136 patients with single aneurysms, which is more than the number rated as showing overall personality impairment
in Chapter V, because in some the changes seemed trivial enough to rate the patient as "Unimpaired" overall. Also, 13 patients who were rated as "Improved" (see later), have verbatim descriptions applied to them. Naturally, more than one verbatim description was made of many patients. (The no aneurysm and multiple aneurysm groups are small and have been excluded from the results given in Chapter V). There are various pitfalls in this method, of which the most obvious is that the comments made by a spouse must depend on the "set" created by the patient's condition. Thus, if a man is hemiplegic and dysphasic, his wife may not consider it necessary to describe him as lacking in vitality. If he seems outwardly normal, she may be at pains to impress on the interviewer that he has really changed in some way, perhaps to emphasise the burden she has to bear; as a helpless invalid may be easier to live with than someone who can still work but is irritable and disinhibited. Another difficulty is that some of those patients were also depressed, as described in Chapter VII. If the depressive state was chronic, this is bound to be reflected in the verbatim descriptions. Although an attempt was naturally made to distinguish between fluctuating affective states and enduring personality changes, the separation cannot be complete and is, in some ways, artificial. With those patients rated as having moderate personality impairment, this was a particular problem. A third disadvantage is that subtleties and complex details are largely ignored; but the advantages seemed to outweigh the disadvantages in this single interview situation.
An attempt was made at follow up to get an idea of the personality of every patient and was unobtainable in only 5, who were impossibly demented and had no relative available. While appreciating the difficulty and uncertain validity of the assessments, each patient was rated on three point scales of below average, average and above average on four personality factors: Proneness to Anxiety; Obsessionalism; Energy; and Outgoingness.

These factors were chosen because they could be described by someone else without having to speak to the patient at all, if, for example, he was demented or aphasic. Also, clinical experience with brain damaged patients had suggested that they were clinically meaningful.

The following questions were asked about everybody for whom another informant was available and modified suitably if only the patient was seen. Other questions were asked arising from the answers and, of course, other relevant information was also given during the interview. The ratings were then made subjectively. The method is not sophisticated and covers only a small range of human variation but it is felt that it does provide at least some quantification of clinical judgments of a type made regularly in clinical psychiatry.
1. What sort of a person was he? How would you describe his temperament?

2. Was he a worrying sort of person? If so - how did that show? What sort of things worried him?

3. Did he have any particular fears - of heights, insects, travelling on tube trains - that sort of thing?

4. Did he tend to remain calm when little things went wrong? (Give examples.)

5. Was he an orderly sort of person? Liked everything to be neat and tidy? (If so) - Was he unusually fussy (or was she unusually houseproud)? (Give examples).

6. Was he the sort of person who did not like a regular routine in life? (Give examples).

7. Was he careless about his appearance, etc.? 

8. Did he check and double check? (Examples of gas taps and light switches).

9. Would you describe him as reserved or shy? How did that show?

10. Did he find it easy to get on with people?

11. How much time did he spend with friends? In clubs, etc.?

12. How did he like to spend his spare time and holidays?

13. What did he like to do at the end of a day's work and at the weekend?

14. Was he the sort of person who was always on the go?

Table I shows the distribution of the ratings on these factors for the whole series.
Table 1

<table>
<thead>
<tr>
<th>Personality Trait</th>
<th>Below Average</th>
<th>Average</th>
<th>Above Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety Prone</td>
<td>54</td>
<td>100</td>
<td>102</td>
</tr>
<tr>
<td>Obsessional</td>
<td>20</td>
<td>112</td>
<td>124</td>
</tr>
<tr>
<td>Energy</td>
<td>12</td>
<td>137</td>
<td>107</td>
</tr>
<tr>
<td>Obsessional</td>
<td>54</td>
<td>109</td>
<td>33</td>
</tr>
</tbody>
</table>

None of these ratings has a normal distribution.

It may be that the author's standards of normality are misplaced, perhaps as a consequence of deriving them from clinical experience with psychiatric patients, rather than that the patients in this series are unusually anxious, obsessional, energetic and outgoing.

Table 36 in Chapter IX shows the distribution in those with and without a past history of migraine; there are only trivial differences, although the author's bias was to expect patients with migraine to be both more anxiety prone and more obsessional than those without. To this extent then the ratings can be regarded as free of bias of that type.

The previous personality ratings for 112 men and 144 women separately are shown in Table 2.
The women are significantly more anxious, when below average and average are grouped together.

\[ \chi^2 = 21.4, \text{ 1 d.f.} \quad p = 0.001 \]

There are no other significant differences.
FAMILY HISTORY OF PSYCHIATRIC ILLNESS

This is defined as any treatment for a psychiatric disturbance of any severity and, in most cases, referred to the use of psychotropic drugs by the general practitioners. The unsupported statement of the patient or other informant was accepted as adequate and it was not checked with the doctor concerned.

FAMILY HISTORY OF PSYCHIATRIC ILLNESS

This is defined as inpatient or outpatient treatment for a disturbance of any severity in a parent, sibling, or child. Again, the unsupported statement of the patient or other informant was accepted.

MIGRAINE AND OTHER HEADACHES

The classification of the Ad Hoc Committee (1962) has been followed with one modification. They described vascular headache of migraine type as having the following characteristics: recurrent attacks widely varied in intensity, frequency and duration. Commonly unilateral in onset; usually associated with nausea and sometimes with vomiting; and sometimes preceded by or associated with conspicuous sensory, motor and mood disturbances. Often familial in incidence.

The two following subgroups are included as migraine in this study:

1. Classic migraine - with sharply defined transient visual and other sensory or motor prodromata.
2. Common migraines — without striking

moderates and less often unilateral.

Other episodic and throbbing headaches, many of

then also probably vascular, are grouped with "other headaches"

and not with migraine, which the ad hoc Committee considered

then to be.
CHAPTER IV

QUALITATIVE ANALYSIS OF THE TRIAL OF OPERATIVE AND CONSERVATIVE TREATMENT

INTRODUCTION TO CARETAKERS

The composition of the series was described in Chapter III - 30 had no aneurysms, 7 multiple aneurysms, and 224 single aneurysms - of whom 61 had A.C., 72 had P.C. and 71 had N.C. aneurysms.

Conservative treatment in this trial means six weeks' rest in bed, with any necessary medical measures, including hypotensives, followed by gradual mobilisation.

Surgical treatment is of three main types: common carotid ligation (C.C.L.); direct attack on the aneurysm (D.A.); and proximal occlusion of the vessel from which the aneurysm arises, used only for anterior communicating aneurysms in this series; the operations used in the cases considered in the comparisons are described later.

Fifteen of those with single aneurysms were for technical reasons regarded as unsuitable for the trial or had an operation in order to evacuate a life-threatening haematoma. These and the seven patients with multiple aneurysms are not considered further here.

When the study was carried out, all cases with A.C. aneurysms and women with N.C. aneurysms were still in the trial if they fulfilled the criteria but those with P.C. aneurysms and men with N.C. aneurysms had been out of the trial and, therefore, treated operatively, since 1959 and
1961 respectively. To increase the size of the groups under comparison, all cases in the two latter categories have been included in the "trial comparisons" if they fulfilled the original trial criteria - even if they were admitted after the trial had been abandoned and surgery recognised as the treatment of choice. Such cases are described as "suitable for trial" in this paper. Of the 224 patients with single aneurysms, 163 were actually in the trial and a further 46 were suitable for trial (but treated surgically). There are consequently 209 patients who may be used to provide valid comparisons of the incidence of psychiatric and other sequelae - in 125 operatively and 84 non-operatively treated cases. Table 3 shows the sex and treatment distribution. (Of the 30 cases with no aneurysm, 14 were men and 16 women.)

**TABLE 3**

<table>
<thead>
<tr>
<th></th>
<th>A.C.</th>
<th>M.C.</th>
<th>P.C.</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>F</td>
<td>M</td>
<td>F</td>
</tr>
<tr>
<td>Op.</td>
<td>16</td>
<td>18</td>
<td>18</td>
<td>21</td>
</tr>
<tr>
<td>Cons.</td>
<td>25</td>
<td>17</td>
<td>10</td>
<td>19</td>
</tr>
<tr>
<td>Totals</td>
<td>41</td>
<td>35</td>
<td>28</td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>76</td>
<td>68</td>
<td>65</td>
<td></td>
</tr>
</tbody>
</table>
In order to check the validity of the comparisons between the 125 operatively treated and 84 conservatively treated cases with single aneurysms, a series of \( \chi^2 \) estimations was made on the incidence of the following 11 pretreatment factors in the two groups: sex; age; side of aneurysm; coma following the haemorrhage; level of consciousness on admission (alert or not alert); the incidence and severity of lateralizing neurological signs; blood pressure on admission (above 160/90 or not); incidence of haematomata and of cerebral arterial spasm on angiography; history of previous subarachnoid haemorrhage; and the results of carotid compression before angiography. No significant differences emerged, either between these two groups or in the distribution of the factors between operative and conservative subgroups for each type of aneurysm separately. They may therefore be justifiably compared for the results of treatment.

Table 4 shows the type of operation used in the cases compared for the trial.
TABLE 4

OPERATION USED IN 125 OPERATIVELY TREATED CASES (TRIAL AND SUITABLE-FOR-TRIAL CASES ONLY)

<table>
<thead>
<tr>
<th></th>
<th>A.C.</th>
<th>H.C.</th>
<th>P.C.</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>F</td>
<td>M</td>
<td>F</td>
</tr>
<tr>
<td>C.C.L.</td>
<td>5</td>
<td>9</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>D.A.</td>
<td>3</td>
<td>5</td>
<td>16</td>
<td>21</td>
</tr>
<tr>
<td>Proximal occlusion</td>
<td>8</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Totals</td>
<td>16</td>
<td>18</td>
<td>18</td>
<td>21</td>
</tr>
</tbody>
</table>

C.C.L. = Common carotid ligation.
D.A. = Direct attack on aneurysm after craniotomy, with clipping or gauze wrapping of aneurysm.

Proximal occlusion = Of the supplying vessel.
1. **Overall Mental Disability**

Using the five-point scale as a measure of overall mental disability, Table 5 shows the results in the 209 aneurysm cases used in the trial comparisons. Comparable figures for the 30 cases with no demonstrated aneurysm are also given.

**TABLE 5**

RESULTS IN 209 ANEURYSM CASES (TRIAL COMPARISONS) AND 30 NO-ANEURYSM CASES

OVERALL MENTAL DISABILITY

<table>
<thead>
<tr>
<th></th>
<th>Absent</th>
<th>Mild</th>
<th>Mod.</th>
<th>Severe</th>
<th>V.Severe</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>P.C.</td>
<td>33 (51)</td>
<td>15 (23)</td>
<td>11 (17)</td>
<td>6 (9)</td>
<td>0 (0)</td>
<td>65 (100)</td>
</tr>
<tr>
<td>N.C.</td>
<td>26 (38)</td>
<td>14 (21)</td>
<td>12 (17)</td>
<td>11 (16)</td>
<td>5 (7)</td>
<td>68 (99)</td>
</tr>
<tr>
<td>A.C.</td>
<td>35 (46)</td>
<td>22 (29)</td>
<td>14 (18)</td>
<td>3 (4)</td>
<td>2 (3)</td>
<td>76 (100)</td>
</tr>
<tr>
<td>All an.</td>
<td>94 (45)</td>
<td>51 (24)</td>
<td>37 (18)</td>
<td>20 (10)</td>
<td>7 (3)</td>
<td>209 (100)</td>
</tr>
</tbody>
</table>

Percentages in parentheses.

Comparing all those with aneurysms to those without and simplifying the results into absent/mild and moderate/severe/very severe, we find that morbidity is significantly greater in those with aneurysms ($\chi^2 = 5.593$ with 1 degree of freedom, $p < 0.02$).
Those with M.C. aneurysms do worse than those with A.C. or P.C. aneurysms. Again simplifying Table 3 as above and considering only the aneurysm cases, then $\chi^2 = 5.345, p<0.1$ with 2 degrees of freedom, not quite reaching conventional levels of significance. If, however, M.C. aneurysms are compared with the others as a whole, then $\chi^2 = 5.320, p<0.05$ with one degree of freedom.

The distribution of central nervous system signs at follow-up is shown in Table 6.

### TABLE 6

**C.N.S. SIGNS AT FOLLOW-UP**

<table>
<thead>
<tr>
<th></th>
<th>Absent</th>
<th>Mild</th>
<th>Mod.</th>
<th>Severe</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>P.C.</strong></td>
<td>40 (62)</td>
<td>12 (18)</td>
<td>6 (9)</td>
<td>7 (11)</td>
<td>65 (100)</td>
</tr>
<tr>
<td><strong>M.C.</strong></td>
<td>27 (39)</td>
<td>22 (32)</td>
<td>7 (10)</td>
<td>12 (18)</td>
<td>68 (99)</td>
</tr>
<tr>
<td><strong>A.C.</strong></td>
<td>49 (64)</td>
<td>20 (26)</td>
<td>3 (4)</td>
<td>4 (5)</td>
<td>76 (99)</td>
</tr>
<tr>
<td><strong>All an.</strong></td>
<td>116 (55)</td>
<td>54 (26)</td>
<td>16 (8)</td>
<td>23 (11)</td>
<td>209 (100)</td>
</tr>
<tr>
<td><strong>No an.</strong></td>
<td>24 (80)</td>
<td>5 (17)</td>
<td>1 (3)</td>
<td>0 (0)</td>
<td>30 (100)</td>
</tr>
</tbody>
</table>

Percentages in parentheses
In patients with aneurysms, the neurological signs at follow up were significantly more frequent and severe than on admission ($\chi^2 = 6.39$, $p < 0.02$, with one degree of freedom). In those without aneurysms, the incidence of the neurological signs was almost exactly as on admission. The fact that no increase occurs in the signs of the no-aneurysm cases indicates that the ratings are probably reliable, as these latter patients have very low rebleed rates and no possibility of postoperative complications. (The ratings of C.N.S. signs on admission are derived from surgical notes made by a number of people over several years, whereas the follow-up examinations were all made by me).

There is a significant difference in the incidence of neurological signs at follow up in the three main aneurysm groups, being greatest in those with N.C. and least in those with A.C. aneurysms. (When grouped as absent/mild and moderate/severe, $\chi^2 = 8.407$ with 2 degrees of freedom, $p < 0.025$).

The fact that those with N.C. aneurysms have a higher incidence of signs is at least a partial - and possibly a complete - explanation for their increased overall mental morbidity, demonstrated in Table 5. Equally, those with no aneurysms have the fewest neurological signs and the lowest mental disability, while those with A.C. and P.C. aneurysms have intermediate positions in these respects. The evidence is not set out fully in this chapter but, as would be expected, both personality and intellectual impairment are clearly associated with brain damage, of which neurological signs are
unequivocal evidence. Psychiatric symptoms of anxiety and depression are not infrequent in those without neurological signs but those with brain damage have a greater tendency to such symptoms than those without. (These aspects are described in detail in later chapters).

There are no significant differences between the overall mental disability of the operatively and the conservatively treated groups, between men and women, nor between those with right-sided as opposed to left-sided aneurysms. A.C. aneurysms are essentially midline structures, although they may damage one side of the brain more than the other on rupture; they are, therefore, not included in this comparison.

Although when the trial material is considered as a whole there are no sizable differences between operative and conservative groups, between men and women, or between right- and left-sided aneurysms, this is not so when those with M.C. aneurysms are considered separately. Patients with M.C. aneurysms do worse overall than those with other aneurysms (Table 5), and it can be seen from Table 7 that this is entirely because of the worse results with surgery in this group. The difference is most pronounced in the case of women with left-sided aneurysms but men with right-sided aneurysms also fare worse with surgery than with conservative treatment.

Table 7 (a) - that is, for women with M.C. aneurysm - has been analysed using Cochran's criterion, testing for the difference in mental disability when grouped as absent/mild and moderate/severe/very severe, and correcting for the differences between right- and left-sided lesions. The test is two-tailed.
and the result is between the 7% and 8% levels of significance (t = 1.779), not quite reaching conventional levels.

### Table 7

**H.C. Analyses in Trial Comparisons.**

**Overall Mental Disability**

<table>
<thead>
<tr>
<th></th>
<th>Absent</th>
<th>Mild</th>
<th>Mod.</th>
<th>Severe</th>
<th>V. Severe</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>(a) Women</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R Op.</td>
<td>4</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>R Cons.</td>
<td>4</td>
<td>4</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>L Op.</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>11</td>
</tr>
<tr>
<td>L Cons.</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td><strong>13(32)</strong></td>
<td><strong>11(27)</strong></td>
<td><strong>7(17)</strong></td>
<td><strong>5(12)</strong></td>
<td><strong>4(10)</strong></td>
<td><strong>40</strong></td>
</tr>
<tr>
<td><strong>(b) Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R Op.</td>
<td>4</td>
<td>1</td>
<td>3</td>
<td>4</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>R Cons.</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>L Op.</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>L Cons.</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td><strong>13(46)</strong></td>
<td><strong>3(11)</strong></td>
<td><strong>5(18)</strong></td>
<td><strong>6(21)</strong></td>
<td><strong>1(4)</strong></td>
<td><strong>28</strong></td>
</tr>
</tbody>
</table>

Percentages in parentheses.
Table 8 shows M.C. aneurysm cases of both sexes, divided by type of treatment for added clarity.

**TABLE 8**

M.C. ANEURYSMS IN TRIAL COMPARISONS
OPERATIVE VERSUS CONSERVATIVE TREATMENT

<table>
<thead>
<tr>
<th></th>
<th>Absent</th>
<th>Mild</th>
<th>Mod.</th>
<th>Severe</th>
<th>V. Severe</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Op.</td>
<td>12 (31)</td>
<td>7 (16)</td>
<td>8 (21)</td>
<td>8 (21)</td>
<td>4 (10)</td>
<td>39 (101)</td>
</tr>
<tr>
<td>Cons.</td>
<td>14 (48)</td>
<td>7 (24)</td>
<td>4 (14)</td>
<td>3 (10)</td>
<td>1 (3)</td>
<td>29 (99)</td>
</tr>
<tr>
<td>Totals</td>
<td>26 (38)</td>
<td>14 (21)</td>
<td>12 (17)</td>
<td>11 (16)</td>
<td>5 (7)</td>
<td>68 (99)</td>
</tr>
</tbody>
</table>

Percentages in parentheses.

Combining the results into two categories of absent/mild and moderate/severe/very severe, the latter contains 52% of the operative and only 27% of the conservative groups. The use of $\chi^2$ test shows the difference to be significant at the 5% level. ($\chi^2 = 3.892, p < 0.05, 1 \text{ d.f.}$)

Comparable separate tables for A.C. and P.C. aneurysm groups are not given but, in fact, those treated by surgery have a lower overall mental disability than the conservatively treated patients. The differences are not significant. They are, however, marked enough to even out the operative versus conservative comparisons when all aneurysms are considered together.

2. Using Separate Rating Scales

The results so far have been set out with the use of only the rating of overall mental disability. As was explained earlier, each patient was originally rated on four scales, each of four points, for personality impairment, psychiatric symptoms, intellectual impairment and physical disability. These are set out as histograms in Figs. 1 and 2. It must be remembered that in the overall mental disability ratings psychiatric symptoms were given relatively less weight, as in many cases they are transient, whereas personality and
intellectual impairment are permanent. The histographic method, with four point-rating scales, makes differences between groups more apparent than did the overall rating method, but there are no major inconsistencies. The rating scales were also assessed statistically using t tests and the results of these will be mentioned where applicable. Fig. 1 shows that the aneurysm cases are more severely affected than those with no aneurysm, and also shows that in the latter group there is very little personality impairment, intellectual impairment or physical disability rated as moderate or severe. One man, the only no-aneurysm case with C.N.S. signs, rated moderately severe at follow up, is responsible for the $\frac{3}{5}$ shown in each of those categories. Patients with no aneurysm have, however, a similar incidence of psychiatric symptoms to patients with aneurysms. Seven of the 30 patients without aneurysms had clinically significant anxiety or anxiety/depressive states at the time of their haemorrhage but only two out of seven are rated here as being worse than before the stroke. (This aspect is dealt with in greater detail in a later chapter.)
All Aneurysm cases in trial comparisons.

- mild disability
- moderate & severe disability

O: operated cases
C: conservative
pers: personality impairment
psych: psychiatric symptoms
intel: intellectual impairment
dis: physical disability

fig. 1

(a) All Aneurysm cases (209).

fig. 2

(a) Ant. Communicating/Ant. Cerebral (76).

(b) Middle Cerebral (68).

(c) Post. Communicating (55).
The columns representing the aneurysm cases in Fig. 1 show a slight worsening in the operative groups, which did not appear in the ratings of overall mental morbidity, but the differences are not significant.

Fig. 2 shows the results in the three main aneurysm groups, from which it can be seen that in the P.C. and A.C. cases, those treated operatively do somewhat better than the others but the differences are not significant. M.C. cases do considerably worse with surgery, however, particularly where moderate damage and severe damage are concerned. Using t tests and considering the sexes and side of aneurysm separately (although the histograms do not show them separately), the significant differences are as follows:

- For personality impairment in women with left-sided aneurysms, operative cases do significantly worse than conservative cases at the 1% level ($t = 2.9$, $n = 17$, $p < 0.01$).
- For intellectual symptoms in the same cases, the difference is significant at the 2% level ($t = 2.8$, $n = 17$, $p < 0.02$). None of the other differences reach conventional levels of significance. (The use of t tests as rating scales is, however, of dubious validity. They are retained here despite that as the method is widely used and the results in keeping with the other method of assessment.)

**Epilepsy**

In the whole case material of 261 patients, there were 22 who had had epileptic disturbances. These all had either an M.C. aneurysm (17 cases out of 71, or 24%), or a P.C. aneurysm (5 cases out of 72, or 7%). The overall incidence of epilepsy in those with single aneurysms was, therefore, 22 out of 224 cases, or approximately 10%. The epilepsy was easy to control in all except four patients and in most there were only a few fits.

There are no significant differences between the operative and conservative groups, but epilepsy is significantly more common in men than in women ($\chi^2 = 8.556$, $p < 0.01$, 1 d.f.)
and with M.C. aneurysm than with P.C. aneurysm ($\chi^2 = 5.665$, $p < 0.025$, 1 d.f.). None of the other differences is significant.

Epilepsy was in all but six cases associated with some abnormal neurological signs, mostly moderately severe or severe, and some other mental disability was present in all but five cases. Fourteen of the 19 patients with epilepsy had had a haematoma demonstrated by angiography - an incidence of 74%. Overall 40% of all patients with M.C. and P.C. aneurysms had haematomata.

Statistical Note

The use of a separate measure of overall mental disability has the advantage that, in making comparisons of the results of operative and conservative treatment, each patient is being considered once only, which makes for clarity. It would not have been suitable to rely on the sum of the other rating scales, as the numbers used are only labels applied to categories and bear no regular and meaningful mathematical relationships to each other.

DISCUSSION

In this study, the operative and conservative groups have been shown to be comparable for all the factors used for stratification in the neurosurgical trial as well as for a few others listed earlier. This is to be expected, as most of the patients had been randomly allocated in the trial itself and the others all chosen as suitable for the trial. The mortality after admission to hospital is high, as pointed out in Chapter I, and for the three aneurysm groups studied the six month survival rates of operatively and conservatively treated combined were approximately: P.C. aneurysm 69%, M.C. aneurysm 72% and A.C. aneurysm 56%. These are better figures than described in the earlier follow up studies, presumably as a result of advances in conservative as well as in operative treatment.
The study shows that the psychiatric morbidity of the survivors is also high. Only 45% of those in the trial comparisons were considered to be unimpaired and 24% mildly affected. Eighteen per cent were moderately, 10% severely and 5% very severely damaged - those last being demented and inaccessible. H.C. aneurysm is associated with a significantly higher morbidity than either of the other main aneurysm types and most of the serious mental disability was associated with neurological evidence of brain damage. Depressive and anxiety states, sometimes very severe, were quite commonly found in the absence of such damage, however. Those patients with no demonstrable aneurysm had a lower physical, personality and intellectual morbidity than the others, a finding which was clearly associated with a lower incidence of brain damage; but still had a rather high incidence of psychiatric symptoms, as was found by Tappura (op. cit) and Björkesten and Troup (op. cit).

Depressive states are considered in detail in Chapter VII.

Thirteen patients were unexpectedly found to have "improved personalities". This is considered in detail in Chapter V, with other aspects of personality change.

The most important practical finding is that patients with H.C. aneurysms did worse with operative treatment than when treated conservatively. This was most noticeable in women but it also occurred in men. Men, however, have a better chance of survival with surgery and it is probable that their increased morbidity is because some of those more severely damaged, who might otherwise have died, have been saved by surgery; and the findings here do not affect the policy of operating in these patients.
In women, on the other hand, there is no increased survival rate with surgery and it is probable that the cause lies in the morphological and neuropathological differences in H.C. aneurysms in women (Crompton, op cit). These aneurysms are more difficult to treat and the added brain damage due to surgical exposure of the aneurysm is presumably responsible for the worse results after operation.

It is understandable that left-sided aneurysms should do worse with surgery, as in the case with women here, when the dominant hemisphere is involved. It is not clear why in men right-sided aneurysms should lead to a higher morbidity. Inspection of the original findings does not suggest a markedly different pattern of disability, although there is a tendency in H.C. aneurysms for right-sided lesions to be associated with a greater incidence of psychiatric symptoms and left-sided lesions to have more intellectual impairment. (This does not apply to aneurysms at other sites, however).

The incidence of epilepsy, 10%, along with its distribution, is very similar to that found by Rose and Sarner (1965), who studied a larger material from the same neurosurgical department. There is a full discussion of the subject in their paper.

Employment status has not been considered separately as a measure of recovery, although always taken into account, and it is dealt with in more detail in Chapter V. Men in some occupations, mainly in large organisations, seemed often to be accepted by the management and carried by their workmates, even when clearly incapable of full work. This was found in the case of a
senior managerial executive who continued to draw full salary although suffering from marked intellectual and personality impairment; but it was much commoner in lower employment levels. Self-employed people doing skilled or professional work seemed to suffer more from any given level of disability. Other patients were found who had retired or had changed their work, on medical grounds, unnecessarily—often because of exaggerated fears of exertion. The whole matter is closely linked with economic conditions and attitudes towards the handicapped, so that one would expect differences between various countries and various periods.

Supplementary Series of 30 Women with H.C. Aneurysms

As just described, women with H.C. aneurysms treated surgically did poorly in the trial compared both to the men in the same group and to both sexes in the other groups. Before the results of this first psychiatric follow up was known, the neurological team altered their management of these patients by delaying operation to the fourth, fifth or sixth days after the bleed, instead of operating as soon as possible. In order not to gain a spurious impression, patients were still allocated to operative or conservative treatment as soon as the diagnosis was made, so that, if a patient allocated to surgery died before operation was performed, she still fell into the operative group.

This change in policy has, in fact, led to a marked improvement in the mortality and neurosurgical morbidity in women with H.C. aneurysms but the trial is still in progress and no
further results have yet been published. (Richardson, 1966, private communication).

In order to check on the psychiatric morbidity with the new surgical policy, a second series of 30 women with M.C. aneurysms was interviewed, the composition of which is described in Chapter III. They were all the women, living in southeast England, who had been in the modified trial up to the time of the study. Nineteen were treated by surgery and 11 conservatively. In the operated group, 5 had common carotid ligation (C.C.L.) and 14 direct attack on the aneurysm (D.A.).

There were no significant differences between the operated and conservative groups between this second series and the first on any of the factors described for the first series: age, side of aneurysm; coma following the haemorrhage; level of consciousness on admission; the incidence and severity of C.N.S. signs; blood pressure on admission; incidence of haematomata or spasm on angiography; history of previous subarachnoid haemorrhage; or the results of carotid compression.

Table 9 shows the results in terms of the overall mental morbidity, laid out with the relevant figures from the first trial for easy comparison.
Table 2.

**SUBFEMORAL A.C. ANEURYSM TRIAL**

**(20 WOMEN ONLY)**

OVERALL MENTAL DISABILITY AND TREATMENT

<table>
<thead>
<tr>
<th>Operation (19)</th>
<th>Overall Mental Disability</th>
<th>Conservative (11)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td></td>
</tr>
<tr>
<td>4 (4) 3 (1)</td>
<td></td>
<td>4 (4) 2 (4)</td>
</tr>
<tr>
<td></td>
<td>Mild</td>
<td></td>
</tr>
<tr>
<td>8 (3) 5 (2)</td>
<td></td>
<td>1 (4) 2 (2)</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td></td>
</tr>
<tr>
<td>1 (2) 0 (2)</td>
<td></td>
<td>1 (2) 0 (1)</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td></td>
</tr>
<tr>
<td>1 (0) 1 (4)</td>
<td></td>
<td>0 (0) 1 (1)</td>
</tr>
<tr>
<td></td>
<td>Very severe</td>
<td></td>
</tr>
<tr>
<td>0 (1) 0 (2)</td>
<td></td>
<td>0 (0) 0 (1)</td>
</tr>
<tr>
<td>10 (10) 9 (11)</td>
<td></td>
<td>5 (10) 5 (9)</td>
</tr>
</tbody>
</table>

Figures in brackets are from the first trial series of 40 women with M.C. aneurysm.

The main difference is in the left-sided operative subgroup. In the first series, 8:11 had moderate, severe, or very severe disability; compared to 1:9 in the second series. The numbers involved are small and formal statistical comparison is, perhaps, inappropriate but, using Yates correction, $\chi^2 = 5.062$, 1 d.f., $p < 0.05$.

In the other subgroups, the second series patients did at least as well as the others and there has been no apparent change in the results of conservative treatment.
It may be that the difference is random but, associated as it is with an improvement in mortality also, it probably represents a genuine improvement in the fate of those operated on. There is, however, no evidence, as yet, that the operated do better than the conservatively treated, as of the former 3:19 have moderate disability or worse, compared to all of the latter.

No more detailed breakdown of the physical, personality, intellectual and affective changes in this series of 30 women is given here. They bore practically the same relationships to each other as did those demonstrated in the first series — which are dealt with in detail in later chapters.
In this chapter, only the patients in the first series of 261 patients are considered. The rating scales and definitions used were described in Chapter III.

RESULTS

Overall Personality Impairment

Of the 261 patients, 100 (41%) were considered impaired, of whom 58 (22%) had mild, 39 (15%) moderate and 11 (4%) severe impairment. A further 13 (5%) showed favourable personality change. They do not enter into any of the considerations of personality impairment. Clinical descriptions will be given in a later section.

1. Association of Personality Impairment and C.N.S. Signs

There was a striking association with neurological signs, as shown in Table 10, and patients without C.N.S. signs had an excellent chance of escaping with little or no personality damage. On the whole, the severity of personality change increases with the degree of neurological signs, although there is considerable overlap and 9 patients with severe signs have absent or mild personality impairment. (Considering personality impairment as absent or present only, and C.N.S. signs as absent, mild, or moderate/severe, then \( \chi^2 = 57.27, 2 \text{ d.f.}, p < 0.0001 \)).
Table 10

ASSOCIATION OF C.N.S. SIGNS AND OVERALL PERSONALITY IMPAIRMENT.

<table>
<thead>
<tr>
<th>C.N.S. Signs</th>
<th>Overall Personality Impairment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Absent</td>
</tr>
<tr>
<td>Absent</td>
<td>111</td>
</tr>
<tr>
<td>Mild</td>
<td>36</td>
</tr>
<tr>
<td>Moderate</td>
<td>2</td>
</tr>
<tr>
<td>Severe</td>
<td>4</td>
</tr>
<tr>
<td>TOTALS</td>
<td>153</td>
</tr>
</tbody>
</table>

Table 11 shows that C.N.S. signs are significantly more common in those with aneurysms than without ($\chi^2 = 12.699$, 2 d.f., $p < 0.01$); if signs are considered as absent, mild, or moderate/severe. It also shows that of the single aneurysm groups signs are significantly more common with M.C. than with A.C. or P.C. aneurysms. ($\chi^2 = 12.469$, 2 d.f., $p < 0.01$).

Table 12 shows that the incidence of personality impairment in the diagnostic groups largely follows that of C.N.S. signs, although patients with P.C. aneurysms are spared severe personality change even with severe C.N.S. signs.
TABLE 11.

**C.H.S. SIGNS IN DIFFERENT ANEURYSM GROUPS**

<table>
<thead>
<tr>
<th>Severity of Personality Impairment</th>
<th>Absent M.C.</th>
<th>Absent P.C.</th>
<th>Absent A.C.</th>
<th>Multiple M.C.</th>
<th>Multiple P.C.</th>
<th>Multiple A.C.</th>
<th>No an.</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>26 (37)</td>
<td>42 (56)</td>
<td>52 (64)</td>
<td>2 (29)</td>
<td>26 (37)</td>
<td>26 (37)</td>
<td></td>
<td>148 (57)</td>
</tr>
<tr>
<td>Mild</td>
<td>25 (35)</td>
<td>14 (19)</td>
<td>21 (26)</td>
<td>3 (42)</td>
<td>3 (10)</td>
<td>3 (10)</td>
<td></td>
<td>66 (25)</td>
</tr>
<tr>
<td>Severe</td>
<td>13 (18)</td>
<td>9 (13)</td>
<td>4 (5)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td></td>
<td>26 (10)</td>
</tr>
<tr>
<td>Totals</td>
<td>71</td>
<td>72</td>
<td>81</td>
<td>7</td>
<td>30</td>
<td>261</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Percentages in parentheses.

TABLE 12.

**ANEURYSM GROUP AND OVERALL PERSONALITY IMPAIRMENT**

<table>
<thead>
<tr>
<th>Severity of Personality Impairment</th>
<th>Absent M.C.</th>
<th>Absent P.C.</th>
<th>Absent A.C.</th>
<th>Multiple M.C.</th>
<th>Multiple P.C.</th>
<th>Multiple A.C.</th>
<th>No an.</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>34 (40)</td>
<td>42 (56)</td>
<td>49 (60)</td>
<td>4 (57)</td>
<td>24 (30)</td>
<td>24 (30)</td>
<td></td>
<td>153 (59)</td>
</tr>
<tr>
<td>Mild</td>
<td>15 (21)</td>
<td>15 (21)</td>
<td>21 (26)</td>
<td>2 (29)</td>
<td>5 (10)</td>
<td>5 (10)</td>
<td></td>
<td>58 (23)</td>
</tr>
<tr>
<td>Severe</td>
<td>15 (21)</td>
<td>15 (21)</td>
<td>7 (9)</td>
<td>1 (14)</td>
<td>1 (3)</td>
<td>1 (3)</td>
<td></td>
<td>39 (15)</td>
</tr>
<tr>
<td>Totals</td>
<td>71</td>
<td>72</td>
<td>81</td>
<td>7</td>
<td>30</td>
<td>261</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
If Table 12 is simplified to consider personality impairment as absent or present only, then it is significantly commoner in those with aneurysm than in those without. ($X^2 = 6.361, 1$ d.f., $p < 0.01$).

In those with single aneurysms, personality impairment is more common and more severe with M.C. aneurysms than with the others and there is a statistically significant difference between M.C. and A.C. aneurysms when personality impairment is grouped as absent/mild against moderate/severe. ($X^2 = 6.774, 1$ d.f., $p < 0.01$). P. C. cases fall between the other two.

Although C.M.S. signs were significantly commoner in association with greater age and raised blood pressure, these factors do not account for the differences between the various groups, (details omitted).

Moderate and severe personality impairment was significantly more common in men than in women, affecting 28: 112 men and 22:149 women ($X^2 = 4.265, 1$ d.f. $p < 0.05$), although C.M.S. signs were in fact slightly commoner in women.

There were no differences between the aneurysm groups in the proportions of those without C.M.S. signs who nevertheless had some personality impairment. Thus, 15:52 A.C. cases with no C.M.S. signs had some personality impairment, compared to 7:25 M.C. and 11:43 P. C. cases.
Central Infarction versus Hern (Superficial) Saccas

When the blood supply through a major artery fails, there may be widespread 'central' infarction in the territory it supplies; which, in this series happened most often after ligation of the common carotid artery for ruptured P.C. aneurysm. Rupture of an H.C. aneurysm is different in that it tends to cause most damage locally in the cortical and subcortical areas, which may be worsened by surgery if the temporal lobe has to be partially removed for good exposure. This local damage is particularly common and severe in women with H.C. aneurysms, because of the morphological characteristics of H.C. aneurysms in that sex, as described in Chapter II. The results of the trial of surgery set out in Chapter IV are in keeping with this difference.

There was significantly less personality damage in those with P.C. aneurysm and 'central' infarction than in H.C. patients, who had M.N.S. signs of similar severity, which is why no P.C. cases had severe personality impairment. Thus, of the 10 patients with severe M.N.S. signs, who had only mild or absent personality impairment, all but one had central infarction and only one an H.C. aneurysm. Further evidence of the relatively greater personality damage with H.C. aneurysms comes from studying separately those patients with absent or mild M.N.S. signs; of 51 H.C. cases with absent or mild signs, 10 had moderate or severe personality change; compared to only 12 of 129 A.C. and P.C. cases with similar M.N.S. signs, a significant difference. ($^2 = 3.692, 1 \text{ d.f.}, p = 0.05$).
From these findings, it seems that "death" of a large part of a hemisphere carries a lesser mental morbidity than does more superficial damage to the temporoparietal regions of apparently equivalent severity, if judged by neurological signs.

**Side of lesion**

There were only trivial differences in the incidence of personality impairment between those with left-sided and right-sided lesions. Considering only those with one-sided aneurysms or unilateral C.M.S. signs, 27:103 with right-sided lesions had moderate or severe personality impairment, compared to 20:98 with left-sided lesions.

2. **Association of Personality Impairment & Intellectual Impairment.**

The method of clinical intellectual assessment and the use of the slightly modified Inglis Paired Associate Test (P.A.T.) and the Benton Visual Retention Test (Benton) are described in Chapter III. More details of the intellectual changes themselves are given in Chapter VI.

Table 13 shows how the two clinical assessments of personality and intellectual impairment are related to each other.
### Table 13

**Clinical Assessment of Intellectual and Personality Impairment**

<table>
<thead>
<tr>
<th>Severity of Personality Impairment</th>
<th>Absent</th>
<th>Mild</th>
<th>Mod.</th>
<th>Severe</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>125 (59)</td>
<td>27 (35)</td>
<td>1 (4)</td>
<td>0 (0)</td>
<td>153 (59)</td>
</tr>
<tr>
<td>Mild</td>
<td>19 (13)</td>
<td>37 (47)</td>
<td>2 (9)</td>
<td>0 (0)</td>
<td>58 (22)</td>
</tr>
<tr>
<td>Mod.</td>
<td>8 (5)</td>
<td>14 (18)</td>
<td>16 (73)</td>
<td>1 (11)</td>
<td>39 (15)</td>
</tr>
<tr>
<td>Severe</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>3 (14)</td>
<td>8 (89)</td>
<td>11 (4)</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>152</td>
<td>76</td>
<td>22</td>
<td>9</td>
<td>261</td>
</tr>
</tbody>
</table>

Percentages in parentheses.

Although there is a very close relationship, there is one interesting discrepancy, in that there was a significant tendency for patients with A.C. aneurysms (who are likely to have frontal lobe damage) to have relatively less intellectual impairment in the presence of personality deterioration. Thus, 7 out of 7 A.C. patients with moderately severe personality impairment had absent or mild intellectual damage, as opposed to 14:30 of the N.C. and P.C. patients. (The numbers are small but do reach significance using Yates correction \(\chi^2 = 5.169, 1 \text{ d.f.}, p < 0.05\).)
Of the seven patients with moderately severe personality change but no intellectual impairment, all were right-handed men and 6 had right-sided lesions, divided equally between the 3 major aneruyn groups.

3. Psychological Test Scores

Patients with severe personality impairment are excluded as only three of them could attempt the tests.

(a) Benton Test. This did not differentiate between those with mild and those with moderate personality impairment, but did distinguish very significantly between those with and those without any degree of impairment, when set out as shown in Table 14.

<table>
<thead>
<tr>
<th>Personality Impairment</th>
<th>Benton Normal Absent</th>
<th>Benton Abnormal Present</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>96</td>
<td>58</td>
<td>144</td>
</tr>
<tr>
<td>Present</td>
<td>33</td>
<td>64</td>
<td>97</td>
</tr>
<tr>
<td></td>
<td>119</td>
<td>122</td>
<td>241</td>
</tr>
</tbody>
</table>

($X^2 = 15.323, 1$ d.f., $p<0.001$)
(b) **Inlaid Mixed Associate Test (I.M.A.T.)**

This test also did not distinguish significantly between those with mild and moderate personality impairment but did between those with and without any degree of impairment; most effectively when set out as in Table 15.

**TABLE 15.**

<table>
<thead>
<tr>
<th>Personality Impairment</th>
<th>Absent</th>
<th>Present</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>103</td>
<td>44</td>
<td>147</td>
</tr>
<tr>
<td>Present</td>
<td>30</td>
<td>56</td>
<td>86</td>
</tr>
<tr>
<td>Total</td>
<td>133</td>
<td>80</td>
<td>213</td>
</tr>
</tbody>
</table>

$\chi^2 = 11.757, 1$ d.f., $p < 0.001$

When used and considered as set out here, both tests are very significantly related to the presence or absence of personality impairment - the Benton to a rather higher degree.

(a) **Benton and P.A.T. Combined.** Those with and without personality impairment are distinguished even more significantly if both test scores are considered together. Of the 79 patients with mild or moderate personality impairment who completed both the Benton and P.A.T., only 14 had normal scores on both; 9 with mild and 5 with moderate
of the 134 patients with no personality deterioration, who did both
tests, 64 had normal scores on both. This
in an even more significant association
than shown by either test separately.
($X^2 = 19.239, 1 \text{ d.f.}, p < 0.001$).

4. Employment Status and Personality Impairment
This was found to be frequently misleading as an
index of personality and intellectual impairment, as indeed
it was as a measure of overall disability, as mentioned in
Chapter IV. Apart from the difficulty of rating housewives
accurately, self-employed people, especially those in more
responsible and intellectual work, suffered much more in this
respect than did those employed by big organisations, which
often were very generous about keeping on their old employees,
especially if they had given long service before the illness.
For example, one man, previously an extremely obsessive
carriage maker and joiner for British Rail, was still in full
time employment although he was apathetic, forgetful, affect-
ively flat and occasionally incontinent. He now worked as a
cleaner, although his wife had been told by his superiors that
he did almost no useful work. (Personality - moderately impaired).
Another man, who had built up an estate agents business single-
handed, was unable to carry on with this and had had to become
a part-time filing clerk, although only mildly impaired.
The ratings made for employment status are shown in Table 16. For men, the definitions are as follows:

(a) No change - either the same work or promotion; patient and informant agree that ability to do the work unimpaired. (Where doubt existed in the qualification, actual job status counted).

(b) Some impairment - either a fall in status, part-time work, or the same work less efficiently performed.

(c) Marked impairment - difficulty in obtaining and/or holding a job but patient capable of useful work.

(d) Unemployable.

In housewives and pensioners, the ratings were necessarily more subjective.
Table 16.

ANEURYSM GROUP & EMPLOYMENT STATUS

<table>
<thead>
<tr>
<th>Employment Status</th>
<th>A.C.</th>
<th>M.C.</th>
<th>M.G.</th>
<th>Mult.</th>
<th>No. am.</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No change</td>
<td>52 (64)</td>
<td>29 (41)</td>
<td>36 (50)</td>
<td>3 (43)</td>
<td>25 (83)</td>
<td>145 (56)</td>
</tr>
<tr>
<td>Some Impairment</td>
<td>17 (21)</td>
<td>11 (15)</td>
<td>20 (28)</td>
<td>1 (14)</td>
<td>3 (10)</td>
<td>52 (20)</td>
</tr>
<tr>
<td>Marked Impairment</td>
<td>6 (7)</td>
<td>17 (24)</td>
<td>12 (17)</td>
<td>2 (29)</td>
<td>2 (7)</td>
<td>39 (15)</td>
</tr>
<tr>
<td>Unemployable</td>
<td>6 (7)</td>
<td>14 (20)</td>
<td>4 (5)</td>
<td>1 (14)</td>
<td>0 (0)</td>
<td>25 (9)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>81</td>
<td>71</td>
<td>72</td>
<td>7</td>
<td>30</td>
<td>261</td>
</tr>
</tbody>
</table>

Percentages in parentheses.

Overall, 56% showed no change in employment status.

Again, patients with M.C. aneurysms fared worse than did the others and those with no aneurysms did markedly better. The impairment of employment status is, of course, due to a combination of physical and mental disability and, although the details are not given, it is correlated with the other measures of impairment, as expected.

SOME CLINICAL ASPECTS OF PERSONALITY IMPAIRMENT

The rating scales given earlier only outline the severity of the changes. Of the 11 patients with "severe" personality change, 8 were grossly demented and so also had "severe" intellectual impairment; of these, one was akinetic
and mute, lying permanently in the fetal position; five were
hemiplegic, deformed and bedfast in chronic sick hospitals;
while two, in a similar state, were nursed at home. Of the
3 with severe personality but only moderate intellectual change,
none was employable or apparently capable of independent life,
but each could speak coherently and attempt the tests given;
although apathetic and prone to catastrophic reactions when
stressed.

The commonest pattern of change in those with mild
or moderate impairment was to be described as having become
more irritable and anxious and to have less vitality, but
other syndromes were also seen, as described later, and some
illustrative case histories are also given.

VERBATIM DESCRIPTIONS OF PERSONALITY CHANGE

As was described in Chapter III, some descriptions
of personality changes given by relatives and other informants
were recorded verbatim, without leading questions, and later
put into 17 categories. This was done in order to avoid any bias
from the investigator contaminating the results of the qualitative
assessment.

Table 17 shows the frequency with which each verbatim
description was used, in descending order of frequency, for the
three single aneurysm groups. The multiple aneurysm and no
aneurysm patients have been excluded because of the small numbers
impaired.
TABLE 17.

CAT. CORIES OF VERBATIM DESCRIPTION IN 139 PATIENTS WITH SINGLE ANEURYSMS

<table>
<thead>
<tr>
<th>Category</th>
<th>Number described</th>
<th>A.C.</th>
<th>E.C.</th>
<th>H.C.</th>
<th>F.C.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less vitality</td>
<td>70:138</td>
<td>23:40</td>
<td>21:42</td>
<td>26:40</td>
<td></td>
</tr>
<tr>
<td>More irritable</td>
<td>58</td>
<td>21</td>
<td>16</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>More easily worried</td>
<td>51</td>
<td>14</td>
<td>21</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Dependent*</td>
<td>25</td>
<td>5</td>
<td>15</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Emotionally labile*</td>
<td>23</td>
<td>4</td>
<td>16</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Less obsessional</td>
<td>22</td>
<td>0</td>
<td>3</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Withdrawn</td>
<td>21</td>
<td>7</td>
<td>6</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Hostile</td>
<td>16</td>
<td>3</td>
<td>6</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Uninhibited*</td>
<td>16</td>
<td>10</td>
<td>1</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Less easily worried*</td>
<td>15</td>
<td>10</td>
<td>0</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Less irritable*</td>
<td>15</td>
<td>10</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Selfish</td>
<td>13</td>
<td>7</td>
<td>4</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Fatuous*</td>
<td>11</td>
<td>1</td>
<td>9</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>More obsessional</td>
<td>10</td>
<td>2</td>
<td>3</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>More affectionate</td>
<td>7</td>
<td>5</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Suspicious</td>
<td>7</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>More vitality</td>
<td>4</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

* indicates significant differences in distribution between the aneurysm groups.

Each of the verbatim personality descriptions shown in Table 17 was correlated with the following factors: sex, age, aneurysm, C.S.S. signs at follow up, side of lesion, clinical intellectual impairment, overall personality impairment and
psychiatric symptoms (of depression or anxiety). They were also correlated with some assessments of premorbid personality, which are described later. Only those findings which seem significant are given here.

The most important results were:

1. A.C. patients are most often described as Uninhibited, Less Easily Worried and Less Irritable, the terms being used least often for M.C. patients. These descriptions are mainly used about patients rated as having mild personality impairment, or in those rated as "improved" (see later), and the difference holds even if only patients with mild impairment are considered, as shown in Table 18, so that the differences cannot merely reflect severity of personality change.

Although the numbers are small, if A.C. cases are compared to M.C. and P.C. combined, the differences easily reach conventional significance, using Yates correction, for "less irritable" ($\chi^2 = 9.003$, 1 d.f., $p < 0.01$); and for "less easily worried", ($\chi^2 = 5.514$, 1 d.f., $p < 0.02$).

The classical idea of a separate frontal lobe syndrome is, therefore, supported by these results, as it is less strongly by the fact that the descriptions "More Affectionate" and "More Vitality" are also more commonly used about A.C. patients.

**TABLE 18.**

<table>
<thead>
<tr>
<th>Aneurysm Group</th>
<th>No.</th>
<th>Less Irritable</th>
<th>Less Easily Worried</th>
<th>Uninhibited</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.C.</td>
<td>15</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>P.C.</td>
<td>15</td>
<td>1</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>A.C.</td>
<td>21</td>
<td>10</td>
<td>9</td>
<td>7</td>
</tr>
</tbody>
</table>
2. Descriptions of "Dependent", "Emotionally Labile", and "Fatuous" are much commoner with M.C. than with P.C. or A.C. aneurysms combined. (If each description is considered separately, using Yates correction where necessary, then \( \chi^2 = 12.0 \) in each: 1 d.f., \( p < 0.001 \)). These descriptions were used almost always in patients rated as having moderate or severe impairment and who had moderate or severe signs; and the association with C.N.S. signs was in each of these at the 0.1% level. The significant tendency for M.C. cases to have relatively more severe personality impairment than the others is again demonstrated by the fact that, of 20 M.C. patients with moderate or severe signs, 13 were described as Fatuous, Dependent or Emotionally Labile; compared to 5 of 16 P.C. cases with similar signs. \( \chi^2 = 4.050, 1 \text{ d.f.}, p < 0.05 \).

3. Men were significantly more often described as "Less Vital" and "More Irritable" and "More Selfish", in keeping with their generally higher incidence of personality impairment, but not as "More Easily Worried" (details omitted).

4. The following verbatim descriptions were correlated with the presence and severity of C.N.S. signs at follow up at the 1% or 0.1% levels of significance: "Less Vital", "More Irritable" and "More Worried"; and these three were also much the most commonly used overall, as shown in Table 17. The three were used together
about 41 patients. "More Selfish" was also correlated at the 1/2 level and "Less Obsessational" at the 3/4 level but they were less often used.

5. Psychiatric symptoms of anxiety and depression (not considered in more detail here) were correlated at the 0.1/ level, with "Less Vitality" and "More Worried", and at the 1/2 level with "More Irritable".

6. Clinical Assessments of Intellectual Impairment also correlated at the 0.1/ level with each of the same three verbatim descriptions.

These three descriptions of Less Vitality, More Irritable and More Worried are not only the component used in this series but are correlated with the other major measures of physical and mental disability and can be taken as describing key clinical features of personality change after the brain damage suffered. When these were marked, they were naturally part of more severe impairment but they were not otherwise related to severity of change, as were Dependent, Labile and Fatuous.

Clinically, irritability could be seen as a defence used by the patient to prevent excessive stimulation or excessive demands for thought or action; the patient preferring characteristically to be left alone and responding with little explosions of ill temper when interrupted. Similarly, when someone was described as 'more easily worried', it appeared that this patient became flustered, anxious and perhaps tremulous when any performance or action was needed, but, when left alone, was not necessarily anxious. The emotional disturbance is situation specific and belongs to the category of catastrophic anxiety - a manifestation
of the damaged person's inability to cope and, like irritability, it leads to few attempts to do anything, with consequent limitation and stabilization of the environment.

The terms 'loss Vitality' and 'withdrawal' describe other aspects of the brain damaged person's behaviour, both of which also lead to less stimulation from the environment and consequently fewer demands for action of any type. Many of the patients with personality deterioration showed a picture of "organic readiness", with chronic shallow depressive mood, often lifting quickly in response to some new stimulus, but rapidly falling back with apparent boredom, loss of interest and easy fatigue (see Chapter VII). In addition, there was often apathy, withdrawal and the irritability already mentioned.

"IMPROVED PERSONALITY"

A good many patients were described by their spouses in terms which made it clear that they regarded them as "improved" in personality; being, for example, less sarcastic and irritable, less tense and anxious, less fussy and over-meticulous and more pleasant to live with generally. Thirteen were put in a separate category as "Improved" in personality. Some illustrative case histories are given later. To be included in this group, there had to be no unfavourable changes, such as loss of drive, socially undesirable disinhibition, impaired working capabilities, etc., and all of the patients were aware of increased emotional well being. In two, minor forgetfulness was present but otherwise there were no detectable intellectual changes. Obviously these verbatim descriptions already mentioned of "uninhibited", "less easily worried"
"less irritable", "more affectionate" and "more vitality" were
cosently applied to the group with improved personality but, in
most the were as described, there were untoward changes in intellect
or drive, which prevented classification as "improved".

Of the 15 patients considered "improved", 8 had A.C.,
A.P.C. and one an H.C. aneurysm. There were 10 men and 3 women.
Two had moderate, four had mild and seven had no C.I.S. signs. Of
those with signs, four were on the left and two on the right. The
previous personalities of these thirteen patients were not all
similar. Five were described as having been markedly anxious,
perfectionistic, withdrawn and sexually inhibited but were energetic
and capable people. They each became less anxious, less withdrawn,
less inhibited sexually and more affectionate; and two became less
perfectionistic. All retained their high drive and none became
flapdash or careless. Two others had apparently been tense and
suspicious men, given to continuously nagging in a domineering manner,
and both became much more friendly and tolerant. One woman had been
an alcoholic for years, having received inpatient treatment, but
she had become able to drink normally and coped well with her
difficult and psychopathic husband, although her right arm and leg
were markedly parietic. One man had previously been extremely
withdrawn, with marked feelings of inferiority and ideas of refer-
ce, and became much more relaxed and normally outgoing. His
wife noticed the change from the earliest post-operative days,
when he was euphoric and disinhibited, settling down over the
course of a few months to the permanent "improvement". Two men
had been for the whole of their adult lives tense, gloomy, easily
fatigued and subject to severe headaches of tension type. Both
became cheerful, much more vigorous and energetic and lost their headaches. In neither could a convincing case for a chronic depressive illness be made out. The last patient of the 13 had had a number of obsessional compulsive rituals for many years, of moderate severity, which he lost completely, becoming generally more relaxed and cheerful.

RELATIONSHIP OF PREMORBIT PERSONALITY RATINGS TO TYPE OF PERSONALITY CHANGE

The method of rating premorbid personality was described in Chapter III. Each patient was rated on four 3-point scales of: Proneness to anxiety, Obsessionalism, Energy and Outgoingness.

These premorbid personality assessments were then correlated with the verbatim descriptions of personality change. There were only 4 significant findings, as set out in Table 19.
(a) Those previously more anxiety prone were more often described as "labile" at follow up.

<table>
<thead>
<tr>
<th>Anxiety Prone</th>
<th>Below Average</th>
<th>Average</th>
<th>Above Average</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not Labile</td>
<td>49</td>
<td>89</td>
<td>78</td>
<td>216</td>
</tr>
<tr>
<td>Labile</td>
<td>3</td>
<td>6</td>
<td>16</td>
<td>25</td>
</tr>
<tr>
<td>Total</td>
<td>52</td>
<td>95</td>
<td>94</td>
<td>241</td>
</tr>
</tbody>
</table>

\( \chi^2 = 7.425, \) 2 d.f., \( p < 0.05 \)

(b) Those previously more obsessional were more often described as "less obsessional" at follow up.

<table>
<thead>
<tr>
<th>Obsessional</th>
<th>Below Average or Average</th>
<th>Above Average</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not Less Obsess.</td>
<td>117</td>
<td>101</td>
<td>218</td>
</tr>
<tr>
<td>Less Obsess.</td>
<td>6</td>
<td>17</td>
<td>23</td>
</tr>
<tr>
<td>Total</td>
<td>123</td>
<td>118</td>
<td>241</td>
</tr>
</tbody>
</table>

\( \chi^2 = 6.247, \) 1 d.f., \( p < 0.02 \)
(c) Those previously more obsessive were less often described as 'fatuous' at follow up.

<table>
<thead>
<tr>
<th>Previously Obsess-</th>
<th></th>
<th>Not Fatuous</th>
<th>Fatuous</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>ionally Below Average or Average</td>
<td></td>
<td>113</td>
<td>10</td>
<td>123</td>
</tr>
<tr>
<td>Above Average</td>
<td></td>
<td>116</td>
<td>2</td>
<td>118</td>
</tr>
<tr>
<td></td>
<td></td>
<td>229</td>
<td>12</td>
<td>241</td>
</tr>
</tbody>
</table>

\(\chi^2 = 5.337, 1 \text{ d.f.}, p < 0.05\)

(d) Those previously more energetic were less often described as 'more easily worried' at follow up.

<table>
<thead>
<tr>
<th>Previously Energetic</th>
<th>Not easily worried</th>
<th>More worried</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Below Average or Average</td>
<td>98</td>
<td>42</td>
<td>140</td>
</tr>
<tr>
<td>Above Average</td>
<td>83</td>
<td>18</td>
<td>101</td>
</tr>
<tr>
<td></td>
<td>181</td>
<td>60</td>
<td>241</td>
</tr>
</tbody>
</table>

\(\chi^2 = 4.595, 1 \text{ d.f.}, p < 0.05\)
(c) There was also a non-significant tendency for those previously more anxiety prone to be described as more anxious afterwards.

<table>
<thead>
<tr>
<th>Previously Anxiety</th>
<th>Not more Anxious</th>
<th>More Anxious</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Below Average</td>
<td>44</td>
<td>8</td>
<td>52</td>
</tr>
<tr>
<td>Average</td>
<td>73</td>
<td>22</td>
<td>95</td>
</tr>
<tr>
<td>Above Average</td>
<td>64</td>
<td>30</td>
<td>94</td>
</tr>
<tr>
<td></td>
<td>181</td>
<td>60</td>
<td>241</td>
</tr>
</tbody>
</table>

\( \chi^2 = \text{not significant} \)
ILLUSTRATIVE CASE HISTORIES

Mild Personality Impairment. No Intellectual Change.

Mr. B. H., aged 54. Right middle cerebral artery aneurysm rupture aged 40. Operation - clips applied to neck of aneurysm. Left-handed. Previous personality - cheerful, stable patient and affectionate Irishman; no neurotic traits, no personal or family history of mental illness. Devoutly Roman Catholic, taking part in parish activities, interested in politics, sport and 'serious reading'. A successful small shop keeper, happily married.

Afterwards became a debt collector, successfully, as too restless and impatient to work in his shop. His wife said he had become factious and excessively jovial in a "meaningless" way but was irritable if thwarted. Lost interest in religion and politics, reading confined to thrillers and light novels, which he read and re-read. No loss of libido but less affectionate.

Normal scores on Benton and I.P.A.T.

No clinical intellectual change. No C.N.S. signs.

Moderate Personality Impairment. No Intellectual Change.

Mr. F.B., aged 54. Rupture of anterior communicating artery aneurysm, filling from the left, aged 49. Small left frontal haematoa. Treated conservatively.

Previously manager of an ironmongers shop; an anxious and withdrawn man, intelligent, with a scholarship to grammar
school. Had been convicted of indecent exposure aged 40
(a few years after marriage). Intermittently impotent but
marriage described as 'happy enough' by his wife. His brother
probably schizophrenic.

Returned early to work but was sacked because he
talked incessantly of sex and made indecent suggestions to
the staff. Shortly afterwards, his wife left him, unable to
tolerate his sexual and aggressive disinhibition; and a week
later he became quite seriously depressed. He was treated as
an inpatient with E.C.T. and imipramine on two occasions within
one year.

Three years later, at follow up, he was garrulous
and circumstantial, disinhibited and cheerful; but there was
no evidence of hypomania. He had knocked in his front door
with an axe the previous week, having left the key at work,
and he showed me the damage smilingly, considering it a great
joke. His home was sparsely furnished but clean. He felt quite
well but was bitter about his wife's desertion and occasional
flashes of anger broke through into his conversation.

He was working full time as messenger for a City firm,
apparently satisfactorily. No intellectual impairment; normal
scores on both psychological tests. C.N.S. signs - slight left
hyper-reflexia, left plantar extensor. (Wife's account taken
from that recorded in mental hospital notes).
Moderate personality and mild intellectual impairment.

Mr. H. J., aged 42. Rupture of anterior communicating artery aneurysm 3 years before, with small right frontal hematoma. Treated conservatively. Previously a driving and very successful executive in an industrial combine; gay in company, fond of tennis and golf, very intelligent but not intellectual. No neurotic traits, but irritable and perfectionistic in character and a difficult man to work for and to live with. Married with two children.

For the first nine months after the stroke, he was vague and grossly indecisive, even about minutiae, such as deciding when to shave. Returned to work while in that stage and was 'carried' by the appointment of a deputy—who was still doing nearly all his work, although the patient remained on full pay.

At follow up, he was apathetic and sad and said that he felt "only 10% the man I was"; "I feel my standards are lower—no one takes any notice of me now!" He did not seem distressed by lack of promotion and spent nearly all his spare time just sitting and staring into space; not even interested in television but was irritable if interfered with or crossed.

He had become more interested in sex, to his wife's pleasure, and more affectionate in a childish and dependent way, sometimes following his wife around the house from room to room like a child. She found him easier to live with and felt guilty at times that she was happier only because he was so changed.
His memory and general grasp were definitely worse than before but this did not show in tests. (Previous level very high). I.P.A.T. - normal score. Benton - two below expected score (rated normal). C.S.X. - mild left-sided signs, with inattention, asterognosia, finger agnosia, slight clumsiness and hyper-reflexia.

Moderate Personality and Moderate Intellectual Change.

Mr. C.C., aged 58; Rupture of right M.C. aneurysm 5 years previously, with a right temporal haematoma. At operation, necrotic brain removed, haematoma evacuated, the aneurysm wrapped in cotton gauze.

Previously he had been a lorry driver, described by his wife as cheerful, popular and perfectionistic, although bad tempered at times. He had had a brief "breakdown" two years before the stroke, with hypochondriacal fears, depression and a severe panic attack; all of which cleared in a few months with tablets.

At follow up, he was very depressed and had apparently shown little change over the past 4 years. He had had inpatient treatment in a mental hospital a few months before with little benefit. He had changed markedly in personality; instead of being obsessionally orderly, he was slovenly and personally dirty, he had become excitable and had hit his wife, which had led his son to strike him, and the family atmosphere was continually tense. He had become forgetful and irresponsible with money and had bought expensive cameras, the instructions of which he could not understand,
although he had been a keen photographer for years. He now found it difficult to follow television programmes and could not concentrate well enough to read. He was "fighting against suicide", he told me; his wife said he had threatened to "take you lot with me". They were continuously quarrelling and she was planning to leave him. There were no disturbances of sleep, appetite, weight, etc., and his tone was self-pitying and resentful. He was working full time as a factory sweeper and complained that his work mates taunted him with being a "nut case".


Benton - score normal.

C.S.S. - left hyper-reflexia, asterognosis and sensory inattention. (No disability complained of).

**Moderate Personality and Moderate Intellectual Impairment.**

Miss K.P., aged 50. Rupture of right posterior communicating aneurysm 5 years before, with marked cerebral arterial spasm - treated conservatively. Grand-daughter of a rich Victorian industrialist, had lived a retired life, helping in family firm. Quiet, gentle, unassuming in personality, intelligent and musical, with a small circle of friends. No neurotic features in the history.

Physically disabled at follow up, with a spastic left hemiparesis, the arm almost useless, gait shuffling. Some grand mal fits, easily controlled.

She had become forgetful and slow and was no longer able to understand or perform her old book-keeping and secretarial work; with a marked inability to follow more than
one thing at a time, she did not seem depressed but was bitter about her fate, with occasional incoherent laughter. At times she flared up into anger at her sister. She was much more easily anxious, not only about being unsteady, etc., but about world affairs, the fate of the business and day to day trivia. She had become more obsessional and quadruple-checked many of her activities. Few interests left, apart from a little sewing; had lost pleasure in music and avoided her friends.

Abnormal scores on I.P.A.T. (30 errors) and scored 2 points below expected on Benton (rated normal).

Improved Personality:

Mrs. G. F., aged 43. Rupture of A.C. aneurysm 3 years previously, with severe and widespread cerebral arterial spasm and a small left frontal haematoma operation. Operation = clips to neck of aneurysm.

Previously a very anxious and "highly strung" woman, impatient, slapdash and restless, prone to depressive spells and troubled by marked inferiority feelings. Her husband was of psychopathic personality, an undischarged bankrupt and often unemployed. She had had inpatient treatment for alcoholism two years before her stroke, with transient benefit, and had been drinking heavily up to the time of her stroke.

After the stroke, she was left with a right weakness, more in the leg than arm, and needed a caliper. Mentally, she was much calmer, coped well with her four children and the housework and with her hemiplegic sister, who had also had a subarachnoid haemorrhage. She no longer suffered from depression.
or anxiety and dismissed her old inferiority feelings with
"I never think of that sort of thing now". She still looked
down on her husband but lived more peacefully with him and she
had no more alcohol than an occasional beer.

Psychological tests - both normal.

In this case, there was some flattening of affective
response, the effects of which were beneficial, given her
personality and circumstances. An equivalent change in some-
one else might not have been regarded as an "improvement".

Improved Personality.

Mr. H.H., aged 54. Rupture of large anterior
communicating aneurysm 4 years before; treated by clipping
left anterior cerebral artery.

A self-made business man; very hard working, tense,
irritable, sarcastic, perfectionistic; "mean and conceited"
and "emotionally hard and unyielding", his wife said. Irritated
by his daughter (then 11 years old) and had forbidden her
friends to visit the house. His father had had a chronic
paranoid illness; and he had had a "nervous breakdown" when 18,
with depression and headaches for two years; untreated.

At follow up, he seemed entirely normal, said he was
happier and more relaxed than before and that he really enjoyed
life now for the first time. He did not say that his illness
had made him reevaluate his life, nor in any way suggest that
he had changed as a reaction to his illness. His business had
continued to flourish, he worked hard without undue fatigue and
had a more active social life.
His wife was almost in tears at interview, as she described the change that had occurred. Previously she and her daughter had been frightened of him, her friends could not visit, as he was so hostile and ill-mannered. Now he was affectionate and sexually more active; relaxed, polite to her mother and her friends. He went riding with his daughter and brought his wife flowers on her birthday. There were definitely no intellectual changes, he was very energetic and still the 'boss' in the home.

He scored normally on intellectual tests and there were no C.N.S. signs.

CONCLUSIONS AND DISCUSSION

In this Chapter, personality impairment has been rather arbitrarily distinguished from intellectual impairment and from psychiatric symptoms - which are described in later chapters - but nevertheless 108 (41%) of the 261 patients were considered impaired. This is a lower rate than that found by Felger et al (1961) in a study of 1,000 patients with mixed disorders, of whom 284 (54%) showed organic brain syndromes, including 47 (52%) of 91, who had had cerebral haemorrhage. Their patients were considerably older than those described here; and the real differences are bigger than shown by these figures, as their method would not have identified many cases of mild impairment. The psychological morbidity of subarachnoid haemorrhage is almost certainly considerably lower than that after cerebral haemorrhage or infarction.
associated with diffuse arterial disease, as would be expected, although no direct comparisons are at present possible.

Logue et al (1963) described in detail the psychiatric morbidity of 79 survivors of subarachnoid haemorrhage from A.C. aneurysms, 66 treated by operation, who were roughly comparable in age, sex and timing of follow up to those described here. Of their patients, 52:79 were neurologically unimpaired, as were 52:61 patients with A.C. aneurysms in this series; and 41:79 had returned to their original work, as had 52:61 in this series. These relatively objective findings confirm the comparability of the groups and are important in that Logue et al, in common with others, consider that the value of surgery for ruptured A.C. aneurysm has already been firmly established. It has, however, been shown in Chapter IV that there were no significant differences in the psychiatric morbidity following operative and conservative treatment for A.C. aneurysms; and similarly, McIsaac et al (1963) have shown that life expectancy and physical condition are not improved by surgery in this group.

In this series, personality impairment was, on the whole, proportional to the severity of the neurological signs, which are the least equivocal evidence of brain damage; and it is interesting that personality change was equally common in all three major aneurysm groups in the absence of signs, although one might have expected that damage to the neurologically "silent" frontal areas would have led to more personality change, even in their absence. (As will be described in Chapter VI, intellectual impairment in the absence of C.N.S. signs does occur more often with A.C. aneurysms).
Patients with aneurysms had significantly more frequent and more severe signs than those without, and correspondingly more personality impairment. Similarly, of the three major aneurysm groups, both C.N.S. signs and personality change were commonest with A.C., least common with M.C., and intermediate with P.C. aneurysms.

In addition to demonstrating the relationship between personality damage and the severity of brain damage, as indicated by neurological signs, there is clear evidence of the importance of the site of damage:

1. There was a significant tendency for patients with A.C. aneurysms to be more affected mentally than others, even allowing for the degree of C.N.S. signs. This could be explained by the finding that patients with central infarction, which occurred largely as a complication of common carotid ligation for P.C. aneurysms, had less personality impairment than those with equally severe signs from M.C. aneurysms. Clinically and anatomically, the area affected by occlusion of the carotid artery includes the territory of the middle cerebral artery - the same area, in fact, as that damaged locally around an M.C. aneurysm. It seems likely that the difference in the effect on personality is due to the fact that, when the main damage is to cortical and subcortical tissues, there is more interference with the function of the brain as a whole than when a larger segment of brain is completely out of action and "silent". This would then be analogous to the improvement which may be brought about in Little's Disease (cerebral diplegia), when complete removal of a diseased
hemispheres may be followed by marked improvement (Ignalina and Lucy, 1963). Certainly, epilepsy after subarachnoid haemorrhage is commoner with M.C. aneurysms than with the others, (see Chapter IV and Rose and Gummer, 1965), so that dysfunction of epileptic type must be commoner. Unfortunately, no M.A.C. studies were possible in this series but it would be useful to attempt a correlation of clinical personality description and M.A.C. findings in a similar material.

2. The existence of a qualitatively different frontal lobe syndrome is also confirmed by this material. The use of the verbatim statements of relatives, as described, effectively excludes the possibility of the findings being the product of the author's bias, even when frontal craniotomy scars betrayed the site of aneurysm at interview. Nor could the findings be due merely to differences in severity of brain damage, as they persist unaltered even when only those with mild personality changes are compared. Patients with A.C. aneurysms were significantly more often described as less irritable, less easily worried and uninhibited, although these terms were also used about a few patients with P.C. and about two with I.C. aneurysms.

The existence of a group of 13 patients, regarded as 'improved' in personality, is another reflection of the frontal lobe syndrome and must be due to a leucotomy effect. If the changes were the result of the patients' reaction to severe illness, leading to a reappraisal of life and to 'turning over a new leaf', one would not expect it to be associated so markedly with one area of the brain. In addition, the quality of the changes is in keeping with those classically described in the
frontal lobe syndrome and similar changes were seen in other patients who were not improved because of accompanying unfavourable changes. The results are, therefore, in direct contradiction to those described by Sicules (1951), derived from cerebral tumour cases, where there were no qualitatively different frontal lobe features; Logue et al (op cit), however, found a similar group in their own series, although their patients had more associated intellectual impairment. After subarachnoid haemorrhage, there may be patchy infarction in areas distant from the territory of the artery mainly concerned and this happens particularly after rupture of P.C. aneurysms, (Crompton, 1953). The existence of a frontal lobe syndrome of classical type after haemorrhage from any aneurysm may therefore still be due to true frontal lobe damage.

It is worth contrasting this true localisation effect to that shown by descriptions of Dependent, Emotionally Labile and Fatuous, which were much commoner with P.C. aneurysms but apparently only because C.N.S. signs and severe personality damage were commoner in these cases.

3. The qualitatively different effects of frontal lobe damage are also demonstrated by the relationship of personality to intellectual impairment, in that A.C. patients had a significant tendency to suffer from personality change without intellectual impairment, compared to the others in whom the two were more often parallel. This finding is paradoxical, in view of the finding to be described in the next chapter that A.C. cases more often have intellectual impairment in the absence of
signs; and the finding mentioned earlier that, in the absence of signs, there are no differences between the aneurysm groups in the incidence of personality impairment.

4. The most frequently used descriptions were of less Vitality, More Irritable and More Anxious, all of which were very significantly associated with neurological signs, intellectual impairment and psychiatric symptoms and which seem to form a 'core syndrome' in brain damaged patients.

5. Personality impairment was significantly more common in men than in women, although C.I.N.I. signs were rather commoner in the latter, a finding for which there is no apparent explanation.

6. The effect of laterality only shows once in this report, in that, of the seven patients with moderately severe personality change who were intellectually unimpaired, all were right-handed and six had left-sided lesions, a finding in keeping with the generally accepted role of the left hemisphere in intellectual functions. There is no support here for the finding by Lishman (1968) that the frontal syndrome was commoner with right frontal damage, in his series of head injured patients.

7. Intellectual impairment, as assessed clinically was closely correlated with the presence and severity of personality impairment. The Benton and the Paired Associate Tests, however, did not distinguish patients with moderate from those with mild personality change, although they distinguished those with impairment from those without at a very high level of significance, especially when considered together. Even then, however, 14:79
patients with personality impairment scored normally on both
tests, which only underlines the generally accepted view that
there are no unequivocal tests of "brain damage" as a unitary
phenomenon.

6. The importance of brain damage to personality
change is clearly shown here but the role of premorbid person-
ality seems relatively small, in apparent contradiction to much
of the literature, especially that on the sequelae of head injury,
in which premorbid personality and life situation factors have
been emphasized. This different emphasis, however, has been
brought about by the concentration here on the reports of other
people, largely excluding the subjective experience and symptoms
of the patient, and compensation factors played no part. In
fact, depression and anxiety were common and, as will be de-
scribed in Chapter VII, were more clearly related to the pre-
morbid personality than were the personality changes described
here. This separation of objective personality change and sub-
jective psychiatric symptoms is in some ways artificial but is
useful in the light it throws on organic factors. Although the
method used and patients studied are very different from the
retrospective case study by Ishman (op cit), the results are
similar in important ways: he too showed that some mental changes
-intellectual impairment, apathy, euphoria and certain behaviour
disorders - depended on the amount of brain damage; whereas
depression, anxiety, fatigue and other symptoms were less closely
related to brain damage. He also confirmed the existence of a
qualitatively different frontal lobe syndrome, although it was
not confined to those with damage to the frontal lobes.
Nevertheless, four statistically significant associations did appear between previous personality and the changes described by relatives and speculative explanations can be offered for them, although they do not seem of much clinical importance.

(a) That the previously more obsessional were described as less obsessional afterwards in keeping with the frequent tendency for brain damaged people to become more careless and disorderly (although some patients become more orderly and obsessional, as happened to ten in this series).

(b) That previously more anxiety prone people were described as more emotionally labile may be due to the lessened control of emotional expression leading to more frequent emotional outbursts in those who are chronically anxious.

(c) That the previously more obsessional were less often described as "fatuous" may reflect a higher level of emotional control in those categorised as obsessional.

(d) Lastly, the previously more energetic were less often described as more easily worried afterwards, which might be taken to mean that to be high in energy goes with a lower level of "neuroticism" - although this is always an unsatisfactory global concept.

Altogether, the results presented in this chapter support classical views about the localization of brain function, in that both general and specific effects may occur with damage to the brain. These effects show a continuum of severity of mental change up to gross dementia, mainly parallel to the degree
of neurological involvement, as demonstrated by C.N.S. signs. (Brain damage does not always lead to such signs but the signs are unequivocal evidence that damage has occurred). The importance of premorbid personality is less here because of the method used but this emphasis on what are essentially behavioural changes is useful in distinguishing organic and reactive psychological factors.
CHAPTER VI
INTELLECTUAL CHANGES AFTER SUBARACHNOID HEMORRHAGE

In this Chapter, as in the previous one, only the first series of 261 patients is considered. The methods, rating scale of clinical intellectual impairment and details of the two psychological tests used are described in Chapter III.

RESULTS

1. Clinical Intellectual Impairment

Of the 261 patients, 109 (41%) were considered to show some overall clinical intellectual impairment; 78 (30%) were mildly, 22 (8%) moderately and 9 (3%) severely impaired.

Table 20 shows the way in which this was closely related to C.N.S. signs.

Table 20.

<table>
<thead>
<tr>
<th>C.N.S. SIGNS</th>
<th>Absent</th>
<th>Mild</th>
<th>Mod.</th>
<th>Severe</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical Impairment</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>116(78)</td>
<td>29(44)</td>
<td>5(24)</td>
<td>2(8)</td>
<td>152(59)</td>
</tr>
<tr>
<td>Mild</td>
<td>31(21)</td>
<td>29(44)</td>
<td>8(30)</td>
<td>10(40)</td>
<td>76(30)</td>
</tr>
<tr>
<td>Mod.</td>
<td>1(1)</td>
<td>7(11)</td>
<td>7(33)</td>
<td>7(26)</td>
<td>22(8)</td>
</tr>
<tr>
<td>Severe</td>
<td>0(0)</td>
<td>1(1)</td>
<td>1(5)</td>
<td>7(26)</td>
<td>9(3)</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td>148(100)</td>
<td>66</td>
<td>21</td>
<td>26</td>
<td>261</td>
</tr>
</tbody>
</table>

Percentages in parentheses.
Overall there is a very significant association between more severe signs and more severe intellectual impairment and, in those without C.N.S. signs, intellectual impairment is absent or mild with only one exception. If Table 20 is simplified by grouping together moderate and severe categories, then \( \chi^2 = 98.965 \), 4 d.f., \( p < 0.001 \). Of those with severe signs, only two escape intellectual impairment. (It should be remembered that mild C.N.S. signs, apart from dysphasia, were only detectable on physical examination and were not obvious at the time the rating for intellectual impairment was made).

Patients with intellectual impairment were in most cases the same as those assessed as showing personality impairment with certain exceptions, which are described elsewhere. Naturally halo effects are likely to have occurred in these two subjective assessments.

There were important differences in the incidence of C.N.S. signs in the various aneurysm groups, which were associated with differences in clinical intellectual impairment, as they were with personality impairment. Thus both C.N.S. signs and intellectual impairment were significantly less common in no aneurysm cases than in those with aneurysms (\( p < 0.05 \)); and of the single aneurysm groups, C.N.S. signs and intellectual change were both more common with Middle Cerebral artery (M.C.) than with Posterior Communicating (P.C.) and Anterior Communicating (A.C.) aneurysms, (\( p < 0.05 \)). (Details omitted as the figures are so similar to those for C.N.S. signs and personality impairment).

There were 12 patients with severe signs who escaped with
absent or with only mild intellectual change, of whom nine had had major cerebral infarction occurring as a complication of common carotid artery ligation for A.C. aneurysm. Only three had A.C. aneurysms, in which the damage to the brain is more often confined to cortical and subcortical tissues.

This finding is also parallel to that described for personality changes.

Although overall the relationship to C.N.S. signs is clear, A.C. aneurysm cases again differ significantly from M.C. and P.C. cases in that they more often have intellectual impairment in the absence of C.N.S. signs than do the others. Thus, 17/52 A.C. cases without signs have intellectual impairment compared to 11/66 of the others ($$^2 = 4.557$$, 1 d.f., $$p < 0.05$$). This is somewhat paradoxical in view of the finding mentioned earlier that A.C. cases had a significant tendency to have personality impairment without intellectual impairment, unlike the M.C. and P.C. cases in which the two forms of impairment were more closely associated.

2. Other Factors and Clinical Intellectual Impairment

There were no significant relationships between clinical intellectual impairment and any of the following factors once degree of C.N.S. signs has been allowed for:

- Side of aneurysm, or side of C.N.S. signs; estimated premorbid intelligence, or sex. As mentioned elsewhere, age and high blood pressure were associated with more frequent and more severe C.N.S. signs and clinical intellectual impairment was commoner in older and hypertensive people but not to a significant degree. (Age and B.P. were not responsible for any of the differ-
ences between the aneurysm groups).

Table 21 shows the relationship between side of brain damage and intellectual impairment for those with definite signs either at follow up or during admission. Right and left refer to the hemisphere involved; 'Neither' includes cases with clearly lateralised aneurysm so long as no signs were recorded at any time. Two patients with severe intellectual impairment had bilateral signs.

**Table 21.**

**INTELLECTUAL IMPAIRMENT & SIDE OF BRAIN DAMAGE**

<table>
<thead>
<tr>
<th>Intellectual Impairment</th>
<th>Right</th>
<th>Left</th>
<th>Neither or (both)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>60</td>
<td>43</td>
<td>49</td>
<td>152</td>
</tr>
<tr>
<td>Mild</td>
<td>35</td>
<td>30</td>
<td>13</td>
<td>78</td>
</tr>
<tr>
<td>Mod.</td>
<td>7</td>
<td>15</td>
<td>-</td>
<td>22</td>
</tr>
<tr>
<td>Severe</td>
<td>2</td>
<td>5</td>
<td>Both (2)</td>
<td>9</td>
</tr>
<tr>
<td><strong>TOTAIS</strong></td>
<td>104</td>
<td>93</td>
<td>64</td>
<td>261</td>
</tr>
</tbody>
</table>

If right and left are compared grouping moderate and severe impairment together, then \( \chi^2 = 6.742, 2 \) d.f., \( p < 0.05 \), and intellectual impairment is significantly commoner with left-sided lesions. However, this significant difference is entirely explained by the poor results in women with left-sided M.C. aneurysms, described in Chapter IV, who had more severe C.H.S.
signs. If severity of signs is allowed for, the difference disappears and side of brain damaged is no longer relevant. (This unexpected finding is discussed later).

3. Psychological Test Results

The manner in which the Ingels Paired Associate Test (P.A.T.) was modified has been described. A total of 213 patients completed both the P.A.T. and the Benton Visual Retention Test, as the modified P.A.T. was not given to the first 29 patients seen, nor to the severely dysphasic and none of those with severe clinical intellectual impairment (dementia) could attempt either test meaningfully. Only those who completed both tests are considered in the following comparisons, except where specifically stated otherwise.

Table 22 shows the relationship between C.N.S. signs and P.A.T. scores, grouped to maximise differences.

**TABLE 22.**

<table>
<thead>
<tr>
<th>C.N.S. SIGNS AND P.A.T. SCORES</th>
<th>C.N.S. SIGNS</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;Normal&quot; 0-10 errors</td>
<td>Absent</td>
</tr>
<tr>
<td>&quot;Normal&quot; 0-10 errors</td>
<td>95 (76)</td>
</tr>
<tr>
<td>&quot;Abnormal&quot; &gt; 10 errors</td>
<td>30 (24)</td>
</tr>
</tbody>
</table>

| Total                          | 125          | 55    | 18    | 15    | 213    |

\( \chi^2 = 3.096, 3 \text{ d.f.}, p < 0.05 \)

Percentages in parentheses.
Table 23 shows the relationship between C.N.S. signs and the Benton Test results, also grouped to maximise differences.

**Table 23.**

<table>
<thead>
<tr>
<th>C.N.S. SIGNS &amp; BENTON TESTS</th>
<th>C.N.S. SIGNS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Absent</td>
</tr>
<tr>
<td>Benton Scores</td>
<td></td>
</tr>
<tr>
<td>&quot;Normal&quot;</td>
<td>92 (74)</td>
</tr>
<tr>
<td>&quot;Abnormal&quot; (3 or more below expected score)</td>
<td>33 (26)</td>
</tr>
<tr>
<td>Percentages in parentheses.</td>
<td>125</td>
</tr>
</tbody>
</table>

Percentages in parentheses. \( \chi^2 = 9.260, \; 3 \text{ d.f.}, \; p < 0.05 \)

The F.A.T. and Benton scores have almost identical overall patterns of distribution in relation to C.N.S. signs but, as will be shown later, this does not mean that the scores were related similarly in each individual, so that the tables give a false impression in that respect. For both F.A.T. and Benton, there is a significant worsening with increasingly severe C.N.S. signs. By altering the groupings used, e.g., by comparing all those with signs to those without, and comparing those with absent and mild signs to those with moderate and severe signs, one test or the other can be made to appear
as more significantly related to the C.N.S. signs; which is a
reminder of the limitations of the method used.

**PSYCHOLOGICAL TEST SCORES AND OTHER FACTORS**

(a) **Innis Paired Associate Test (P.A.T.)**

Scores on this test were not related significantly to
sex, side of lesion or to side of C.N.S. signs in those with
signs. Scores were worse in older people at the 1% level of
significance but so were C.N.S. signs. They were not related
to enuremic group, except in so far as they were associated
with more severe signs, with one exception. In patients with-
out C.N.S. signs, A.C. cases had (non-significantly) worse
P.A.T. scores than had H.C. and P.C. cases; a similar finding
to that made for clinical intellectual impairment. (Of patients
without C.N.S. signs, 16/49 A.C. cases made >10 P.A.T. errors,
compared to 14/76 H.C. and P.C. cases. \( \chi^2 = 3.242, 1 \text{ d.f.} \)
0.1; \( p > 0.05 \)).

Table 24 shows the lack of significant relationship
between P.A.T. scores and side of brain damage.

**TABLE 24.**

<table>
<thead>
<tr>
<th></th>
<th>Right</th>
<th>Left</th>
<th>Neither</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>P.A.T. 0-10</strong></td>
<td>38</td>
<td>34</td>
<td>76</td>
<td>146</td>
</tr>
<tr>
<td><strong>errors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>&gt; 10 errors</strong></td>
<td>20</td>
<td>26</td>
<td>19</td>
<td>67</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>58</td>
<td>60</td>
<td>95</td>
<td>213</td>
</tr>
</tbody>
</table>

Comparing right to left then \( \chi^2 = 0.964 \) (non-significant)
This is more evenly distributed than was the case with clinical intellectual assessment, where a spurious relationship with left-sided brain damage was produced by the predominance of women with severe C.I.O. signs from left-sided M.C. aneurysms.

The most striking association of the P.A.T. was with estimated premorbid intelligence. Table 25 shows that errors were very significantly more common in those of below average intelligence premorbidly, (rated on the basis of educational, personal and work history).

**TABLE 25.**

**BACKGROUND INTELLIGENCE AND P.A.T. ERRORS**

<table>
<thead>
<tr>
<th>P.A.T. Errors</th>
<th>Below Average</th>
<th>Average</th>
<th>Above Average</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-10</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17 (39)</td>
<td>69 (76)</td>
<td>40 (77)</td>
<td>146</td>
<td></td>
</tr>
<tr>
<td>&gt; 10</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>27 (61)</td>
<td>28 (24)</td>
<td>12 (23)</td>
<td>67</td>
<td></td>
</tr>
<tr>
<td></td>
<td>44</td>
<td>117</td>
<td>52</td>
<td>213</td>
</tr>
</tbody>
</table>

Percentages in parentheses.

(If the average and above average groups are combined, then $\chi^2 = 23.17$, 1 d.f., $p < 0.001$). None of the other factors considered appeared to be involved in this difference.
(b) **Benton Visual Retention Test.**

Scores on this test are already adjusted for age and estimated premorbid intelligence. They were not related significantly to side of lesion or to side of C.H.S. signs, as shown in Table 26, although there is a trend for those with left-sided lesions to score worse.

**Table 26.**

**Side of Brain Damage and Benton Scores**

<table>
<thead>
<tr>
<th>Side of Damage</th>
<th>Right</th>
<th>Left</th>
<th>Neither</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benton Scores</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>40</td>
<td>34</td>
<td>71</td>
<td>145</td>
</tr>
<tr>
<td>Abnormal</td>
<td>18</td>
<td>26</td>
<td>24</td>
<td>68</td>
</tr>
<tr>
<td></td>
<td>58</td>
<td>60</td>
<td>95</td>
<td>213</td>
</tr>
</tbody>
</table>

Comparing right to left then $X^2 = 1.80$ (not significant).

Comparisons within the single aneurysm groups show that women with left-sided M.C. aneurysms already known to do particularly badly in this series, did worse than others on the Benton Test and this group again is responsible for what little difference is present.
Sex

Sex was an important factor, as women had considerably worse scores. Considering all the 231 patients who did the test, 61.44% women (42.1%) had abnormal scores, compared to only 22.407 men (21.2%). \( \chi^2 = 17.17, 1 \text{ d.f.}, p < 0.001 \). Overall the incidence of C.N.S. signs was almost identical in men and women and, although some of the difference was due to the poor scores in women with H.C. aneurysm, already mentioned, this was not the sole cause. Women also scored worse than men in the A.C. and no aneurysm cases; and when severity of C.N.S. signs was allowed for. It did not apply, however, to those with no C.N.S. signs. As an example, in M.C. patients with mild signs, 11:16 women and 0:19 men had abnormal scores; but of those with no signs, only 2:12 women and 1:13 men had abnormal scores. Curiously this sex difference did not apply to P.C. aneurysm cases, although these patients had rather worse scores than did the other diagnostic groups.

Aneurysm Site

Site of aneurysm is also important in that, of the A.C. patients with mild signs (and therefore with mainly frontal lobe damage), only 2:21 had abnormal Benton scores, compared to 18:37 H.C. and P.C. patients with similar signs. \( \chi^2 = 8.944, 1 \text{ d.f.}, p < 0.01 \). This difference is not due to sex or any other factors considered and is in contrast to the findings for clinical intellectual impairment and on the P.A.T., where A.C. patients did worse than H.C. and P.C. patients, at least in the absence of signs.
RELATIONSHIPS BETWEEN PERSONALITY IMPAIRMENT AND MEASURES OF INTELLECTUAL IMPAIRMENT.

(a) Clinical Intellectual Impairment.

The close association between this and personality impairment has been described in Chapter V.

(b) Personality Impairment and Psychological Test Results.

Tables 27 and 28 show the relationships between personality impairment and the P.A.T. and Benton Test scores, respectively. The tests distinguish between those with and without personality impairment; the P.A.T. with a higher degree of statistical significance. Neither distinguished between those with mild and those with moderate impairment.

**TABLE 27.**

<table>
<thead>
<tr>
<th>Personality Impairment</th>
<th>P.A.T. Errors</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0-10 errors</td>
</tr>
<tr>
<td>Absent</td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>103</td>
</tr>
<tr>
<td></td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>146</td>
</tr>
</tbody>
</table>

\[ \chi^2 = 12.32, 1 \text{ d.f.}, p < 0.001 \]
TABLE 20.

<table>
<thead>
<tr>
<th>Personality Impairment</th>
<th>Absent</th>
<th>Normal</th>
<th>Abnormal</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>98</td>
<td>35</td>
<td>133</td>
</tr>
<tr>
<td></td>
<td></td>
<td>47</td>
<td>33</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td></td>
<td>145</td>
<td>68</td>
<td>213</td>
</tr>
</tbody>
</table>

$\chi^2 = 5.185$, 1 d.f., $p < 0.05$

RELATIONSHIP BETWEEN CLINICAL INTELLECTUAL IMPAIRMENT AND THE P.A.T. AND BENTON TEST.

Tables 29 and 30 show the relationships of the P.A.T. and Benton Test to clinical intellectual impairment.

TABLE 29.

<table>
<thead>
<tr>
<th>P.A.T.</th>
<th>Clinical Intellectual Impairment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Absent</td>
</tr>
<tr>
<td>0-10 errors</td>
<td>110 (88)</td>
</tr>
<tr>
<td>&gt; 10 errors</td>
<td>15 (12)</td>
</tr>
</tbody>
</table>

$\chi^2 = 41.29$, 2 d.f., $p < 0.001$

Percentages in parentheses.
TABLE 30.

BENTON AND CLINICAL INTELLECTUAL IMPAIRMENT

<table>
<thead>
<tr>
<th>Benton Scores</th>
<th>Normal</th>
<th>Absent</th>
<th>Mild</th>
<th>Moderate</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(78)</td>
<td>97</td>
<td>40</td>
<td>8</td>
<td>145</td>
</tr>
<tr>
<td>Abnormal</td>
<td>(22)</td>
<td>28</td>
<td>28</td>
<td>12</td>
<td>68</td>
</tr>
<tr>
<td></td>
<td>(41)</td>
<td></td>
<td>(41)</td>
<td>(60)</td>
<td>(32)</td>
</tr>
</tbody>
</table>

\[ \chi^2 = 15.114, 2 \text{ d.f.}, p < 0.001 \]

The P.A.T. is, therefore, more closely related to the clinical assessment of both personality impairment and intellectual impairment (especially the latter) than is the Benton Test; although they are similarly related to C.N.S. signs.

Considering the single aneurysm groups separately, there was one very significant difference on the Benton Test. Of the patients with absent or mild clinical intellectual impairment, P.C. cases do significantly worse; as 23:39 P.C. cases and only 26:118 A.C. and M.C. cases have abnormal Benton scores.

\[ \chi^2 = 18.51, 1 \text{ d.f.}, p < 0.001 \]

As P.C. patients do not show the sex difference described earlier, this cannot be the explanation; nor are any of the other factors considered relevant. There are no differences of this type apparent for the P.A.T.
Relationship between Benton Test and P.A.T.

This is shown in Table 31 and, if this is compared to Tables 29 and 30, it is clear that the P.A.T. is much more, and the Benton rather more closely related to clinical intellectual impairment than the two Tests are to each other (based on the value of $\chi^2$ for each table).

TABLE 31.

BENTON AND P.A.T. SCORES

<table>
<thead>
<tr>
<th>P.A.T.</th>
<th>0-10 errors</th>
<th>&gt;10 errors</th>
<th>Normal</th>
<th>Abnormal</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>111</td>
<td>35</td>
<td>146</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>34</td>
<td>33</td>
<td>67</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>145</td>
<td>68</td>
<td>213</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$\chi^2 = 13.482, 1 \text{ d.f., } p < 0.001$

Considering only the single aneurysm cases, we find again that it is the P.C. cases which stand out. A normal Benton accompanies a normal P.A.T. in 76/89 A.C. and M.C. cases but in only 13/30 P.C. cases ($\chi^2 = 20.34, 1 \text{ d.f., } p < 0.001$). Again, differences in sex and C.N.S. signs do not explain the finding which is parallel to that found in comparing the P.A.T. and Benton to clinical intellectual impairment.
It is now generally agreed that there is no single test for "brain damage" but only tests for different aspects of mental function, (see Piercy (1964) and Yates (1966) for reviews on this subject). To a clinician the least equivocal evidence of brain damage comes from a history of, for instance, leucotomy or tumour removal or from the presence of C.N.S. signs; but any of these may be accompanied by no detectable changes in the patient's mental state. In clinical practice, no psychological test is more valuable in assessing the mental effects of brain damage than is the account of some person who knows the patient well and describes changes of the type outlined in Chapter V of this thesis or other characteristic changes.

This project was not undertaken to study the validity and inter-relationships of clinical intellectual assessment and of the two tests used but it does have the advantage of studying a relatively homogeneous population, the tests were carried out by one person and the site of brain damage was known with some certainty. Disadvantages are that the clinical ratings of C.N.S. signs and personality and intellectual impairment may have been distorted by halo effects (see later); and that the intellectual impairment ratings may have been biased by the results of the Benton and P.A.T., although these tests are unlikely to have affected the ratings for signs and personality change.
Comparisons with other published studies are largely inappropriate. Only Toce et al. (1964) have described/intellectual changes after subarachnoid haemorrhage in any detail, in their study of patients with A.C. aneurysms. They used more complex testing and a more sophisticated analysis of the data and distinguished memory disorder from other intellectual functions, whereas they are not considered separately here.

Memory is a very complex concept and, although clinically the Wernicke type of defect, with a more or less pure impairment of new verbal learning, has been firmly linked with lesions of the hippocampal-amygdillary body system (e.g., Harbison, 1963), in most patients with even mild cerebral damage, memory disorders are inextricably linked with impairment of other intellectual functions. It is even difficult to be sure that the tests we use do measure what they are supposed to. For example, ten patients in this study, when given the P.A.T. were unable to give correct answers, even after thirty trials, but were recorded as spontaneously repeating the three pairs of words correctly in the given order at the end. (This was not done systematically and may well be more common). It is known that serial learning of this type is easier but the situation just described could as well be attributed to a failure of the patient to "abstract", (Goldstein, 1952) as a failure to remember. Dysphasia itself is also a form of intellectual disturbance, whether purely expressive or mixed expressive and receptive, and it complicates matters by interfering with many tests of other intellectual functions.

However, so long as we remain aware of these difficulties of definition and of concept, there is no point in pursuing them.
Clinical intellectual impairment was present in 41% of the patients in this series; and the principal findings about it and the two psychological tests used can be considered under four headings: their relationship to the extent and to the site of brain damage; to the various other factors considered; and to each other.

1. **Intellectual Changes and Extent of Brain Damage.**

   The method used here to assess extent of brain damage is the severity of neurological signs, which is naturally less accurate than weighing the amount of brain removed at operation; and, as mentioned before, some halo effect might be expected, especially when faced by a patient with severe C.N.S. signs. Nevertheless, the importance of C.N.S. signs shows very clearly, not only in the clinical assessments but in the unbiased P.A.T. and Benton Test Scores. Rather few studies of brain damaged patients do attempt to assess the degree of brain damage in non-psychological ways but this aspect of the results is in keeping with the work of, for example, Chapman and Wolff (1959), who assessed the amount of brain removed surgically and found it proportional to the psychological deficit. In this series, only 148 patients with no C.N.S. signs had more than mild intellectual impairment; and only 7152 with moderate or severe C.N.S. signs escaped intellectual impairment of some degree.

2. **Intellectual Changes and Site of Brain Damage.**

   (a) **Frontal Lobe Damage.**

   In Chapter V, the existence of a qualitatively distinct
Frontal lobe syndrome was demonstrated and distinct differences are also shown here for intellectual change. Patients with A.C. (anterior communicating artery) aneurysms, who mainly suffer frontal lobe damage, significantly more often sustained intellectual impairment without C.N.S. signs than did the other patients with aneurysms; a finding in keeping with the known "neurological silence" of the frontal lobes. A.C. patients without signs also had (nearly significantly) worse P.A.T. scores than the others; and, considering only patients with mild signs, A.C. patients made very significantly fewer errors on the Benton Test—a finding in keeping with those described by Benton (1955) in his test handbook. A.C. patients also tended to have personality change without intellectual impairment in this series, as may happen after leucotomy, but this 'splitting' of function cannot be investigated further in this material, owing to the lack of exact knowledge of which frontal and para-frontal areas are damaged and the lack of independent assessment by more than one observer.

Other

The only psychometric study of patients with ruptured aneurysms discovered is that by Naughton (1954). He studies 51 patients a few weeks after the stroke, using Ravens Matrices, the Graham-Kendall Memory for Designs Test, the Terman-Merrill oral vocabulary and the Stanford-Binet sentence repetition Test. His A.C. group (17 cases) showed no important defect of vocabulary but did badly on the other three tests. Perhaps sentence repetition would be most like the P.A.T. but really all these tests are so different that one cannot generalise with confidence (see later).
(b) Hemispheric Infarction versus Cortical and Sub-cortical Damage.

Patients with P.C. aneurysms have relatively less intellectual impairment than those with M.C. aneurysms, allowing for the severity of C.N.S. signs, as was found for personality impairment (see Chapter V) - and presumably for the same reason that "dead" tissue interferes less with function than an area prone to aberrant activity.

(c) P.C. Aneurysm and Benton Test

Although patients with P.C. aneurysms had relatively less overall intellectual impairment than M.C. patients, as just outlined, they had worse scores on the Benton Test than the others, allowing for degree of C.N.S. signs and for intellectual impairment, and they more often had abnormal Benton scores in the presence of normal P.A.T. scores. In fact, it is the P.C. cases which are mainly responsible for the poor degree of overlap between the Benton and P.A.T. There is no obvious explanation for these findings and no relevant literature has been discovered but they are so marked that they may well reflect some aspect of localisation of cerebral function. In any case, they remind us of the complexity of the problem and the need for caution in making generalisations about abstract "cerebral functions" or "abilities", from psychological tests which may well have idiosyncratic and specific relationships to site of brain damage and to other variables (see later).

The older "mass action" theories of cerebral function,
which denied the importance of site of damage, have been modified in recent years. It is now generally agreed that both extent and site are important, a view supported by the evidence from this series of patients. However, current views are not supported in the next section.

(d) **Intellectual Changes and Side of Brain Damage.**

Pierce (1964) and Yates (1966), in their important reviews, show that most investigators find that verbal deficits are more marked with dominant hemisphere lesions; and, although there is less general agreement on this, that damage to the non-dominant hemisphere leads to more impairment on non-verbal tasks, especially those of a visuo-spatial nature. My expectations at the beginning of this study were that clinical intellectual impairment and the P.A.T. would be more impaired with left, and the Benton with right hemisphere lesions; and the tests were selected partly for this reason on the advice of psychologist colleagues. In fact, no such relationship appears when the presence and severity of C.N.S. signs is allowed for. It seems unlikely that the atypical findings here are due to differences arising from actual tissue pathology variations but rather to differences in the extent and site of damage and to factors specific to the tests and methods of assessment themselves. As Yates (op cit) has said, it is unwise to generalise too freely from the results of one or two tests and to assume that they are measuring some strictly definable mental function.

In one sense it is encouraging that the expected results were not obtained, as it indicates that the clinical ratings were
relatively free of bias, in that respect at least.

Naughton (op cit) studied 17 with right-sided and 17 with left-sided aneurysms, as well as an A.C. group mentioned earlier. His findings supported those made here in one important way, in that those with right-sided lesions did not score worse on the two visuo-spatial tests (Havens Patricee and Graham-Hendall) than did those with left-sided lesions. However, his left-sided cases did have worse scores on the verbal tests, unlike these, but test differences may well explain this.

3. Intellectual Change and the other Factors Considered.

It is interesting that age did not seem to be of importance once degree of C.N.S. signs had been allowed for, probably because most of the patients did not suffer from generalised arteriosclerotic or senile brain damage but only from the single trauma of their subarachnoid haemorrhage. Otherwise there are only two important findings under this heading:

(a) P.A.T. and Premorbid Intelligence.

Patients estimated as of below average premorbid intelligence scored very significantly worse on the P.A.T. than did the others, a difference which cut across all other categories. The finding is not surprising but it is more marked than expected from data which Inglis has distributed to colleagues in the past (Inglis, unpublished data); and indicates that premorbid intelligence should probably be allowed for in interpreting the results of any paired associate test.
(b) **Benton and Sex Difference.**

The finding that women scored worse than men on the Benton Test at the 0.1% level is more surprising, although it was not true of those with P.C. aneurysms, who tended to have worse scores anyway. It is possibly explained by a difference in the organization of cerebral function in men and women, as has been demonstrated for Raven's Matrices, Memory for Designs Test and the Goodenough Draw-a-Man Test.

It seems that in women results on these tests may be more affected by cerebral lesions than in men, because, in the former, performance on them is dependent more on a specific spatial factor, whereas, in men, performance is related more to "general intelligence" (Smith, 1964).

4. **Inter-relationships of Clinical Intellectual Impairment, P.A.T. and Benton Test.**

Overall the P.A.T. was much more closely related to clinical assessment of intellectual impairment than was the Benton, although both tests are related similarly to C.N.S. signs, presumably because clinical assessment also relies largely upon the spoken word. Conversely, the P.A.T. and Benton were much less closely related to each other than the former was to clinical assessment. Unfortunately, the clinical assessments were recorded after the two tests had been done and, although a conscious effort was made not to be swayed by their results, it is probable that there was some halo effect — presumably from the P.A.T.
CONCLUSIONS

Intellectual impairment of varying degrees is common after subarachnoid hemorrhage, occurring in 41% of this series on clinical assessment. It is closely associated with neurological evidence of brain damage and, depending on the method of assessment, is influenced by the site and extent of brain damage, premorbid intelligence and sex. Surprisingly, none of the three measures used are influenced by the side of the brain damaged.

As appeared in Chapter V, during the study of personality changes, these data further support the idea that there is no single measure of brain damage, only measures of different aspects of mental function. Comparisons with other studies cannot be close, because of differences in the test used, which, despite superficial resemblances, seem to measure different functions.
CHAPTER VII

AFFEETIVE CHANGES AFTER SUBARACHNOID HEMORRHAGE

In this Chapter also, as in Chapters V and VI, only the first series of 261 patients is considered. The rating scales and definitions used are described in Chapter III.

RESULTS

Of the total case material of 261 patients, 69 (26%) were rated as having been depressed in the follow up period: 31 (12%) mildly, 30 (12%) moderately, and 8 (3%) severely. The data have been grouped in a number of ways in order to identify factors associated with the development of depression, of which the most important seems to be the presence of brain damage, a concept which can be defined in various ways but clinically must be recognised by its effects on the physical or mental state.

(a) C.N.S. Signs as Evidence of Brain Damage.

There were 113 (43%) patients with C.N.S. signs, of whom 42 (37%) were rated as depressed; compared to only 27 (14%) without signs. ($\chi^2 = 11.748$, 1 d.f., $p < 0.001$). The breakdown is shown in Table 32.
If this table is simplified, we find that depression is significantly more common in those with moderate or severe signs (24.47 or 51%) than in those with mild signs (14.66 or 27%), $\chi^2 = 6.588$, 1 d.f., $p < 0.05$. However, it is not more common in those with severe signs (12.26 or 46%) than in those with moderate signs (12.21 or 57%), and there is no tendency for more severe depression to occur in those with the more marked signs.

Thirty six patients were recorded as having C.N.S. signs while in hospital but not at follow up. The incidence of depression in that group was 6.36 (17%), almost identical with the incidence in all those with no C.N.S. signs at follow up (16%). Thus,
 transient neurological signs were not related to the development of depressive states.

(b) Abnormal Mental State as Evidence of Brain Damage

The presence or absence of neurological signs is not a satisfactory way of defining brain damage, as undoubted damage can occur without leading to such signs, particularly with lesions in the frontal lobes.

In earlier chapters of this thesis, it was shown that on the whole the patients with neurological signs were those who also suffered from personality and/or intellectual changes but that there were some important exceptions. If intellectual and personality impairment are related separately to the incidence of depression, as was done for C.N.S. signs in Table 32, then very similar figures to these are produced (details omitted), as would be expected in view of the overlap described. However, the two psychological tests used, the Benton Visual Retention Test and the Inglis Paired Associate Test, were not significantly related to the depression, (details omitted), which suggests that it is the personality changes which are more important, as described later.

(c) Mental & Physical Changes Combined as Evidence of Brain Damage.

In order to include those with significant mental changes but no C.N.S. signs, the following definition of brain damage is used from now on: patients are considered as brain damaged if they had neurological signs at follow up; or if, even in the absence of
such signs, they had been rated as "moderate" or "severe" on either of the two scales of personality and intellectual impairment; or as "mild" on both scales. Thus, a single rating of mild intellectual or personality impairment is ignored.

Using the new definition, 136 of 261 (52%) were brain damaged, compared to 43 who had C.N.S. signs. Derived as it is from C.N.S. signs and personality and intellectual impairment, "brain damage" naturally bears the same relationship as did those three findings to the various factors considered and described in the earlier chapters. Thus, brain damage is significantly more common in those with aneurysms than in those without (p < 0.001); is significantly commoner in those with M.C. than in those with A.C. and P.C. aneurysms combined; and is significantly more common in older and in hypertensive people.

It was not significantly associated with the following: side of aneurysm or C.N.S. signs; operative or conservative treatment overall (although there were important differences between the aneurysms groups in this respect; see Chapter IV); or with a personal or family history of treatment for psychiatric disturbance.

There were no differences between the brain damaged and non-damaged groups on the premorbid personality ratings described in Chapter III.

Table 33 shows that, of the 136 brain damaged patients, 52 (38%) were depressed, compared to only 17 (14%) of the non-brain damaged. This is a rather more highly significant association than was found when C.N.S. signs alone were taken as the criterion of "brain damage". (χ² = 20.21, 1 d.f. p < 0.001).
Table 33 also shows that mild depression is commoner in the brain damaged, although this is not significant.

**TABLE 33.**

<table>
<thead>
<tr>
<th>Depression</th>
<th>Absent</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brain Damaged</td>
<td>84</td>
<td>25</td>
<td>21</td>
<td>6</td>
<td>136</td>
</tr>
<tr>
<td>Not Brain Damaged</td>
<td>128</td>
<td>6</td>
<td>9</td>
<td>2</td>
<td>125</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td>192</td>
<td>31</td>
<td>50</td>
<td>8</td>
<td>261</td>
</tr>
</tbody>
</table>

Even if those with mild depression are counted as non-depressed and the moderate and severe depressives grouped together, depressive states are still significantly more common in the brain damaged, ($\chi^2 = 6.350, 1 \text{ d.f.}, p < 0.05$).

**Other Factors in the Development of Depression**

(a) Aneurysm Group.

The incidence of depression and brain damage in the various aneurysm groups is shown in Table 34. Depression is most common with P.C. aneurysm, although brain damage is commoner in the N.C. group. The single aneurysm groups do not differ significantly from each other in terms of age, sex or blood pressure or previous personality ratings.
<table>
<thead>
<tr>
<th></th>
<th>A.C.</th>
<th>H.C.</th>
<th>P.C.</th>
<th>No an.</th>
<th>Hult.</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brain Damaged (Depressed)</td>
<td>10/40</td>
<td>10/48</td>
<td>16/37</td>
<td>4/6</td>
<td>2/5</td>
<td>52/136</td>
</tr>
<tr>
<td></td>
<td>(25%)</td>
<td>(37%)</td>
<td>(49%)</td>
<td>(67%)</td>
<td>(40%)</td>
<td>(38%)</td>
</tr>
<tr>
<td>Not Brain Damaged (Depressed)</td>
<td>5/41</td>
<td>3/23</td>
<td>7/35</td>
<td>2/24</td>
<td>0/2</td>
<td>17/125</td>
</tr>
<tr>
<td></td>
<td>(13%)</td>
<td>(13%)</td>
<td>(20%)</td>
<td>(9%)</td>
<td>(0%)</td>
<td>(14%)</td>
</tr>
<tr>
<td>TOTALS</td>
<td>15/81</td>
<td>13/72</td>
<td>23/72</td>
<td>6/30</td>
<td>2/7</td>
<td>69/261</td>
</tr>
<tr>
<td></td>
<td>(19%)</td>
<td>(34%)</td>
<td>(35%)</td>
<td>(23%)</td>
<td>(29%)</td>
<td>(26%)</td>
</tr>
</tbody>
</table>

Depression was significantly more common with P.C. than with A.C., analysed overall \( \chi^2 = 5.172 \) d.f. \( p < 0.05 \); and in the brain damaged groups \( \chi^2 = 4.761 \) d.f. \( p < 0.05 \).
Other differences are not significant.

(b) Premorbid Personality Ratings.

The method was described in chapter III. Table 35 shows that the depressed were as a group rated significantly more anxious and less outgoing than the others, but no differently for energy and obsessiveness.
<table>
<thead>
<tr>
<th>AUXILIARY PRESS</th>
<th>OBSESSIOANAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Below Average</td>
</tr>
<tr>
<td>Not Depressed</td>
<td>45</td>
</tr>
<tr>
<td>Depressed</td>
<td>9</td>
</tr>
</tbody>
</table>

\( x^2 = 7.0403, 2 \text{ d.f.}, p < 0.05. \)

<table>
<thead>
<tr>
<th>ENERGY</th>
<th>OUTGOINGNESS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Below Average</td>
</tr>
<tr>
<td>Not Depressed</td>
<td>9</td>
</tr>
<tr>
<td>Depressed</td>
<td>3</td>
</tr>
</tbody>
</table>

Not significant

\( x^2 = 6.4455, 2 \text{ d.f.}, p < 0.05. \)
There were no differences on those ratings between any of the single aneurysm groups; but the no aneurysm patients were significantly more often of lower energy, their strokes more often took place at a time of emotional turmoil and they had more frequently a past history of psychiatric treatment.

These factors are probably related to the rather high incidence of depression they show, despite a low incidence of C.N.S. signs; (detailed tables are given in Chapter VIII).

Although P.C. aneurysm patients did not differ on the personality ratings, a number of those depressed had neurotic character disturbances, which may be related to their rather high incidence of depression (but see later).

(c) Previous History of Psychiatric Treatment

This consisted mainly of psychotropic drugs from the general practitioner. It was significantly more common in the depressed (16/69 depressed versus 2/4192 others. \( \chi^2 = 4.421 \), 1 d.f., p 0.05). There were only small differences between the single aneurysm groups.

(d) Other Factors.

No significant differences were found between the groups of depressed and non-depressed patients in any of the following factors: age; sex; marital state; a history of treated psychiatric illness in first degree relatives; operative or conservative treatment for aneurysm; side of the aneurysm; or of neurological signs; hypertension or migraine.
Depressed patients did not spontaneously express fears of recurrent subarachnoid haemorrhage more often than the non-depressed. Many of those who did so had anxiety states (see later), although of course many depressed patients had also had significant anxiety symptoms and very many patients admitted such a fear when questioned.

There were important differences between the brain damaged and non-brain damaged depressed in the various factors just considered, which will be discussed in a later section.

Clinical Features of Depression in those with Brain Damage.

The rating of depression as mild, moderate and severe conceals important differences in the clinical pictures of those with and without brain damage. In those with such damage, there was in nearly all cases an associated or underlying personality change - which was indeed an important diagnostic feature of the group, (Chapter V).

Mild Depression.

In the 23 patients with brain damage and mild depression, there was in all but four a chronic, shallow depressive mood, often lifting quickly in response to some new stimulus but rapidly falling back with apparent boredom, loss of interest or easy fatigue. They were usually apathetic but irritable and frequently withdrawn, so that they would often sit alone and unoccupied, brooding unhappily for hours at a time. They were mainly well aware of their changed temperament and of how difficult they were to live with. Anxiety was common and was almost always "situational" in
that it was provoked by any task or demand made upon them with which they felt unable easily to cope. At times they would become more markedly depressed and might weep or go off on their own - sometimes walking out of the house and disappearing for hours at a time. These spells usually lasted only a few hours and were often, but not always, in response to a family quarrel or some other upset. All but four of these 25 patients had at times wished that they were dead but none had attempted suicide. There was rarely any marked disturbance of appetite, weight or sleep. Only 17 had received treatment for their mental state, 13 from their G.P., one as an inpatient in a chronic sick hospital and one had been admitted to a local mental hospital for assessment and treatment. Ten of these patients felt that they had been helped by treatment but their relatives rarely agreed. The clinical picture did not seem to change with the passage of time nor was it less frequent in those followed up after a longer period.

This state of "organic moodiness" then is a combination of personality change with depressive mood and verges at one end of a spectrum into personality change characterised by flatness of affect and apathy, and at the other into more severe and prolonged depressive states.

**Moderate Depression.**

Twenty one brain damaged patients were rated as having moderately severe depression. In 15 it arose recurrently from a background of "organic moodiness", each spell lasting up to several
months. A few seemed per seerently more depressed. Three had had inpatient, seven outpatient and nine O.P. treatment. All but four had had sleep disturbance and appetite or weight loss but rarely severe. One had made a serious suicidal attempt with barbiturates and 10 others claimed to have contemplated suicide. Of the 6 patients without "organic moodiness", 4 made a complete recovery; of the others, 6 had made a worthwhile improvement.

Severe Depression.

Of 6 brain damaged patients with severe depression, 5 had had inpatient treatment; the sixth had a severe physical disability and had been nursed at home for years in a state of severe agitated depression, dramatically improved by imipramine just before my visit. All showed a worthwhile response to treatment but only one made a complete affective recovery. All had had severe disturbances of sleep, appetite and weight and one had made a serious suicidal attempt.

Clinical Features of Depression in those not Brain Damaged.

Mild Depression.

Of the 6 with mild depression without brain damage, 5 women and one man, all had had O.P. treatment. In 4, the depressive states had been transient, in reaction to environmental stress, and similar to disturbances before the subarachnoid haemorrhage. Two had chronic mild anxiety/depressive states with agoraphobic symptoms and both expressed fears of recurrent haemorrhage in this context.
Moderate Depression.

Of the 9 with moderately severe depression, 8 were women. One had had inpatient, five outpatient and three GP treatment. In four women and one man, all of whom had had ruptured P.C. aneurysms, the depressive states were unusual, in that the patients had not been subject to depression before and that, from the time of the haemorrhage, they had been almost continuously depressed, all but one responding poorly to treatment. The four women mentioned disturbances which could have been hypothalamic in origin: altered sensitivity to temperature and disturbances of menstruation. The man described a change of sleep habit, in that he tended to sleep for 16 hours at a stretch, and also complained of curiously sticky and "ropy" saliva. (He lost his complaints completely when treated with amitriptyline by the author). None of these patients showed any clinical evidence of hypopituitarism.

Four of these women, one of whom had an "unusual" severe depression, as indicated above, had rather/neurotic character disorders; although they had not been depressed before.

Severe Depression.

This occurred in two women, both with P.C. aneurysm. One had a past history of hypomania while on steroid hormone therapy, and was in hospital for a year with a manic depressive (bipolar) illness, which began within days of her haemorrhage. The other had no past history of depression but developed an intractable agitated depression within days of the haemorrhage and was finally leucomatized after three years with only moderate
Depression in the Brain Damaged: Other Factors

(a) Reaction to Disability.

In individual cases it naturally seemed that physical handicap or intellectual or personality change led to chronic stresses which were in themselves depressing. Overall however, the role of these factors was difficult to demonstrate. Thus of the 7 patients who had severe C.M.S. signs but no organic mental changes, only one was depressed. Of the 6 most severely depressed brain damaged patients, two had no C.M.S. signs, two mild signs and two severe signs. Similarly, there was no evidence that people of high previous intelligence were more likely than others to become depressed if they suffered only mild intellectual impairment - a possibility suggested by the work of Billson (1960) with head injury patients. This applied using the Benton, P.A.T. and clinical assessment of impairment, (figures omitted).

Of the 10 most severely brain damaged - that is, with both severe intellectual and personality change combined with moderate or severe C.M.S. signs - none was depressed, because the severity of the damage led to affective flattening, apathy or emotional lability; and to relative inaccessibility.

(b) Aneurysm Group.

Depression in the brain damaged was almost significantly less common with A.C. aneurysm than with M.C. and P.C considered
together, as shown in Table 54, ($^2 = 3.49$), probably because of the qualitatively different personality change in the A.C. group. The rather higher incidence in the A.C. group may be due to personality differences, as mentioned, or could possibly be associated with hypothalamic damage, as mentioned earlier for none of the non-brain damaged depressed. (P. C. aneurysms often involve the hypothalamus directly when they rupture, (Chapter II).

(c) Premorbid personality.

Table 56 shows that, of the brain damaged patients, those with depression were rated as significantly more often energetic than the non-depressed.
TABLE 36.

PERSONALITY RATINGS IN THE BRAIN DAMAGED

<table>
<thead>
<tr>
<th></th>
<th>ANXIETY PRONE</th>
<th>OBSESSINOAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Below Average and Average</td>
<td>Above Average</td>
</tr>
<tr>
<td>Depressed</td>
<td>29</td>
<td>23</td>
</tr>
<tr>
<td>Not Depressed</td>
<td>49</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>78</td>
<td>53</td>
</tr>
<tr>
<td></td>
<td>Not significant</td>
<td>Not significant</td>
</tr>
</tbody>
</table>

ENERGY OUTPUT

OUTGOINGNESS

<table>
<thead>
<tr>
<th></th>
<th>Below Average and Average</th>
<th>Above Average</th>
<th>TOTAL</th>
<th>Below Average and Average</th>
<th>Above Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressed</td>
<td>28</td>
<td>24</td>
<td>52</td>
<td>11</td>
<td>41</td>
</tr>
<tr>
<td>Not Depressed</td>
<td>56</td>
<td>23</td>
<td>79</td>
<td>15</td>
<td>64</td>
</tr>
<tr>
<td></td>
<td>84</td>
<td>47</td>
<td>131</td>
<td>26</td>
<td>105</td>
</tr>
</tbody>
</table>

$\chi^2 = 3.8922, 1$ d.f.

$p < 0.05$.

(Not significant)

(The ratings are grouped into 2 x 2 tables as the numbers are small)
(d) **Other Factors.**

There were no differences even approaching significance between the depressed and non depressed brain damaged patients, using any of the following factors: sex; age; past history or family history of psychiatric treatment; side of aneurysm or of C.N.S. signs; operative or conservative treatment; hypertension; or migraine. If the moderately and severely depressed are compared to those with mild depression (that is, those mainly with "organic moodiness"), then the more severely depressed do show greater background disturbance, in that they more often have a previous history of psychiatric treatment (8:27 versus 3:25); and a positive family history (11:27 versus 6:25). The numbers are too small for useful statistical comparison but the trends are in the expected direction.

**Depression in the Non Brain Damaged - Various Factors.**

There were only 17 depressed compared to 108 not depressed in the patients without brain damage. Although the number depressed is small, it is sufficient to show some marked differences between them and the others. Yates correction is used in the statistical comparisons where appropriate.

(a) **Premorbid Personality Ratings.**

Table 37 shows that, for the non brain damaged patients, the depressed are significantly more often described as more anxiety prone, of lower energy and less outgoing. Although the numbers concerned are small, the differences are so marked that
they led to statistically significant findings of more anxiety prone and less outgoing for the much larger number of all those depressed (Table 35).

**Table 35.**

DEPRESSIVE SYMPTOMS IN THE NON BRAIN DAMAGED.

<table>
<thead>
<tr>
<th></th>
<th>ANXIETY PRONE</th>
<th>OBSESSIONAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average &amp; Below Average</td>
<td>Above Average</td>
</tr>
<tr>
<td>Depressed</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>Not Depressed</td>
<td>72</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td>76</td>
<td>49</td>
</tr>
<tr>
<td>$\chi^2 = 11.319$, 1 d.f., $p &lt; 0.001$</td>
<td>Not significant</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>ENERGY</th>
<th>OUTGOINGNESS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average &amp; Below Average</td>
<td>Above Average</td>
</tr>
<tr>
<td>Depressed</td>
<td>14</td>
<td>3</td>
</tr>
<tr>
<td>Not Depressed</td>
<td>51</td>
<td>57</td>
</tr>
<tr>
<td></td>
<td>65</td>
<td>60</td>
</tr>
<tr>
<td>$\chi^2 = 7.374$, 1 d.f., $p &lt; 0.01$</td>
<td>$\chi^2 = 14.456$, $p &lt; 0.001$.</td>
<td></td>
</tr>
</tbody>
</table>
(b) Sex.

Only 2:17 of the non brain damaged depressives were men, compared to 53:108 of those not depressed, \( \chi^2 = 8.354, 1 \text{ d.f.}, p < 0.01 \).

(c) Past History of Psychiatric Treatment.

This was obtained in 5:17 depressed and 11:108 not depressed; a non significant difference in the expected direction.

(d) Other Factors.

No differences even approaching significance appeared when the depressed and not depressed without brain damage were compared on the following factors: side of aneurysm; operative or conservative treatment; age; hypertension; or migraine. Of the 11 rated moderately or severely depressed, 7 had P.C. aneurysms, all but one of whom had persistent or recurrent depression. The numbers are too small for statistical analysis.

ANXIETY SYMPTOMS

No detailed breakdown of the anxiety symptoms is given here for several reasons. Firstly, anxiety is a common accompaniment of depression in this series as in ordinary clinical psychiatry. Secondly, anxiety and depression were not distinguished in the past and family histories of the patients. Thirdly, and most importantly, because so many brain damaged patients have some "situational bound" or "stimulus bound" anxiety of catastrophic type, as discussed later.
In this section, patients with anxiety symptoms in the period leading up to the stroke are excluded, except for two in whom distinct and prolonged worsening occurred. Patients in whom the anxiety symptoms were rated as 'mild' are also excluded.

Twenty-eight patients had moderate or severe anxiety symptoms, equally distributed between brain damaged and non-brain damaged groups (16:136 versus 12:125). Seven of the 28 had "pure" anxiety states, not associated with either depression or brain damage; 5 had evidence of brain damage without depression; 5 had associated depression (moderate or severe) without brain damage; 11 had both brain damage and depression.

These moderate and severe anxiety symptoms occurred in 18 women and 10 men, a proportion not significantly different from that in the whole series. Of the 12 patients with anxiety but without brain damage, 10 were women and only 2 were men; of the 16 with brain damage, there were 8 of each sex. This sex difference is the same as that found for depressive symptoms.

Clinically, those with anxiety symptoms fell into three main classes. Characteristic of the brain damaged patients was "catastrophic" anxiety, mounting to uncontrollable levels at times, when faced by difficult or unfamiliar situations. In those with depression and no brain damage, there were mixtures of the two symptoms, often with phobic features. Of the 7 patients with "pure" anxiety states, 5 were women and all had had psychiatric treatment for their symptoms. Five had severe agoraphobia limiting them largely to the house but in four this improved within 18 months, although none had lost all symptoms.
and one was still unable to go out of the house alone at a three year follow up. The others could go out of the house but had abandoned many activities - including housework and shopping, were constantly tired and could be described as "neurasthenic". All but 5 patients with moderate or severe anxiety but without brain damage expressed spontaneously a great fear of another stroke, including all those with "pure" anxiety. This spontaneous expression was relatively uncommon in the series as a whole and in the depressed patients also but very many patients admitted to a fear of recurrent haemorrhage on questioning. Each of those with pure anxiety states claimed to have been told by a doctor or nurse that activity was dangerous and to be avoided; and, although this is against the policy of the neurosurgical unit, it is nevertheless still advised by many. Some patients had been advised never to put their heads below the level of their hearts or never to lift a heavy shopping basket, etc; and even advice to "take things easily" seemed to have produced unnecessary invalidism at times. On the whole, however, most patients had ignored such advice with robust common sense and they had gradually taken up normal activities and many had been effectively reassured at follow up visits to the neurosurgical outpatient clinic.

One of the principal personality changes described in the brain damaged patients was an increased proneness to anxiety (Chapter V). In almost all, as mentioned already, this was situational, in that, when confronted by demands for activity, for concentration or for emotional response, depending of course on
the nature of the disability, they become anxious and often 'irritable'. This anxiety is not "free floating" or related to fears or to emotional conflicts, etc., of long standing but is really a milder form of catastrophic anxiety. A typical example is that of a patient with a visual field inattention defect, who is effectively blind in one half field when presented with simultaneous stimuli from both sides. When crossing the road, such a patient is bewildered, frightened and has mounting anxiety, made worse by the fact that he cannot understand or explain his disability, as he is not blind when he is out of the situation. The almost invariable response of patients is to limit activity, to withdraw from others and thus to keep the environment simple and under control. In most cases, this situation bound anxiety can be distinguished from neurotic anxiety with fair confidence.

**SUMMARISED RESULTS**

The principal findings can be summarised as follows:

1. Depression occurred in 691261 (26%) overall and was significantly more common in the brain damaged, whether this was defined in terms of C.N.S. signs only or using a criterion derived from C.N.S. signs combined with personality and intellectual change. Transient C.N.S. signs were not associated with later depression. More severe brain damage was associated with more frequent but not with more severe depression; and no simple overall association between severity of depression and physical or mental disability could be demonstrated.
2. Differences in the incidence of depression in the brain damaged group, other than those due to variations in the severity of the brain damage, could be explained partly by the following:

(a) The nature of the personality change in those with A.C. aneurysm, who more often showed a frontal lobe syndrome;

(b) previous personality differences; and

(c) possibly by direct hypothalamic involvement in the case of the P.C. aneurysm group.

3. In the brain damaged patients, mild depression was in most cases an inseparable part of the organic personality change which had occurred ("organic moodiness") and more severe depressive states also arose from this background. In the non brain damaged, the mild depressive states were similar to those the same patients had had before their stroke but there was again a possibility that hypothalamic damage was involved in some of the more severe cases, who had not been depressed before.

4. There were important differences between the brain damaged and non brain damaged depressives in personality and in the degree of background disturbance:

(a) The brain damaged depressed were rated as more energetic than those brain damaged but not depressed; but the non brain damaged depressed were rated as more anxiety
prone, less energetic and less outgoing than their fellows.

(b) A past history of psychiatric treatment was commoner in the non brain damaged depressives but not in the brain damaged depressives overall. However, in the latter group, the more severely depressed tended to have had more psychiatric treatment in the past.

(c) Depression in the brain damaged was equally present in men and women but, in the non brain damaged, was almost confined to women.

(d) Side of brain involved, greater age and increased blood pressure were not related to depression, even though the latter two were associated with more brain damage.

5. Anxiety symptoms were very common, especially in the form of 'situation bound' anxiety in the brain damaged but have not been analysed in detail. As with depression, marked anxiety symptoms were equally present in men and women with brain damage and almost confined to women otherwise.
DISCUSSION.

The high incidence of depressive symptoms reported here (26%) cannot validly be compared statistically to the incidence in the community shown by population surveys, because of the differences in method and selection. However, in this series, 9 of 136 brain damaged and 3 of 125 non-brain damaged patients were admitted to hospital for treatment of depression; an overall rate of approximately 46 per 1,000 over a mean follow-up period of 2.8 years; and the outpatient referral rate was also 46 per 1,000. The Ministry of Health Annual Report for 1959 for England and Wales showed an inpatient admission rate of 1.78 per 1,000 for all diagnostic categories per annum. Even allowing for the 2.8 year mean follow-up period, the rates in this series of patients seem very strikingly high and these comparisons provide objective backing for the otherwise essentially subjective ratings used.

Only two studies of subarachnoid haemorrhage can be usefully considered here. That by Short et al (1968) showed that 18 out of 29 patients (62%) were more irritable or more prone to anxiety and depression but insufficient detail is given for further comparisons. Logue et al (1968) found that 10/79 patients with A.C. aneurysms were more depressed than before the illness and their cases seem to have been comparable in other respects to the 81 A.C. patients studied here, of whom 15 were rated as depressed. The difference may be random or may be due to the methods of rating, where mild depressive states and organic personality change are so interwoven. (In this series, A.C. patients had the lowest incidence of depression).
As mentioned earlier, no psychiatric follow up of large unselected series of patients with other cerebrovascular accidents have been carried out but Roth (1955) states that 30% of patients with arteriosclerotic psychoses show affective changes; and Tourney et al (1958) found that in 24 patients with occlusive cerebral vascular disease, 9 (37%) suffered from depression, 12 from significant anxiety and 20 had some intellectual impairment; so that the morbidity was even higher than in this series.

There is still uncertainty about the role of "organic factors" in depressive states in the elderly, when arteriosclerotic brain disease is common; and there is an extensive literature on this subject, reviewed by Post (1965), who found that in his own series of elderly depressives, focal, presumably vascular, cerebral pathology was more frequently present on admission than "expected" and concluded from this and other findings that cerebral arteriosclerosis was aetiologically associated with the affective syndrome. Kay, Roth and Hopkins (1955), however, took the view that cerebral degeneration of the kind found in the senile and arteriosclerotic psychoses is unlikely to be an aetiological factor of any importance in the causation of affective psychoses in late life. A large scale follow up of patients after cerebrovascular accidents is needed but a study of patients who have had a subarachnoid haemorrhage is relevant, because it is a cerebrovascular illness, but, unlike other such illnesses, is non progressive and not so markedly associated with widespread arterial disease nor with cerebral degenerative processes. In fact, age in this series was not important except in so far as
older people tended to have more C.N.S. signs.

It is widely accepted that, in terms of constitutional and hereditary loading, those who develop affective psychoses in later life have manifested a greater resistance to the responsible gene until undermined by the new stresses of old age and bodily infirmity (Post, op cit). This study is not concerned with affective psychosis as such, nor are the patients old, but the results could be interpreted as compatible with that hypothesis. Certainly, the incidence of depressive states, including those severe enough to need hospital treatment, seems to be considerably higher in this series of patients than in the general population. In addition, Post made the point that in the aged "organic depression" was not a syndrome necessarily characterised by shallow and labile affect or merely a passing phase of some dementing process. Instead, depression in those with organic brain damage can be very severe, suicide is not uncommon and "the patient's thought content may be indistinguishable from that seen in other elderly melancholics". Certainly that applies to the patients in this series too, who frequently developed severe depression against a background of mild organic affective change.

It is impossible to be sure of the reason for the increased incidence of depression in the brain damaged, uncertain as we are about the real nature of depressive states in any case. Naturally one can 'understand' the relationship in terms of a reaction to the illness, with loss or impairment of physical and mental abilities, but overall no simple relationship to severity of disability was apparent. In psychoanalytic terms, brain damage,
with its impairment of function, is recognised by the ego as
causing an "impoverishment of the sum total of narcissistic
libido and as a crucial blow to self esteem". This blow is
then equivalent to that represented by the loss of some love
object in classical psychoanalytic terms; and repeats the
injury to infantile narcissism, which occurred in childhood;
and predisposed the individual to depression in later life;
(see Brodin (1952) for discussion of this subject and further
references). As an explanation, this seems to say little more
than that the patient predisposed to depression feels diminished
and damaged by his illness and becomes depressed as a result.
In any case, no clinical data which could be related to the
theory were collected.

A family history and personal past history of psychiatric
troubles were surprisingly unimportant in the brain damaged group,
although they were more prominent in those with more severe
depression. Interestingly, men and women were equally affected
in the brain damaged group, although men were rare in the non-
damaged depressives.

The previous personality ratings in the brain damaged
group also produced a surprising result, in that the depressed
were described as more often energetic. Hillbom (op cit) found
that, in his series of head injured patients, depression was more
common in intelligent patients, presumably because they reacted
more intensely to the intellectual impairment. In this series,
intelligence did not appear as a relevant factor but perhaps more
energetic people suffer more from the limitations imposed by their
disability in an analogous way. Altogether however, although we
can understand the depression as a reaction to disability, it is probably not a complete explanation; and we are left with the familiar problem of reconciling these two aspects of understanding and explanation, as set out by Jaspers (1962).

The 17 depressed patients without brain damage were more like the depressed patients one sees in ordinary clinical practice; mainly women with vulnerable personalities (anxious, low in energy and withdrawn), and a marked personal and family history of psychiatric treatment. Again, there is the faint possibility of hypothalamic involvement but very little is really known of the effects of hypothalamic disturbance on mood, although it is at present a fashionable area of the brain to invoke.

Altogether, it seems to the author that brain damage is depressing in itself, in some uncertain way which goes beyond reactivity and understanding in psychological terms.
In this Chapter both series of patients, the first of 261 and second of 30 patients, are considered. Rating scales and definitions will be found in Chapter III. The neurosurgical notes of a further 203 patients were also studied, as described in the text.

It is well known that strokes can occur in paroxysms of rage or other strong emotion, possibly because of a sudden rise in blood pressure. Many other strokes occur at rest, even during sleep, or during vigorous physical exertion. This Chapter presents evidence that in patients with subarachnoid haemorrhage the role of emotional stress may be greater in those with normal angiograms than it is in those with cerebral berry aneurysms. The no aneurysm group of patients was included as a control group, of a sort, without any expectation of, or indeed any interest in, psychosomatic aspects of precipitation of the haemorrhage. They had had the same frightening experience of a stroke "out of the blue", had been through the same routine of (usually) urgent admission, physical examination, compression of the carotid arteries for 10' on each side, followed by bilateral carotid and unilateral vertebral angiography, and then had had 6 weeks rest in bed, as did the conservatively treated aneurysm cases. Both groups of patients could equally fear recurrence. They had, therefore, had very similar emotional stresses to the aneurysm patients and it was hoped that this would be useful in assessing
such things as fear of coitus, of physical effort, of travelling, etc., and their relation to anxiety and depression.

The aneurysm and no aneurysm groups had little else to distinguish themselves by and all patients were supposed to be advised that activity was not harmful, although many received conflicting advice outside hospital. One might expect that those who knew and understood that they had an aneurysm might have more fear of recurrence than those who knew they did not - but this type of sophisticated knowledge did not seem important when they were interviewed.

FIRST STUDY

Unfortunately no specific questions were asked at follow up about the patients' circumstances and mental state at the time of the stroke, as it had not occurred to me that this was of interest. However, a number of patients spontaneously claimed that their illnesses had been due to mental stress, in some cases very striking. (In each of the cases quoted, the account was supported by a relative).

There were four particularly dramatic episodes in the 261 patients:

1. A woman of 50, in personality described as bustling, managing and always worrying. She had always been frigid and she and her husband seemed very ambivalent towards each other. She had been in perfect health when a policeman came to the door and asked if he could use the telephone. She asked why and he told her that the woman next door, who was her closest friend, had hanged herself in the lavatory. She said, "Oh my God", and had her sub-
2. A woman of 49, in personality described as very energetic, houseproud, outwardly calm but inwardly tense, sensitive, devoted to her family and home, with few outside interests. She was watching television, when an aeroplane on a test flight was shown exploding in mid-air. She believed that her son was in it (although he was, in fact, off sick) and she too put her hands to her head and had her stroke. (Multiple aneurysms. Her second haemorrhage).

3. A woman of 35, in personality tense and moody, very obsessional and worrying; of low energy in that very easily fatigued, who had suffered for years from a chronic anxiety state and was very unhappily married. She had her subarachnoid haemorrhage within a minute of being told by her husband that he was going to divorce her for adultery – which she thought he knew nothing about. (No aneurysm).

4. Another woman, rather tense and worrying but otherwise stable, had her haemorrhage a few hours after a telephone call from hospital, which told her that her son had been seriously injured in a motor cycle crash. She had immediately after the phone call developed a panic anxiety state, which persisted until the stroke. (No aneurysm).

Another two were less dramatic and not so clearly related to an acute stress.

5. A woman of 46, again described as tense and anxious,
had nursed her husband for over a year as he slowly died of cancer. Her subarachnoid haemorrhage occurred in the last few hours of his life, as she knew he was dying. (No aneurysm).

6. Another very anxious and obsessive woman of 47, plagued all her married life by obsessive thoughts of sex and obscene words, developed an acute anxiety state after some sexual experience with her husband, which neither could bring themselves to describe to me. She had her haemorrhage three days later, in a state of great anxiety and tension. (No aneurysm).

Three others, two of them women and each with no aneurysm, said spontaneously that they were anxious and depressed at the time of the stroke; but there were no convincing time relationships mentioned, nor any other details recorded.

Three others with no aneurysm had past histories of affective disorders, treated by their general practitioners, but did not link their strokes with anxiety or depression.

Altogether, 11:30 patients with no aneurysm gave some history of an affective disturbance before the stroke, as just outlined. Of the 231 aneurysm patients, only one described a dramatic association and a further 28 had at some time had G.P. or other treatment for an affective disturbance, although none of these linked them to the onset of the stroke; a difference significant at the 1% level, when affective disturbances in the immediate and more distant past are grouped together. ($\chi^2 = 11.88$, 1 d.f., $p < 0.01$).

If we allow for the fact that more aneurysm cases had memory impairment at follow up and exclude those with more than
mild intellectual impairment (that is, with more than forgetfulness for recent events mainly), the figures are as follows: no aneurysm cases, 11:29; aneurysm cases, 29:192. (χ² = 9.063, 1 d.f., p < 0.01).

Clearly it is possible that it may only be the frequency of complaints which is being compared here, rather than the true incidence of affective disturbance but, even so, this could well reflect personality differences.

It is interesting that all of those describing dramatic precipitating stress were women; as were all but 12 of the 35 others who gave a past history of some treated affective disturbance.

**Previous Personality Assessment**

Table 38 shows the previous personality ratings for those with and without aneurysms, grouped to maximise any differences.

The no aneurysm cases do rate significantly more 'below average' on energy (a characteristic perhaps related to "neuroticism" as a global concept) and they are also non-significantly more anxiety prone. Unfortunately, the numbers are too small to be really convincing.
### Table 56.

#### ASSESSMENT OF PREVIOUS PERSONALITY:
**ANEURYSM VERSUS NO ANEURYSM**

<table>
<thead>
<tr>
<th>Anxiety proneness</th>
<th>Below average and Average</th>
<th>Above Average</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No aneurysm</td>
<td>14</td>
<td>16</td>
<td>30</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>140</td>
<td>86</td>
<td>226</td>
</tr>
<tr>
<td></td>
<td>154</td>
<td>102</td>
<td>256</td>
</tr>
</tbody>
</table>

\( (X^2 = 2.518, \text{ Not significant}) \)

<table>
<thead>
<tr>
<th>Obsessionalism</th>
<th>Below Average</th>
<th>Average</th>
<th>Above Average</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No aneurysm</td>
<td>2</td>
<td>11</td>
<td>17</td>
<td>30</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>18</td>
<td>101</td>
<td>107</td>
<td>226</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>112</td>
<td>124</td>
<td>256</td>
</tr>
</tbody>
</table>

(almost identical)

<table>
<thead>
<tr>
<th>Energy</th>
<th>Below average</th>
<th>Average and Above average</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No aneurysm</td>
<td>5</td>
<td>25</td>
<td>30</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>7</td>
<td>219</td>
<td>226</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>244</td>
<td>256</td>
</tr>
</tbody>
</table>

\( (X^2 = 7.597, 1 \text{ d.f., p < 0.01}) \)

<table>
<thead>
<tr>
<th>Outgoingness</th>
<th>Below Average</th>
<th>Average</th>
<th>Above Average</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No aneurysm</td>
<td>9</td>
<td>13</td>
<td>8</td>
<td>30</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>55</td>
<td>96</td>
<td>75</td>
<td>226</td>
</tr>
<tr>
<td></td>
<td>64</td>
<td>109</td>
<td>83</td>
<td>256</td>
</tr>
</tbody>
</table>

(Almost identical)
SECOND STUDY

The second study of 30 women with M.C. aneurysms, as described in Chapter IV, was carried out because such patients had been found in the first study to fare badly with surgery; and as some surgical details had been changed in the interim, we wished to check on the recent surgical morbidity. This smaller study was carried out in exactly the same way as the first, except that, because of the findings just described, each patient and other informant were carefully asked about her activities, experiences and state of mind just before the stroke.

RESULTS

Only one dramatic precipitant was described. A 44-year-old woman, described as a worrying person, houseproud, tense and troubled by religious preoccupations, who had returned one day from shopping to find that her difficult 18-year-old step-son was dismantling his bicycle on the carpet. She began to nag him, a row developed and he stepped forward and threatened to strike her. At the height of this fracas, she had her subarachnoid haemorrhage.

One other was on her way to see her sick daughter in hospital, which she had often done, and was standing on the railway platform, somewhat concerned but in no way distressed. One was lighting the fire but remembers that she was worried about a quarrel she had had with her husband the previous night—but again, as she said, "it was nothing unusual". Otherwise there was no mention of any emotional stress. Some could
remember nothing but one was during coitus (a well recognised precipitant); 11 were doing household tasks or getting dressed; one was laughing while enjoying herself at a party; and others were doing various other things. Thus only one of these 30 M.C. patients described any convincing emotional precipitant; unlike the 30 no aneurysm patients, of whom no specific questions were asked but of whom 4 described dramatic and 2 described non-dramatic connections spontaneously.

In the two studies, there were, therefore, a total of 5 very convincing and 2 fairly convincing accounts of an emotionally precipitated subarachnoid haemorrhage, in each case supported by a relative. Only two of these were recorded in the neurosurgical admission notes.

THIRD STUDY

This was done by studying the neurosurgical folders of 102 patients with and 101 without aneurysms, who had had a proven subarachnoid haemorrhage and had been followed up by the neurosurgical unit (see below). They were all taken from 1957–1958, in order to exclude M.A.O.I. reactions; and were selected in alphabetical order from the files. Most patients were admitted as emergencies and a relative or friend was available to give some history. Naturally, in a fair number of cases, the patient has been found collapsed or unconscious and no details of the circumstances were known.

All patients with subarachnoid haemorrhage admitted to Atkinson Morley's Hospital are followed up with great care, because
of the special research interest taken in this illness. After personal outpatient follow up has stopped, letters are still sent each year to the patient asking for information; and if no reply is obtained, letters are sent to relatives, general practitioners and, if necessary, to local executive councils to check on change of address, etc. Thus, over the years, a considerable amount of information is available.

Table 39 shows that the groups were well matched for age and sex; and also some findings relevant to personality and circumstances.
TABLE 39.

**DIFFERENCES IN ADMISSION NOTES OF 102 ANEURYSM AND 101 NO ANEURYSM CASES**

<table>
<thead>
<tr>
<th></th>
<th>102 Aneurysm Cases</th>
<th>101 No Aneurysm Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F</td>
<td>56</td>
<td>57</td>
</tr>
<tr>
<td>M</td>
<td>46</td>
<td>44</td>
</tr>
<tr>
<td><strong>Age (Years)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;30</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>30-44</td>
<td>22</td>
<td>20</td>
</tr>
<tr>
<td>45-59</td>
<td>53</td>
<td>50</td>
</tr>
<tr>
<td>60+</td>
<td>21</td>
<td>25</td>
</tr>
<tr>
<td><strong>Marital State</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>11</td>
<td>22 Single ( Pearson  =  4.539,  p &lt; 0.05 )</td>
</tr>
<tr>
<td>Sep.</td>
<td>2</td>
<td>2 Sep.</td>
</tr>
<tr>
<td>Div.</td>
<td>1</td>
<td>3 Div.</td>
</tr>
<tr>
<td><strong>Physical activity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>described explicitly, e.g., tennis, polishing table, bending. Excludes &quot;at work&quot;, &quot;at home&quot;, etc.</td>
<td>25</td>
<td>14 ( Pearson  =  7.702,  0.05 &lt; p &lt; 0.1 )</td>
</tr>
<tr>
<td><strong>Clear statement of abnormal emotional state</strong></td>
<td>1 occurred during E.C.T.</td>
<td>2 (said to have agitated depression)</td>
</tr>
<tr>
<td><strong>Latter from patient or G.P. refers to treatment of psychiatric symptoms</strong></td>
<td>54/67 survivors</td>
<td>16/92 ( Pearson  =  3.25 survivors 0.05 &lt; p &lt; 0.1 )</td>
</tr>
</tbody>
</table>
Results

1. Significantly more of the no aneurysm cases were single.

2. In almost significantly more of the aneurysm cases, a physical activity was explicitly described as going on at the time of the stroke.

3. In almost significantly more of the no aneurysm cases, psychiatric treatment was mentioned by the G.P. or by the patient in the follow up letters they had sent to the hospital, (mostly treatment by the G.P. in the form of tranquillisers or antidepressant drugs). This is more striking when it is remembered that the aneurysm cases have much more brain damage and consequent physical and organic mental disability, as described in earlier chapters.

Clear statements of an abnormal emotional state were uncommon but two of the three (both with agitated depression) were no aneurysm cases. The occurrence of subarachnoid haemorrhage during electroconvulsive treatment must be very rare and the notes did not make it clear at exactly what stage of the treatment it occurred, nor how much the convulsion had been modified.

Discussion and Conclusions

The role of emotional stress and individual psychopathology in precipitating subarachnoid haemorrhage and other strokes was discussed by Ecker (1954) and by Seidenberg and Ecker (1954), who thought that emotional turmoil could cause cerebral arterial spasm and, in turn, lead to the stroke itself, although unfortunately some of their case histories were unconvincing.
They also attempted to relate cerebral arterial spasm, demonstrated by angiography, to personality structure and emotional stress; but could not control for such variables as the effect of blood in the subarachnoid space, accidental variations in the timing of radiographs nor the incidence of similar psychopathology without a stroke. We are left, from their studies, with the fact that strokes can certainly be precipitated by overpowering emotional stress, as almost every experienced physician would agree.

The present study again demonstrates that subarachnoid haemorrhage can be precipitated by powerful emotional turmoil, which, however, seemed important only in women, unlike the cases described by Goldenberg and Ecker.

There is also circumstantial evidence here that emotional and personality factors were more important in patients with normal angiograms than in those with aneurysms. The no aneurysm cases in the personally followed up series more frequently had a history of emotional disorder and more often described a dramatic precipitant of the haemorrhage.

In the case record study, the patients without aneurysms more often received psychiatric treatment afterwards, although they suffered less from neurological disability and brain damage; and they were less often described as engaged in some physical activity at onset. They were also significantly more often unmarried. It is well known that unmarried people have more major mental illness, as demonstrated repeatedly by first admission rates to mental hospitals (Brooke, 1959), and similar findings have come from non-hospital populations, (Shepherd et al
1966). It is therefore unlikely that the hospital admission rates are solely because the unmarried have no-one to look after them at home but also reflect personality characteristics and environmental stress. Søegaard (1946), in particular, has taken the view that potential psychiatric patients often show personality traits which lessen the chances of marriage.

Finally, in the personally followed up series, the assessment of previous personality suggested that the no aneurysm cases were significantly less energetic and non significantly more anxiety prone than the aneurysm cases, although the numbers were rather small for convincing analysis.

Subarachnoid haemorrhage occurring in people with normal angiograms has a number of distinctive features. It rarely recurs, is not usually associated with progressive cerebral arteriopathy, the prognosis is good and it seems from this study that emotional disturbance is an important causal factor. A study is now under way in which, as patients with subarachnoid haemorrhage are admitted, their relatives are interviewed before angiography are known and genuinely unbiased personality assessment made.

**SUMMARY**

The role of emotional factors in the precipitation of subarachnoid haemorrhage has been studied from a personally conducted follow up of 291 patients and from case note study of 203 patients. It is clear that emotional turmoil may lead directly to subarachnoid haemorrhage on occasions and it is possible that
Emotional factors are more important in patients with normal angiograms than in those in whom angiography demonstrates a berry aneurysm.
CHAPTER IX

SUBARACHNOID HEMORRHAGE & MIGRAINE

Definitions used are given in Chapter III.

1. Incidence of Migraine.

Several authors have considered that migraine can be caused by cerebrovascular malformations, both angiomas and aneurysms, especially if posteriorly placed, (see Ozer et al., (1964)). Lees (1962), however, found no solid relationship between angiomas and migraine and Bull (1966) states that the incidence of migraine in patients with proven cerebrovascular malformations is the same as that in the general population - which has been variously estimated, often at around 10% (Wolff, 1963).

In this series, most patients were asked about headache of various types before and after the haemorrhage; and after a while all spouses were also questioned, in order to have a control series matched for social class, age and sex. The results are set out in Table 40.
### TABLE 40.

**PAST HISTORY OF MIGRAINE AND OTHER HEADACHES**

<table>
<thead>
<tr>
<th>1. Incidence of Migraine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients 43:254 (17%)</td>
</tr>
<tr>
<td>Men - 11:106 (10%)</td>
</tr>
<tr>
<td>Women 32:114 (26%)</td>
</tr>
<tr>
<td>Spouses 19:153 (12%)</td>
</tr>
<tr>
<td>Men - 4:93 (5%)</td>
</tr>
<tr>
<td>Women 15:88 (17%)</td>
</tr>
</tbody>
</table>

(No significant difference)

<table>
<thead>
<tr>
<th>2. Migraine by Aneurysm Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>P.C. 19:71 (27%)</td>
</tr>
<tr>
<td>M.C. 7:68 (10%)</td>
</tr>
<tr>
<td>A.C. 8:77 (10%)</td>
</tr>
<tr>
<td>No an. 6:30 (20%)</td>
</tr>
</tbody>
</table>

P.C. versus M.C./A.C. 

\[ \chi^2 = 9.615, 1 \text{ d.f.}, \]

\[ p < 0.01. \]
Migraine was non-significantly more common in the
patients than in the spouses and in women than in men, as
occurs in the general population. Only the P.C. aneurysm
group stands out with an incidence of 27%, compared to the
other patients and the spouses who were very close in this
respect. This finding, therefore, supports the older clinical
view that cerebrovascular malformations can lead to migraine,
especially if posteriorly placed.

2. Relief of Migraine and Other Headache.

I expected, before beginning the study, that headache
would be a common symptom. In fact, it was soon obvious that
it was as common for patients to describe relief of headache,
mainly migrainous, which may have troubled them severely for
many years. Most headache which developed in the follow up
period was of typical tension type, found in association with
anxiety and depression, and not considered in more detail here.
Also, 5 patients developed typical migraine for the first time
after their haemorrhage.

Altogether, of the 43 patients with a past history
of migraine, 24 experienced complete and lasting relief; 11
experienced partial relief; and 8 no relief.

Of 30 patients with "other headache" (as mentioned in
Chapter III, most of these patients could have been described
as having "vascular" headache), 18 experienced complete and 9
partial relief.

Those with migraine who obtained partial relief were
interesting, in that 8 of the 11 lost their headache but continued to have typical fortification spectra, paraesthesiae, and sometimes nausea and vomiting.

Relief of migraine was not more common in those with I.C. than with other aneurysms or no aneurysms; nor was it much more common with operation than without, although one might have expected common carotid ligation to be most effective in this way.

Altogether, these findings also provide firm evidence for a causal connection between the cerebrovascular abnormality and migraine in this series.

3. Previous Personality and Migraine

There is a widespread impression, which the author shared, that migraine is particularly common in tense, driving and perfectionistic people; that it is more common just after a period of intense effort and concentration; or that it is related in some other way - often specific to the patient - to emotional stress, (Wolff, 1963). Certainly these aspects are commonly present in patients seen in psychiatric practice who have migraine.

However, the personality ratings used in this series do not show any differences between those with and without migraine, as set out in Table 41.
<table>
<thead>
<tr>
<th></th>
<th>Below Average</th>
<th>Average</th>
<th>Above Average</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety proneness</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Migraine</td>
<td>3</td>
<td>14</td>
<td>23</td>
<td>45</td>
</tr>
<tr>
<td>No migraine</td>
<td>46 (44.5)</td>
<td>66 (82.4)</td>
<td>79 (84.1)</td>
<td>211</td>
</tr>
<tr>
<td></td>
<td>54</td>
<td>100</td>
<td>102</td>
<td>256</td>
</tr>
<tr>
<td>Obsessionalism</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Migraine</td>
<td>2</td>
<td>16</td>
<td>26</td>
<td>44</td>
</tr>
<tr>
<td>No migraine</td>
<td>18 (16.5)</td>
<td>96 (92.3)</td>
<td>98 (102.2)</td>
<td>212</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>112</td>
<td>124</td>
<td>256</td>
</tr>
<tr>
<td>Energy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Migraine</td>
<td>2</td>
<td>25</td>
<td>17</td>
<td>44</td>
</tr>
<tr>
<td>No migraine</td>
<td>10 (9.9)</td>
<td>112 (112.9)</td>
<td>90 (88.2)</td>
<td>212</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>137</td>
<td>107</td>
<td>256</td>
</tr>
<tr>
<td>Outgoingness</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Migraine</td>
<td>12</td>
<td>19</td>
<td>13</td>
<td>44</td>
</tr>
<tr>
<td>No migraine</td>
<td>52 (52.7)</td>
<td>90 (89.8)</td>
<td>70 (68.4)</td>
<td>212</td>
</tr>
<tr>
<td></td>
<td>64</td>
<td>109</td>
<td>83</td>
<td>256</td>
</tr>
</tbody>
</table>

Figures in brackets are "expected" figures, if distribution had been random. All differences are trivial.
It may be that these ratings do not measure the relevant aspects of personality, although three of them have some apparent relationship to those features described by Wolff. Another possible explanation, more likely in my opinion, is that in many of these patients the migraine was specifically related to the cerebrovascular anomaly; but that the ratings might detect differences if used on a different population of migraine sufferers. In any case, the author's bias towards the relevant personality factors has not distorted the ratings, which encourages the belief that, when they do show significant differences (as in the depressed patients described in Chapter VII), those differences are meaningful.
In this section, only a few comments on the study as a whole will be made before proceeding to the summary, as the results and the relevant literature have already been discussed chapter by chapter.

The study has had at least two major disadvantages: firstly, the lack of operational definitions of some important scales; and, secondly, the lack of independent ratings by another observer, which could have helped to outweigh that drawback. These have been partly overcome by the use of two well known psychological tests in considering intellectual impairment; and the method of verbatim personality description seems to be free from serious subjective bias. As was mentioned in the text, there seemed good reason to assess affective changes subjectively rather than to go solely by treatment offered and the latter did provide support for the ratings of depression in most cases. Nevertheless it is probable that halo effects have occurred, especially in the clinical assessments of personality and intellectual change. However, the fact that there were several unexpected results - for instance, the lack of right/left effects in the rating of intellectual impairment and the lack of personality differences between those with and without migraine - suggests that the possibility of subjective bias need not invalidate the findings.
The advantages of the study include the large size of the population studied - 261 cases in the main series, large enough to make statistical comparisons feasible - although the size also meant that each patient was seen only once and, therefore, could not be studied in depth. The series was subjected to no special selection and, in most cases, the author did not know the exact site of an aneurysm or indeed whether there was an aneurysm present or not. The follow up in the patient's home, the relatively standardised interview and physical examination and the fact that another informant was seen in so many cases were all of benefit. It is hoped that these help to counterbalance the disadvantages outlined above and any others which may have arisen from the author's own clinical shortcomings.
The thesis deals mainly with the psychological sequelae of subarachnoid haemorrhage, including a comparison of the results of operative and conservative treatment for ruptured cerebral berry aneurysm. It also considers the role of emotion in precipitating subarachnoid haemorrhage, and deals with some aspects of migraine in relation to personality and berry aneurysms.

The results are presented in nine chapters, the last six of which contain the results.

Chapter I

The literature on subarachnoid haemorrhage is reviewed, dealing briefly with the historical and then with the neurosurgical aspects before considering the small psychiatric literature on the subject.

Chapter II

Some details of the neuropathology of subarachnoid haemorrhage are given, indicating the areas of brain principally affected by the rupture of cerebral berry aneurysms in different sites.

Chapter III

The case material and methods used are described. Most of the patients had been in the comparative trial of surgery versus conservative treatment.

Two series of patients, the first of 261 and the second
of 30 were seen. The first was made up of 81 with anterior communicating artery (A.C.) aneurysms; 71 with middle cerebral artery (M.C.) aneurysms; 72 with posterior communicating artery (P.C.) aneurysms; 7 with multiple aneurysms (mult. an.) and 30 with normal angiograms (no an.). There were 149 women and 112 men, in age from 19-67 years. The second series consisted of 30 women with middle cerebral aneurysms, in age from 20-62 years. Follow up was from six months to six years after the haemorrhage.

All but one patient was seen personally and in 272 another informant was seen. In all but 10 cases the patients were seen in their own homes. No special selection was used. The author was unaware of the exact diagnosis in most cases.

Details of the method of examination are given. It included the Benton Visual Retention Test (Benton), a paired associate word learning test (P.A.T.); and each patient was examined neurologically and blood pressure measured.

Each patient was rated for physical disability, C.N.S. signs, personality impairment, intellectual impairment, psychiatric symptoms, overall mental disability and employment status. Pre-morbid personality was assessed on four factors; and a qualitative assessment of personality change also made.

Chapter IV

The morbidity was high: 45% had no overall mental disability, 24% had mild, 16% moderate, 10% severe and 3% very severe mental disability.

Morbidity was mainly associated with C.N.S. signs and
patients with no aneurysm were least affected. Epilepsy occurred in 10% of those with single aneurysms.

Patients with H.C. aneurysm did significantly worse with operative than with conservative treatment, especially women with left sided aneurysms. The second series of 30 women with H.C. aneurysm showed that a change in the operative management had been followed by a lesser morbidity, which did not exceed that of conservative treatment.

Chapter V

Personality impairment was absent in 5%, mild in 22%, moderate in 15%, and severe in 4%. It was mainly proportional to C.N.S. signs and no aneurysm cases were least affected. It was associated with intellectual impairment in most cases.

In addition
(a) a qualitatively different frontal lobe syndrome occurred and 13 patients had "improved" personalities—presumably a leucotomy effect,
(b) patients with deep hemispheric infarction from P.C. aneurysms had relatively less impairment than M.C. cases with more superficial damage,
(c) a "core" syndrome is described of lowered vitality, increased irritability, increased anxiety proneness, intellectual impairment and C.N.S. signs,
(d) frontal lobe damage (from A.C. aneurysms) more often led to personality change without intellectual impairment.
(a) personality impairment was relatively commoner in men,
(b) previous personality showed up as relatively unimportant, probably because of the reliance on the accounts of other informants.

Chapter VI

Clinical intellectual impairment was absent in 59%, mild in 30%, moderate in 8% and severe in 3%. There was very considerable overlap with personality impairment.

In addition to a general overall relationship to CNS signs similar to that found for personality impairment, both clinical intellectual impairment and the psychological test results showed some special relationships to site of brain damage and other factors.

(a) A.C., aneurysm cases more often had intellectual impairment without CNS signs; and had better scores on the Benton Test.
(b) P.C., aneurysm cases had worse scores on the Benton Test.
(c) Women did worse on the Benton Test.
(d) Below average premorbid personality was associated with poor scores on the P.A.T.
(e) Clinical intellectual impairment was more closely related to the P.A.T. than to the Benton Test.
(f) As with personality impairment, deep hemispheric infarction led to less intellectual impairment than did more superficial damage.
(g) The most surprising finding was the relative unimportance of the side of brain damage.

Chapter VII

Depression was absent in 74%, mild in 11%, moderate in 12% and severe in 3%. It was commoner in those with CBS signs and even more so in those with brain damage, as defined by a combination of CBS signs and personality and intellectual impairment.

In addition:
(a) no simple overall relationship to the degree of brain damage is demonstrated,
(b) mild depression in the brain damaged is an inextricable part of the personality change usually, whereas in the non-brain damaged it is mostly similar to depression experienced before the stroke,
(c) the previous personality of the brain damaged depressives was more energetic than that of those not depressed,
(d) the previous personality of the non-brain damaged depressives was more anxious, less energetic and less outgoing than those not depressed,
(e) in the brain damaged, depression and anxiety were equally common in men and women; in the non-brain damaged, affective symptoms were much commoner in women.
Chapter VIII

Several patients attributed their strokes to severe emotional turmoil and five of the accounts were very convincing, others less so. There is some evidence that emotionally precipitated haemorrhages are commoner in patients with no aneurysms, who also have a higher incidence of previous affective disturbance and more post haemorrhage psychiatric symptoms than would be expected, considering how little organic brain damage they suffer. (This point also appears in other chapters).

Chapter IX

No differences in personality are shown between those with and without a past history of migraine.

Migraine is commoner in those with F.C. aneurysms.

Relief of migrainous headache is common after subarachnoid haemorrhage.
REFERENCES


Ruptured Intracranial Aneurysm: Community Study.
Lancet 2 1254-1257.

Crompton, H.R. (1962) (a) The Pathology of Ruptured Middle Cerebral Aneurysms; with Special Reference to the Differences between the Sexes.
Lancet 2 421-425.

Crompton, H.R. (1962) (b) Intracerebral Haematoma Complicating Ruptured Cerebral Berry Aneurysm.

Crompton, H.R. (1963) (a) Central Nervous System Lesions consequent on Ruptured Cerebral Berry Aneurysm.
M.D. Theses, London.

Crompton, H.R. (1963) (b) Hypothalamic Lesion following Rupture of Cerebral Berry Aneurysm.
Brain. 86 301-314.


Das Weitere Schicksal von 1,000 zerebralen Insulten.

Nervenzt. 33 203-208.


Goldstein, K. (1952) The Effect of Brain Damage on the Personality.
Psychiatry 15 245.

Hornby (1952) Intracranial Aneurysms. Thomas Springfield.


I am grateful to the many people who made this study and thesis possible. The patients examined were under the care of Mr. Wylie McKissock, who originally suggested the work, Mr. L. S. Walsh and Mr. A. E. Richardson; and all gave every help and much valuable advice. Professor Desmond Curran and later Professor Arthur Crisp gave me every encouragement and provided me with all necessary facilities. I have had essential statistical advice from Mr. P. M. Payne and Professor H. Gwynne Jones. Dr. Michael Rutter read and criticised some earlier drafts and Dr. Peter Hays gave helpful advice.

Financial support for computer analysis has been provided by St. George's Hospital and its Medical School and I was supported by N.I.H. Bethesda, Maryland, U.S.A. for the first two years of data collection (Grant No. NB 03136-06).

To all these and to many others, including the patients and their relatives, I would like to express my thanks.
LUMBAR AIR ENCEPHALOGRAPHY IN CHRONIC SCHIZOPHRENIA:
A CONTROLLED EXPERIMENT

BY
P. B. STOREY

Reprinted from
THE BRITISH JOURNAL OF PSYCHIATRY
Vol. 112, No. 483, February 1966
Lumbar Air Encephalography in Chronic Schizophrenia: 
A Controlled Experiment

By P. B. Storey*

INTRODUCTION

The lumbar air encephalogram (L.A.E.G.) was introduced by Dandy in 1919, and in 1929 the first report of its use in schizophrenia was published by Jacobi and Winkler. Since then numerous papers have appeared, most of which have claimed to demonstrate cerebral atrophy in chronic schizophrenic patients. All these studies have suffered from the fact that no adequate series of normal controls has yet been collected. Most of the authors concerned made no attempt to use controls, nor did they consider the possibility of observer error or of the bias which may exist when the reporting radiologist knows the diagnosis and is looking for abnormalities.

Notwithstanding these and other objections, an impressive amount of evidence has been collected. In particular Huber (1957) produced the most convincing support for the theory, and this study was planned as a direct result of his monograph. In it care has been taken to include a control series, to exclude bias, and to assess observer error.

REVIEW OF THE LITERATURE

Thirty-two papers have been found which have appeared on this subject, and twenty-nine have supported Jacobi and Winkler in their original contention: that chronic schizophrenics show cerebral atrophy. Only the most important will be mentioned here.

Lemke (1936) studied 100 cases, rating ventricular size on a five-point scale; and using, as controls, patients with epilepsy, migraine, hysteria and psychopathy. Of the schizophrenics 85 per cent. showed cortical atrophy, and 50 per cent. dilated ventricles; of the controls only 20 per cent. were similarly affected. The most severe changes were in those with the most severe defect. As some of his cases had been studied by Jacobi and Winkler eight years before and showed no increase in the degree of atrophy, he concluded that in some patients there was an inborn cerebral anomaly which adversely affected the schizophrenic process.

Huber (1957), in a detailed monograph, describes his findings in 190 schizophrenics. They were an unselected series of cases who had had little or no somatic treatment, and who showed no evidence of brain damage. He rated them for defect on a scale based essentially on social competence, and found that the degree of atrophy was proportional to the severity of defect. He emphasized in particular the importance of broadening of the third ventricle; and also found that the different clinical types of schizophrenia showed varying degrees of atrophy. Groups of patients were effectively matched for age, and the author gave the necessary technical details and measurements. By demonstrating differences within the series he partly overcame the lack of outside controls, but the absence of “blind” assessment of the films and of any statistical evaluation are drawbacks of this work.

Recently there have been two negative studies, both as a result of Huber’s work. Peltonen (1962) compared the films of 644 patients from a Finnish mental hospital, of whom eighty-six were schizophrenic. The films were examined in ignorance of the diagnosis, and only the third ventricle was measured. It was shown conclusively that the schizophrenics did not differ from the other patients—depressive, epileptic, neurotic and psychopathic.

* Based on a dissertation submitted in part fulfilment of the requirements of the Academic Postgraduate Diploma in Psychological Medicine of the University of London, 1963.
Selection of Patients

fractional techniques are less distressing than the older studies have been published which fail to support air in most of his cases. It seems unlikely as he only used 30-40 ml. of air in only ten. They suggest that his results stem from technical factors, such as injecting too much air and thus inflating the ventricles. Huber did, in fact, consider this possibility, but it seems unlikely as he only used 30-40 ml. of air in most of his cases.

The Encephalogram after E.C.T.

It has been considered unlikely that E.C.T. can cause cerebral atrophy. The subject is reviewed in the handbook by Bellak (Bellak, 1958). In addition a number of histopathological studies have been published which fail to support the idea: Rowland and Mettler (1949); Peyton et al. (1949); and Corsellis and Meyer (1954). Many of the papers describing cerebral atrophy in schizophrenics were, of course, written before the introduction of E.C.T.

The Present Study

Ethical considerations

Encephalography is considered safe in the absence of raised intracranial pressure, but it remains a painful procedure in many cases, even though the modern fractional techniques are less distressing than the older methods. It was thought to be justified to submit patients to this if both they and their relatives knew that it was only an investigation, and gave permission independently. The patients were all referred by the consultant in charge as being both indubitably schizophrenic and likely to understand the proposal. Letters were then sent to the next of kin seeking approval, and if this was forthcoming the patient was interviewed. It was explained in simple terms as e.g., “trying to find out more about the cause of your illness”; the lumbar puncture was described (many patients knew what these were already), and they were warned to expect headache and several days in bed. No attempt was made to persuade unwilling relatives or patients. Out of fifty-four relatives approached, forty-nine patients knew what these were already, and they were instructed in the procedure. So far as was known these patients had not been shown later to have any serious intracranial disease. The provisional diagnosis of these patients for which they had had the encephalograms included “epilepsy”, “recent transient giddiness” and “severe migraine”. The provisional diagnosis did not affect selection as a control. They are approximately matched for age with the patients (see Table III).

Radiological technique was the same as for the experimental subjects.

Radiological Technique

After premedication and local anaesthesia the lumbar route was used. 10 ml. of C.S.F. and 40 ml. of air were the approximate maxima exchanged, using a fractional technique. Films were taken of the standard Lysholm positions at a focus-film distance of 36 inches (90 cm.). Potter-Bucky and iris diaphragms were used.

Selection of Controls

These were taken from the records of the radiological department. They were the first eighteen found, from patients under the age of 45 years, which had been reported as “normal” in the course of the routine work of the department. So far as been known these patients had not been shown later to have any serious intracranial disease. The provisional diagnosis of these patients for which they had had the encephalograms included “epilepsy”, “recent transient giddiness” and “severe migraine”. The provisional diagnosis did not affect selection as a control. They are approximately matched for age with the patients (see Table III).

Selection of Patients

The patients had to meet the following criteria:

1. they should be considered to have chronic schizophrenia in the opinion of both the referring consultant and the author, and to have spent at least two years in hospital. (Illustrative cases are included in Table VIII.)

2. they should be not more than 45 years old, to minimize the age effects.

3. they should have no clinical evidence of brain damage; and have had no leucotomy.

By any normal standards these patients were severely affected, although the most severe cases had to be excluded on ethical grounds, but attempts were made to distinguish varying grades of severity. They were rated on two scales devised by Wing (1960 and 1961), in one of which the Sister or Charge Nurse rates patients on behaviour on the ward which indicates “socially embarrassing” and “socially withdrawn” behaviour; in the other the psychiatrist rates them on four symptoms: flatness and incongruity of affect; poverty of speech; incoherence of speech; and the expression of coherent delusions. The diagnostic categories of paranoid, hebephrenic, and catatonic were also used.

LUMBAR AIR ENCEPHALOGRAPHY IN CHRONIC SCHIZOPHRENIA

Ansink et al. (1963) examined the films of forty-three chronic schizophrenics using the same criteria as did Huber, and found anomalies in only ten. They suggest that his results stem from technical factors, such as injecting too much air and thus inflating the ventricles. Huber did, in fact, consider this possibility, but it seems unlikely as he only used 30-40 ml. of air in most of his cases.
ment, is another problem. Features such as rounding of angles, or pooling of air over the cortex, are of great importance in the skilled examination needed; but it is in these that observer error and bias are most likely to appear.

It was therefore decided to have two independent observers, and to use a blind technique. Only one subjective judgment was made—that of normality/abnormality—and three measurements. These were: the maximum breadth of the third ventricle in the antero-posterior view; and the breadth of the bodies of the lateral ventricles. They were measured using a transparent rule on an illuminated screen.

The films of patients and controls were mixed, and then examined separately by two radiologists, A and B, who did not know which films were which. After correlation of these observers’ readings, all the films were re-examined by A. He was told which measurements were in dispute, and his final reading was taken as the “agreed score” for the comparison of patients and controls.

It was apparent to the author, who watched both radiologists, that in cases where the difference was more than 2 mm. they were measuring from slightly different parts of the ventricles, and that errors of less than 2 mm. were inherent in the technique used.

RESULTS

These are in three main groups: the correlation of the observations of the two radiologists; the comparison of patients and controls; and differences between patients.

1. Observer Correlation

For this the total of 36 A.E.G. is used (Table I).

(a) Third Ventricle Breadth

Mean breadth measured by A—6·47 mm. (S.D. 1·96). Mean breadth measured by B—5·97 mm. (S.D. 1·76). The product moment correlation coefficient = 0·6. In 27 out of the total 36 there was agreement within 1 mm., and in 32:36 agreement within 2 mm.

It is clear that inter-observer agreement in these measurements is very good.

(b) Lateral Ventricle Breadth

To avoid “averaging out” any abnormality, only the larger of the two ventricles is considered here. Mean breadth measured by A—18·74 mm. (S.D. 2·81). Mean breadth measured by B—17·32 mm. (S.D. 3·01). The product moment correlation = 0·74. In 23:36 there was agreement within 1 mm., and in 29:36 agreement within 2 mm.

The use of $\chi^2$, with Yates’ correction, shows that the difference between the two observers is significant at the 5 per cent. level. ($\chi^2 = 4·346$, with one degree of freedom.) The difference found by B between the patients and controls does not reach significance.

2. Comparison of Patients and Controls

For this the “agreed score” is used, as described above (Table I).

(a) Third Ventricle

Mean breadth for patients—6·8 mm. (S.D. 1·92). Mean breadth for controls—5·9 mm. (S.D. 1·73). This difference is not significant. ($t = 0·37$.)

(b) Lateral Ventricle

Again only the larger of the two ventricles is considered here.

Mean breadth for patients—18·3 mm. (S.D. 2·8). Mean breadth for controls—18·6 mm. (S.D. 2·26). This difference is not significant. ($t = 0·34$.)

(c) Abnormalities as Described by Radiologist B

Of the four controls considered abnormal by B, one showed cortical atrophy and three
ventricular dilatation. Of the seven patients three were thought to show ventricular dilation and five cortical atrophy. One patient showed both abnormalities. Some details are given in Table II and it can be seen that no relationship exists between the presence of the abnormalities and the duration, severity, or clinical type of illness.

3. Differences within the Series of Patients

(a) Repeat Encephalogram

One patient had had an encephalogram performed three and a half years previously, at the start of her illness. These earlier films were obtained, but no differences could be demonstrated between the two sets.
### Table 1(b)

<table>
<thead>
<tr>
<th>Name</th>
<th>Radiologist A.</th>
<th>Radiologist B.</th>
<th>Agreed Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### (b) Age of Patient and Duration of Illness

The relationship between these factors and ventricular size is shown in Table IV. It is clear that neither factor is of importance.

### (c) Severity of Illness

Table V shows that using the two rating scales devised by Wing, which were described above, there is no increase in size of ventricles in the more disturbed patients. On the mental state rating in fact, the third ventricle mean breadth is greater in the less severely affected, at the 1 per cent. level of confidence.

### (d) Type of Schizophrenia

The mean third ventricular breadth in the paranoid patients was greater than in the hebephrenics at the 5 per cent. level of con-
TABLE II
Patients Considered Abnormal by Radiologist B.

<table>
<thead>
<tr>
<th>Name</th>
<th>Age</th>
<th>Reason</th>
<th>Measurements</th>
<th>Type of Schizophrenia</th>
<th>Duration</th>
<th>Mental State Rating</th>
<th>S.E.</th>
<th>S.W.</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.S.</td>
<td>27</td>
<td>Lateral ventricles sl. enlarged (generalized)</td>
<td>3rd V. 6 mm. Lat. V. 20 mm.</td>
<td>Hebephrenia</td>
<td>3 yrs.</td>
<td>Sub-group 2</td>
<td>-7</td>
<td>-4</td>
</tr>
<tr>
<td>P.P.</td>
<td>31</td>
<td>Lateral ventricles enlarged: sl. bilat. generalized cortical atrophy</td>
<td>3rd V. 5 mm. Lat. V. 19 mm.</td>
<td>Hebephrenia</td>
<td>14 yrs.</td>
<td>1 (b)</td>
<td>0</td>
<td>-10</td>
</tr>
<tr>
<td>E.K.</td>
<td>31</td>
<td>Sl. R. cortical frontal atrophy</td>
<td>3rd V. 6 mm. Lat. V. 20 mm.</td>
<td>Unclassified</td>
<td>5 yrs.</td>
<td>1 (b)</td>
<td>-3</td>
<td>-7</td>
</tr>
<tr>
<td>E.D.</td>
<td>45</td>
<td>Cortical atrophy, mod. generalized</td>
<td>3rd V. 9 mm. Lat. V. 20 mm.</td>
<td>Paranoid</td>
<td>12 yrs.</td>
<td>1 (a)</td>
<td>-15</td>
<td>-6</td>
</tr>
<tr>
<td>J.M.</td>
<td>35</td>
<td>Frontal atrophy with ventric. dilation. Sulci normal</td>
<td>3rd V. 10 mm. Lat. V. *</td>
<td>Paranoid</td>
<td>6 yrs.</td>
<td>2</td>
<td>-6</td>
<td>-14</td>
</tr>
<tr>
<td>B.D.</td>
<td>32</td>
<td>Sl. bilat. cortical frontal atrophy</td>
<td>3rd V. 5 mm. Lat. V. 15 mm.</td>
<td>Catatonic</td>
<td>6 yrs.</td>
<td>5</td>
<td>-2</td>
<td>-11</td>
</tr>
<tr>
<td>M.S.</td>
<td>30</td>
<td>Patchy post-frontal cortical atrophy</td>
<td></td>
<td>Hebephrenic</td>
<td>7 yrs.</td>
<td>2</td>
<td>-9</td>
<td>-11</td>
</tr>
</tbody>
</table>

Mean age of this group: 33 years. Mean duration of this group: 7-6 years.
Mean age of whole series: 31-2 years. Mean duration of whole series: 7-8 years.
6 out of 7 of this group fall into the "less severely disturbed" subgroup.
No relationship demonstrated between "abnormal" encephalogram and age, duration, or severity.

TABLE III
Age of Patients and Size of Ventricles

<table>
<thead>
<tr>
<th>Age</th>
<th>Number</th>
<th>Mean Third Ventricle</th>
<th>Mean Lateral Ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td>19-25</td>
<td>4</td>
<td>5·3 mm.</td>
<td>15·8 mm.</td>
</tr>
<tr>
<td>26-30</td>
<td>3</td>
<td>7·7 mm.</td>
<td>19·7 mm.</td>
</tr>
<tr>
<td>31-35</td>
<td>7</td>
<td>6·7 mm.</td>
<td>18·0 mm.</td>
</tr>
<tr>
<td>36-40</td>
<td>3</td>
<td>7·3 mm.</td>
<td>20·3 mm.</td>
</tr>
<tr>
<td>41-45</td>
<td>1</td>
<td>9·0 mm.</td>
<td>20·0 mm.</td>
</tr>
</tbody>
</table>

Age of Controls and Size of Ventricles

<table>
<thead>
<tr>
<th>Age</th>
<th>Number</th>
<th>Mean Third Ventricle</th>
<th>Mean Lateral Ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td>(17)-25</td>
<td>4</td>
<td>6·5 mm.</td>
<td>20·0 mm.</td>
</tr>
<tr>
<td>26-30</td>
<td>3</td>
<td>3·7 mm.</td>
<td>16·6 mm.</td>
</tr>
<tr>
<td>31-35</td>
<td>6</td>
<td>8·0 mm.</td>
<td>18·2 mm.</td>
</tr>
<tr>
<td>36-40</td>
<td>2</td>
<td>6·5 mm.</td>
<td>16·5 mm.</td>
</tr>
<tr>
<td>41-45</td>
<td>3</td>
<td>7·0 mm.</td>
<td>19·0 mm.</td>
</tr>
</tbody>
</table>

(f) E.C.T. and Insulin Coma Therapy

Almost all the patients had been in several different hospitals, and the total amounts of treatment they had had was not known accurately. However, a group of seven was formed who were known for certain to have had at least thirty E.C.T. or insulin comas. Most had had considerably more, as is shown in Table VI.

The mean values of the ventricular measurements of this group were very slightly less than those for the series as a whole. Third vent. 6·7 mm.; and lat. vent. 17·3 mm.; compared to 6·8 mm. and 18·3 mm. for the series.
TABLE IV

Severity Ratings on the Wing Scale (Mental State) — I. less severe  
subgroups 1(a), 1(b) and 2  
II. more severe  
subgroups 1(c), 3, 4 and 5  
Duration Grouping — 6— = 2–6 years  
— 6+ = more than 6 years

I. Less Severe

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Duration</th>
<th>3rd Vent.</th>
<th>Lat. Vent.</th>
</tr>
</thead>
<tbody>
<tr>
<td>E.D.</td>
<td>45</td>
<td>6+</td>
<td>9 mm.</td>
<td>20 mm.</td>
</tr>
<tr>
<td>G.M.</td>
<td>30</td>
<td>6—</td>
<td>7 mm.</td>
<td>18 mm.</td>
</tr>
<tr>
<td>H.C.</td>
<td>37</td>
<td>6+</td>
<td>9 mm.</td>
<td>20 mm.</td>
</tr>
<tr>
<td>E.K.</td>
<td>31</td>
<td>6—</td>
<td>6 mm.</td>
<td>20 mm.</td>
</tr>
<tr>
<td>J.M.</td>
<td>35</td>
<td>6—</td>
<td>10 mm.</td>
<td>*</td>
</tr>
<tr>
<td>P.P.</td>
<td>31</td>
<td>6+</td>
<td>5 mm.</td>
<td>19 mm.</td>
</tr>
<tr>
<td>A.W.</td>
<td>32</td>
<td>6+</td>
<td>8 mm.</td>
<td>20 mm.</td>
</tr>
<tr>
<td>G.H.</td>
<td>19</td>
<td>6—</td>
<td>5 mm.</td>
<td>17 mm.</td>
</tr>
<tr>
<td>G.D.</td>
<td>36</td>
<td>6+</td>
<td>6 mm.</td>
<td>20 mm.</td>
</tr>
<tr>
<td>R.S.</td>
<td>27</td>
<td>6—</td>
<td>6 mm.</td>
<td>21 mm.</td>
</tr>
<tr>
<td>M.S.</td>
<td>30</td>
<td>6+</td>
<td>10 mm.</td>
<td>20 mm.</td>
</tr>
<tr>
<td>F.E.</td>
<td>34</td>
<td>6—</td>
<td>6 mm.</td>
<td>17 mm.</td>
</tr>
</tbody>
</table>

Mean age: 32:2 years.  
Mean 3rd vent. breadth: 7:2 mm.  
Mean lat. vent. breadth: 19:3 mm.

II. More Severe

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Duration</th>
<th>3rd Vent.</th>
<th>Lat. Vent.</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.T.</td>
<td>23</td>
<td>6—</td>
<td>5 mm.</td>
<td>14 mm.</td>
</tr>
<tr>
<td>L.M.</td>
<td>40</td>
<td>6+</td>
<td>7 mm.</td>
<td>21 mm.</td>
</tr>
<tr>
<td>E.T.</td>
<td>23</td>
<td>6—</td>
<td>5 mm.</td>
<td>16 mm.</td>
</tr>
<tr>
<td>P.B.</td>
<td>35</td>
<td>6+</td>
<td>7 mm.</td>
<td>17 mm.</td>
</tr>
<tr>
<td>B.D.</td>
<td>32</td>
<td>6—</td>
<td>5 mm.</td>
<td>15 mm.</td>
</tr>
<tr>
<td>D.C.</td>
<td>22</td>
<td>6—</td>
<td>6 mm.</td>
<td>16 mm.</td>
</tr>
</tbody>
</table>

Mean age: 29:2 years.  
Mean 3rd vent. breadth: 5:8 mm.  
Mean lat. vent. breadth: 16:5 mm.

(*=no measurement possible)

It is obvious that, using this scale of severity, there is no increase in size of the ventricles in the more severely ill patients, and that in fact the mean values for Group I are greater than for Group II. The difference in the means for the third ventricles (but not the lateral ventricles) is significant at the 1 per cent. level of confidence (t=3.06 p=<0.01).

TABLE V

Rating on the Socially Embarrassing Behaviour (S.E.) and Social Withdrawal (S.W.) Scales

Those five patients who show the most extreme scores on the two scales have been selected.

<table>
<thead>
<tr>
<th>Name</th>
<th>Score</th>
<th>3rd Vent.</th>
<th>Lat. Vent.</th>
<th>Name</th>
<th>Score</th>
<th>3rd Vent.</th>
<th>Lat. Vent.</th>
</tr>
</thead>
<tbody>
<tr>
<td>E.D.</td>
<td>-15</td>
<td>9 mm.</td>
<td>20 mm.</td>
<td>J.T.</td>
<td>-14</td>
<td>5 mm.</td>
<td>14 mm.</td>
</tr>
<tr>
<td>G.M.</td>
<td>-16</td>
<td>7 mm.</td>
<td>18 mm.</td>
<td>J.M.</td>
<td>-14</td>
<td>10 mm.</td>
<td>*</td>
</tr>
<tr>
<td>L.M.</td>
<td>-20</td>
<td>7 mm.</td>
<td>21 mm.</td>
<td>G.H.</td>
<td>-16</td>
<td>5 mm.</td>
<td>17 mm.</td>
</tr>
<tr>
<td>E.T.</td>
<td>-13</td>
<td>5 mm.</td>
<td>16 mm.</td>
<td>A.W.</td>
<td>-11</td>
<td>8 mm.</td>
<td>20 mm.</td>
</tr>
<tr>
<td>D.C.</td>
<td>-18</td>
<td>6 mm.</td>
<td>16 mm.</td>
<td>B.D.</td>
<td>-11</td>
<td>5 mm.</td>
<td>15 mm.</td>
</tr>
</tbody>
</table>

Mean 6:8 mm. 18:2 mm.  
Mean 6:6 mm. 16:5 mm.

* Measurement impossible.  

The means of the whole series of patients are 6:8 mm. (S.D. 1:73) for the 3rd ventricle and 18:3 mm. (S.D. 2:20) for the lateral ventricle. It is apparent that these scores do not deviate significantly from each other or from the whole series.
TABLE VI

E.C.T., Insulin Coma Therapy, and the Encephalogram

"Heavily Treated Patients"
The following list is of patients who are known to have had at least the stated number of treatments.

<table>
<thead>
<tr>
<th>Name</th>
<th>E.C.T.</th>
<th>Insulin</th>
<th>Third Ventricle</th>
<th>Lateral Ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td>E.D.</td>
<td>30</td>
<td>113</td>
<td>9 mm.</td>
<td>20 mm.</td>
</tr>
<tr>
<td>G.M.</td>
<td>34</td>
<td>30</td>
<td>7 mm.</td>
<td>18 mm.</td>
</tr>
<tr>
<td>J.T.</td>
<td>35</td>
<td>38</td>
<td>5 mm.</td>
<td>14 mm.</td>
</tr>
<tr>
<td>P.P.</td>
<td>4</td>
<td>77</td>
<td>5 mm.</td>
<td>19 mm.</td>
</tr>
<tr>
<td>B.D.</td>
<td>45</td>
<td>30</td>
<td>5 mm.</td>
<td>15 mm.</td>
</tr>
<tr>
<td>M.S.</td>
<td>7</td>
<td>35</td>
<td>10 mm.</td>
<td>20 mm.</td>
</tr>
<tr>
<td>D.G.</td>
<td>1</td>
<td>55</td>
<td>6 mm.</td>
<td>16 mm.</td>
</tr>
</tbody>
</table>

Means 6-7 mm. 17-3 mm.

Mean for series 6-8 mm. 18-3 mm.
(S.D.1.73) (S.D.2.20)

There is no indication from these admittedly imperfect data that somatic treatments are associated with ventricular dilation.

DISCUSSION

Unfortunately a review of the literature on the subject of air encephalography and cerebral atrophy in chronic schizophrenia does not lead to any firm conclusions about its value. Most of the earlier work published has had such obvious drawbacks that it cannot carry much weight, and the more careful studies of Huber (1959) on the one hand, and Peltonen (1963) and Ansink et al. on the other, are contradictory.

The present study was planned in the hope that some aspects could be clarified; and in the sections dealing with agreement and disagreement between observers a certain amount has been achieved. The agreement between the two radiologists is high when they are measuring, but falls when they are asked to make subjective assessments of abnormality. It is clear that the results obtained by B—seven out of eighteen showing cerebral atrophy—are in keeping with many of the earlier papers published. It is not possible to say which of these observers is more nearly right, and the lack of a good control series is likely to remain a stumbling block for all such studies. The controls used here were derived from patients submitted to encephalography for diagnostic reasons, and it is possible that they would have larger ventricles than would a randomly selected series. The converse, however, could be true: that the radiologist in his search for abnormalities might have interpreted large ventricles as evidence of disease, and thus caused our controls to have smaller ventricles than normals. The former seems more likely, but cannot be established.

In a large series worthwhile comparisons can be made within it, comparing groups of different severity for example. With only eighteen...
BY P. B. STOREY

**Table VIII**

**Sample Case Protocols**

D.G. act. 22. Duration 5 years.
Increasing withdrawal act. 14, auditory hallucinations and ideas of persecution followed. Complaints of interference with her thoughts, and of inability to think clearly. Has changed little over the last few years, except for a lessening in the frequency of screaming bouts, and increased incongruity of affect. 

**Diagnosis:** Hebephrenic schizophrenia.

**Ratings—Medical, Subgroup**

<table>
<thead>
<tr>
<th></th>
<th>SE (—16)</th>
<th>SW (—10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Third ventricle</td>
<td>6 mm.</td>
<td>6 mm.</td>
</tr>
<tr>
<td>Lateral ventricle (larger)</td>
<td>16 mm.</td>
<td>16 mm.</td>
</tr>
<tr>
<td>Radiologists A. and B.</td>
<td>normal</td>
<td>normal</td>
</tr>
</tbody>
</table>

E.D. act. 45. Duration 12 years.
Onset with “queer mouth movements”, incoherent speech, passivity feelings, and delusions of persecution. Very hostile to her parents, and at times violent. Frequent bouts of terror associated with auditory hallucinations in the earlier years. At present comparatively well in hospital, but relapses on going home. 

**Diagnosis:** Paranoid schizophrenia.

**Ratings—Medical, Subgroup**

<table>
<thead>
<tr>
<th></th>
<th>SE (—15)</th>
<th>SW (—6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Third ventricle</td>
<td>9 mm.</td>
<td>9 mm.</td>
</tr>
<tr>
<td>Lateral ventricle</td>
<td>20 mm.</td>
<td>20 mm.</td>
</tr>
<tr>
<td>Radiologists A. and B.</td>
<td>abnormal</td>
<td>abnormal</td>
</tr>
</tbody>
</table>

Cortical atrophy, moderate, all areas.

J.M. act. 35. Duration 6 years.
Gradually increasing suspiciousness and withdrawal at onset. Delusions of persecution with a strong homosexual flavour and auditory hallucinations developed later. Marked feelings of passivity, ideas of reference and some incongruity of affect. 

**Diagnosis:** Paranoid schizophrenia.

**Ratings—Medical, Subgroup**

<table>
<thead>
<tr>
<th></th>
<th>SE (—6)</th>
<th>SW (—14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Third ventricle</td>
<td>10 mm.</td>
<td>10 mm.</td>
</tr>
<tr>
<td>Lateral ventricle</td>
<td>no measurement</td>
<td>no measurement</td>
</tr>
<tr>
<td>Radiologists A. and B.</td>
<td>abnormal</td>
<td>abnormal</td>
</tr>
<tr>
<td>Frontal atrophy with ventricular dilation.</td>
<td>Sulci normal.</td>
<td>Sulci normal.</td>
</tr>
</tbody>
</table>

Patients this is less valuable, particularly as so many of the variables cannot be accurately assessed. Using the factors which are well established—age of patient and duration of illness—we find that they are not associated with encephalographic changes. The others—severity of illness, type of schizophrenia, and amount of physical treatment—are much less reliable. Only one statistically significant finding is made: that paranoid patients have broader third ventricles than hebephrenics. The series is small, and the finding unlikely, it can probably be safely dismissed. With regard to the severity of the illness it should be said that all these patients were markedly affected by any criteria, and previously published work would have led one to expect quite severe abnormalities. This study provides no support for the existence of cerebral atrophy in schizophrenic patients.

The most effective method of further research in this field would probably be for those workers already in possession of large numbers of well documented cases to reassess both the encephalograms and the clinical state of the patients, using independent observers and a blind technique. As far as possible they should rely on simple and objective measurements, keeping subjective judgments to a minimum. Further developments in echo-encephalography may enable large numbers of normal controls and patients to be simply and painlessly examined.

**Summary**

The literature on the encephalographic demonstration of cerebral atrophy in chronic schizophrenic patients is briefly reviewed. The absence of adequate control series is discussed.

Eighteen patients and eighteen controls were examined by lumbar air encephalography. The controls were films previously described as “normal” following diagnostic L.A.E.G. for various reasons. Two observers examined the films independently using a blind technique. When measuring, their correlation was good; but they differed significantly in their subjective assessments of abnormality. No significant differences were found between patients and controls. No relationship was found within the patient series which linked the size of the ventricles and such factors as: age, duration of the illness, severity of illness, or amount of somatic treatment. Paranoid patients were found to have broader third ventricles than had hebephrenics, but this is dismissed as being probably accidental.

Some suggestions for further research are made.
ACKNOWLEDGMENTS

I should like to thank the many people who have helped to make this study possible. Dr. M. Shepherd suggested it in the first place, and has given much encouragement and advice. Dr. R. D. Hoare arranged and performed the encephalograms, and with Dr. J. Ambrose, reported on the films. Dr. G. B. Barker and Dr. T. W. Bewley, consultants at Tooting Bec Hospital, recommended the patients for the investigations, and gave every help; as did the administrative and nursing staff of that hospital. I am also most grateful to Drs. J. A. N. Corsellis, J. K. Wing, M. L. Rutter and M. G. Gelder, who gave valuable advice, and to Miss N. Hemsley who performed the statistical calculations.

Last, but not least, I am indebted to the patients who allowed the investigation to take place.

REFERENCES


P. B. Storey, M.B., M.R.C.P., D.P.M., Senior Lecturer in Psychiatry, St. George's Hospital, London, S.W.1

(Received 8th February, 1965)