THE ROLE OF CEREBRAL DEGENERATION IN MENTAL DETERIORATION

A clinico-neuropathological study

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PREAMBLE

This study is based on the clinical and pathological examination of 24 elderly, deteriorated men who lived together during the last months of their lives in a ward of a mental hospital. The ward was one which had been reserved for the care of senile dements and other patients requiring considerable nursing attention.

The purpose of this study was to see if a clinico-pathological method could be found which would go some way to solving the problem of the relationship of the clinical symptoms of senile dementia and the degenerative changes of the brain.

This subject was considered worth investigating because there is still so much confusion surrounding it. Whilst previous work has shown that cerebral atrophy, senile plaques, neurofibrillary tangles and vascular disease are seen to a variable degree in the brains of the elderly, at present, it is not possible by examining a brain, to state with certainty whether one is looking at a normally aged brain or a pathologically aged brain. On the clinical side the definition of normal and abnormal ageing is just arbitrary. In fact, Post (1965) avoided defining ageing beyond stating that 'normal ageing merged imperceptibly with abnormal ageing' and 'that dementia in old age, especially of the senile type might not be an illness, but merely an exaggeration and acceleration of a statistically normal process'.

Because there is no clearer definition of senile dementia than a 'progressive intellectual deterioration including memory loss' it was necessary to investigate different aspects of the patients which lent themselves most readily to measurement. Since the patients in the present study were deteriorated, and verbal communication was often unsatisfactory, a behavioural assessment was made the prime investigation. Supplementary

investigations were carried out by using memory tests, and other psychological measurements.

Ideally an attempt to investigate the relation of mental function and cerebral degeneration in the aged would include both the alert elderly as well as the obviously demented but this is impracticable as it could not be completed unless a period of about 10 years is allowed for the patients' follow-up. The present study is limited to investigation of one end of the spectrum, i.e. the most deteriorated patients were chosen.

There has been only one previous prospective clinico-pathological study of patients who were dementing but this made use of the single examination of each patient. The present study is the first investigation in which repeated monthly examination of the patients was carried out so that one could see whether the patients did deteriorate further, if so, what was the pattern of their deterioration and finally whether the measurable abilities in such deteriorated patients bore any meaningful relationship to their pathology.

In order to understand why a study of this sort which appears to tackle such basic questions is only now being carried out for the first time it is necessary to understand the historical development of knowledge in this field.

HISTORICAL REVIEW

Although the physical and psychological aspects of old age have been described by writers and thinkers since antiquity, one of the earliest descriptions of abnormal ageing appeared as late as the 17th century: William Salmon (1646-1713) was a self-styled "Professor of Physick" who practised near St. Bartholomew's Hospital to catch patients who could

either not be admitted or were discharged un-cured. He described a patient with senile dementia under the heading of "Defects of the imagination, Reason and Memory in a man Superannuated". His description was of a man whom he had known for many years and who when he grew older showed striking loss of memory, being unable to recognise the Doctor or remember anything which he told him. He also showed lability of mood. Salmon considered him to be "decayed in his intellectuals" and believed that age might be "partly the cause".

> "Sir John was not mad or distracted like a man in Bedlam, yet he was so depraved in his intellect that he was become not only a perfect child in understanding but also foolish withal, as his laughing and crying without any reasonable cause did show, and by this deprivation becoming childish and foolish, he acted little better than the part of a mad man or a man Non-Compos-Mentis."

The first notable description of abnormal ageing which included a description of the changes in the brain is to be found in Kraepelin's Text Book of Psychiatric Illness (1896). He described the illness 'Altersblödsinn' as usually beginning between 65 and 75 years. He noted that in some possibly constitutionally weaker individuals or those who had been weakened by a stressful life and overwork and had become worn out, the illness could start as early as the end of the fifth decade. Kraepelin designated this younger group as cases of 'Senium praecox'.

His clinical description of Senile Dementia was extensive and included the features which are currently accepted under the term. He drew attention to the patient's deterioration, his memory disturbance with a profound impairment of recent memory, a tendency to dwell in the

past and the late onset of a progressive dysphasia. He noted the variable moods ranging from depression to elation with bewilderment as a frequent feature. He commented on the extreme emaciation of many patients and their poor appetite.

Kraepelin described the brain as showing definite macroscopic and microscopic atrophy. Brain weight was reduced about 200g. and there was a comparable loss of volume. The meninges were usually thickened, and the gyri were uniformly narrowed. Kraepelin noted atheroma of the large vessels and haemorrhages of varying size within the brain. The neurones appeared atrophied and were often deeply pigmented. There was also a marked gliosis.

In 1896 he already stressed the ill-defined boundary between the clinical features of normal ageing and the earliest evidence of a dementing process. In a later edition of his textbook (1904) he wrote: "... at present we are unable to answer the question as to the clinical position of this group of cases with certainty, but perhaps it will be possible to do so some day with the help of pathological anatomy".

The considerable advances which had been made in the development of the compound microscope, and the improvement in staining techniques had set the stage for Kraepelin's prediction to be justified. In 1906 Alzheimer in a concise note reported a case which resembled senile dementia except for the fact that the patient was in her early fifties.

> "This 51 year old woman had become unjustifiably jealous of her husband. Shortly after this there was evidence of impaired memory. She became unable to manage in her home, moved furniture about endlessly and hid various articles. She believed that someone wanted to kill her.
>
> On admission to hospital she was bewildered and totally disorientated.

Paranoid ideas were present and at times she seemed to be hallucinated. She was severely dysphasic and showed disturbance of reading and writing. Physical examination revealed no other abnormality. Her dementia became more profound and she was mute and incontinent before she died some 4½ years after admission."

When Alzheimer examined the brain after the patient's death, he found that there was a generalised atrophy and arteriosclerosis of the larger vessels. Silver staining of the sections revealed apart from a neuronal loss and gliosis, senile plaques and neurofibrillary tangles. Although senile plaques had been described before, this was the first time anybody had noted tangles. This case, which was the forerunner of other similar cases, was considered to be an example of presenile dementia and Kraepelin (1910) named the condition "Alzheimer's disease". Plaques had in fact been described earlier (Blocq and Marinesco 1892, Redlich 1898) in the brains of epileptics in which they had been considered to be incidental findings.

There seemed little doubt at the time that the pathological findings were responsible for the clinical picture although the respective part played by the various abnormalities was not known.

Elderly patients who had been admitted to mental hospitals with varying degrees of intellectual deterioration were now being found, after death, to have the same changes as patients with Alzheimer's disease. This discovery sparked off repeated discussions as to the relationship of Senile Dementia to Alzheimer's disease. Were they separate disease entities as is still believed by some authors today (Mutrux 1958, King 1960) or were the pathological changes merely part of a continuum found with ageing and hastened by unknown factors in the presenile dementias?

Alzheimer (1911) himself favoured the latter view and its adherents included Grünthal (1936), Wilson (1940) and Newton (1948) and Neumann and Cohn (1953).

Towards the end of the last century and until about the 1920's most neuropathological work was being carried out not by pathologists but by psychiatrists who were looking at the brain for the causes of G.P.I., the schizophrenias and the other functional disorders. As a shift from a purely organic approach to a more dynamic one was made by the psychiatrists, neuropathology became more and more the province of the pathologist. Consequently much of the debate concerning the relative importance of the cerebral pathological changes in relation to senility and senile dementia was then contributed by pathologists who worked from retrospective clinical records.

The pathologists contributed several single cases or small groups of cases showing the basic pathology and clinical features but with one or more unusual findings, e.g. focal atrophy, (Liebers 1933, Divry et al 1935, Rothschild and Kasanin 1936), cases with tangles but no plaques (Goodman 1953, Raskin and Ehrenburg 1956a), cases with plaques but no tangles (Weimann 1921), cases with congophilic angiopathy of the familial type (Worster-Drought et al 1940, 1944, Van Bogaert et al 1940, Luers 1947) and the non-familial type, (Corsellis and Brierley 1954, Neumann 1960). Three cases of senile plaque formation and systemic amyloid were described (Marinesco cited by Haberland 1964, Krucke 1950, Haberland 1964).

In the larger series of cases (Uyematsu 1923, Neumann and Cohn 1953, Newton 1948) there were brief clinical descriptions of the individual cases but these investigations were not standardised so that close

clinical comparisons could not be made. These reports cannot contribute much to clarifying the relationship of the severity of the clinical disturbance to the severity of the pathology.

A few pathologists thought quantification of plaques might elucidate the problem. Some contented themselves with recording a subjective impression grading plaque frequency as mild, moderate or severe. Two important studies of actual counting were those of Simchowicz (1924) and Grünthal (1926). Because different techniques were employed for counting the results cannot be meaningfully compared.

To make the picture more confusing reports were coming in of senile changes being present in young patients (Barrett 1913) (Ferraro and Jervis 1941). The youngest case ever described (Malamud and Lowenburg 1929) was a boy who developed scarlet fever when aged seven years. At 15 years he showed personality change, confusion and restlessness and disturbance of speech. He died at 24 years and his brain showed typical plaques and neurofibrillary tangles.

Cases were being described where the senile changes were present in conditions other than Alzheimer's disease:- plaques and tangles together being found in mongolism (Jervis 1948), Creutzfeldt-Jakob disease (McMenemey 1963). Plaques without tangles were found in kuru (Greenfield 1963) and cases of carcinoma (Neubürger and Rösch 1934-35). Neurofibrillary tangles alone were described in a staggering variety of diseases. Hirano and Zimmerman (1962) include in their list post-encephalitic parkinsonism, amyotrophic lateral sclerosis, idiopathic Parkinsonism as well as G.P.I., tabes, juvenile paresis, cholera, dysentery, intestinal tuberculosis, rabies, cranial trauma, pellagra, Tay-Sachs, disseminated sclerosis, hereditary cerebellar ataxia and cachexia.

It thus became of extreme interest to know what the incidence of these changes was in the non-demented elderly in order to assess whether they played a significant role in the development of dementia or whether they could be regarded as incidental findings. In fact very little work was published on the cerebral findings in normal people. This can be understood when one realises that post-mortem examinations are only carried out on patients in hospital or where because of the manner of their death an examination is carried out for medico-legal purposes. Such coroner's post mortems usually include a routine examination of the brain which does not entail the use of special staining techniques to show microscopic senile changes. Even if these cases were to be examined for the presence of plaques and tangles there still remains the insoluble problem of obtaining an exact record of their mental state before death.

One of the most noteworthy investigations of the brains of non-demented patients was that by Gellerstedt (1933). His sample of 50 patients who died over the age of 65 years came from general hospitals. The mental state was assessed retrospectively from the clinical records and in addition some relatives were interviewed to establish if there had been any memory impairment. His interesting finding was that plaques were found in 84% of his subjects. Gellerstedt commented that the frequency was higher than previous work would have suggested. In addition he also noted that tangles were not uncommon in non-demented people. As he did not quantify his findings it cannot be known what the severity of these pathological changes were.

Rothschild (1937) in a study of 50 'healthy' brains between the ages of 55 and 86 found plaques in 25 (very scanty in 12); they were found more readily as age advanced. This aspect of his paper is not really acceptable however since the patients who formed his control group

were all diagnosed as suffering from some form of mental illness, including 5 with "psychoses with cerebral arteriosclerosis" and 2 with symptomatic psychoses. The author gave no details of their clinical history or mental state at the time of death.

There have been isolated case reports of centenarians. Critchley (1929) found only a very few plaques in two subjects aged 99 and 102. Schukru-Aksel (1936) found no plaques in the brain of a man reputed to be 130 years old when he died. Other cases collected by Critchley (1929) included macroscopic descriptions of the brain only. In all this there is insufficient information to indicate that these patients were 'normal' at the time of death and not demented.

The work of Dayan (1970) is of interest. He examined a series of cases labelled senile dementia as well as a group of cases who died accidentally, e.g. following road accidents. He found that the incidence of plaques and tangles tended to rise with increasing age and was greater in demented patients. These results are, however, based on only the examination of sections from the right middle frontal gyrus at the level of the caudate nucleus and the right ammon's horn at the level of the lateral geniculate body.

An additional cause of controversy about the respective significance of the senile changes has been the presence of vascular disease. Arteriosclerotic degeneration of the brain was noted by early anatomists. Griesinger's volume on psychiatry (English edition of 1867), regarded as a forerunner of modern textbooks, contains a small paragraph, under the heading "Apathetic Dementia", where he used the term senile dementia as describing "Those cases in which a primary state of mental weakness has for a long time preceded an attack of apoplexy or of encephalitis."

Kraepelin in 1896 spoke of the effect of cerebral arteriosclerosis on the mental state. He described cases with increasing dementia, others which were indistinguishable from G.P.I. Headache, dizzyness, memory loss were features and permanent paralyses were noted. He considered the illness to begin in the late forties or early fifties. He mentioned Binswanger's sub cortical or white matter form of arteriosclerosis (1894) and Alzheimer's findings. Alzheimer (cited by McMenemy 1963) thought that dementia in the aged was caused by arteriosclerosis in some cases.

In a textbook of psychiatry published in 1932 (Cannon and Hayes) patients with dementia due to arteriosclerosis were divided into those whose symptoms were the result of:-

1. Arteriosclerotic atrophy of the brain manifested either by 'headache and fatiguability with vertigo' or 'a more severe form in which confusion, hallucinations, and disorientation were associated with irritative phenomena such as apoplectiform and epileptiform convulsions ... and where the patients passed into a profound dementia'.
2. Senile cortical devastation following from sclerosis affecting the small cortical end arteritis which resulted in small areas of softening or atrophy. Here 'the mental symptoms can vary greatly. Confusion, irritability, apoplectiform attacks and focal pareses are among the symptoms. This too can give way to dementia. Physical signs are more obvious than mental disturbances in this variety'.

Walton (1966) considered the term Cerebral Atherosclerosis to describe a 'progressive degenerative disorder resulting from widespread atheroma of the cerebral vasculature with consequent intermittently progressive ischaemia of the brain. The condition causes an irreversible

dementia but it is not possible to accept this diagnosis as a cause of dementia arising in the presenium or in the elderly unless there have been transient episodes of confusion, paraesthesiae, aphasia or paresis, to indicate that one or more 'little stroke' has occurred".

It is thus seen that though the effect of large vessel disease is regarded as responsible for some cases of dementia, this and the possible effect of small vessel disease await further clarification.

An attempt to reclassify psychiatric disorder of the elderly was made by Roth (1955). See Table 1. He felt that too little emphasis had been placed on the affective disorders as a separate nosological category by earlier writers. One of the results of this was that he circumvented the problem of whether his patients who were suffering from senile dementia had plaques and tangles or not by defining a condition "Senile Psychosis" in which there was a progressive "failure in the common activities of everyday life and a clinical picture dominated by failure of memory and intellect and disorganisation of a personality, where these were not attributable to specific causes such as infection, neoplasm, chronic intoxication or cerebrovascular disease known to have produced cerebral infarction".

Since the cause of dementia in the elderly is unknown it seems to be wise to detach the pathological associations from the clinical picture in order to reconsider the problem. However, the logic of Roth's action is not followed through by his acceptance of a group designated "arteriosclerotic psychosis". Although he defined it well he still assumed that in these cases it was the vascular abnormality alone which was responsible for the dementia.

CATEGORIES OF MENTAL DISORDER IN OLD AGE AS CLASSIFIED BY ROTH (1955)

1. Affective Psychosis
2. Senile Psychosis
3. Late Paraphrenia
4. Arteriosclerotic Psychosis
5. Acute Confusion
6. Other disorders (miscellaneous)

TABLE 1

Corsellis was interested to see whether Roth's criteria held up (personal communication 1970) and whether Roth's diagnostic groups remained clear-cut entities when related to their pathology. At the same time Corsellis' broadly-based study tackled the whole problem of the relationship of atrophy, plaques and tangles to mental function. He examined the brains of 300 patients who had been diagnosed as suffering from an organic or functional condition during life. (The clinical diagnosis was based on Roth's scheme). He found that the incidence of all types of degenerative change was higher in his organic group.

This study made two main points. The first was that patients who were diagnosed during life as having some progressive degenerative condition showed a far greater incidence and degree of brain atrophy, vascular change, plaques and tangles than his functional group. The second point was that these brain changes were present to some extent in patients diagnosed as suffering from schizophrenia, manic depressive psychosis, and other non-organic states.

Although, the organic changes in some of his functional group may have been due to the onset of a dementing process whilst in hospital there is also the possibility that the few plaques seen in these patients were a function of their age. It cannot be known whether testing a short while before death would have revealed symptoms of intellectual deterioration.

Corsellis thought that intellectual deficit was caused by a cumulative effect of the organic changes and he did not attribute a special significance to any one change. This study, the largest yet carried out, indicated that the whole problem of organic change and its effects could be taken no further unless precise clinico-pathological

investigations were made.

CLINICAL QUANTIFICATION

In the early fifties there was a changing approach to psychiatric diagnosis. It was thought that a quantified method for multiple psychiatric diagnosis might be helpful. The term 'diagnosis' was used by Wittenborn (1950) in a limited descriptive sense and the diagnostic method which he proposed was not concerned with aetiological, prognostic or dynamic considerations. He thought that use of specially designed rating scales would identify the existence of clustering tendencies among psychiatric symptoms. If these clustering tendencies retained their essential characteristics for different raters and for patients in different hospitals they could provide the basis for an effective multiple diagnostic procedure. Advances in statistical methods had paved the way for the complicated procedures which the analysis of such data would require.

At the outset Wittenborn (1951) enunciated certain precautions which should be observed in the design of these scales.

1. The rating scales must sample the important symptoms in the patients.

2. The rating scales must be restricted to currently discernible behaviour so that their repeated use can reveal changes in the patients. If they were not restricted to currently discernible behaviour, their appropriate use would be limited by the adequacy of the patient's social history.

3. The scales must provide ratings which are relatively independent of the insights and sophistication of the rater. The

use of the scales cannot be based on dynamic interpretations or any inferences which require a skilful use of some particular theory or consideration of prior events.

4. The rating scales must provide ratings which are relatively independent or any bias or theoretical persuasion of the rater. The halo effect is ever present and one can only attempt to minimise it.

5. The rating scales must be simple in form.

6. The rating scales must be reliable.

By adhering to this approach a method of clinical observation was developed which was a far cry from the detailed descriptive approach used in the past. Wittenborn,and others (Lorr et al 1953, 1955, 1957, 1960; French et al 1970; Heninger et al 1970) have been concerned with differentiating neurotic patients from schizophrenic and other psychotic patients. They did not concern themselves with geriatric patients. However, their careful work with its accent on objectivity, their exploration of the problems of reliability and validity of their scales provided a basis for the development of a purely behavioural questionnaire for administration to geriatric patients. Although there were often ambiguities in these early questionnaires the importance of this work was that one could learn from its defects and make modifications as indeed the authors themselves are constantly doing. Also, it suggested a new way of approaching the problem of the relation of dementia and ageing to cerebral pathology.

QUANTITATIVE CLINICO-PATHOLOGICAL STUDIES:

Roth and his colleagues (Blessed 1968) were probably directly or indirectly

influenced by the work of Corsellis and the authors of rating scales when they began their clinico-pathological correlative study. Their work was novel in that it was a prospective study which for the first time attempted to quantify both the clinical and the pathological factors. They attempted to investigate the relationship of organic change to dementia.

Their inquiries were conducted on patients admitted to a psychiatric hospital, a geriatric hospital and a number of wards in a general hospital. An attempt was made to place the patient into a diagnostic category, employing the criteria outlined by Roth in 1955. Secondly an attempt was made to describe, in quantitative terms, the degree of intellectual and personality deterioration shown by the patient. The first method of quantification, using questionnaires, was based upon the patient's ability to cope with personal, domestic and social activities. His ability to deal with the practical tasks of everyday life during the previous six months was ascertained by questioning a close relative or friend. Although "the score was kept up to date as far as possible in patients who survived by observations recorded by the nursing staff on the ward in the period between the original score and the pre-terminal state", no details were given about the interval between the final assessments and the patient's death.

The second method of quantification was based upon the performance by the patient in a number of simple psychological tests of orientation, remote memory, recent memory and concentration.

A mean plaque count was calculated after death by taking sections from both sides of the brain. Though this included sections from the frontal, parietal, temporal and occipital regions only five fields were sampled on each of 10 slides. These fields were selected arbitarily and errors

may arise if the distribution was not even (Hollander 1968).

Roth et al found a highly significant correlation between mean plaque counts and scores for dementia and performance in the psychological tests. But among their severely demented subjects and those diagnosed clinically as senile dements (26) correlations between psychological and pathological measures declined sharply. For example there were six patients who scored zero on their 'test scores' yet whose mean plaque counts range from 14 to 42. Similarly there were 13 patients whose dementia scores ranged from 16 to 24 and who had a mean plaque count ranging from 6 to 50. A possible source for the decreasing correlation may be that the tests used were unable to differentiate severe deterioration.

The next publication by this group of workers (Tomlinson, Blessed, Roth 1968) was the first paper where quantitative methods were used to assess the intellectual capacity of a non-demented population. Sixteen females and 12 males aged between 65-92 years (mean 75 years) from a geriatric unit of a general hospital were compared with patients from a mental hospital. The patients showed good intellectual preservation. Brain weights and ventricular size were not greatly different from those reported for younger subjects. Cerebral atrophy was usually slight or absent. Only four patients showed notable retraction of the brain from the dura and atrophy in these cases was largely limited to the frontal and parietal convolutions.

Many brains showed softening and senile changes including plaques, tangles and granulo-vacuolar degeneration alone or in combination. Patients with functional psychosis and acute confusion did not show greater pathological changes than those dying of purely physical diseases.

There was no evidence that softening or senile changes contributed

to the development of confusion or depression.

The net finding of all this work suggests that cerebral degenerative changes can be seen in non-demented patients but occur with greater severity in demented patients, but this generalisation falls far short of answering the question; how much atrophy to how many plaques to how many tangles to how much vascular disease causes how much, if any, mental deterioration?

Roth's work has shown the need for prospective studies. Ideally these would include a large random sample of elderly people. This type of investigation might take anything up to 10 years before completion and require a large staff. Although this was considered impracticable with the resources of the author of the present study it was felt that some other aspects of the problem could be tackled.

It was considered desirable to produce a method which differentiated the most severely demented patients from one another clinically and pathologically. A prospective study in which serial observations of the patients were made had never been done before and it was felt that this might prove helpful in obtaining an assessment of the patients' abilities based on a number of repeated observations rather than a single one and also on information as to the pattern of their deterioration.

It seemed that the most suitable available persons on which to conduct such a study was a group of elderly patients who had been admitted to hospital for a variety of reasons and who included patients suffering from both organic and functional psychiatric disorder.

Previous workers such as Rothschild /(1937) stressed the importance of obtaining information about the personality of the patient before the illness. Whilst the previous history of the patient, including his family

history, social history and previous illnesses are essential for complete understanding of the patient it seemed more important, for assessment of severe deterioration to standardise their present situation by assessing all the patients on a single ward and to concentrate on gauging their current mental state.

Because several of the patients were too deteriorated for conventional assessment of their mental function a behavioural method was used which took into account their limited powers of communication and function.

MATERIAL AND METHODS

Elm House, a small ward with 29 beds was selected for the study. The reasons for this are as follows:

The use of a single ward facilitated liaison with the nursing and ancillary staff. The nursing staff were trained to assess the patients' behaviour and it was their observations which were used to rate the behavioural questionnaire. The nursing staff were also more easily trained to administer the Tooting Bec Memory and Orientation questionnaire.

It was considered best to start with a small group of patients treating it as a pilot study and then select the most pertinent aspect of the study for administration on a wider scale.

By using a single ward one could control for environment. In fact, after the start of the study it became general practice to transfer the patients to an intensive care unit if they developed acute medical complications or became terminal. As this did not affect more than about one to three weeks of the patients life it did not influence more than one, or at the most, two monthly ratings.

The Ward

Elm House is a single storey building. There is a large day room which leads through a locked door on to an open verandah. At one end of the ward a door gives way to a passage from which leads off a kitchen, the nurses office and a small dining room. At the other end of the day room a passage leads to the bathroom and lavatories, the clinical room and the dormitory. The dormitory has 26 beds separated by lockers and there are three single side rooms.

Criteria for admission to Elm House

1. The patients were males over 60 years of age with one or two exceptions.

2. The patients required considerable nursing attention either continuously, as might be the case with a demented patient, or periodically, as may be the case with chronic schizophrenic or manic depressive patients.

A 12-bedded ward, Harper 2, was available for the physically ill patients. Two of the patients in this study were admitted to the ward during the last month of their lives and one patient spent some three weeks in this ward six months before his death. Apart from these episodes all the patients were assessed while on Elm Ward. It was hoped that this would standardise their environment to some extent. (During the study six patients were transferred to other wards or discharged and so were lost to the study. All the patients who died came to post mortem, the relatives being very co-operative and understanding about this).

Patients' Daily Programme

The patients were awoken and dressed in the morning by the night staff, (generally two or three). They were then seated in chairs which were placed around the walls of the day room. All but the most deteriorated patients went to the dining room for breakfast, the remaining patients being fed in the day room. After breakfast the patients spent the rest of the day in the day room, leaving it for lunch and dinner and to go to the toilet or the bathroom. On fine, warm days, which are few in this country, they were seated in their chairs on the verandah. There were two offical visting days during the week although no visitor would ever be turned away on unofficial visiting days. Once a week an occupational

therapist came to play games with them for about an hour and the music therapist also came weekly. During the latter's visits the patients were encouraged to play percussion instruments, sing and dance in time to music from a record or piano. Industrial therapy was begun about a quarter of the way through the project, but since this had to be supervised by the nursing staff it did not take place regularly and at most, about once or twice a week. Although there were no books or newspapers lying about the patients were exposed to information about current events by way of radio or television, one of which was on the whole day. From time to time the patients had a film show.

Nursing Staff.

At any one time there was one charge nurse, two deputy charge nurses and a maximum of half a dozen junior nurses. The night staff consisted of two or three nurses; there was one part-time nurse, a jovial scotsman in his late 50's who was the only member of staff who was on the ward throughout the project. All the other staff changed during the investigation. Explanatory sessions were given to each new batch of nurses who were participating by the charge nurse and myself.

Design of Ward Behaviour Questionnaire.

The ward behaviour questionnaire consisted of 90 questions of which 82 investigated the patients' behaviour and the remaining 8 concerned drug administration. (Sample questionnaire in appendix).

The questions were broadly divided into two groups, physical items and emotional items. A physical item is one where a piece of behaviour is assessed independently of the mood with which it may be associated. An emotional item was defined as a piece of behaviour in which the mood associated with it is assessed. The sub categories within these

two divisions were as follows:

1. PHYSICAL ITEMS OF BEHAVIOUR

A. Primary Physical Items of Behaviour

i. Speech.

ii. Self-care.

a. Feeding
b. Excretion
c. Washing
d. Dressing

iii. Sociability - (Communication)

a. Social
b. Anti-social

B. Secondary Physical Items of Behaviour

i.. Orientation.

ii. Locomotion - mobility.

iii. Initiative.

C. Miscellaneous Physical Items of Behaviour

II. EMOTIONAL ITEMS OF BEHAVIOUR

Mood.

III. DRUGS

The responses in Section A were influenced to some extent by the patients' abilities as found in Section B.

PRIMARY PHYSICAL ITEMS

i. Speech.

This included questions 1-9 and was an investigation into the quantity and quality of the patient's speech.

ii. Self-care.

a. Feeding. This includes questions 19-23. To some extent whether

a patient feeds himself or not depends upon the rate at which he does so. If he is very slow he may be fed, at least partially. Therefore, questions 21 and 22 were put in as a check to see if the patient would feed himself in a situation where his rate of feeding would matter less.

b. Excretion. Questions 24, 25 and 26. The period of observation for continence of urine was limited to that day, but continence of faeces was observed over 24 hours. As bowel actions occurred less frequently than the passage of urine it was felt that a longer period of observation was necessary. A register was kept where faecal incontinence during the night was recorded.

c. Washing. Questions 29-31. The patient's response in this situation was recorded at the time by the nurse in attendance. In this way it did not matter when the patient's bath had taken place, as a record of his behaviour would have been made at the time.

d. Dressing. Questions 27 and 28. As an attempt was made to obtain as much of the information as possible from the day staff rather than the night staff the question was phrased to consider the patients undressing which is carried out by the day staff rather than his dressing behaviour which is supervised by the night staff.

iii. Sociability - (Communication).

a. Social. Questions 32-36. Obviously some of the other questions, i.e. questions 9, 38, 39, 46 etc. could also be considered as measures of sociability but they have not been considered in this way in the present system.

b. Anti-social. Questions 50-53.

SECONDARY PHYSICAL ITEMS

i. Orientation.

Questions 16, 17, 18, 55, and 56.

ii. Locomotion - mobility.

Questions 10 and 13.

iii. Initiative.

Question 40. (Questions 9, 38 and 39 also tests of initiative).

MISCELLANEOUS ITEMS

Restlessness/Lethargy. Questions 14, 15, 41 and 42.

Response to request. Question 37.

Sense of Proprietaryship. Question 43.

Musicality. Question 44.

Ball game. Question 45 and 46.

Inappropriate Behaviour. Questions 47 and 48.

Sexual Behaviour. Questions 49 and 54.

EMOTIONAL ITEMS OF BEHAVIOUR

Mood. Questions 58-82.

It was impossible to know whether a particular affect was appropriate or inappropriate so that these questions were condensed.

DRUGS

Questions 83-90.

Method of Rating

The patients were selected in alphabetical order and rated once a month. Because of this, the interval between two tests ranged from four weeks to seven weeks. The latter situation would occur if the patient was tested in the first week of one month and the last week of the subsequent month. In practice this meant that almost every afternoon during the week the rater would go to the ward, sit down and interview at least two members of staff in the duty room and use their answers to complete the questionnaire regarding three selected patients. Those questions which

still could not be answered because the nurse who had witnessed the behaviour had gone off-duty would be filled in when the nurse in charge had obtained the necessary information the following day. Occasionally this information was not acquired and then the answer would be filled in as 'don't know'. Throughout the study the nursing staff were encouraged by the rater not to hesitate to say they did not know. Although the rater scored the question as stated in the questionnaire, it was necessary sometimes to ask supplementary questions to get the answer. For example, when asked if the patient had spoken words or sentences, an attempt was made to get the nursing staff to give a verbatim report of the patient's speech.

Apart from obtaining information direct from the nursing staff, registers, which had been filled in previously, were referred to. These registers gave answers about the patient's faecal continence, bath behaviour, behaviour with visitors, music therapy and ball game response.

Scoring of Ward Behaviour Questionnaire

All questions were rated as 'Yes', 'no', 'don't know' or 'not applicable'. For details of the scoring of individual questions see appendix. The following questions were scored to give a behaviour profile.

Speech

Questions 1-9. No = 0 Yes = 1

Self-care - feeding

Questions 19-23. If the patient feeds himself without help, score as 1. If the patient feeds himself and also requires assistance, score as ½. If the patient had to be entirely fed, score as 0.

Self-care - excretion

Questions 24, 25 and 26. Patient incontinent of urine = 0. Patient continent of urine = 1. Patient incontinent of faeces = 0. Patient

continent of faeces = 1.

Self-care - washing

Question 29. No = 0 Yes = 1.

Questions 30 and 31. No = 1 Yes = 0.

Self-care - dressing

Questions 27 and 28. If the patient undressed himself without assistance = 1. If he attempted to undress but needed assistance = ½. If the patient made no attempt to undress = 0.

Sociability - social

Questions 32 and 36. In an encounter between two patients the observers would frequently not know who had initiated the contact. Therefore the patient scored one mark if question 34 or question 35 was rated as yes.

Orientation

Questions 16, 17 and 18. If found way or could point to all three, scored 1. If found way or could point to any one of three, scored ½. If found way or could point to none, scored 0.

Locomotion

Question 10. Yes = 1 No = 0.

Initiative

Question 40. Yes = 1 No = 0.

Miscellaneous - Ball Game

Questions 45 and 46. Patient scored 1 mark if rated Yes for either question 45 or 46.

Items other than those listed below were not used in the final analysis as they did not seem to contribute to differentiating the patients.

The following items were extracted from the Ward Behaviour Questionnaire and quantified as described above to give a Behavioural profile:

1. Speech present.

2. Spoke words.
3. Spoke sentences sensibly.
4. Speech relevant.
5. Most of speech sensible.

6. Attempted to feed self.

7. Attempted to dress self.

8. Attempted to wash self.

9. Continent of urine.
10. Continent of faeces.
11. Lavatory know-how.

12. Made contact with staff.
13. Made contact with patients.
14. Made contact with others.

15. Orientated on ward.

16. Walked unaided.

17. Left chair spontaneously.

18. Caught or threw ball.

Thus a final total of 18 items was obtained. This date was handled as follows:

1. Monthly 'Mean' Score of Ward Behaviour Questionnaire.

A total score was obtained for each monthly rating (maximum 18). This score was then converted to a fraction and expressed as a percentage. The denominator was the sum of the items which were applicable and excluded the items rated as 'Don't know' and 'Not applicable', e.g. if two questions were not applicable and the patient had scored a total of eight then his monthly mean would be 8/16 x 100 = 50%. 50% was thus the monthly weighted mean. (This method was considered to be a more valid way of handling the data than always using 18 as the denominator).

These monthly means were then used (a) to obtain a Total Mean Percentage Score and (b) they formed the basis of comparing patients' patterns of deterioration.

2. Total Mean Percentage Score of Ward Behaviour Questionnaire (WBQ)

The monthly means for each patient were taken to obtain a Total Mean, e.g. if a patient's scores for four months were 40%, 30%, 50%, 40%, then his total mean was 40%. This total mean was used for comparison with the severity of the patient's pathology and to compare the patients with one another. An analysis of variance was used.

3. Percentage Frequency Performance

The number of times a patient performed a particular item out of the number of times he was tested, was calculated and expressed as a percentage, e.g. if a patient attempted to wash on four of the five occasions he was rated his percentage frequency performance was 4/5 x 100 = 80%. If, however, on one occasion bathing had been rated 'not applicable' (because there had not been any bathing) his score would be 4/4 x 100 = 100%.

The percentage frequency performance was used to compare single items, e.g. bathing, continence of urine with the patient's pathology.

Inspection of this data showed that the scores in the percentage frequency performance tests were non-parametric and for this reason an analysis of variance was inappropriate. The data was therefore analysed using a 'Trend Chi Squared Test' (Maxwell, 1961). This variant of Chi Squared is particularly useful where the data to be analysed forms a logical progression as is true of the neuropathological data. The number of subjects in some of the four pathological categories was small and therefore prior to data analysis these categories were always reduced to three by combining the two smallest sequential groups. Even so the number in the categories is small and the results should be interpreted with caution.

TOOTING BEC QUESTIONNAIRE

Design of Tooting Bec Questionnaire

This questionnaire was designed during a study (conducted at the Tooting Bec Hospital, London), correlating oxygen utilisation of elderly dements with their psychometric scores. It was found to correlate just as well as a number of sub-tests of the much more elaborate Wechsler Bellevue Intelligence Scale. The

questionnaire (for a full description see Doust et al., 1953) consists of twenty-two items covering information of past and recent personal life events as well as some past and current historical matters.

Administration of Questionnaire

The patients were tested monthly by the nursing staff and both junior and senior members were involved. The questions on recent events were changed from time to time so as to remain topical.

The advantage of this questionnaire is that it is short, can be administered by nursing staff and that as the patient's exact reponse, whether verbal or non-verbal, is recorded, the questionnaire can be marked at leisure.

Scoring of Tooting Bec Questionnaire

Full Tooting Bec Questionnaire - One point was given for each correct answer.

Short Tooting Bec Questionnaire - this included the following questions:

1. What is your name?
2. How old are you?
3. Are you married?
4. What was your work?
5. What year is it now?
6. What is the name of this hospital?
7. Can you give me the dates of the Second World War? Start. Finish.
8. Who is on the throne of England?
9. (Some question relating to recent World events)
   a. Where is there fighting at present?
   b. (Another topical question)
10. What was the address I asked you to remember earlier?

Score - One for each question answered correctly: Nos. 7 and 9 score one point if one answer is correct (maximum one). Thus a maximum total score of 10 could be obtained. This data was handled as follows. Three types of scores were derived:

1. Monthly Mean Percentage Score of Tooting Bec Questionnaire.

2. Total Mean Percentage Score of Tooting Bec Questionnaire.

3. Percentage Frequency Performance of Tooting Bec Questionnaire.

Monthly Mean Percentage of Tooting Bec Questionnaire.

The monthly score for each patient was converted to a percentage.

Total Mean Percentage Score of Tooting Bec Questionnaire.

A single mean was derived from a patient's monthly scores and it was this mean which was used to compare the patients with one another and with their degree of cerebral degeneration.

Percentage Frequency Performance of Tooting Bec Questionnaire.

The number of correct responses to a particular question during the period the patient was tested was totalled and converted to a percentage.

LANGUAGE DISORDER

Disturbance of speech was mentioned by Alzheimer in his classic case of 1906. His patient was "severely dysphasic and showed disturbance of reading and writing". Since then its frequent occurrence in other patients from Alzheimer's Disease and senile dementia has been noted (Mayer-Gross, Slater and Roth, 1960; Sjogren et al, 1952; Larsson et al, 1963).

There are numerous questionnaires available which test various aspects of the impairment, but none of these has been correlated against post-mortem findings (Head, 1926; Goldstein, 1948; Weisenberg and McBridge, 1935; Penfield and Roberts, 1959; Schuell, 1960; Eisenson, 1954; Bay, 1960; Klein and Mayer-Gross, 1957).

As the pathology in patients with senile dementia or Alzheimer's disease is diffuse, tests for language dysfunction were not undertaken with a view to determining the localisation of a particular disorder. Rather, an attempt was made to elicit what the patient could and could not do in a number of tests. For this purpose Eisenson's 'Examining for Aphasia' (1946) was chosen. In this manual a disturbance is scored as 'complete', 'severe', 'moderate', 'little', or 'none'. There is no numerical scoring system. The questionnaire is long and often each item is tested

MODIFICATION OF EISENSON'S 'EXAMINING FOR APHASIA'

1. Recognition (12 marks)

Visual: common objects; pictures; colours; shapes; numbers; letters; words; sentences.

Auditory: recognition of sounds.

Body scheme.

Right-left orientation.

Tactile: recognition of common objects.

2. Ability to Comprehend Instructions (2 marks)

Auditory verbal comprehension.

Written verbal comprehension.

3. Ability to Perform Tasks (3 marks)

Non-verbal apraxia.

Verbal apraxia.

Constructional apraxia.

4. Ability to speak (8 marks)

Automatic speech.

Spelling.

Writing name.

Dictation.

Copying.

Arithmetic.

Clock-setting.

Oral reading.

Table 2

with three or more questions. This was felt to be an advantage as it would give a patient a greater chance to score in a particular section.

The questionnaire was slightly modified and arbitrarily quantified: see table 2.

If a patient could complete all the tests in a subtest he received one mark. If he made one or more errors he scored half a mark and if none were correct he scored zero. The maximum number of marks was twenty-five.

The initial assessment of the subjects included the following examinations:

1. Physical examination.
2. Examination of the Special Senses.

a. Vision. An optician, Mr. Mitchell, examined every patient to assess visual acuity and the general state of the eye including the retina. In the case of some very deteriorated patients the eye chart was of no use and here one could merely assess the patients' reaction to light and fingers being thrust in front of them. It was not possible to test the visual fields of such patients. In some cases one could only deduce that they were not blind because they would take a proferred object, or for example, find their way to the door and open it without hesitation. The vision of all the patients in the samplewas considered adequate for the study.

b. Hearing. This was tested by an E.N.T. specialist, Mr. Punt. Again some patients were difficult to test, but extreme deafness could be excluded. The exception was case 60, who though deaf, could lip-read and read written material).

3. Mental State Examination.

A semi-structured interview was used which was based on the U.S./U.K. Mental State Interview Schedule 1968 P.S.E. 8th edition (January 1968)

P.S.S. Diagno 1 (November 1967). The advantage of using this questionnaire, which was not designed for administration to a geriatric population was that the questions were stereotyped and that each patient, no matter what his level of comprehension or cooperation, was subjected to an interview which lasted approximately the same time.

It was thought that if the patients were questioned in all areas one might elicit information which would not be given without specific questioning. The mental state summary for each patient (see appendix) was based on this interview.

Psychological Tests

Tests used.

1. Wechsler Adult Intelligence Scale.
2. Figure Form-board tests.
3. Token test.

W.A.I.S.

This test was used because it is one of the most commonly employed intelligence tests and the patient's score would be meaningful to other psychiatrists.

Figure Form-board tests.

Two figure, three figure and five figure form board tests were administered because it was thought that the patients might find these tests more engaging and that they would measure comprehension and spatial ability (details of method in appendix).

Token Test.

This test devised by De Renzi and Vignolo (1962) modified by Bollere Vignolo (1966) and was thought to be another simple way of measuring comprehension.

PATHOLOGY

Post-mortem examinations were carried out by C.J.B. in 20 cases and J.A.N.C. The brains were fixed in formalin. Macroscopic examination of the coded brains was performed by J.A.N.C. and included assessment of atrophy and large vessel degeneration. Cerebral atrophy included assessment of cortical atrophy and ventricular enlargement, grades 0-3 as in Corsellis (1962).

0 = no cortical atrophy	0 = no ventricular enlargement
1 = slight cortical atrophy	1 = slight ventricular enlargement
2 = moderate cortical atrophy	2 = moderate ventricular enlargement
3 = severe cortical atrophy	3 = severe ventricular enlargement

Cortical atrophy and ventricular enlargement were added together giving a range from 0 to 6. In tabulation, a total of 1 or 2 ranked as slight cerebral atrophy (degree 1), 3 or 4 as moderate (degree 2), and 5 or 6 as severe (degree 3). The assessment was based on the appearance of the brain as a whole and not on that of a particular region.

Assessment of arteriosclerotic change was made by examination of the basilar and internal carotid arteries with their main branches and graded 0 to 3. 0 = no evidence of vascular change, 1 = slight, 2 = moderate, and 3 = a severe degree.

Following macroscopic examination sections were cut and stained for histological examination. The sections included the following areas: Frontal, parietal, temporal and occipital lobes. Basal ganglia (usually at more than one level). Cerebellum, midbrain, pons and medulla.

Large blocks with an area of some 12 to 16cm^2 were embedded in paraffin wax. Blocks for cutting frozen sections were taken in all cases.

The sections cut were fixed and stained by the laboratory technicians. The following stains were used:

On all blocks: Haematoxylin and eosin (H&E), iron haematoxylin and van Gieson's mixture (v.G), Luxol-fast-blue for myelin counter-stained with the periodic acid Schiff method (LFB/PAS), the Palmgren, Glees or Bielschowsky's method for nerve fibres and senile plaques. Congo red for amyloid, Mallory's phosphotunstic acid haematoxylin for glial fibrils, Nissl's stain for cells, Heidenhain stain for myelin.

On selected blocks: Perl's method for iron.

On frozen sections: von Braunmuhl for plaques and tangles.

The microscopic examination was carried out by the author on coded histological sections. The code was broken after the histological investigations had been completed and the clinical data evaluated.

Histological assessment of the following was made:

1. The presence and intensity of cerebral senile changes, i.e. senile plaques and neurofibrillary tangles.

2. The presence and intensity of cerebral change, especially of the smaller vessels.

3. The presence and intensity of focal cerebral damage related to vascular change.

4. The presence either of other specified neuropathological entities or of complicating factors.

The histological changes were assessed in the same way as at the macroscopic level - four degrees for each type of lesion, none, slight, moderate, severe (0, 1, 2, 3). See photographs.

An independent histological assessment was made by J.A.N.C. and agreement was reached as to the final rating.

Routine histological sections from the other viscera were taken and assessed to exclude gross pathology.

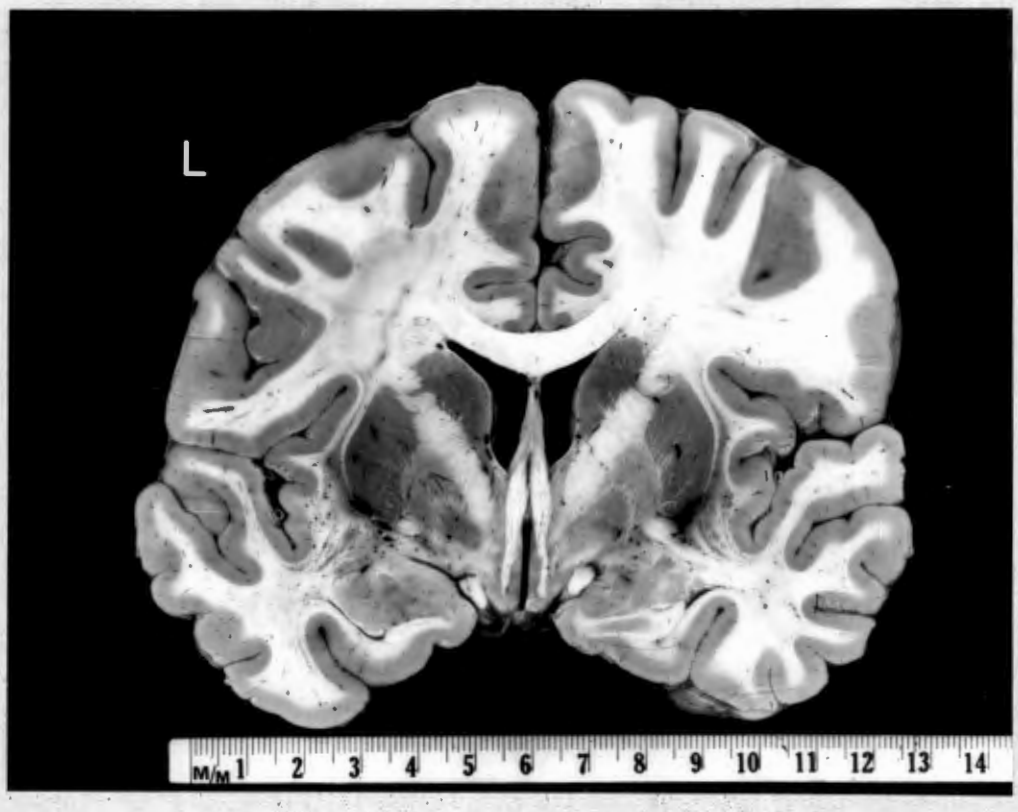

Fig. 1

Coronal section at level of intraventricular foramina showing a normal brain.
Note the width of the cortical ribbon and the size of the ventricles, which are within normal limits.

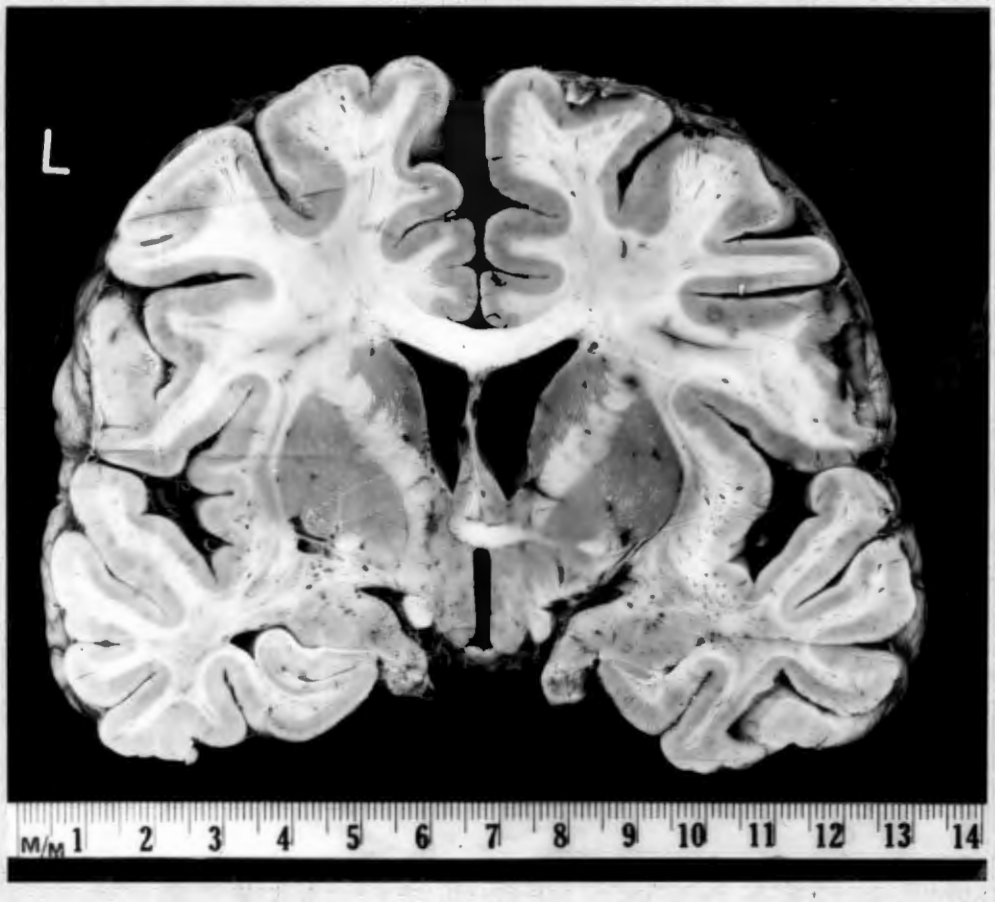

Fig. 2

Case 73
Coronal section at level of intraventricular foramina showing a slight degree of cortical atrophy.
The lateral ventricles are normal but the third ventricle is slightly dilated.

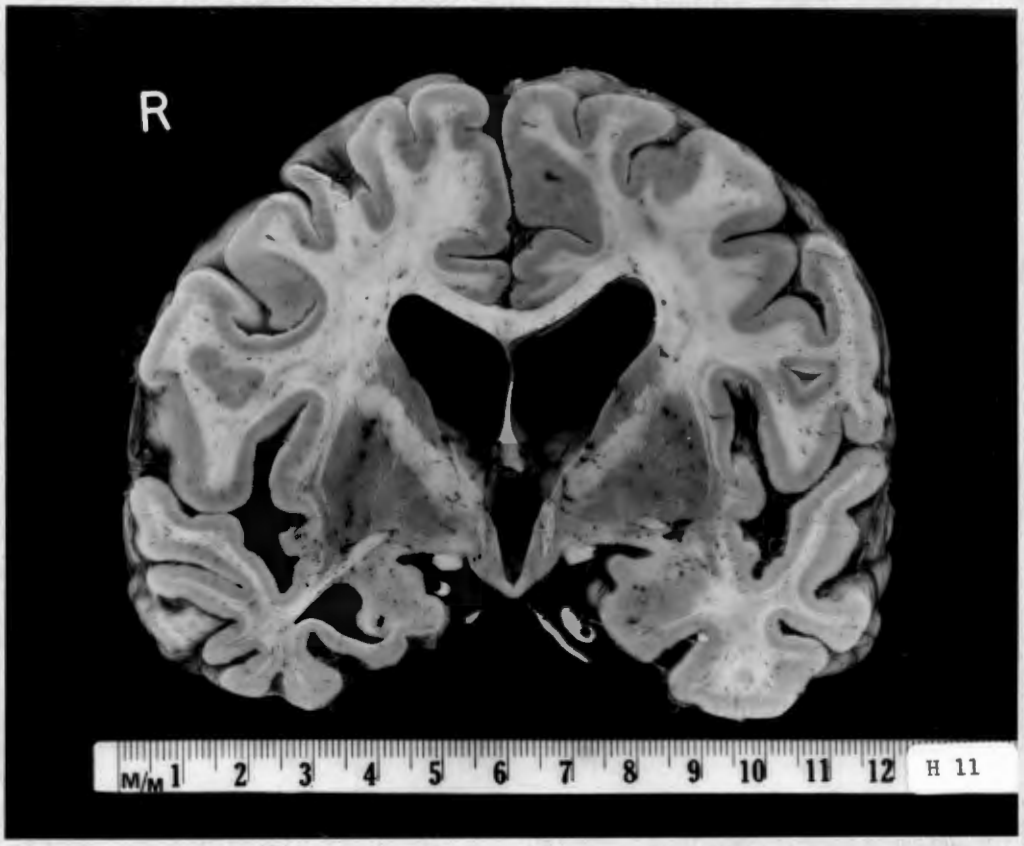

Fig. 3

Case 11
Coronal section at level of intraventricular foramina showing moderate cortical atrophy and moderate dilatation of ventricles. Note the considerable atrophy of the right temporal lobe compared with the left.

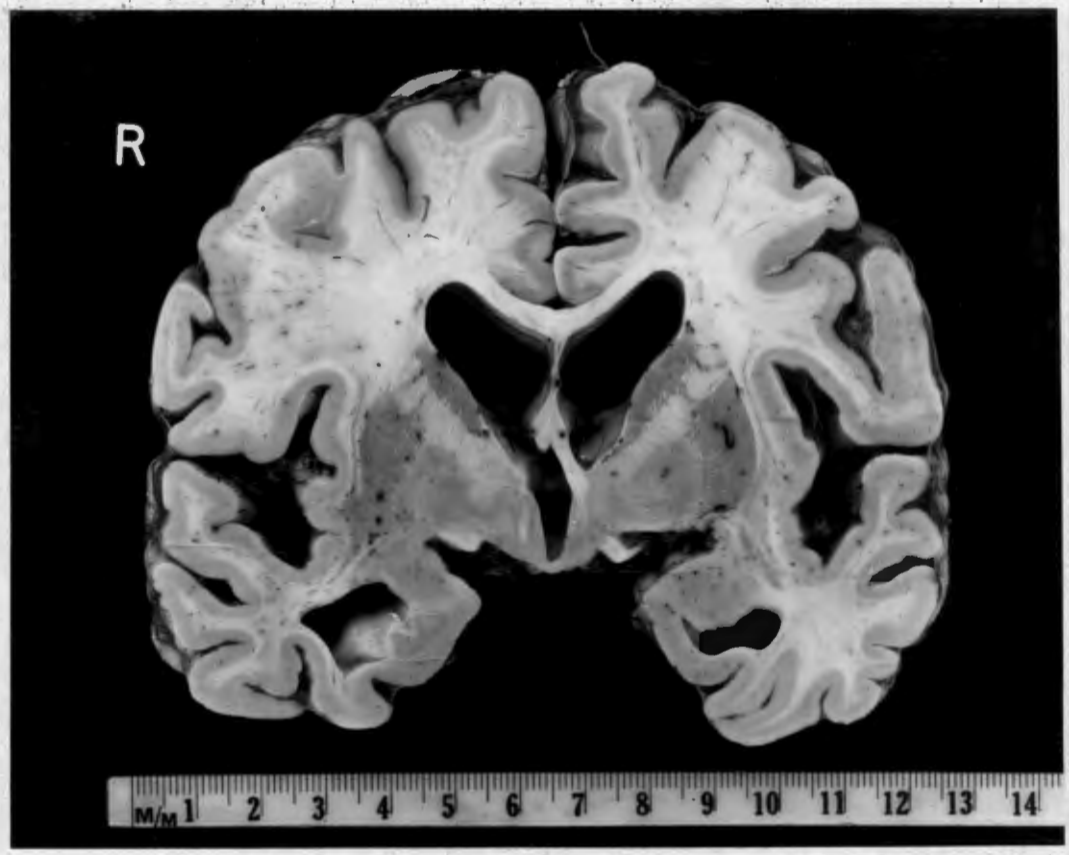

Fig. 4

Case 47
Coronal section at level of intraventricular foramina showing severe cortical atrophy and severe dilatation of ventricles.

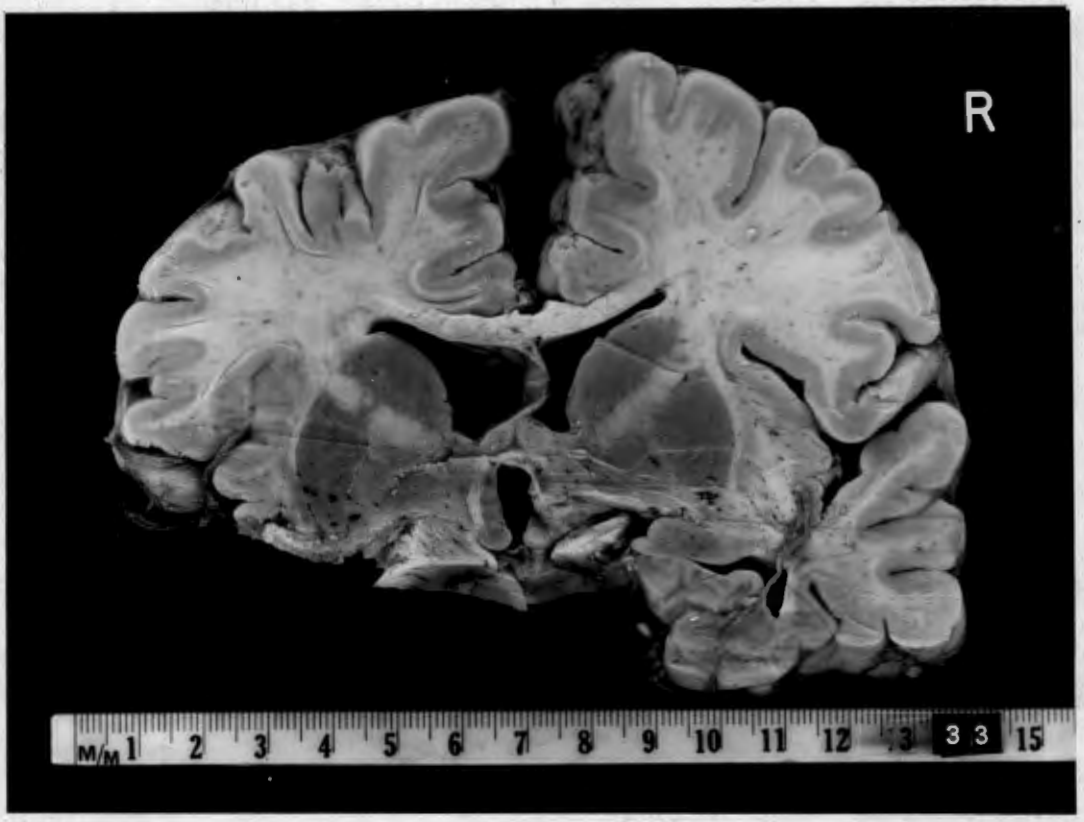

Fig. 5

Case 33
Coronal section at level of intraventricular foramina showing severe vascular disease. The left temporal lobe has been completely destroyed by occlusion of middle cerebral artery. Note the slight cerebral atrophy of the unaffected gyri.

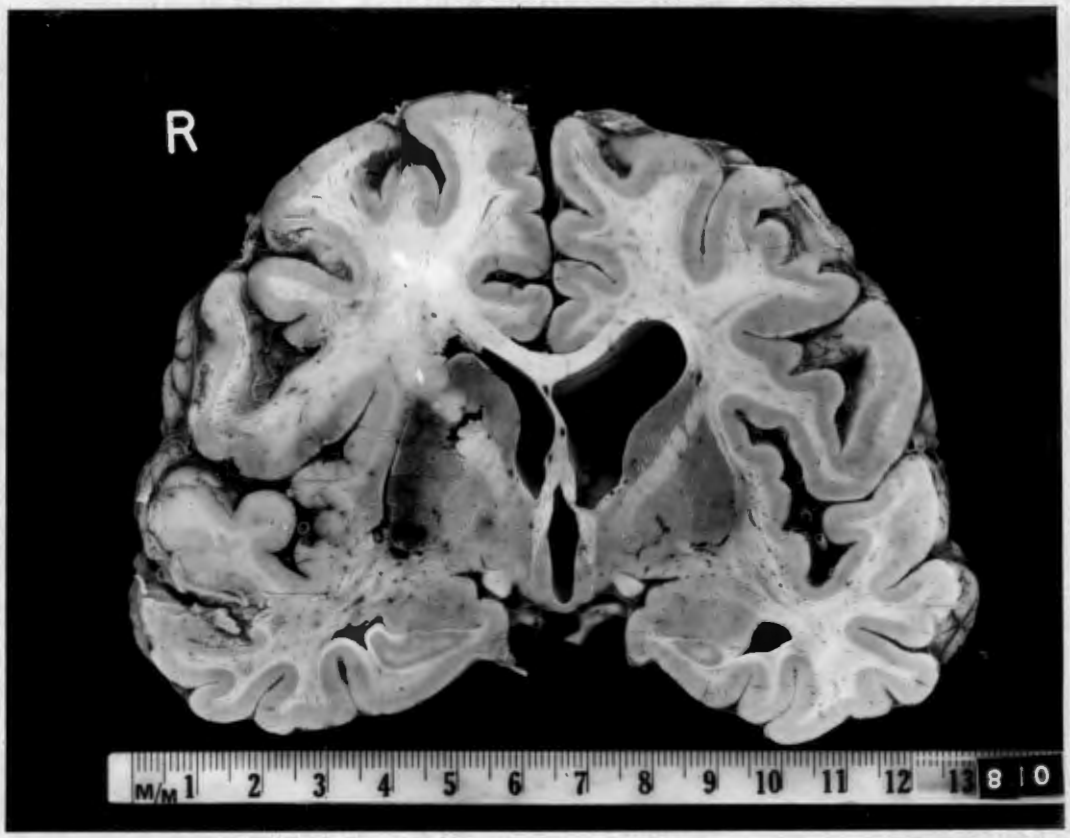

Fig. 6

Case 80
Coronal section at level of intraventricular foramina showing severe necrosis of right putamen internal capsule and caudate nucleus. (Note brain swelling of right hemisphere indicating recent infarction). The opercular cortex and insula is also softened. This is the result of a right middle cerebral artery occlusion.

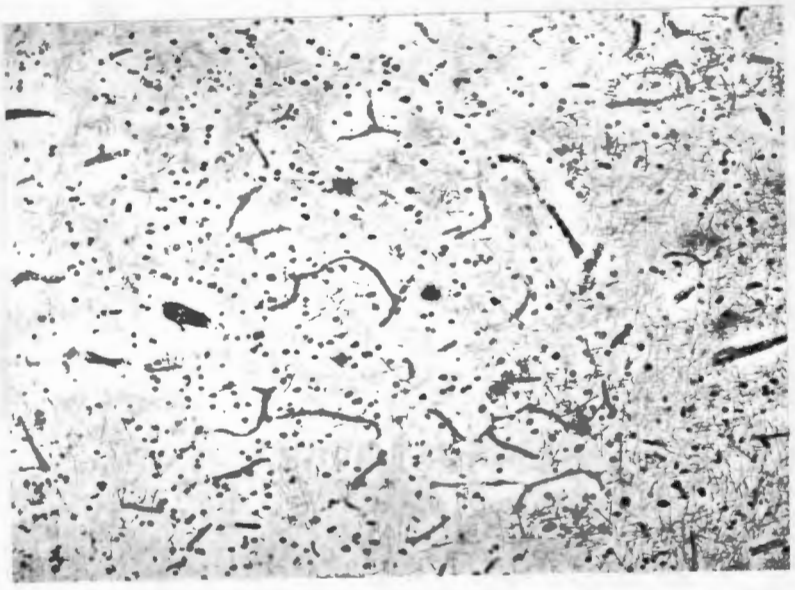

fig. 7

Case 80.

This field shows a slight degree of senile plaques (von Braunmühl x 100).

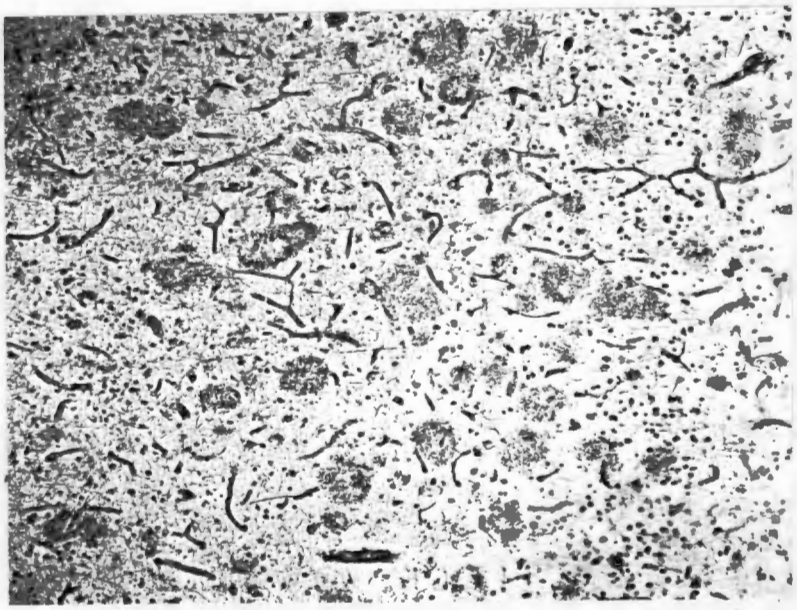

fig. 8

Case 47

This field shows a severe degree of senile plaques (von Braunmühl x 100).

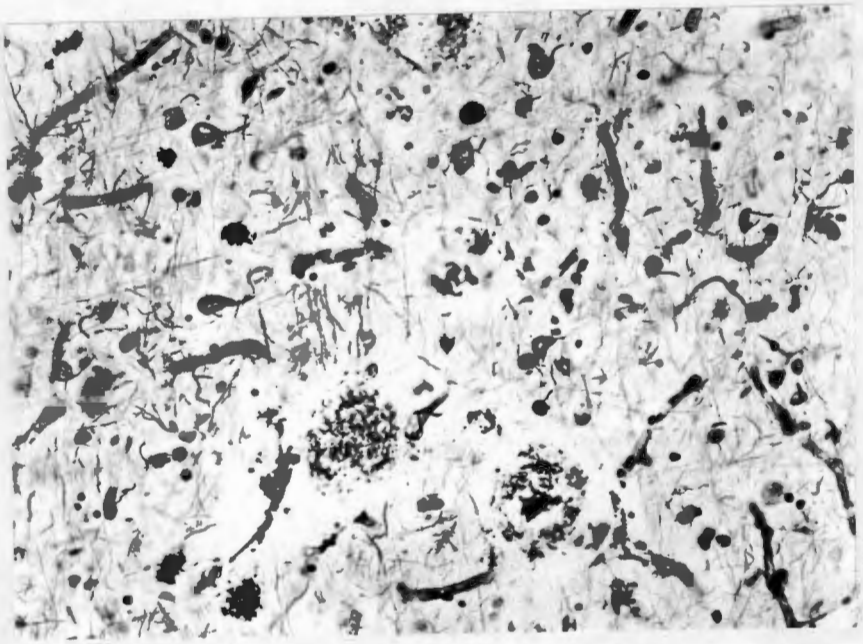

fig. 9

Case 45

This field shows a severe degree of neurofibrillary tangles and a few plaques.
(von Braunmühl x 250).

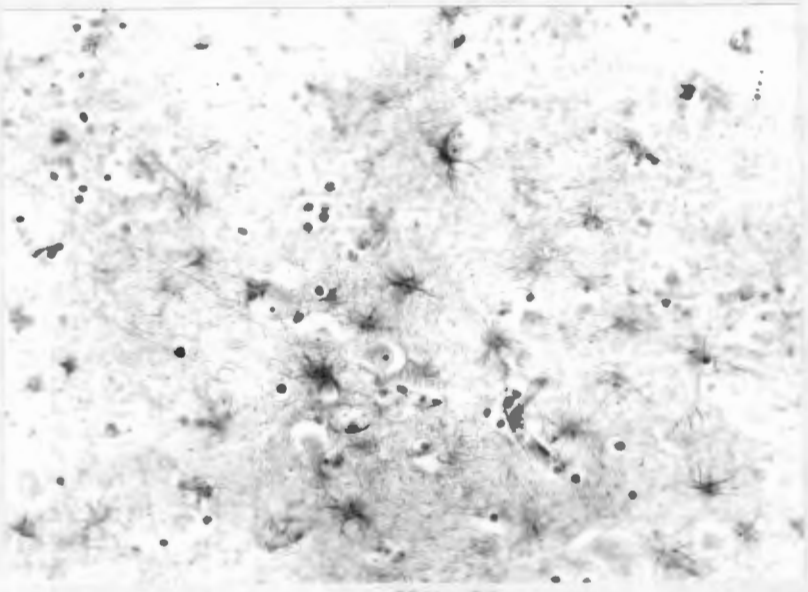

fig. 10

Case 94

This field shows many astrocytes (arrows)
(PTAH x 300).

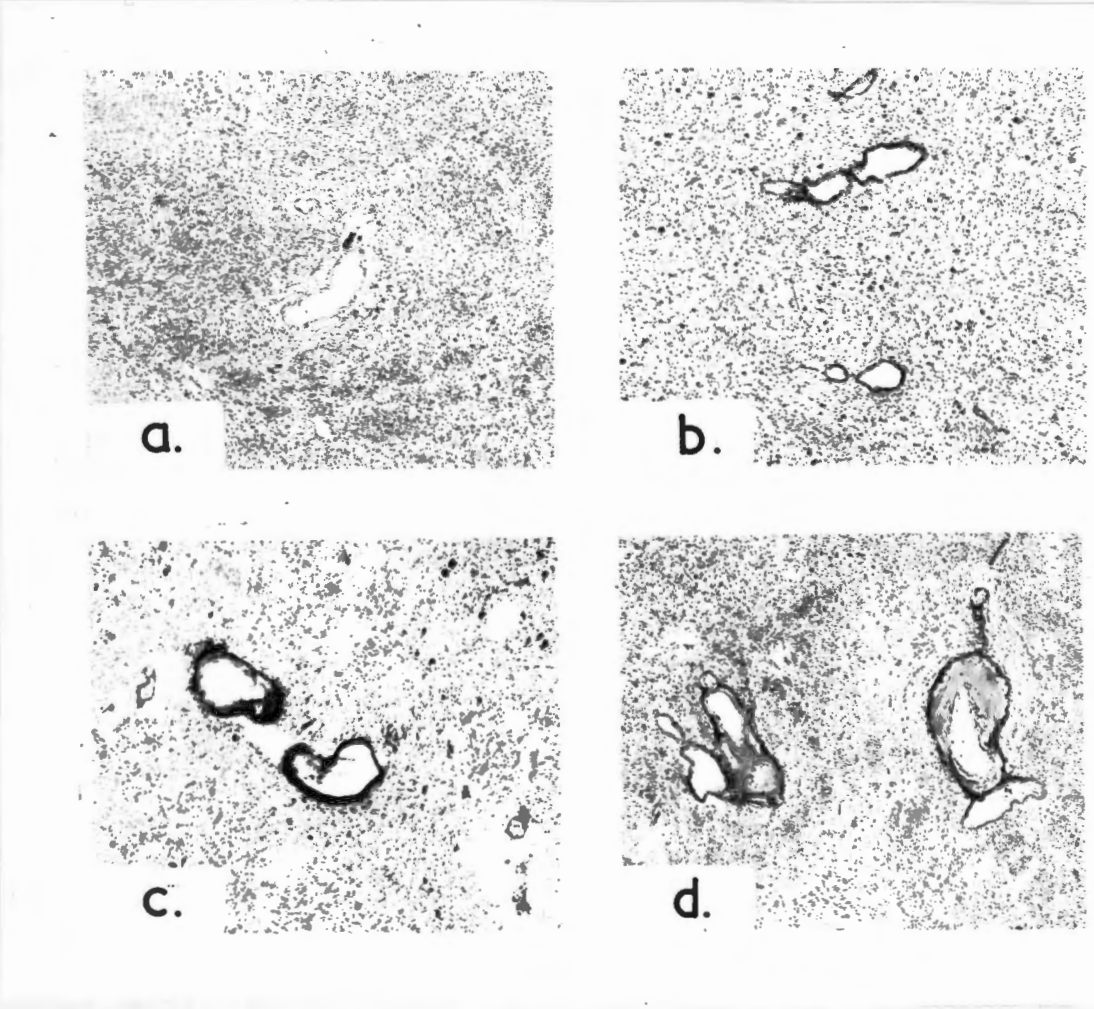

fig. 11

Showing (a) normal (b) slight (c) moderate and (d) severe small vessel disease.

Reproduced from 'The Ageing Brain' by J.A.N. Corsellis (1962).

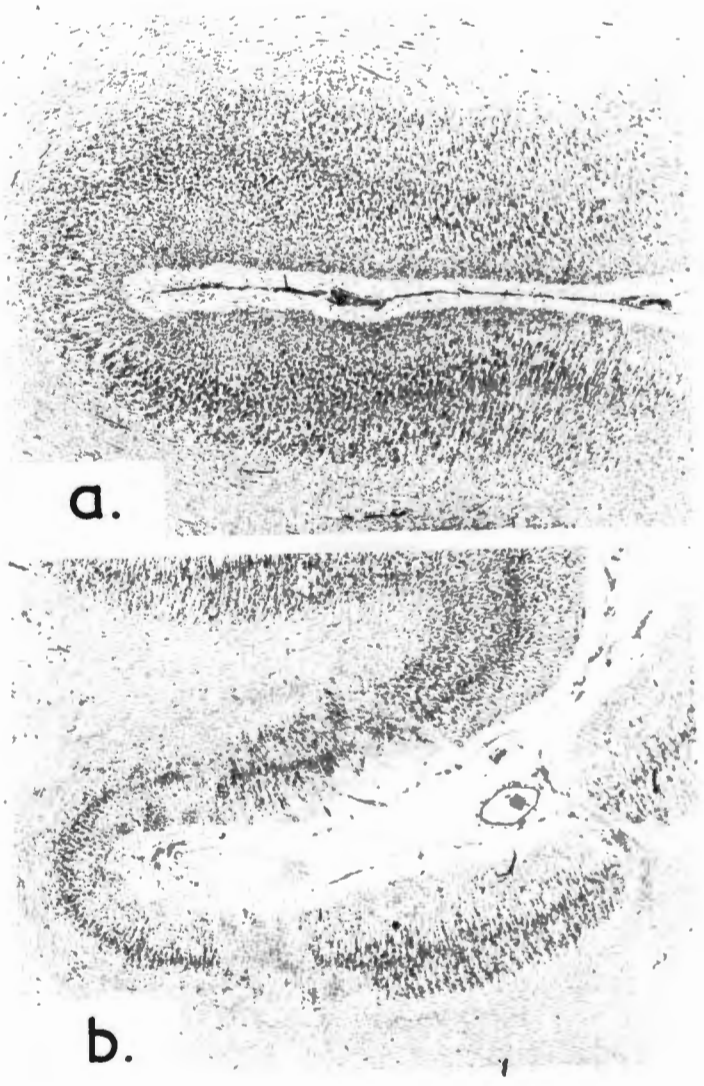

fig. 12

Showing (a) a normal cortex and (b) neuronal loss.

Reproduced from 'The Ageing Brain' by J.A.N. Corsellis (1962)

METHODOLOGICAL LIMITATIONS

The aim was to extract clinical and morbid anatomical facts, about a group of elderly patients, in order to gain further information about the relationship of deterioration in the elderly to the cerebral pathology.

The clinical data was selected in order to quantify, as far as possible, the main clinical components of the patients deterioration, e.g. speech, self-care, memory, orientation. The morbid anatomical data was based on assessment of cerebral degeneration, e.g. cerebral atrophy, senile plaques, neurofibrillary tangles, vascular disease.

The Sample

The sample was random in the sense that every patient in a particular environment was included. It was not random for the population as a whole.

Because a single ward was used the environment was relatively controlled. But this obviously does not mean that the patients were always exposed to an identical situation.

The sample was regarded as homogeneous in so far as it was an all male sample aged over 60 years. No case was rejected because of the presence of multiple pathology, partly because this is to be expected in later life but mainly because it was the patient's function which was being compared with his own pathology.

The Ward Behaviour Questionnaire

The questions were worded simply. Accuracy of observations was facilitated by asking whether a particular piece of behaviour occurred during a given period rather than how often it did so.

The questions give information about certain defined aspects of function

and do not lend themselves to generalisation. The information depended mainly on the observations of the staff but the use of registers increased accuracy of the observations. A single rater was used throughout. There was no previous work to suggest how often the patients should be tested and it was found that one month was a suitable interval. Random checks were made when patients were retested after a day and after a week; the results were virtually unchanged.

The Tooting Bec Questionnaire

The test is regarded valid as a test of memory and information but it should be emphasized that it is concerned only with those aspects of memory which require verbal responses.

Pathological Assessment

The pathological assessments were carried out on coded material.

The problems of shrinkage during fixation and histological processing are an unknown variable. It is usually assumed that different parts of the brain shrink to the same extent when subjected to identical fixing and staining techniques, as were used in the present study, but this may not be so.

The semi-quantitative assessments based on a subjective four point scale are recognisably limited but at present, no alternative superior method exists which is practical.

Quantitative assessments of plaques by counting had been personally attempted on other material (Hollander 1968). It was found that error may arise because it is difficult to assess which areas and how much of each are required to obtain a representative sample. What to count is also a problem and may lead to error. The classical plaque or neurofibrillary tangle is easily recognised but histology is essentially a three dimensional subject and therefore all manner of cuts through plaques and tangles can occur giving rise to fragments of all shapes

and sizes. When is a fragment too small? Is plaque material lying on either side of a longitudinally cut small vessel two plaques or one? Plaque counts in the cortex can include fields which are counted as cortex although they did not contain neurons. Examples of this are lumena of blood vessels, holes and cysts. If these areas are scored as 'fields having no plaques' they slightly bias the data in this direction.

Direct plaque counting requires many fields to be counted as there may be a considerable variation in plaque count from field to field as well as from slide to slide. It is for these reasons that the semi-quantitative method was selected. The material was independently checked twice by two observers and a high measure of agreement was reached. There were half a dozen cases where discussion ensued before a final decision was reached regarding one aspect of the assessment, e.g. plaques or tangles or vascular disease.

DESCRIPTION OF SAMPLE

a) Age of Sample at Death

There were 24 patients. The mean age of the subjects was 74·12 years (SD 6·57). The youngest subject was 63 years and the oldest was 84 years. See Table 3. (page 43)

b) Duration of Final Admission

The mean period of time in hospital prior to death was 2·75 years. The shortest period of admission was 3 weeks and the longest period of admission was 32 years. See Table 3. (page 43)

c) Interval between Last Examination and Death

i) Ward Behaviour Questionnaire (WBQ)

The mean interval between the last test and death was 3·26 weeks. The shortest interval was 4 days and the longest interval was 7 weeks. See Tables 4 and 5. (page 44, 45)

ii) Tooting Bec Questionnaire (TBQ)

The mean interval between the last test and death was 3·20 weeks. The shortest interval was 4 days and the longest interval was 8 weeks. See Table 6. (page 45)

iii) Mental State Examinations

The patients were assessed within six months of their death. Ten patients were not given the formal mental examination although they were interviewed by the author.

iv) Physical Examination

All the patients who had this examination were tested within 6 months before death except 2 patients who were tested 8 months before they died.

v) Language Disorder Tests

All the patients who received this test were tested within 6 months of death. (The reason why not all the patients in this study had formal mental state examinations, language disorder tests and psychological investigations was because they died before the final methodology in these investigations was decided.)

d) Number of Times Tested

i) Ward behaviour Questionnaire

The mean number of occasions on which each patient was tested was 4. The most any patient was tested was 11 times.

ii) Tooting Bec Questionnaire

The mean number of occasions that each patient was tested was 4·4. The most any patient was tested was 11 times.

DURATION OF FINAL ADMISSION AND AGE AT DEATH IN YEARS

Case	Duration final admission	Age at death
77	1·66	76
36	1·66	76
33	0·79	74
71	2·04	74
45	2·17	64
95	0·66	80
96	0·33	73
11	1·83	83
94	0·35	77
61	32·0	83
60	0·5	84
47	6·0	65
16	1·21	73
62	0·92	75
26	0·58	73
86	1·5	64
43	0·42	73
73	1·17	67
46	0·06	63
98	7·0	83
14	1·33	76
80	0·75	77
3	0·5	81
10	0·66	65

TABLE 3

NUMBER OF WEEKS BETWEEN LAST TEST AND DEATH

Case	WBC	T/B	Mental	Physical	Language
77	3•0	4•0	-	20•0	-
36	3•0	4•0	5•0	2•0	4•0
33	2•0	1•42	5•0	1•0	-
71	5•0	3•0	20•0	5•0	-
45	3•0	6•0	12•0	32•0	12•0
95	3•0	2•0	12•0	12•0	4•0
96	3•0	1•42	-	15•0	-
11	0•57	1•57	16•0	32•0	20•0
94	7•0	5•0	-	13•0	8•0
61	0•71	1•71	-	17•0	6•0
60	4•0	7•0	12•0	22•0	12•0
47	0•71	4•0	16•0	3•0	16•0
16	7•0	8•0	-	9•0	-
62	6•0	4•0	-	8•0	5•0
26	3•0	4•0	-	27•0	-
86	2•0	1•14	1•0	0•8	-
43	4•0	0•85	-	22•0	-
73	3•0	0•71	-	33•0	-
46	4•0	4•0	-	4•0	4•0
98	2•0	2•0	-	1•0	2•0
14	0•0	0•71	12•0	16•0	20•0
80	5•0	1•28	4•0	5•5	-
3	1•28	4•0	4•0	1•0	-
10	6•0	5•0	20•0	20•0	20•0

TABLE 4

Interval between last WBQ test / Death	Number of Patients
Under 1 week	4
Under 2 weeks	1
Under 3 weeks	3
Under 4 weeks	7
Under 5 weeks	3
Under 6 weeks	2
Under 7 weeks	2
Under 8 weeks	2

TABLE 5

Interval between last TBQ test / Death	Number of Patients
Under 1 week	5
Under 2 weeks	6
Under 3 weeks	1
Under 4 weeks	7
Under 5 weeks	2
Under 6 weeks	1

TABLE 6

RESULTS

The results are described in the following sequence:-

A. CLINICAL

1. The Ward Behaviour Questionnaire.
2. The Tooting Bec Questionnaire.;

B. PATHOLOGICAL

1. Cerebral Atrophy.
2. Neurofibrillary Tangles.
3. Senile Plaques.
4. Large Vessel Disease.
5. Small Vessel Disease.

C. CLINICO-PATHOLOGICAL CORRELATES OF A AND B

D. PHYSICAL EXAMINATION

E. MENTAL STATE EXAMINATION

F. MEDICATION

G. LANGUAGE DISORDER

H. PSYCHOLOGICAL EXAMINATION

A. CLINICAL

1. Ward Behaviour Questionnaire

a) Monthly Mean Scores of Ward Behaviour Questionnaire:

The monthly mean (weighted) percentage scores for each patient on each occasion they were tested is shown in Table 7. (page 48) The range of scores for each patient varied from 6 to 67; see Table 8. (page 50).

The mean range of scores for the 20 patients who were tested more than once was 26.2.

b) Total Mean Percentage Scores of Ward Behaviour Questionnaire:

The mean for the group was 59·41. The highest score was 93 and the lowest score was 23, see table 9, (p.49).

Distribution of Total Mean Percentage Scores:

This can be seen in table 10, (p.50).

2. Tooting Bec Questionnaire

a) i Total Mean Percentage Scores of Tooting Bec Questionnaire (Full):

The mean for the group was 17·8%. The highest score was 72% and the lowest score was 4%. The scores were not evenly distributed. There was a gap between the top scorer and the second highest who had 38%.

ii Total Mean Percentage Scores of Tooting Bec Questionnaire (Short):

The mean for the group was 28·7%. The highest score was 83% and the lowest score 5%.

When the Full and Short Questionnaire scores were compared there was found to be a correlation coefficient of 0·94. Therefore, in further discussion only the results of the short form will be quoted.

Distribution of Mean Percentage Scores:

This can be seen in table 11, (p.50).

b) Monthly Means of Tooting Bec Questionnaire (Short):

The mean percentage scores for each patient on each occasion they were tested is shown in Table 12 . The range of scores for each patient varied from 0 to 50. See Table 13, (p.52).

The mean range of scores for the 20 patients who were tested more than once was 24.

SHOWING MEAN PERCENTAGE SCORES ON WARD BEHAVIOUR QUESTIONNAIRE IN MONTHS BEFORE PATIENTS' DEATH

Case	1	2	3	4	5	6	7	8	9	10	11
73	90·6	90·0		96·8							
60	100·0	100·0	100·0	83·3	75·0						
14	66·6	85·3	97·0	97·0	100·0	94·0	94·0	94·0			
98	91·6	83·3									
33	64·7		88·2	83·0	90·6	91·1					
43	81·25	75·0			76·6						
11	50·0	52·7	63·8	55·5	73·5	88·8	80·5	67·6	64·7		
96	76·5	69·4									
46		72·7									
3		71·8	62·5		68·75						
95	62·0	65·0	62·5	44·0	86·0	58·0	75·0				
62		45·45	64·7	64·7			71·9				
80	71·8	50·0									
94	61·7	63·8	63·8	38·2							
71	35·7		53·3	50·0	56·3						
10	30·5	30·5	26·5	44·0	63·8	94·1					
26	46·7										
16		46·6	21·4			70·0					
36	31·25	50·0	41·0	53·0							
61	43·3										
77	42·8										
45	44·0	56·0	37·0	53·0	53·0	35·0	23·0	40·0	34·0	15·0	56·0
86	17·6	20·0	31·3	35·3							
47	7·69	23·3	21·4	21·4	40·0						

TABLE 7

RESULTS OF WARD BEHAVIOUR OBSERVATIONS

Case	No. of tests	Mean % Score	Range (%)
73	3	93	90-87
60	5	92	75-100
14	8	91	67-100
98	2	88	83-92
33	5	84	65-91
43	3	78	75-81
11	9	75	50-89
96	2	73	69-77
46	1	73	
3	2	68	63-72
95	7	65	44-86
62	4	62	45-72
80	2	61	50-72
94	4	57	38-64
71	4	49	36-56
10	6	48	27-94
26	1	47	
16	3	46	21-70
36	4	44	31-53
61	1	43	
77	1	43	
45	11	40	15-56
86	4	26	18-35
47	5	23	8-40

TABLE 9

Range of Monthly Mean Scores (WBQ)	No. of patients
0 - 25	10
26 - 50	8
59	1
67	1

TABLE 8

Distribution of total Mean Percentage Scores (WBQ)	No. of patients
0 - 25	1
26 - 50	9
51 - 75	8
76 - 100	6

TABLE 10

Distribution of total Mean Percentage Scores (TBQ)	No. of patients
0 - 25	11
26 - 50	11
51 - 75	1
76 - 100	1

TABLE 11

RESULTS OF TOOTING BEC QUESTIONNAIRE OBSERVATIONS

Case	No. of tests	Mean Percentage Score	Range
73	4	62·5	50 - 70
60	4	47·5	10 - 60
14	9	83·0	60 - 90
98	2	40·0	
33	5	36·0	20 - 60
43	5	32·0	10 - 40
11	8	36·0	20 - 40
96	3	50·0	40 - 60
46	1	20·0	
3	3	26·0	0 - 50
95	8	10·0	0 - 20
62	6	56·0	10 - 70
80	3	26·0	0 - 50
94	4	17·5	10 - 20
71	6	18·3	10 - 20
10	6	10·0	10 - 30
26	1	20·0	
16	3	10·0	10 - 20
36	4	10·0	10 - 20
61	1	30·0	
77	1	30·0	
45	11	7·3	0 - 20
86	4	7·5	0 - 20
47	4	5·0	0 - 10

TABLE 12

Range of T/B Mean Percentage Scores	No. of patients
0 - 25	13
26 - 50	7

TABLE 13

NEUROPATHOLOGICAL FINDINGS IN THE BRAIN

Cause of Death	No. of patients
Broncho-pneumonia	17
Suppurative bronchiolitis	2
Myocardial infarction	1
Dissection of aorta + left ventricular failure	1
Bronchial Carcinoma	1
Haemochromatosis + pneumonia	1
Duodenal Ulcer and left ventricular failure	1

TABLE 14

B PATHOLOGICAL

Cause of Death

Most of the patients died from a lung infection. For details see Table 14 (p52). Individual details are in Appendix

Macroscopical Findings

i Atrophy. All degrees of atrophy were found. The distribution can be seen by reference to Histogram (a) - Table 15 (p.54).

ii Arteriosclerosis. All degrees of change were found. One patient had no change, nineteen slight, three moderate and one severe change. See Histogram (b) - Table 15.

Microscopical Findings

i Senile plaques. Four patients had no plaques, four slight, five moderate and eleven severe. See Histogram (c) - Table 15.

ii Neurofibrillary tangles. The distribution of neurofibrillary change was as follows. Seven patients had no tangles. Nine patients had slight, four moderate and four severe neurofibrillary tangle change. See Histogram (d) - Table 15.

iii Small vessel disease. Two patients had no disease of their small vessels. Fourteen patients had slight, four had moderate and four had severe small vessel disease. See Histogram (e) - Table 15.

To Show Degree Of Cerebral Pathology In Twenty-Four Patients

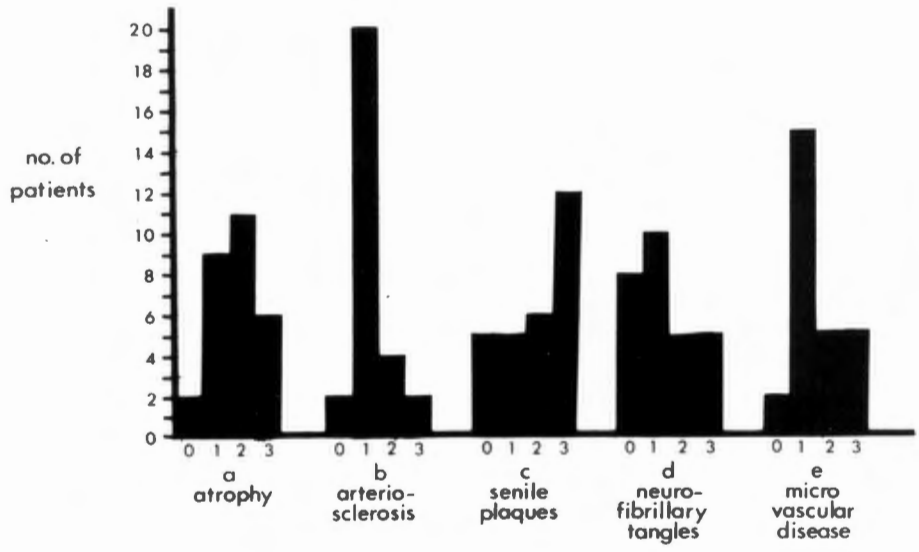

Table 15

C. CLINICO-PATHOLOGICAL CORRELATIONS

i) Clinical Correlates

1. Total Mean Percentage of Ward Behaviour Questionnaire.
2. Total Mean Percentage of Tooting Bec Questionnaire.
3. Percentage Frequency Performance of the Ward Behaviour Questionnaire (i.e. frequency of performance of individual items).
4. Language Disorder Score.

These four clinical groups were compared with the

ii) Pathological Correlates Graded (0-3) of

1. Cerebral atrophy.
2. Neurofibrillary tangle formation.
3. Senile plaque formation.
4. Vascular disease, macroscopic and microscopic.

The number of patients having any particular grade of cerebral pathology was small and so for statistical analysis subjects scoring 0 or 1 were grouped together, as were subjects scoring 2 or 3, and the differences in their clinical tests were compared using an Analysis of Variance.

Relationship of Ward Behaviour to Neuropathology

1. Correlation between Total Mean Percentage Score of Ward Behaviour Questionnaire and Cerebral Atrophy:

The relationship between these is shown in Table 16. The difference was statistically significant at the 0.001% level (see Table 17) (p57).

The Relationship of the Total Mean Percentage Score of the Ward Behaviour Questionnaire to Cerebral Atrophy

Case	Total Mean Percentage Score of W.B.Q.	Degree of Cerebral Atrophy
73	93	1
60	92	2
14	91	0
98	88	1
33	84	1
43	78	1
11	75	2
96	73	1
46	73	1
3	68	2
95	65	1
62	62	2
80	61	2
94	57	3
71	49	2
10	48	3
26	47	2
16	46	2
36	44	3
61	43	2
77	43	2
45	40	3
86	26	2
47	23	3

TABLE 16

The Relationship of the Ward Behaviour Questionnaire to Cerebral Pathology

Pathology	Degree of Pathology 0, 1.		Degree of Pathology 2, 3.		F/Ratio	Degrees of Freedom	P.
	Mean	S.D.	Mean	S.D.			
Cerebral atrophy.	71·5	9·96	51·5	17·4	18·94	1·22	<0·001
Neurofibrillary tangles	69·75	16·9	44·13	16·80	12·3		<0 01
Senile plaques	66·75	13·64	58·44	23·25	<1		Not sig.
Large vessel disease	60·80		63·25				
Small vessel disease	58·68		66·25				

TABLE 17

2. Relationship between Total Mean Percentage Score of Ward Behaviour Questionnaire and the degree of Neurofibrillary Tangle Involvement:

This is shown in Table 18. The difference was statistically significant at the 0·01% level (see Table 17).

3. Relationship between Total Mean Percentage Score of Ward Behaviour Questionnaire and the severity of Senile Plaque Formation:

This is shown in Table 18. The subjects with fewest plaques had higher mean scores on the Ward Behaviour Questionnaire but the difference did not reach statistical significance.

4. Relationship between the Ward Behaviour Questionnaire and Large Vessel Disease:

The relationship is shown in Table 19. The difference did not achieve statistical significance.

5. Relationship between the Mean Percentage Score of the Ward Behaviour Questionnaire and Small Vessel Disease:

This can be seen in Table 19. The difference did not achieve statistical significance.

6. Individual Items. The relationship between the frequency performance of individual items and pathology is shown in Tables 38-53, (see p. 105) and discussed there.

Relationship of Tooting Bec Questionnaire to Neuropathology

1. Relationship of Total Mean Percentage of Tooting Bec Questionnaire to Cerebral Atrophy:

This is shown in Table 20. The difference was statistically significant at the 0·05% level (see Table 22)(p.63).

2. Relationship of Total Mean Percentage of Tooting Bec Questionnaire to Neurofibrillary Tangles:

This can be seen in Table 21, (p.62). The relationship was statistically significant at the 0·05% level (see Table 22).

The Relationship of the Total Mean Percentage Score of the Ward Behaviour Questionnaire to Senile Plaques and Neurofibrillary Tangles

Case	W.B. Questionnaire Mean % Score	Senile Plaques	Neurofibrillary tangles
73	93	0	0
60	92	3	1
14	91	2	1
98	88	3	1
33	84	2	1
43	78	3	2
11	75	0	1
96	73	2	0
46	73	0	0
3	68	2	1
95	65	1	0
62	62	1	1
80	61	1	0
94	57	1	0
71	49	3	2
10	48	0	0
26	47	3	2
16	46	3	3
36	44	3	3
61	43	2	1
77	43	3	1
45	40	3	2
86	26	3	3
47	23	3	3

TABLE 18

The Relationship of the Total Mean Percentage Score of the Ward Behaviour Questionnaire and Large and Small Vessel Disease

Case	Ward Behaviour Questionnaire	Large Vessel Disease	Small Vessel Disease
73	93	1	1
60	92	1	1
14	91	1	1
98	88	1	1
33	84	1	3
43	78	1	2
11	75	2	1
96	73	1	1
46	73	3	3
3	68	1	3
95	65	1	1
62	62	1	2
80	61	2	3
94	57	1	2
71	49	0	0
10	48	1	0
26	47	1	2
16	46	1	1
36	44	2	1
61	43	1	1
77	43	1	1
45	40	1	1
86	26	1	1
47	23	1	1

TABLE 19

To Show Relationship of Tooting Bec Score and Cerebral Atrophy

Case	Mean % of short T.B. Questionnaire	Degree of Cerebral Atrophy
14	83	0
73	63	1
62	56	2
96	50	1
60	48	2
98	40	1
33	36	1
11	36	2
3	33	2
43	32	1
77	30	2
61	30	2
80	27	2
46	20	1
26	20	2
71	18	2
94	18	3
36	10	3
10	10	3
95	10	1
16	10	2
86	8	3
45	7	2
42	5	3

TABLE 20

The Relationship between Tooting Bec Questionnaire and Senile Plaques and Neurofibrillary Tangles

Case	Mean % of short Tooting Bec	Senile Plaques	Neurofibrillary Tangles
14	83	2	1
73	63	0	0
62	56	1	1
96	50	2	0
60	48	3	1
98	40	3	1
33	36	2	1
11	36	0	1
3	33	2	1
43	32	3	2
77	30	3	1
61	30	2	1
80	27	1	0
46	20	0	0
26	20	3	2
71	18	3	2
94	18	1	0
36	10	3	3
10	10	0	0
95	10	1	0
16	10	3	3
86	8	3	3
45	7	3	2
47	5	3	3

TABLE 21

The Relationship of the Tooting Bec Questionnaire to Cerebral Pathology

Pathology	Degree of Pathology 0, 1.		Degree of Pathology 2, 3.		F/Ratio	Degrees of Freedom	P.
	Mean	S.D.	Mean	S.D.			
Cerebral atrophy	26•5	20•84	13•44	9•805	4•47		0•05
Neurofibrillary tangles	23•06	16•19	7•25	4•24	7•22		0•05
Senile plaques	17•13	13•64	18•13				not sig.
Large vessel disease	30•35		23•25				not sig.
Small vessel disease	28•62		30•25				not sig.

TABLE 22

3. Relationship of Total Mean Percentage of Tooting Bec Questionnaire to Senile Plaques:

This can be seen in Table 21. The difference did not reach statistical significance..

4. Relationship between the Mean Percentage Score of the Tooting Bec Questionnaire and Large Vessel Disease:

The relationship can be seen in Table 23. The differences were not statistically significant (see table 22).

5. Relationship between the Mean Percentage Score of the Tooting Bec Questionnaire and Small Vessel Disease:

This can be seen in Table 23. The differences were not statistically significant.

The Relationship of Tooting Bec Questionnaire to Vascular Disease

Case	Mean % of short Tooting Bec	Large Vessel disease	Small Vessel disease
14	83	1	1
73	63	1	1
62	56	1	2
96	50	1	1
60	48	1	1
98	40	1	1
33	36	1	3
11	36	2	1
3	33	1	3
43	32	1	2
77	30	1	1
61	30	1	1
80	27	2	3
46	20	3	3
26	20	1	2
71	18	0	0
94	18	1	2
36	10	2	1
10	10	1	0
95	10	1	1
16	10	1	1
86	8	1	1
45	7	1	1
47	5	1	1

TABLE 23

D. PHYSICAL EXAMINATION

General Appearance

The striking feature was the thin and wasted appearance of the patients. Only seven of the 24 could be considered to look healthy and of these two (Patient 11 and patient 62) were obese.

Four patients (numbers 86, 80, 3 and 62) were terminal at the time of the examination.

One patient (number 95) had a bluish grey complexion. He was found to have haemochromatosis subsequently.

Cardiovascular System

The mean blood pressure for the group was 140/90. Only two patients had a diastolic over 110 and they were cases 94, (180/110), and 61,(210/140). Five patients were in cardiac failure at the time of examination. They did not include the hypertensive patients.

Respiratory System

Five patients had evidence of a respiratory infection. One patient had just sustained a pneumothorax following rupture of an emphysematous bullae.

One patient had bronchial carcinoma confirmed by bronchoscopy. Three other patients had signs of chronic bronchitis.

Gastro-Intestinal Tract

One patient had hepatosplenomegaly; he was the patient with haemochromatosis. Another had had a colostomy. There were no other significant findings in the gastrointestinal tract examination of the remainder.

Genito-Urinary System

There were no obvious abnormalities.

Bones and Joints

One patient (case 14) had severe arthritis with ulnar deviation of

both hands, and arthritis of both knees. Another patient (case 95) had gross osteoarthritis of the left knee.

Central Nervous System

Twenty-two patients were fully conscious at the time of the examination. The others, though conscious, were toxic and drowsy and in their terminal illness.

Skull

No abnormalities were noted on inspection and palpation.

Speech

Slurred speech was noted in one patient (case 43). This may have been due to his medication. He had no other gross neurological abnormality. Standard test phrases to test for dysarthria were of limited value because the patients' cooperation was limited. However, no patients were considered to be dysarthric.

Cranial Nerves

Smell was not tested.

Vision. All the patients could see. Severely demented patients (cases 36, 47 and 86) were assessed by noting that they picked up objects placed in front of them. The quality of their vision could not be assessed further. The pupils were all small.
They all reacted sluggishly to light and accommodation.

The fundi appeared normal although again all the vessels were narrower than in younger age groups. Only one patient (case 61) with a B.P. 210/14) showed stage two retinopathy.

Fields. Two patients (cases 71 and 10) appeared to have a left hemianopia. Most of the patients could not be tested for field defects.

Eye movements were normal and no patient had nystagmus.

The other cranial nerves appeared normal except for one patient who was deaf, one patient who had some wasting of his tongue and one patient who

showed wasting of his sternomastoids.

Motor System

The most striking feature of this group of patients was the severe generalised muscle wasting seen especially in eight patients. (Cases 36, 71, 47, 62, 14, 80, 3, 10). The muscle wasting did not seem to be confined to any single group. (One patient (Case 45) showed fasciculation. Five patients (Cases 95, 33, 62, 80 and 3) showed weakness of upper and lower limbs. Five patients would not cooperate (Cases 36, 71, 47, 86 and 10) sufficiently for adequate assessment. An increase in tone of a fluctuating nature was found in three patients. Two patients had localised rigidity following a previous cerebrovascular accident and one patient had a flaccid paresis after an acute cerebrovascular accident. A slight patient tremor was noted in one patient (case 96). Three patients (Cases 96, 16,11 and 62) were Parkinsonian.

Reflexes

One patient who was examined terminally (case 80) had bilateral extensor plantars. One patient with a recent cerebrovascular accident on an old cerebrovascular accident had a unilateral extensor plantar response. An extensor plantar response as an isolated finding was seen in a patient with Parkinsonism (case 11). Six of the more demented patients had a grasp reflex and five sucking reflexes (cases 36, 71, 45, 47 and 80 had both reflexes and case 10 had grasp reflex only).

Sensation

Apart from responding to pin prick it would be difficult to comment on the group as most would not cooperate or were too ill to do so for the tests of vibration, position sense etc.

Coordination

The finger/nose test and heel/knee tests performed by four patients were normal. One patient (case 77) showed poor coordination but no other evidence of neurological abnormality.

E. MENTAL STATE

The mental state was difficult to assess. The majority of patients examined in this study were remote and inaccessible by normal standards. Most times the only responses were to questions which could be answered by a 'yes' or 'no'. Only in rare cases was the patient's attention held for more than a few seconds unless constant fresh verbal stimuli were presented. Often any attempt by the patient to communicate was hampered by his speech disability. However, the impression was gained that their silence concealed gross impoverishment of thought. Their talk apart from swear words, which remained to the end, was devoid of colour and lacked subtlety.

It was impossible to tell whether these patients were depressed or not in the conventional sense. There was no evidence of euphoria or lability of mood in the present sample. Some patients appeared irritable when questioned. Thought content could not be explored in any meaningful way. Only two patients (cases 33 and 45) showed any tendency to dwell in the past. There was no evidence of hallucinations.

Since memory and orientation were tested elsewhere the patients' response to these questions will not be discussed here.

It could not be determined whether the patients had any insight into their condition. Certainly none was expressed. The conclusion in the case of most of the patients was that they were 'deteriorated' and 'demented'.

It can be seen that a mental state assessment based on this conventional approach does not easily allow meaningful comparisons to be made between patients. A single assessment of this sort, at a variable time before the patients death becomes a meaningless formality.

The problem of assessing the inarticulate, unresponsive patient who is not catatonic or stuporose can be overcome if an examination includes more behavioural items and a suggested proforma is given in the appendix.

F. MEDICATION

All except two patients (Cases 98 and 61) were treated with medication at some time during the study. Twelve patients were on a phenothiazine preparation (Chlorpromazine or Thioridazine) at some stage. Night sedation was prescribed sparingly and twelve patients are recorded as having been given this, on one or more occasions, the night before assessment of their Ward Behaviour. Eight patients received anti-biotics terminally. There was no indication that medication affected the patients' performance.

The patients' medication was as follows:

1. Case 73. Thioridazine 50mg t.d.s; Ampicillin 250mg 6 hourly (terminally).

2. Case 60. Digoxin 0·25 mg b.d; Hydrosaluric 5mg daily.
   Comment: Treatment of his cardiac failure may have been responsible for improved scores.

3. Case 14. Thioridazine 25mg t.d.s; Ferrous Gluconate 300mg b.d; Magnesium trisilicate 20ml q.d.s; Nitrazepam 10mg nocte.

4. Case 33. Thioridazine 50mg t.d.s; Orphenadrine hydrochloride 50mg t.d.s; Ampicillin 250mg 6 hourly terminally; Nitrazepam 10mg night before final assesment.
   Comment: Lowered performance on day of rating may have been the result of drowsiness.

5. Case 43. Nitrazepam 5mg the night before the final rating.

6. Case 11. Nitrazepam 10mg nocte prior to first assessment. Orphenadrine Hydrochloride 50mg t.d.s. (last four assessments).

7. Case 96. Thioridazine 50mg t.d.s; Orphenadrine Hydrochloride 50mg (first assessment); Nitrazepam 10mg prior to final assessment.

8. Case 46. Thioridazine 50mg t.d.s; Dihydrocodeine tartrate 60mg 6 hourley.

9. Case 3. Chlorpromazine 25mg t.d.s; Orphenadrine Hydrochloride 50mg t.d.s; Frusemide 20mg mane; Chloral Hydrate 15gr nocte (first rating); Tetracycline 250mg 6 hourly; Digoxin 0·25mg twice daily; Morphine Sulphate 10mg t.d.s. (terminally).

10. Case 95 Digoxin 0·25mg daily; Slow K 1ml daily.

11. Case 62 Benadryl 25mg twice daily; Dichloral phenazone 1300mg nocte (first three ratings); Aminophylline 15gm 6 hourly; Frusemide 25mg mane (terminally).

12. Case 80. Thioridazine 50mg t.d.s; Orphenadrine Hydrochloride 50mg t.d.s.

13. Case 94. Nitrazepam 10mg nocte (prior to last rating).

14. Case 71. Thioridazine 50mg t.d.s. (second, third and fourth ratings); Nitrazepam 10mg nocte (prior to last rating).

15. Case 10. Chlorpropramide 250mg daily.

16. Case 26. Thioridazine 25mg t.d.s; Nitrazepam 10mg (night before rating).

17. Case 16. Choline Theophyllinate 100mg t.d.s; Nitrazepam 10mg (second rating); Tetracycline 250mg q.d.s. (terminally).

18. Case 36. Nitrazepam 10mg nocte (prior to first, second and fourth ratings); Thioridazine 25mg t.d.s. (last rating).

19. Case 77. Tetracycline 250mg 6 hourly; Slow K 1200mg daily; Lasix 20mg daily.

20. Case 45. Thioridazine 50mg t.d.s; Nitrazepam 10mg nocte (prior to all except fourth, fifth, sixth and seventh ratings); Tetracycline 250mg 6 hourly (tenth rating).

21. Case 86. Haloperidol 1·5mg b.d; Orphenadrine Hydrochloride 50mg b.d; Tetracycline 250mg 6 hourly (terminally).

22. Case 47. Thioridazine 25mg t.d.s. (first, third, and fifth ratings); Nitrazepam 10mg nocte (prior to second and fourth ratings); Tetracycline 250mg 6 hourly.

LANGUAGE DISORDER

Eleven patients who had been tested came to post mortem. The scores ranged from 96% to zero. One patient (case 95) was tested three times over a period of 6 months, all his scores are included. The other patients were tested once.

Table 24 shows the patients' scores and it can be seen that the patients who scored highest on language function also scored higher on Ward Behaviour. (An exception was case 95) who had cerebral haemochromatosis.

Table 25 shows the results within the sub-tests.

Patient No.	Language Score %	Ward Behaviour Score %	Degrees of Atrophy	Degrees of Tangles	Degrees of Plaques
14	96	91	0	1	2
60	93	92	2	1	3
11	68	75	2	1	0
94	48	57	3	0	1
62	38	62	2	1	1
61	24	43	2	1	2
10	32	48	3	0	0
36	14	44	3	3	3
95	10, 0, 2	65	1	0	1
45	2	40	3	2	3
47	0	23	3	3	3

Table 24

The Relationship of Language Score to Ward Behaviour Questionnaire Score and Cerebral Pathology.

RESULTS OF TESTS FOR LANGUAGE DISORDERS

Recognition of: Case Nos.	14	60	11	94	62	61	10	36	95	45	47
1. Common objects	1	1	½	½	½	1	½	0	½ 0 0	½	0
2. Pictures	1	1	0	1	½	1	½	0	0 0 0	0	0
3. Colours	1	½	½	1	½	½	½	½	0 0 0	0	0
4. Shapes	1	1	1	1	½	0	0	0	0 0 0	0	0
5. Numbers	1	1	½	1	½	½	½	0	½ 0 0	0	0
6. Letters	1	1	1	½	1	½	½	½	½ 0 0	0	0
7. Words	1	1	½	½	½	½	1	½	0 0 0	0	0
8. Sentences	1	1	½	0	1	NT	½	NT	0 0 0	0	0
9. Recognition of sounds	1	NA	½	½	½	½	½	0	0 0 0	0	0
10. Body scheme	1	1	½	0	1	0	0	½	0 0 0	0	0
11. R-L orientation	1	1	½	½	½	0	0	0	0 0 0	0	0
12. Tactile agnosia	1	1	½	½	0	½	½	0	0 0 0	0	0
Ability to comprehend instructions:											
13. Auditory verbal	1	NA	1	½	½	0	½	0	0 0 0	0	0
14. Written verbal	1	1	½	½	0	0	½	0	0 0 0	0	0
Ability to perform tasks:											
15. Non verbal apraxia	1	1	1	½	½	½	½	½	0 0 ½	0	0
16. Verbal apraxia	1	NA	1	0	½	0	½	½	0 0 0	0	0
17. Constructional	1	1	1	1	0	NT	0	NT	0 0 0	0	0
Ability to speak:											
18. Automatic speech	½	1	1	0	½	0	0	0	0 0 0	0	0
19. Spelling	½	NA	1	0	½	0	0	0	0 0 0	0	0
20. Writing name	1	1	1	1	0	0	0	0	0 0 0	0	0
21. Dictation	1	NA	½	0	0	0	0	0	0 0 0	0	0
22. Copying	1	1	1	1	0	0	0	½	0 0 0	0	0
23. Arithmetic	1	1	½	0	½	0	½	0	0 0 0	0	0
24. Clock setting	1	1	½	1	0	0	0	0	0 0 0	0	0
25. Oral reading	1	1	½	0	0	0	½	0	0 0 0	0	0
Total scores =	24	19.5	17	12	9.5	6	8	3.5	25 0 0.5	0.5	0

Table 25

Psychological Tests

Sixteen subjects were tested using the W.A.I.S. Figure Form Board and Token Tests. In summary the findings were as follows:-

Five subjects were unable to make any reasonable attempt and scored 0.

Eight subjects made some attempt but did not complete the tests.

Three subjects completed the tests satisfactorily.

Only three out of sixteen subjects tested were able to complete the tests satisfactorily, therefore it was decided that these tests were not helpful in assessing the subjects. They were discontinued and reliance placed on behavioural assessments. Details of the results are in the Appendix.

DISCUSSION

General

Previous studies have shown that the senile degenerative changes of cerebral atrophy, neurofibrillary tangles and senile plaques are found, to some extent in the brains of the non-deteriorated elderly (Gellerstedt 1933, Blessed et al 1968) and that they are more common and more marked in patients with a clinically recognisable dementing condition (Corsellis 1962, Grunthal 1926, Simchowicz 1910, Tomlinson et al 1968). Less is known about the relationship between the degree of cerebral degeneration and the degree of clinical deterioration.

The present study was begun in an attempt to answer this question and it was clear that there would have to be quantitative assessments of both clinical and pathological features. It was realised that a single examination of the patient, at a variable time before death was unsatisfactory, so a long-term prospective study was undertaken, in which repeated standardised examination of the patients was carried out.

The elderly patients already on a single (29 bedded) ward in a psychiatric hospital were selected for study. This group included patients admitted to hospital for a variety of conditions. They had come to the ward direct because they had been unable to look after themselves at home and required considerable nursing attention, or because they had deteriorated whilst on another ward and were transferred. Twenty four of these patients came to post-mortem and it is the clinical and pathological findings of these patients who were observed during the last months of their lives, which forms the basis of this study.

The clinical and pathological features which were quantified and compared might be influenced by a number of variables so an attempt to control them was made.

Previous workers have used mixed samples (Blessed et al 1968, Corsellis 1962) and have not described any differences due to the sex of their cases,

but in the present study on all male population was selected.

In order to avoid the influence of a different environment on the patients' beahviour this was limited as far as practical by studying patients on a single ward with a shared milieu.

The sample was elderly. Six patients were in their sixties, twelve in their seventies and six in their eighties at the time of death. The clinical and pathological findings did not correlate positively with age. This agrees with the findings of previous workers who examined elderly patients (Corsellis 1962, Roth 1968). Yet one might have expected differences between the 60 year old group and the 80 year old group. The younger group might, if suffering from Alzheimer's Disease have been more clinically and pathologically deteriorated and it might be relevant that the three most detiorated patients (cases 45, 86 and 57) were in their sixties, all of whom were suffering from Alzheimer's Disease.

The duration in hospital of the sample ranged from 3 weeks to 32 years. This finding indicated that the patients were suffering from different conditions at the time of admission and that some of the chronic patients had aged in hospital. The duration in hospital tells us little about the duration of the illness because, in conditions like Alzheimer's Disease and Senile Dementia the onset is usually insidious and hospital admission is often precipitated by social factors.

A physical examination was carried out in all cases. The results are interpreted cautiously because firstly, a single examination at a variable time before death renders the findings not strictly comparable unless the clinical picture is static. Secondly, the nature of the illness was such that several signs could not be elicited because of the patients extreme deterioration. What emerged, however, was:

1. The striking paucity of hypertensive patients in the sample. (Only two patients had a diastolic pressure over 110mmHg.)
2. The infrequency of gross localising neurological signs. (Only one patient had a hemiplegia and this was terminal.)
3. The relative infrequency of extra-pyramidal signs.
4. The presence of severe generalised muscle wasting in a large number of patients.
5. The confirmation that positive grasp and sucking reflexes were good indicators of severe brain damage.

The chief importance of the physical examination in this group has thus demonstrated the absence of localising signs clinically and this correlates well with the diffuseness rather than the localised nature of senile dementia. These observations reaffirm the wasting characteristic of senile dementia (Sjogren et al 1952) and this may well have been an important factor in the progressive difficulty in walking. This is a question which requires further investigation.

The present study showed that the conventional mental state assessment of severely demented patients is of limited value in comparing the patients. Because this had been anticipated the Ward Behaviour questionnaire and the Tooting Bec Memory and Information questionnaire were used as the main clinical measures.

The construction of items for the Ward Behaviour Questionnaire, in order to give a dementia profile, was helped little by the accepted definitions of dementia, e.g.

1. The word 'dementia' derives from the Latin dementia, ae, (demens) (Lewis and Short 1945).
2. "...although initially synonymous with madness, insanity, lunacy and folly, it became synonymous with delirium in the early part of the 17th century. The term is now limited to those who show primary memory loss due to disorders in brain tissue and the term stresses the irreversibility

of the intellectual defects." (Hinsie and Campbell 1960).

3. "Dementia is the term applied to a diffuse deterioration in the mental functions manifesting itself primarily in thought and memory and secondarily in feeling and conduct..... the clinical picture varies somewhat according to the previous temperament of the patient, the age of onset, the localization, rate of progress, and the nature of the causal disorder." (Brain 1962).

The items of Speech, Self-care, Socialisation, Orientation, Initiative were selected to investigate the residual presence or absence of the most basic functions. The questions had to be appropriate for even the most deteriorated patient and at the same time, allow distinctions to be drawn within the patient group. The questions were as simple and unambiguous as possible and limited to observations over a set period. The test is valid if, like the definition that 'intelligence is that which an intelligence test measures',(Hilgard 1962) the patients residual function is taken as the function which the test measured.

The method of using repeated assessments was useful for three main reasons. Firstly an average of the patient's performance was obtained. Secondly, information as to how often a patient performed a particular item was gained, and thirdly, the pattern of deterioration could be seen.

It was surprising to find that the average scores on the Ward Behaviour Questionnaire i.e. The Total Mean Percentage Score showed a large variation ranging from 93% to 23%. A visitor entering the ward might, in all probability, have been struck by the appalling lifelessness and immobility of the patients. The patients were usually seated around the sides of the room, one or two might be standing, or trying to stand up, one or two others might be walking about in an apparently aimless fashion. The patients would appear listless and vacant and there would be a dreadful apparent uniformity of behaviour. Though the behaviour of the patients may, to a greater or lesser extent, have been the result

of institutionalisation, they shared a common environment, and the behavioural assessment demonstrated differences which correlated well with the degree of cerebral pathology. The poorer the performance on behavioural assessment the more severe was the cerebral pathology. Previous workers have found difficulty in separating their most deteriorated patients from one another because they scored nought. It is suggested that by using repeated simple investigations, as in the present study, this problem can be overcome, because the patient is given more than one occasion to score.

The frequency with which particular items of the Ward Behaviour Questionnaire were performed varied. Items such as speech present, speaking words relevantly and mainly sensibly were always performed correctly by over 50% of the group. Sentences, however, were always spoken correctly by 25%. Although 50% of the patients always knew what to do when placed on the toilet, less than 25% were always continent of urine and faeces. Whilst over 50% always attempted to feed themselves only four patients always attempted to wash and only one to undress. There was a striking difference between the number of patients who always made some contact with the staff (84%) and the number who never made contact with the staff (0%). Only a third of the patients always walked without the aid of a nurse and only one patient was always orientated. These findings are discussed in more detail in relation to the pathology on page 105.

Whilst the concept of a progressive deterioration is considered the main feature of Senile Dementia and Alzheimer's Disease little is in fact known about the rate of deterioration. There are excellent clinical descriptions of the dissolution of function until a stage of profound dementia is reached (Mayer-Gross, Slater and Roth, 1960) but there is not much information about the patients' residual function, i.e. if in fact all patients are deteriorated to the same degree at the time of death, and

whether their rate of final deterioration is the same.

It was thus most interesting to find, in the present study, that the patients varied more from one another in their degree of deterioration than in their actual rate of final deterioration.

The pattern of the patients' deterioration was assessed by comparing the Monthly Mean Scores of the Ward Behaviour Questionnaire. The monthly scores which each patient obtained can be seen in Table 7, page 48 and Table 26, page 81a shows the histograms for each patient. The patients have been placed in order of their Total Mean Percentage Scores.

The histograms show the difference in the degree of deterioration in the patients. They also show that there was little fluctuation during a patient's final months irrespective of his degree of deterioration. It is as though each patient achieved an individual plateau at least four months before he died with relatively little further deterioration. Only one patient (10) deteriorated precipitously.

If one compares the first observation with the last it is seen that 13 patients (nos. 73, 14, 33, 11, 95, 62, 10, 16, 36, 86, 47, 71, 45) deteriorated but seven patients improved before death. The reasons for this improvement in one group and deterioration in the other requires further investigation.

The Monthly Mean Scores of the Tooting Bec Questionnaire were used similarly to draw histograms and these can be seen in Table 27 The differences in the degree of deterioration as shown by the Ward Behaviour histograms are reflected by the Tooting Bec Questionnaire histograms. The Tooting Bec histograms also demonstrate a similar pattern of variation for each patient.

It is recognised that because of the arbitrary nature of the items in the test the histograms cannot be said to be a true linear representation of the degree of deterioration. They do however show that the pattern of

To Show The Pattern Of Deterioration Of 24 Patients On The Ward Behaviour Questionnaire

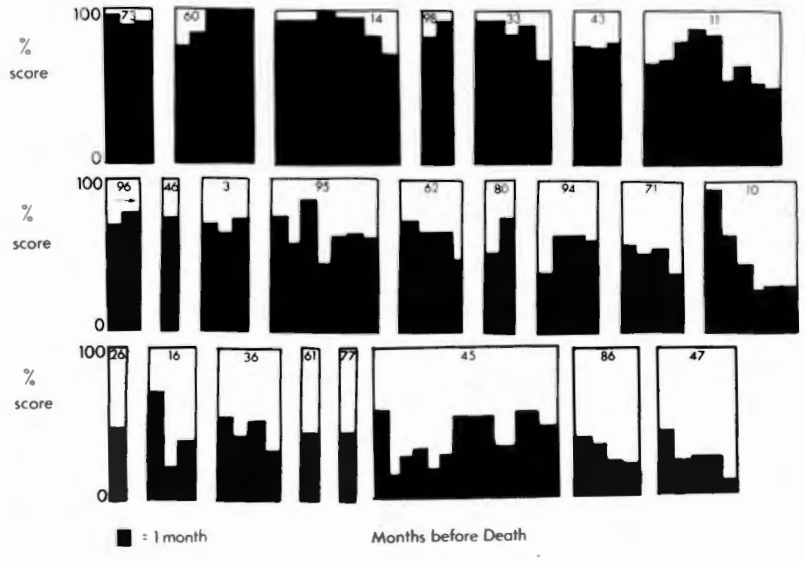

Table 26

To Show The Pattern Of Deterioration Of 24 Patients On The Tooting Bec Questionnaire

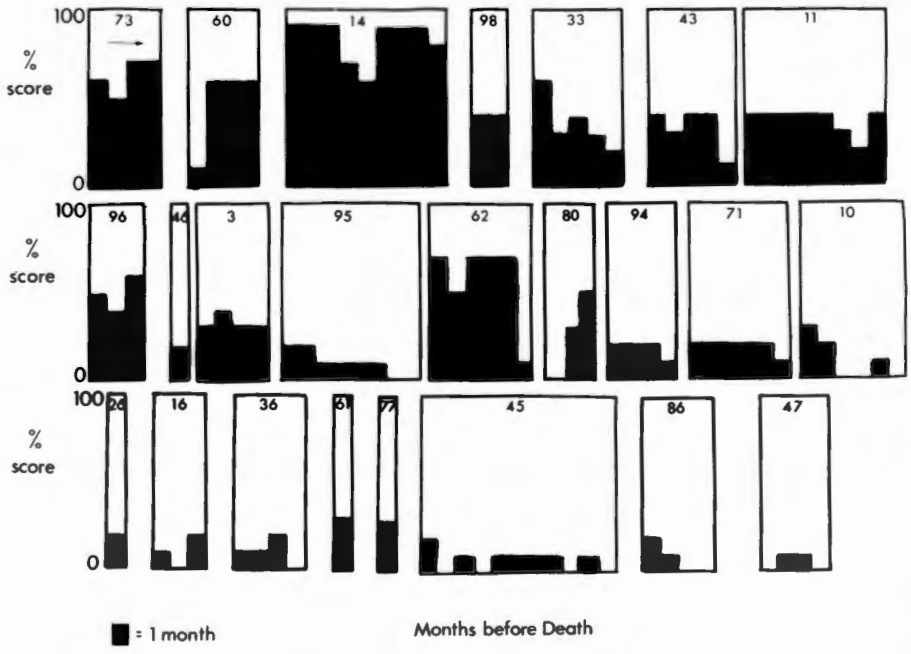

Table 27

deterioration, a few months before death is not identical for each patient, but that each patient varies relatively little before death.

The remaining results of the Ward Behaviour Questionnaire and the Tooting Bec Questionnaire are discussed in conjunction with the pathology later (see pages 105 - 124).

Although all the patients had cerebral changes these were largely in the cerebral hemispheres while the brain stems tended to be better preserved. It was particularly interesting that the degree of physical and mental deterioration varied greatly at the time of death and it seems improbable that death could be considered primarily as the result of direct cerebral degeneration. Moreover, infection was commonly found, 17 patients having died with bronchopneumonia.

THE RELATIONSHIP OF CEREBRAL ATROPHY TO CLINICAL DETERIORATION.

Cerebral atrophy graded as nil, mild, moderate or severe was found to correlate positively with clinical deterioration as measured on the Ward Behaviour Questionnaire ($p=<0.001$) and the Tooting Bec Questionnaire ($p=<0.05$). The relationship of the pathology to the clinical scores of the Ward Behaviour Questionnaire can be seen in Table 28, page 83. This shows that of the five patients who scored over 80% on behavioural function one had no atrophy and three only slight atrophy. The patient who had moderate atrophy is anomalous and will be discussed later. Four of the eight patients scoring between 60% and 79% had slight atrophy and the other four had moderate atrophy. It was only when one reached the nine patients who only scored between 40% to 59% that four patients with severe atrophy appear. The other five had moderate atrophy. Finally of the two cases scoring between 20% to 39% one had moderate atrophy and the other severe atrophy.

The trend can be seen that the lower the patient scored on Ward Behaviour, i.e. the more disabled he was, the more atrophy his brain showed

RELATIONSHIP OF WB CHART SCORE AND CEREBRAL PATHOLOGY

		Cerebral Atrophy				Senile Plaques				Neurofibrillary Tangles			
Degrees of Damage		0	1	2	3	0	1	2	3	0	1	2	3
WBC Score	No. of patients												
80+	5	1	3	1	0	1	0	2	2	1	4	0	0
60+	8	0	4	4	0	2	3	2	1	4	3	1	0
40+	9	0	0	5	4	1	1	1	6	2	2	3	2
20+	2	0	0	1	1	0	0	0	2	0	0	0	2

TABLE 28

after death. There were no patients scoring under 60% with less than moderate atrophy.

A problem surrounding the significance of cerebral atrophy is the extent to which the degree of atrophy is reflected in the clinical picture. Although there is little evidence that there is a direct relationship between cerebral atrophy and clinical symptoms, several observations point in this direction. "Some shrinkage of the brain with age is generally accepted but its relation to mental function is not known". (Greenfield, 1963). Post (1965) states "It is common knowledge that memory declines with rising age". Thus there are two observations made repeatedly which indicate an association, if not a direct relationship, between atrophy and clinical impairment.

Since Corsellis (1962) had emphasized that cerebral atrophy was a feature not only of Senile Dementia but also other organic states diagnostic labels were avoided in the present study. The work is therefore not strictly comparable with previous studies where these labels have been used. However, some of the findings of earlier work can be used to evaluate the present findings.

Gellerstedt (1933) in his work on the brains of 50 elderly subjects who died in the wards of a general hospital and were regarded as normal found no atrophy in 28 cases, mild atrophy in 5 and severe atrophy in 17. Although 66% thus had mild atrophy, the relatively symptomless 34% suggest that there may be no definite relationship between intellectual impairment and cerebral atrophy and that cerebral atrophy to a severe degree need cause no symptoms.

An example of considerable atrophy (moderate) with relatively little impairment was seen in the present study. Patient 60 had a mean percentage score of 92. He was a patient who had improved after admission to hospital and was on the point of being discharged when he fell, broke his leg and died. This sort of patient with marked brain damage yet little clinical impairment was described by Rothschild (1937). He postulated that the previous personality was an important factor in controlling observable intellectual deficit: the same extent of brain damage might manifest differently in different personalities. The present writer is sceptical that the adequacy of the personality influences ability to recall the date, be orientated in space and be capable of attending to the basic items of self-care. The relationship of patient 60's clinical picture to his cerebral pathology remains inexplicable.

Tomlinson et al (1968) who examined the brains of groups of non-demented and demented patients found that there was less likelihood of cerebral atrophy in their non-demented patients and if present it was less severe than in their demented group. However the overlap can be seen in Table 29 and their work shows that the relationship is not on a one to one basis.

Corsellis (1962) who compared the incidence of cerebral degenerative change in a mental hospital series found that cerebral atrophy to "some degree was found in three quarters of all the patients". However, the contrast between his organic and functional division was marked, for whereas

from QUANTITATIVE MEASURES OF DEMENTIA AND OF SENILE CHANGE

Blessed, Tomlinson and Roth (1968)

Neurofibrillary Tangles

	Dements	Non-dements
Not found	14	11
Few in hippocampus only	8	6
Moderate in hippocampus only	9	9
Severe in hippocampus	19	0
Present in general cortex	31	3

Table 29

from QUANTITATIVE MEASURES OF DEMENTIA AND OF SENILE CHANGE

Blessed, Tomlinson and Roth (1968)

Cerebral Atrophy

	Nil	Mostly para-sagittal	Slight generalized	Generalized atrophy Moderate	Severe	Obvious temporal lobe atrophy
Controls	13	11	4	0	0	0
Dements	20	14	8	5	3	14

Table 30

on the organic side moderate or severe atrophy was found in nearly half, atrophy of this extent was seen in only sixteen percent of the functional division. Furthermore, he found that his senile group had more moderate or severe atrophy (71%) than his vascular group (39%), with his mixed vascular group (65%) coming in between. Corsellis thus showed clearly that some conditions are associated with a greater degree of cerebral atrophy than others. If one assumes that his organic group, at the time of death, were more clinically impaired than his functional group, the findings would be good evidence of a direct relationship of atrophy to clinical function. Obviously one can take the position that one cannot make this assumption, but though both a depressed patient and a chronic schizophrenic may behave like a demented patient and be wrongly diagnosed, their presence in the organic group would only have the effect of shifting the result to the left, i.e. that Corsellis' percentage of patients in his organic group with severe and moderate atrophy might have been an underestimate.

How then do the findings, that cerebral atrophy of all degrees may be symptomless but is more likely to be associated with symptoms support the results of the present study? All the patients, bar one, had some degree of atrophy and they were all impaired clinically. The patient without atrophy (no. 14) scored 91% on the Ward Behaviour Questionnaire and 83% (the highest score) on the Tooting Bec Questionnaire indicating that he was only mildly impaired. Apart from this patient, clinical impairment assessed over a period was associated with atrophy and the more impaired the function the greater the degree of atrophy. This would suggest that whilst there is a group of people who may, even with severe atrophy, not appear grossly demented, a demented patient is more likely to have cerebral atrophy. The greater the dementia the more severe will be the atrophy. Since the findings of the present study concern a small number of patients the results must be regarded cautiously.

The method of assessing cerebral atrophy in the present study, by inspection, is semi-quantitative. It was considered the method of choice as it is a relatively rapid way of making an assessment of the whole brain and at present no satisfactory method exists for the accurate and objective measurement of cerebral atrophy. Physical methods such as assessing the weight and volume of the brain and the volume of the cranial cavity are laborious and not sufficiently reliable to warrant their use. Because of the limitations of the method used in the present study, the results must be interpreted with caution, but in the absence of a more precise technique it is felt that this simple grading has produced useful information.

The exact nature of the loss incurred in cerebral atrophy is unknown. Comparison of coronal atrophied and unatrophied sections suggest that there is a loss of both cortex and white matter. Microscopically the loss of cortical tissue appears to be due to a loss of neurones with glial proliferation and alterations in the fluid content.

Quantitative assessments of neuronal populations have been attempted, (Monagle, et al, 1974) The interpretation of the results presents difficulty because (1) when making comparisons between brains it is impossible to ensure that identical areas have been assessed; (2) by the same token, it is difficult to compare the results of different workers; and (3) in virtually all cases the neuronal counts have been made in the absence of any information as to the cognitive function of the patient at the time of death.

With more advanced techniques becoming available for particle counting, the relationship of diffuse cell loss to cerebral function may become clearer. However, the problem of the similarity of the clinical picture in illnesses where there is localised damage such as Pick's Disease as against conditions where there is diffuse damage such as Senile Dementia and Alzheimer's Disease, remains. It may well be that it is not only the loss

of cells but also their situation which is important.

THE RELATIONSHIP OF NEUROFIBRILLARY TANGLES TO CLINICAL DETERIORATION

The presence of neurofibrillary tangles graded as nil, mild, moderate or severe was found to correlate positively with clinical deterioration as measured on the Ward Behaviour Questionnaire and the Tooting Bec Questionnaire. The relationship of the pathology to the behavioural scores can be seen in Table 28. This shows that the five patients scoring over 80% included one patient with no tangles and four patients with slight. Eight patients scored between 60 and 79% and of these four had no tangles, three slight tangles and one a moderate degree of tangles.

Nine patients scored between 40 and 59% and in this group of more deteriorated there were two patients with severe neurofibrillary tangle involvement, three patients with moderate, two with slight and two patients without tangles. It is thus seen that in this low scoring group all degrees of neurofibrillary change are represented. The two patients who had no tangles in this group were nos. 10 and 94. Patient 10 was grossly atypical. Whilst he rapidly reached a stage of extreme deterioration (see fig.26) his brain only showed severe atrophy and was free from tangles and senile plaques. Patient 94 was interesting in that he too had severe atrophy, no tangles and only slight plaques. (It is this sort of picture which makes one feel that neuronal loss might be responsible for the deterioration and that tangles and plaques are incidental findings). The two patients who had only slight neurofibrillary change were patient 61, who had moderate plaques, and patient 77, who had severe plaques. In both these cases there was a considerable amount of brain damage other than tangles which may have accounted for their low scores on the Ward Behaviour Questionnaire.

It was interesting to see that in all the cases except one (patient 11) the grading of tangles was lower than that of plaques in any one patient.

But as has been stated (Wiesniewski, Terry, Hirano, 1970) many more tangles are visible under the electron microscope than with lower magnification, so that one has to be cautious in making too much of this observation.

The findings of the present study indicate that with increasing clinical deterioration, the degree of neurofibrillary change rises. Tomlinson et al (1970) showed a higher incidence of neurofibrillary change in a group of demented elderly subjects compared with a group of non-demented controls. Severe involvement was found in 31 out of 50 dements and three out of 28 controls. The degree of overlap can be seen (see fig 29), page 86. Because these workers did not comment on whether there was any relationship between the degree of dementia and the degree of neurofibrillary tangles, a comparison with the present work cannot be made.

Corsellis (1962) found a greater incidence of tangles in his organic as compared to his functional group. Furthermore, the incidence and degree of involvement was greater in his senile group than in his vascular group. Again, if one assumes that his senile group was more deteriorated than his vascular group and certainly, than his functional group, then his findings support the concept that the more deteriorated the patient the more tangles he is likely to have.

Various workers have noted a relationship of tangles to ageing (Simchowicz, 1910; Grunthal, 1927; Corsellis, 1962; Tomlinson et al, 1968; Dayan, 1970). Corsellis (1962) found that in his functional group the incidence of tangles did increase with age (p 0.01) but the overall incidence of neurofibrillary change in this division was very slight. No relationship with age was found in his organic division. Dayan (1970) found a statistically significant relationship between increasing age (over 60 years) and increasing occurrence of tangles, in a group of non-demented subjects. It would appear that a few tangles can occur with ageing and that in such cases they are relatively symptomless. The non-specificity of tangles is

suggested by their presence in conditions other than Senile Dementia and Alzheimer's Disease (see chapter 1). In such conditions the possibility that mild dementia is present but masked cannot be excluded. The development of symptoms may depend, however, not only on the absolute number of tangles but also on their situation within the brain.

THE RELATIONSHIP OF SENILE PLAQUES TO CLINICAL DETERIORATION.

Senile plaques are believed to increase with age (Dayan, 1970) and occur less frequently in normal populations (Gellerstedt, 1933; Tomlinson et al, 1970; Dayan, 1970) and are present to a greater degree in patients suffering from a dementing process than in patients suffering from a functional psychiatric disorder (Corsellis, 1962; Tomlinson, 1968).

A high statistical association between senile plaques and clinical scores was found by Roth and his colleagues. Roth had examined 60 patients before death and the relation to their mean plaque count can be seen in Table 32, page 93.

The same degree of correlation was not found in the present study. Table 28, page 83 shows that all degrees of plaque involvement were found in the 13 patients scoring over 60% on the Ward Behaviour Questionnaire. But when one inspects the incidence of plaques in the lower scoring group it can be seen that six of the nine patients scoring between 40% and 59% had severe plaque involvement with the remaining three patients having nil, mild or moderate plaques respectively. Again the patients with nil and mild plaques included patient 10 who was atypical. There were two patients scoring between 20 and 40% and both had severe plaque involvement. There was a trend therefore in the present study towards an association between the Ward Behaviour score and the incidence of senile plaques, in that the patients who scored lowest had the most plaques. But the correlation was not great enough to be statistically significant.

RELATIONSHIP BETWEEN WARD BEHAVIOUR CHART SCORE (WBC)

AND CEREBRAL PATHOLOGY (ARTERIAL DISEASE)

		Large Vessel Disease				Small Vessel Disease			
Degrees of Damage		0	1	2	3	0	1	2	3
WBC Score	No. of patients								
80+	5	0	5	0	0	0	4	0	1
60+	8	0	5	2	1	0	3	2	3
40+	9	1	7	1	0	2	5	2	0
20+	2	0	2	0	0	0	2	0	0

TABLE 31

from QUANTITATIVE MEASURES OF DEMENTIA AND OF SENILE CHANGE,

Blessed, Tomlinson and Roth (1968)

Diagnostic Groups	No. of cases	Mean Plaque Counts	Mean Dementia Scores
Senile Dementia	26	20·85	13·92
Functional Cases	12	2·75	2·42
Delirious States	14	2·64	2·00
Physically ill subjects	8	5·13	2·25

o = fully preserved capacity
+28 = extreme incapacity.

TABLE 32

As can be seen from the Table 18 (p 59) three patients 60, 98 and 43 scored relatively highly (92%, 88% and 78% respectively) on ward behaviour yet had severe plaque deposition. It thus appears that severe plaque deposition is not necessarily associated with greatest intellectual impairment. On the other hand, the most impaired patients (lowest four) all had severe plaque involvement. If the patients are divided into two groups, it can be shown that whilst there is no correlation between the upper half of the behavioural scores and senile plaques, there is a correlation between the lower half and senile plaques. The numbers in the two groups were too small to place any emphasis on this finding.

The lack of agreement between Roth and the present writer may be due to differences in the techniques of clinical and pathological assessment and/or differences in the sample; Roth's sample contained both men and women.

An important point is that found by Corsellis (1962). Analysis of his figures shows that the severity of plaque formation is not strictly proportional to the severity of atrophy. Since Ward Behaviour correlated very well with atrophy in the present study it would be unlikely also to correlate with senile plaques. There does however appear to be a relationship between the three states of severe atrophy, severe plaque deposition and greater intellectual impairment (see Table 33, (p.95).

To summarise, no positive correlation was found between senile plaques and the degree of clinical impairment. However, it was apparent that whilst severe deposition of plaques could be found in relatively less demented patients, the most demented patients all had severe plaque deposition.

THE RELATIONSHIP OF THE WARD BEHAVIOUR QUESTIONNAIRE TO VASCULAR DISEASE.

There was no apparent relationship between the clinical scores and the degree of vascular disease, i.e. large vessel disease, small vessel

RELATIONSHIP OF WARD BEHAVIOUR SCORE TO CEREBRAL ATROPHY AND SENILE PLAQUES WHERE A = 80% +, B = 60% +, C = 40% +, D = 20% + ON WBQ

	Senile Plaques			
	0	1	2	3
Cerebral Atrophy	A B	B	A AB	AB
	B	BB	BC	A CCCC D
	C	C		CC D

TABLE 33

disease, macroscopic and microscopic infarcts. The findings will be briefly summarised below.

RELATIONSHIP OF THE WARD BEHAVIOUR QUESTIONNAIRE AND LARGE VESSEL DISEASE.

This showed that of the five patients scoring over 80% all had slight atheroma of the large vessels (see Table 31). Eight of the patients scored between 60 and 79% and of these five had slight, two had moderate and one had severe large vessel disease. Nine patients scored between 40 and 59% and of these one (patient 71) had no large vessel disease, seven had slight and one moderate large vessel disease.

Therefore in the group as a whole, only one patient was free from large vessel disease, 19 had slight, three had moderate large vessel disease. Only one patient had severe large vessel disease.

Botton, cited by Corsellis (1962), showed that 90% of patients dying in their sixties or later had some degree of large vessel degeneration. Corsellis himself found that in his functional group, the incidence of large vessel disease increased with age. In his organic division he found that the relative amount of cerebral vascular disease was greater in every age group and the proportions in the moderate and severe sections were constantly higher in the functional division.

The findings in the present study might be a function of age, such a function has been indicated by the two previous workers. The three patients with moderate and the one patient with severe large vessel disease did not have the lowest scores on the Ward Behaviour Questionnaire: 75%, 61%, 44%, 73% respectively. However, because all the patients were clinically deteriorated to some extent one cannot exclude the possibility that large vessel disease may have been responsible in part. It must be emphasised that there is no clear indication that the degree of large vessel disease bore any relation to the degree of deterioration of the patients in this

sample.

RELATIONSHIP OF THE WARD BEHAVIOUR QUESTIONNAIRE AND SMALL VESSEL DISEASE.

Twenty-two of the 24 patients had some degree of small vessel disease. Five patients who scored over 80% on Ward Behaviour included four with slight and one with severe small vessel disease (see Table 29). Eight patients scored between sixty and 79% and of these three had slight, two moderate and three had severe small vessel disease. Nine patients scored between 40 and 59% and of these two had no vascular disease, five had slight and two had moderate small vessel disease.

There is thus no clear relationship between the severity of vascular disease and clinical deterioration. Corsellis' study showed that there was some degree of small vessel disease in each of his diagnostic categories except the affective one.

THE RELATIONSHIP OF THE WARD BEHAVIOUR QUESTIONNAIRE AND CEREBRAL INFARCTS.

There were 11 patients who had infarcts, 8 patients had both macroscopic and microscopic infarcts, one patient had only a macroscopic infarct and two patients had only microscopic infarcts. The range of Ward Behaviour scores in these patients was from 93 to 23 and there was no positive relationship between the degree of damage and Ward Behaviour. The findings can be seen in Table 34, page 98.

The impression gained from the present study is that vascular disease played a small part in influencing the degree of deterioration. Tomlinson (1970) believed arteriosclerotic dementia was over-diagnosed clinically. He wondered whether his method of pathological assessment had underestimated the part played by cerebral softening in producing dementia. However, he considered it certain that the diagnosis of arteriosclerotic dementia was made more frequently on clinical grounds than could be confirmed pathologically.

RELATIONSHIP OF WARD BEHAVIOUR QUESTIONNAIRE AND CEREBRAL INFARCT.

Case No.	WBQ	Infarcts Macro	Micro
73	93	0	1
98	88	0	1
33	84	3	3
43	78	2	3
11	75	1	1
46	73	2	2
3	68	1	1
80	61	3	3
94	57	2	1
61	43	1	1
47	23	1	1

Table 34

Birkett (1972) compared the mental symptoms of two groups of aged hospital patients. One group had senile brain disease but no infarcts and the other had brain infarcts but no positive evidence of senile brain disease. Birkett found they could be differentiated clinically by retrospective examination of their case histories. In the senile group the natural history was from an onset with a variety of psychotic symptoms to progressive confusion and dementia with death from pneumonia. The infarct group had less tendency to confusion and dementia and died from other causes. Neurological features predicted brain infarcts more accurately than did mental features.

Although the cases in the present study were mixed vascular-senile the degree of deterioration bore a closer relation to the degree of senile change than to the degree of vascular disease. This finding does seem to indicate that where a diagnosis of dementia is made the pathological cause is more likely to be senile than vascular.

THE TOOTING BEC QUESTIONNAIRE AND ITS RELATIONSHIP TO CEREBRAL PATHOLOGY.

Introduction.

Memory is traditionally divided into three stages: registration, retention and recall or recognition. It is only the third of these phases which is amenable to direct observation.

The commonly used psychological test procedures such as the Bender Visual Motor Gestalt Test (1938), the PALT (Inglis, 1957) and the Modified Word Learning Test (Walton and Black, 1957) have all been found unsuitable for measuring the performance of severely demented persons. This was because the patients were often dysphasic or mute and could not or would not respond sufficiently to make it possible to score their performances adequately. Blessed et al (1968) devised two simple questionnaires to assess memory and orientation. The one used information about the patient, obtained from an informant, the other contained simple questions which the

patient had to answer verbally. These were the first prospective tests of memory to be compared with a quantitative pathological assessment, i.e. senile plaques. The findings will be discussed elsewhere.

The Tooting Bec Questionnaire was used in the present study. The questionnaire has not been fully standardised or validated. The original workers found that correct answers given on more than nine items excluded the presence of dementia; its presence was confirmed by patients answering eight or less questions correctly.

The patients in the present study were all in the category of dementia scoring under nine. Only one patient had a higher score (patient 14). The test thus appears suitable in revealing demented patients and supports the initial findings.

Because the scores were so low a scoring system was devised to give a shortened version. This was found to correlate with the Ward Behaviour Questionnaire (r = 0.76). This has practical implications; the questionnaire can be administered by a nurse or ancillary staff member after a short briefing and does not require the elaborate organisation necessary for behavioural ratings. It would appear to lend itself to use on a wide scale.

The main problems affecting its validity are that the test is dependent on comprehension and ability to speak as well as willingness to do so. Since it seems that the ability to use words meaningfully is one of the last abilities to go completely, the test may indeed be an accurate, if limited, test of memory and general information.

THE RELATIONSHIP OF THE TOOTING BEC QUESTIONNAIRE TO CEREBRAL ATROPHY.

The Tooting Bec Questionnaire was found to correlate positively with Cerebral Atrophy ($p < 0.05$). The relationship of the mean percentage scores to the pathology can be seen in Table 35, page 102.

The one patient who scored over 80% had no atrophy. One patient

scored between 60 and 79% and he had slight atrophy. There were four patients who scored between 40 and 59% and of these three had slight atrophy and six had moderate atrophy. There were nine patients who scored between five and 20% and of these one had slight atrophy (patient 95), three had moderate atrophy and five had severe atrophy.

As with the comparison of atrophy and behaviour there was a tendency for patients who had lower clinical scores to have more atrophy. Patient 95 who scored 10% yet had only slight atrophy was suffering from two distinct disease processes affecting his brain. (The cerebral haemochromatosis may have been an important factor affecting his performance.

Whilst the association of score to atrophy correlated best at either extremes there were a group of patients scoring between 20 and 59% with mild or moderate atrophy. This suggests that either the questionnaire was not subtle enough to distinguish the patients clinically or that other pathological factors were involved.

THE RELATIONSHIP OF THE TOOTING BEC QUESTIONNAIRE TO NEUROFIBRILLARY TANGLES.

The Tooting Bec Questionnaire was found to correlate positively with neurofibrillary tangles ($p<0.05$). The relationship of the mean percentage scores to the pathology can be seen in Table 35.

One patient scored over 80% and he had a slight degree of tangles. One patient had between 60 and 79% and he had no tangles. Four patients scored between 40 and 59% and of these one had no tangles and three had slight tangles. Nine patients scored between 20 and 39% and of these two had no tangles, five had slight tangles and two had moderate tangles. There were nine patients who scored between five and 19% and of these three had no tangles, two had moderate tangles and four had severe tangles.

In spite of a degree of overlap there was some relationship between the degree of tangles and the scores on the Tooting Bec Questionnaire.

RELATIONSHIP OF TOOTING BEC QUESTIONNAIRE AND CEREBRAL PATHOLOGY

		CEREBRAL ATROPHY				SENILE PLAQUES				NEUROFIBRILLARY TANGLES			
DEGREES OF DAMAGE		0	1	2	3	0	1	2	3	0	1	2	3
TB Score	No. of pts.												
80+	1	1	0	0	0	0	0	1	0	0	1	0	0
60+	1	0	1	0	0	1	0	0	0	1	0	0	0
40+	4	0	2	2	0	0	1	1	2	1	3	0	0
20+	9	0	3	6	0	2	1	3	3	2	5	2	0
0	9	0	1	3	5	1	2	0	6	3	0	2	4

TABLE 35

RELATIONSHIP OF TOOTING BEC QUESTIONNAIRE AND CEREBRAL PATHOLOGY

(ARTERIAL DISEASE)

		LARGE VESSEL DISEASE				SMALL VESSEL DISEASE			
DEGREES OF DAMAGE		0	1	2	3	0	1	2	3
TB Score	No. of pts.								
80+	1	0	1	0	0	0	1	0	0
60+	1	0	1	0	0	0	1	0	0
40+	4	0	4	0	0	0	3	1	0
20+	9	0	6	2	1	0	3	2	4
0	9	1	7	1	0	2	6	1	0

TABLE 36

The results showed that those patients who had the highest scores on the questionnaire, i.e. those scoring over 40% had a smaller number of tangles than those with lower scores. The group of patients scoring under 20% included six patients who had a moderate or severe number of tangles but also three patients without tangles. Two of these three patients (95 and 10) had atypical pathology. However the poor performance of the third patient (94) does not appear to be related to neurofibrillary tangles; he did have severe atrophy.

Investigation of boxers (Corsellis, Bruton, Freeman-Browne, 1973) showed that there was severe neurofibrillary change especially in the antero-medial temporal grey matter of the brain. The clinical histories of these patients revealed a considerable but varied degree of memory impairment. These patients might by any standard be considered to have severe localised involvement. The fact that they were probably not as globally deteriorated clinically as the patients in the present sample, suggests that the site of the tangles as well as the degree of change, influences the total degree of dementia. It would be of interest to assess the degree of tangle involvement in a non-deteriorated group.

THE RELATIONSHIP OF THE TOOTING BEC QUESTIONNAIRE TO THE DEGREE OF SENILE PLAQUES.

The Tooting Bec Questionnaire did not correlate positively with the degree of senile plaques. The relationship of the mean percentage scores to the pathology can be seen in Table 35, (p. 102).

There was one patient scoring over 80% and he had moderate plaques. There were four patients who scored over 40%, one of whom had slight plaques, one moderate and two severe. There were nine patients whose score was between 20 and 39% and of these two had no plaques, one had slight, three had moderate and three severe plaque involvement. It can thus be seen

that whilst the bulk of the patients scoring under 20% had severe plaques, patients with none or slight plaques were atypical. When, however, one looked at the patients scoring between 20 and 39% no clear relationship could be found. As the patients who scored over 80% had moderate plaques, the relation between plaques and the Tooting Bec Questionnaire is not clear.

THE RELATIONSHIP OF THE TOOTING BEC QUESTIONNAIRE AND ARTERIAL DISEASE.

There was no clear relationship between the degree of vascular disease and the scores on the questionnaire. Nineteen of the patients had slight large vessel disease and 14 had slight small vessel disease. Only one patient was free from large vessel disease and two patients from small vessel disease. The relationships can be seen in Table 36.

DISCUSSION OF INDIVIDUAL ITEMS OF WARD BEHAVIOUR QUESTIONNAIRE

SPEECH

Speech present. The relationship between whether a patient spoke and the pathology can be seen in Table 38. There was a positive correlation with cerebral atrophy ($p<.025$). All but six patients spoke on all the occasions they were tested, and it is of interest that even the most deteriorated patient spoke some words 25% of the time that he was examined. Brain (1963) defines mutism as a complete loss of speech in a conscious patient. By this definition none of the patients was mute during the period of investigation and one month before death over 80% of the patients still spoke on the occasion they were examined. See table 37.

Sentences and Words. The relationship to the pathology can be seen in the Table 39 /and 40, page 114. The ability to speak a sentence positively correlated with cerebral atrophy ($p<.025$). One month before death 79% of the patients spoke words sensibly but only 38% spoke a whole sentence. The ability to speak sentences declined noticeably during the last three months before death. It must be stressed that these results tell one whether a patient spoke sentences or words but do not tell one how often.

Relevance. The relationship of relevant speech to pathology shows that those patients with severe atrophy spoke relevantly less than 100% of the time, relevant speech positively correlated with cerebral atrophy ($p<0.01$). One month before death 75% of the patients still said something that was relevant, see fig. 41. This supports the impression that comprehension of spoken speech was present within one month of death.

Sensible speech. The relationship to the pathology can be seen in the Table 42. Relevant speech positively correlated with cerebral atrophy ($p<0.025$). Most of that which was said by 67% of the sample was understandable one month before death. The impression of the nursing staff was that when the patient spoke,

Percentage of Patients performing an Item of the Ward Behaviour Questionnaire one month before death

	ITEM	PERCENTAGE
1.	Contact with staff	96
2.	Speech present	88
3.	Contact with others	87
4.	Spoke words	79
5.	Speech relevant	75
6.	Fed self	71
7.	Most speech sensible	67
8.	Washed	56
9.	Continent of faeces	52
10.	Left chair	50
11.	Lav know-how	45
12.	Contact with patients	42
13.	Spoke sentences	38
14.	Walked unaided	33
15.	Undressed	29
16.	Orientated (partially)	25
17.	Threw/Caught ball	23
18.	Continent of urine	21

TABLE 37

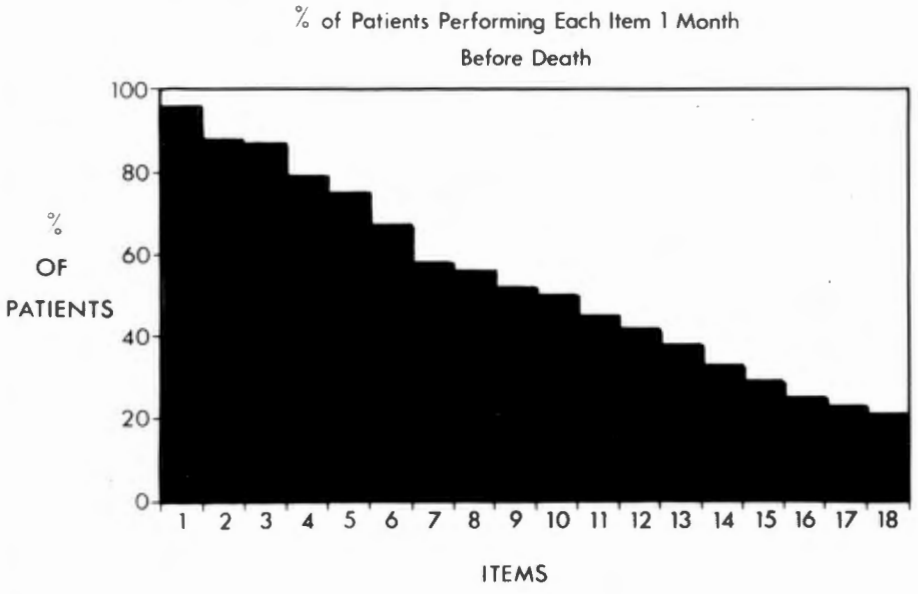

Table 37

(for key to items see page 106)

it was usually understandable, i.e. there were more recognisible words than neologisms or mutterings.

SUMMARY

The patients with less impairment of speech tended to have less brain damage of all types, and significantly less cerebral atrophy. It is likely that factors other than brain damage influence the results. The effect of being in an institutional setting with relatively little social stimulation cannot be ignored.

Some speech remained with all the patients. 35% spoke sentences but in 41% speech was reduced to isolated words during the last month of their life. 75% of the patients spoke relevantly and to the point at least once during the last months of their lives. It may be that these patients who appear oblivious to their surroundings and circumstances are, in fact, more aware than is realised.

SELF-CARE

Feeding. Seventy-one percent of all the patients made some attempt to feed themselves during the last month before death. Because the patients placed food in their mouths one cannot assume that they knew what they were doing in all cases. Several of the patients showed orality, they picked up objects indiscriminately and placed them in their mouths. It is therefore interesting to see, that in spite of the fact that some of the feeding might have been a mechanical primitive action, the patients who fed themselves least often were those who had the greatest brain damage. See Table 43. Feeding positively correlated with cerebral atrophy ($p<0.01$).

Failure to feed may have been due to the fact that the patients did not recognise the food for what it was, that they did not feel hungry (the ravenous patients described elsewhere, Mayer-Gross et al, (1960) were not seen here), or that they were too apathetic to feed.

Washing. The relationship of the patients' attempt to wash to the pathology can be seen in Table 44. Four patients always attempted to do so and four patients never did, the remaining patients attempted to do so on some occasions. There was a trend that the patients who attempted to wash less frequently had more atrophy.

One month before death fifty-six percent of the patients still attempted to wash even though this may have been reduced to patting away at some part of their anatomy with a piece of soap or a flannel.

Dressing. All the patients bar one did not attempt to undress on at least one occasion. One month before death only 25% attempted to do so, see fig. 37. Ten of the patients who never attempted to undress had moderate or severe plaques and tangles indicating that failure to undress is related to brain damage. / See table 45. Because nearly all the patients had impairment in this ability, patients with apparent dementia who do attempt to undress probably have a better prognosis than patients who do not. Ability to undress positively correlated with both plaques and tangles ($p<0.025$).

Urinary and Faecal Incontinence. Incontinence of urine and faeces may be due to organic causes, psychological factors or a combination of the two. Failure of control has been described by Post (1965) and Mayer-Gross et al (1969) as almost always present in the really deteriorated patient. This was certainly the case in the patients in this study. Urinary incontinence has been described as being more common than faecal incontinence and this was borne out by the present results (see Table 37).

The relationship of urinary continence to cerebral pathology can be seen in Table 46. There was a trend for those patients who were never continent to have moderate or severe brain damage of all types. There was a positive statistical correlation with senile plaques ($p<0.025$). One month before death only 21% of the patients were still continent of urine. Because such a large proportion of the patients were incontinent of urine this cannot be used

to prognosticate or compare deteriorated patients. One patient (case 45) who was investigated for a year was already incontinent twelve months before death.

Assessment of faecal incontinence is made difficult in the hospital situation because with regular toiletting 'accidents' can be avoided and a false picture of continence may emerge. In the present sample 52% of the patients were still continent of faeces one month before death, see table 37 (p.106). These findings would suggest that when a patient is clean there is no indication of the imminence of death.

The relationship of faecal incontinence to the cerebral pathology is shown in Table 47. There was a positive correlation with cerebral atrophy ($p<0.05$).

Lavatory Know-how. There was a positive correlation with inability to use the lavatory and cerebral atrophy ($p<0.001$) and senile plaques ($p<0.05$). See table 48.

SOCIABILITY

Ninety-six percent of the patients made contact with the staff within the last month of death. Eighty-seven percent of those who had visitors made some contact with them during this period but only 42% of the patients communicated with one another.

The number of times a patient made contact in relation to the pathology can be seen in Table 49. The patients who always made contact with other patients tended to have less brain damage of all types.

Assessment of communication is difficult and in this case only a minimal contact was considered necessary for the patient to score. The assumption had been that the quite deteriorated patient would be totally inaccessible. In fact there were only two occasions when there was no response at all by the patient to the nursing staff. In all other cases the patient spoke to the staff or looked up at him when addressed and at least appeared aware of his presence.

ORIENTATION

Twenty-five percent of the patients were able to find their way or point correctly to the lavatory, bedroom or bed, within the last month of life. Whilst the findings of the present study are in keeping with the well documented descriptions of the disorientation of the demented patient (Mayer-Gross et al 1969) they do show that some patients retain a minimal spatial ability until death.

One patient was always orientated and he had no cerebral atrophy (see Table 50. Five patients were never orientated and these had a varying degree of atrophy; four of the five had severe plaques. There would appear to be some relation between the degree of brain damage and disorientation for place.

LOCOMOTION

Thirty-three percent of the patients walked unaided during the last month. The reason why some maintain the ability to walk and others do not is unknown. Examination of Table 51 shows that those patients with more brain damage of all types tended to walk unassisted less frequently than those with less damage. Five of the patients with none or slight atrophy and/or tangles always walked without help. There was a positive statistical correlation with inability to walk unaided and the degree of neurofibrillary tangles ($p<0.01$).

The results in this study would suggest that a patient with considerable mental deterioration need not automatically be regarded as chair-bound.

INITIATIVE

Fifty percent of the patients attempted to leave their chair without being asked to do so. There was a trend for those patients who always attempted to leave their chair to have less brain damage than those who never attempted to do so. See table 52.

Whilst this function may have been a measure of restlessness it was also

considered to be one which might indicate how much initiative the patients' still had.

BALL GAME

Twenty-three percent of the patients caught or threw a ball during the last months of their life. The question did not distinguish the more brain damaged from the less, as patients who always caught or threw the ball included those with severe brain damage of all types. / See table 53. It is interesting that even with gross damage, an action as skilful as this procedure could be carried out successfully. (This would indicate that simple activities of this sort can be employed with even the most deteriorated patients to combat their apathy).

SUMMARY

Of the ten items which showed a statistically significant correlation failure to perform six correlated positively with cerebral atrophy and failure to perform one item correlated positively with atrophy and plaques. This suggests that even if cerebral atrophy is not the sole cause of clinical deterioration, it is the most important neuropathological factor.

RELATIONSHIP OF FREQUENCY OF PERFORMANCE OF AN ITEM TO CEREBRAL PATHOLOGY

DEGREES OF:

% Frequency of	ATROPHY *				SENILE PLAQUES				TANGLES			
	0	1	2	3	0	1	2	3	0	1	2	3
Speech Present	100	100	100	100	100	100	100	100	100	100	100	100
		100	100	82	100	100	100	100	100	100	100	100
		100	100	80	100	100	100	100	100	100	100	80
		100	100	75	71	75	100	100	100	100	82	25
		100	100	50	50			100	75	100		
		100	100					100	71	100		
		71	100					100	50	100		
			100					100		100		
			100					82		100		
			100					80				
			25					25				

* = $p < .05$

TABLE 38

RELATIONSHIP OF FREQUENCY OF PERFORMANCE OF AN ITEM TO

CEREBRAL PATHOLOGY

DEGREES OF:

Frequency of	ATROPHY				SENILE PLAQUES				TANGLES			
	0	1	2	3	0	1	2	3	0	1	2	3
entences	87	100	100	50	100	100	100	100	100	100	100	33
		100	100	33	100	50	87	100	100	100	50	0
		100	80	9	55	25	50	100	50	100	9	0
		100	55	0	33	0	0	80	50	100	0	0
		100	50	0	14			50	33	87		
		50	33					33	14	80		
		14	25					9	0	55		
			0					0		25		
			0					0		0		
			0					0				
			0					0				

Table 39

		ATROPHY			SENILE PLAQUES				TANGLES			
ords	100	100	100	75	100	100	100	100	100	100	100	100
		100	100	64	100	100	100	100	100	100	100	50
		100	100	50	100	75	100	100	100	100	100	40
		100	100	40	57	50	100	100	75	100	64	0
		100	100	33	33			100	57	100		
		100	100					100	50	100		
		57	100					100	33	100		
			100					64		100		
			100					50		100		
			50					40				
			0					0				

Table 40

RELATIONSHIP OF FREQUENCY OF PERFORMANCE OF AN ITEM TO CEREBRAL PATHOLOGY

DEGREES OF:

% Frequency of:	ATROPHY ***				SENILE PLAQUES				TANGLES			
Relevance	0	1	2	3	0	1	2	3	0	1	2	3
	100	100	100	75	100	100	100	100	100	100	100	67
		100	100	45	100	100	100	100	100	100	100	0
		100	100	17	100	75	100	100	100	100	45	0
		100	100	0	29	50	100	100	75	100	0	0
		100	100	0	17			100	50	100		
		100	100					67	29	100		
		29	100					45	17	100		
			67					0		100		
			50					0		100		
			0					0				
			0					0				

*** = $p < .01$

Table 41

RELATIONSHIP OF FREQUENCY OF PERFORMANCE OF AN ITEM TO CEREBRAL PATHOLOGY

DEGREES OF:

% Frequency of	ATROPHY **				SENILE PLAQUES				TANGLES			
	0	1	2	3	0	1	2	3	0	1	2	3
Speech-Sensible	100	100	100	75	100	100	100	100	100	100	100	67
		100	100	33	100	100	100	100	100	100	100	0
		100	100	18	100	75	100	100	75	100	18	0
		100	100	0	57	0	0	100	57	100	0	0
		100	100	0	33			100	33	100		
		57	100					67	0	100		
		0	100					18	0	100		
			67					0		100		
			0					0		100		
			0					0				
			0					0				

** = p < ·025

TABLE 42

% Frequency of	ATROPHY ***				SENILE PLAQUES				TANGLES			
	0	1	2	3	0	1	2	3	0	1	2	3
Feeding	100	100	100	87	100	100	100	100	100	100	100	87
		100	100	87	100	100	100	100	100	100	100	75
		100	100	64	93	87	75	100	100	100	64	30
		100	100	64	89	83	50	100	87	100	0	16
		100	89	30	64			87	75	100		
		93	83					75	73	89		
		75	75					64	64	83		
			50					30		50		
			16					16		0		
			0					0				
			0					0				

** = p < ·01

RELATIONSHIP OF FREQUENCY OF PERFORMANCE OF AN ITEM TO CEREBRAL PATHOLOGY

DEGREES OF:

% Frequency of	ATROPHY				SENILE PLAQUES				TANGLES			
	0	1	2	3	0	1	2	3	0	1	2	3
Washing	87	100	100	50	71	67	100	100	100	100	100	25
		100	100	27	67	50	87	100	71	100	33	0
3 DK. Cases	26	71	67	17	17	50	33	100	50	87	27	0
	46											
	73	33	67	0		50	0	33	50	67		0
		0	50	0				27	17	67		
		0	50					25		50		
			33					0		33		
			25					0		0		
			0					0		0		
			0					0				

TABLE 44

% Frequency of	ATROPHY				SENILE PLAQUES **				TANGLES **			
	0	1	2	3	0	1	2	3	0	1	2	3
Dressing (Undressing)	78	50	100	8	55	100	78	87	100	87	16	0
		50	87	0	50	25	25	16	50	78	0	0
		25	55	0	50	16	0	0	50	55	0	0
		16	25	0	8	0	0	0	25	25	0	0
		0	16	0	0			0	8	16		
		0	0					0	0	0		
			0					0	0	0		
			0					0		0		
			0					0				
			0					0				
			0									

** = $p < .025$

TABLE 45

RELATIONSHIP OF FREQUENCY OF PERFORMANCE OF AN ITEM TO CEREBRAL PATHOLOGY

DEGREES OF:

% Frequency of	ATROPHY				SENILE PLAQUES **				TANGLES			
	0	1	2	3	0	1	2	3	0	1	2	3
Urinary Continence	87	100	100	33	100	25	100	100	100	100	0	0
		100	55	0	71	0	87	50	71	100	0	0
		71	25	0	55	0	0	0	33	87	0	0
		50	0	0	33	0	0	0	0	55	0	0
		0	0	0	0			0	0	50		
		0	0					0	0	25		
		0	0					0	0	0		
			0					0		0		
			0					0		0		
			0					0				
			0					0				

** = p < ·025

TABLE 46

	ATROPHY *				SENILE PLAQUES				TANGLES			
Faecal Continence	75	100	100	91	100	33	100	100	100	100	100	50
		100	100	80	89	33	100	100	100	100	91	50
ANA . Case 71		100	89	50	80	25	75	100	80	100	57	50
ADK, Case 61		100	50	50	71	0		91	71	89		50
		71	50	25	0			667	25	75		
		67	33					50	0	33		
		0	33					50	0	33		
			0					50		0		
			0					50				
								0				

* = p < ·05

TABLE 47

RELATIONSHIP OF FREQUENCY OF PERFORMANCE OF AN ITEM TO CEREBRAL PATHOLOGY

DEGREES OF:

% Frequency of		ATROPHY ****			SENILE PLAQUES *				TANGLES			
	0	1	2	3	0	1	2	3	0	1	2	3
Lav. Know-how.	100	100	100	33	100	100	100	100	100	100	100	0
		100	100	25	100	100	100	100	100	100	25	0
IDK. Case 46		100	100	0	44	50	100	100	100	100	0	0
		100	100	0	33	25	100	25	100	100	0	0
		100	50	0				0	33	100		
		100	44					0	25	100		
			25					0		50		
			0					0		44		
			0					0		0		
			0					0				
			0					0				

* = p < ·05
*** = p < ·001

TABLE 48

% Frequency of		ATROPHY			SENILE PLAQUES				TANGLES			
	0	1	2	3	0	1	2	3	0	1	2	3
Social	50	100	100	75	100	100	100	100	100	100	100	100
Communication with Other Patients		100	100	64	100	100	80	75	100	80	75	67
		100	100	17	17	25	50	75	100	60	64	25
With staff - all patients scored 100%		80	75	0	11	0	0	67	100	50	33	0
		50	67	0	0			64	17	50		
		33	60					60	0	25		
		0	25					50	0	11		
			25					33		0		
			11					25		0		
			0					0				
			0					0				

TABLE 49

RELATIONSHIP OF FREQUENCY OF PERFORMANCE OF AN ITEM TO CEREBRAL PATHOLOGY

DEGREES OF:

% Frequency of	ATROPHY				SENILE PLAQUES				TANGLES			
	0	1	2	3	0	1	2	3	0	1	2	3
Orientation	100	67	87	37	67	75	100	87	67	87	0	0
		50	44	17		50	20	50	25	100	0	
7 NA. Cases 46		20	0			50	25	0	75	50		
71												
26		0	50	0	44	37	0	0	50	20		
16												
36		25	50		17			0	37	44		
61												
47								0	17	0		
		75								50		
										0		
			0									
			0									

TABLE 50

DEGREES OF:

% Frequency of	ATROPHY				SENILE PLAQUES				TANGLES			***
	0	1	2	3	0	1	2	3	0	1	2	3
Locomotion - unaided	100	100	100	100	100	100	100	100	100	100	100	67
		100	80	100	100	100	100	100	100	100	37	50
2 NA. Cases 77		100	50	67	71	33	80	80	100	80	0	50
and 46												
		100	44	50	44	33	0	67	100	80	0	33
		80	33	37				50	100	44		
		71	33					50	100	33		
			33					37		33		
			0					33		0		
			0					0				
*** p < ·01			0									

TABLE 51

RELATIONSHIP OF FREQUENCY OF PERFORMANCE OF AN ITEM TO CEREBRAL PATHOLOGY

DEGREES OF:

% Frequency of	ATROPHY				SENILE PLAQUES				TANGLES			
	0	1	2	3	0	1	2	3	0	1	2	3
Initiative:	100	100	100	100	100	100	100	100	100	100	44	67
Leaving Chair		100	80	75	86	75	100	80	100	100	25	50
		86	67	50		33	80	67	100	80	0	25
		80	55	44	67	0	0		86	80	0	0
		67	33	25	55			50	75	55		
		0	25					44	67	33		
			0					25		0		
			0					25		0		
			0					0				
			0					0				
								0				

TABLE 52

% Frequency of	ATROPHY				SENILE PLAQUES				TANGLES			
	0	1	2	3	0	1	2	3	0	1	2	3
Ball Game	100	100	100	100	50	100	100	100	75	100	100	100
		50	100	75	40	75	50	100	50	100	50	50
1 DK. Case	98	50	50	44	29	0	50	100	50	100	44	0
2NA Case	46	50	50	40	0	0	0	50	40	50	0	0
	77	0	29	0				50	0	29		
			0					44	0	0		
			0					0		0		
			0					0				
			0					0				

TABLE 53

DISCUSSION OF ITEMS OF TOOTING BEC QUESTIONNAIRE

The Tooting Bec memory questionnaire relies on a patient's ability to reproduce information acquired in the past and subsequently stored. Performance depends on the subject's level of general intelligence, as well as his inclination to cooperate and his ability to do so by using spoken speech to give the responses (Post, 1965).

In the present study the patient's correct response on at least one occasion was compared with the degree of brain damage. The results showed that incorrect responses correlated positively with cerebral pathology. This method gave patients who were tested more than once a greater chance of giving a correct answer, but was helpful in obtaining a score from the most deteriorated patients and also gave the benefit of the doubt to patients who failed to respond for some reason or another on a particular occasion. It was seen that many more patients could answer the first three questions (see Table 54, /Page 127) than the rest. It appears that past personal events are best retained. All twenty-four patients remembered their surname at least once during the period of investigation. It may be that this is retained because it is one of the earliest pieces of information which is acquired and remains unchanged. It also receives reinforcement every time the patient is addressed on the ward. It would be interesting to observe if women recall their name equally well and whether they would give their maiden or married name. A christian name was rarely given and this might indicate that the patients considered themselves in a formal situation in spite of their deterioration. Only the six most severely demented, remote and inaccessible patients failed to give their name within a month of death (cases 36, 71, 45, 95, 47 and 10). (For the pathology see Table 55). It was surprising to find that even patients with moderate atrophy could retain their name.

Twenty-three of the patients recalled their marital status correctly, at least once. Of course this question did give the patient a fifty percent chance of being right because the response was limited to 'yes' or 'no'.

Sixteen out of twenty-four patients remembered their previous occupation. It was surprising that patients who remembered this every time they were asked included those with moderate atrophy. The patients who never remembered included both patients with slight atrophy and severe atrophy. This indicates that the degree of loss of cerebral tissue alone did not account for the quality of the response.

There was a large drop in the number of patients who answered the first three questions and the remaining seven. The answers which required new learning were less well remembered. Six patients gave their age correctly (Cases 14, 96, 60, 73, 62 and 43). The pathology is shown in Table 55, page 128. Eight out of twenty-four patients (Cases 11, 60, 98, 62, 33, 73 and 14) knew they were in Runwell Hospital or could give the name of the ward but only two patients knew which year it was. The patients may well have heard the words 'Runwell Hospital' or 'Elm Ward' more frequently than the date.

Six patients gave the name of the Queen yet eight patients gave a correct answer to where fighting was taking place or knew that men had landed on the moon. Recall of an address five minutes later was only correctly performed by five patients. The date of the last war was remembered by only two patients.

Cosin et al (1957) found that in the case of personal orientation memory loss followed a fairly constant pattern. The temporal localisation of recent events is the first thing to be lost, followed by the subjects ability to give his own age. Marital state and occupation of self or spouse follow and own name is the last to be lost. The findings of the present study are thus not dissimilar and in addition provide pathological data

which points to a relation between memory and cerebral degeneration.

It has been shown in the present study that all aspects of memory are affected in patients with generalised cerebral degeneration. The mamillary and hippocampal zones and the circuits which link these with the anterior nucleus of the thalamus forming the limbic system are believed to be involved in the ability to register and retain new impressions. (Brierley, 1966; Hoenig et al., 1962; Whitty, 1962) and these areas were certainly affected in the patients in the present study. The relative importance of generalised cortical atrophy and limbic lobe atrophy is yet to be determined and would be a fruitful avenue for further work.

THE RELATION OF LANGUAGE DISORDER TO CEREBRAL PATHOLOGY

If there is a disturbance of memory the assessment of language disorder becomes more complicated. It is partly for this reason that little is known about the type of dysphasia which is seen in senile dementia. Mayer-Gross, Slater and Roth (1960) have described three stages in the breakdown of speech in senility.

1. Naming difficulties and lack of precision.
2. Reduction of speech to simple phrases, in which sounds and propositional forms remain intact, but which contain little meaning. Speech tends to go on endlessly and to be repetitive. Patients can comprehend the general trend of a conversation but miss the details.
3. Comprehensible speech is limited to one or two sensible utterances or to echolalia. There is much repetition of inarticulate material.

Cameron (1938a/ and b) compared the performance of seniles with that of Schizophrenics and children on tests of sentence completion and noted striking differences between the three groups. The failures made by seniles were mainly due to disorientation and circumstantiality.

Williams (1965) believes with other observers that disturbances of articulation, expression, comprehension and of the written language may occur independently of one another and for this reason it was considered worth testing for the different functions even though the pathology was expected to be diffuse.

The present study differed from previous ones as it attempted to determine what residual function remained rather than gauge how impaired the patients were.

Agnosia

All the patients bar one could recognise at least one object. The test

did not show any differences between visual, auditory or tactile agnosia (see p.74).

Receptive Dysphasia

It was interesting that both auditory and visual instruction were equally comprehended.

Apraxia

Although all but three patients were defective in the tasks some measure of function was present in all but two patients.

Expressive Dysphasia

The performance in these tests was poorest for the group as a whole. Here no more than five patients at any one time could perform the tests.

The performance of the eleven patients in the present study was of interest since it revealed that whilst there was evidence of severe dysphasia in at least nine patients, some element of comprehension and speech was present in all but one.

The scores (p. 73) appeared to reflect the scores of the Behavioural questionnaire in that the patients who scored highest on the Ward Behaviour Questionnaire had the higher scores for language function. An exception was patient (case 95) whose low score was indicative of his lack of responsiveness.

Testing was a lengthy procedure but it was rewarding to observe the efforts made by the patients so that a negative score was either a wrong answer or an inability to give a correct response rather than a disinclination to cooperate.

The sample of patients is too small to draw many conclusions but what has been shown is that no matter how deteriorated a patient his performance can be quantified and seems to bear a meaningful relationship to his cerebral pathology.

TO SHOW NUMBER OF PATIENTS WHO RESPONDED CORRECTLY

TO ITEMS ON THE TOOTING BEC QUESTIONNAIRE

	Question	Number of patients answering correctly at least once
1.	Name	24
2.	Status	23
3.	Previous occupation	16
4.	Orientation of place	8
5.	Recent event	8
6.	Age	6
7.	Name of Monarch	6
8.	Recall of an address	5
9.	Orientation in time	2
10.	Date of beginning or end of Second World War	1

TABLE 54

CEREBRAL PATHOLOGY

	Macroscopic			Microscopic				
Case	Cerebral atrophy	Large Vessel Disease	Focal lesions Infarcts	Senile Plaque	Tangles	Micro vasc. disease	Focal lesions	Neuronal loss
73	1	1	0	0	0	1	1	nil
60	2	1	0	3	1	1	0	yes
14	0	1	0	2	1	1	0	yes
98	1	1	0	3	1	1	1	nil
33	1	1	3	2	1	3	3	nil
43	1	1	2	3	2	2	3	yes
11	2	2	1	0	1	1	1	yes
96	1	1	0	2	0	1	0	yes
46	1	3	2	0	0	3	2	0
3	2	1	1	2	1	3	1	yes
95	1	1	0	1	0	1	0	yes
62	2	1	0	1	1	2	0	yes
80	2	2	3	1	0	3	3	yes
94	3	1	2	1	0	2	1	yes
71	2	0	0	3	2	0	0	yes
10	3	1	0	0	0	0	0	0
26	2	1	0	3	2	2	0	yes
16	2	1	0	3	3	1	0	yes
36	3	2	0	3	3	1	0	yes
61	2	1	1	2	1	1	1	yes
77	2	1	0	3	1	1	0	yes
45	3	1	0	3	2	1	0	yes
86	2	1	0	3	3	1	0	yes
47	3	1	1	3	3	1	1	yes

TABLE 55

THE RELATIONSHIP OF CEREBRAL DEGENERATIVE CHANGE TO CLINICAL DETERIORATION.

Evaluation of Results

As the relationship of cerebral atrophy, neurofibrillary tangles and senile plaques to clinical deterioration have been discussed individually it does not mean that they act separately. In fact it is possible that there is a summation of effect. This is supported by evidence from the present study that the most clinically deteriorated patients had the most senile degenerative change of all types.

However, the various pathological factory do not appear to carry the same weight. It seems that at the present time cerebral atrophy reflects best the unknown factors which cause mental deterioration.

Although the present study shows no positive correlation between vascular disease and clinical deterioration, the influence of the former on the latter cannot be entirely ruled out since the patients were all deteriorated to some degree. It does seem that arteriosclerosis has been over-diagnosed as the cause of dementia in the past.

Because the numbers in the present study are small, more definite conclusions cannot be drawn at this stage. Yet in the present sample, the finding that cerebral atrophy with loss of brain substance is fundamental to clinical deterioration, has emerged very clearly.

CONCLUSION AND SUMMARY

A prospective study of male patients on a psycho-geriatric ward was carried out by making repeated quantitative clinical assessments. Twenty-four patients came to post-mortem and quantitative pathological examinations of their brains were made after the brains had been given code numbers. The clinical and pathological results were then compared.

The Ward Behaviour Questionnaire included questions about the patient's most basic functions and the simplicity of the questions made them suitable for even the most deteriorated patient.

The Tooting Bec Questionnaire tested the patient's memory, orientation and general information. The questions were easily administered by even the most junior nurse and the patient's replies documented. As with the ward behaviour questionnaire, the repeated administration of the questionnaires enabled the most deteriorated patient to score.

The physical examinations showed that the patients were not hypertensive, that they had marked muscle wasting and variable non-localising neurological signs. The presence of grasp and sucking reflexes in the most deteriorated patients were found to be associated with severe brain damage.

Examination confirmed that assessment of the mental state provided little information to distinguish the patients meaningfully from one another: they were too deteriorated.

The tests of language function produced a range of results which paralleled the patients scores on the ward behaviour questionnaire. The sample was small but the findings suggest that this method could be developed in further studies.

The psychological tests were found to tax the patient's co-operation and require modification before further use. They did, however, make it clear that conventional psychological tests are inappropriate for severely deteriorated patients.

The results showed that most patients had achieved a plateau of deterioration some six months before death. The residual functions one month before death showed that eighty-eight percent of the patients could still say a few words, seventy-one percent made some attempt to feed themselves. Fifty-two percent were continent of faeces but only twenty-one percent were continent of urine. Thirty-three percent walked unaided and twenty-five percent showed some orientation on the ward. The fact that the staff could still establish contact with ninety-six percent of the patients indicates that even in severe dementia the patient is not continuously inaccessible.

The pathological assessment was done by inspection using the criteria employed by Corsellis (1962) and all except one of the brains had cerebral atrophy and all except one large vessel disease. Microscopically seventeen patients had some degree of neurofibrillary tangles, and all but four had some degree of senile plaques. All but two patients had some degree of small vessel disease and eleven patients had macroscopic or microscopic infarcts.

The clinico-pathological comparisons revealed that performance on the Ward Behaviour Questionnaire and Tooting Bec Questionnaire correlated with the degree of atrophy ($p < 0{\cdot}001$ and $p < 0{\cdot}05$ respectively) and the degree of neurofibrillary tangles ($p < 0{\cdot}05$) in both cases. There was no statistical correlation with senile plaques.

There was no correlation between the clinical scores and the degree of vascular disease and this suggests that the role of vascular disease, as a cause of mental deterioration, may have been over-emphasised.

It has been shown that the degree of clinical deterioration is best reflected by the degree of cerebral atrophy. The precise nature of the tissue lost is not known and awaits further study.

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APPENDIX I

Case No. 3

Male aged 81 Admitted 25.5.69 Died 9.1.70

Family History

Unknown

Past History

Unknown

History of Final Illness

Transferred from another hospital where he had been for a year. On admission he was suffering from grandiose delusions. During his stay his mental and physical condition gradually deteriorated. He appeared to have several cerebrovascular episodes, which were followed by increasing left-sided weakness. BP 160/90. He deteriorated progressively, developed 'flu, followed by pneumonia and died five months later.

Mental State on Examination

A thin, old man, wheeled into interview. Left hemiplegia. Comprehension appeared good, but replies limited to short sentences. Denied depression but appeared sad. No ideas of worthlessness, guilt or suicide. Disorientated in time and place, thinking he was a visitor in a hotel. No hallucinations.

Diagnosis

Arteriosclerotic dementia.

Cause of Death

Bronchopneumonia.

Post Mortem Findings

Nervous System

Brain weight after fixation 1330g.

Macroscopic Appearance: Normal size brain. Moderate widening of sulci over both convexities, least marked parieto-occipitally. Central gyri above the lateral fissure softened and necrotic (2cm diameter). Vessels at base, slight patchy atheroma. Moderate enlargement of lateral ventricles, right slightly more than left. Slight thinning of cortical ribbon. Slight widening of sulci in posterior frontal regions and around insula. On the right side some softening of cortex and white matter above the lateral fissure, which tracks back along the parietal convexity almost to the occipital pole. It involves the middle third of the parietal lobe. Slight atrophy of thalamus. The amygdaloid nuclei and hippocampi a little atrophic.
Substantia nigra, average on the right but reduced pigmentation on the left.

Microscopic Appearance: There were a moderate number of plaques and slight degree of tangles throughout the cortex. There was mild cell loss and gliosis.

The left occipital section has numerous small holes. There was an old infarct between the claustrum and insular cortex on the left. The white matter generally showed demyelination and astrocytic gliosis. The small vessels were fibrosed with greatly enlarged perivascular spaces in some areas.

Other Viscera

Respiratory System: Trachea slight inflamed but the large and small bronchi were acutely inflamed and showed typical plum-coloured appearance seen in viral infections. Lungs - both showed lower lobe bronchopneumonia. There was also some slight pulmonary oedema in both upper lobes.

Cardiovascular System: There was considerable hypertrophy of the left ventricular wall. Coronary vessels showed severe atheromatous change, no evidence of myocardial infarction. Myocardium generally fibrotic. Aorta - extreme atheromatous change and there was a small dissecting aneurysm of its abdominal part.

Case No. 10

Male aged 64 Admitted 2.3.70 Died 7.1.70

Family History

Nil known.

Past History

Fractured right femur 1965. Prostatectomy 1967. Benign myodenosis 1967. Then developed diabetes.

Admitted to general hospital 1968 where diagnosis of hypertension and presenile dementia was made. Unable to look after himself. Deteriorated progressively and was admitted to hospital. On admission a little restless, grossly dysphasic and appeared to have difficulty in understanding. Incontinent, poor memory. BP 170/90. Diabetic. He deteriorated progressively, losing two stone within six months of admission. He was totally disorientated, doubly incontinent. He then became pyrexial and died a week later.

Mental State on Examination

A thin, neatly dressed man. Shook an outstretched hand with his left. Gazed fixedly at interviewer. Frequently dysphasic, many of his remarks a simple reiteration of the interviewer's questions. Not depressed, or hallucinated. Deluded and disorientated in time, but not for place. Smiled several times with an element of hopelessness. Eyes filled with tears at one point. Mood inappropriate, laughed when asked what was the trouble.

Diagnosis

Organic dementia. Dysphasia.

Cause of Death

Bilateral bronchopneumonia.

Post Mortem Findings

Very thin, elderly man.

Nervous System

Brain weight after fixation: 1340g.
Macroscopic Appearance: Normal size brain. Leptomeninges thickened over both convexities. Brownish in colour with poles severely atrophic. Granular texture; severely atrophic at both poles. Left convexity more severe than right. Temporal lobes, parietal and occipital lobes and posterior frontal lobes relatively spared. Severe enlargement of lateral ventricles particularly marked in anterior horns. Septum considerably stretched but not perforated. Third ventricle moderately widened, measuring up to 6mm maximum. Cortical ribbon considerably narrowed in both convexities, left more than right. White matter not well demarcated from the cortex in the severely atrophic areas. The corpus callosum slightly narrowed. Deep grey matter showed no focal lesion, but striatum appears a little atrophic. Head of caudate shrunken. Thalamus a little atrophic. Aqueduct, mid-brain slightly dilated.
Cerebellum;unusually deep grooves around tonsils.
No other evidence of brain swelling, presumably an anatomical variant.

Microscopic Appearance: The cortex was very porous and showed disorganisation of the neurons and marked depopulation. There was severe cortical gliosis. The white matter was porous. There were frequent widened perivascular spaces.
The small vessels themselves showed slight fibrosis.

Diagnosis: Pick's disease.

Other Viscera

Respiratory System: Bronchi filled with pus. Patchy consolidation of lower lobes and considerable pulmonary oedema.

Case No. 11

Male aged 83 Admitted 15.9.69 Died 17.10.70

Family History

Nil known.

Past History

Nil known.

History of Final Illness

He became increasingly forgetful, difficult to manage and incontinent at night over the two years preceding admission. For three months before admission he attended as a day patient.

On admission he was a frail, weak, old man with a shuffling gait. BP 140/90. He became progressively more infirm, falling occasionally, requiring full nursing care. He developed broncho-pneumonia and died.

Mental State on Examination

A cleanly dressed, round-faced man, looking younger than his years. Walked with assistance. He smiled and shook an outstretched hand in greeting. Throughout the interview he sat quietly, occasionally passing a hand over his face. He maintained eye to eye contact, and returned a smile, although his face was otherwise expressionless. His speech was mainly limited to monosyllabic responses and comments on the difficulty of the questions, and he was unable to give a proper account of himself. His mood appeared bland and cheerful, at times puzzled. When contradictions in his replies were pointed out to him he looked unconcerned.

Diagnosis

Organic dementia.

Cause of Death

Bilateral broncho-pneumonia.

Post Mortem Findings

Nervous System

Brain weight after fixation 1380g.

Macroscopic Appearance: Normal size brain. Slight generalised widening of sulci over both convexities. Right temporal pole considerably more atrophic than rest of brain and more so than left temporal pole. Atrophy of right uncus greater than those of left. Vessels at base terribly tortuous and moderate patchy atheroma most marked around both carotids, walls of which are 1-2mm thick but luminae fully patent. Moderate enlargement of the lateral and third ventricles, left larger than right, except for right inferior horn which is exceptionally dilated. Cortical ribbon round right temporal pole narrow. There is a softening in the white parietal white matter dorso-lateral to the posterior horn some 1 x 0.5cm. This is probably an old vascular lesion and there are other patchy areas of white matter necrosis

scattered through the hemispheres. Hippocampi on right a little smaller than left. Aqueduct slightly dilated. Substantia nigra average pigmentation.

Microscopic Appearance: The cortex in the anterior and posterior temporal regions, especially in the right, appeared spongy but in other areas the neuronal population was good. There was a moderate degree of fibrosis in the cortex. There was severe white matter gliosis throughout the brain. There was an old infarct in the white matter of the right frontal region. There was sub-pial fibrosis and the vessels were mildly fibrosed. The lateral nuclei of the right thalamus was porous. The vessels in the mid-brain and cerebellum were slightly fibrosed with widened perivascular spaces.

Case No. 14

Male aged 76 Admitted 12.6.69 Died 26.10.70

Family History

Nil known.

Past History

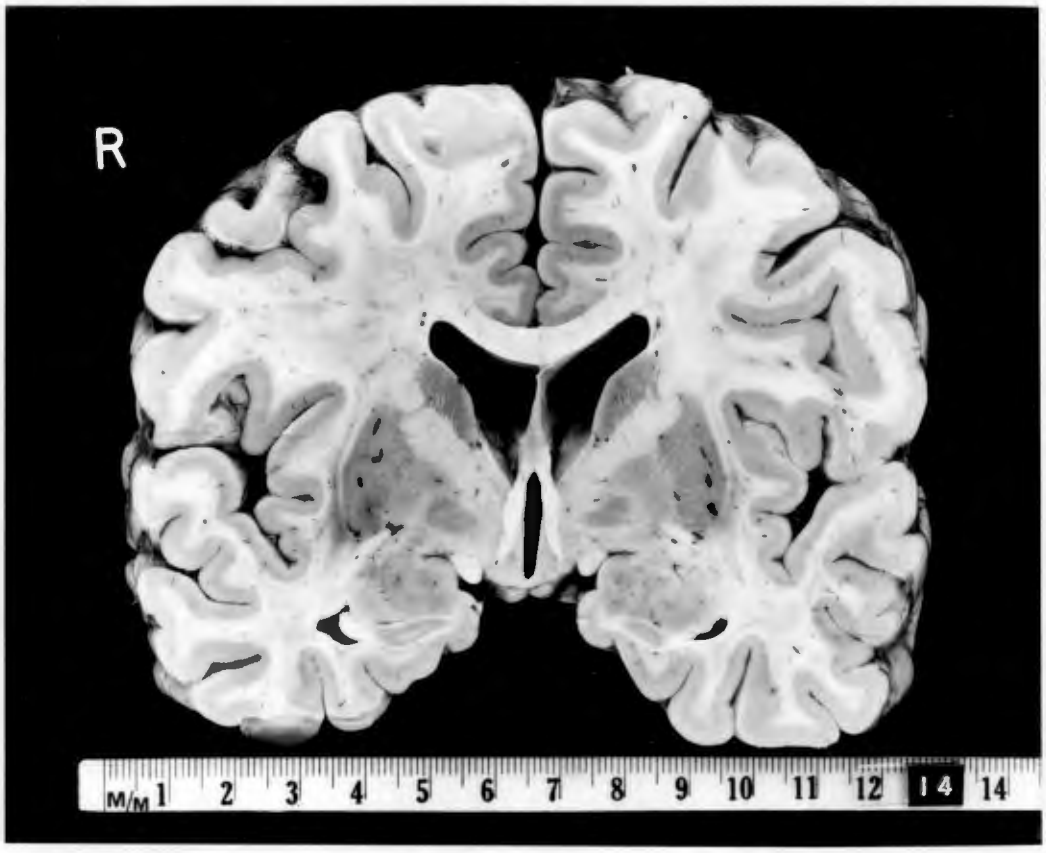
1914 War - ? shell shock, but no actual indication of intellectual deterioration. In 1953 he was admitted to hospital for investigation of abdominal pain, but he was so aggressive and agitated that the operation was postponed and he was discharged home. His behaviour continued to be aggressive and argumentative and on one occasion he insisted that all the lights be switched off because they caused him abdominal pain. He was readmitted to hospital the same year when a stricture of the oesophagus was noted. His mental state had features suggestive of an organic disorder but there was no measureable intellectual deterioration. He was agitated and hypomanic. On the 22.10.64 he was readmitted to hospital after slashing his left wrist and causing ulnar palsy. He was agitated and depressed and he was readmitted to hospital in 1966, 1967 and 1968, on each occasion complaining of epigastric pain and depression, with disturbances of appetite and sleep. He had lost weight. He was treated with ECT. In 1968, however, an EEG was generally abnormally suggestive of a degenerative process. He was hospitalised again in April 1969 and was finally admitted on 12.6.69.

History of Final Illness

It is difficult to date the onset of the final illness, but during the seven years preceding his death he was in and out of hospital, being treated for depression and abdominal pain. There was no evidence of intellectual deterioration until an EEG done in 1968. Finally admitted 12.6.69 because his wife was unable to look after him. He had a few falls whilst in hospital, becoming gradually more feeble, developed shortness of breath and died.

Mental State on Examination

A thin, neatly dressed, polite man with severe arthritis in his hands. Good rapport was established. Admitted to feeling very depressed with suicidal ideas but no feelings of self guilt or blame. He was preoccupied with his bowels and



Case 14

Coronal section at level of intraventricular foramina showing non-atrophied brain.

ulcer. He expressed paranoid ideas about the surgeon who had refused to operate on him seven years previously. He was correctly orientated in time and place, but had little insight, thinking his trouble was due to his bowels.

Diagnosis

Depressive illness with marked anxiety.

Cause of Death

Inhalation of vomit and bilateral bronchopneumonia, oesophageal stricture, and hiatus hernia.

Post Mortem Findings

Left paramedian abdominal operation scar.

Nervous System

Brain weight after fixation 1320g.

Macroscopic appearance: Normal size brain. Rather prominent sulci, not materially widened, gyri look well-formed but not atrophic. Some tortuousity of vessels at base. Very slight flecks of atheroma. Coronal cuts show enlargement of both lateral ventricles but brain in all other respects appears normal. Cortical ribbon normal thickness. No other macroscopic abnormality.

Microscopic appearance: There was a moderate degree of senile plaques and a slight degree of neuronal tangles in the frontal parietal, temporal and hippocampal regions. There were no tangles in the occipital cortex. There was evidence of neuronal disorganisation and slight neuronal loss. There was slight gliosis of the cortex. The white matter was slightly porous throughout the brain, but especially in the anterior temporal regions. There was slight fibrosis of the small vessels. There were plaques in the amygdaloid and small softening of the right thalamus.

Other Viscera

Respiratory system: Larynx and trachea occluded by partially digested food. Lungs both showed considerable pulmonary oedema and lower lobe consolidation. Pus exuded from the cut ends of the small bronchi and bronchioles. Myocardium and valves normal. Coronary vessels showed minimal atheroma. Aorta showed moderate atheromatous change.

Adrenals: Small adenoma (1cm diameter) of left adrenal.

Case No. 16

Male aged 72 Admitted 22.11.68. Died 8.2.70

Family History

Nephew had mental illness.

Past History

Lost left eye in first World War.

History of Final Illness

Six years after retirement became gradually forgetful. Had to be washed and dressed by wife. Sat in chair most of day and slept. Became incontinent of urine and was wandering in streets inadequately dressed before his admission.

On admission he was an elderly man who was unable to give an account of himself. He was totally disorientated in time and place and person, and incontinent of urine. He was deluded thinking he was still fighting the Germans. He deteriorated gradually becoming incontinent of faeces. Two months before death he developed 'flu followed by pneumonia and died.

Mental State on Examination

An elderly man who walked to the interview and sat quietly throughout. There was no spontaneous speech and replies were monosyllabic. He denied depression and no delusions or hallucinations were elicited. He was disorientated for time, place and person, could not remember an address after two minutes. No insight.

Clinical Diagnosis

Organic Dementia.

Cause of Death

Bilateral broncho-pneumonia.

Post Mortem Findings

Elderly well nourished man. Several decubitus ulcers on left and right buttocks. Tattoos on both arms. Atrophy left optic nerve.

Nervous System

Macroscopic appearance: Normal size brain. Very marked widening of sulci at both frontal poles spreading back to precentral gyrus, sparing posterior half frontal lobe more than anterior. Antero-lateral surface temporal lobe markedly atrophic. Parietal-occipital areas relatively spared. Basal vessels slightly tortuous. Slight patchy atheroma.

On stripping leptomeninges from left hemisphere cortical atrophy seen to be severe, particularly left F1 and F2 and antero-lateral part of temporal lobe. Cortex here particularly roughened and frontally has brownish colour.

Moderate enlargement of lateral ventricles, slight of third. Considerable atrophy around insula of both sides. Cortical ribbon in temporal lobe narrowed. White matter - slight suggestion of porosity. Thalami a little small.

Amygdaloid nuclei and hippocampi a little atrophied but medial temporal parts of temporal lobe less affected than more laterally.

Mid-brain - aqueduct moderately dilated 2-3mm across.

Cerebellum - small area folial necrosis postero-ventral to dentate nucleus some 4mm across in right hemisphere.

Diagnosis: Moderate atrophy.

Microscopical Appearance: Fibrosis of small and medium sized vessels of meninges. Some congophilic.

Cortex - severe senile plaques and tangles throughout. Marked cell loss. Fibrosis small vessels. Some vessels congophilic with perivascular plaques.

White matter - astrocytic gliosis of both frontal and occipital lobes. Widened perivascular spaces especially in right occipital lobes.

Amygdaloid - numerous plaques, tangles and congophilic vessels in right amygdaloid, none in left.

Basal ganglia; tangles in right globus pallidus.

Hypothalamus - few plaques left mamillary body.

Cerebellum - loss of Purkinje cells maximally at base of sulci. Few plaques in superficial and deeper layers of cortex. Several congophilic vessels with perivascular plaques.

Other Viscera

Lungs - pulmonary oedema, broncho-pneumonia both lower lobes.

Case No. 26

Male aged 73 Admitted 22.7.69 Died 15.2.70

Family History

Nil relevant.

Past History

Not known.

History of Final Illness

Developed depressive illness 6 years before admission. Then became progressively more forgetful failing to recognise his wife and home. He attended a day hospital for four years but became too agressive and difficult to manage.

On admission to hospital he was very confused and disorientated. Left homonymous hemianopia, B.P. 170/90. He deteriorated progressively and had to be washed, dressed and fed. He became incontinent of urine and faeces. His weight decreased from 16 stone to 9 stone over the seven months before he died suddenly.

Mental State on Examination

A tall, thin man wheeled into interview. He shook hands but offered no spontaneous speech. Replies limited to a few words. Unable to give proper account of himself. Admitted to depression "I don't know why". Completely disorientated in time, place and person.

Clinical Diagnosis

Organic Dementia.

Cause of Death

Left ventricular failure.
Haemorrhage into dissecting aneurysm of aorta.

Post Mortem Findings

Very tall (6'6") poorly nourished man.

Nervous System

Macroscopic Appearance: Normal sized brain (weight after fixation 1360g). Moderate widening of sulci over both convexities.
Vessels at base somewhat tortuous with a few flecks of atheroma.
Slight enlargement of lateral ventricles, third ventricle slightly widened. Cortical ribbon slightly narrowed especially in medial temporal region.

Microscopic Appearance: Fibrosis of large and small meningeal vessels - some congophilic. Severe plaques and tangles throughout cortex; many plaques congophilic. Scattered areas of porosity, most marked in occipital region, associated with astrocytosis. Neuronal loss, especially in hippocampus.
White matter showed demyelination.
Putamen porous.
Moderate small vessel disease throughout white matter. Some vessels fibrosed in amygdaloid and putamen.
Brainstem - fibrosis of overlying meninges.

Other Viscera

Respiratory system: Lungs.
Macroscopic appearance - gross pulmonary oedema in lower lobes. Apex of upper lobe consolidated having the appearance of a tuberculous lesion but no cavitation or pus visible. Apex of left lobe adherent to pleura.
Microscopic appearance - normal architecture replaced by fibrous material in which can be found mitotic cells and some macrophages. Tubercle bacilli not seen.

Cardiovascular System. Coronary vessels show minimal atheromatous change. Minimal atheroma of ascending and arch of aorta; abdominal aorta has severe atheromatous change. Bifurcation of aorta has a dissecting aneurysm which contained about a pint of recent blood clot.

Case No. 33

Male aged 74 Admitted 7.5.69 Died 29.2.70

Family History

Father died aged 64 of pneumonia. Mother died age 84 of 'old age'.

Past History

Born in England. Went to Australia at age 15. Worked as book-keeper and store-keeper until first World War. Developed trench feet during war; multiple

terminal phalanges affected - big toe amputated. Tuberculosis after war and was given 100 percent disability pension and stopped working. Married twice.

Admitted Runwell Hospital when aged 63 years (16.5.61). Had become irritable, worried by noise, easily awakened, loss of appetite and weight. Repeated episodes of left-sided incoordination and stiffness and numbness - four minutes duration - treated phenobarbiturates. Diagnosis of anxiety reaction in a schizoid personality made. His depression and appetite gradually improved and he was discharged.

History of Final Illness

Readmitted because of increasing confusion and restlessless (7.6.69). On admission very talkative - sometimes hostile, sometimes apologetic. BP 170/80. Poor reflexes right limb, left hemianopia, some loss of sensation left leg.

Mildly dysphasic, slightly paranoid. Memory for time and recent events reasonably intact. Orientated for place, but lost way about ward.

One month after admission had a haematemesis- hiatus hernia diagnosed after investigation. Three months after admission noted to be disorientated with left hemianopia and left apraxia. Two months before death developed chest infection. Became over-talkative, completely disorientated and abusive. Developed right pneumothorax. Transferred to general hospital for aspiration but died two days later.

Mental State on Examination

Tall, untidy, scruffy looking man who was restless throughout interview. Kept jumping up saying he had to go for a little walk. Speech showed pressure of talk and though coherent it was mainly irrelevent.

He was irritable and mildly depressed. He believed himself to be at work and would be returning home that evening. He then asked for envelopes so that he could write to his wife. Spoke about being in Australia and was worried about his financial affairs and the condition of his feet. He was disorientated in time.

Admitted to hearing voices, often threating, occasionally asking him questions, usually that of a friend. Admitted to visual hallucinations of people and animals 'mostly fish and birds in colour'. He seemed to confabulate. When asked if there were animals in the corner of the room he said 'yes', and that they had sharp beaks. No insight.

Diagnosis

Organic Dementia.

Cause of Death

Broncho-pneumonia; spontaneous pneumothorax. Ruptured emphysematous bullae. Right middle cerebral infarction.

Post Mortem Findings

Moderately nourished old man; left paramedian operation scar. Big and little toes absent right foot. Puncture mark third rib interspace anteriorly.

Nervous System

Brain weight after fixation: 1240g.

Macroscopic Appearance: Normal size brain, slightly atrophic. Slight patchy atheroma of basal vessels. Cortex and white matter of right temporal pole and fusiform gyrus softened, necrotic; adjacent tissue scarred.

Middle cerebral infarction appears mainly restricted to below lateral fissure. Frontal lobes do not appear affected. No gross other abnormality of cortex or white matter. Lateral ventricles moderately dilated. Posteriorly infarction spreads back almost to occipital pole on lower half of convexity.

Summary: slight atrophy, slight vascular disease, right middle cerebral infarction.

Microscopic Appearance: Right anterior temporal region missing - right hippocampus present partially but no cortex due to effect of right middle cerebral infarction.

The cortex showed cell loss in the hippocampal region. There was scattered gliosis. Moderate numbers of plaques were seen in all cortical areas and slight tangles in the left temporal region, insula and fusiform gyrus.

Right hippocampus and fusiform gyrus partially missing due to patchy infarction which also involved the parahippocampal region.

Right occipital cortex contained at least six small infarcts which were slit-shaped, round or irregular in shape and contained foam cells and altered blood.

Subarachnoid haemorrhage over right occipital cortex.

White matter gliosed and there were several small holes and perivascular spaces in the frontal, temporal and occipital lobes.

Some of the cortical infarcts in the occipital area extended into the white matter.

Vessels throughout the brain were severely fibrosed.

Thalamus - very porous and riddled with enlarged, perivascular spaces as was the putamen.

Right amygdaloid contained plaques and a few tangles.

Brainstem - necrosis of one cerebral peduncle and one corpora quadrigemina.

Cerebellum showed loss of Purkinje cells, small vessels were fibrosed.

Other Viscera

Respiratory System: Partial expansion of right lung. Gross chronic bronchitis and emphysema with many bullae in both lungs. Right basal broncho-pneumonia.

Cardiovascular System: Small septal scar in myocardium. Moderate coronary atheroma.

Digestive System: Many pigment stones in the gallbladder. Small bowel firmly adherent to the anterior abdominal wall at the site of the operation scar. A little blood in the stomach from terminal erosions.

Case no. 36

Male aged 77 Admitted 15.4.69. Died 19.1.70

Family History

Father and two sisters all deteriorated and became confused with increasing age.

Past History

Senior civil servant in Housing section until retirement at 65 years. Wilful, always had a bad temper. Smoked 1 oz. tobacco a day. No alcohol. Interests - gardening.

History of Final Illness

Two years before admission had tonsillitis. Deteriorated after this. Gave up reading newspapers, could not concentrate on television, unable to shop - could not recognise money values. Became afraid to be alone. Awoke at night telling wife there were people in the room. Could not wash or shave himself - gradually more unco-operative. Unable to walk unaided for nine months preceding admission.

On admission he had a cataract of the left eye. There was marked peripheral wasting of all four limbs and his jerks were sluggish. He was incontinent of urine. Appeared to understand speech but his own was difficult to comprehend. BP 120/90. Subsequent to admission he became doubly incontinent, could only walk with assistance, fed himself slowly and messily. He suddenly collapsed, showed Cheyne-Stokes breathing and died four days later.

Mental State on Examination

He sat hunched up throughout the interview and displayed no change of facial expression. He was unable to answer any questions except by making a humming noise. He was,however, able to smoke a cigarette and to push away a proffered hand.

Diagnosis

Organic Dementia.

Cause of Death

Suppurative bronchiolitis. Dementia.

Post Mortem Findings

Elderly, well-nourished man.

Nervous System

Brain weight after fixation 1160g.

Macroscopic Appearance: Small brain - severe generalised atrophy. Tortuous vessels at base with slight patchy atheroma. Moderately severe dilatation of ventricles. Few cysts (1mm diameter in both putamina).

Microscopic Appearance: Severe plaques, severe tangles, moderate congophilic angiopathy. Slight small vessel fibrosis, slight gliosis, seen throughout brain. Depopulation of cells in hippocampus.

Other Viscera

Respiratory System: Lungs - complete consolidation of lower lobes by broncho-pneumonia. Pulmonary oedema upper lobes. Microscopic - pneumonia. Liver - microscopic appearance: centrilobular necrosis and congestion.

Case No. 43

Male aged 72 Admitted 23.7.69 Died 10.1.70

Family History

Nil known.

Past History

Nil relevant.

History of Final Illness

He was transferred from a general hospital where he had been admitted six weeks previously with a diagnosis of arteriosclerotic dementia. He had become increasingly forgetful, confused and incapable of caring for himself. On admission he was confused, disorientated with poor concentration and slurred speech. He was incontinent of urine. One month after admission he had a grand mal fit. Two months later he fell, sustained a Colle's fracture of the right arm. He developed 'flu five weeks later and died after a fortnight.

Mental State on Examination

An elderly, thin man who was disorientated in time and place. His replies were mainly monosyllabic. He said he was the son of a fisherman and had a happy life. He denied depression and no delusions were elicited though he could easily be led to confabulate. He was not hallucinated.

Diagnosis

Organic Dementia.

Cause of Death

Pneumonia.

Post Mortem Findings

Fairly well-nourished elderly man.

Nervous System

Brain weight after fixation 1495g.

Macroscopic appearance: Minimal widening sulci frontally. Slight patchy atheroma basal vessels. Lateral ventricles moderately dilated; normal cortical ribbon. Small cysts in white matter posterior half. Shrunken left hippocampus. Summary - slight atrophy, slight vascular disease, plus multi-focal white matter softenings.

Microscopic appearance: Severe plaques, moderate tangles throughout cortex. Severe congophilic angiopathy. Moderate small vessel disease. White matter contained scattered old focal infarcts in frontal, anterior temporal, hippocampal and occipital regions. Infarcts associated with severe gliosis. Plaques in cerebellum. Midbrain and pons, few areas of softening 1-2mm diameter.

Other Viscera

Cardiovascular system: Heart weight 335g. Severe atheroma of coronary vessels. Old myocardial infarct posterior wall left ventricle. No recent infarct.

Aorta - main arteries - moderate atheroma.
Microscopic appearance - heart muscle showed necrosis and some fibrosis.

Respiratory system: Bronchi inflamed with pus. Consolidation of lower lobes with inflammatory reaction. Spleen, congested with haemorrhage.

Case No. 45

Male aged 64 Admitted 8.7.68 Died 3.9.70

Family History

Sister died after cerebrovascular accident.

Past History

Merchant seaman from 14 years old. Heavy drinker since early life.

History of Final Illness

Personality deteriorated over 10 years preceding admission. On admission: confused, restless, unable to give any account of himself; laughs and giggles; completely disorientated. BP 130/80.

He deteriorated progressively and after one year was incontinent of urine and faeces. He had a series of urinary and respiratory infections, the final one being one month before his death.

Mental State on Examination

Patient appeared puzzled at times during interview. Responses elicited after repeated (4 to 8 times) questions. Answered correctly to his name, said he was fine. Some questions brought forth long, totally incoherent monologues sounding like a foreign language (jargon dysphasia). He was cheerful, laughed and tried to involve interviewer in his jokes. He placed one of his fingers on his cheek with his other hand and then slid the finger into his mouth and tried to chew it as though it were a foreign object. He shook a proferred hand and drank a cup of tea. No delusions, hallucinations or obsessional phenomena elicited; attention and concentration good. He appeared to listen intently, when asked if ill, said "No, fine, fine".

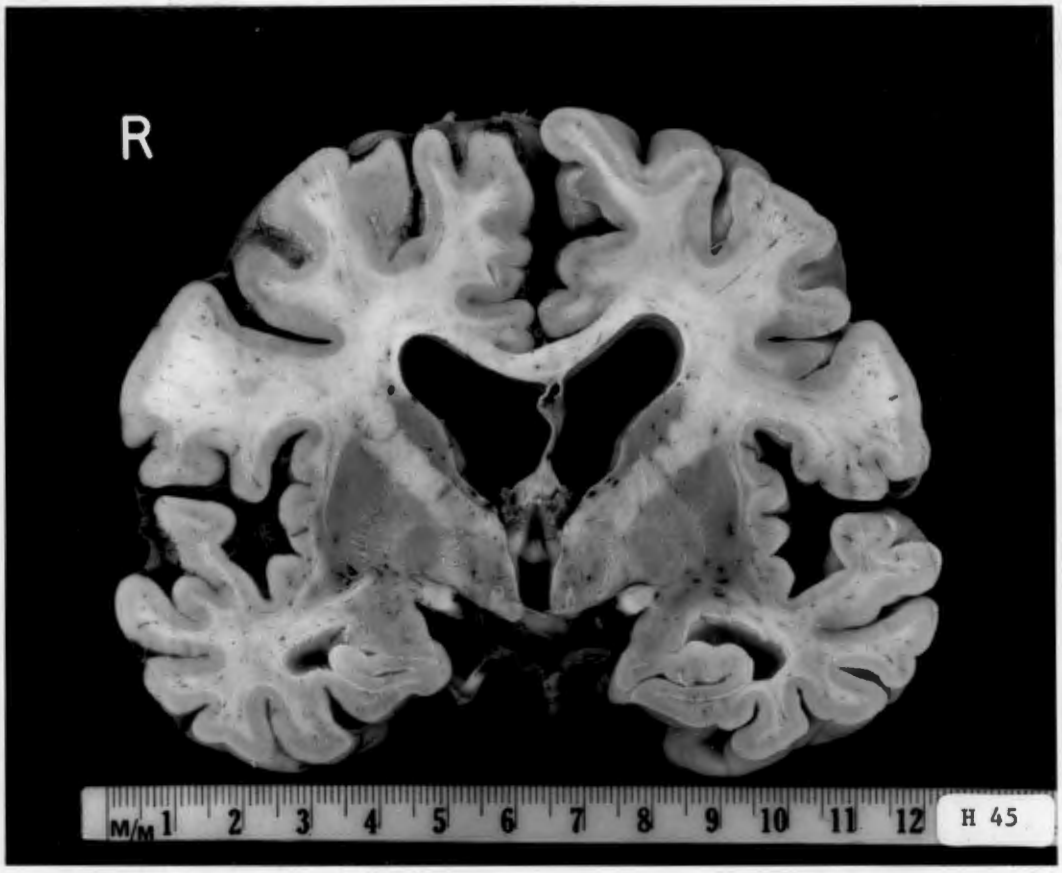
Memory - disorientated for time, place and person (stated correctly, "I have been here for years").

Clinical Diagnosis

Pre-senile organic dementia.

Cause of Death

Bilateral broncho-pneumonia. Chronic urinary trace obstruction from prostatic hypertrophy, bilateral renal medullary necrosis. Alzheimer's Disease.



Case 45

Coronal section at level of intraventricular foramina showing severe atrophy.

Post Mortem Findings

Nervous System

Brain weight after fixation 1180g.

Macroscopic appearance: Severe generalised atrophy, most marked frontal and temporal and parietal regions, less marked in occipital region, temporal lobes pointed at poles; considerable widening of sulci in temporal lobes. Vessels at base - a little tortuous.

Moderate enlargement of lateral ventricles, third ventricle widened up to 8mm. Cortical ribbon thinned. White matter - no macroscopic abnormality. Corpus callosum thinned particularly anteriorly. Hippocampi rather small. Cerebellum slight generalised widening of sulci, vermis and both lateral lobes.

Microscopic appearance: Fibrosis of meninges and extensive sub-pial fibrosis of cortex and severe astrocytic gliosis more marked on left than right. Thin cortex but less severely depopulated than one would have envisaged from macro. Severe distortion of plaque throughout. Moderate deposition of tangles. Patchy cell loss in hippocampi. White matter porous, moderate gliosis with thin demyelination. Atherosclerosis of large meningeal vessels. Slight fibrosis of small vessels in widened cortex and white matter.

Respiratory System

Both lower lobes showed confluent bronchopneumonia.

Urinary System

Bilateral renal papillary necrosis - Left kidney more severely than right. Bladder contained infected urine and showed haemorrhagic cystitis. Prostate moderately enlarged and firm.

Case No. 46

Male aged 61 Admitted 15.9.67. Died 8.10.69

Family History

Father died. Bright's Disease.
Mother died. Aged 74. Pneumonia, but did not know what she was doing last few years.

Past History

Worked as football club steward. Enuretic after marriage, stopped two years after wife left. Smoked 40 cigarettes daily.

Wife left patient 14 years before admission and patient lived with sister.

History of Final Illness

Nine months before admission he had a stroke followed by several others. His left side was affected, walked with difficulty. Became doubly incontinent;

speech difficult to understand. Required constant supervision, ate ravenously, became very restless. Was therefore admitted to hospital. On admission he was confused, slow to respond. Disorientated in time and place and incontinent of urine and faeces. BP 200/110. Chronic bronchitis and emphysema. Increased jerks on the left. Left extensor plantar. Five days after admission he had a 'blackout' followed by facial asymmetry, BP 190/100. Both plantars flexor. Three months later he fractured his left leg, which was pinned and plated. He deteriorated steadily and 11 months after admission he had difficulty in swallowing solids. This was further investigated because of increasing shortness of breath, weight loss and persistent cough. The left main stem bronchus was found to be completely obliterated by a growth. He died 2 months later.

Mental State on Examination

A breathless old man who was friendly and co-operative. Replies limited to short sentences. Denied feeling depressed and was not deluded or hallucinated. He was completely disorientated in time and place, could not give his age and could not recall recent or past events.

Diagnosis

Arteriosclerotic Dementia. Carcinoma of lung.

Cause of Death

Lobar pneumonia due to carcinoma of left lung.

Post Mortem Findings

Emaciated man, decubitus ulceration right sacrum. Nervous system. Brain weight after fixation 1175g.

Nervous System

Macroscopical Appearance: small brain, unusually elongated front to back (11cm) and correspondingly narrow from side to side, 16cm. Sulci moderately widened.

Slight patchy atheroma of vessels at base. Moderate enlargement of lateral ventricles.

Right caudate and putamen shrunken and scarred by old softening. Left striatum a little porous. Both thalmi very shrunken; the right shows two small cysts, 2mm across. Right putamen necrotic posteriorly. Brownish staining of medial part of cerebral peduncles which appear atrophied.

Medulla normal apart from small right cortico-spinal lesion.

Cerebellum; softening of folia around undersurface of vermis and some generalised atrophy, most marked in vermis.

Summary: moderate vascular disease and focal damage.

Microscopic Appearance: Severe small and large vessel fibrosis with widened perivascular spaces throughout the brain. White matter showed moderate patchy demyelination and gliosis. There were scattered micro infarcts and the left putamen had large holes.

Other Viscera

Respiratory System: The oesophagus was invaded by neoplastic tissue growing backwards from the carcinoma of the lung. This oesophageal tumour was 5 x 3cm and virtually blocked its lumen. The trachea and both bronchi were filled with greenish pus. The lower lobe of L. lung blocked by bronchial carcinoma which affected the L.L.L. bronchus. The pleura of the L. lung was adherent all over.

Pleura and pleural cavities on the right were normal. The lower lobe of the left lung showed evidence of suppurating lobar pneumonia with several small abscesses measuring approximately 0.5cm in diameter.

Cardiovascular System: Coronary vessels showed moderate atheroma. No evidence of myocardial infarction, past or recent.

The stomach contained two chronic gastric ulcers, one approximately 7cm across, and the other 1cm across. The smaller ulcer had penetrated almost through the gastric wall. Although not perforated it was very near to doing so.

Case No. 47

Male aged 59 Admitted 4.1.64 Died 8.1.70

Family History

Nil known.

Past History

Always odd, mean, bad tempered and demanding, no affection for daughter. Frequent changes of job. Drank at week ends. Dirty in personal habits.

History of Final Illness

Admitted to hospital because of further deterioration in personal habits and inability to cope with him. On admission dysphasic and euphoric, grossly impaired in insight and judgements. Short attention span. Could not maintain connected conversation. Recall very poor. Disorientated in time and space. BP 130/80.

Three years after admission had grand mal attack. Four months later fractured right femur which was pinned and plated. Fell frequently and had further grand mal attack six months later.

Deteriorated progressively becoming unable to walk, doubly incontinent, required full nursing care. Developed 'flu and died three weeks later.

Mental State on Examination

A thin, white haired man, wheeled into the interview. Mainly sat motionless, occasionally glanced at the interviewer. Pawed his tray and placed any object placed on it into his mouth indiscriminately. He replied in the affirmative when asked if he had any daughters and if he wanted to phone his wife, but for the rest remained mute. When he did reply it seemed he was emerging from a state of impaired consciousness. He would look up and his eyes would open wider and he would stare in a perplexed way but after one or two seconds would sink back into inaccessibility. He droned (Mmmmmmmmm ..) almost constantly.

Diagnosis

Organic Dementia.

Cause of Death

Bronchial pneumonia.

Post Mortem Findings

Thin and wasted.

Nervous System

Brain weight after fixation 1220g.

Macroscopical Appearance: Rather a small brain. Moderate generalised atrophy of both hemispheres. All lobes affected, least marked orbitally. Vessels at base atheroma. Severe enlargement of lateral and third ventricles. Cortical ribbon appears narrowed, marked widening of sulci. Lateral fissure widely opened. White matter shows prominent vessels of rather sieve-like appearance in the digital white matter. Deep grey matter (caudate, putamen, thalamus) generally atrophic. Softening of cortex and white matter in right superior frontal gyrus above the anterior horn. This lies in anterior cerebral territory but does not stretch rostrally to the tip of the anterior horn and it spares the cingular gyrus. Patches of cortical softening at tip of left occipital pole 2cm across. Small patch of haemorrhagic infarction in same situation on right. Amygdaloid nuclei small and atrophic. Aqueduct of mid-brain slightly dilated.

Microscopical Appearance: Fibrosis of meninges. Severe plaques and severe tangles throughout cortex. Cortex thin and porous in places, especially in hippocampi. Astrocytic gliosis. Hippocampi shows loss of cells in subiculum and granulo-vacuolar degeneration. White matter astrocytic proliferation. Fibrosis of small vessels throughout brain with widened perivascular spaces. Patchy astrocytic gliosis. Patchy infarction of both occipital lobes and right frontal lobe.

Cerebellum: congophilic plaques. Loss of myelin in dentate. Fibrosis of vessels.

Other Viscera

Respiratory System: Severe congestion of tracheal and bronchial mucosa with much mucopurulent exudate. Consolidation of right lung and patchy consolidation of left lung.

Cardiovascular System: Moderately patchy atheroma of coronary vessels. No evidence of infarction. Slight atheroma of aortic cusps.

Case No. 60

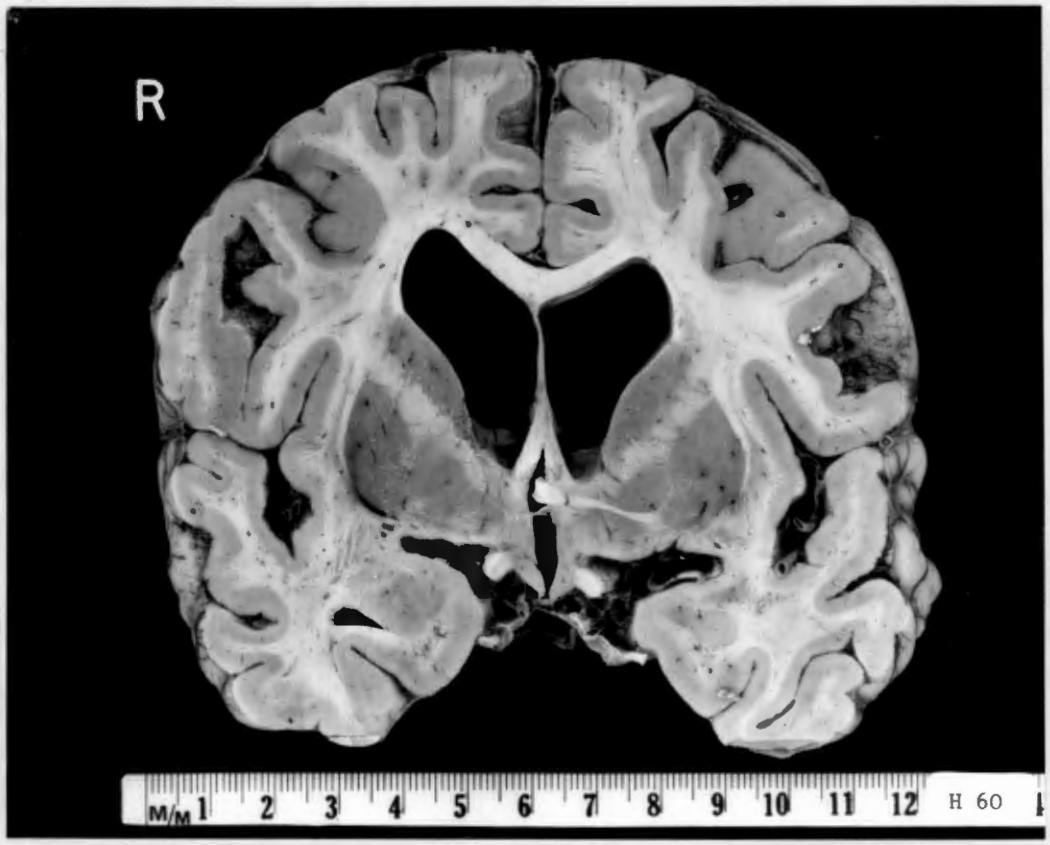
Male aged 84 Admitted 24.3.70 Died 10.9.70

Family History

Nil relevant.

Past History

Lived alone, visited by mental welfare officer. Very dirty, deluded, thinking neighbours were pumping gas into the house. Otherwise rational. Received treatment for ulcer on sole of foot, and oedema right ankle. Meals on wheels. Refused to go into old persons home. Deaf.



Case 60

Coronal section at level of intraventricular foramina showing moderate atrophy.

History of Final Illness

Was unable to cope and admitted after domicillary visits. On admission general condition poor, pulse 120 per minute, irregular. BP 140/90. Short of breath, cough, exematous lesions legs; varicose veins. Right leg stiff. Agitated, confused, accusing neighbours of widespread gambling, and people trying to get him out and disturb him. Recent memory impaired, speech coherent, could read and understand questions, thought neighbours were trying to gas him. Not incontinent, able to dress, feed and wash himself. Moderately steady on feet. Fell and fractured left femur 22.8.70. Transferred general hospital. On return agitated, depressed, incontinent, bed sores. Wound grossly infected and patient died 3 weeks after fracture.

Mental State on Examination

Friendly, deaf old man who answered written questions coherently. Denied depression and hallucinations. Thought neighbours were trying to gas him. Orientated for place but not time. Could recall address after a few minutes.

Diagnosis

Paranoid illness. Mild dementia.

Cause of Death

Pneumonia. Fractured femur.

Post Mortem Findings

Average nutrition. Healing surgical scar along left side with gauze drain inserted.

Nervous System

Brain weight after fixation 1400g.

Macroscopic appearance: Normal sized brain. Vessels at base moderately atheromatous. Left carotid, considerably stenosed, internal lumen about 2mm across, wall on one side also 2mm. Scar right frontal pole measuring some 1.5cm across. Situated at orbito-frontal margin and centred 2-2cm lateral to midline. Cortex indented but no appreciable displacement.

Moderate enlargement of all ventricles. Slight generalised widening of sulci, both convexities. Thalamus porous with few micro-cysts. Midbrain and aqueduct dilated 2-3mm.

Microscopic Appearance: The cortex of the frontal, temporal and occipital regions was gliosed. There were moderate plaques throughout the cortex and in the amygdaloid. There were slight tangles in the amygdaloid. The white matter was porous and gliotic in the frontal, anterior temporal, hippocampal and occipital regions. There was slight fibrosis of the small vessels throughout, including the brain stem.

Other Viscera

Respiratory System: Trachea and main bronchi contain much muro-purulent material. Consolidation in right, middle and lower lobe.

Cardiovascular system: Large heart (385g). Considerable hypertrophy of myocardium, particularly on left side. Patchy white fibrosis; scarring of myocardium. Severe atherosclerosis of coronary vessels which are considerably narrowed in places. Some atheroma around aortic cusps.

Right femur fractured and pinned. Thin fluid coming from wound but no evidence of more than mild infection.

Case No. 61

Male aged 84 Admitted 1937 Died 1.9.69

Family History

Nil known

Past History

Nil known

History of Final Illness

Admitted to hospital with a history of becoming increasingly depressed. On examination depressed and hallucinated. BP 105/80. Mild emphysema, chronic otitis media left ear.

Diagnosis of involutional melancholia. However, clinical picture became more that of schizophrenia with auditory hallucinations. Twenty years after admission described as socially withdrawn and apathetic, unable to give adequate account of himself; evidence of thought disorder, irrelevant replies to questions, aurally hallucinated, poverty of ideation. In spite of all this considered an excellent ward worker. Gradually deteriorated and transferred to ward providing greater care. In 1960 he showed memory impairment for recent events, auditory hallucinations, disorientation in time, place and person, and became increasingly immobile. Became progressively weaker, more disorientated. Incontinent of urine; required help in washing, dressing and feeding before death.

Mental State on Examination

Wheeled into interview. Elderly, remote old man staring blankly into space. Replies limited to 'yes' and 'no'. Difficult to assess. He was not obviously depressed; not possible to illicit whether he was deluded or hallucinated.

Diagnosis

Schizophrenia. Organic Dementia.

Cause of Death

Myocardial infarction, coronary thrombosis.

Nervous System

Brain weight after fixation 1170g.

Macroscopical Appearance: Moderate widening of sulci over both convexities. Moderate dilatation of lateral ventricles, less marked of third. Old rusty softening at apex of right caudate and putamen anteriorly. Systic softening in right putamen, some 6mm across. Left putamen also contained small patches of softening in dorsal half. Slight patchy atheroma of vessels at base.

Microscopic Appearance: There were moderate plaques and slight tangles, more marked in the anterior temporal region except for plaques, which were also severe in the occipital region.

Cortex showed some neuronal loss, was spongy, porous and gliotic. The white matter was porous with a few fibrosed vessels. There was an old infarct in the left occipital cortex. Both putamen were cystic as were the thalami. The vessels were fibrosed with widened perivascular spaces, also noted in external capsule. A few meningeal vessels were congophilic.

Other Viscera

Respiratory System: Gross pulmonary oedema both lungs.

Cardiovascular System: Wall of left ventricle congested and necrotic, showing evidence of recent myocardial infarction. Coronary vessels, right coronary artery - gross atheromatous change. Descending branch of left coronary artery blocked by recent antemortem thrombus. Mitral valve showed evidence of rheumatic heart disease.

Gastrointestinal System: Liver showed nutmeg pattern of congestive cardiac failure. Spleen showed evidence of congestive cardiac failure.

Genito urinary System: Left kidney single cyst at lower pole 5cm diameter, contained fluid.

Case No. 62

Male aged 76 Admitted 4.4.69 Died 3.3.70

Family History

Nil known.

Past History

Admitted because wife went into hospital and patient unable to look after himself. On examination well-preserved man with staring look. Co-operative, disorientated in time and place, answers in monosyllables. Dressed and undressed inappropriately. Urinated in various places. Speech slurred. Unable to give history. BP 140/80. Gradually deteriorated, becoming incontinent, with more marked Parkinsonian face and tremor of left hand.

Four months after admission fell and fractured femur. Six weeks after operation walking with aid, hardly spoke, became ill with 'flu. Gradually deteriorated, developed bronchitis, pulmonary oedema and died.

Mental State on Examination

Elderly Parkinsonian man. He sat quietly throughout interview. Stared fixedly ahead, unblinking, and only answered questions monosyllabically after lengthy pauses.

Denied depression. No delusions or hallucinations elicited. Knew he was in hospital but otherwise disorientated for place and person. Unable to give account of himself.

Cause of Death

Bilateral broncho-pneumonia. Subdural haematoma.

Post Mortem Findings

Thin and wasted man. Pressure sores both heels and right buttocks. Healed scar left thigh.

Central Nervous System

A layer of partially organised blood clot subdurally over right fronto-parietal region. Cortex slightly impressed in this area.

Brain weight after fixation 1400g.

Macroscopical appearance: Normal sized brain. Sheet of sub-dural haemorrhage overlying both convexities, thicker on right than left. Right dorsal convexity brownish and slightly compressed by subdural. Moderate widening of sulci fronto-parietally, less marked in temporal and occipital regions. Basal vessels, thick walled, minimal atheroma. Moderate enlargement of lateral ventricle and third ventricle. White matter slightly porous, most marked occipitally. Hippocampi a little small, cerebellum - suggestion of folial atrophy particularly on dorsal surface.

Microscopical appearance: Slight fibrosis of meninges and sub-arachnoid blood over the right occipital lobe. Cortex showed cell loss with gliosis, which was specially marked in the hippocampi at the lobe of H1. There were a few plaques in frontal and temporal lobes and a deposition of tangles in frontal lobes. The white matter showed demyelination and fibrosis. Small vessels throughout were severely fibrosed with wide perivascular spaces. Numerous perivascular spaces, the parenchyma around these was thin. Brainstem - fibrosis of small vessels in the medulla. Cerebellum, loss of Purkinje cells and some fibrosis of small vessels.

Other Viscera

Respiratory System: Patchy consolidation of both lower lobes.

Cardiovascular System: Slight hypertrophy of left ventricular myocardium, slight patchy atheroma of coronary arteries.

Case No. 71

Male aged 74 Admitted 4.1.68. Died 20.2.70.

Family History

Father died of meningitis.

Past History

Coach builder. Car hire and garage proprietor until retirement.
1956 Adenocarcinoma of rectum with metastases; colostomy made.

History of Final Illness

During the two years prior to admission he became increasingly forgetful and confused and was unable to look after himself. Began to go back to period before his marriage, forgot to turn on gas tap after lighting a match, would drive car in and out of garage saying he had someone to pick up. In November said a large amount of money was missing. Wandered out to the car park, picked up by the police and admitted to hospital. On admission he was disorientated in time and place and showed a poor memory for recent events. His condition deteriorated, he became incontinent of urine and unable to walk. He developed broncho-pneumonia and died three weeks later.

Mental State on Examination

A tall, elderly man whose attention was difficult to hold. There was no spontaneous speech and his replies were monosyllabic. He was totally disorientated in time and place and had no memory for past or recent events. He stared fixedly ahead throughout most of the interview.

Diagnosis

Organic Dementia.

Cause of Death

Suppurative bronchiolitis. Senile dementia, carcinoma of the rectum excised.

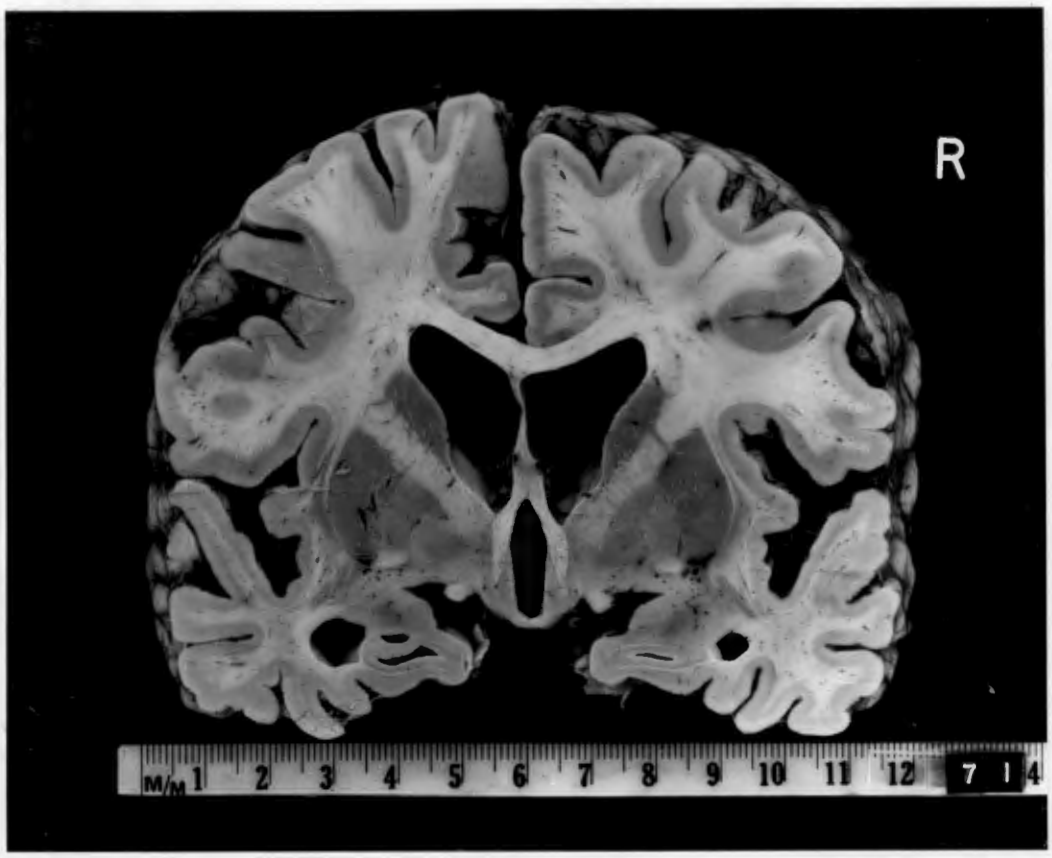
Post Mortem Findings

Elderly man, fairly well nourished.

Nervous System

Macroscopical appearance: Normal size brain. Moderate atrophy, more marked in frontal region and anterior temporal region. Basal vessels not thickened. Slight thinning of cortical ribbon. Moderate enlargement of lateral ventricles. Slight dilatation of third ventricle. Moderate dilatation fourth ventricle. Subcortical white matter - moderate number of pinpoint size cystic spaces.

Microscopical appearance: Severe plaques, moderate tangles throughout cortex. Plaques and tangles in amygdaloid. Neuronal population good except slight cell



Case 71

Coronal section at level of intraventricular foramina showing moderate atrophy.

loss in hippocampi. Severe gliosis frontal area, moderate gliosis temporal and occipital areas. Both putamen porous. Brainstem: substantia nigra very poorly pigmented with a lot of free lying pigment. Louwey bodies.
Cerebellum - lipofuscin in cells; suggestion of tangles. Blood vessels normal.

Other Viscera

Respiratory System: Bronchi filled with pus. Lungs congested with pus exuding from smaller bronchi and bronchioles.

Cardiovascular System: Coronary vessels minimal atheroma. Moderate atheroma abdominal part of descending aorta.

Case No. 73

Male aged 67. Admitted 17.11.68. Died 7.1.70

Family History

Brother committed suicide after car in crash killing passenger.

Past History

During 1940s patient fell out of car, head gashed, refused hospital treatment. Wife said "never been the same since".

History of Final Illness

Did not work for 6 years before admission, was eating very very little. Admitted after attacking wife. Presenile dementia diagnosed 6 years before admission. On examination: Elderly man BP 130/90. L. extensor plantar. Unsteady gait. Three days after admission collapsed in chair ? coronary thrombosis. BP 110/70. Pale, purpura of forearms - both plantars down. Aggressive at times. Three months after admission fell while leaving toilet BP 120/90 lying, 75/60 standing. Developed ankle oedema, BP 150/100 and then diarrhoea and dehydration. Occult blood in stools. Episodes of vomiting - diagnosis of hiatus hernia. Developed 'flu two weeks before death and then pneumonia.

Diagnosis

Organic Dementia.

Cause of Death

Broncho-pneumonia. Organic Dementia.

Mental State on Examination

Well looking elderly man who replied coherently to questions. He was neither depressed, deluded, not hallucinated. He knew where he was but was disorientated for time.

Post Mortem Findings

Nervous System

A few flecks of atheroma. Brain weight after fixation 1410g.

Macroscopical appearance: Rather a small brain. Left parietal convexity shows a patch of blood staining measuring some 4cm diameter, appears to be sub-arachnoid. Adjacent tissue feels a little soft. Slight generalised atrophy of both hemispheres. Vessels at base a little tortuous. R. carotid, few flecks of atheroma; L. carotid contains blood clot which is difficult to remove and may be ante-mortem. Both unci rather deeply grooved some 4mm lateral to their medial borders.

Coronal cuts: Ventricles normal size. Very slight widening of sulci. Small cyst at base of R. putamen 2-3mm diameter.

Microscopical appearance: Slight subarachnoid bleeding L. frontal and L. occipital regions. Medium sized and small vessels fibrosed. Vessels patchily congophilic in L. occipital region.

Slight cell loss in occipital region. Marked fibrosis small vessels and there were fibrotic veins. Parenchyma spongy and reticulated appearance - peri-vascular cuffing in L. anterior temporal region.

Marked demyelination in both occipital regions, more marked on L. than R. - not seen in frontal parietal or temporal regions.

Vessels throughout white matter fibrosed.

Other Viscera

Respiratory System: Bilateral consolidation. Moderate pulmonary oedema.

Cardiovascular System: Severe atheroma of coronary vessels. Severe atheroma of aorta. Small dissecting aneurysm of ascending and arch of aorta.

Case No. 77

Male aged 76 Admitted 4.3.70. Died 2.8.70

Family History

Father died aet 60 of pneumonia.
Mother mildly demented before dying aged 78.

Past History

Wheelwright 25 years; then caretaker of band stage until aged 65 years. Retired and did light work until 3 years before admission.

History of Final Illness

During 3 years prior to admission he became increasingly confused and forgetful. There was noticeable deterioration after an episode of 'flu 4 months before admission. He did not recognise his home, was incontinent, not recognising the W.C. when led there, displayed an unpredictable temper and was restless.

On admission he was a cheerful, co-operative man with no spontaneous speech - he answered mono-syllabically. Disorientated in time and place. Washed self but needed help with dressing, did not recognise nursing staff, continent.

BP 170/90. Poor co-ordination; R.L discrimination lost.

A month after admission developed a urinary infection then a chest infection with evidence of cardiac failure. Two months later he developed broncho-pneumonia and died.

Mental State on Examination

A cheerful, elderly man who kept showing his name in his shoes. He was suspicious that his wife might go away with someone else. Disorientated in time and place, not hallucinated.

Diagnosis

Organic Dementia.

Cause of Death

Congestive cardiac failure. Ischaemic heart disease. Broncho-pneumonia.

Post Mortem Findings

Elderly well-built man.

Nervous System

Brain weight after fixation 1470g.
Macroscopic appearance: Normal size brain. Slight narrowing frontal gyri. Moderate enlargement lateral and third ventricles - septum split posteriorly. Cortex round anterior tip of inferior horn has little or no white matter and appears atrophic.
Amygdaloid nuclei small.
Elsewhere slight widening of sulci. White matter normal.

Microscopic appearance: Severe plaques formation throughout brain. Slight tangles in temporal cortex (hippocampus). Slight fibrosis of small vessels. Gliosis of cortex. Depopulation of hippocampi.

Other Viscera

Respiratory System: Larynx and trachea filled with pus. Broncho-pneumonia in lower lobes and acute suppurative bronchiolitis. Micro - inflammatory cells.

Cardiovascular System: Patchy atheroma coronary vessels - no infarcts - some Right ventricular hypertrophy.

Digestive System: Liver - nutmeg pattern of congestive cardiac failure.

Case No. 80

Male aged 77 Admitted 15.4.69. Died 19.1.70

Family History

Nil known.

Past History

Nil known.

History of Final Illness

During the two years prior to admission he had become increasingly forgetful, confused, incapable of caring for himself. He had been unable to walk for the 9 months preceding admission.

On admission he was confused, drowsy and unco-operative. He was disorientated in time but knew he was in hospital. He had a cataract of the left eye. There was marked peripheral wasting of all four limbs. His reflexes were sluggish and his plantar responses flexor. His response to pain was blunted over the left forearm. He was incontinent of urine.

He improved over the first month becoming better nourished and more co-operative. He crawled about. He seemed to understand speech but his own was very difficult to understand. He became doubly incontinent but walked with assistance, fed himself slowly and messily and crawled unaided to the bedroom and toilet.

He suddenly collapsed with a stroke resulting in left-sided paralysis. He then developed pneumonia and died 4 days after his collapse.

Mental State on Examination

He sat hunched up throughout the interview and displayed no change of facial expression. He was unable to answer any questions except by making a humming noise. He was, however, able to smoke a cigarette and to push away a proffered hand.

Diagnosis

Organic Dementia.

Cause of Death

Suppurative bronchiolitis. Cerebrovascular accident.

Post Mortem Findings

Nervous System

Brain weight after fixation 1290g.

Macroscopic appearance: Normal size brain. The frontal, and to a lesser extent anterior parietal sulci, are moderately widened, both convexities. Temporal and occipital least marked. Undersurface of the left cerebellar hemisphere has an area of tissue destruction with retraction of cortex, measuring some 3cm antero-posterior by 1.5cm across.

The right cerebral hemisphere shows recent softening of cortex in territory of middle cerebral artery but particularly below the lateral fissure.

Vessels at base are moderately tortuous and show moderate patchy atheroma.

Microscopic appearance: Slight fibrosis of meninges. Evidence of neuronal loss in frontal cortex. There were no neurofibrillary tangles but a slight degree of plaques in the temporal lobe.

There was marked vascular disease. The large vessels in the temporal lobe were arteriosclerotic. Medium and small sized vessels throughout the meninges and the cortical and white matter were fibrosed with widened perivascular spaces. There were numerous infarcts: one infarct in the right frontal region, a large (0.5cm) area of haemorrhage in the right putamen, visible to the naked eye.

There was destruction of the superior and part of R. middle temporal lobe, the insula, and the area connecting the basal ganglia with the temporal lobe. The haemorrhage was in the distribution of the middle cerebral artery. The hippocampus appeared unaffected.

There was a large infarct over the lateral convexity of the right occipital lobe involving two adjacent lobes and extending into the white matter.

There was an area of softening and fibrosis of the left occipital lobe. The globus pallidus and amygdala were normal.

There was an area of old softening in the dentate nucleus. The midbrain had an old infarct near the substantia nigra.

Other Viscera

Respiratory System: Trachea and main bronchi filled with pus. Lungs showed marked emphysema and suppurative bronchiolitis.

Cardiovascular System: All chambers were dilated but especially left and right auricles and right ventricle. Coronary vessels showed moderately severe atheromatous change. Myocardium was fibrotic and there was evidence of two old myocardial infarcts in the posterior wall of the left ventricle. Aorta showed moderately severe atheromatous change.

Case No. 86

Male aged 66 Admitted 6.6.68 Died 13.3.69

Family History

Nil relevant.

Past History

Twenty-one years with the B.B.C. as studio attendant. Retired. Widower since 1966.

History of Final Illness

Stroke 17 months before admission. Had residual hemi-paresis, became aggressive and was retired from work. Deteriorated further, becoming confused, incontinent. On examination, very confused, dysphasic old man, with a lot of spontaneous talk which was incomprehensible. Gradually deteriorated further, developed 'flu, followed by severe broncho-pneumonia and died.

Mental State on Examination

A small, gray haired old man who sat in the chair and failed to answer any questions except concerning his name and status. He continually ground his teeth and made loud sucking noises. From time to time he would grimace. At one point he tried to grab the interviewer's pen.

Diagnosis

Dementia.

Cause of Death

Influenzal tracheo-bronchitis and broncho-pneumonia. Pre-senile Dementia.

Post Mortem Findings

Nervous System

Brain weight after fixation 1180g.

Macroscopical Appearance: Moderate generalised atrophy; some orange-yellow staining of meninges over left posterior ventral gyrus. Vessels at base show few flecks atheroma. Moderately severe dilatation of ventricles. Cortex generally thin; gyri atrophied especially in both temporal lobes. Sub-cortical white matter reduced in amount. Corpus callosum slightly thinned. Caudate nucleus slightly atrophic; more so on right. Hippocampi small. Mid-brain; aqueduct slightly dilated.

Microscopical Appearance: The cortex throughout contained a severe degree of plaques and tangles. There was evidence of neuronal loss and the cortex had numerous holes. There were plaques in the amygdaloid nucleus and medial aspect of caudate nucleus. There were tangles in the amygdaloid. Some of the small vessels in the cortex and white matter were fibrosed. Vessels in the fusiform gyrus, putamen and the base of the inferior temporal sulci were patchily congophilic.

Other Viscera

Respiratory System: Trachea and main bronchi plum coloured, typical appearance of viral tracheo-bronchitis. Lungs - confluent bronchial pneumonia in lower lobes. Some pulmonary oedema.

Cardiovascular System: Slight atheromatous change of coronary vessels.

Case No. 94

Male aged 77 Admitted 29.1.70 Died 5.6.70.

Personality

Belligerent, dogmatic. No interests.

Family History

Father died in old age.

Mother died early 80's, family used to avoid her.

Past History

Menial job until war, where he worked in factory. General hand on Southend Pier until retirement.

Appendicectomy 1966. After operation developed chest infection and a severe confusional state.

History of Final Illness

During three years before admission he became gradually more forgetful and apathetic. He was restless and confused at night, wandered out and became incontinent of urine.

On admission he was a feeble, emphysematous old man, disorientated for time, place and person. He was incontinent of urine and faeces, and had to be dressed. BP 180/120. He deteriorated further, developed pneumonia and died eight months after admission.

Mental State on Examination

An old man who walked slowly in short steps, and who sat quietly throughout interview but only replied monosyllabically to questioning. Gross poverty of speech. He was totally disorientated for time, place and person. He looked depressed and anxious. No insight.

Diagnosis

Organic Dementia.

Cause of Death

Pneumonia.

Post Mortem Findings

Nervous System

Brain weight after fixation 1320g.

Macroscopical Appearance: Normal size brain. Severe atrophy of both hemispheres - more marked frontal pole and along convexity. First frontal gyrus and adjacent sulci remarkably shrunken back to central fissure. Some widening of parietal sulci, particularly on left. Temporal and occipital convexity noticeably less affected on both sides.

Vessels at base thin-walled and tortuous - patches of thickening with atheroma.

Cortical ribbon markedly narrowed. Moderate dilatation of lateral and third ventricles. Sub-cortical white matter - a few small cystic spaces especially in temporal lobe. Deep grey matter cystic, large cystic softening 1cm diameter in right putamen; several similar softenings left putamen. Substantia nigra poorly pigmented especially laterally.

Microscopical Appearance: Few plaques in temporal area. Cortex diffusely gliosed, sub-pial fibrosis. White matter severely gliosed throughout. Small vessels fibrosed throughout and markedly widened perivascular spaces. Two old infarcts left frontal cortex and one at right fronto-cortico-medullary junction; several softenings in right hippo. Temporal lobes; loss of cells in subiculum.

Basal ganglia: Putamen full of holes. Small micro infarcts with foam cells. Gliosis, pallidum porous.

Other Viscera

Respiratory System: Bronchi inflamed. Bilateral broncho-pneumonia and bronchiolitis.

Cardiovascular System: Slight left ventricular hypertrophy. Moderate atheroma of aorta.

Urinogenital System: Large solitary cyst 5cm diameter at upper pole.

Reticulo-Endothelial System: Several calcified omental nodes.

Case No. 95

Male aged 80 Admitted 3.3.70 Died 1.11.70

Family History

Sister died aged 81 with dementia.

Past History

1961 stroke. Appeared to recover.

History of Final Illness

Six years before admission had a second stroke and after this was very aggressive towards wife and then gradually became confused, disorientated. One year before admission fell and cut his arm badly, ? stroke. There was a rapid deterioration. Speech became incoherent, he was unable to recognise members of the family. Forgot to light gas, unable to wash or dress himself and became doubly incontinent - lay in bed fully clothed. On admission he had a right increased knee jerk, right extensor plantar. BP 140/100. Deaf, confused, unable to give account of himself. Walked with right-sided limp. Four months after admission fell out of bed, lacerated scalp, found to have broncho-pneumonia. Two months before admission blood pressure 110/60, became more enfeebled and died.

Mental State on Examination

Dyspnoeic old man with pronounced bluish-grey complexion. Sat down unaided, shook hands and sat quietly throughout interview. Appeared to understand questions but only replied to those which required a 'yea' or 'nay'. Did not appear deaf. No spontaneous speech. Speech slurred at times, admitted to being depressed and wanting to die. Looked dejected. No paranoid ideas, no hallucinations. Disorientated for time, place and person. (Was dysarthric, dysphasic and demented.)

Diagnosis

Organic dementia, cardiac failure.

Cause of Death

Broncho-pneumonia and carcinoma of liver, haemochromatosis.

Post Mortem Findings

Elderly man, slight icteric with generalised slate-grey pigmentation of skin, marked senile purpura on hands. Small, old right sub-dural haematoma containing 20-30ml colourless fluid enclosed in a thin membrane. Flecks of atheroma in larger vessels.

Nervous System

Brain weight after fixation 1210g.

Macroscopic appearance: Slightly small sulci, a little widened, more marked in anterior three-quarters than occipitally. Slight brownish discolouration frontal pole. Vessels at base slightly tortuous, slight patchy atheroma. Moderate enlargement of lateral ventricles. Slight widening of sulci. Thalami a little atrophic.

Area of haemorrhage at origin of middle cerebellar peduncle 1cm by 8mm cross section, extending 8mm from above-down. May be small vascular malformation.

Brownish, cystic area 1cm by 5mm antero-lateral part of lower pons, ? connected to malformation. Left pyramid brownish and shrunken.

Microscopic appearance: Cortex has few plaques in frontal, anterior temporal and hippocampal areas.

White matter, severe gliosis. Fibrosis of small vessels with widened perivascular spaces. Old infarcts in anterior temporal region and occipital region. Cerebellum - fibrosis of vessels.

Brainstem - vascular anomaly in pons.

Other Viscera

Respiratory System: Moderate congestion and pulmonary oedema. Left lung and lower lobe right lung almost black.

Cardiovascular System: Heart - dilatation of right auricle, right ventricle, left auricle; myocardium "nutmeg" colour, moderate atheroma of coronary vessels. Moderately severe atheroma of aorta.

Alimentary System: Liver (Weight 1525g). Finely, nodulated cirrhosis with single neoplastic mass some 15cm diameter in Right lobe. Fibrotic and brown coloured. Liver stained +ve with Perls stain for iron.

Reticulo-endothelial System: Lymph nodes purple-brown.

All viscera contained iron.

Case No. 96

Male aged 73 Admitted 28.8.69. Died 27.12.69

Family History

Nil known

Past History

Nil known

History of Final Illness

Onset of illness not known. Transferred from a nursing home after found wandering half clad in road in a deteriorated condition. Incontinent of urine. On examination marked facial and general muscular rigidity. BP 140/80. Completely disorientated in time and place. Syncopal three months after admission. Pyrexial two weeks before death. Few creps right lung. ? appendicitis 5 days before death, then became unconscious. BP 70/60. Died.

Mental State on Examination

Friendly, elderly man with Parkinsonian faces. Unable to give account of himself. Sat quietly throughout interview, no spontaneous speech, replies mainly monosyllabic. Few sentences. Not depressed or obviously hallucinated. Deluded and disorientated for time and place. Thought he was in a hotel. Gave year as 1893. No insight.

Diagnosis

Organic Dementia. Parkinson's disease.

Cause of Death

Left ventricular failure. Intestinal haemorrhage from duodenal ulcer.

Post Mortem Findings

Pale, well nourished man.

Nervous System

Brain weight after fixation 1400g.
Macroscopic appearance: Normal size brain. Patchy atheroma and generalised thickness of basal vessels. Moderate widening of sulci over both convexities, most marked fronto-parietally and temporally. Slight enlargement of lateral ventricles. Cortical ribbon slightly narrowed generally.
Mid-brain - considerable loss of pigment in substantia nigra. Most marked on right side.

Microscopic appearance: There were moderate plaques throughout the cortex, including the amygdaloid, but sparing the occipital cortex. Small plaques had congophilic cores. There was gliosis of the cortex. The white matter was severely gliotic and porous. There was fibrosis of the small vessels throughout the brain, some being congophilic.

Other Viscera

Respiratory System: Lungs - pulmonary oedema.

Cardiovascular System: Moderate atheroma of pulmonary vessels.

Alimentary System: Stomach - large, chronic duodenal ulcer in first part of duodenum; removal of blood clot showed small gaping artery. Remainder of duodenum, small and large intestines contained large quantity of blood.

Case No. 98

Male aged 81 Admitted 3.10.62 Died 8.10.69

Family History

Nil known

Past History

Nill known

History of Final Illness

Onset not known. Transferred from a general hospital after major fit. Endogenous depression with nihilistic delusions and hypochondriasis diagnosed. On admission grossly resistive, inaccessible, agitated, marked perserveration of speech. BP 120/70.
Chronic bronchitis with emphysema, cervical spondylosis, osteoarthritis of knees and elbows. Grasp reflex. Major fit day after admission, then incontinent of urine, confused with poor memory for recent and past events, paranoid ideas. Two months later found on floor whilst on visit to home, ? CVA. Became increasingly confused and 2 years after admission was unable to walk, unsteady when standing. Haematuria 1964/1968. Still speaking sentences, continent of urine but unable to wash or dress, knew where he was two months before death. Developed pneumonia month before he died.

Mental State on Examination

A thin, elderly man unable to give clear account of himself, fatuous and facile, not obviously depressed, suspicious at times, not hallucinated, made poor rapport. Disorientated for time, place and person.

Diagnosis

Organic dementia.

Cause of Death

Broncho-pneumonia. Carcinoma of lung.

Post Mortem Findings

Thin, wasted body.

Nervous System

Brain weight after fixation 1320g.
Macroscopic appearance: Rather small brain, moderate generalised narrowing of gyri with some widening of sulci over both convexities, less marked at frontal poles. Slight patchy atheroma of vessels at base. Slight enlargement of ventricles, third normal. Cortical ribbon minimally narrowed. White matter - normal. Softening 2cm diameter right parietal lobe; soft purulent-looking centre. ? secondary or abscess.

Microscopic appearance: Severe plaques, moderate tangles, moderate congophilic angiopathy. Slight small vessel fibrosis. Right parietal region has secondary deposit extending from cortical surface to white matter. Micro-infarcts right frontal. Moderate gliosis throughout. Plaques in amygdaloids.

Other Viscera

Respiratory System: Left lung replaced by carcinoma appearing to derive from bronchi. Mediastinal glands involved (microscopy - anaplastic neoplasm).

Alimentary System: Liver weighed 2700g. Massive infiltration by secondary deposits. Gallbladder and ducts healthy.

Mesenteric glands: Secondary deposits in para-aortic glands. (Right iliac almost occluded by severe atheroma)

SUGGESTED PROFORMA FOR MENTAL STATE ASSESSMENT OF DETERIORATED PATIENT

General Appearance and Behaviour

Brief description of physical appearance of patient.

Speech

Is there any spontaneous speech?

Does he comprehend questions?

Are his replies understandable/sensible?

Are his replies relevant?

Are his replies delayed and the questions require repeating?

What abnormalities are there in the delivery of his talk?

What abnormalities are there in the content of his talk?

Sample of talk.

Mood

Determine this as best possible.

Thought content

Delusions where they can be elicited.

Hallucinations

If detectable.

Compulsive phenomena

Memory and Orientation

Ten questions from Tooting Bec.

Insight

Where this can be elicited.

Get patient to 1) Write his name.

2) Read a sentence.

3) Copy the time on a watch.

Find out within the first week whether the patient

1) Attempts to undress himself, wash himself, feed himself.

2) Can find his way to the lavatory, bedroom or bed.

3) Makes contact with other patients.

4) Is continent of a) urine day after admitted; b) faeces.

5) Can walk without the help of a nurse.

APPENDIX II

Figure Form Board Tests

Total maximum score for the three tests: 50 points.

I. Three-figure Form Board Test (3 pieces)

The patient has to complete each stage by placing the appropriate block in the empty space. See diagram.

Scoring One point for each block placed correctly.
Bonus points if each stage is completed within a time limit as follows:

Stage I within two minutes = ½ point
Stage II within two minutes = ½ point
Stage III within two minutes = 2 points.

Maximum possible score 8 points.

II. Two-figure Form Board Test (9 pieces)

Scoring One point for each block placed correctly.
Bonus points given for stage IV only as follows:

Completion within three minutes = 3 points
Completion within five minutes = 2 points
Completion within ten minutes = 1 point.

Maximum possible score 20 points.

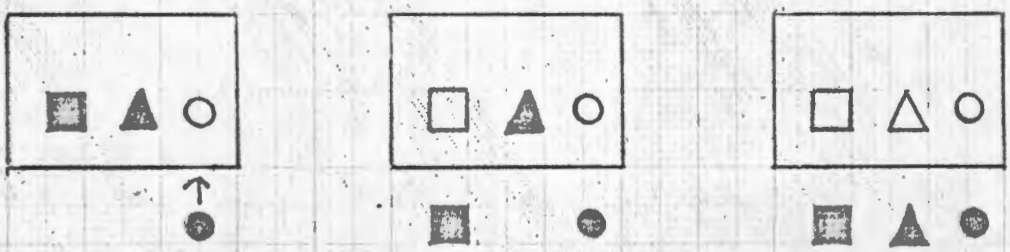
III. Five-figure Form Board Test (11 pieces)

Scoring One point for each block placed correctly.
Bonus points given for stage III only as follows:

Completion within three minutes = 1 point
Completion within five minutes = 2 points
Completion within ten minutes = 1 point

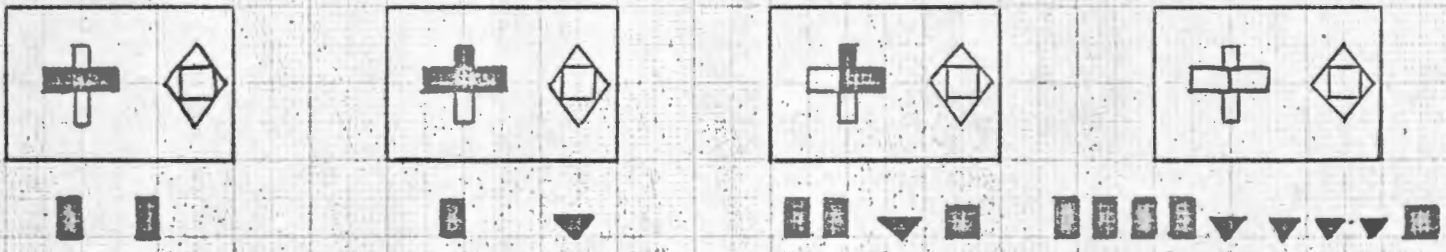
Maximum possible score 22 points.

I. Three-figure Form Board Test



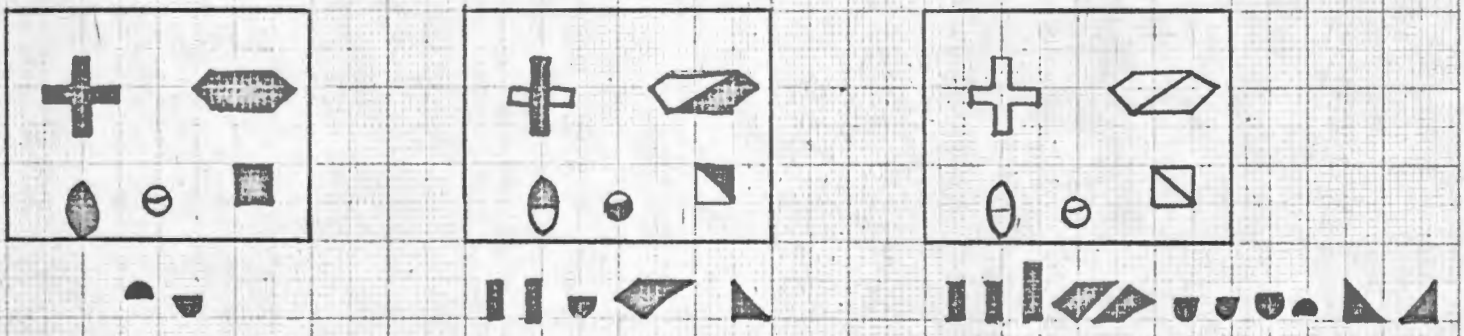
Stage I Stage II Stage III

II. Two-figure Form Board Test



Stage I Stage II Stage III Stage IV

III. Five-figure Form Board Test



Stage I Stage II Stage III

Results of Psychological Testing

1. Patient no. 86

Could not perform any of the tests.

2. Patient no. 33

W.A.I.S. Scaled scores Information 7
Comprehension 2.
Arithmetic 3.
(No response for rest of test).
Performance Score 0
Verbal Score 68

Figure Form Board: only obtained one point in first part. Did not continue.
Token Test: Performed 5 of the first 10 tests, but after this was quite unresponsive.
The patient was too distractible to perform the tests.

3. Patient no. 36

Was untestable. He did not co-operate on any of the tests.

4. Patient no. 96

W.A.I.S. Verbal 101
Performance 85
Full scale score 94
Figure Form Board 28%
Token Test 86%

5. Patient no. 71

Failed to score on anything except two items out of the first ten questions of the Token Test and did not co-operate further.

6. Patient no. 45

Unable to perform any of the tests.

7. Patient no. 95

Did not attempt any of the tests.

8. Patient no. 73

Scored 62% on token test but then tired and did not complete it.

9. Patient no. 80

W.A.I.S. Scaled scores Information 5
Comprehension 2
Did not attempt the other tests of the W.A.I.S.
Figure Form Board Test = 21%
Token Test 19% - he showed sporadic concentration.

10. Patient no. 14

W.A.I.S. Verbal score 95
Performance Score 94
Full scale score 94
Figure Form Board Test 100%
Token Test 94%

11. Patient no. 47

W.A.I.S. Block design scalred score 5
Object assembly scaled score 1
Picture completion scaled score 1
Similarities scaled score 4
Verbal score 0
Performance Score 63
Figure Form Board 52%
Token Test 0

12. Patient no. 13

Only completed parts 1, 2 and 3 of Token test for which he received a score of 30 out of 30 and then refused to co-operate further.

13. Patient no. 16

Unable to complete any of the questions.

14. Patient no. 62

W.A.I.S. Scaled score Information 6
Comprehension 1
Arithmetic 4
Similarities 0
Digit Span 1
Vocabulary 2
Raw Score Picture Completion 1
Object Assembly 1
Other tests 0
Verbal score 70
Performance score 69
Full scale score 68
Token Test 54%. Patient stopped co-operating half-way and answered randomly.

15. Patient no. 11

W.A.I.S. Verbal Score 81
Performance Score 80
Full scale score 79
Figure Form Board Test 74%
Token Test 50%

16. Patient no. 94

Figure Form Board 100%
Token Test 3% then refused to co-operate further.

0 = 1 = DK = NA =

NAME: CONSULTANT:

DATE: INITIALS OF NURSES OBSERVING PATIENT:

WARD:

Abbreviations: DK = Don't Know; NA = Not Applicable

The number corresponding to the correct answer should be ringed. Where "Not Applicable" is the appropriate answer details should be filled in along side.

UNUSUAL OCCURRENCE SINCE THE PATIENT WAS LAST RATED:

A1. Did the patient speak to-day?
No 0
Yes 1
DK 8
NA 9

A2. Was the patient's speech relevant to-day?
No 0
Yes 1
DK 8
NA 9

A3. Was most of what the patient said to-day sensible?
No 0
Yes 1
DK 8
NA 9
(did not speak)

A4. Did the patient say one or two words sensibly to-day?
No 0
Yes 1
DK 8
NA 9
(did not speak)

A5. Did the patient speak sentences sensibly when he spoke to-day?
No 0
Yes 1
DK 8
NA 9
(did not speak)

A6. Was the patient's rate of speech within normal limits to-day?
No 0
Yes 1
DK 8
NA 9
(did not speak)

A7. Did the patient speak very much more rapidly than a healthy person to-day?
No 1
Yes 0
DK 8
NA 9
(did not speak)

NAME OF PATIENT: DATE:

A8. Did the patient speak much more slowly than a healthy person to-day?
No 1
Yes 0
DK 8
NA 9
(did not speak)

A9. Did the patient speak without first being spoken to to-day? (Speech need not have been coherent)
No 0
Yes..... 1
DK 8
NA 9

A10. Did the patient walk without the help of a nurse to-day?
No 0
Yes 1
DK 8
NA 9
(bed-ridden)

A11. Did the patient walk without repeated encouragement to-day?
No 0
Yes 1
DK 8
NA 9
(did not walk)

A12. Did the patient, to-day, walk very much more rapidly than a healthy person of the same age?
No 1
Yes 0
DK 8
NA 9
(did not walk)

A13. Did the patient, to-day, walk very much more slowly than a healthy person of the same age?
No 1
Yes 0
DK 8
NA 9
(did not walk)

A14. Was the patient extremely restless to-day? (walking about continually)
No 1
Yes 0
DK 8
NA 9
(did not walk)

A15. Was the patient extremely lethargic to-day?
No 1
Yes 0
DK 8
NA 9

A16. Did the patient find his own way /or point to the lavatory at least once to-day?
No 0
Yes 1
DK 8
NA 9

A17. Did the patient find his own way /or point to the bedroom last night?
No 0
Yes 1
DK 8
NA 9

NAME OF PATIENT: DATE:

A18. Did the patient find his bed last night; or point to it to-day?
No 0
Yes 1
DK 8
NA 9
(Does not walk Bed-ridden

A19. Did the patient attempt to feed himself to-day? (Breakfast or lunch)
No 0
Yes 1
DK 8
NA 9

A20. Was the patient fed to-day?
No 1
Yes 0
DK 8
NA 9

A21. Did the patient drink when a cup of tea or glass of water was placed in front of him to-day?
No 0
Yes 1
DK 8
NA 9

A22. Did the patient eat a biscuit without assistance to-day?
No 0
Yes 1
DK 8
NA 9
(not given a biscu

A23. Did the patient eat very much more slowly than a healthy person to-day?
No 1
Yes 0
DK 8
NA 9

A24. Was the patient incontinent of urine to-day?
No 1
Yes 0
DK 8
NA 9

A25. Was the patient incontinent of faeces over the past 24 hours?
No 1
Yes 0
DK 8
NA 9

A26. Did the patient know what to do when he was in the lavatory or was given a bottle or commode during the past 24 hours?
No 0
Yes 1
DK 8
NA 9

A27. Did the patient attempt to undress himself last night?
No 0
Yes 1
DK 8
NA 9

A28. Did the patient require help to undress last night? (Includes nurse's adjustments)
No 1
Yes 0
DK 8
NA 9

NAME OF PATIENT: DATE:

A29. Did the patient attempt to wash himself when he had his last bath?
No 0
Yes 1
DK 8
NA 9

A30. Did the patient resist being washed when he had his bath?
No 1
Yes 0
DK 8
NA 9

A31. Was the patient indifferent to being washed when he had his bath?
No 1
Yes 0
DK 8
NA 9

A32. Did the patient make spontaneous contact with the staff to-day?
No 0
Yes 1
DK 8
NA 9

A33. Did the patient make contact with the staff with encouragement to-day?
No 0
Yes 1
DK 8
NA 9

A34. Did the patient make spontaneous contact with other patients to-day?
No 0
Yes 1
DK 8
NA 9

A35. Did the patient make contact with other patients if approached by them to-day?
No 0
Yes 1
DK 8
NA 9

A36. Did the patient make contact with friends or relatives when visited during the past week?
(Includes with or without encouragement)
No 0
Yes 1
DK 8
NA ,.... 9
(no visitors)

A37. Did the patient respond soon after being asked to go to the dining room to-day?
No 0
Yes 1
DK 8
NA 9
(eats in chair)

A38. Did the patient assist another patient during the past 24 hours?
No 0
Yes 1
DK 8
NA 9

A39. Did the patient assist the staff in cleaning and tidying the ward during the past 24 hours?
No 0
Yes 1
DK 8
NA 9

NAME OF PATIENT: DATE:

A40. Did the patient ever leave his chair without first being asked to do so to-day?
No 0
Yes 1
DK 8
NA 9

A41. Did the patient sit motionless for an hour or longer to-day?
No 1
Yes 0
DK 8
NA 9

A42. Did the patient exhibit any repetitive movements whilst in his chair to-day?
No 1
Yes 0
DK 8
NA 9

A43. Did the patient move the position of his chair to-day?
No 0
Yes 1
DK 8
NA 9

A44. Did the patient respond when there was singing by singing, humming or tapping?
No 0
Yes 1
DK 8
NA 9
(no singing)

A45. Did the patient attempt to catch the ball when it was thrown to him yesterday?
No 0
Yes 1
DK 8
NA 9

A46. Did the patient attempt to throw the ball back to the nurse yesterday?
No 0
Yes 1
DK 8
NA 9

Has the patient shown any of the following during the past 24 hours?

A47. Handling faeces.
No 1
Yes 0
DK 8
NA 9

A48. Drinking out of inappropriate containers.
No 1
Yes 0
DK 8
NA 9

A49. Handling genitals in public.
No 1
Yes 0
DK 8
NA 9

A50. Striking people without apparent cause.
No 1
Yes 0
DK 8
NA 9

NAME OF PATIENT: DATE:

(Has the patient shown any of the following during the past 24 hours?...contd.)

A51. Destructive behaviour.
No 1
Yes 0
DK 8
NA 9

A52. Hit back or defended himself when attacked.
No 0
Yes 1
DK 8
NA 9

A53. Hit back when verbally provoked.
No 1
Yes 0
DK 8
NA 9

A54. Touching other people's genitals.
No 1
Yes 0
DK 8
NA 9

A55. Misidentification.
No 1
Yes 0
DK 8
NA 9

A56. Evidence of living in the past.
No 1
Yes 0
DK 8
NA 9

A57. Wandering.
No 1
Yes 0
DK 8
NA 9

A58. Appropriate laughter.
No 0
Yes 1
DK 8
NA 9

A59. Inappropriate laughter.
No 1
Yes 0
DK 8
NA 9

A60. Appropriate crying.
No 0
Yes 1
DK 8
NA 9

A61. Inappropriate crying.
No 1
Yes 0
DK 8
NA 9

NAME OF PATIENT: DATE:

(Has the patient shown any of the following during the past 24 hours?...contd.)

A62. Appropriate anger.
No 0
Yes 1
DK 8
NA 9

A63. Inappropriate anger.
No 1
Yes 0
DK 8
NA 9

A64. Sadness or depression.
No 1
Yes 0
DK 8
NA 9

A 65. Cheerfulness.
No 0
Yes 1
DK 8
NA 9

A66. Euphoria.
No 1
Yes 0
DK 8
NA 9

A67. Appropriate fear.
No 0
Yes 1
DK 8
NA 9

A68. Inappropriate fear.
No 1
Yes 0
DK 8
NA 9

A69. Perplexity or bewilderment.
No 1
Yes 0
DK 8
NA 9

A70 Suspicion.
No 1
Yes 0
DK 8
NA 9

A71. Hostility.
No 1
Yes 0
DK 8
NA 9

A72. Sullenness.
No 1
Yes 0
DK 8
NA 9

NAME OF PATIENT: DATE:

A73. Silly giggling fatuous behaviour.
No 1
Yes 0
DK 8
NA 9

A74. Guilt.
No 1
Yes 0
DK 8
NA 9

A75. ~~Screaming~~. shouting
No 1
Yes 0
DK 8
NA 9

A76. Expressionless face.
No 1
Yes 0
DK 8
NA 9

A77. Irritability.
No 1
Yes 0
DK 8
NA 9

A78. Pleasure.
No 0
Yes 1
DK 8
NA 9

A79. Anxiety.
No 1
Yes 0
DK 8
NA 9

Has the patient taken any of the following drugs this week?	No	Yes
A80. Phenothiazine.	0	1
A81. Anti-parkinsonian drug.	0	1
A82. Night sedative.	0	1
A83. Anti-biotic.	0	1
A84. Anti-depressant.	0	1
A85. Anti-convulsant.	0	1
A86. Sympathomimetic.	0	1
A87. Analgesic.	0	1

Smiling
Swearing
Resistive to physical attention

... Name ... Date ...

AGNOSIA

ISUAL

Common objects

Penknife

Fork

Key

Coin

Pictures

Apple

Spoon

Jug

Car

Colours

Red

Blue

Yellow

Green

Black

Forms

Triangle

Square

Star

Circle

Numbers

9

14

428

1569

41

Letters

B

L

J

A

E

C

Words

Cat

Knife

Picture

Car

Sentences:

I have a hat.

The girl has a cat.

The car sped swiftly down the road.

Perseverance is essential to success.

Date ...

AGNOSIA

(continued)

AUDITORY

Recognition of Sound

Coughing

Humming

Whistling

Hand-clapping

Jingling of keys or money

Word identification (Body Scheme)

Ear

Nose

Head

Elbow

Right - Left Orientation

Show me your: R hand

L eye

R thumb

L little finger

TACTILE

	Left hand.	Right hand.
Penny		
Marble		
Latchkey		
Spoon		
Small Comb		

Name ... Date ...

RECEPTIVE DYSPHASIA

AUDITORY VERBAL COMPREHENSION

1. What do people wear on their heads?

   Shoes Stockings Shirts Hats

2. What do birds move when they fly?

   Feet Wings Beak Claws

3. What is a small child called?

   Lamb Baby Pony Kitten

4. In what month is Christmas?

   December June April October

5. What is the colour of snow?

   Black Green White Red

WRITTEN VERBAL COMPREHENSION

Clap your hands.

Cough.

Smile.

Nod your head.

Name ... Date ...

APRAXIAS

dedness:

NON-VERBAL APRAXIA

Stick out tongue

Tap with fingers

Clap hands

Close eyes

Show teeth

Write with a pencil (note handedness)

Fold a paper in half

Tie shoelace

Open a lock

Cut with scissors

(Pretended actions:)

Drink water from glass

Comb hair

Use a spoon

Smoke a cigarette

VERBAL APRAXIA

Repeat 9

14

428

1569

Cup

House

Telephone

Elephant

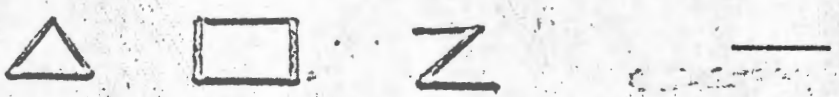
A cat drinks milk.

I may go fishing on Sunday.

We admired the flowers in the garden.

Australia is the smallest of the continents.

CONSTRUCTIONAL APRAXIA

Copy with matchsticks 

DICTATION

Name ... Date .....

EXPRESSIVE DYSPHASIA

1. Automatic speech

Count 1 - 10

Alphabet

Days of week.

Mon. Tues. Wed. Thurs. Fri. Sat. Sun.

Song

2. Spelling

Let Feeling Cabinet Suppose

3. Writing

(a) Write your name

Write ... (patient's name if he cannot do above)

Copy A E F W V K P Z

(b) Dictation

Words. Write: This month is

Most girls like to sew

Forms. Draw: Triangle Square Circle

Numbers. Write: 9 1 7 2 14 (Score 1 mark each)

4. Arithmetic

$$\begin{array}{cccc} 7 & 6 & 21 & 14 \\ +3 & \times 9 & \div 3 & -5 \end{array}$$

5. Clock Setting (1 mark each)

5.30 Quarter to eight. 12.15

6. Oral Reading

9 JUL 1975

FIGURE FORM BOARD TESTS

FOR MEASUREMENT OF DETERIORATION IN A GERIATRIC POPULATION

TOTAL NAME DATE

3 FIGURE FORM BOARD

Stage	Blocks Placed	Actual No. of Blocks	Time
1		1	( )
2		2	( )
3		3	( )

Total +

COMMENTS

2 FIGURE FORM BOARD

Stage	Blocks Placed	Actual No. of Blocks	Time
1		2	
2		2	
3		4	
4		9	( )

Total +

COMMENTS

5 FIGURE FORM BOARD

Stage	Blocks Placed	Actual No. of Blocks	Time
1		2	
2		6	
3		11	( )

Total +

COMMENTS