LANGUAGE PATHOLOGY

IN

ALZHEIMER TYPE DEMENTIA

AND

ASSOCIATED DISORDERS

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I hereby declare that
I have composed this thesis myself
and that the work here reported is my own.

A B S T R A C T

The management of dementia poses immediate problems of health care in institutions and the community. Although speech therapists have an increasing commitment to psychogerontology there are no instruments that measure or describe progressive language breakdown. Current concepts of language disturbance are based on focal lesions and are inappropriate for the diffuse neuroanatomic lesions causing dementia.

A diagnostic examination that measures and describes language pathology according to established criteria is described. It has face content value and internal consistency. It is also discriminating and provides standardisation for the aphasic, demented and healthy elderly.

"Aphasia" is established as an unsuitable term for the language of the demented following factor analysis of groups of aphasics and dements. Further investigation establishes that linguistically Alzheimer's Disease is significantly different from disorders that may be associated with it either psychologically, physiologically or anatomically.

The language of dementia is established as a disturbance of written and verbal comprehension, syntax, naming and abstract operations. There is evidence of the increasing dissociation of language operations in problem solving.

Progressive language disability in Alzheimer's Disease is described both cross sectionally and as a four year longitudinal study identifying those abilities that are more, or less, resistant to the dementing process.

The study discusses the selective impairment of verbal and non verbal skills that determine effective communication and argues their implication for management by speech therapists and other professionals.

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that I would inscribe this work,

"..... he shall mould
Thy spirit, and by giving make it ask"

CONTENTS

CHAPTER 1

INTRODUCTION

There is a vast body of literature concerning aphasia following focal brain disease but few studies of the status and breakdown of language in neuropsychiatry.

Sjogren et al (1952) in their clinical study of "Morbus Alzheimer and Morbus Pick" noted language disturbance in 88% of their cohort, Sim and Sussman (1962) noted aphasic deficits in 47% of patients in their series that attempted to differentially diagnose the dementias and Kaszniak et al (1978) has demonstrated language pathology as a predictor of mortality in senile and presenile dementia.

The literature recognises a paucity of research into the language of dementia either as small, large or longitudinal studies (Brookshire and Manthie 1980, Obler and Albert 1981, Appell et al 1982, Bayles 1984). An MRC report on Senile and Presenile Dementia (Lishman 1977) discussing "Problems Awaiting Solution" demanded a clear delineation of possible core areas of disability including "the characteristics of language disturbance in dementia" (P.5). More recently there has been a call to develop measuring instruments that define, with greater precision, speech disorders in dementia (Arie 1982).

Language pathology in the dementia is historically documented. Alzheimer (1907) paid forceful attention to language disturbance in a 51 year old patient whose syndrome now bears his name.

> "When shown objects she could name them relatively correctly. However, even her perceptions were disturbed. Immediately after naming the objects she would forget them. She drifted from one line to the next while reading - either enunciating the individual letters or speaking in a meaningless tone. While writing, she repeated single syllables, omitting others and quickly became confused. She used perplexing phrases when speaking or made paraphrasic errors ("milk pourer" instead of "cup"). She would hesitate during speech. She did not understand some of the questions put to her. She appeared to have forgotten the use of several objects."

Pick (1892), in his paper "On the Relation between Aphasia and Senile Atrophy of the Brain" emphasised the language abnormality of a 71 year old patient:

> "In observing the speech disorder we lay greatest emphasis on the fact that we are not dealing with the disorder which can exclusively, or even primarily, be attributed to simple mnestic effects of the senile process, but rather, it more closely parallels those which are the result of focal lesions; it resembles those disorders which Wernicke-Lichteim described as transcortical sensory aphasia in so far as we could determine that the patient's primary symptoms were loss of understanding of speech and writing, paraphasia and partly retained ability to speak."

The last half of the 19th century was a period of intense medical interest in the study of language and the fundamental anatomy of the brain. Charcot, the great physician of the Hospital of Salpetriere, proposed one of the earliest psychological models of language function in his Clinical Lectures on Senile and Chronic Diseases (1881). His student, Seglas, used it as the basis to

his Des Troubles du Language Chez les Alienes (1892), the first major attempt to describe and categorise language disturbances in the demented that related those disturbances to changes in the brain.

The study of aphasia grew out of this ferment. In 1861 Paul Broca, in support of the localisationist model of the relationship between brain topography and brain function reported a single post mortem study of a speechless patient with a large frontal lesion. For the next 100 years there continued an established debate between "Holists" who argued that cortical tissue was equally potential, and "Localisationists" who argued that there were areas of the brain that were specialised for the execution of specific abilities. These two schools have their parallels in cognitive psychology: those who see a concept of intelligence as a general ability pervading intellectual functioning after Spearman (1923), and those who hold to Thurstone's (1938) model of primary mental abilities. More recently non invasive metabolic and blood flow studies of speech and language (Risberg and Ingvar 1973, Ingvar and Schwartz 1974, Knopman et al 1980, Mazziotta et al 1982) have supported concepts of functional cortical systems as the basis of higher mental abilities.

"Aphasia" is a psychological term that describes language pathology following focal lesions of the dominant hemisphere. It has been borrowed to describe disorders of language in organic brain syndrome, psychosis and the language of developmental delay. Many linguists, neurologists and psychiatrists prefer the word to describe language pathology accompanying focal lesions (Lesser 1978, Lhermitte and

Gautier 1969). Weinstein and Khan (1952) talk of "non aphasic" disorders of language" to describe the output of the demented, a term which appears contradictory. Critchley (1964) suggested "dyslogia" be used to describe the language of the demented.

It is necessary to differentiate disorders of speech and language as neuromuscular disorders affecting articulation and prosody, or the breakdown in formulation of verbal or graphic symbols necessary for communication and thought (Sokolov 1972).

There are four problems facing the speech pathologist investigating the language of dementia. The first is aetological. Aphasic syndromes are usually caused by focal vascular, neoplastic or traumatic lesions but the aetology of Alzheimer Type Dementia (ATD), although increasingly described, remains uncertain.

Second, the onset of dementia is insidious and it is difficult to differentiate, linguistically, from normal ageing.

The third problem is nosological. There are few systematic studies of language in dementia and fewer into language sub types of the disorder. Aphasic disorders with focal disease produce distinctive patterns of linguistic deficit that have long been classified into syndromes, but diffuse lesions of the cortex produce less apparent symptomatology. Although a few studies suggest characteristics of demented language there are none that conclusively differentiate the dementias on linguistic criteria (Perez et al 1975, Skelton-Robinson and Jones 1984, Bayles et al 1985, Holland et al 1985).

Finally, language pathology in dementia raises the question of the relationship between language, cognition and thought. Dementia has been described as a disturbance of memory, intellect and personality. In aphasia personality is retained, memory may be selectively impaired, and intelligence, although often reduced, is not grossly disturbed. Roth and Meyers (1975) observed that physicians must differentiate between dementia and other organic syndromes. The speech pathologist needs to differentiate pathological language from primary mental disorders. (Strub and Black 1981).

The four problems raise three working hypotheses that may be expressed as statements:

(1) that aphasia is not a feature of language in dementia

(2) that language pathology differs according to the disease that causes it

(3) that language will deteriorate in progressive dementia.

Language may deteriorate in senescence and breakdown in vascular and degenerative disease. Alzheimer's disease is the most significant of the degenerative dementias and in the western world is more prevalent than vascular dementia. This thesis will begin by describing Alzheimer's disease historically, epidemiologically, neuropathologically, its behavioural effects and methods of investigation.

Because Alzheimer type changes exist in normally ageing brains, and because dementia has been described as a continuum of normal ageing, or a speeding up of the normal ageing process, language breakdown also needs to be examined on a continuum. There are no reported studies of the longitudinal breakdown of demented language, particularly demented language of Alzheimer type. Nor are there any studies that systematically compare the language of ATD to the language of aphasia, normal ageing or disease in an attempt to investigate its nature. There are no studies that compare language breakdown with the neuropathological passage of the disease.

Communication changes occur in normal ageing and as secondary to vascular and degenerative disease. Chapter 3 discusses the effects of normal and abnormal ageing on voice, speech and language. It both evaluates the literature reporting language pathology in dementia and evaluates recent developments in scanning and imageing language and brain function.

Chapter 4 describes an instrument used to assess neurolinguistic acts in normal, aphasic and dementing populations. It argues a rationale for test inclusion according to established criteria for test construction in the field of language pathology. The chapter also provides a description of subject groups used to standardise the language battery and experimental groups used to delineate and compare language performance. It then discusses appropriate statistical procedures for the identification, description, comparison and mapping of language in neuropsychiatry.

The fifth chapter gives the results of the investigation of language performance in selected groups including the predictive power of the test battery. It describes how on the standardised format of the Language Scales individuals may be compared to normal or appropriately impaired populations. It highlights the lack of an even distribution of language scores arguing the use of non parametric statistical analysis and describes the neurolinguistic performance of aphasic, demented and controlled populations. It correlates language acts to severity of cognitive impairment and demonstrates on an inter-correlation matrix the internal consistency and comparability of declining scores.

Whether the language of dementia can be called "aphasia" is discussed in Chapter 5 following factor analysis of the disorders. Following this quantitive analysis the quality of language in ATD is both illustrated and discussed. Again, neurolinguistic data of ATD is examined both longitudinally and cross-sectionally. The interaction of various linguistic and spatial abilities that are used to problem solve are examined for dissociation of function. Finally the language of ATD is cross compared with the language of other diseases that might mimic it to demonstrate its uniqueness.

The working hypotheses stated in this chapter are considered and discussed in Chapter 6 and recommendations for further research are both stated and elaborated.

CHAPTER 2

THE SYNDROME OF ALZHEIMER TYPE DEMENTIA

2.1 INTRODUCTION

Dementia is characterised by changes in personality and intelligence. Fisher (1968) widened the definition to include linguistic capacity defining dementia as "a reduction in any or all of the higher verbal and non verbal cerebral activities". Such loss of cognitive abilities requires cortical and sub cortical dysfunction producing a failure in the relationship between words and meaning, (aphasia) recognition and perception (agnosia) and intention and action (apraxia).

In dementia cognitive deficits are partly recognised by the failure of planning, inflexibility, perseveration, decreased learning ability, impoverished ideation, the loss of both abstract thinking and conceptual shift. Emotional changes may include a coarsening of personality, blunting of emotion, querulousness, pathological lability or euphoria and the personality being reduced to a caricature of the individual's worst features (Roth and Myers 1975).

In the last decade dementing illness, and ATD in particular, has been better understood by clinical criteria that differentiate neuropathologies (Hachinski et al 1975, Glen and Christie 1979, Gustafson and Nilsson 1982). Such clinical criteria are increasingly supported by investigations including CT scanning, NMR scanning, EEG, cerebral blood flow and metabolic studies.

2.2 AETIOLOGY AND EPIDEMIOLOGY

Dementia is a psychosyndrome secondary to trauma, infective or toxic states, deficiency disease, hypoxia, space occupying lesions or vascular, endocrine, metabolic and degenerative disorders. Such disorders may be primarily cortical, as Alzheimer's and Pick's Disease or subcortical as Parkinson's and Wilson's disease. The syndromes and classifications of dementing illness have recently been reviewed by Kellet (1982) and Bridges and Jolly (1985). They are summarised in Table 2.1.

2.2.1 Prevalence

The epidemiology of dementia is becoming increasingly socially and economically significant as more people live to a longer age and the cognitive ability of the elderly changes over the generations (Schaie and Gribbin 1975).

A study by Kay et al (1970) in the Newcastle area gave a total prevalence of 6.2%, increasing from 2.3% of the population in their sixth decade to 22% in the eighth to ninth decade. There are indications that the population increase is now dramatic above the eighth decade (Mortimer 1983). The prevalence of ATD plateaus in the ninth decade (Tomlinson and Kitchener 1972) and it is more severe and progressive in the younger patient (Constantinidis 1977).

Mortimer (1983) notes that, using 4.15% as a median prevalence rate of dementia in his epidemiological review of patients over 65 that the disease prevalence is comparable to Parkinson's disease and exceeds any other degenerative neurological disease.

Table 2.1

A CLASSIFICATION OF THE CAUSES OF THE DEMENTIA SYNDROME

1 Intracranial Space-occupying Lesions

Subdural haematoma, brain abcess, tumours primary and metastatic
Normal pressure hydrocephalus

2 Traumatic

Single severe head injuries, repeated less severe injuries, eg 'brain-damaged boxers'

3 Infections

Brain abcess, meningitis, encephalitis
Neurosyphilis, subcute sclerosing panencephalitis, progressive multifocal leucoencephalopathy
Creutzfeldt-Jakob disease, Kuru

4 Vascular Disorders

Multi-infarct dementia, occlusion of the carotid artery
Binswanger's disease, arteriovenous malformations
Inflammatory conditions of blood vessels: SLE thromboangiitis obliterans

5 Hypoxia and Anoxia

Anaemia, post anaesthesia, carbon monoxide poisoning, cardiac arrest, respiratory insufficiency

6 Metabolic Disorders

Renal failure, hepatic failure, remote effects of carcinomas

7 Toxic States

Intoxication with heavy metals: lead, mercury, manganese
Organic compounds: nitrobenzenes, anilene compounds, bromine hydrocarbons, tri-ortho-cresyl phosphate, carbon disulphide, carbon tetrachloride
Drugs; bromides, barbiturates, phenacetin and a very large number of others, especially in combinations
Alcohol

8 Endocrine Disorders

Myxoedema, Cushing's syndrome, hypopituitary syndromes, hypoglycaemia, parathyroid disorders

9 Deficiency Diseases

Pellagra, Wernicke-Korsakoff states
Vitamin B_{12} deficiency concentration camp syndrome

Table 2.1 (Contd.)

10 Miscellaneous

Multiple sclerosis, muscular dystrophy, Whipple's disease familial calcification of the basal ganglia

11 Degenerative Disorders of the CNS

(a) Principally cortical:

Alzheimer's disease, Pick's disease, Schilder's disease

(b) Principally subcortical:

Parkinson's disease, Huntington's chorea, Wilson's disease, progressive supranuclear palsy, Friedreich's ataxia

Tomlinson et al (1970) found half the brains of their institutionalised sample has lesions characteristic of ATD at autopsy.
This figure has been confirmed by the WHO report of 1981 which reports that 50-60% of the cases seen in clinical practice show characteristic ATD. Multi Infarct Dementia (MID) accounts for 15% of the cases and a mixed type of dementia for 10%. St. Clair and Whalley (1983) reported 89 deaths at the Royal Edinburgh Hospital from 1959-78 with a diagnosis of presenile dementia. On the basis of neuropathological findings 46 were diagnosed ATD, 27 MID and 16 were mixed. The average age of death was 61.1, 65.3 and 66.7 years respectively. Seventy five percent of the ATD cases and 66% of the MID cases were female.

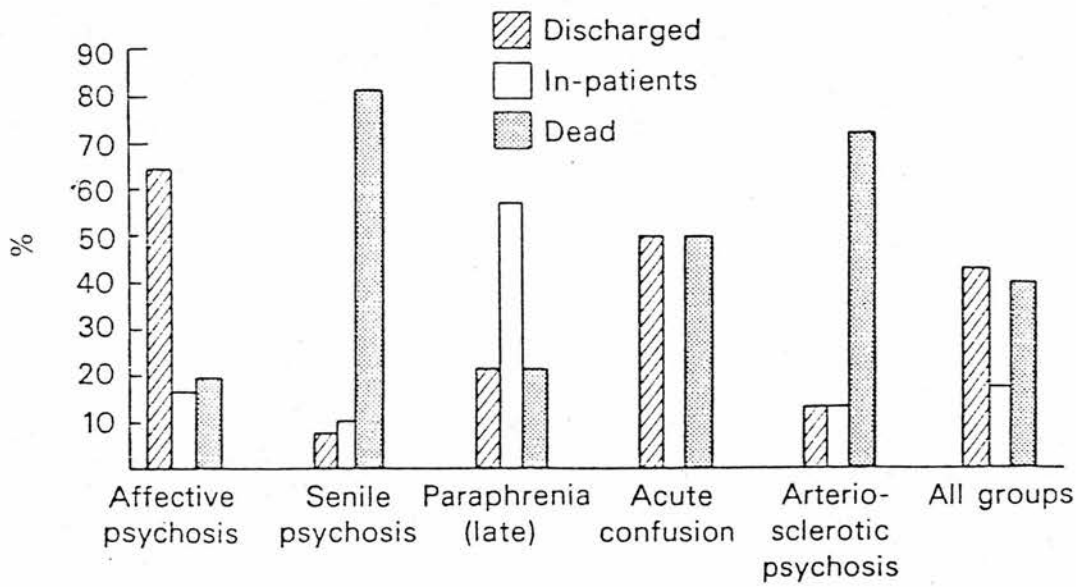
At present there are an estimated half million demented people in the UK (Bridges and Jolly 1985) and half of these account for ATD, at one in twenty at the age of 65 and one in four at the age of 80 (MRC Report 1983). In the UK most of these may be in the community and not reported to their GP's (Williamson et al 1964) while in some countries (for example, Australia) they may be institutionalised (Henderson and Jorm 1986).

2.2.2. Incidence

Incidence, or the frequency of new cases, requires a monitored study of those existing patients. Kay (1972) suggests an annual incidence rate of 1.4% for a combination of senile and arteriosclerotic dementia over 65 years. The incidence is confirmed by other studies. Mortimer (1983) and Larsson et al (1963) have estimated the lifetime risk of contracting senile dementia at 1.8% for males and 2.1% for females.

Figure 2.1

LIFE EXPECTANCY OF DEMENTED PATIENTS. STATUS OF 318 PATIENTS TWO YEARS AFTER ADMISSION
(After Miller 1984)



Kay (1972) estimated that dementing males have only about a third, and females a quarter, of the life expectancy of normals. Nielsen et al (1977) suggested the mean survival time of patients with senile dementia is 2.6 years compared with 7.8 years for normals. Figure 2.1 reports life expectancy after Miller (1984)

Barclay et al (1985) gave 3.4 years as the 50% survival duration of cases with ATD. Survival of institutionalsed patients is increasing (Blessed and Wilson 1982, Christie 1982, Christie and Train (1984), perhaps because common infections as pneumonia are better managed (Gruenberg 1977, 1978).

2.2.3 Familial Studies

Sjogren et al (1952) and Pearce and Miller (1973) have reported features of ATD including onset age of presenile forms, duration of illness and familial aggregates. They are also reported by Pratt (1967), (1970) as "six times in sibships, eight times in two generations, four times in three generations and one in five generations". The Sjorgren study placed morbidity risks at 10.7% for parents and 3.8% for siblings as compared to the Swedish general population risk of ATD at .1%. The rates are generally confirmed by a Swiss study by Heston (1976). Heston and Mastri (1977) reported higher risk by sampling wider severity groups. In one twin study Kallmann (1953), quoted Matsuyama (1983), reports an 8% risk for dizygotic twins and 42.8% for monozygotic twins with a frequency of 6.5% for siblings and 38% for parents.

Brietner et al (1984) demonstrated the risk of dementia in first degree relatives was less than half for ATD probands with language disturbance compared with zero risk in those relatives of ATD patients without language disorder.

2.3 HISTORICAL BACKGROUND

Senile dementia as a clinical condition was first described by Esquirol (1838):

> "senile dementia is established slowly, it commences with infeeblement of memories, particularly the memory of recent impressions, sensations are feeble, the attention, at first fatiguing at length becomes impossible, the will is uncertain and without compulsion, the movements are slow and impractical."

The neuropathological condition was described by Alois Alzheimer in 1907, the clinical condition was described pregressively in three

phases by Sjogren et al (1952), the medical epidemiological condition was described by Tomlinson, Blessed and Roth in 1968 and 1970.

2.4 DIFFERENTIAL DIAGNOSIS

Although Alzheimer Type Dementia follows neuropathological diagnosis clinical rating scales (Hachinski et al 1974, Glen and Christie 1979, Gustafson and Nilsson 1982) have demonstrated remarkable validity and reliability in the differential diagnosis of dementia (Harrison et al 1979).

Frackowiak et al (1981) was able to subdivide dements into vascular and degenerative groups, relate the number of infarcts to Hachinski's Ischaemic Score, and correlate reduced cerebral blood flow and cerebral oxygen utilisation with the severity of dementia. Vascular patients demonstrate greater focal abnormalities of oxygen uptake (although they vary individually) and wider focal neurological signs, aphasia, dysarthria, gait disturbance and brisk reflexes. Aetiologically, Ladurner et al (1982) demonstrated that only hypertension correlated significantly with MID. The degree of cognitive decline or dementing illness depends on the localisation, extent, nature and chronicity of lesions.

Many physiological and psychological investigative techniques indicate not so much a differentiation of the dementias but the severity of the dementia. Ingvar and Gustafson (1970) determined

that decreases of the mean cerebral blood flow in dementia were related to the degree of dementia rather than type. Frackowiak et al (1981) have noted Positron Emission Tomography differentiating ATD from controls, but no correlation between type of dementia and magnitude of oxygen metabolism. Perez et al (1976) were not able to differentiate ATD from MID on WAIS subtests.

In early ATD the brain may appear normal but in advanced cases may suffer a loss of 400-500 gms compared with normals. Cortical atrophy is marked by shrinkage of the gyri and widening of sulci. Tissue loss is mainly in grey matter, white matter loss being secondary. The temporal, frontal and to a lesser extent, the parietal lobes are involved. Primary projection areas, the sensory motor, auditory and calcarine areas are spared (Brody 1955, Bowen et al 1979). There is moderate widening of the ventricles, especially the temporal and temporo parietal horns, reflecting a loss from white matter tracts. Temporal areas are particularly affected, the hippocampus and the amygdaloid are shrunken (Ball et al 1985). In elderly ATD atrophy is a less useful indicator since age related changes in normal and non demented peopple may be equal to that in ATD. Hubbard and Anderson (1981) noted that above 80 years the temporal lobe was the only cortical region significantly smaller in ATD. Regional changes in presenile forms are therefore similar, though less pronounced, than late forms (Brun 1983).

Microscopic and macroscopic changes in ATD and MID show marked differences. Changes in MID are asymmetrical whereas ATD changes are predictable in both hemispheres. Pick's disease produces changes frontally and temporally but not to the cingulate or parietal areas (Brun 1983).

It seems that ATD is a pathology of the cortico-limbic area with regional and particular association zones involved, especially the temporal area which can demonstrate up to 20% cortical shrinkage (Hooper and Vogel 1976, Bowen et al 1979).

Association areas account for over 70% of the cortical surface. A decrease of this tissue is responsible for reduced integration of specific information into meaningful wholes and therefore diminishes cognitive function (Scheibel 1983).

2.4.1 Microscopic Changes

The characteristic microscopic changes in ATD are neurofibrillary tangles (NFT) and senile plaques (SP). Neurofibrillary tangles also occur in Downs Syndrome, dementia Puglistica and in the Parkinsonian dementia complex of Guam.

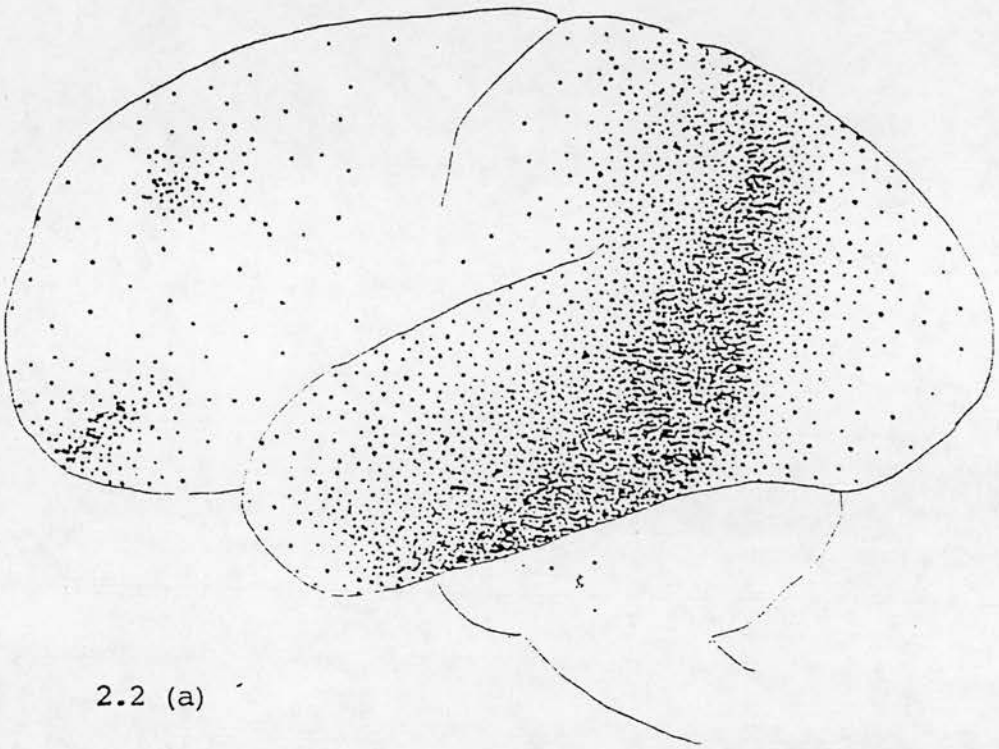
NFT's associated with ATD are most abundant in the archicortex as opposed to the neocortical regions. Small numbers of NFT's may occur in the hippocampus of the normal elderly. In early or moderate ATD the pyramidal neurons are most involved particularly on cortical layer three. In more severe cases NFT's are involved in all cortical layers and both pyramidal cells and granular interneurones.

The effects of these microscopic patterns as seen by light microscopic changes (See Figure 2.2) have been correlated to reduced glucose metabolism demonstrated by Positron Emission Tomography (Ferris et al 1981) and regional Cerebral Blood Flow using Xenon ^{133} either

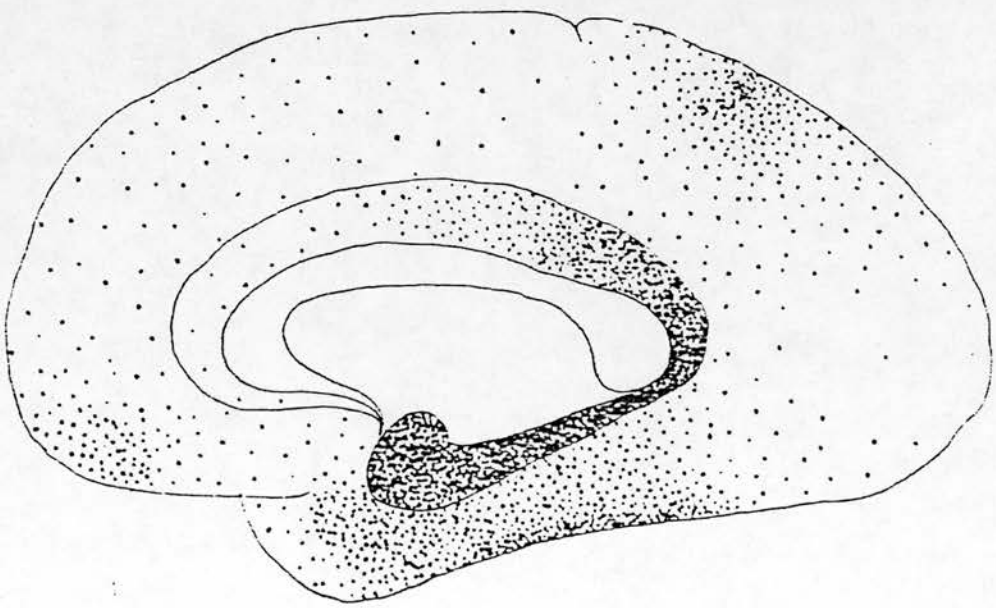
Figure 2.2

SCHEMATIC REPRESENTATIONS OF DISTRIBUTION AND SEVERITY OF DEGENERATION ON (a) LATERAL AND (b) MEDIAL ASPECT OF THE BRAIN IN AN AVERAGE ALZHEIMER CASE. THE DARKER THE AREA, THE MORE PRONOUNCED THE PLAQUE COUNT. WHITE AREAS ARE SPARED, WITH ONLY BASIC CHANGE DISCERNIBLE

(After Brun 1983)



2.2 (a)



2.2 (b)

injected (Ingvar and Gustafson 1970, Ingvar et al 1978) or inhaled (Risberg 1980).

The other characteristic feature of ATD is the marked increase in the occurrence of senile plaques. They also occur in normally aged brains and their number has been shown to be related to the degree of dementia (Blessed et al 1968). The plaques are surrounded by degenerating neurites and often accompanied by spongiosis, neural loss and blurring of the cortical cytoarchitecture. Perry and Perry (1985) have recently identified aluminium associated with silicon in the amyloid core but it remains uncertain whether this is a specific stimulus to plaque production or if it is just a form of mineralisation.

Granulo-vacuolar degeneration is reported as a striking abnormality in the neurons of the hippocampus in ATD (Tomlinson and Kitchener 1972). Their incidence in ATD exceeds that of age matched controls, and, along with NFT's, are present in the posterior half of each hippocampus.

The passage of the disease is not uniform but the order of severity extends from the hippocampus and amygdala the inferior temporal area (Brodman 20, 21), post central parietal areas (Brodman 7b, 19 39), posterior cingulate areas (Brodman 23) and the frontal lobes anterior to the precentral gyrus. The anterior cingulate (Brodman 24), sensory motor cortex (Brodman 4-7a) and calcarine area (Brodman 17) are relatively spared (Brun 1983).

Pearson et al (1985) has described NFT's and SP's occurring in the temporal, parietal and frontal areas, and while NFT's predominantly

occur in clusters in layers three and especially five, SP's occur unclustered in layers two and three. Since the pyramidal cells of layer three project intracortically and those of layer five project extracortically, and since tangles cluster while plaques do not, there is a suggestion that the disease may spread by pyramidal fibres. The tangles may represent an early degeneration of specific cortical pathways and the plaques, spread randomly by pyramidal fibres, degenerating structures beyond the cortex.

Ferry (1985) has reviewed the passage of Alzheimer type changes through the cortex suggesting a spread of fibres linking pyramidal cells and moving from areas where plaque and tangle counts are rarest, the primary projection zones, to the association areas of the temporal, parietal and frontal lobes. Pearson et al (1985) and Ball et al (1985) have suggested its spread from the hippocampal area.

2.4.2. Neurochemical Aspects

A finding in the neocortex of ATD brains at postmortem, and temporal lobe biopsy tissue from ATD brains (Sims et al 1980), demonstrates a reduction of choline acetyltransferase (CAT). CAT is responsible for reduced synthesis of the neurotransmitter acetylcholine (ACh) from its immediate precursor acetyl-coenzyme A. The enzyme is predominantly localised in synaptic terminals to which it is carried by slow axonal transport following synthesis in the cell body. In ATD there is a marked reduction of CAT at 20% of that in the normal hippocampus and the reduction of CAT is related

to the plaque count (Perry et al 1978). The neurochemical hypothesis for the aeteology of ATD has recently been reviewed by Perry (1984).

The neurochemical changes in ATD are not restricted to the cholinergic system although a reduction in cholinergic markers remains the most extensive and consistent abnormality (Perry 1984). There are reductions of cholinergic activity in the cortical cholinergic projections from the basal forebrain. There are also reductions in markers for the ascending noradrenergic and serotonergic systems with cell loss in the locus ceruleus and extensive NFT formation in the raphe nuclei (Hardy et al 1985). Only one intrinsic neurotransmitter, somatostatin, has been shown to be reduced in ATD. Deficits in neurotransmitter systems other than the cholenergic system appear to be more pronounced in younger cases of ATD (Rossor and Iversen 1986).

The formation of memories may relate to the specialisation of certain neurones which signal to each other over synapses by neurotransmitters. As a result of learning some synapses may become more efficient in the transmission of information between neurones and this enhanced efficiency may be a prerequisite to memory. Cholinergic neurotransmission has been associated with the facilitation of memory acquisition and storage.

Deutsch (1974) and Deutsch and Rogers (1979) have strongly argued that cholinergic transmission is involved in learning and memory in animals and that amnesia is consequent upon less cholinergic activity in the brain. Recent research suggests memory improvement

with enhancement of cholinergic activity. Cooper (1984) has recently reviewed studies of the effects of cholinergic drugs on memory. Physostigmine, an acetylcholinesterase (AChE), which increases ACh at synapses improves long term memory and studies with scopolamine, an antagonist which blocks the effects of ACh, impairs memory. There is, however, little indication that choline treatment causes significant improvement.

2.4.3 Psychological Measures

Intelligence has been described by Horn and Cattell (1966) as "fluid" or "crystallised". "Fluid" ability is independent of education and measured by tests of inductive reasoning. It is vulnerable to destruction. "Crystalline" intelligence includes habits that are based on experience or general information. They are in need of continual referral and are better preserved. This model appears to be demonstrated by dements who give better performances on information scores of the WAIS. Such preservation of the passive lexicon over the years makes favourable comparisons with younger age groups (Baer 1972, Goldstein and Shelly 1975, Cunningham 1975). Verbal ability may therefore suggest premorbid intelligence (Nelson and O'Connell 1978).

This dichotomy of "crystalline" and "fluid" abilities compares with Goldstein's (1948) "concrete" and "abstract" attitudes. Walker (1982) gave the Colour Form Sorting Test (Goldstein and Sheerer 1941) to a group of elderly normal controls, a group of mild and moderately demented subjects and determined a linear progression in normals of failure from a fail rate of 10% at 60-64 years to 92% at 80+ years. Failure in the demented groups was 100%.

Hart and Spearman (1914) and Eysenck (1945) attempted to find an underlying factor responsible for diminished performance in senile dementia and concluded a failure of /g/ factor. Using Cattell's (1943) distinction between tests of fluid and crystalline ability one may assume /g/ saturated with crystalline ability. Gustafson and Hagberg (1975) argued from their investigations of presenile dements that there were three factors: amnesia-apraxia, amnesia-confusion, and agraphia-alexia.

Psychological testing in dementia has traditionally used multi-factorial standardised intelligence tests as, for example, The Wechsler Adult Intelligence Scale (Wechsler 1958). The scale has consistently shown poorer performance of diagnosed dements against the normal elderly, with a discrepancy between "verbal" and "performance" scores. Verbal abilities are less vulnerable (Bolton et al 1966, Ron et al 1979). The use of the Mill Hill Vocabulary Scale and the Progressive Matrices have also confirmed vocabulary scores as consistently better than the matrices (Miller 1977). Roth and Hopkins (1953) suggested the differential between verbal and performance scores may differentiate ATD. Miller (1977) has noted ATD patients show greater variability of scores and Reisberg (1983) has commented on the progressively diminishing scores of serial testing.

Alexander (1973) determined Paired Associate Learning, Nonsense Syllable Learning and Block Design"the best measure of discriminating brain damaged subjects from those with functional disorders". Crookes (1974) argued Block Design to be the best predictor of early dementia. Perez et al (1975) ranked Block Design above Similarities,

Performance IQ, Information, Picture Arrangement, Verbal IQ, Full IQ, Age, Education, Picture Completion and Arithmetic as being sensitive to dementia.

Bayles and Boone (1982) have argued that specific language tasks from the WAIS are more sensitive than the performance tasks, especially Similarities and Nonsense Syllable Learning and only mental status questionnaires have the discriminant value of such language tests. They have argued that Block Design, as a test of reasoning, demands the patient to grasp relations and test an hypothesis. Vocabulary tests only demand word definition but Verbal Similarities demand sensitive reasoning by judging commonalities. Failures on such tests demonstrate failure of language and thought. The relationship of thought and language has been debated indefinitely but it is impossible to assess language comprehensively and not assess thought and it is generally very difficult to assess thought without assessing language.

2.4.3.1 Neuropsychological Investigations

The strength of the Luria Neuropsychological Investigation is that it has pass or fail criteria and investigates the impairment of "cortical functions" that underpin clusters of tasks that rely on particular functions. This is in accord with Luria's interest in the factors that underlie constellations psychological processes. Its weakness is that it is not standardised nor does it have standardisation of procedure (Golden et al 1980).

Two studies have investigated dementia using Luria's battery. Ernst et al (1970) reported in abstract form the Luria battery's assessment of nine dements. It indicated phonemic hearing normal but comprehension of logical and grammatical structures impaired. They cited visuo-spatial and mnestic deficits responsible for reduced comprehension. Repetition and automatic speech were impaired. There was a "poverty" of speech and "a lack of language initiative ". The study concluded dements were separable from aphasics by memory loss, visuo-spatial deficits, echolalia and perseveration.

Blackburn and Tyrer (1985) were able to differentiate ATD from Korsakoff syndrome and age matched controls with sub sections of the Christensen (1975) Luria Neuropsychological Battery and an orientation scale from the Wechsler Memory Scale (Wechsler 1945). The battery did not differentiate, clearly, normals and Korsakoff patients except on tests of memory, logical thinking and orientation. Patients with ATD showed reduced scores that did not correlate to age or duration of illness. Reduced performance on all subtests in the battery differentiated degrees of severity within the Alzheimer cohort. Blackburn and Tyrer suggest the value of such neuropsychological measures in describing and predicting the rate of deterioration for clinical management.

2.4.3.2 Memory

Memory loss is one of the most prominent clinical findings in dementia (Roth 1980). Miller (1977) reported failure of short and long term memory. Albert et al (1981), Wilson et al (1981) and

Moscovitch (1982) have all reported a failure of remote memory although Boyd (1936) had suggested recent memory was more vulnerable than remote memory. Kral (1978) described initial memory loss as "benign senescent forgetfulness" meaning a loss of memory for details but retention of episode compared to malignant memory loss being a loss of both recent and remote information. Senescent forgetfulness is common to 80% of the elderly population and is reportedly pathological in up to 15% of that population (Reisberg 1983).

Inglis (1957) reported memory failure in presenile dementia was related to coding new information rather than storage or retrieval. Miller (1973, 1975) following Milner et al's (1968) work on memory loss following bilateral ablation of the basal temporal areas, demonstrated the failure to pass information into storage from intact short term memory. He determined that short term storage is impaired in ATD as tested by recall of word lists and that there is also a difficulty of putting information into storage.

Failure of immediate memory may relate to attention (Caird and Inglis 1971), reduced acoustic coding (Miller 1971, 1972) or rehearsal in the "executive system of working memory" (Morris 1968). There is evidence of a recency effect in dementia (Miller 1971) as well as a failure of serial order recall (Morris 1984) or recall with interference (Corkin 1982).

2.4.3.3. Perception

Perceptual disturbances in dementia have been noted by Williams (1956) and Ernst et al (1970) who describe praxic and gnostic disabilities while Gustafson and Nilsson (1982) have noted dressing

apraxia as a disturbance of the perception of space. Williams (1956) also noted dements' poor performance on spatial tests using mazes. Lawson et al (1968) commented on the distractability in demented patients and Birren and Botwinick (1951) noted poor copying skills related to motor speed.

2.4.3.4 Rating Scales

The purpose and design of rating scales has been reviewed by Robinson (1979). He defines three types of scale, those that assess cognitive ability, as that of Blessed et al (1968), those that assess behavioural functioning, as the Clifton Assessment Scale (Pattie and Gilleard 1975) and those that assess mood change, as the Hamilton Depression Scale (Hamilton 1960).

Diagnostic scales also attempt to classify organic psychiatric disorders. The Hachinski Index (Hachinski et al 1975) aims to differentially identify MID by an ischaemic score of 13 features. The scale is reported to have a validity and clinical practicality: Harrison et al (1979) differentiated vascular and non vascular dements by the Index, Loeb et al (1980) recommended it as a valuable clinical tool for differential diagnosis and Rosen et al (1980) validated it clinico pathologically. Frackowiak et al (1981) found all but one of their cohort could be classified by the score confirmed by CT and autopsy findings.

Glen and Christie (1979) have reported inclusion and exclusion criteria in the indentification of patients presenting ATD, Pick's disease and MID based on age, medical history, neurological and physical examination, EEG, CT and additional psychological measures.

A scale has been reported which includes the Hachinski Index by Gustafson and Nilsson (1982). It differentiates ATD, Pick's disease and MID on a 12, 9 and 13 point scale respectively.

Other scales have reported patterns of breakdown in dementing illness describing not only the symptoms of ATD but the clinical progression of the disease. Sjogren et al (1952) divided the disease into three clinical stages based on cognitive decline. Stage I is characterised by disturbances of memory and spatial disorientation. Stage II is characterised by increasing disorientation and disturbance of praxis, gnosis and language while Stage III has vegetative status. These stages are reported and confirmed by Pearce and Miller (1973) and Reisberg (1983).

2.4.4. Electroencephalography

The value of special diagnostic procedures as electroencephalography, averaged evoked potentials, cerebral blood flow and positron emission tomography over CT studies is that they demonstrate brain function in dynamic states. EEG has demonstrated its use as a test sensitive to specific disease aiding diagnosis by exclusion. Muller and Schwartz (1978), quoted Busse (1983), have commented that diffuse slow EEG activity suggests Alzheimer's disease, while intermittent lateralised activation is suggestive of flow problems related to arteriosclerosis. Johannesson et al (1977, 1979) have noted that patients with Alzheimer's disease have progressive slowing of the EEGs late into dementia while Pick's disease may present a normal EEG late into the illness. There is also a good

correlation of EEG to cerebral blood flow and metabolism in old age (Orbrist et al 1963), and a relationship between EEG and mental impairment: focal abnormalities exist in the EEG of vascular rather than non vascular dementia (Roberts et al 1978, and Johannesson et al 1979).

Investigations of the use of auditory evoked potentials in the diagnosis of Alzheimer's disease have focused on the P3 positive wave occurring at approximately 300 milliseconds following a novel stimulus. P3 latency appears to depend on the time the subject takes to complete the identification and evaluation of the significance of the signal. The amplitude is influenced by psychological variables including the unexpectedness of the stimulus as well as its complexity being the difficulty of the task required of the subject (Blackwood et al 1985, Patterson et al (1983). St Clair et al (1985) in comparing the effects of P3 on Alzheimer patients with Korsakoff syndrome and normal ageing found longer latency and smaller amplitude in ATD.

2.4.5 Computerised Tomography and Nuclear Magnetic Resonance

Computerised Emission Tomography (CT)demonstrates degrees of cortical atrophy and ventricular enlargement particularly after the seventh decade in normals (Barron et al 1976). The diagnosis of senile dementia using CT has been inconsistent and its value has been for exclusion criteria (Caird 1985). While relationships between ventricular size and cortical atrophy are reported as a poor predictor of mental decline (De Leon et al 1979, Jacoby and Levy 1980),

Soininen et al (1982) and Mann (1973) have noted ventricular dilation and not cortical atrophy show better correlation to intellectual failure, but Ron et al (1979) has disputed this. Caird (1985) has commented that ventricular widening is evident in ATD and infarcts are demonstrated in MID.

Some conclusions may be drawn from studies of CT and ATD. Advancing age is associated with cerebral atrophy in both normal ageing and dementia although dements show greater atrophy than normals of equivalent age (Fox et al 1975). However, there is overlap and the degree of atrophy provides little information about dementia in individual patients. Whether ventricular dilation or sulcal widening is better related to dementia is not certain. In all, studies have suggested the role of CT in the evaluation of dementia is limited to ruling out infarcts and mass lesions (Wilson et al 1982). The later development of Nuclear Magnetic Resonance (NMR) scanning spectography has allowed the imageing of brain tissue content in coronal, sagittal and transverse axial views. It also makes the possibility of measuring cortical thickness both locally and generally. NMR studies of the normal brain are still in their infancy but Besson et al (1983) has differentiated ATD from MID on proton density using NMR.

2.4.6 Cerebral Blood Flow

ATD is accompanied by reduced cerebral metabolism, blood flow and onset of psychiatric symptoms (Gustafson and Risberg 1974, Gustafson 1979). Ingvar and Gustafson (1970) found reduced flow over temporal, parietal and occipital borders and Hagberg and Ingvar (1976) found reduced regional cerebral blood flow, especially in

grey matter, proportional to cognitive reduction as measured by a battery of intelligence tests. There are strong correlations between specific cognitive tasks and regional cerebral blood flow. Reduced flow over temporal regions in patients with memory disturbance, and reduced flow over the posterior cortex, confirms Luria's (1966) and Gustafson et al's (1978) classical theories of dynamic functional organisation of the cerebral cortex (Risberg and Ingvar 1973).

Cerebral blood flow studies have also shown correlations of flow to severity, duration of illness, and degree of atrophy (Meyer 1983). It can be used to differentiate MID from pseudo dementia and ATD (Yamaguchi et al 1980).

Reductions in cerebral flow may be greater over the dominant hemisphere at specific loci depending on the nature of the task, but hemispheric asymmetry in ATD has not yet been unequivocally established. (Lassen et al 1960 , Ingvar 1983). Flow remains high in the peri Rolandic area, the areas of the Sylvian fissure and primary occipital areas (Ingvar and Lassen 1979, Brun 1983), also suggesting ATD as a disease of the association areas (Ingvar et al 1975). Such observations confirm neuropathological investigations that ATD seldom presents sensory motor deficits, cortical deafness or cortical visual agnosia (Wolstenholme and O'Connor 1970).

CBF studies have been able to differentiate ATD in which there are recognised flow deficits from pseudodementia where flows are more normal (Gustafson and Risberg 1979, Mathew et al 1980). Pick's disease shows reduced uptake in temporal and frontal areas by both

inhalation and injection methods, while MID demonstrates bilateral abnormalities rather than symmetrical deficit and focal flow diminution (Gustafson and Risberg 1974, 1979).

2.4.7 Positron Emission Tomography

Positron Emission Tomography (PET)is a technique which can be used to measure brain functions including the metabolism of deep structures. Positron labelled compounds are used to measure blood flow, oxygen uptake and glucose utilisation. Glucose utilisation is mapped with 18 fluoro deoxy-glucose (FDG) injected as a tracer. There is no clear evidence of age related changes in oxygen and glucose metabolism (De Leon et al 1983). Several studies have compared glucose utilisation in ATD with that of age matched control subjects (Ferris et al 1980, Foster et al 1983, Foster et al 1984).

Foster et al (1984) reported, compared with control subjects, a 10%-49% reduction in glucose metabolism in ATD. The posterior parietal and contiguous portion of the posterior temporal and anterior occipital lobes were most affected with the frontal cortex relatively spared. Patients with visuo spatial or language deficits demonstrate left-right asymmetries. They also suggested that glucose metabolism occurs before cognitive impairment, but in late stages small metabolic decrements are associated with marked deterioration of intellectual function.

Certainly hypometabolism exists in the tertiary posterior cortex and is a consistent finding in ATD. This may be responsible for the similarity of ATD and the effects of cerebro vascular disease in the dominant posterior cortex (Benson et al 1982).

Changes in ATD as described by CBF and PET using glucose utilisation have demonstrated loss of metabolism in the classical language areas of frontal, temporal and parietal lobes (Gustafson et al 1978, Foster et al 1983). There remains the problem raised by Foster et al (1983) on the variability of the relationship of cortical metabolism to cognitive tasks as demonstrated on batteries of tests which are multifactorial in design.

CHAPTER 3

LANGUAGE IN NORMAL AGEING, CEREBROVASCULAR AND DEGENERATIVE DISORDERS

3.1 INTRODUCTION

Both "aphasia" and "dementia" are psychological terms that describe the behavioural effects of disease processes. Thus Jackson (1878) observed of aphasia that "to locate the damage which destroys speech and to localise speech are two different things" and Levy (1975) commented "dementia is a psychological concept that cannot be comprehended in anatomical and psychological terms, and attempts to do so lead to hopeless confusion." Now, with increasingly sophisticated language assessments and advances in neurological imageing techniques it is possible to understand the relationship between linguistic functioning and brain physiology (Kertesz 1983). This marriage of dynamic imageing and cognitive processes has fostered the infant science of neurolinguistics, broadly defined as the study of the brain correlates of language (Thatcher 1980).

The study of normal and abnormal language function has depended upon diseases of neoplastic and vascular origin, the effects of missile wounds (Luria 1970), electrical stimulation (Penfield and Roberts 1959, Mateer 1983) and CT (Benson 1967, Mohr et al 1975, Naeser and Hayward 1978, Kertesz 1979, Mazzocchi and Vignolo 1979, Naeser et al 1981). In vivo studies of CBF and PET have been published by Ingvar and Schwartz (1974), Maly et al (1977), Lassen et al (1978), Gustafson et al (1978),

Soh et al (1978), Skinhøj and Larsen (1980), Metter et al (1981), Foster et al (1983).

Dysarthria has been described by Darley et al (1975), but has not been localised by imageing techniques. Localisation studies of the alexias have been reviewed by Greenblat (1983) and the dysgraphias by Vignolo (1983). The effects of focal brain lesions on"intelligence"is still disputed (Le Brun and Hoops 1974): many scanning studies include subtests of standard measures of intelligence when investigating metabolism (Hagberg and Ingvar 1976, Maly et al 1977, Foster et al 1983)

3.2 LOCALISATION

Jackson's comment was pertinent to neurological debate on language representation in the nineteenth century. The "localisationists" school, following phrenology, considered specific areas of the brain were responsible for discrete mental functions. Broca in 1861 demonstrated that speech could be localised in the inferior portion of the third frontal gyrus. Wernicke, in 1874, described two types of aphasia, motor and sensory, which were separable. He localised sensory aphasia to the posterior third of the left upper temporal convolution. In 1881 Exner suggested a writing centre in the second frontal convolution adjacent to the hand area on the precentral motor strip.

Opposing this position the "holists" argued that brain functions were equipotential and determined by mass action. Their position was

stated by Marie in 1906. It has been clinically demonstrated in animals by Lashley in 1926. It has also been supported by German and English neurologists (Bay 1964, Critchley 1970) and American aphasiologists (Schuell et al 1964).

In 1965 Geschwind published his "Disconnection Syndromes in Animals and Man" and revived interest in the localisationist-connectionist model of human cerebral functioning. He argued cortical tissue is divided into specific areas of functional importance and linked by complex networks of association fibres. Russian neuropsychologists, while expressing strong antilocationists opinions, have suggested that the brain acts in complex functional dynamic systems of primary, secondary and tertiary zones each differentiated by cytoarchitecture (Luria 1970). Cerebral blood flow studies have supported such a dynamic concept of cortical functioning in language (Lassen et al 1978), although there is dispute over the nature of tertiary overlapping zones of cross modal functioning (Larsen et al 1978). Positron tomography has also established a dynamic theory of language function related to a theory of metabolic function which also make Luria's (1966) theories of interactive neuropsychological interpretation of aphasia take on greater significance (Tikofski 1984).

3.3. COMMUNICATION DISORDERS

Competent communication skills involve synergy of voice, speech and language. Voicing demands acoustic and supraglottic features of frequency intensity and rhythm. Speech involves articulation

and prosodic realisation. Language is the symbolic formulation of communication by linguistic and paralinguistic phenomena.

3.3.1 Voice

Voice is the acoustic realisation of the transformation of breath support into energy that is shaped by glottic and supraglottic structures into pitch, loudness, intonation and rhythm. Dysphonia following senile changes of the larynx has been documented but there is no reported evidence of dysphonia in ATD. The possibility of aphonia in frontal lobe disease has been raised by Penfield and Roberts (1959) and Sapir and Aronson (1985).

Walker (1982) has reviewed the literature of voice changes in the elderly. Fundamental frequency increases from the fifth to the eighth decade with accompanying increase of variability and phonation time ratio (Mysak 1959) and in men rather than women (McGlone and Hollien 1963). Ryan (1972) noted changed intensity and Huchinson et al (1978) noted increased hypernasality with advancing age. Morris et al (1971) has noted the diminished ventilatory capacity and the increased susceptibility of the elderly to respiratory disease affecting breath support that underpins vocal quality. At laryngeal level Mysak (1959) and Hollien and Shipp (1972) have attributed increased fundamental frequency of voice in the aged male to vocal fold atrophy. Histological evidence for such changes in the folds and cartilage has been presented by Bach et al (1941) and Zemlin (1968). Luchsinger and Arnold (1965) have noted changes of laryngeal speech musculature.

3.3.2. Speech

Decreased speech rates as a feature of neuromuscular function is noted in the normal elderly (Yairi and Clifton 1972). Speed and accuracy of articulation in normals is diminished beyond the sixth decade (Ptacek et al 1966) because of loss of neuromuscular control (Ryan 1972) and reduced sensory feedback (Mysak and Hanley 1958, Ryan and Burk 1974). Horenstein (1971) and Schow et al (1978) have suggested that increased hesitancy may indicate an increasing search with a rise of bucco-facial programming.

Dysarthria in dementia of Alzheimer Type has been reported at a rate of 10-30% in studies by Irigaray (1967) and Gustafson et al (1978). Sjogren et al (1952) reported two thirds of their sample dysarthric. Brookshire and Manthie (1980) note dysarthria in dementia as secondary to cerebrovascular disease. Golper and Binder (1981) and Obler and Albert (1981) note dysarthria to be a feature of the sub cortical dementias rather than ATD.

3.3.3. Speech Perception

Perceptual studies of speech rate, fluency, intensity and general voice quality suggest the healthy elderly are perceptually different from the young (Ptacek et al 1966, Shipp and Hollein 1969). Ryan and Burk (1974) relate laryngeal air loss, laryngeal tension,

mean fundamental frequency, voice tremor, slow articulation rate and imprecise consonants as predictor variables of age related to speech perception.

Decreases in processing both verbal and non verbal stimuli in the normal elderly have been demonstrated by Konkle et al (1977). Reduced perception of speech occurs when speech is temporally extended or compressed (Corso 1977). Feldman and Reiger (1967) showed the temporal response to tones diminished after the sixth decade. The normal elderly are also poor at comprehending auditory information by rate (Schmitt and McLosky 1981) and sentence complexity (Maxim 1985). Such disability may be related to presbycuisis (Corso 1970), attention (Rabbitt 1965), ambiguity (Scholes 1978) and inflexibility (Davies and Grunwell 1973).

No known studies of speech perception in ATD are reported.

3.3.4 Language in Normal Ageing

Measurement of language changes in normal elderly are rare and a search of the literature on language changes in normal senescence and early dementia uncovers only occasional case reports and even less frequent attempts at systematic investigation. It is not clear which changes in ageing are normal and which are not. Birren (1970) considered that in the absence of neurological disease cognitive ability should remain at a high level beyond eighty.

Walker (1982) gave the Minnesota Test for Differential Diagnosis of Aphasia (Schuell 1965) to the normal elderly. She noted dysphasic errors suggested a common disturbance of normal and abnormal language in ageing; the difference was of degree rather than type. Duffey et al (1976) gave the Porch Index of Communicative Ability (Porch 1967) to a group of normal elderly and obtained a negative correlation between age and performance on gestural, verbal and graphic sections. Wertz et al (1971) found a deterioration with age on part five of the Token Test of De Renzie and Vignolo (1962). This measures the comprehension of language structures of increasing complexity. The WAIS (Wechsler 1958) normative data suggests that although verbal abilities are more resistant than performance abilities to normal age change both verbal and performance abilities drop in the third decade, the rate of decline increasing with increasing age. Performance scores decline more rapidly.

Comprehension is lost as a function of hearing acuity (Corso 1977), speech discrimination and perception (Ryan and Burk 1974, Stevenson 1975, Corso 1977), as well as masking (Groen 1969) and competing stimuli (Bergman et al 1976). Moreover, comprehension in ageing is affected by diminished auditory memory (Horn 1979). Memory on the WAIS as assessed by Digit Recall decreases after the fifth decade (Bortwinick and Storandt 1974, Craik 1977). When constraints are placed on such tests, and concentration is demanded, such as digit recall in reverse, a greater dampening occurs (Broadbent and Heron 1962).

The generation of word strings in the elderly diminishes with age (Schaie and Zelinski 1980) although the lexicon increases with age (Smith 1957, Reigel 1968), or at least remains stable (Fox 1947). Failure to generate tasks of word fluency suggests that active vocabulary diminishes while passive or recognition vocabulary remains. This pattern has been explained by Horn and Catell (1966) in terms of fluid and crystalline intelligence.

3.3.5. Language in Abnormal Ageing

"Aphasia" as a term to describe the language pathology in schizophrenia, developmental language disorders and dementia has been considered imprecise by neurologists and linguists (Lhermitte and Gautier 1969, Lesser 1978). Other linguists have argued the discipline of aphasiology to be a discipline only for linguists. Jacobson (1964) commented "aphasia is first and foremost a disorder of language and as linguistics deal with language it is linguists who have to tell us what the exact nature of these diverse disintegrations is". Lenneberg (1975) has argued that linguistic models are speculative and tell us nothing of brain mechanisms in speech. Brain (1961) has indicated the difficulty of psychological phenomena expressed in terms of anatomy and physiology. The study of anatomical aphasia producing lesions does not tell us about the physiological mechanisms supporting language and, because aphasiology has borrowed from other disciplines, its descriptions are hybrid: "transcortical motor aphasia" is a combination of the language of anatomy, physiology and psychology.

Aphasia

Disturbances of language in focal neurological lesions are the aphasias whose syndromes have been described and classified

with increasing uncertainty (Head 1926, Schwartz 1984, Caramazza 1984). Such classifications have provided a taxonomy for aphasia as either a unitary or multi focal pathology(Head 1926, Bay 1962, 1964, 1967, Luria 1964, 1970, Benson 1967, Schuell 1974). The linguistic and taxonomic/localisation of aphasiology has recently been described by Lesser (1978) and Kertesz (1979)

The elderly are more susceptible to injury related changes of language: 80% - 90% of CNS injuries after the age of 50 are due to strokes and 70% of strokes occur after the age of 70 (Langton and Hewer 1977). The posterior cortex appears more vulnerable in age related strokes producing more fluent Wernicke-type aphasias (Obler et al 1978). However capacity for improvement of language is inversely related to age (Walker and Williams 1980) although age may not be the sole indicator of language recovery (Lefeure 1957).

Schizophrenia

Disturbances of language are also recognised in the non-organic psychoses. The neurology of psychotic speech has been reviewed by Critchley (1964). He quotes Runke and Nijan (1958) who considered the disturbed interhuman relationships in schizophrenia could be reduced to an aphasia. More recently schizophrenic discourse has been analysed in terms of cohesion of verbal output (Rochester and Martin 1979). This is not necessarily aphasic in nature (di Simoni et al 1977) but poses a great problem for the listeners (Rochester et al 1977, Rutter 1985). More recently linguistic analysis of schizophrenic speech suggests major differences from normal speech (Morice and Ingram 1982). Faber and

Reichstein (1981) identified neurolinguistic disturbance of comprehension and repetition in schizophrenics suggesting aphasia in schizophrenia. The basis of the problem of schizophrenic speech is language representation of disordered thought and only recently has a description of thought disorder in linguistic terms been addressed (Andreasen 1979).

Dementia

Miller (1981) has noted that research in dementia has focused on memory disturbance and that there has been little on language disturbance and nothing on perception, information processing, visuo spatial ability and thinking.

There have been several descriptions of the general features of language in dementia. Stengel (1964) and Irigaray (1967) describe a lack of verbal spontaneity and speech initiative with a slowness or a refusal to respond. Generally language of the demented is considered circuitous, vague and empty (Obler and Albert 1981) but fluent (Golpher and Binder 1981). Echolalia is more common than mutism (Allison 1961). If present mutism is, like echolalia, a late feature of the disorder (Constantinidis et al 1978).

Semantics

It is generally considered that the naming errors dements make are similar to the paraphasias found in forms of Wernicke type aphasia (Albert 1980). Martin and Feido (1983) could find no evidence of

phonemic errors in ATD. Nicholas et al (1985) found the empty speech of ATD contrasted with the paraphasias of Wernicke type aphasia. Semantic errors are similar to errors produced by lesions of the posterior cortex, and in particular, the tertiary zones of the temporo-parieto-occipital borders of the left hemisphere (Luria 1966, Whitehouse et al 1978). Naming errors in anything but mild ATD have not been investigated until recently (Martin and Feido 1983, Grober et al 1985, Pietro and Goldfarb 1985).

Anomia has been reported to be common in dysphasia and in generalised brain damage (Benson 1979). Critchley (1964) and Stengel (1964) have noted anomia as the reason that demented language is "empty". Martin and Feido (1983) found mildly impaired Alzheimer patients impoverished on tasks of confrontation naming but within the normal range of the WAIS Verbal Similarities task. Irigaray (1967) and Stengel (1964) noted that nouns were selectively lost rather than adjectives, pronouns, prepositions and conjunctions, while Allison (1962) and Schwartz et al 1979) noticed that abstract nouns were more vulnerable than concrete ones. Skelton-Robinson and Jones (1984) have pointed out that the loss of nouns relates to their frequency in the lexicon. Allison (1962) and Stengel (1964) reported that the dements were likely to use vague generalisations like "thing" rather than search for appropriate labels and also add personal possessive labels as "my tie", or "your pen". Tissot et al (1967) similarly noted the attempt of dements to make nouns more concrete by providing them with verbal labels as "drawing pencil" or "drinking cup". This suggests the use of verbal paraphasias to aid the

search for nouns. Schwartz et al (1979) confirmed the observation of the extension of verbal labels arguing that in the loosening of semantic ties and concept formation it represented a loss of the associative links between words and the things they represent.

Miller and Hague (1975) described decreased word fluency in dementia on tasks that demand Thurstone's /w/ factor. Borkowski et al (1967) argued, in this test, that less familiar letters produce fewer word streams than common ones. Martin and Feido (1983) noticed that not only this measure of fluency but ideational fluency - the ability to produce streams of words in semantic fields - is reduced. However Miller and Hague (1975) also determined measures of oral fluency were normal in spontaneous speech, that is, words that occurred infrequently in word streams were distributed normally in discourse.

Several authors have noticed the increased latency of naming in word fluency tasks (Lawson and Baker 1968, Miller and Hague 1975) and several have noted the degree of anomia parallels the degree of dementia (Martin and Feido 1983, Bayles and Tomoeda 1983).

Nebes (1985) has investigated semantic deficits in ATD and concluded the deficit is not one of loss of a semantic memory but of access to lexical store. ATD patients could be "primed" or cued to retrieve words, and at the level of recognition, ATD patients have little difficulty, at least in mild stages, pointing to named objects. Nevertheless Bayles and Boone (1982) and Skelton-Robinson and Jones (1984) have argued the value of impaired naming in the diagnosis of dementia.

Overman (1979) was able to classify alchoholic dementia using naming tasks to differentiate patients with alchoholic dementia from ATD and MID.

The reasons for the breakdown of semantic fields remains unclear. Luria (1966), following the Pavlovian concept, argued that failure of selection of semantic terms represented a breakdown in a "law of strength", the pathological brain is unable to select from competing stimuli so that weak stimuli arouse strong associations and this lack of selectivity results in paraphasia. Warrington (1975) noted that the redundancy of language in dementia may be a result of the individual's need to qualify lexemes in unsteady and diminishing fields, so the ability to make broad classifications is retained, but the ability to make more precise selection within semantic fields is lost.

A third possibility of dual representation of names was advanced by Lissauer (1890) and is quoted by Warrington (1975) with supporting evidence from Wilkins and Moscovitch (1978). Visual engrams may be matched to verbal analogues for concrete but not abstract nouns. The misaligning of an auditory analogue to a visual engram has been interpreted by Stengel (1964) and Rochford (1971) as producing an agnostic component in anomia. The suggestion is that whereas aphasia was a difficulty of access and retrieval of nouns dementia was a difficulty of recognition. However, Barker and Lawson (1968) noted dements could gesture the use of objects they could not name and Appell et al (1982) commented that names could be phonetically cued. This suggests dements do have an internal representation of objects they cannot label.

Phonemic paraphasias are sound substitution errors. Tissot et al 1967, Ajuriaguerra and Tissot (1975) and Nicholas et al (1985) found no real evidence of phonemic paraphasia and Constantinidis et al (1978) found phonemic paraphasia rarer than semantic paraphasia or word substitution errors. Semantic paraphasias include circumlocution and generic terms or vague reference (Irigaray 1967, Schwartz et al 1979, Bayles 1985). Critchley (1964) mentioned the semantic paraphasias of dements are quite unlike those of aphasics. Schwartz et al (1979) suggested paraphasias and anomia represented breakdown at pre speech level.

Logoclonia, echolalia and perseveration, but seldom mutism, have been described by Allison (1962). Fuld et al (1982) have drawn attention to intrusion errors in the discourse of 89% of patients with Alzheimer's disease. Smith et al (1979) and Fuld et al (1982) note these intrusion errors or learned words that are unrelated to specific tasks perseverate into discourse as a distinguishing feature of ATD. The number of intrusions is shown to be increased by scopalamine in normal subjects while in ATD the number of intrusions appears sensitive to cholinergic therapy (Fuld 1983). However intrusions occur in multiple infarct dementia and aphasia where perseveration is far more common (Yamadori 1981). Intrusions have been reported by Luria (1965) as a contamination of the activity of the frontal lobes.

Syntax

Syntactic errors may be agrammatic, where utterances are short with the omission of function words, or paragrammatic, as fluent and copious speech with function words and affixes often misused

(Howard 1985). Discohesive speech is a failure of sentence clauses and phrases to relate to each other by conjunctive or lexical reference (Rochester et al 1977).

Compared with semantic loss syntax in dementia is remarkably preserved (Irigaray 1967, 1973, Ernst et al 1970, Warrington 1975, Whitacker 1976, Schwartz et al 1979, Bayles 1982).

Selective impairment of semantic memory would suggest that the grammar and lexicon are therefore independent (Whitacker 1976 and Schwartz et al 1979). Whitacker (1976) has argued for functional independence of syntax in diffuse pathology as judged by the ability to change and correct syntactic structures in repetition tasks. Schwartz et al (1979) described a patient who produced errors of semantic comprehension, but who was also able to operate on sentences indicating comprehension of grammatical transformations and appropriate morphological endings. They suggested there was a filter for grammatical structures independent of cognition. Preserved grammar suggested "a tighter wiring" of syntax than semantics in the brain. This suggests that dements may not be asyntactic listeners like Broca's aphasics (Boller et al 1979) and the normal elderly who are forced to rely on the context to comprehend (Maxim 1985).

Constantinidis et al (1978) noticed a proportion of sentences and phrases in dementia that were "left hanging", with the loss of plural markers and grammatical non agreement that may be interpreted

as a loss of short term memory with a resultant loss of cohesion. Irigaray (1973) commented on the flexibility of speech patterns in dementia and the failure of dements to alter word order and so produce sentences that were forced and inelegant. To date, however, there are no studies which investigate linguistic breakdown in terms of syntactic errors or loss of cohesion in the duration of dementia.

Pragmatics

The loss of the use of language pragmatically has also been noted. Obler (1980) considered that limited syntax produced "stimulus boundedness" or a reliance on context and a failure to generate novel utterances. Critchley (1964) also noted the loss of speech initiative and the restriction of the language of the demented to statements and requests while Obler (1980) noted the increased loss of questions. This pragmatic divorce is between language and the purpose it represents.

The penultimate stage of language deterioration in the demented preceding mutism appears to be repetition, substitution and perseveration of verbal stereotype (Ajuiaguerra and Tissot 1975). Speech is reduced to a syllabic palilalia that has been described following intellectual deterioration with bilateral lesions of the basal ganglia and grey matter of both hemispheres (Boller

et al 1975). It presents as endless repetition of syllable word or phrase both verbally and graphically and may accompany late Alzheimer's disease (Meyer-Gross et al 1969), and has also been indexed to linguistic decline. However, there remains little literature on the localisation of palilalia. It may be sub cortical (Brown 1972) or frontal (Luria 1965).

3.3.6. "Aphasia" in Dementia

Some authorities have described the language of dementia in terms of aphasia. In 1892 Pick mentioned his patient as presenting transcortical sensory aphasia, Constantinidis et al (1978) suggested anomic aphasia and Kertesz (1979) has suggested that in ATD early language disturbance presents initially as anomic aphasia but not Transcortical Motor or Broca's aphasia. Nicholas et al (1985) have argued the empty speech of ATD is not characteristic of Wernicke's aphasia but more often similar to anomic aphasia. However Obler (1981) presented Irigaray's (1973) data of senile dements with no expressive aphasic signs.

Ernst et al (1970) and Irigaray (1967) have concluded that there is little comprehension deficit in early dementia for speech sounds and report little evidence of expressive phonemic disturbance.

Hagberg and Ingvar (1976) commented of their investigation of 55 organic dements that language was lost late in the progression of ATD, however Weschler (1977) and Allison (1962) have reported cases of language loss as a presenting feature.

3.3.7. Language in Associated Cortical Dementias

Communication disorders in diseases other than Alzheimer's disease, Pick's disease and dialysis dementia are reported only by rare case studies. In dialysis dementia they present as an initial complaint of articulatory disturbance to aphasia, aphemia and finally mutism. These difficulties were described by Rosenbek et al (1975) and Madison et al (1977) as cortical stammering with variable dysarthric signs and by Baratz and Herzog (1980) as ataxic dysarthria from damaged cerebral connections. Mutism in dialysis dementia seems to be related to bilateral lesions to the basal ganglia.

Holland et al (1985) described progressive dissolution of language in a patient with Pick's disease over 12 years. Neuropathological findings were left hemispheric atrophy especially over the anterior two thirds of the anterior temporal area. Here 50% neuronal loss was noted. Atrophy of the first third of the middle and inferior temporal gyri, shrinkage of the left fronto parietal lobe, the right superior temporal gyrus and diffuse right atrophy in the frontal and parietal areas was also described.

Medially there was a widening of the sulci particularly the left superior frontal gyrus and the left paracentral lobule. Their patient retained cognitive ability including the ability to calculate, read, write and remember late into the disease. Communication deficit was marked with mutism, confirming the observation of Tissot, Constantinidis and Richard (1975), quoted Holland et al (1985) who reported 19 of their 32 cases with Pick's disease mute.

Benson (1979) has reviewed the psychiatric and neurological causes of mutism. The neurological causes appearing to be damage to Broca's area and multiple lacunar infarcts in the basal ganglia and upper brain stem. Frontal lobe damage is found in akinetic mutism. However, there is no compelling evidence differentiating elective and non elective mutism in dementia.

3.4 EVALUATION OF LANGUAGE STUDIES OF DEMENTIA

There are certain weaknesses in the published studies of language pathology in dementia. Appell et al (1982) has commented they are based on unstandardised observations, restricted areas of language, atypical cases, mixed samples and are without quantitive data. Their investigation used a clinically well defined group and a standardised aphasia battery. However no indication of the severity of dementia of their 25 institutionalised patients was given. The study concluded that half the cohort had disturbed

language; that mild ATD demonstrated retained fluency and preserved articulation but poor naming, poor comprehension, and preserved syntax. They agreed with the observation that language in ATD is fluent but empty. Naming errors were similar to those of aphasia but without phonemic paraphasias. Successful naming seemed to be concrete and better than verbal fluency, agreeing with observations of Allison (1962) and Gustafson et al (1978). Comprehension was worse for abstract operations and performance was poor on sequential commands. There was little echolalia, mutism or syllabic perseveration. There was no correlation between age and the degree of language pathology agreeing with the observation of Hagberg and Ingvar (1976) but there was a correlation of length of hospitalisation and degree of language pathology.

A summary of the weakness of all studies includes primarily a complete lack of a longitudinal study of language breakdown in dementia. There are only cross-sectional observations Bayles (1984). Many studies are clinical impressions without scientific methodology (Stengel 1943 and 1964, Critchley 1964). There is an assumption that language following focal lesions can be validly compared to language following generalised lesions (Appell etal 1982). Many studies define language in terms of verbal ability as measured by intelligence tests (Bayles and Boone 1982). Studies have been conducted with different aims that bias methodology and results: Ajuriaguerra and Tissot (1975) attempted to compare language breakdown in adults with language acquisition in children, while Irigaray (1967) attempted to produce a

performance grammar for dementia. Studies seldom relate the degree of dementia to the degree of language pathology. Experimental groups have been poorly defined in terms of type of dementia or type of disease processes causing those dementias. Finally, studies rarely include descriptions of concomitant neurological deficits, or correlations of language to age, institutionalisation, duration of severity of illness.

3.5 INVESTIGATIVE TECHNIQUES

3.5.1 Dichotic Listening and the WADA Technique

Two tests for language dominance have been developed in the last twenty years. Dichotic listening (Kimura 1967, Broadbent 1971) involves the carrying of separate messages to the left and right ears and theoretically to the contra-lateral hemispheres. The procedure has produced statistically significant evidence for the language dominance of the left hemisphere.

The WADA technique involves injection of sodium amytal, a fast acting barbituate, into the internal carotid arteries producing ispilateral cerebral hemisphere paralysis, hemiplegia, hemiaesthesia, hemianopia and, if introduced to the dominant hemisphere, aphasia (Wada 1949, Wada and Rasmussen 1960). The test has been used to determine the role of the left hemisphere in speech (Milner, Branch and Rasmussen 1964) and memory (Milner 1967). The results show an absence of speech representation bilaterally in right handed

subjects and high bilateral representation for ambidextrous subjects. Non right handed subjects show 48% speech representation in the left hemisphere, 38% in the right and 14% bilaterally. Goodglass and Quadfasal (1954) and Roberts (1969) have confirmed that in non right handers left hemisphere language dominance is still more common than right: approximately a 60/40 split (Benson 1979).

3.5.2 Neurosurgical Correlations

Penfield and Rasmussen (1950) mapped the motor and sensory periRolandic areas by electrical stimulation of the exposed cortex. Penfield and Roberts (1959) using the same technique produced speech arrest, hesitancy, slurring and defects of naming and repetition. This allowed them to determine the speech areas being in the temporoparietal, inferior frontal and supplementary motor area of the dominant cortex. They were also able to elicit spontaneous vocalisation from bilateral stimulation of the Rolandic and supplementary motor areas. Stimulation of the central speech area has been recently reviewed by Mateer (1983). She has confirmed the role of the left periSylvian area and adjacent zones in language. This is consistent with findings of clinical studies of aphasia.

Penfield and Roberts (1959) regarded the thalamus as an integrating centre between frontal and temporo-parietal areas. Bilateral stimulation of the thalmic nuclei produced anomia, perseveration and disturbance of short term memory and speech sequencing. Their

study, and the results of steriotaxic surgery in Parkinsonism, implicate the role of the thalamus, and especially the ventro-lateral nuclei in the organisation of language function (Bell 1968, Vikki 1978). Results of steriotaxic surgery on the basal ganglia have produced speech disorders resulting from lesions of the globus pallidus, including poor oral formulation, disorders of expression with anomia, pausological and fluency disturbance. The role of subcortical language mechanisms has been reviewed by Ojemann (1976) while recent interest in subcortical dementia and language has been raised by Obler and Albert (1981).

Milner (1967) described the differential effects of left frontal and left temporal lobectomy on word fluency and verbal memory. She demonstrated the role of the dominant frontal lobe in spontaneous speech rate confirming the observation of Luria (1966) who had established the role of the frontal lobe in fluency and the role of the second and third temporal convolutions, together with the hippocampal gyri, in verbal recall and verbal learning. Right temporal lobectomy produces deficits of perception, memory, pattern recall and tonal perception.

3.5.3 Radionucleide Scanning and Computerised Axial Tomogrpahy

Early studies of the use of brain scans to localise lesions producing aphasia were reviewed by Benson and Pattern (1967). Benson (1967) described a fluency/non-fluency dichotomy of aphasia on the basis of frontal versus posterior lesions but Karis and Horeinstein (1976) who could not confirm this paradigm. Kertesz et al (1977) and

Benson and Pattern (1967) noted, however, there are difficulties in imageing lesions after acute stages when lesions tend to "fade and disappear". Kertesz has nevertheless preferred radionucleide to CT imageing.

Kertesz (1979) reports the use of isotope scans imageing lesions producing Broca's aphasia, nominal aphasias showed deeper lesions. He concluded "the results are rather gratifying in that we were able to demonstrate distinct areas for Broca's and Wernicke's aphasias and to show that lesions of global aphasias involved both these regions while conduction aphasias have lesions in between" (Kertesz 1979, page 549). (See Plates 3.1 and 3.2)

Computerised tomography is a technique which uses a computer to combine X-ray readings taken from many different angles to represent a cross section of the brain. The method as a study of a neuroanatomical basis of aphasia has been described by Kertesz (1979) and Damasio (1981) who considered the technique useful in determining the site and type of aphasia producing lesions but not the functional neuroanatomical nature of the lesion.

Mohr et al (1975) used CT to describe thalamic aphasia while Naeser et al (1978) correlated site and size of cortical lesions to aphasic deficits as measured by the BADE and Token Test. Kertesz et al (1977) correlated lesion size with severity of aphasia. Severity positively correlated with lesion size, and

Plate 3.1 Summary of maximum overlap of aphasia producing lesions on anterior and posterior templates.
(After Kertesz 1979)

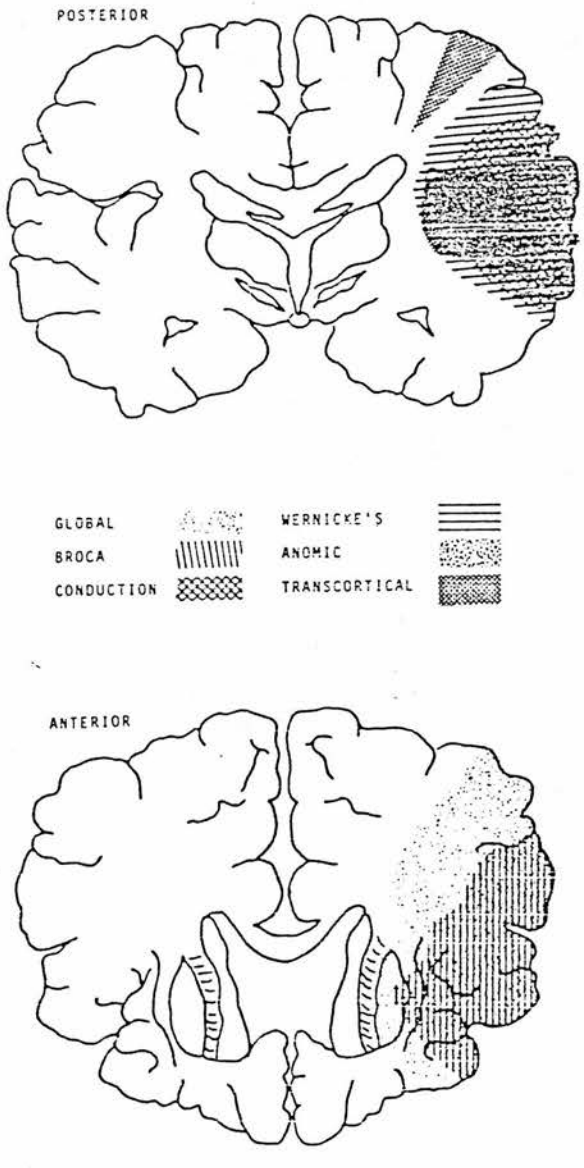
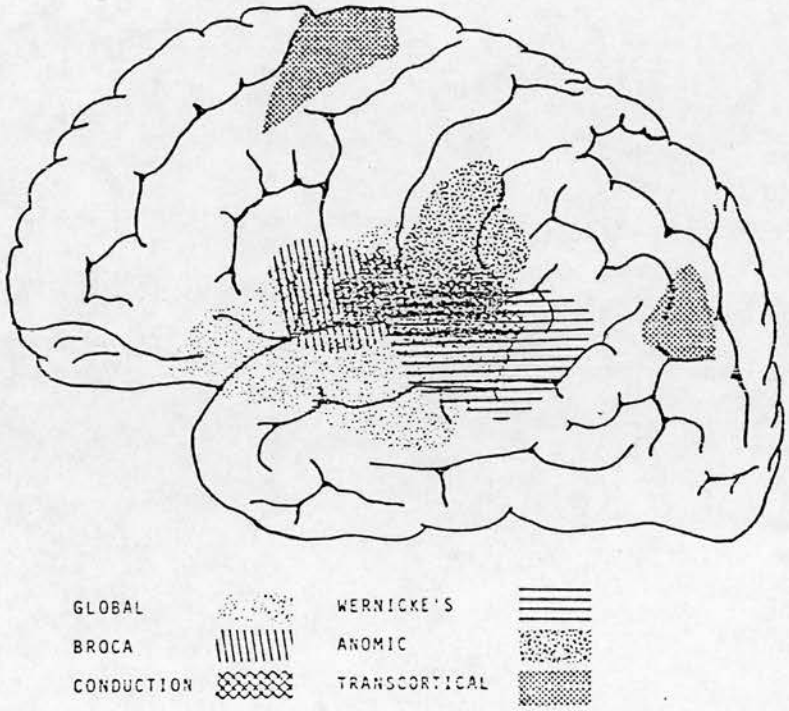


Plate 3.2 Summary of maximum overlap of aphasia producing lesions on lateral templates. (After Kertesz 1979)



the size of lesion was considered more important in determining the degree of the aphasia than the type of aphasia. Kertesz suggested redionucleide scans were better for acute lesions but CT was better for chronic lesions. However, Shirley (1979) found the Porch Index of Communicative Ability (Porch 1967) correlated to lesion sites and the Western Aphasia Battery (Kertesz 1980) to lesion size. He concluded that the Western Aphasia Battery and the Boston Aphasia Diagnostic Examination (Goodglass and Kaplan 1982) demonstrated "outmoded concepts of localisation". This strong criticism has been justified by CBF and PET studies.

3.5.4 Cerebral Blood Flow

Metabolic studies of CBF, using Xenon by injection or inhalation methods have supported a dynamic theory of the functional organisation of the cerebral cortex in terms of language representation, cognitive processing and the general role of language in the programming and regulation of behaviour (Hagberg and Ingvar 1976, Gustafson et al 1978). The technique has allowed the observation of dynamic functioning of the lateral cortex by non invasive means through the use of radioactive isotopes.

Studies in normal activation of speech, reading and problem solving have indicated that in listening there is increased bilateral flow over the auditory cortex and Wernicke's area which mediates the understanding of speech.

In speech there is increased flow over the articulatory areas of the motor and somato-sensory cortex as well as the temporo- parietal cortex, the supplementary motor area and the inferior areas of the frontal lobes. The difference between right and left hemisphere flow is more diffuse perfusion over the non dominant mouth-tongue-larynx area which coalesces with the auditory cortex.

Automatic speech makes less demands on "language" by being non propositional. Larsen et al (1978) have demonstrated that in automatic speech mean hemispheric flow on the left side does not change significantly but flow on the right does. Patterns of flow over motor and auditory perception areas are similar bilaterally. The left hemisphere shows significantly more involvement of the premotor cortex. There is therefore the suggestion that in automatic speech flow is less than in propositional speech. The supplementary motor area acts as a tertiary area involved in programming all speech acts.

Cerebral blood flow in abstract thinking depends on the nature of the task (Risberg and Ingvar 1968) but all tasks demonstrate increased cerebral flow as well as specific flow to the posterior cortex. Particular tasks that demand abstract problem solving, rearrangement of data and concentration, as digit repetition in reverse and the Progressive Matrices of Raven, demand higher global flow.

In tests that demand mental effort cortical flow increases over temporal and temporo-occipital regions which are known to be involved in

hearing, auditory memory, speech and other highly integrated sensory activities (Penfield and Rasmussen 1950, Luria 1966) as well as frontal motor speech areas, and in particular the peri Rolandic and anterior frontal cortex which is "utilised in conscious intellectual processes" (Penfield and Jasper 1954) and in logical operations (Luria 1966). The Progressive Matrices as reasoning tests involve visual perception of figures and complex analysis of the given problems and, when a satisfactory solution is found, the chosen figure is verbalised. Thus in addition to problem solving the test involves verbal functions, including covert "inner" speech (Sokolov 1972). In this test pre Rolandic and anterior frontal regions show increased flow, they are known to be important for integrated and complex visual activities like reading and spatial thinking (Luria 1966).

Although voluntary muscle movements in speech, writing or typing cause bilateral flow to increase the pre motor areas, the upper frontal lobe and supplementary motor areas have a specific role in planning and regulation of tasks: their uptake is increased when merely thinking about the motor task. Such tasks suggest the role of the supplementary motor area as the programmer of dynamic movements, the primary sensory cortex as the controller of movements and the primary motor cortex as the executor of movement. The role of the supplementary motor area as a "programmer" has received particular attention in localisation studies. Fluency is disturbed in frontal lobe disease, and in particular, the left supplementary motor area appears to have a role in verbal rather than ideational fluency (Newcombe 1969).

Psychological tests that demand abstract thinking involve the association cortex in normals (Risberg and Ingvar 1973). In ATD these areas are functionally low at rest (Hagberg and Ingvar 1976), in fact there is deactivation during psychological testing and reading (Ingvar et al 1975). This failure of activity has been called "intellectual steal" (Ingvar et al 1975) and may relate to dements attempting to use different strategies, for example, not seeing an abstract reasoning test as a problem but perceiving it and inducing greater activity in the primary sensory regions.

Meyer et al (1978) studied patients with internal carotid occlusion, MID and focal epilepsy and compared their cerebral flow patterns to healthy controls. They attempted to define cerebrovascular reserve to function in psychological testing. Despite decreased flow related to cortical weight there was no significant reduction of flow in MID compared to patients with carotid disease.

A Gustafson et al (1978) study of presenile dementia, including Pick's and ATD, related CBF to language disturbance. They concluded that blood flow was reduced over the dominant hemisphere and found language disturbances in the presenile dementias conformed to the localisation principles of brain lesions leading to aphasia. Verbal abilities failed with general blood flow reduction. Receptive disturbances were associated with decreased flow in the post central regions and expressive disturbances with decreased flow in frontal and anterior temporal regions. The failure of speech regulation or inner speech was associated with decreased frontal flow. Patients with jargon and logorrhea had a global decrease in CBF.

Their patients with ATD who had receptive and expressive disturbances such as naming errors, abstract word finding errors, and stereotyped speech had reduced flow in temporal and occipital areas but not precentral areas. Patients with Pick's disease demonstrated frontal and temporal reduction with central areas spared. Those Pick's patients with disinhibition and who were unable to regulate their behaviour showed frontal flow deficiency.

3.5.5. Positron Emission Tomography

The advantage of positron emission tomography over CT is that it demonstrates disturbances of functional systems away from the site of the lesion. It has also advantages over previous CBF techniques in imageing deep structures and not just the lateral cortex. PET has been used to investigate normal brain function and a variety of psychiatric and neurological conditions. Cerebral metabolic studies using PET have demonstrated changes in dementia in temporal, frontal and parietal areas. The technique has also related diminished CBF and diminished glucose utilisation in Alzheimer's disease to cognitive impairment (Hagberg and Ingvar 1976, Ferris et al 1981, Ferris and De Leon 1983).

Metter et al (1981) reported that, in aphasics, glucose metabolism was reduced beyond the lesions imaged by CT. Forster et al (1983) was able to demonstrate that language was associated with a reduction in glucose metabolism of the left frontal, temporal and parietal areas in ATD. They found glucose utilisation was 19% lower in the left temporo-parietal cortex compared with the same area

in the right hemisphere. In ATD patients with constructional apraxia glucose metabolism was 31% lower in the right hemisphere than the left. The reductions were particular to the parietal and temporal areas. They found no relationship between glucose hypometabolism and WAIS scores, perhaps because the WAIS is multifactorial. There was a relationship between reaction time and glucose metabolism. They concluded that glucose metabolism shows focal and diffuse changes in ATD related to deficits of language and visuo constructive tasks.

Haxby et al (1985) have demonstrated lateral asymmetry of function in patients with ATD compared to controls using regional glucose utilisation values. Patients with language impairment demonstrated reduced glucose utilisation in left frontal, temporal and parietal association areas while patients with reduced visuo spatial abilities demonstrated lower right parietal utilisation. Discrepancies between language and visuo spatial deficits in ATD are related to asymmetrical reduction of cerebral metabolism.

In conclusion PET studies show that in ATD there is reduced glucose metabolism that is significant in frontal and temporo parietal areas (Benson et al 1983). The reduction in metabolism correlates with the degree of atrophy as measured by CT scanning (Alvari et al 1981) and with the severity of dementia (Benson et al 1981). Changes in glucose metabolism can be related to the type of dementia (Benson 1982) and to impairments of language (Forster et al 1983, Huxby et al 1985).

CHAPTER 4

METHOD OF INVESTIGATION

4.1 INTRODUCTION

Two methods of analysis of adult pathological language systems can be described: the linguistic analysis of speech samples and the assessment of speech acts. The first method has only recently been addressed in aphasiology (Jacobson 1964, Lesser 1978, Newman and Epstein 1985) and in schizophrenia (Rochester et al 1977, Morice and Ingram 1982), but not in dementia. Demented language has been assessed by the verbal scales on tests of adult intelligence and by aphasia batteries. Appell et al (1982) used the Western Aphasia Battery (Kertesz 1980) to study language functioning in Alzheimer's disease, Walker (1982) used the Minnesota Test for Differential Diagnosis of Aphasia (Schuell 1965), Wertz (1978) used the Porch Index of Communicative Ability (Porch (1967) and Hodkinson et al (1984) used the Aphasia Screening Test (Whurr 1974).

Three current neurolinguistic test batteries are used to investigate language pathology in aphasia: the Neuro-sensory Centre Examination for Aphasia (Spreen and Benton 1969), the Boston Diagnostic Examination for Aphasia (Goodglass and Kaplan 1982), and the Western Aphasia Battery (Kertesz 1980). An over-view of these most current tests shows their content to be remarkably similar (See Table 4.1). As early as 1967 Benton commented

Table 4.1

COMPARISON OF SUBTESTS OF

MAJOR APHASIA BATTERIES WITH LANGUAGE SCALES

TASK	Language Scales	Neurosensory Centre Comprehensive Examination for Aphasia	Boston Diagnostic Aphasia Examination	Western Aphasia Battery
Memory - Orientation Concentration	Rating Scale			
Automaticisms	Automatic sequences		Automatic sequences	
Comprehension	Object Identification by Name & Function Token Test	Object Identification by Name Token Test	Complex Ideational Material Word Discrimination Commands	"Yes-no" questions Auditory word recognition Sequential Commands
Auditory Memory	Repetition of Sentences Repetition of Digits - Forward Reverse	Repetition of Digits Forward & Reverse Sentence Repetition Articulation	Repetition of Words Repetition of Phrases Oral Agility	Numbers Words Sentences
Spontaneous Speech	Description of Function Sentence Construction	Description of Use Sentence Construction	Melodic line, phrase length, articulatory agility, grammatical form, paraphasias in running speech, word finding	Fluency Information Content
Naming	Confrontation Naming Tactile Naming - Right and Left Word Fluency	Visual Naming Tactile Naming - Right and Left Word Fluency	Confrontation Naming Fluency in Controlled Association Sentence Completion Responsive Naming	Object Naming Word Fluency Sentence Completion Responsive Speech
Reading	Reading Words Reading Sentences Word Recognition Sentence Comprehension	Reading Sentences for Meaning Oral Reading(Sentences) Oral Reading(Names) Reading Names for Meaning (pointing)	Reading Sentences and Paragraphs Word Reading Word Picture Matching Word Recognition Symbol and Word Discrimination Comprehension of Oral Spelling Spelling Dictation	Reading Comprehension of Sentences Reading Commands Aloud Performing Commands Written Word: Stimulus - object choice matching Picture Stimulus - written word choice matching Word Stimulus - picture choice matching Phonetic Association Letter Discrimination Spelled Word Recognition Spelling
Writing	Automatic Writing Spelling Dictation Writing to Copy	Writing Names Writing to Dictation Visual graphic Naming Writing from Copy	Mechanics of Writing Narrative Writing Sentences Written to Dictation Spelling to Dictation Recall of Written Symbols Primer-level Dictation Copy	Writing on Request Written Output Writing on Dictation Writing of Dictated or Visually Presented Words Recall of Written Symbols Writing of Dictated letters or numbers Copy

Table 4.1 (contd.)

TASK	Language Scales	Neurosensory Centre Comprehensive Examination for Aphasia	Boston Diagnostic Aphasia Examination	Western Aphasia Battery
Calculation	Calculation		Recitation, Singing Arithmetic	
Praxis	Gesture Oral Apraxia Ideomotor Apraxia		Intransitive limb Buccofacial Transitive limb Serial actions	Upper limb Buccofacial Instrumental Complex
Drawing	Constructional Apraxia		Drawing to Command	Drawing

that, unlike their concepts the content of aphasia tests has never presented difficulty. Oddly enough few, if any, of the most popular test batteries include in their rationale reasons for test inclusion. This is in marked contrast to the theorising that preceded attempts to measure intelligence earlier this century. Benton (1967) and later Kertesz (1979) have indicated criteria for tests for aphasia, that they should

(1) explore all potentially disturbed modalities
(2) discriminate aphasia sub types
(3) be graded to assess difficulty
(4) be administered in one setting
(5) provide explicit instructions
(6) minimise the effects of education and so measure language performance as purely as possible
(7) discriminate between normal, demented and aphasic language
(8) have internal consistency and comparability of scores
(9) have face content validity
(10) provide standardisation

Kertesz (1979) comments that most aphasic tests should include

(1) a description of spontaneous or conversational speech
(2) a measure of information value
(3) a measure of fluency
(4) auditory comprehension
(5) naming

(6) repetition

(7) reading comprehension

(8) writing

(9) arithmetic

4.2. THE LANGUAGE SCALES

The Language Scales constructed for this research encompass the ten parameters. They provide measures of:

1. Comprehension at word and sentence level in both spoken and written modalities
2. Expressive verbal language at word and sentence level.
3. Symbolic function in reading, reading comprehension and calculation
4. Non verbal meaning by gesture
5. Remote, recent and immediate (auditory) memory
6. Verbal intelligence by a fluency test of controlled word association and non verbal intelligence by Block Design (Weschler 1958) and the Progressive Coloured Matrices (Raven 1965).

The Language Scale includes subtests of language and cognitive performance. These may be subdivided into tests for auditory agnosia (2), receptive verbal language (3), expressive verbal language (7), gesture (1) immediate recall (3), reading (4),

writing (4), praxis (3), calculation (1). It also includes the Coloured Progressive Matrices of Raven (1965), Block Design (WAIS 1958) and the Dementia Rating Scale of Blessed, Roth and Tomlinson (1968).

The battery was originally a modification of the Neurosensory Centre Comprehensive Examination for Aphasia of Spreen and Benton (1969). Its advantages over other assessments of aphasia have been outlined by Walsh (1978) as having standardised information, explicit test procedures, exact scoring criteria, and a profile format that allows comparison of individuals to groups of aphasics, brain damaged non-aphasics and normals. It has stimulus material that includes everyday objects which can be manipulated. Its selection of tests interpret recognised aphasic syndromes and these tests often have constraints that measure speed and appropriateness of response.

The Language Scales take an hour to administer, they explore auditory, gestural, visual and verbal modalities and their subtests are graded in difficulty.

The ability of the test to exclude the effects of intelligence and learning and so measure language performance as purely as possible is suggested by the perfect or near perfect scores achieved by healthy controls on 22 of 31 subtests. Nine subtests have a standard deviation greater than 1 but all demonstrate narrower ranges and higher scores than any pathological group.

Each of the subtests, and the reasons given for inclusion, are described along with procedure of delivery and scoring criteria below.

4.2.1. Materials

Test Booklet with Profile Sheets, Stopwatch.

Objects: Tray A Razor, Pipe, Torch, Bell, Glasses, Tweezers, Whistle, Watch

Tray B Gun, Scissors, Pen, Sponge, Fork, Cup, Brush, Box of Matches

Tokens: Twenty tokens of wood, in two shapes, circles and squares, and two sizes, large and small, and five colours, red, blue, yellow, white and green

The Objects and Tokens are displayed on Plates 4.1 and 4.2.

Cards with written names of objects of Tray B and cards with written instructions for tests 20/21.

A set of KOH Blocks and cards with WAIS designs

A set of Standard Coloured Matrices of Raven

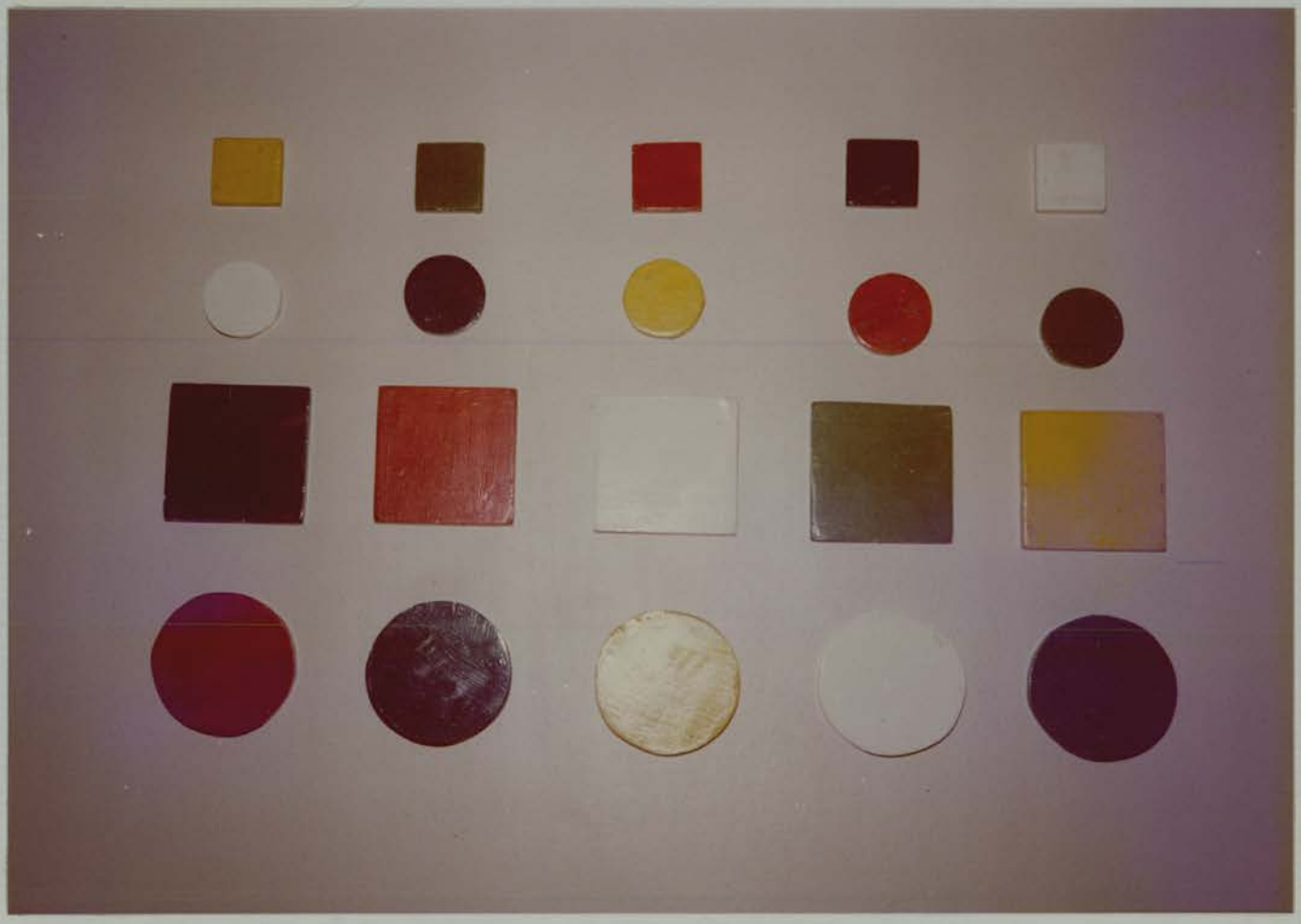
4.2.2. Subtests

1. Dementia Rating Scale (Memory, Orientation, Concentration

Rationale: The dementia rating scale is similar to that of Roth

Plate 4.1.

Plate 4.2



and Hopkins (1953). Lloyd (1970) used it to differentiate dementing and non dementing subjects. A shorter version has been produced by Hodkinson (1972). Blessed, Roth and Tomlinson (1968) correlated scores on this scale to senile plaque counts in the grey matter. Qureshi and Hodkinson (1974) used a short version of it to demonstrate severity in institutionalised and non institutionalised patients. Frackowiak et al (1981) used it as a measure of severity of dementia and related scores to oxygen uptake.

The scale assesses orientation in person, place and time and recall from remote and recent memory with a five minute "hold" constraint. It demands recall of personal and non personal information and automatic repetition by counting and reciting. There is a concentration test that demands reversal of automatic sequences.

Scoring: Each correct piece of information has a weighing of one to give a maximum score of 40.

2. and 3. Identification of Name/Identification by Function

Rationale: These are tests of verbal agnosia (Brain 1961) demanding visuo-auditory matching. By pairing the objects with identical ones the task can test for visual object agnosia.

Administration: The patient is asked to point to the objects of Tray A by names and Tray B by function.

Scoring: A score of one is given for each correct response. The maximum score for each test is 8.

4, 5 and 6 Token Test

Rationale: This is a short version by Spellancy and Spreen (1969) of a test of comprehension designed by De Renzie and Vignolo (1962). The test is designed to measure comprehension of instructions of increasing length and spatial, temporal and logico-grammatical complexity. Commands do not load the memory and redundancy is eliminated. The test is sensitive to disruptions of linguistic processes and can identify concomitant disabilities that may mask aphasic disorder or subtle symbolic processing disability. Part F of the test can in itself identify left hemisphere damage misclassified as nonaphasic because of the subtletyof linguistic impairment (Boller and Vignolo 1966).

Administration: Tokens are presented in set order and verbal instructions are given as on the answer sheet. The test is discontinued if Part A indicates agnosia or confusion of colour or shape.

Scoring: One point is scored for each part of the instruction. The carrier phrase of Parts A-E is excluded. Thus

"Show me the SMALL WHITE CIRCLE" is accredited 3 points and

"PUT the RED CIRCLE ON the GREEN SQUARE" is accorded 6.

The maximum score for Parts A-E is 67 and for Part F is 96.

The total score is 163.

7 Sentence Repetition

Rationale: Sentence repetition tests the integrity of the auditory system and the organisation of motor speech movements. It has been used as a basis of differentiating peri Sylvian from transcortical aphasias and reviewed by Benson (1979). Errors of repetition have been interpreted as conduction aphasia producing literal or nominal paraphasias. Warrington and Shallice (1969) have suggested the deficit to be one of auditory memory span while Strub and Gardener (1974) have argued the deficit to be linguistic.

The ability to repeat may depend on the predictability of the stimulus (Goodglass and Kaplan 1982). Newcombe and Marshall (1976) were able to demonstrate the greater difficulty of left hemisphere damaged patients to repeat sentences with increasing syntactic complexity. The average adult is able to repeat sentences of 28 to 29 syllables (Lezak 1976). Recently Vargo and Black (1984) were able to provide normative data for the sentences used in this particular battery for neurologically impaired patients and have indicated that intelligence, and to a lesser degree memory, may be related to performance.

Administration: The patient is instructed to repeat the listed sentences. The test is discontinued after three successive failures.

Scoring: A score of 1 is given for each sentence correctly repeated. The maximum score is 22.

8 and 9 Digit Repetition (Forward and Reverse)

Rationale: Digit span has traditionally been included in examinations of intelligence. It is easy to administer and score and measure immediate auditory memory span for unrelated material compared to sentence repetition which is a measure of recall in the meaningful context of a sentence. Digit repetition is more sensitive to left than right sided or diffuse damage and remains sensitive to brain injury. A difference between digits forward and in reverse suggests a concentration deficit. Normal ageing processes do not affect auditory memory span but does affect concentration and mental tracking (Lezak 1976).

Administration: The patient is asked to repeat strings of listed digits. Two trials are given for each series and failure at two consecutive trials constitutes termination criteria. Patients who are expressively aphasic may be asked to point to the digits written down.

Scoring: One point is awarded for each trial passed. The maximum score for each test is 14. Should the total number of trials for each test be added, the combined score may be normed against WAIS data.

10 Automatic Speech

Rationale: Material rote learned can be recalled unthinkingly and effortlessly and is the least perishable of learned verbal habits (Lezak 1975). It is also least vulnerable to brain damage (Luria 1970). Luria comments that such activity is a verbo-motor habit and"does not constitute speech in the true sense of the word" because it is not propositionally formulated.

Administration: The patient is asked to count to 20 and to recite the days of the week.

Scoring: Two points are given for both the ability to count and the ability to recite. One point is deducted for errors of sequence. The maximum score is four.

11 Confrontation Naming

Rationale: The study of naming ability has a long history in aphasia testing being considered a disturbance of memory function (Goldstein 1924), agnosia (Rochford 1971), symbolic dysfunction (Head 1926), or conceptual impairment (Bay 1962). The inability to name may also be related to phonological complexity, levels of abstraction, pictureability of stimulus, word frequency and word length (Kertesz 1979, Benson 1979). Errors of the substitution of incorrect target phonemes (phonemic or literal paraphasia) often have features of the target and may be, for example, errors of voice, place or manner of articulation. Semantic paraphasias, or word substitution errors, are often in the same generic class. They are nominal paraphasias if nouns, verbal paraphasias if a verb form is given, a circumlocution if the response refers to the noun, and a neologism if the response is meaningless jargon. The loss of substantial nouns and the substitution of indefinite words produces vague and rambling discourse called "empty speech".

Administration: The patient is asked to name the objects in Tray A. If there are any errors Tray B is presented. The

patient is not allowed to touch the objects in order to restrict the task to visual confrontation naming.

Scoring: Paraphasias are not accepted and one point is credited for each correct response. If Tray A is named successfully the score is doubled. The maximum raw score is 16.

12 Description of Function

Rationale: This task differs from confrontation naming because it requires the patient to abstractualise the nature of the object. It seems that nouns rather than verbs are compromised in brain damage. The reasons for this are unclear. Luria's application of the Pavlovian principle of the Law of Strength, states there may be fewer competing responses for verb than noun forms and therefore the stimulus range is smaller (Luria 1973).

Administration: The patient is shown each object from Tray A and asked what he would do with it. The items have been selected for noun-verb disagreement. If there are any errors Tray B is presented.

Scoring: A score of 1 is credited for each correct response. If Tray A is named successfully the score is doubled. The maximum score is 16.

13 and 14 Tactile Naming (Right) and Tactile Naming (Left)

Rationale: This is a test for asteriognosis or tactile agnosia described by Fredericks (1973) and Beauvois et al (1978). A comparison with confrontation naming will discriminate a sensory - perceptual from a language deficit. Brain (1961) considered differences between tactile agnosia and asteriognosis as the former being a disturbance of both superficial and deep sensibility while asteriognosis results from sensory impairment. He noted it may be impossible physiologically to distinguish cortical sensory function and cortical perceptual function. Tactile agnosia is associated with lesions of the contralateral parietal lobe. Left hand agnosia may also be associated with callosal lesions and bilateral tactile agnosia in right handed persons may result from lesions of the left hemisphere. Brain (1961) concluded that the left hemisphere appears dominant for tactile recognition. Geschwind (1965) has indicated that different scores for each hand indicate a disconnection, the failure of a stimulus palpated in the dominant hand to be perceived by the non dominant hemisphere by a lesion of callosal fibres.

Administration: The patient is instructed to close his eyes. Objects from Tray A are placed in the right hand and then objects from Tray B in the left hand. He is required to name each object on presentation.

Scoring: One point is scored for each object to produce the maximum score of 8 for each hand.

15. Gesture (Object Use)

Rationale: The inability to gesture the use of objects has been described as ideational apraxia (Morlaas 1921 quoted De Renzie et al 1968). Many feel that ideational apraxia is a severe form of ideomotor apraxia (Sittig 1931, quoted De Renzie et al 1968) while some consider it to be a disturbance of symbolic function (Goldstein 1948), that is, an aspect of aphasia as an impairment of symbols of comprehension and expression crossing all modalities.

Administration: The patient is asked to demonstrate the use of the objects in Tray A, if there are any errors Tray B is presented.

Scoring: Partial gesture is not accepted and a score of 1 is given for clarity of execution. The maximum score is 16.

16. Fluency

Rationale: Vernon (1971) identified five fluency factors as ideational fluency, associational fluency, expressional fluency, oral fluency and word fluency. Word Fluency is Thurstone's /w/ factor, the producing of as many words with certain characteristics as possible in a limited time. In this test list words beginning with F, A and S are asked with a minute for each letter. Howes (1964) has reported diminished speech rates in aphasia and Borkowski et al (1967)

has noted the test not only sensitive to aphasia but intelligence levels in patients with cerebral disease. Difficult and uncommon letters discriminate higher intelligence while easy or common letters discriminate normal and brain damaged populations. Struss and Benson (1983) have described linguistic impairment on this test in terms of precise frontal lobe deficits. Miller (1977) determined that although fluency rates in such tests were diminished in dements their frequency in spontaneous speech was not.

This test is given with behavioural limitations, the patient is instructed not to repeat himself, not to give proper nouns and not give words that differ only by changing the final morpheme. The breaking of such constraints is suggestive of frontal lobe pathology (Walsh 1978).

Administration: Patients are instructed to repeat as many words beginning with a given letter for a minute but not to repeat themselves, not to give the names of people or places and not to give words that are the same but with different endings. The letters administered are F, A and S. Scoring the total sum of all admissible words, the ceiling is 60.

17 Sentence Construction (Masselon Test)

Rationale: Agrammatic, telegraphic language is characteristic of frontal lesions. Paragrammatic speech which is fluent but marked with errors of morphological endings and word order is

characteristic of the language of posterior lesions (Howard 1985). Paucity of output, or inertia of speech, is characteristic of pre-frontal lesions producing the syndrome of transcortical motor aphasia or dynamic aphasia (Luria 1970). Dynamic aphasia may reflect a reduced ability to convert thought into sentences through a lack of "linear scheme of phrase". Luria (1970) argued the condition to be a disturbance of inner speech with its characteristics of abbreviated forms and predicative function.

Administration/Scoring: The patient is required to generate a sentence that includes stimulus words. An attempt is accredited with 3 points if the sentence is grammatically acceptable. Two bonus points are added if the task is completed within ten seconds and one if the task is completed within 20 seconds. The test has a time constraint of 30 seconds. The maximum raw score is 25.

18/19 Reading Words and Word Recognition

Rationale: Luria (1970) noted that reading words is less impaired in traumatic aphasia than writing because it is an overlearned and automatic skill. Critchley (1964) noted that reading vocabulary was more likely to be retained in dementia than spoken vocabulary. Reading demands particular abilities of sound letter synthesis and forward perception to allow correct pronunciation of phonemes. Visual defects or neglect of visual attention, optikinetic function, perception of forms, spatial relations and symbolic significance may impair word recognition (Kertesz 1979).

Aphasic alexia refers to the reading, and writing, disorders in aphasia and usually accompanies the particular aphasic syndrome.

Alexia may also exist with or without agraphia or associated with aphasia. Alexia without agraphia is pure word blindness. It appears as a result of lesions of the dominant occipital lobe and the splenium of the corpus callosm disconnecting the right hemisphere (Benson and Geschwind 1969). Alexia with agraphia is part of a Gerstmann Syndrome and the dominant angular gyrus is usually involved.

Administration: Eight words are presented on cards, the patient is required to read them aloud and match them to appropriate objects.

Scoring: A score of 1 is given for each correct response. The maximum score is 8 for each test.

20/21 Reading Sentences and Sentence Comprehension

Rationale: Since the most common mistake in testing reading ability is to equate it with comprehension (Kertesz 1979) this test is included to indicate the ability to read and understand or, in patients with severe articulatory disorders, the ability to act on written instructions read sub vocally.

Administration: The patient is required to read an instruction relating to the Tokens and act accordingly.

Scoring: A score of 1 is given for each sentence read and a score of 1 for each part of the written instruction, as in Part F of the Token Test. The maximum scores are 7 for reading and 32 for comprehension.

22. Automatic Writing

Rationale: Written language is probably the most complicated and fragile of all language skills (Brain 1981) although aphasias will, in the face of verbal impairment, often manage automised sequences and sign their names, write their addresses, and take telephone numbers (Ulatowska et al 1979). As writing tasks become more conscious disruptions become more evident (Luria 1970, Benson 1979).

Administration: The patient is required to write his name and address.

Scoring: The maximum score is 5 for Christian Name, Surname, House Number, Street and Town.

23 Spelling

Rationale: This task demands patients to demonstrate an inner auditory schema of words and write them. Graphic skills can be judged in terms of calligraphy as the mechanics of writing or orthographically as spelling. Parietal syndrome produces calligraphic disruption and difficulty on copying tasks (Critchley 1953). Orthographic disturbance is generally reflected as paragraphia in temporal lobe disturbances (Luria 1970) and perseveration in frontal lobe disturbances (Benson 1979).

Administration: Objects in Tray A are presented to the patient who is instructed to write their names. If the name is supplied it is noted and scored normally.

Scoring: The writing of names is scored by giving a credit of 3 to each word correct, 2 for words with one spelling error and 1 for words with two errors. Calligraphy is judged generously in patients with right hemiplegia or paresis. The maximum score is 24.

24/25 Dictation and Copying

Rationale: Writing to dictation demands complex psychological mechanisms of holding an auditory trace which is unstable, compared to writing to copy where the visual trace is stable.

Aphasic agraphia is marked in spontaneous writing and dictation rather than copying which differentiates orthographic and calligraphic errors by direction and rotation of words and letters (Critchley 1953, Kertesz 1979). In aphasia, dysgraphia correlates well with calculation, reading, the Raven's Coloured Progressive Matrices, drawing and comprehension, and less with praxis and naming. There appears to be no relationship with fluency or repetition (Kertesz 1979).

Administration/Scoring: The patient is required to write the dictated sentences:

This is a very nice day.

This brick building was built last year.

A score of 1 is given for each word correctly spelled and legible, although this is judged generously in patients with motor disability. 1 point is subtracted for each word duplicated. The maximum score is 13. The patient is then shown cards with two sentences for copying:

I am very hungry.

The colour of the walls is green.

A score of 1 is given for each word copied correctly and legibly. 1 point is subtracted for all words duplicated. The maximum score is 11.

26 Calculation

Rationale: Calculation has been described by Critchley (1953) as the ability to process symbolic thought. The task demands high level operations, the transfer of arithmetical symbols, spatial orientation of numbers in vertical and horizontal columns, and memory of multiplication values and function of systems for borrowing or transferring when adding or subtracting. Poor calculation appears to correlate with reduced ideation (Sittig, 1921) quoted Grewel 1973) and spatial disorders (Heneshen 1920 quoted Grewel 1973). It may be asymbolic as failure to recognise signs: digit notation may accompany disturbances of inner language, and (according to Poppelreuter(1917), quoted Grewel 1973) always accompany aphasia. Occipital, temporal and parietal acalculia have been described (Critchley 1953) while Heneshen described it as a frontal syndrome.

Administration: The patient is required to complete ten arithmetical problems demanding six simple and four complex processes.

Scoring: The maximum score is 10, one point for each correct calculation.

27 Oral Apraxia

Rationale: Apraxia is defined as the inability to perform purposive movements without misunderstanding or motor impairment.

Oral apraxia, or bucco-facial apraxia, has been implicated in Broca's aphasia (de Renzie et al 1966). Geschwind (1965) considered this a sympathetic apraxia that spared the limbs but affected the representation of the face on the motor strip that lies closer to Broca's area.

Administration: The patient is instructed to make simple oral movements, if necessary these are demonstrated.

Scoring: Two points are scored for each act, whether demonstrated or on verbal request, judged correct. 1 is given for approximations. The maximum score is 20.

28 Ideomotor Apraxia

Rationale: This test demands the execution of simple gestures on demand or by imitation. Ideomotor or Limb apraxia may be responsible for the diminished ability of aphasics to gesture (Goodglass and Kaplan 1963) although there is also an argument that failure to gesture results from central impairment of all communication systems (Finkleberg 1870 , quoted Goodglass and Kaplan 1963)

It is noted that imitation plays a large part in the nature of apraxia and patients may perform on action spontaneously but not voluntarily, they may lick their lips, wipe their brow or issue a series of fluent movements when there is demonstrable oral or limb apraxia. Benson (1978) has described a model for

unilateral and bilateral limb apraxia suggesting lesions between Wernicke's area and the dominant motor association area are responsible for bilateral apraxia, between the dominant motor association area and the motor cortex for preferred limb apraxia, and of the corpus callosum for apraxia of the non dominant limb.

Administration: The patient is instructed to perform simple gestures with his arm and if necessary, the task is demonstrated.

Scoring: Two points are scored for each act, whether demonstrated or on verbal request, judged correct. 1 is given for approximations. The maximum score is 20.

29 Constructional Apraxia

Rationale: Constructional apraxia has been described as "a disturbance is formulative activities such as assembling or drawing, in which the spatial form of the product proved to be unsuccessful without their being a disturbance of single movements" (Benton 1969). It is associated with lesions of the non speech hemisphere although unilateral neglect may result from lesions of the contralateral hemisphere.

There may be two kinds of constructional apraxia, that for the two dimensional and that for three dimensional tasks (Benton 1969). Drawing is a test of two dimensional ability using common figures from the Benton Visual Retention Test (Benton 1963). They

demand synthesis of size, shape and linear patterning. Problems that arise are poor quality of line, poor closure or overlapping, perceptual loss and scattering of components, reversals, rotations, misalignments and distortions of size. Warrington et al (1966) has argued that drawing should be differentiated from constructive tasks in constructional apraxia.

Lesions producing such behaviours seem, on the whole, to be right sided parieto-occipital producing fragmented, scattered drawings and are associated with dressing apraxia or neglect (Benton 1969).

Administration: The patient is required to copy figures presented on six cards. They are of increasing difficulty of dimension and sequences.

Scoring: The maximum score is 20, 2 points for each of the first four figures and 2 points for each of the three figures on the last two cards, the figures varying in size, complexity and rotation. One point is awarded for approximations.

30 Block Design

Rationale This test is taken from the WAIS as a test of three dimensional apraxia after Critchley (1953). Benton (1969) has argued that, if intelligence be "the capacity to analyse a situation to discover methods of solving it and synthesising the details into a consistant unity", tests of three dimensional

construction are better indicators of cerebral disease than the full WAIS scale for they demand components and spatial analysis, component assembly, sustained attention and planned activity. Matarazzo (1972) has commented that the test correlates best to the WAIS verbal scale of Comprehension, Vocabulary and Information, and those who perform it best do not see the patterns as wholes but as units. Fuld (1983) reports patients with ATD will score less on Block Design than a scaled score of 5. Perez et al (1975) found Block Design the best test in discriminating ATD, MID and vertebro-basilar insufficiency.

Administration/Scoring: The patient is required to assemble ten patterns from stimulus pictures, the first two are modelled and bonus points are given for rapid performance on the final three. The maximum score is 48.

31 The Coloured Progressive Matrices of Raven (1977)

Rationale: The RCPM are added as a test of non verbal intelligence with the minimum of instruction and the minimum response. They are effective in identifying organically impaired patients (Zimet and Fishman 1970) and correlates highly with non verbal and visuospatial reasoning (Colonna and Faglioni 1966, Archibald et al 1967). There is evidence (Zangwell 1964, McQuigan 1970, Sokalov 1972) that they demand sub vocal rehearsal and thus are an index to "inner language", that plans, regulates, accompanies and monitors behaviour. The

Matrices demand the ability to match, close and reason and are ideal for use with patients with language deficits for they correlate highly with verbal intelligence.

Administration/Scoring: There are 36 plates in the test, each scores 1 credit. The maximum score is therefore 36.

4.2.3 Profile Forms

All patients in the study, along with controls, were initially assessed and the statistical data was then used to standardise the Scales in percentiles. This allows for any one patient to be profiled against Normals, Dements and Aphasics.

4.3 SUBJECTS

Cases represented in five experimental groups include healthy controls, dements, aphasics, and a diagnosed group with Korsakoff psychosis.

4.3.1 Controls

Age-matched normal subjects were recruited through the Ladywell Medical Practice and the Leith Medical Practice after this research proposal passed the North Lothian Ethics Committee. Controls were selected at random from the Age/Sex files of the practices. All were free of all psychotropic medication and had no history of neurological disease. See Table 4.2.

Table 4.2

NORMAL CONTROLS: WHOLE GROUP

	Number	Mean Age (Yrs)	Age Range (Yrs)
Male	39	65	44 - 86
Female	22	69	45 - 87
TOTAL	61	67	44 - 87

4.3.2 Dements

Sixtysix demented patients recently diagnosed with behavioural and clinical criteria, and referred by GPs for psychogeriatric consultation, and admitted to the Royal Edinburgh Hospital, or the City Hospital, Edinburgh, or to acute wards of The National Hospital for Nervous Diseases, Queens Square, London, or The Department of Neurology, St Mary's Hospital, Paddington, were constituted to standardise the Language Scales.

An experimental group of 20 primarily presenile dements referred to the Royal Edinburgh Hospital from 1981 to 1984 was also constituted. Patients were initially seen by a consultant and following a presumptive diagnosis of dementia were admitted to the brain metabolism unit for a period of two to three weeks. They were given a complete medical and psychological examination and a diagnosis of ATD made if they met the following criteria.

(1) Onset of symptoms under the age of 65 with memory impairment as a presenting feature

(2) Steadily progressing deterioration

(3) Absence of a history suggestive of another type of dementia

(4) Absence of focal neurological signs and hypertension

(5) CT scan normal or showing cerebral atrophy and no additional pathology

(6) EEG showing no focal activity

(7) Haematological, biochemical and CSF parameters within laboratory normal value

(8) Normal ECG

(9) Psychological testing showing deficits in some or all of the following: orientation, immediate memory, logical memory, paired associate learning, with or without apraxia or aphasia

(10) All patients free of psychotropic medication

Demented patients who failed to meet these criteria were diagnosed as possible ATD, MID, Pick's disease or a dementia of unknown aetiology.

With the permission of Dr Daffyd Thomas of St Mary's Hospital, Department of Neurology, London, nine patients who fulfilled the criteria were added to the ATD group making a cohort of 20.

The 66 dements used to standardise the test battery are described in Table 4.3. The ATD cohort are described in Table 4.4 and those who were subsequently tested, for up to four consecutive

years are described in Table 4.5. Table 4.6 describes the ATD cohort in terms of age, sex, handedness and occupational status. Table 4.7 gives the dates of test-retest examinations.

A cohort of 12 patients with evidence of cerebral infarcts on CT scan and thus a possible diagnosis of MID was also constituted. It is described in Table 4.8.

Table 4.3

DEMENTIA: WHOLE GROUP

	Number	Mean Age (Yrs)	Age Range (Yrs)
Male	28	66	43 - 83
Female	38	71	45 - 91
TOTAL	66	69	43 - 91

Table 4.4

ATD: WHOLE GROUP

	Number	Mean Age (Yrs)	Age Range (Yrs)
Male	8	60	54 - 71
Female	12	61	54 - 67
TOTAL	20	60	54 - 71

Table 4.5

ATD: TEST - RETEST GROUP

	Number	Mean Age (Yrs)	Age Range (Yrs)
Male	4	58	54 - 64
Female	7	60	54 - 67
TOTAL	11	59	54 - 67

Table 4.6

PATIENT, DATE OF BIRTH, SEX, HANDEDNESS AND OCCUPATION OF ATD COHORT

Patient	Age at Initial Testing (Yrs)	Sex	Handedness	Occupation
1	63	F	R	Housewife
17 *	54	F	R	Housewife
32 *	64	F	R	Farm Worker
28 *	64	M	R	Railwayman
33 *	62	F	R	Cleaner
29 *	58	F	R	Clerkess
27 *	67	F	R	Nurse
31 *	61	F	R	Tailoress
30 *	56	M	R	Storeman
3	54	M	R	Oil Refinery Worker
2	59	M	R	Driver
4	54	M	L	Unknown
5	58	M	L & R	Electrician
6	67	M	R	Builder
7	67	F	R	Housewife
8	65	F	R	Housewife
9	54	F	R	Shopkeeper
10	62	F	R	Housewife
11	71	M	R	Chef
12	65	F	R	Housewife

* Asterisk denotes patients on test - retest trial (see Table 4.7)

Note: The profiles of cases 17, 32, 33, 29, 27, 31 and 30 are presented in Chapter 5 Part 2.

Table 4.7

ATD: TEST RETEST DATES

Name	Test 1	Test 2	Test 3	Test 4
17	1. 8.83	6. 9.84	13. 9.85	
32	15. 1.82	31. 8.83	16. 3.84	12. 9.85
28	30.12.83	28.10.84	17. 9.85	
33	6. 8.82	5. 9.83	5. 8.84	16. 9.85
29	8. 2.82	9. 2.83	13. 1.84	
27	8. 7.82	4. 8.83	8. 8.84	
31	7. 6.83	10. 8.84	18. 9.85	
30	7. 7.82	1. 9.83	26.10.84	13. 9.85

Table 4.8

MID: WHOLE GROUP

	Number	Mean Age (Yrs)	Age Range (Yrs)
Male	3	72	69 - 77
Female	9	72	63 - 77
TOTAL	12	72	63 - 77

4.3.3. Aphasics

A population of forty five patients with dysphasia following circumscribed left hemisphere lesions was assembled from Speech Therapists Clinics at the Royal Infirmary of Edinburgh, The Astley Ainslie Hospital, Edinburgh, The National Hospital for Nervous Diseases, Queens Square, London, and St Mary's Hospital, Paddington, London. The numbers, mean ages and age range of all the population of dysphasics used to standardise the battery is given as Table 4.9.

From this population an Experimental Group of 23 was selected according to the following criteria:

(1) Radiological imageing indicating focal left hemisphere lesion through embolus, haemorrage, or space occupying mass

(2) Normal haematology, including B_{12} and folate, normal renal hepatic and thyroid function tests and VDRL screening

(3) Evidence of dysphasia in both verbal and written language

(4) Patients free of psychotropic medication

From this experimental group 13 patients who demonstrated preserved repetition with either dysfluency (Transcortical Motor Aphasia) or poor comprehension (Transcortical Sensory Aphasia) after Kertesz (1983) were created. They are described in Table 4.10

The other 10 aphasics constituted a test-retest population, and each member of the group was assessed and reassessed at not less than 3 months and not more than 2 years. They are described at first testing in Table 4.11.

Table 4.9

PATIENTS WITH CIRCUMSCRIBED LESIONS

	Number	Mean Age (Yrs)	Age Range (Yrs)
Male	24	64	38 - 78
Male	21	65	44 - 83
TOTAL	45	64	38 - 83

Table 4.10

TRANSCORTICAL APHASICS

	Number	Mean Age (Yrs)	Age Range (Yrs)
Male	5	59	38 - 78
Female	9	58.2	25 - 76
TOTAL	13	58.5	25 - 78

Table 4.11

TEST-RETEST APHASICS

	Number	Mean Age (Yrs)	Age Range (Yrs)
Male	6	58.1	38 - 78
Female	4	62.5	44 - 68
TOTAL	10	60.3	38 - 78

4.3.4 Korsakoff Psychosis

An Experimental Group of fifteen subjects with a diagnosis of Korsakoff Psychosis was selected from patients of the Royal Edinburgh Hospital. Fourteen were In-Patients and two were Out-Patients.

All patients in this group satisfied the criteria of St Clair et al(1985)

(1) A definite history of prolonged alcohol abuse, usually in excess of twenty years, together with a past episode of either acute confusion of Wernicke's encephalopathy with or without the presence of peripheral neuropathy

(2) A severe anterograde amnesia

(3) Absence if history suggestive of progressive illness no alcohol abuse at least six months prior to testing

(4) No previous psychiatric history or evidence of mental deficiency

(5) Normal haematological and biochemical tests including B_{12} and folate, normal renal, hepatic and thyroid function tests and negative VDRL

(6) EEG showing no focal activity

(7) No psychotropic medication for at least four weeks before testing

Numbers, mean ages and age ranges are given at Table 4.12.

Table 4.12

KORSAKOFF PSYCHOSIS: WHOLE GROUP

	Number	Mean Age (Yrs)	Mean Range (Yrs)
Male	12	64	57 - 69
Female	3	70	59 - 79
TOTAL	15	67	57 - 79

4.4 STATISTICAL BACKGROUND

The Introduction has delineated the problems to be faced and questions to be asked about the very nature of language pathology in dementia. This chapter has described procedures in the construction of tests of neurolinguistic measurements.

One major criterion for tests of language dysfunction is that they aught to discriminate between normal, demented and aphasic language by assigning individuals to diagnostic categories through differential prediction. The problem of "aphasia" in dementia has received special interest by neurologists and linguists suggesting a difference in the two disorders is more than just a question of labelling. A statistical procedure for classifying aphasics against dements is a dyscriminant function analysis.

If "aphasia" is identified against normal language the analysis aught to indicate which of the test variables are the most powerful predictors of pathological language function. Since there are 31 variables the test battery multiple regression will determine the most

powerful predictors and they will classify the populations differentially as patients with aphasia producing lesions, patients with diffuse lesions and so again by discriminant function analysis classify aphasics and dements.

Two methods of profiling an individuals performance against a larger population are the use of Z scores, which is appropriate for distributions that are normal, and percentiles that are both appropriate for skewed distributions are not affected by deviations from normality. Profiles are produced for normal, aphasic, and dementing populations because the variation of scores between the three groups is too great to be condensed on to one form. The profile formats on individual patients is presented in Appendix I.

Another criterion of a language battery is that a relationship should exist between tests that tap similar abilities demonstrating that there is an internal consistency and comparability of scores. Such relationships can be presented in a correlation matrix. The basis of correlations remains unclear; relationships may exist on an anatomical, physiological or psychological similarity. The weakening of relationships in the correlation may be a marker for the deterioration and dissociation of abilities in complex multifactorial tasks.

Whether the language of dementia may be called aphasia depends on the aetiology, site of lesion, history and nature of the disorder. Statistically a test - retest design aught to test the hypothesis that aphasics improve while dements deteriorate. To determine whether aphasics do improve over time on repeated measures the Wilcoxon Matched Pairs Signed Ranks Test is employed.

Whether aphasia and the language of dementia are different in their nature can be determined by factor analysis. Classification of factors assigns variables to classes that appear homogeneous on the basis of variance. The classification may reflect the design structure of the Language Scales or it may cluster variables according to underlying psychological function as, for example, visuo spatial ability, writing ability, praxis or comprehension. It is generally considered the first factor is the most sensitive index of task difficulty, complexity or severity of disorder. Once factors are assigned to clusters they may be "christened" according to similarity of function.

Factor analysis allows examination of underlying features and relationship of test variables. If aphasia were analogous to both focal and diffuse damage one would expect factors to be common to both groups with strong similarity in rank factor loadings. Failure of such commonality lends to the conclusion that the two conditions are fundamentally different.

Degree of impairment in progressive neurological disease may be examined either cross-sectionally, as "mild", "moderate" or "severe", or longitudinally by tracing the natural history of the disorder. Cross sectional staging may be determined from a histographic distribution of scores of a given population on a dementia rating scale. Test variables can then be correlated to that staging degree. Impairment may relate to other variables, length of onset, age, institutionalisation, or premorbid history. Of these variables only severity and age can be

compared to language abilities. Onset of ATD requires family information, premorbid history was not documented or eliminated in selection criteria and none of the patients were institutionalised.

As the introduction outlines, longitudinally the history of ATD has not been documented, in this study a test for trends will indicate significance of progressive severity of impairment of language acts. The trend test described by Page (1963) is employed.

Finally, the literature has described ATD in terms of other diseases producing cognitive impairment. In order to make a comparison and to test the null hypothesis that there is no difference between pathological groups a Mann Whitney U Multiple Comparisons test is employed between ATD patients, normals and three other abnormal groups. The median scores are noted and the Mann Whitney procedure provides the significance of the difference of the scores between each population and patients with ATD.

CHAPTER 5

RESULTS OF INVESTIGATION

5.1 THE PREDICTIVE POWER OF THE LANGUAGE SCALES

5.1.1 Discriminant Function Analysis: Aphasics and Controls

To determine the precision of the Language Scales in differentially discriminating pathological groups a stepwise discriminant function analysis using the BMDP M (1977) was employed. The test data and score configurations of the 45 aphasics, 61 controls and 66 dements was used to determine this diagnostic classification.

"Aphasia" is meaningful in terms of how a population with focal lesions and pathological language compares with healthy controls with ho history of neurological disease. Thus in order to determine the nature of aphasia 45 aphasics and 61 controls were pooled for a discriminant function analysis.

The Dementia Rating Scale was omitted from the discriminant function analysis. Stepwise regression indicated that 21 of the Language Scale's 30 variables were predictive: the two most powerful being Word Fluency, and Sentence Construction.

These two variables correctly classified 90.6% of the cases. The two strongest predictors were as Word Fluency, accessing a semantic or lexical word store, and Sentence Construction, a syntactic measure of language.

Table 5.1 below summarises the classification Matrix for aphasics and controls by stepwise regression analysis and Figure 5.1 is a histogram of canonical variance.

Table 5.1

CLASSIFICATION MATRIX

	% Correct	Controls	Aphasics
Controls	98.4	60	1
Aphasics	80.0	9	36
TOTAL	90.6	69	37

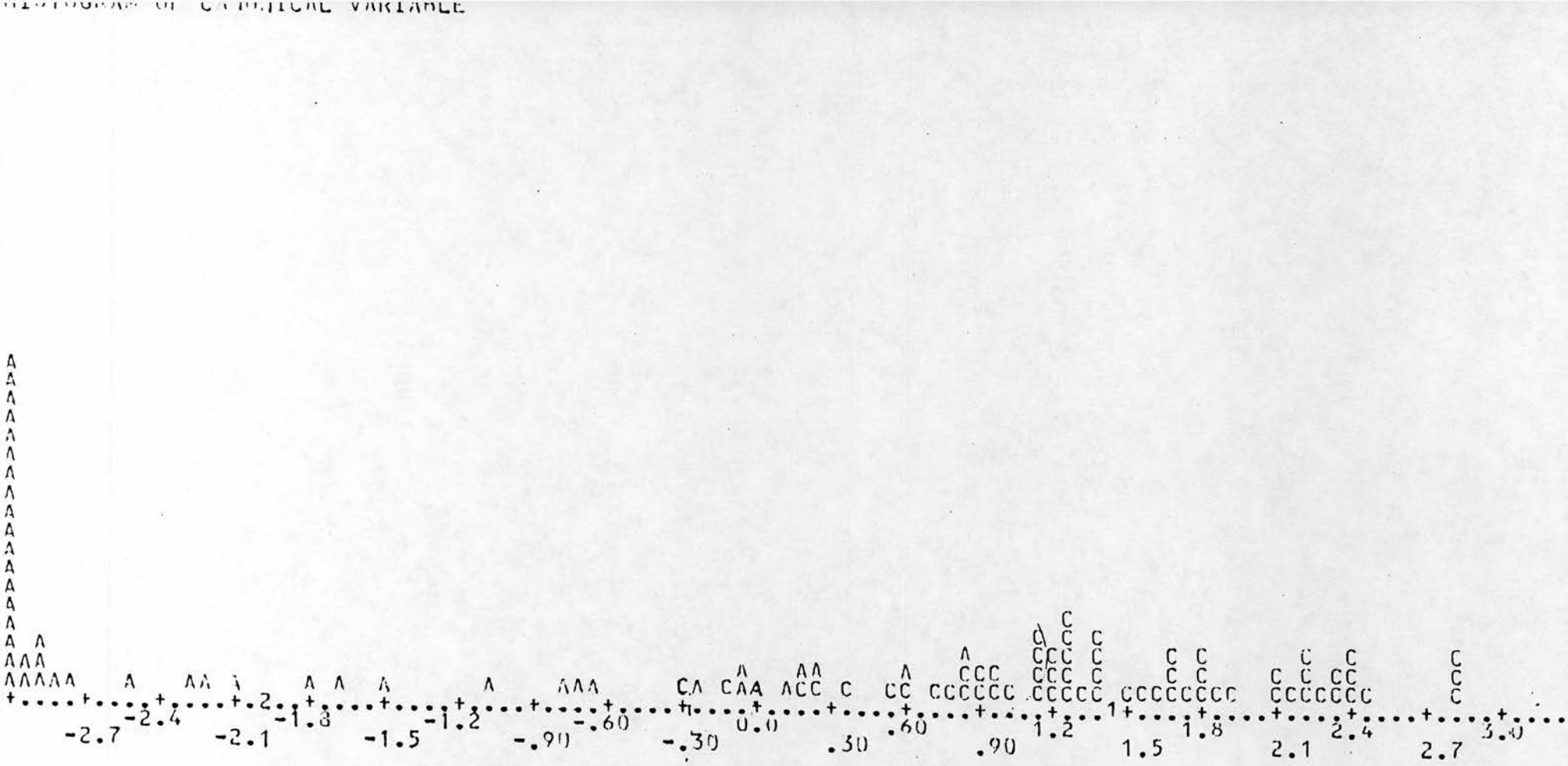
5.1.2 Discriminant Function Analysis: Aphasics and Dements

The two variables of Word Fluency and Sentence Construction were then introduced to the demented population by stepwise discriminant analysis. Table 5.2 summarises the classification matrix of dements as aphasics using the two variables. They correctly classified 86.5% of the cases as aphasic or demented. Figure 5.2 is a histogram of the canonical variable.

Table 5.2

CLASSIFICATION MATRIX

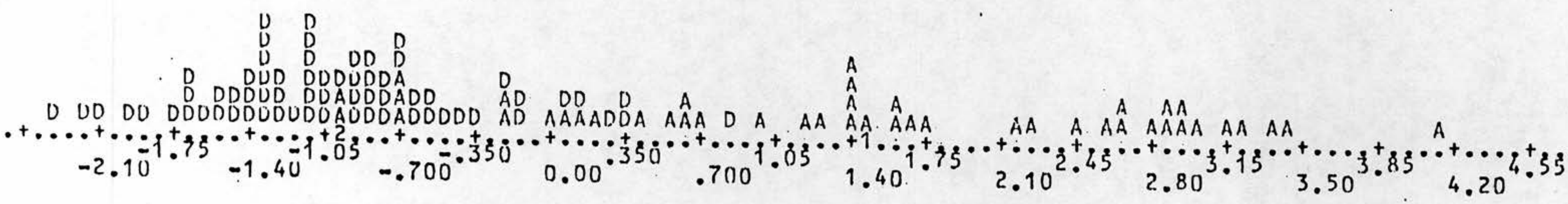
	% Correct	Aphasics	Dements
Aphasics	75.6	34	11
Dements	93.9	4	62
TOTAL	86.5	38	73



Discriminant Function Analysis: Aphasics/Controls: Histogram of Canonical Variable.

Figure 5.1

Figure 5.2



Discriminant Function Analysis: Dements/Aphasics: Histogram of Canonical Variable.

5.1.3 Discussion

One control was reclassified as an aphasic and 9 aphasics were reclassified as controls. Eleven aphasics were reclassified as demented and 4 dements were reclassified as aphasic.

A combination of the analyses suggests mild aphasics may operate within normal limits on these assessments, although it is rare to have healthy controls reclassified as aphasics for language scales are designed for maximum performance of the non neurologically impaired. High level aphasics without focal signs but general reduced cortical ability may mimic dements despite maximum scores on the Dementia Rating Scale.

The two tasks that were used to discriminate the populations are "cognitive" in that Word Fluency is a measure of Thurstone's /w/ factor and Sentence Construction demands a working memory able to hold three units.

5.1.4 Discriminant Function Analysis: Dements and Controls

Another analysis indicated the Language Scales were able to classify all controls correctly but nine dements were reclassified as controls on six variables: Dementia Rating Scale, Confrontation Naming, Tactile Naming (Left), Spelling, Constructional Apraxia and the RCPM. The Dementia Rating Scale alone classified 62.9 of the dements while the other five variables correctly classified 42.8% of the cases.

13.6% of this randomly selected "demented" population can be considered still within normal limits. Walker (1982) found 14.8% or 12 of her normal cohort of 82 demonstrated mild intellectual impairment. Figure 5.3 presents a histogram of canonical variables of a discriminant function analysis of controls and dements.

The fact that all controls in this study were correctly classified is an indication of the selection criteria that eliminated individuals with any history of neurological disease. Of the 9 dements who were recalssified as normal 5 were brought into hospital for investigation of onset of ATD, affective disorder, vertebro-basilar insufficiency, right and left parietal infarcts and a metabolic disorder that resolved within twelve months. One was a clear case of Pick's disease who on initial assessment was well orientated and one had a queried diagnosis of ATD with right parietal signs. One produced a score on the Set Test (Isaacs and Kennie 1973) which is within the normal range. The last patient was diagnosed MID and died within six months.

5.1.5 Conclusions

The scales were also able to correctly classify 90.6% of cases as either aphasic or normal and 86.5% of cases as aphasic or demented. Those aphasics who were reclassified as demented were all recovered aphasics, "high level" aphasics or aphasics without strong focal signs of language impairment. The 4 patients reclassified as aphasic rather than demented were one who presented language pathology as an initial feature in ATD, one ATD with linguistic deficits generally more severely apparent than global cognitive

deficits, one with Pick's disease with indication of a pathology corresponding to the supplementary motor area and one MID with specific language and memory deficits.

Figure 5.3

Discriminant Function Analysis: Controls/Dements: Histogram of Canonical Variable

CHAPTER 5 (contd.)

RESULTS OF INVESTIGATION

5.2 PROFILING LINGUISTIC DISABILITY

5.2.1 Standardisation of Data

Standardised linguistic data for dementia does not exist. There are standardised scores for aphasics (Goodglass and Kaplan 1982, Spreen and Benton 1969) and "brain damaged non aphasics" (Spreen and Benton 1969).

To standardise the Language Scales the raw scores for the normal, aphasic and demented populations were converted to separate scales as percentiles.

This procedure has practical advantages over the use of Z scores. Percentiles are not affected by deviations from the norm in the distribution of subtest scores. It is easier to read percentile scores. Unlike percentiles, Z scores need to be understood in terms of a standard deviation above or below a zero mean. Gradations in percentiles can be finer and interpolations between decile markings easier. Language scores produce skewed distributions with the median score at the fiftieth percentile. Percentile profiles are now commonly established in aphasia testing (Goodglass and Kaplan 1982).

It was necessary to produce three profile forms because many dementing and aphasic scores fell below the entire distribution of the control group.

The profiles allow for the immediate indication of the severity of language disorders in all groups as well as the grading of strengths and weaknesses over particular tests. There is also a clear display of the passage of the disorder on test retest procedures.

Raw scores were not corrected for age and education. The controls were matched with dements and aphasics, as far as was possible, for age and education. Testing in Edinburgh indicated a surprising uniformity of educational opportunity and duration: few people tested did not leave formal education at 14 or 15 years. The language tasks were chosen to be culturally neutral. High level cognitive tasks including the Token Test, Fluency, RCPM and Block Design are possibly unaffected by socio-economic background and educational opportunity (Orgass and Poek 1966, Lezak 1976, Maly et al 1977).

Some tests do correlate negatively with age in the normal population. They include at

.001	Token Test Part F
	Token Test Total
	Repeating Sentences
	Constructional Apraxia
	Block Design
	RCPM
.01	Spelling

.05	Token Test A - E
	Repeating Digits (Forward)
	Repeating Digits (Reverse)
	Confrontation Naming
	Fluency
	Oral Apraxia

5.2.2 Measures of Severity of Dementia

The 66 dements in this study were assigned to four groups determined by the distribution of scores on the Rating Scale. Scores fall into four categories and are described in Table 5.3 below.

Table 5.3

DISTRIBUTION OF RATING SCALE SCORES TO SEVERITY CATEGORIES

	Range	No of Cases
Normal	40 - 38	2
Mild	37 - 25	22
Moderate	24 - 15	34
Severe	14 - 0	8

5.2.3 Profiles

24 Profiles are presented as Appendix I to indicate the variabilities of patterns of language performance in differing diseases on a standardised format. Where possible scans imageing lesion sites and examples of deficits are provided. A commentary describes the language pathology:

Right Hemisphere Lesion	Case 1
Aphasics	
Conduction Aphasia	Case 2
Transcortical Motor Aphasia	Case 3
Dysfluent Aphasia	Case 4
Fluent Aphasia	Case 5
Transcortical Sensory Aphasia	Case 6
Herpes Simples Encephalitis	Case 7
Huntington's Chorea	Case 8
Third Ventricle Hydrocephalis	Case 9
Prefrontal Leucotomy	Case 10
Pseudodementia	Case 11
Vertebro Basilar Disorder	Case 12
Multi Infarct Dementia	Cases 13 and 14
Pick's Disease	Cases 15 and 16
Alzheimer Type Dementia	Cases 17 to 24

Note in Appendix I:

References to specific tests in the commentaries are given in capitals. Scaled Scores are taken from the WAIS. The mean scaled score for a person of similar years is 10.

CHAPTER 5 (contd.)

RESULTS OF INVESTIGATION

5.3 A NEUROLINGUISTIC DESCRIPTION OF DEMENTIA

5.3.1 Distribution of Scores

Goodglass and Kaplan (1982) note that scores for language measures in aphasia are variable and statistical data tends to be skewed. Means and medians are not nearly identical and scores do not cluster around the middle of the distribution. It is because such data is not normaly distributed that non parametric statistics are appropriate in analysing pathological language on test batteries.

Table 5.4 gives the range, median, mean and SD of all aphasics, dements and controls used in this study. Visual inspection of the means and medians indicates that, like aphasia, the language scores of the demented are also skewed. To investigate this further a test of skewedness using the formula after Yule and Kendall (1950)

$$m_3 = \frac{\sum_{i-1}^{n} (x_i - \bar{x})^3}{n}$$

$$\sqrt{\beta_1} = \gamma_2 = \frac{m_3}{s_3}$$

where S = the standard deviation was completed on eight of the variables. These variables reflect comprehension, semantic retrieval, fluency, syntactic competence, reading comprehension, constructional apraxia and non verbal intelligence. The variables and skewedness are listed below.

SKEWEDNESS OF NEUROLINGUISTIC VARIABLES

Variables	Skewedness
Token Test Total	- 1.11
Confrontation Naming	- 2.32
Fluency	+ 1.14
Sentence Construction	- 0.45
Reading Comprehension	- 1.50
Constructional Apraxia	- 0.06
Block Design	+ 1.15
Reven's Coloured Progressive Matrices	+ 0.62

Skewedness diminishes as the degree approaches zero. The tests of "intelligence" are all positively skewed. "Language" measures are all negatively skewed. Those tests that demand greater problem solving and effort are positively skewed while negatively skewed variables are those which normals generally produce complete scores.

5.3.2 Description of Data: Aphasia, Dementia, Controls

Table 5.4 presents a description of the range, median, mean scores and standard deviations of 66 dements, 45 aphasics and 61 healthy controls tabled in Chapter 4 and used to standardise the test battery.

Table 5.4

		APHASIA N = 45				DEMENTIA N = 66				CONTROLS N = 61			
	Max	Range	Medn	Mean	S.D.	Range	Medn	Mean	S.D.	Range	Medn	Mean	S.D.
Age		25-85	63	63.7	11.5	45-90	70	69.1	10.7	45-90	67	66.5	12.4
Rating Scale	40					8-40	24	23.8	8.0	36-40	40	39.0	0.9
Iden. Name	8	1-8	8	7.5	1.2	5-8	8	7.7	.7	8	8	8.0	0.0
Iden. Funct.	8	2-8	8	7.4	1.6	3-8	8	7.6	1.0	8	8	8.0	0.0
Token T. A-E	67	0-67	45	41.7	19.3	0-67	62	59.8	8.5	62-67	67	66.6	.8
Token T. F	96	0-96	61	51.7	33.6	0-96	85	79.5	14.3	83-96	95	94.3	2.5
Token T. Total	163	0-163	106	93.4	51.7	0-163	147	139.3	22.1	145-163	162	160.9	3.1
Sent. Rep.	22	0-16	8	6.8	5.2	2-18	12	11.7	2.6	10.21	15	15.6	2.6
Digit. Rep.	14	0-9	3	3.7	2.2	2-11	6	5.8	1.7	3-14	7	8.0	2.7
Digit Rep. R	14	0-7	1	1.6	1.8	0-8	4	3.7	1.7	2-13	7	7.2	2.6
Auto Speech	4	0-4	4	3.3	1.3	3-4	4	3.9	0.2	4	4	4.0	0.0
Con. Naming	16	0-16	12	9.2	6.7	1-16	15	14.1	3.3	14-16	16	15.9	0.3
Des. Funct.	16	0-16	13	9.1	7.1	0-16	16	14.6	3.2	16	16	16.0	0.0
Tac. Nam. R	8	0-8	0	2.7	3.3	0-8	8	6.8	1.8	8	8	8.0	0.0
Tac. Nam. L	8	0-8	6	4.4	3.6	0-8	8	7.1	1.7	8	8	8.0	0.0
Gesture	16	8-16	16	15.3	1.8	12-16	16	15.7	.8	16	16	16.0	0.0
Fluency	60	0-32	2	5.8	8.3	0-40	10	13.8	10.7	5-60	35	36.9	12.1
Sent. Constr.	25	0-25	0	7.2	9.6	0-2	19	15.5	9.0	22-25	25	24.8	0.5
Read Words	8	0-8	8	5.6	3.3	5-8	8	7.9	0.4	8	8	8.80	0.0
Word Recog.	8	0-8	8	7.4	1.7	5-8	8	7.8	0.6	8	8	8.0	0.0
Read Sent.	7	0-7	3	3.4	3.3	2-7	7	6.5	1.1	7	7	7.0	0.0
Sent. Comp.	32	0-32	26	20.5	11.6	0-32	28	28.1	6.6	30-32	32	31.9	0.3
Auto. Writ.	5	0-5	3	3.1	1.9	1-7	5	4.5	1.3	5	5	5.0	0.0
Spelling	24	0-24	3	9.7	10.2	0-24	21	19.1	5.5	19-24	24	23.6	0.9
Dictation	13	0-13	4	5.8	5.8	0-13	11	10.4	3.3	9-13	13	12.9	0.6
Copying	11	0-11	11	8.1	4.7	1-11	10	9.5	2.0	9-11	11	10.8	0.5
Calculation	10	0-10	5	5.1	3.7	0-10	7	7.2	2.5	7-10	10	9.8	0.6
Oral Aprax.	20	7-20	18	17.1	3.5	8-20	20	19.1	2.1	18-20	20	19.8	1.3
Ideom. Aprax.	20	12-20	20	18.8	1.9	13-20	20	19.3	1.4	20	20	20.0	0.0
Const. Aprax.	20	0-20	19	16.7	5.2	1-20	16	14.4	4.9	17-20	20	19.5	.8
Block Design	48	0-35	18	17.3	10.2	0-45	8	9.9	10.4	10-45	28	29.9	8.9
RCPM	36	8-36	25	23.5	7.3	4-36	17	17.4	6.1	18-36	32	28.9	5.0

5.3.3 Discussion

On 31 subtests 61 healthy controls gave perfect scores on 12 subtests while another 10 subtests demonstrated as SD of less than 1. Scores for the remaining 9 tests compare favourably with normative data in those batteries from which they were derived. In no cases do the mean and median scores suggest the population is not normally distributed.

The severity and type of aphasia that standardised aphasia test batteries, and from which some of the tests on the language scales were derived, is not known. The Spreen and Benton (1977) data compares favourably with that of the Language Scales although it is apparent that right hemiplegics were excluded from their data where Tactile Naming (Right) is similar to Tactile Naming (Left) and writing scores are also resultantly higher. Moreover, this population had a fifteen point lower median score on the Token Test suggesting that it is more impaired than that of Spreen and Benton. The same appears true for Word Fluency and Sentence Construction. A study by Kertesz (1979) reports an investigation with the RCPM whose population appears to have more severely impaired than the one used in this study. There is a close approximation of the distribution of RCPM scores in this study and those of a recent study of the Raven IQ and recovery in non acute strokes (David and Stilbeck 1984).

There are high intercorrelations on tests that sample similar abilities: between the two tests of non verbal intelligence, $r_s = 0.85$ $p < 0.001$; tests that demand the ability to generate word strings (Word Fluency) and Sentence Construction,

$r_s = 0.77$ $p < 0.001$; Ideomotor Apraxia and the ability to Gesture, $r_s = 0.47$ $p < 0.001$; and the ability to comprehend verbal versus written instructions (Token Test and Sentence Comprehension), $r_s = 0.70$ $p < 0.001$. The Rating Scale was not given to aphasics who are at an obvious disadvantage on verbally mediated tests of orientation and memory.

The 66 dements produced a greater number of lower range scores than controls. Since no standardised data for language in dementia exists other than in this investigation comparisons to other studies cannot be made.

The scores are internally consistent over the tests that demand similar abilities: non verbal intelligence tests correlate at $r_s = 0.67$ $p < 0.001$; Word Fluency and Sentence Construction at $r_s = 0.64$ $p < 0.001$; tests of Ideomotor Apraxia and Gesture at $r_s = 0.39$ $p < 0.01$; and verbal and written comprehension (Token Test and Sentence Comprehension) at $r_s = 0.80$ $p < 0.001$.

5.3.4 Severity of Language Impairment in Dementia

Factors that may relate to the degree of language impairment in dementia are age, duration, rate, and severity of illness.

There was no relationship between test scores and age that was significant. Constantinidis (1978) observed that language pathology in presenile cases is "quicker in onset, more serious and less regular in its chronological evolution".

A difficulty of establishing duration of illness lies in the reliance of reported histories of patients by relatives (Blackburn and Tyrer 1985). Unfortunately it appears that too often

individuals are not reported to GPs until the disease is well marked (Williamson et al 1964).

Profiles of individual patients with a diagnosis of ATD suggests the disease progresses at individual rates.
Serial investigations need to relate language disturbance not just periodicy of testing but reflect severity as well.

The 31 variables that constitute the Language Scale may be divided into seven sections:

(1) Rating Scale (Orientation)

(2) Comprehension

(3) Auditory Memory

(4) Expressive Verbal Language

(5) Symbolic Language

(6) Praxis

(7) Non Verbal Intelligence

The correlations of severity of impairment, as measured by the Rating Scale, and the 30 language variables are given in Table 5.5.

Table 5.5

SEVERITY OF LANGUAGE DISABILITY IN DEMENTIA

(Correlation with Rating Scale)

N = 66

High (rho more than .40)		Low (rho less than .40)	
Token Test Total	.61	Copying	.28
Word Fluency	.61	Repeating Digits	.29
Calculation	.61	Identification of Names	.34
Tactile Naming (R)	.60	Coloured Matrices	.35
Sentence Comprehension	.60	Gesture	.36
Token Test Part F	.60	Ideomotor Apraxia	.37
Description of Function	.55	Word Recognition	.37
Confrontation Naming	.54		
Dictation	.54		
Token Test Part A-E	.52		
Digit Repetition Reverse	.52		
Sentence Construction	.49		
Sentence Repetition	.48		
Spelling	.46		
Constructional Apraxia	.45		
Automatic Writing	.44		
Tactile Naming (L)	.42		
Block Design	.41		
Identification Function	.41		
Automatic Speech	.40		

Reading Words, Reading Sentences and Oral Apraxia failed to reach significance.

Correlations: $r_s = .25$ $p < 0.05$, $r_s = .32$ $p < 0.01$, $r_s = .40$ $p < 0.001$

Comprehension is particularly impaired when measured by the Token Test, $r_s = 0.61$ $p < 0.001$, or by the understanding of written instructions, $r_s = 0.60$ $p < 0.001$. Token Test A - E, or the memory component in this task of comprehension, is highly correlated with severity of dementia $r_s = 0.52$ $p < 0.001$. Sentence Repetition, demanding similar abilities of comprehension of immediately remembered material, is also strongly correlated to severity $r_s = 0.48$ $p < 0.001$. However, the concepts in instructions are less understood (Token Test F, $r_s = 0.60$ $p < 0.001$).

Other abilities which appear impaired in dementia are Calculation, Word Fluency and Tactile Naming (Right). All suggest parietal lobe dysfunction. Failure to calculate is part of the triad of the Gertsman Syndrome that may follow lesions to the dominant angular gyrus. The role of the tertiary zone of the temporo-parieto-occipital area in naming has been discussed as has the ability to name objects through tactile stimilulation. In the right hand it demands cortical synergy of transference of information across the posterior fibres of the corpus callosum into the left temporo-parietal hemisphere for processing.

The failure of language in dementia is a feature of temporo parietal dysfunction. Neuropathological observations of the continued compromising of the temporo parietal cortex are well established in the literature. Benson et al (1982) has described language features of angular gyrus syndrome that may mimic ATD.

Individual case studies also suggest that in dementia comprehension is affected. Profile number 29 (see Chapter 5.2) demonstrates the effects of progressive dementia on language. Word Fluency, Calculation, Sentence Construction and the Token Test are variables noted in Table 5.5 where rho is greater than .60. They are precisely those abilities that fail in relation to the Dementia Rating Scale in Profile 29.

The hierarchy of scores in Table 5.5 delineates the variability of "intelligence measures". Word Fluency, or /w/ factor, correlates highly with severity at $r_s = .61$ $p < 0.001$. Block Design, that demands construction and assembly, is as strongly correlated at $r_s = .41$ $p < 0.001$. The Matrices that demand sub vocal reasoning, visual matching and visual closure, but no constructional assembly is weakest at $r_s = .35$ $p < 0.01$. Language measures of intelligence are more vulnerable than performance and non verbal measures.

The weakest correlations exist with Oral Apraxia (articulatory apraxia producing muteness is clinically observed only in severe dementia), Word Recognition and and Copying. These tasks appear linguistic but may not be, for words may be encoded in a grapheme store independent of comprehension.

In normals the correlation of the ability to read and act on instructions is 1. In dements a dissociation of function is noted: comprehension of written instructions correlates with severity at $r_s = 0.60$ $p < 0.001$ but the reading of those instructions fails to reach significance.

5.3.5. Interrelationship of Language Abilities in Dementia

Table 5.6 to Table 5.11 show patterns of intercorrelations within the remaining sections of the language scales. The Matrix consists of 82 correlation co-efficients so that 4 correlations at 5% level will be obtained by chance.

Observations of the correlation Matrices indicate for Comprehension all subtests but one correlate above $p < 0.01$. The intercorrelation of the Token Test is particularly high. Auditory memory is also intercorrelated significantly and greater than $p < 0.01$.

Expressive Verbal Language is intercorrelated at $p < 0.001$ between all Naming tasks, the ability to generate word strings as Word Fluency, and the ability to put words into acceptable grammatical sequences. A weaker correlation exists between speech which is unpropositional and the conscious coding of words into syntactic form; the correlation of Automatic Speech to Sentence Construction is $r_s = 0.3$ $p < 0.05$.

Table 5.6 C O M P R E H E N S I O N

	IN	IF	TT A-E	TT-F	TTT
IN	1	.68	.34	.39	.40
IF		1	.37	.32	.41
TT A-E			1	.81	.92
TT-F				1	.91
TTT					1

KEY:

IN	Identification by Name
IF	Identification by Function
TT A-E	Token Test Part A-E
TT-F	Token Test Part F
TTT	Token Test Total

Table 5.7

AUDITORY MEMORY

	SR	DR	DRR
SR	1	.59	.44
DR		1	.38
DRR			1

KEY:
SR Sentence Repetition
DR Digit Repetition
DRR Digit Repetition (Reverse)

Table 5.8

EXPRESSIVE VERBAL LANGUAGE

	AS	CN	DF	TNR	TNL	WF	SC
AS	1	.38	.51	.23	.32	.36	.30
CN		1	.70	.63	.66	.49	.41
DF			1	.58	.53	.51	.46
TNR				1	.68	.50	.51
TNL					1	.49	.44
WF						1	.64
SC							1

KEY:
AS Automatic Speech
CN Confrontation Naming
DF Description of Function
TNR Tactile Naming (Right)
TNL Tactile Naming (Left)
WF Word Fluency
SC Sentence Construction

Table 5.9

READING WRITING AND CALCULATION

	RW	WR	RS	SCom	AW	S	D	C	Cal
RW	1	.46	.28	.31	-.09	.16	.27	-.07	.23
WR		1	.10	.45	.44	.27	.43	.21	.33
RS			1	.40	.20	.26	.40	.28	.18
S Com				1	.48	.66	.63	.47	.60
AW					1	.50	.54	.42	.62
S						1	.74	.45	.56
D							1	.64	.58
C								1	.47
Cal									1

KEY:		
	RW	Reading Words
	WR	Word Recognition
	RS	Reading Sentences
	S Com	Sentence Comprehension
	AW	Automatic Writing
	S	Spelling
	D	Dictation
	C	Copying
	Cal	Calculation

Table 5.10

APRAXIA

	G	OA	IA	CA
G	1	.25	.39	.18
OA		1	.51	.23
IA			1	.29
CA				1

KEY:
G Gesture
OA Oral Apraxia
IA Ideomotor Apraxia
CA Constructional Apraxia

Table 5.11

NON VERBAL INTELLIGENCE

	Cal	CA	BD	RCPM
Cal	1	.61	.64	.58
CA		1	.81	.69
BD			1	.67
RCPM				1

KEY:
Cal Calculation
CA Constructional Apraxia
BD Block Design
RCPM Reven's Coloured Progressive Matrices

For Tables 5.6 to 5.11

$r_s = 0.25 \quad p < 0.05$

$r_s = 0.32 \quad p < 0.01$

$r_s = 0.41 \quad p < 0.001$

Reading, Writing and Calculation may be regarded as "symbolic language" since graphemes and numerals encode linguistic forms as symbols. Although there is a higher correlation between Reading Words and Word Recognition $r_s = 0.46$ $p < 0.001$, and between Reading Sentences and Sentence Comprehension $r_s = 0,40$ $p < 0.01$, there is no correlation between the ability to read a single word and the ability to read a sentence. A high correlation exists between the ability to write to Dictation and Sentence Comprehension or understanding of written instructions $r_s = 0.63$ $p < 0.001$, Spelling $r_s = 0.74$ $p < 0.001$ and Copying $r_s = 0.64$ $p < 0.001$. The ability to calculate correlates significantly with other graphic tasks but not significantly to reading ability.

The apraxia section includes gesture as an index of ideation. Gesture correlates most significantly with Ideomotor Apraxia $r_s = 0.39$ $p < 0.01$, weakly with Oral Apraxia but not with Constructional Apraxia. Indeed the correlation of Constructional Apraxia as measured by drawing ability is only $r_s = 0.29$ $p < 0.05$ for Ideomotor Apraxia and without a significant correlation with Oral Apraxia. Oral Apraxia and Ideomotor Apraxia are however significantly correlated at $r_s = 0.51$ $p < 0.001$.

The Non Verbal Intelligence section has included Constructional Apraxia and Calculation because these tasks demand visuo spatial synthesis and abstract planning. The intercorrelation between Calculation, Constructional Apraxia, Block Design and the Progressive Coloured Matrices are all at $p < 0.001$ significance.

5.3.6 Conclusions

The medians, means and SD's of the populations used to standardise the Language Scale indicates that scores for controls are normally distributed. Scores are skewed for both aphasic and demented groups.

The difference of distribution of scores over the three whole groups of dements, dysphasics and controls indicates the Language Scales' face and content value. The intercorrelations between subtests demonstrate the battery's internal consistency and compatability of scores.

Correlations of the six sections of the Language Scales to severity in dementia as measured by the Rating Scale indicate verbal and written comprehension, Fluency, Calculation and right handed asteriognosis are more impaired than measures of auditory memory and non verbal intelligence.

CHAPTER 5 (contd.)

RESULTS OF INVESTIGATION

5.4 "APHASIA" AS A FEATURE OF DEMENTIA

5.4.1 Introduction

Although many authors have described language pathology in dementia in terms of classical aphasic syndromes the labels may be inappropriate.

There are good reasons for arguing that language pathology in focal and diffuse damage may be different. First, the aetology of the diseases that cause them differs. The aphasias are generally a result of acute trauma or of vascular disease, and while language disturbance may relate to vascular disease in MID, it is often multifocal and insidious.

Second, language pathology in focal and diffuse disease may differ because the factors that involve language may be differently affected by the very nature of the disease. Aphasia may be more "purely" a disturbance of language, while in dementia language may be disturbed because of an underlying and broader "cognitive" impairment. This is the basis of the argument that anomia in aphasia is a disturbance of access to lexical store and in dementia it is a disturbance of visual perception.

Third, the natural history differs. Aphasics generally improve, or performance plateaus as the neurological condition becomes stable.

5.4.2 The Natural History of Aphasia

Table 5.12 represents a test re-test design for 10 aphasics all reassessed after not less than 3 months and not more than 2 years. The Table describes the range, median, mean and SD's of each condition. The Wilcoxon Matched Pairs Signed Ranks Test was used to test the difference between these related groups.

The trend of the means and medians indicates general improvement. However, because of a small sample producing too many ties in 13 calculations measures could not be used to compute significance. Nine measures were not significantly small enough to reject the Null hupothesis.

Comprehension, as measured by the Token Test, Confrontation Naming, Sentence Construction and Calculation all show a significant difference at $p < 0.10$. Memory scores by Sentence and Digit Repetition (Forward) as well as Word Fluency show a significant difference at $p < 0.05$. Thus the significant improvement in aphasics relates to Comprehension as well as the improvement of semantic and syntactic encoding, memory, abstract ability in calculation and fluency. Indeed the greatest improvement appears to be of instructions that demand understanding of length of utterance as Token Test A-E at $p < 0.02$.

Table 5.12

TEST RETEST DESIGN FOR APHASICS

N = 10

AGE		Mean Age 59.9				Mean Age 61.1				Wilcoxon		
TEST	Max	Range	Medn	Mean	SD	Range	Medn	Mean	SD	N	T	Sig
Iden. Name	8	8	8.0	8.0	0	8	8.0	8.0	0	0		
Iden. Funct.	8	8	8.0	8.0	0	8	8.0	8.0	0	0		
Token T. A-E	67	0 - 60	51.0	43.3	20.5	15 - 65	54.0	51.9	15.6	9	3.0	.02
Token T. F	96	0 - 90	63.5	57.6	30.4	0 - 90	78.0	63.1	34.8	10	13.5	NS
Token T. Total	163	0 - 160	112.5	100.9	50.1	20 - 160	129.5	115.0	48.2	10	10.0	.10
Sent. Rep.	22	0 - 16	5.5	5.5	5.7	2 - 16	9.5	8.5	5.4	9	5.5	.05
Digit Rep.	14	1 - 5	3.5	3.3	1.7	0 - 8	3.0	3.4	2.5	9	4.5	.05
Digit Rep. R	14	0 - 3	0	0.9	1.2	0 - 7	1.5	2.2	2.7	3		
Auto Speech	4	0 - 4	4.0	3.1	1.7	2 - 4	4.0	3.4	0.9	4		
Con. Naming	16	0 - 16	13.5	9.9	7.1	4 - 16	15.5	13.4	4.1	5	0	.10
Des. Funct	16	0 - 16	13.5	10.3	6.6	0 - 16	14.0	12.5	5.1	5	2.0	NS
Tac. Nam. R	8	0 - 8	1.0	3.3	3.9	0 - 8	4.0	4.0	4.2	2		
Tac. Nam. L	8	0 - 8	8.0	5.5	3.5	0 - 8	8.0	6.7	2.8	2		
Gesture	16	1 - 16	16.0	15.8	0.6	15 - 16	16.0	15.9	0.3	2		
Fluency	60	0 - 22	1.0	3.0	4.5	0 - 24	3.5	6.8	8.2	7	1.5	.05
Sent. Constr.	25	1 - 8	8.0	7.4	8.5	0 - 24	13.0	11.0	10.1	7	3.0	.10
Read Words	8	1 - 8	8.0	6.0	3.3	1 - 8	8.0	6.8	2.3	3		
Word Recog.	8	8	8.0	8.0	0	7 - 8	8.0	7.9	0.3	1		
Read Sent.	7	0 - 7	5.5	4.2	3.2	0 - 7	4.5	3.9	3.2	4		
Sent. Comp.	32	8 - 32	30.0	25.9	9.2	0 - 30	30.0	25.8	9.9	8	8.0	NS
Auto. Writ.	5	2 - 4	3.5	3.1	2.0	0 - 5	5.0	4.0	1.8	4		
Spelling	24	3 - 24	19.0	12.7	10.4	0 - 24	17.0	14.2	9.9	7	7.0	NS
Dictation	13	0 - 13	10.5	7.0	6.0	0 - 16	8.0	7.1	6.9	4		
Copying	11	0 - 11	11.0	8.4	4.5	9 - 11	11.0	10.5	0.9	6	4.5	NS
Calculation	10	0 - 10	6.0	5.6	3.0	2 - 10	8.0	7.0	2.8	6	1.5	.10
Oral Aprax.	20	7 - 20	18.0	17.0	4.1	8 - 20	19.0	17.3	3.9	5	5.0	NS
Ideom. Aprax.	20	13 - 20	20.0	19.1	2.2	12 - 20	20.0	19.6	0.8	3		
Const. Apraxia	20	17 - 20	20.0	19.0	1.4	18 - 20	20.0	19.7	0.7	5	2.0	NS
Block Design	48	5 - 35	24.0	23.2	8.9	8 - 36	26.0	24.3	8.7	9	12.0	NS
RCPM	36	17 - 34	26.0	26.0	5.1	22 - 36	27.0	28.7	5.1	8	11.0	NS

5.4.3 Factor Analysis: Dementia

Factor analysis matches groupings of variables that are common to two populations. The method is appropriate for comparison of demented and aphasic groups since the number of cases in both groups is greater than the number of variables used.

A series of factor analyses using varimax rotation (BMDP M 1977) was used to investigate the variables of the Language Scales. Table 5.13 below presents a factor analysis based on the measures with the exception of the Dementia Rating Scale, since this test was not given to the aphasic group and the Token Test Total which is a summation of Token Test Parts A-E and Token Test Part F. Seven factors are isolated in the analysis of the dementing population. The percentage of variance explained by each factor is given at the foot of each column.

5.4.4 Discussion

The first factor clearly relates to non verbal visuo-constructional and organisational abilities that require concentration and planning. Constructional Apraxia, Block Design and the Progressive Matrices have loadings from .70 to .85. The Fluency factor (.63) is represented less strongly although it demands concentration for controlled word association. Calculation (.56) also demands the abstract manipulation of symbols while Repeating Digits (Reverse) (.57) requires manipulation of numbers with a concentration constraint. Similarly Part F of the Token

Table 5.13

FACTOR ANALYSIS OF LANGUAGE SCALE VARIABLES,

EXCLUDING RATING SCALE MEASURES

DEMENTIA

Factor 1		Factor 2		Factor 3	
Block Design	.85	Calculation	.51	Word Recog.	.85
RCPM	.82	Sent. Rep.	.78	Iden. Funct.	.84
Constr. Apraxia	.79	Copying	.73	Iden. Name	.75
Fluency	.63	Dictation	.70	Sent. Comp.	.54
Digit Rep. (R)	.57	Spelling	.69	Token Test A-E	.51
Calculation	.56	Digit Rep.	.67	Con. Naming	.43
Token T F	.54	Auto. Speech	.64		
Sent. Comp.	.45	Auto Writ.	.54		
Token Test A-E	.43				
Sent Constr.	.45				
Gesture	.42				
% Variance	21.5		20.1		16.6

Factor 4		Factor 5		Factor 6	
Read Words	.77	Token Test F	.43	Sent Comp.	.43
Tactile Nam. R	.74	Token Test A-E	.41	Read Words	.51
Tactile Nam. L	.73	Oral Apraxia	.82	Read Sent.	.80
Con. Naming	.70	Des. Funct.	.67		
		Sent. Constr.	.61		
% Variance	13.8		12.6		8.5

Factor 7	
Auto. Speech	.40
Ideom Apraxia	.80
Gesture	.40
% Variance	7.0

Test (.54) also requires operations on spatial, temporal and logico-grammatical concepts or "quasi spatial" analysis. Weakly represented is the ability to combine words into grammatical form as Sentence Construction (.45). These non verbal language mediated tasks tend to represent dominant parietal function: the verbal mediation of spatial relationships.

The second factor is strongly determined by writing tasks, Copying sentences (.73) writing to Dictation (.70), writing the names of stimulus objects as Spelling (.69) and Automatic Writing of name and address (.54). Less strongly represented is Calculation. It has, in this test, a writing component. Sentence Repetition is strongly represented at .78 and may be compared to writing to dictation but without a graphic component. Repetition of Digits (.67) is not dissimilar to Sentence Repetition but that the stimulus is not bound by grammatical context. Two automaticisms are also less strongly represented, Automatic Speech (.64) and Automatic Writing (.54).

The third factor appears to reflect recognition through identification of Objects by their Name (.75), Function (.84) and Word Recognition of a written name (.85). Less strongly represented are comprehension beyond single word level as Token Test Part A-E, which demands comprehension by length of instruction. Written commands as in Reading Comprehension represented similarly at .54. Word Recognition is represented strongly at .85 but Confrontation Naming is represented weakly at .43.

The fourth factor is a verbal naming factor. All tasks demanding this ability are represented strongly here as Reading Words and Confrontation Naming. Tactile Naming follows where objects are presented to the right (.74) and left (.73) hand with no visual input.

The fifth factor reflects auditory comprehension. All parts of the Token Test are represented as are Sentence Construction (.61). The factor here perhaps represents the ability to understand the instruction rather than complete the task. Oral Apraxia (.82) is strongly represented but may reflect comprehension of task rather than oral agility.

The six factor is exclusively a reading factor; Reading Words (.51) and Reading Sentences (.80). Sentence Comprehension appears less strongly at .43 and Word Recognition weakly, but not listed in the Table at .30.

The seventh factor reflects motor gestural ability, be those gestures intransitive, as Ideomotor tasks (.80), or the Gesture of the use of objects ideationally (.40). Ideational gesture is resistant to dementing processes and occurs weakly with this factor together with overlearned abilities such as automaticisms at .40.

5.4.5 Factor Analysis: Aphasia

Table 5.14 presents the results of the factor analysis based on all verbal and non verbal measures of the aphasic group. Six

Table 5.14

FACTOR ANALYSIS OF LANGUAGE SCALE VARIABLES,
EXCLUDING RATING SCALE MEASURES

APHASIA

Factor 1		Factor 2		Factor 3	
Con. Naming	.88	Dictation	.49	Calculation	.49
Tactile Nam. L	.85	Digit Rep.	.84	Block Design	.88
Des. Funct.	.81	Fluency	.80	RCPM	.86
Read Words	.76	Digit Rep. R	.76	Constr. Apraxia	.85
Auto Speech	.73	Sent. Constr.	.75	Ideom. Apraxia	.56
Read Sent.	.60	Sent. Rep.	.66	Copying	.56
Dictation	.50	Tactile Nam. R	.60	Auto. Writ.	.53
Sent. Constr.	.42	Calculation	.51	Oral Apraxia	.48
Sent. Rep.	.56	Auto Writ.	.50		
Tactile Nam. R	.54	Spelling	.45		
Spelling	.48	Oral Apraxia	.47		
% Variance	24.5		22.9		18.4

Factor 4		Factor 5		Factor 6	
Token Test A-E	.84	Iden. Funct.	.83	Read Sent.	.48
Token Test F	.81	Iden. Name	.77	Word Recog.	.81
Sent. Comp.	.47	Gesture	.73	Sent. Comp.	.57
% Variance	14.2		10.9		8.9

factors are isolated in the analysis of the aphasic population and the percentage of variance explained by each factor is given at the foot of each column

5.4.6. Discussion

Factors one and two reflect oral language performance with factor one strongly loading semantic or naming ability as Confrontation Naming at .88 and Tactile Naming (Left) at .85. Tactile Naming (Right) is less strongly represented in both factor one and factor two. Factor one also includes oral expressive ability as verbal naming and Description of Function (.81), automatic sequences (.73), Reading Words (.76) and Reading Sentences aloud (.60). Less strongly represented are Sentence Repetition (.56), Sentence Construction (.42) and the writing of names of objects as Spelling (.48), or writing to Dictation (.50). These last two variables are also represented in factor two, Spelling at .45 and Dictation at .49.

Factor two reflects fluency as the ability to formulate word strings. Word Fluency is loaded at .80 and strings of numbers presented as Repeating Digits at .84. The factor also includes a test where strings of words are presented in a set order as Sentence Construction at .75. Oral Apraxia (.47) Automatic Writing (.50) and Calculation (.51) receive near equal loading between the second and third factor.

The third factor represents measures of abstract operations. It includes tests of non verbal intelligence, Block Design (.88) and Progressive Matrices (.86). Constructional Apraxia is included

(.85) being the ability to organise, plan and draw lines and dimensions in space. This "conceptual" aspect is supported by Calculation, less strongly at .49. Three tests of apraxia are also represented: Constructional Apraxia (.85), Oral Apraxia (.48) and Ideomotor Apraxia (.56). They figure here as tasks not of comprehension but rather as tasks of execution, as do two tasks of writing that demand less effortful operations, as Copying (.56) and Automatic Writing (.53).

The fourth factor is exclusively loaded with auditory comprehension of instructions by length, Token Test Part A-E (.84) and by complexity Token Test Part F (.81). It also includes, less strongly loaded, a task of Sentence Comprehension for written instructions (.47).

The fifth factor is concerned with recognition or agnosia. Identification by Name (.77), Identification by Function (.83), and the ability to Gesture to indicate the use of objects (.73) are included.

The sixth factor represents reading ability as Word Recognition (.81), Reading Sentences (.48) and Sentence Comprehension of written material (.57).

5.4.7. Conclusions

Summarising, the factors from the analysis of dements are:

Factor	1	Visuo-Construction
"	2	Writing

Factor	3	Recognition
"	4	Naming
"	5	Comprehension
"	6	Reading
"	7	Motor-Gesture

and for Aphasics:

Factor	1	Naming
"	2	Fluency
"	3	Visuo-Construction
"	4	Comprehension
"	5	Recognition
"	6	Reading

The dementing and aphasic populations demonstrate patterns that are unequal and unaligned. Generally in factor analysis the first factor strongly represents task difficulty and task complexity (Hodkinson et al 1984) or the degree of severity of the disorder (Goodglass and Kaplan 1982). In the dysphasic population lexical word search and oral abilities account for 24.5% of the variance. The demented populations first factor is clearly related to spatial orientation and the ability to solve problems and represents 21.5% of the variance.

5.4.8. Factorial Ranking of Analyses

Factor analysis allows the examination of underlying patterns or relationships in bodies of data, clustering it into groups according

to intercorrelations (Nie et al 1975). If the variables that describe the language of aphasic and dements were similar, one would expect strong similarities by rank factor loadings, and the interpretation could be made that dements were truely "aphasic".

Table 5.15 represents the factor loadings of dements and aphasics aligned to the thirty variables used in the Language Scales. The first column represents the dements the second the aphasics. It will be observed that the pattern of test performance for the two groups is not similarly ranked suggesting that "aphasia" is not an aspect of the dementing syndrome. Whereas aphasia appears to be a disturbance of linguistic components of semantics, fluency and grammar, the language of the demented appears to be a disruption of cognitive ability.

5.4.9. Conclusions

The notion that "aphasia" is a term applicable to the language of the demented is unsupported. Not only is the natural history of aphasia and demented language different but neurolinguistic variables that are used to measure the two conditions demonstrate differences of clustering, distribution and rank.

Table 5.15

SUBTESTS OF BATTERY WITH FACTORIAL RANKS BY TEST ORDER; THE FIRST NUMBER FOR DEMENTS AND THE SECOND FOR APHASICS

Subtests	Dements	Aphasics
Identification by Name	18	25
Identification by Function	17	24
Token Test Part A-E	20	21
Token Test Part F	7	23
Token Test Total	8	22
Sentence Repetition	9	12
Repeating Digits	13	8
Repeating Digits Reverse	5	10
Automatic Speech	14	5
Connfrontation Naming	24	1
Description of Function	26	3
Tactile Naming (right)	22	13
Tactile Naming (left)	23	2
Gesture	30	6
Fluency	4	9
Sentence Construction	27	11
Reading Words	21	4
Word Recognition	16	27
Reading Sentences	28	6
Sentence Comprehension	19	28
Automatic Writing	15	20
Spelling	12	29
Dictation	11	7
Copying	10	19
Calculation	6	14
Oral Apraxia	25	30
Ideomotor Apraxia	29	8
Constructional Apraxia	3	17
Block Design	1	15
Coloured Progressive Matrices	2	16

CHAPTER 5 (contd.)

RESULTS OF INVESTIGATION

5.5 THE QUALITY OF LANGUAGE IN ALZHEIMER TYPE DEMENTIA OF PRESENILE ONSET

5.5.1 Language and Clinical Staging

The progressive clinical features of ATD have been described in three stages by Sjorgren et al (1952), and more recently by Pearce and Miller (1973), Hughes et al (1982) and Reisberg (1983). Pearce and Miller (1973) have described these stages:

Stage 1 is characterised by an amnesic syndrome in 100% of cases, reduced spontaneity in 71% of cases and spatial disorientation in 59% of cases. The stage lasts two to four years and then merges into the second stage.

Stage 2 characterised by progressive dementia in 100% of cases and a failure of higher mental functions. It is accompanied by focal features including receptive dysphasia in 80% of cases, amnestic dysphasia in 82% of cases, dyspraxia in 66% of cases, perseveration in 66% of cases, dysgraphia in 52% of cases and dyslexia in 46% of cases.

Stage 3 demonstrates advanced dementia with a profound memory disturbance in 100% of cases.

The progression is accompanied by increasing extra pyramidal signs, gait disturbance, epilepsy and incontinence. Although ATD is a progressive disorder longitudinal studies have not been reported (Bayles 1984). There are cross sectional reports of language impairment in ATD (Irigaray 1973, Whitacker 1976, Bayles 1982, 1984, Ajuriaguerra and Tissot 1985).

Bayles (1984) has summarised these studies into "Early Stage Dementia" with forgetfulness, disorientation in time, memory deficit, language avoidance, language disinhibitions but preserved syntax and phonology. "Middle Stage Dementia" demonstrates disorientation in time and place, short term memory deficit, empty or irrelevant discourse with substantiative nouns being replaced by vague terms. Phonology is retained, there are errors of morphology but not syntax. Semantic but not phonemic paraphasias are also present. There is a loss of pragmatics, while verbal perseveration and ideational repetition is common. Language becomes egocentric. In "Late Stage Dementia" the patient is completely disorientated, wandering, echolalic, palilalic or mute with both syntax and phonology impaired. These stages are not dissimilar to the qualitative information presented in this study.

The particular difficulty of classifications of "Early", "Middle" and "Late" is that the terms are not well defined. In this study

they have particular reference to groupings of the distribution of scores on the Dementia Rating Scale and are labelled as "Mild", "Moderate" and "Severe". The score ranges include:

40	-	38	Normal
37	-	25	Mild
24	-	15	Moderate
14	-	0	Severe

Qualititive analysis of language behaviour investigates errors of lexical and grammatical analysis. The third component of linguistic study, phonology, was not assessed in this study. Lexical errors include anomia (the inability to retrieve names), nominal paraphasia (the substitution of one noun for another from the same semantic field), verbal paraphasia (the substitution of verb for noun forms), circumlocutory and perseverative responses.

Language breakdown can similarly be described as clinical signs in three stages. The division here has been determined by the distribution of scores on the Rating Scale.

Markers for Mild dementia include:-

Disorientation in time and place

Stable personal history, but impaired non personal one

Reduced auditory memory

Comprehension limited to five pieces of information and vulnerable for operations of increasing length with temporal spatial or logico grammatical concepts

Occasional verbal paraphasia

Dyscalculia for complex arithmetical problems

Ideational apraxia for sequential tasks

Reduced ability to solve problems that demand subvocal reasoning or visuo motor constructive ability

Markers for Moderate dementia include:-

Increasing disorientation in time and place

Reduced auditory and recent memory

Impaired concentration

Comprehension limited to three pieces of information and progressive inability to process basic prepositional concepts

Semantic paraphasias and circumlocutory responses particularly with a disturbance of the "abstract attitude" in language represented by retrieval of verb forms

Right tactile asteriognosis

Ideational disturbance at gesture level

Retained oral fluency with diminished word and ideational fluency

Reading ability dissociated from meaning

Dysgraphia with othographic and calligraphic errors

Constructional apraxia

Dyscalculia

Odeomotor (limb) apraxia

Reduced ability to solve verbal and constructional tasks

Markers for Severe dementia include:-

Disorientation in person, place and time

Inattention

Impaired long term memory

Impaired verbal and graphic automaticisms

Anomia, paraphasia and circumlocution

Bilateral asteriognosis

Paragrammaticisms, agrammaticisms, palalalia, mutism

Single word recognition with alienation of word meaning

Acalculia

Agraphia

Ideational, ideomotor, constructional apraxia

Agnosia

5.5.2 Language Errors in Alzheimer Type Dementia

Specific errors from the Language Scales are referred to in individual case reports in Chapter 5.2 Certain comment needs to be made on the type of error produced in the six sections of the Language Scales.

The errors and behaviours described below are taken from the patients with moderate dementia. It contains the clearest examples of language errors.

5.5.2.1 Remote Memory

Remote memories are the most durable in ATD, not because of "Ribot's Law" of a direct relationship of memory stability to time, but perhaps because long term information is better rehearsed. It would appear that those events which were most relevant in particular stages of patients' lives often intruded into later events, much like the nature of intrusion errors described by Fuld et al (1982). Alzheimer patients may misname the monarch as "Mary", or the Prime Minister as Baldwin, Churchill or Lloyd George. Such responses, although clear errors are paraphasic - the category is correctly named but the selection within it is at error.

5.5.2.2 Perception

Verbal agnosia, or the alienation of word meaning, a failure to recognise single words presented auditorily, is rare in ATD. It produces errors reflecting confusion of semantic categories. Two patients, when asked to point to a whistle, pointed to a bell, there was no visual agnosia, neglect or field defect.

5.5.2.3 Comprehension

Comprehension is frequently impaired in moderate dementia. Differentiated scores in the Token Test between failure of the understanding of instructions because of their length, and because of their complexity, suggests that is is not only a failure of auditory memory but also the inability to process the spatial, temporal and logico grammatical concepts that language contains. This disturbance of quasi spatial relationships has been noted in parietal symptomatology by Luria (1966).

5.5.2.4 Auditory Memory

There are three measures of auditory memory, Sentence Repetition, Digit Repetition (Forward) and Digit Repetition (Reverse) and ATD patients find repeating digits in reverse order the most difficult. This gap between reverse operations and repeating digits forward indicates a disturbance of concentration (Lezak 1976). Sentence Repetition is less impaired in dementia because sentences are aided by grammatical context. Whitacker (1976) in a study of repetition

ability in a severely demented patient noted the ability of the patient to correct syntactic errors when repeating in spite of a total loss of propositional language and suggested that a grammatical filter may operate on repeated language when cognition is lost.

5.5.2.5 Semantics

In ATD the temporal convolutions appear selectively impaired. Naming responses in ATD tend not to include phonemic paraphasia and patients with ATD do not produce fluent neologistic jargon that are common in focal lesions of Wernicke's area. The nominal and verbal paraphasias which occur in late dementia suggest that the disease progresses posteriorally to Wernicke's area. Examples of such paraphasias for moderate and severe dementia are given below:-

MODERATE DEMENTIA : NAMING ERRORS

Target	Nominal Paraphasia	Verbal Paraphasia	Circumlocution
Razor	razor blade	shaver	to cut hair for doing your face for shaving
Tweezers	pincers nippers pliers	pluckers clippers	cut eyebrows to clear splinters
Bell	gong		to call with it ring put it to work
Gun			shoot kids play with it
Sponge			for washing yourself thing for washing
Torch	lamp lantern	lighter	flash thing

MODERATE DEMENTIA : NAMING ERRORS (contd.)

Target	Nominal Paraphasia	Verbal Paraphasia	Circumlocution
Pen	propelling pencil		for writing an old type of nib
Matches	cigarettes		smoke them
Cup	jug		tea or coffee
Whistle	rattle		
Paintbrush			for teeth

SEVERE DEMENTIA : NAMING ERRORS

Target	Nominal Paraphasia	Verbal Paraphasia	Circumlocution
Torch	battery		Flashing
Razor		shaver	the man shave for your face
Pipe			Put things in it To stuff it in and puff
Pen	pencil pointed cane		
Sponge			Soft
Watch	clock		
Scissors			cutting you have long nails
Cup			Eating Drinking Eat from it
Whistle	bell		Put in mouth
Bell	whistle		To make a noice
Brush			For painting
Glasses			Speak To look at For eyes
Fork			Cooking

It should be noted that few of the naming errors suggested misperception of the object.

Unlike aphasia, recall of verbs is as difficult as the recall of nouns. This might be expected in diffuse damage (Schwartz et al 1979). There is also a marginal difference between left and right tactile naming. Generally there is greater difficulty of recalling the names of objects presented to the right hand. This may reflect the neurological complexity of shifting information from the non dominant to dominant hemisphere for recalling names or asymmetry of hemispheric involvement.

5.5.2.6. Fluency

Word fluency as Thurstone's /w/ factor is sensitive to brain damage (Borkowski et al. 1967). The test is one of "divergent thinking" (Vernon 1971) and it has behavioural constraints. Performance on this test is reduced and responses are marked by impoverished generation of words but not violation of test constraints. The two patients with Pick's disease both produced obscenities for the F.A.S. stimuli and both broke the constraints by perseveration and presentation of proper nouns.

5.5.2.7. Syntax

Observation of syntax in ATD, as tested by the Masselon Test, demonstrate difficulty of holding the stimulus words in memory in order to construct the sentences rather than failure to order

them grammatically. In support of this a correlation between the ability to repeat unrelated digits and the ability to generate sentences for apparently unrelated words is $r_s = .43$ $p < .05$. It is the highest correlation of any language variable to the sentence construction task.

5.5.2.8 Reading

Five types of dyslexia are described in the literature. Deep dyslexia refers to errors of semantic name reading (boy = girl), derivational errors (art = artist), visual errors (shape = sharp), visual semantic errors (fragment = fracture), visual then semantic errors (sympathy = orchestra) and function word errors (off = of). A factor that may influence such errors is concreteness of the concept (nouns are better read than verbs). Phonological dyslexia is the misreading of graphemes. Surface dyslexia is produced where similar letters may produce confusion of sounds as (pog = dog) or (bog = dog) where apparent substitutions still produce a meaningful word. A fourth dyslexia is spelling the letter phenomenon rather than reading the word. Finally there is a neglect dyslexia (through = thorough).

The patients in this study were asked to read high imageability words. There was little evidence of surface dyslexia, letter by letter reading (apart from the one subject who presented language pathology as the initial feature of ATD), or neglect dyslexia, despite apparent neglect in copying geometric shapes. There was no real evidence of semantic error reading, or deep dyslexia, but phonological errors were made for example:

fork	flpk
scissors	kIsgs
matches	ʃgs
sponge	sppndjdi

Dements can read lexically being able to access whole words with recognition of visual word form. Warrington (1975) has reported dements making reading errors of low imageability words. Schwartz et al (1980) has noticed the functional dissociation of word reading and understanding that are noted in this investigation. They also noted greater dependence of dements on the rules of spelling and thus an increasing difficulty of reading irregular words and note this as a precursor to deterioration of the phonological store. The nature of progressive dyslexia in dementia awaits systematic investigation.

5.5.2.9 Writing

ATD patients become increasingly dysgraphic. Writing reflects verbal language deficit. Three agraphias may be distinguished. "Pure Agraphia" has been described following lesions of "Exner's centre" (Vignolo 1983). Dysgraphia may be present as "Aphasic Agraphia". This disturbance follows lesions to the angular gyrus and may present errors of semantics or othography. Agraphia may also be present as a "Spatial Agraphia" related to constructional deficits associated with bilateral parietal lesions and reflected in poor alignment of recognisable letters.

In ATD it is uncommon for patients to attempt to write of objects they cannot label. If the name is given they produce phonemic-graphemic errors in spite of their speech being free of

phonemic paraphasia. This suggests a difficulty of transcoding rather than phonemic retrieval. Morphological errors, and particularly rules of inflection, are often lost. Spatial agraphia is rare despite progressive constructional apraxia and visuo-spatial deficit.

Figure 4 is the writing of ATD with severe dementia. Orthographic errors are noted, omissions of double letters in "glasses" and "tweezers", "watch" and "whistle" are not recognisable, "pipe" is not inflected. Dictation is written more as an approximation of the way the sentence was heard rather than as aphasic agraphia. Copying is without error suggesting the difficulty is not spatial or calligraphic but rather phonemic-graphemic. There was evidence of a right neglect and rotational disorder.

Figures 5a, 5b, and 5c show the writing of three consecutive years in a single ATD patient. The first year 5a demonstrates the tendency of dements to "tag" names to stabilise instability in lexical retrieval so "bell" becomes a "ship bell", "torch" is inflected but otherwise the writing is without error. The second year 5b is without error, but for one intrusion error in the second dictated sentence on "new year". The third year 5c automatic writing was contaminated and the maiden name was given. The writing is less assured, "spectacles" is changed to "glasses" and "bent" substituted for "built".

The general calligraphy reflects the loss of assuredness but not to the extent that the writer's drawing is impaired which shows

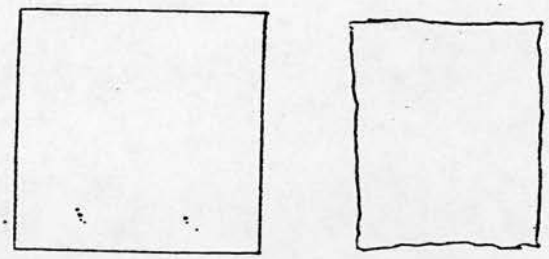
Figure 4

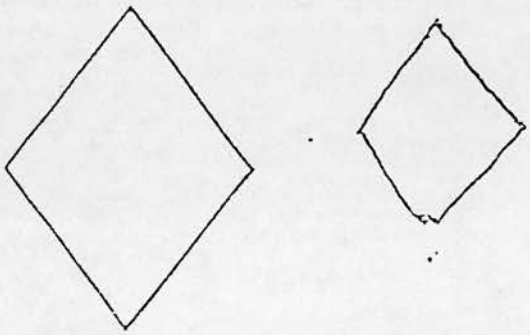
Bell.
R
Torch.
Razer
Wiser
Wathe
Glases.
Twesers.
Pib
This is a fernes day.
This Brick Bildungr. Sas Bild. last ree.
&.
This.

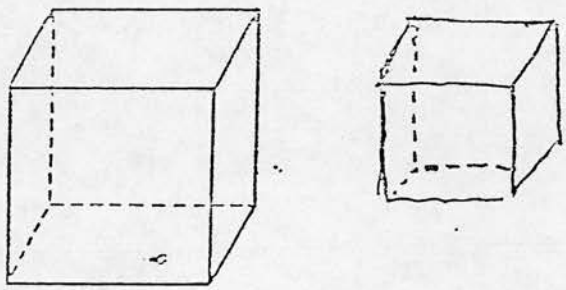
This is a.

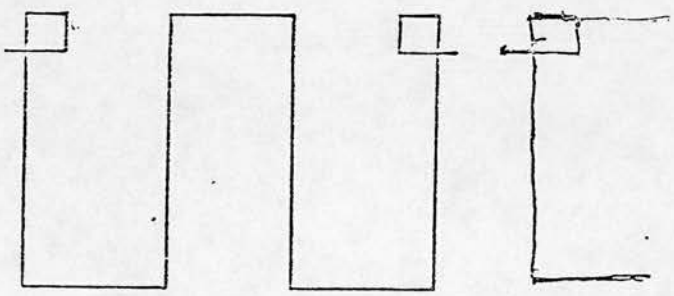
I am very hungry
The colour of the walls is green.

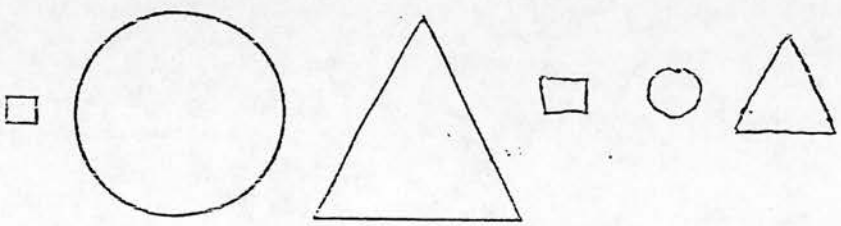
Figure 4 (cont.)

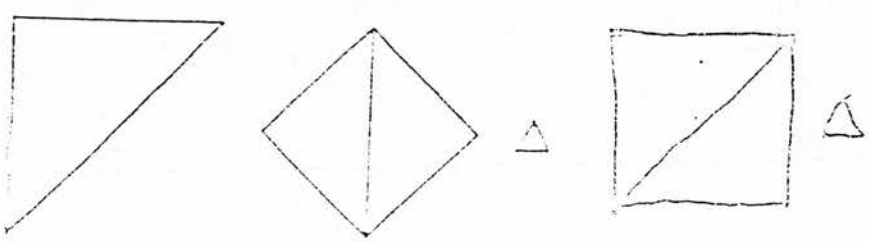












Ships Bell.
Torch.
Glasses.
Watch
Pipe
Tweezers
Razor.
Whistle

This is a very nice day
This Brick Building was
built last year.

I am very hungry
The Colour of the Walls is
Green.

Figure 5a (cont.)

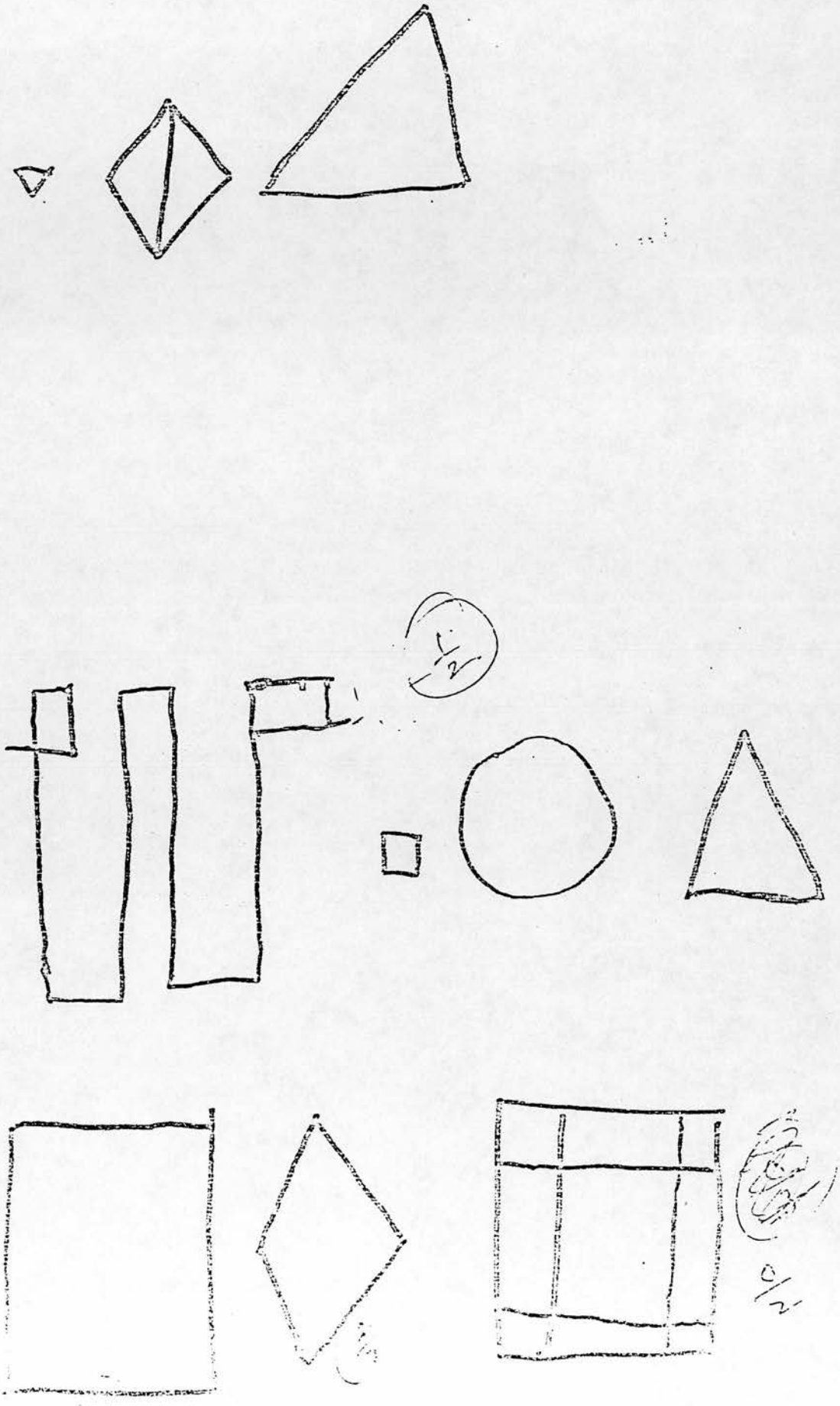


Figure 5b

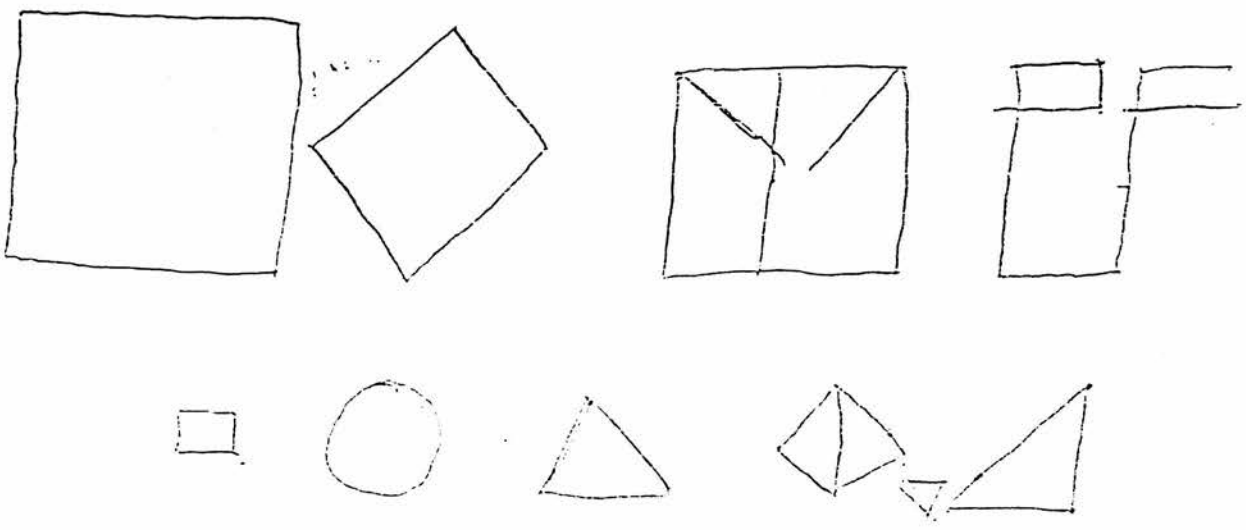
Pipe, whistle Scissors.
Bell. Spectacles Razor.
Torch, Match

This is a very nice Day.

This Brick Building was Built.
last new Year.

I am very Hungry.

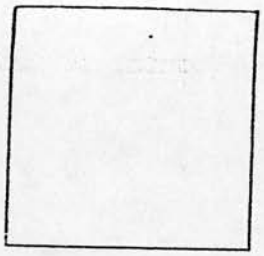
The colour of the Walls is
green.

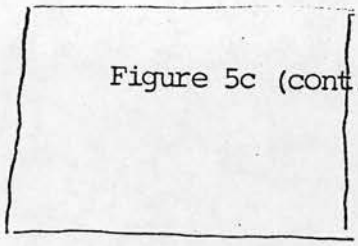


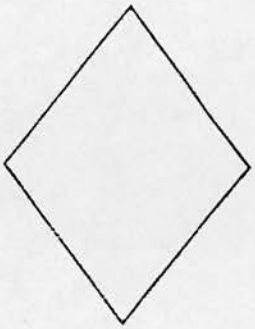
- Bell.
- Torch.
- Razor.
- Pipe
- Whistle
- Tweezers
- Spare Glasses.
- Watch
- This is a very nice Day.
- This Brick Building was bent last year.

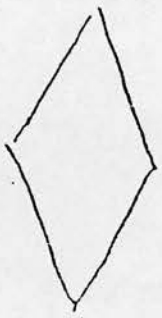
I am very Hungry
The Colour of the Walls
is green.

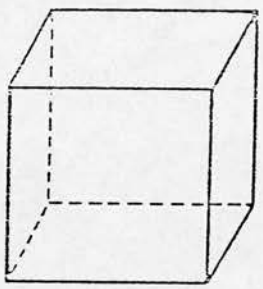
Figure 5c (cont.)

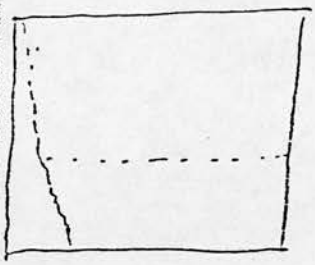


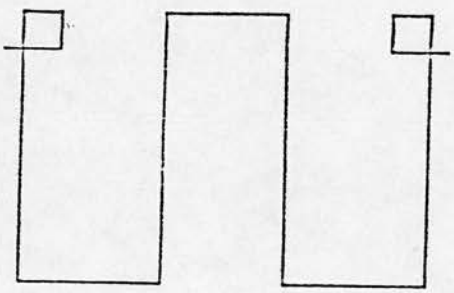


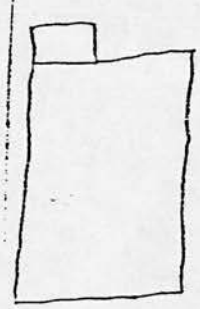


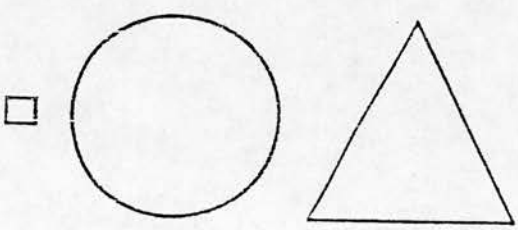


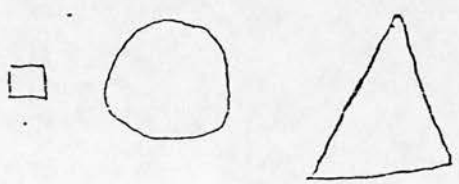


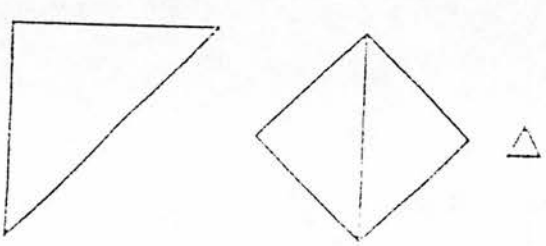


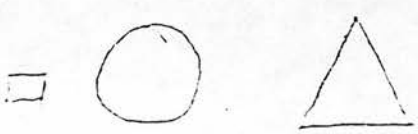












perseveration. This patient demonstrated a drawing disability from initial to final assessment with increased misperception of form in designs. The two dimensional square and rhombus presented no difficulty. Simplification, neglect and perseveration of direction in the small lateral squares are evident on Design 4. The cube lacks perspective in the first year, the second and third years it is increasingly fragmented. The first sequenced figures are inverted for the first and second year and finally there is neglect and perseveration.

Figures 6a and 6b demonstrate a two year decline in ATD. The third year the patient was agraphic. Despite this patient being a professional nurse, writing shifts from print to script, words are misspelt, letters omitted and, in copying, perseverated and substituted. The patient has a complete constructional apraxia demonstrated by accompanying drawing designs. A year later 6b orthography has deteriorated with letter substitutions, omissions and paragraphia as the substitution of "bell" for "whistle". Again dictation appears to be a distorted but not neologistic written jargon. Copying is marked with scrawling and deletions. Drawing ability demonstrates a failure to complete two dimensional tasks.

Figures 7a, 7b, 7c show the progression over 3 years of an ATD patient. 7a is without error but for disagreement of verb auxiliary and tense in the second dictated sentence. Drawing ability demonstrates over simplification of the cube. In the second year there were errors of orientation and automatic writing. Apart from one error in "razor" the performance is without error despite a constructional

apraxia. In the second year the patient has lost perspective, design 4 is simplified by central neglect while the final figure has an increased number of lines and overdrawings. In the third year there was increased disorientation in automatic writing. orthographic errors and dictation is marked with omissions and the disagreement of auxiliaries and verbs. Copying ability was retained but marked with false starts. The patient demonstrated a constructional apraxia for all but one dimensional figures.

Figures 8 a, b and c illustrate the dysgraphia in ATD where the presenting feature was language pathology. In 8a automatic writing was preserved. Spelling is marked with phonemic paragraphias, "dell" for "bell", the initial letter is a substitute of place of articulation but voice and manner are retained. The word "pipe" contains a double substitution of a voiced bilabial stop for its unvoiced cognate. "Glasses" contains a substitution of an unvoiced for a voiced glottal stop while "torch" has a substitution that is either random or unvoiced bilabial plosive for its alveolar cognate. Thus the graphemic errors are related by feature as voicing and place to targets, manner of articulation is correct. Other words are approximations as shown in writing to dictation although it appears that substitutions are not entirely random. The patient demonstrated no real constructional apraxia but writing was micrographic. When drawing there was an error in perspective in the reproduction of a cube, there was also scrawling and overdrawing of lines.

Figure 6a

RAZAR. 2

PIPE 3

BELL. 3

TORCH. 3

GLASS. 1

WATCH. 3

Whistle 3

Jewelry 4

This is a very nice DAY.

This BRICK Building
was Built
LAST YEAR.

I AM VERY SORRY.
HE could not help the rain

Figure 6a (cont.)

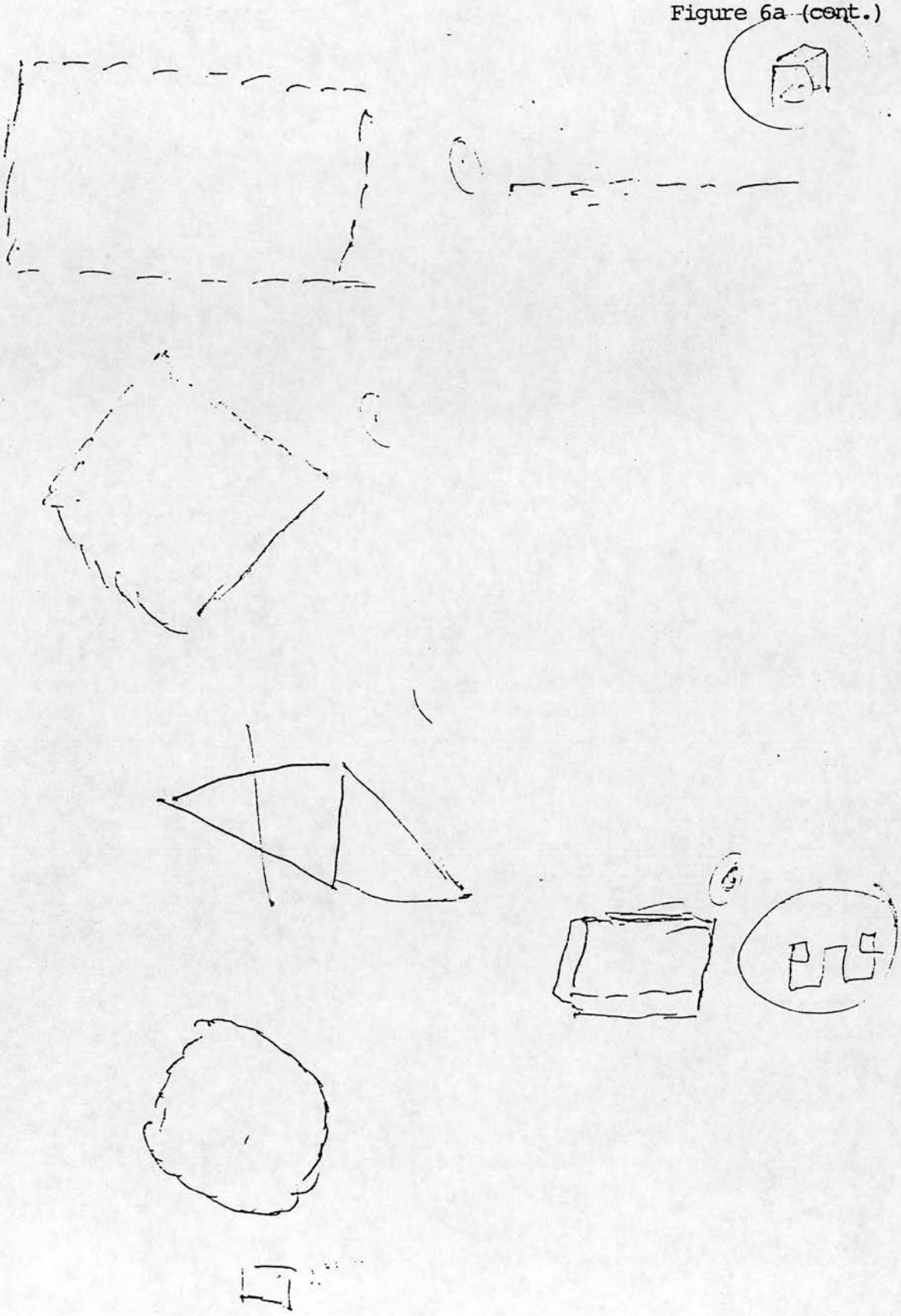


Figure 6b

BELL. RAZOD

pipe

TORCH

BELL (white)

Iwers

whisal

---

watch Glasser.

Thusary ni do

This Brick building

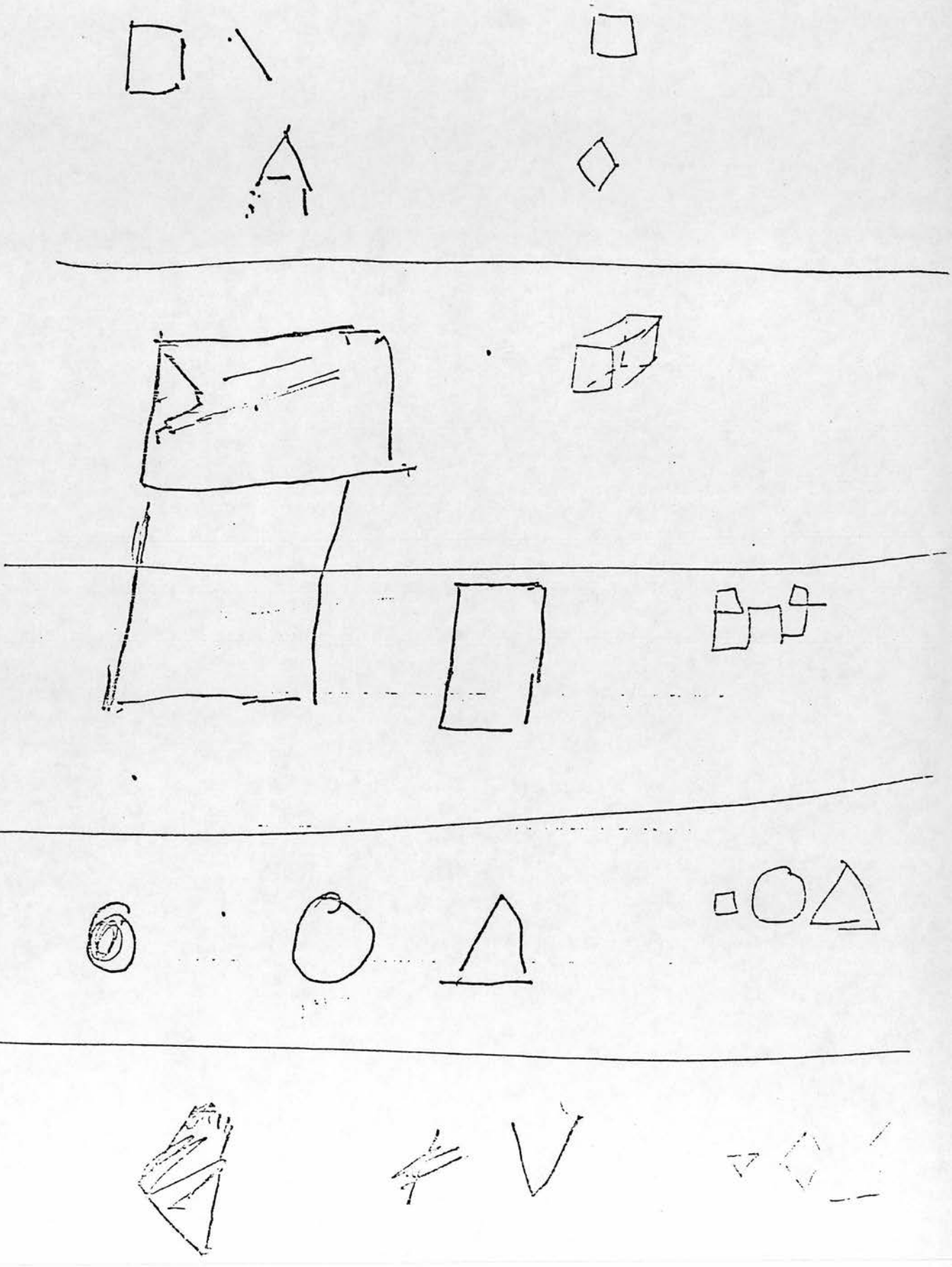
was Built Last Year.

I am very hungry

— — — — — —

~~The

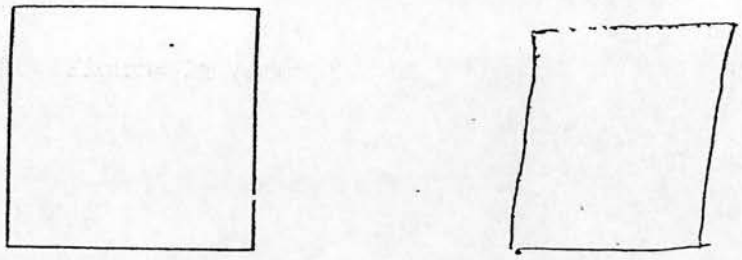
Figure 6b (cont.)

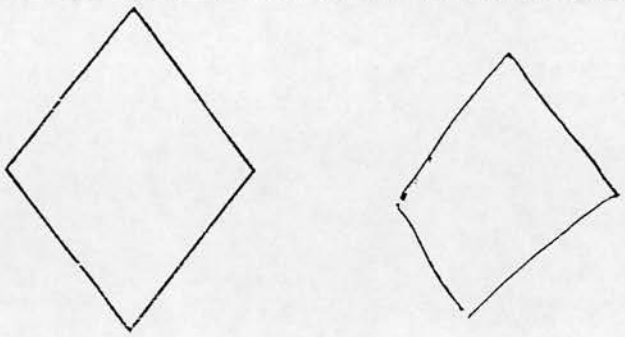


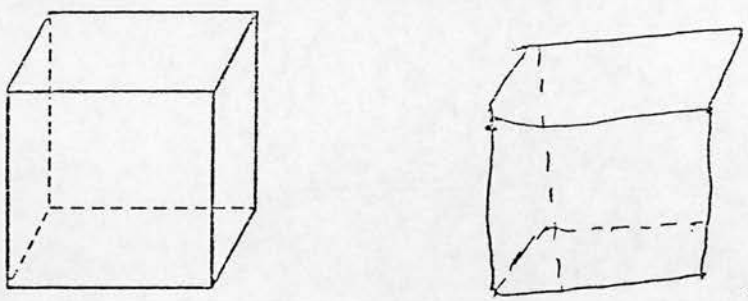
Bell
Torch
Razor
Pipe
Spectacles
Tweezers.
Whistle
Watch

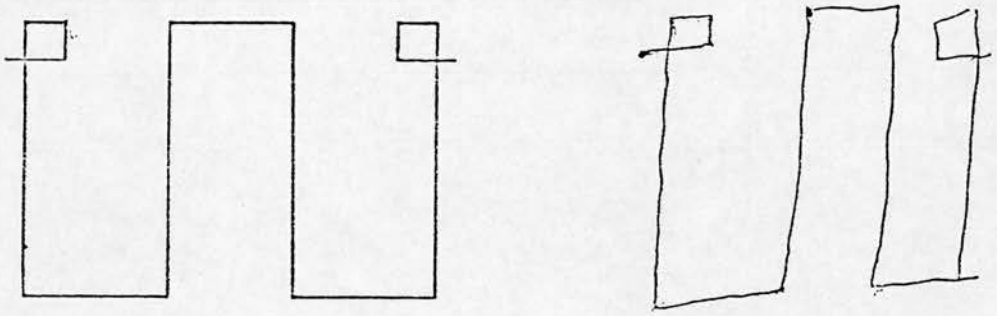
This is a very nice day.
This brick building was build last year.
I am very hungry.
The colour of the walls is green.

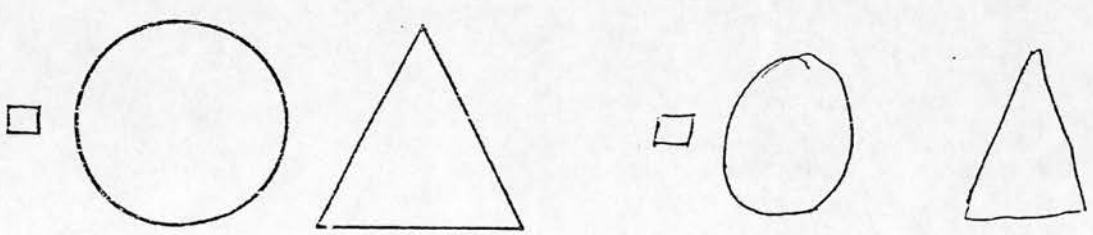
Figure 7a (cont.)











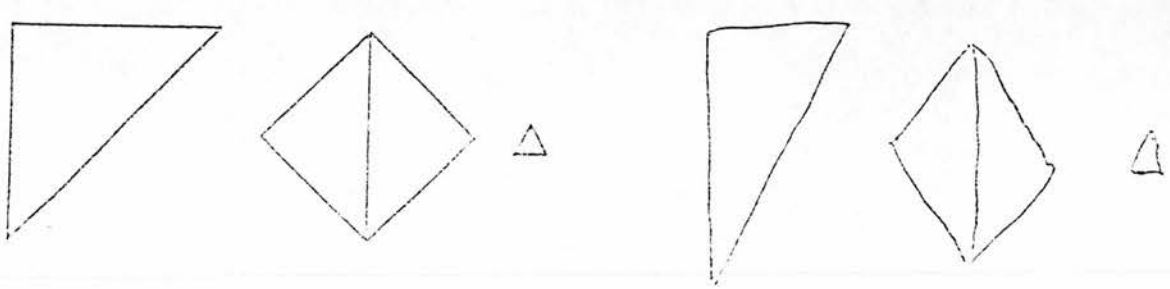
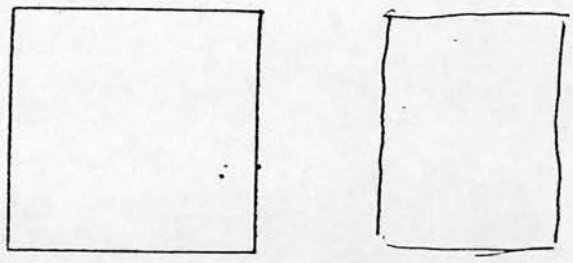


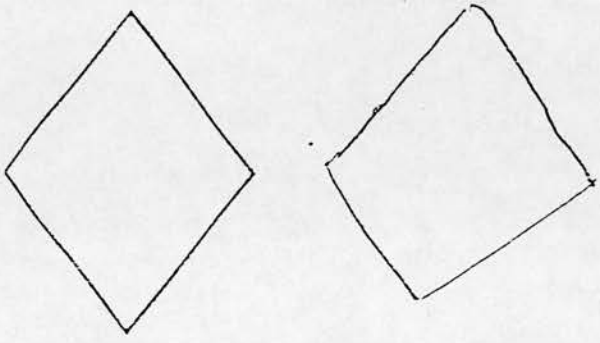
Figure 7b

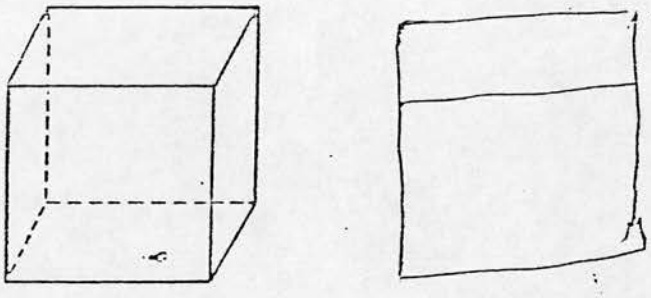
Bell
Razer.
Pipe
Torch
Tweezers.
Whistle
Watch
Glasses

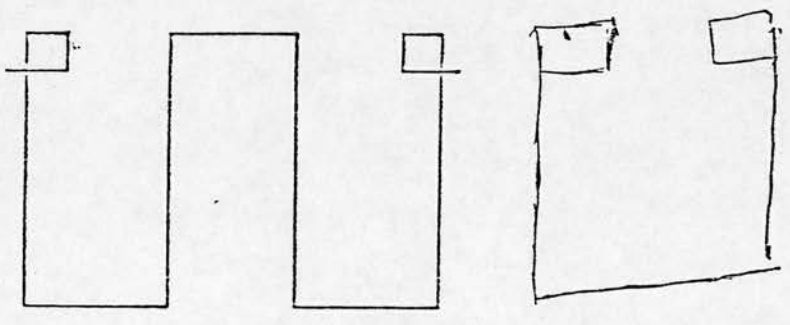
This is a very nice day.
This brick Building was built
last year.
I am very hungry.
The colour of the walls is green

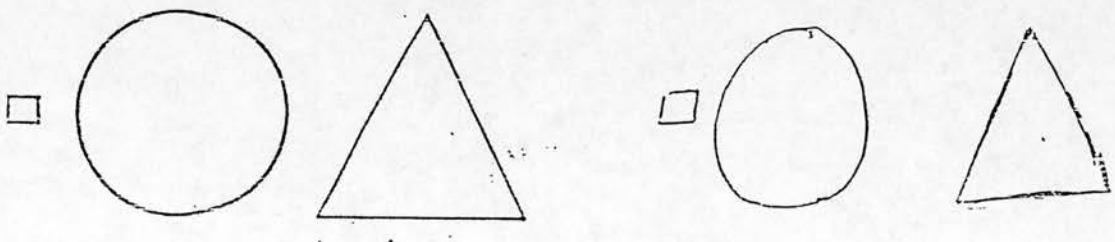
Figure 7b (cont.)











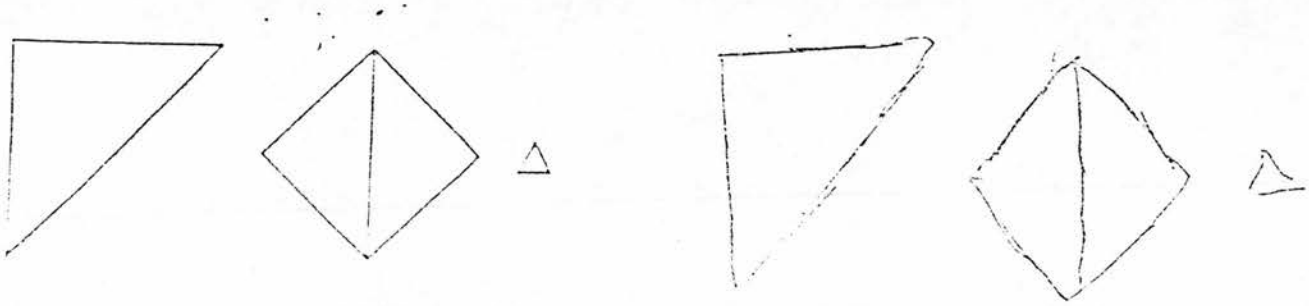
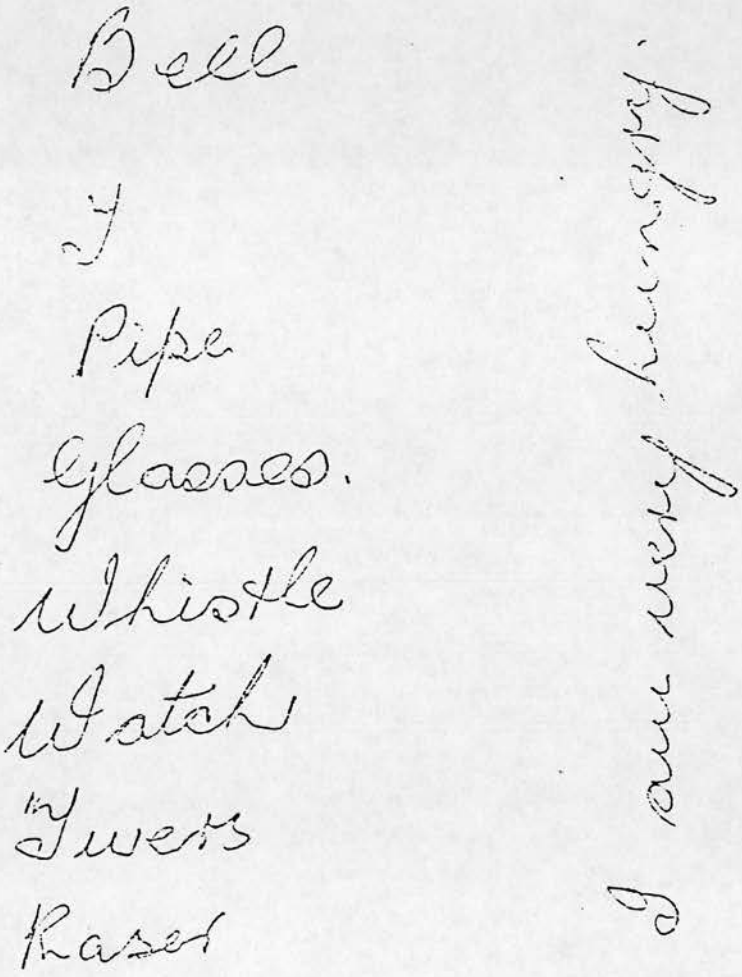


Figure 7c



This is a very nice day.
This truck but
was budding last year.

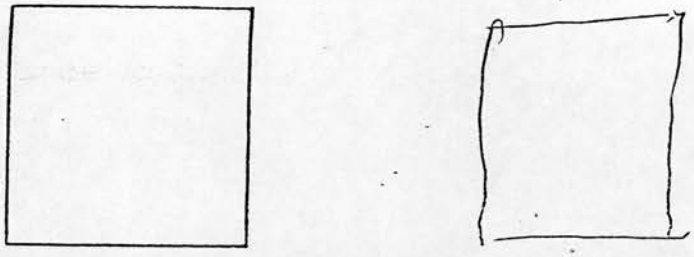
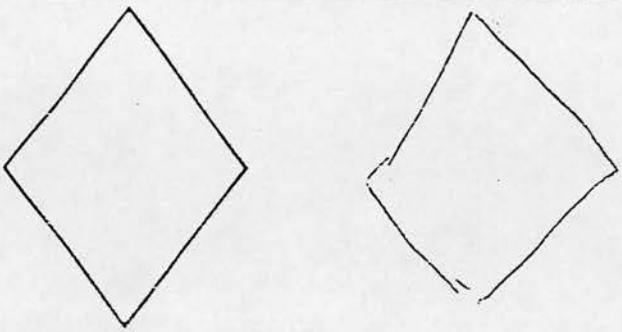
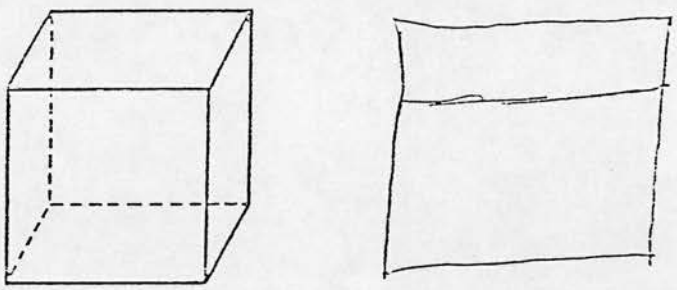
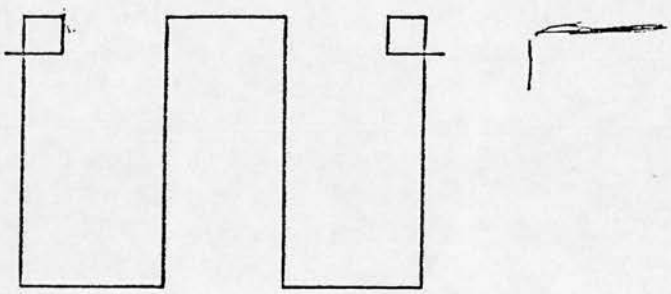
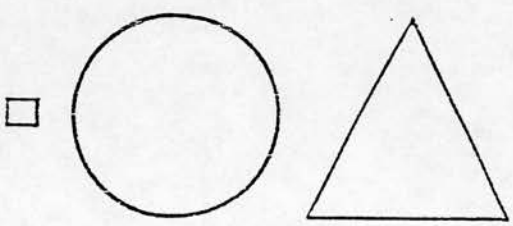


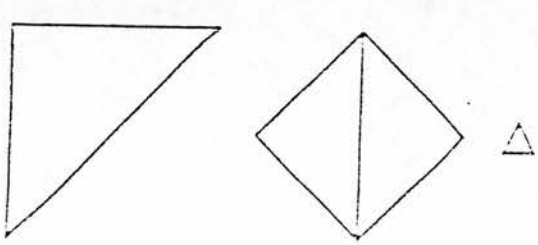
Figure 7c (cont.)











The second year (8b) demonstrates consistency of error. In "bell" the unvoiced alveolar stop /t/ is substituted. In "pipe" the second unvoiced bilabial plosive /p/ is substituted for a voiced alveolar stop /d/. "Torch" is consistent in its substitution of feature and writing to dictation shows substitution of related features rather than randomised errors. Again there is no constructional apraxia apart from scrawling. The cube is complex and lacks perspective. Writing to copy is without real error. In the third year the ability to write automatically and write words to dictation is lost but the ability to copy remains. Calligraphic errors are noted with the gradual increase in disturbance of drawing ability as shown by scrawling and difficulty of drawing angles, curved lines were used to join intersections.

Figures 9a and 9b and Figure 10a show the writing in two cases of Pick's disease. Figure 9a is without error apart from one spelling mistake in "tweezers" and a verb auxillary disagreement in the second dictated sentence. Drawing ability suggests an increased difficulty in drawing three dimensional as compared to two dimensional figures. Writing 9b shows a further deterioration of orthographic capacity after a year. There are corrections and perseveration in dictation and copying. Drawing ability has decreased with scrawling, lack of planning perspective, neglect and finally perseveration. A similar pattern of corrections is noted along with perseveration in Figure 10a. Automatic writing is retained. Orthographic errors are evident in dictated sentences with perseveration to the point of incompletion of the task. The task

Figure 8a

Dell
Rosat
Bike
Classes.
Misel
Dweets.
Wacho
ROCK.

this is a wetch much day
mise Brich dilleing was BiLt last Lick
I am very hungry.
The colour of the walls is green.

Figure 8a (cont.)

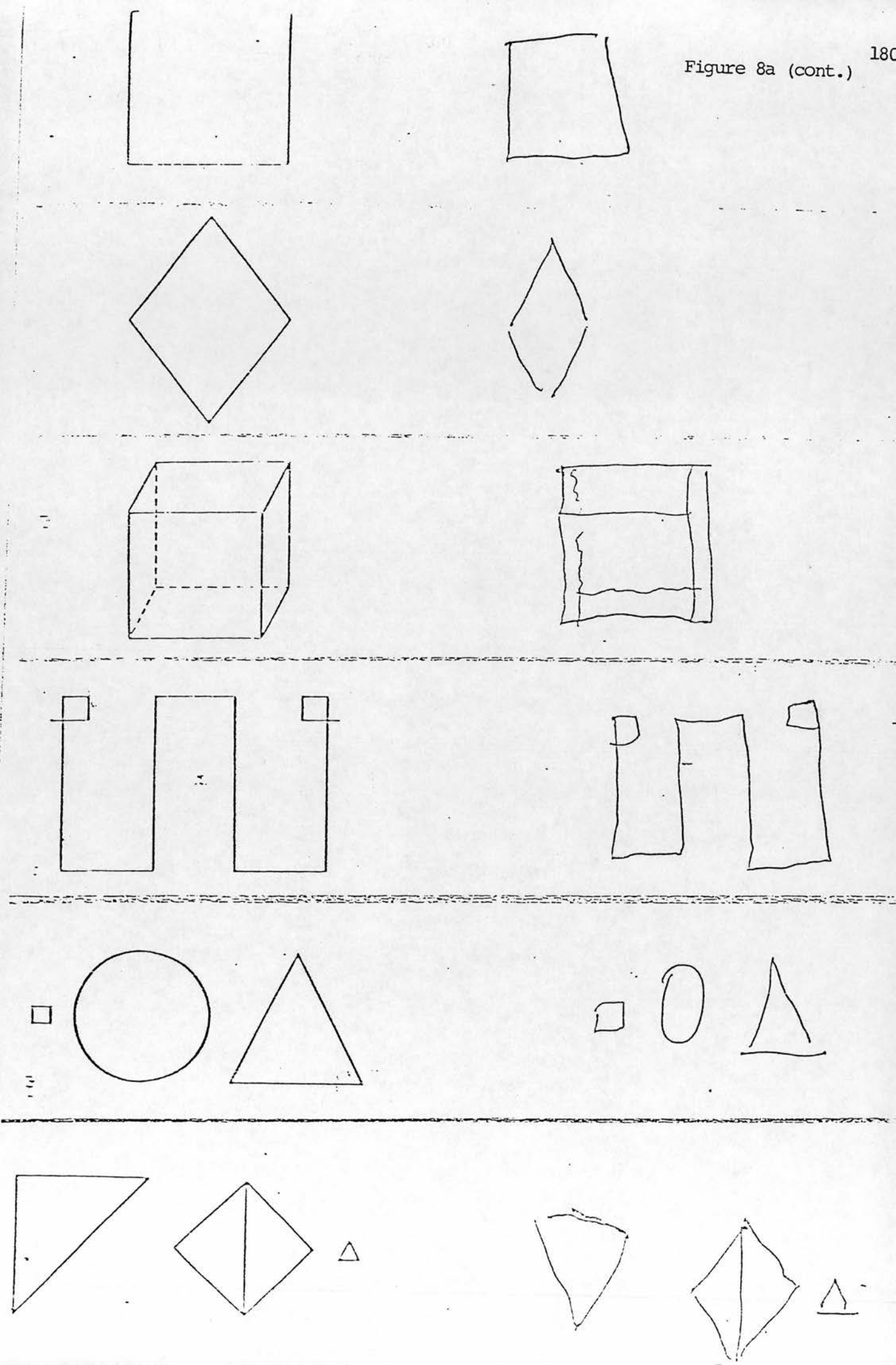


Figure 8b

Tell.
Bide.
Mackor
Mussel
PORCK
Cassee.
Teeras.
Passet

This is aweery nis Paaul.

This dirk Billdin was. Bilid

Hast goo T.

I am very hangry.

The colour of the walls is green.

Figure 8b (cont.)

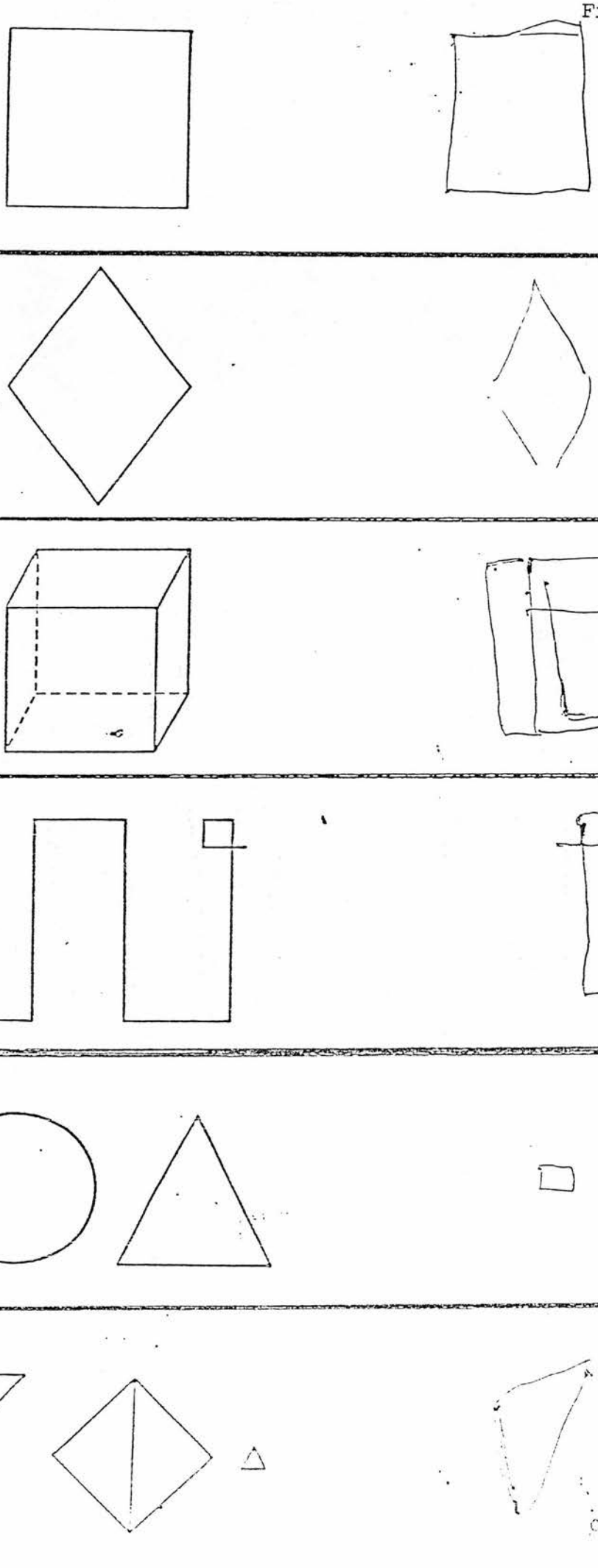


Figure 8c

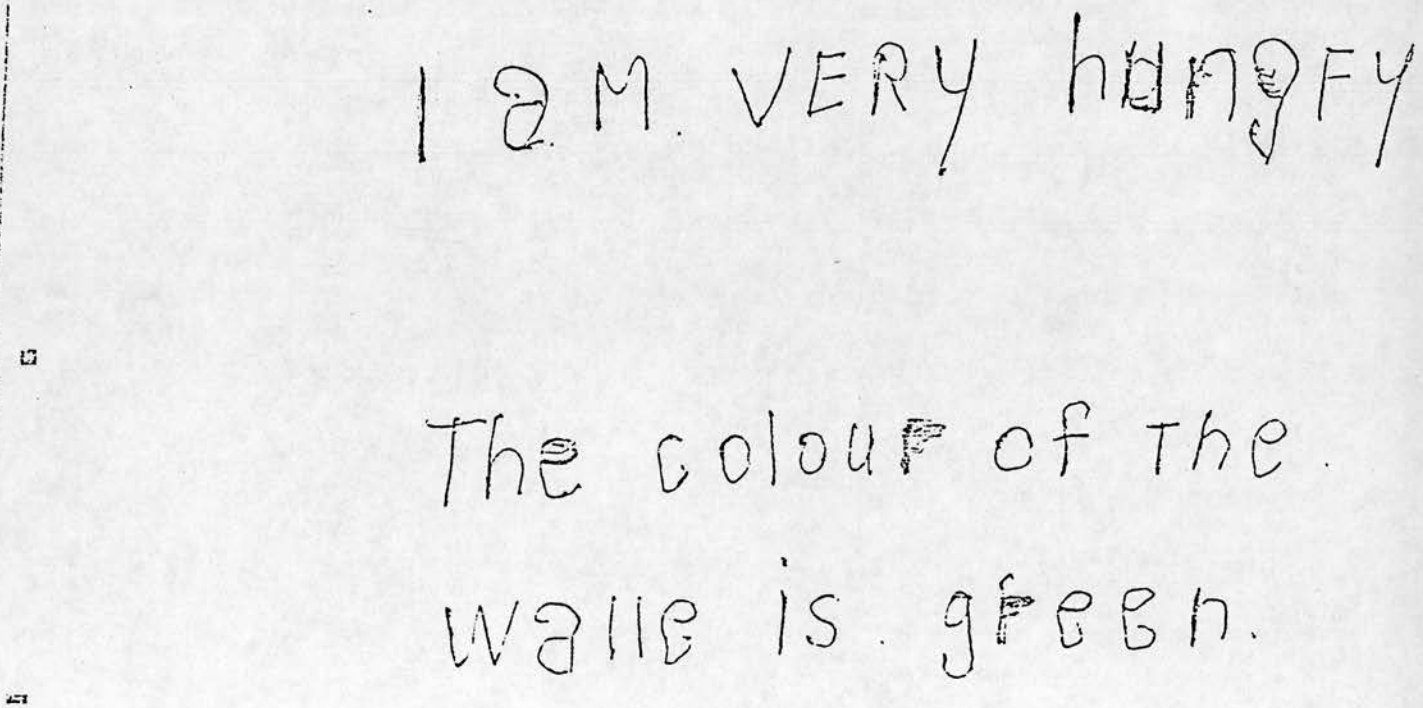
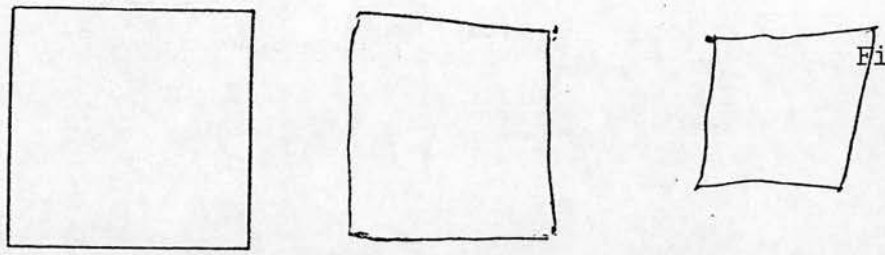
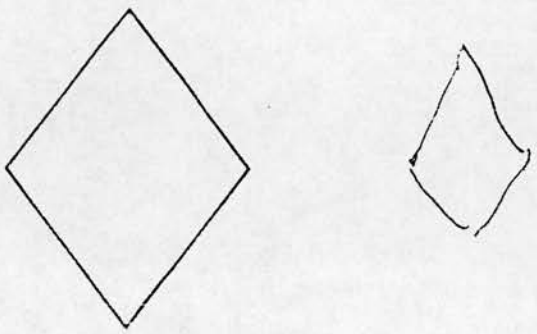
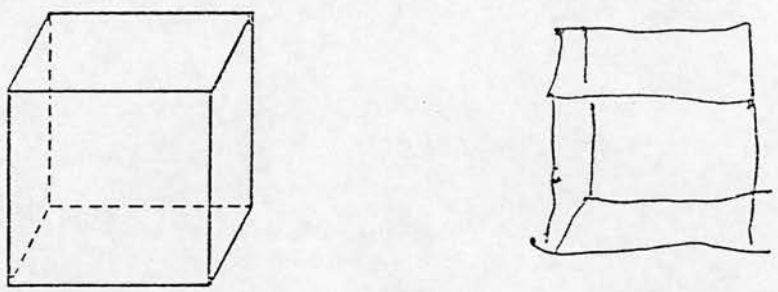
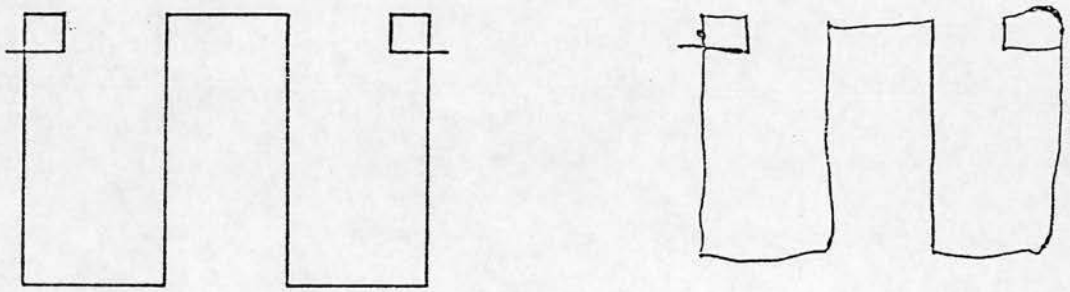


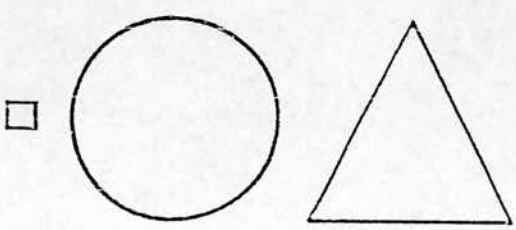
Figure 8c (cont.)

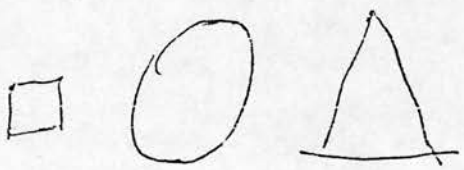


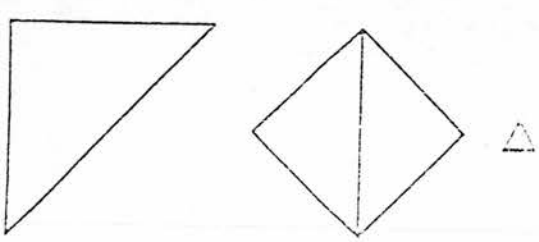












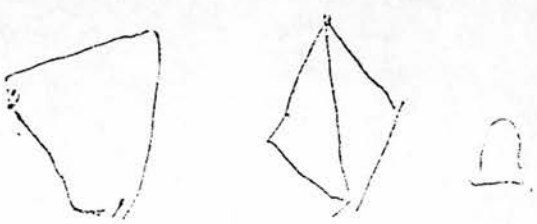


Figure 9a

BELL
RAZOR
WHISTLE
PIPE
TORCH
WATCH
GLASSES.
TWISTERS.

THIS IS VERY NICE DAY
THIS BRICK BUILDING WAS BUILD LAST YEAR.

I'M AM VERY HUNGRY

THE COLOUR OF THE
WALLS IS GREEN.

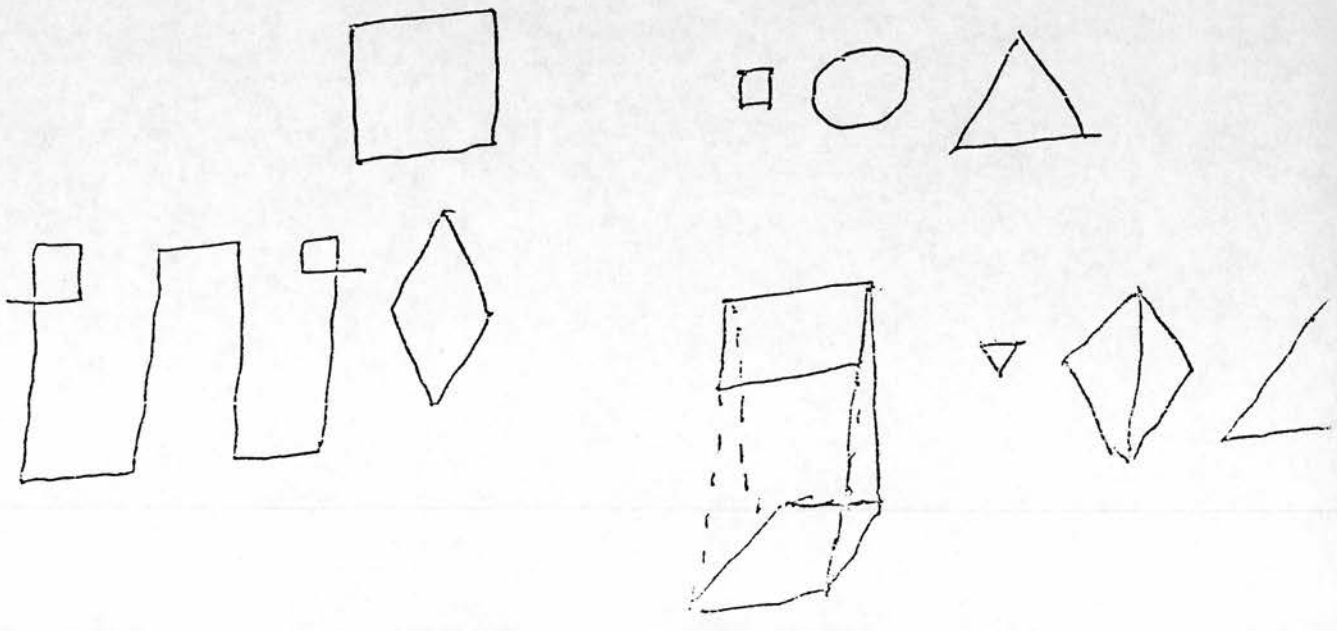


Figure 9b

BELL

TORCH

RASOR

WHISTE

PIPE

WATCH

GLASSES

TEWST

THIS RS VERY AS TOBS

THIS BRICK BUILDING WAS BUILT

LAST YEAR

BUILD

I AM VERY HUNGER

I AM WALLS VERY

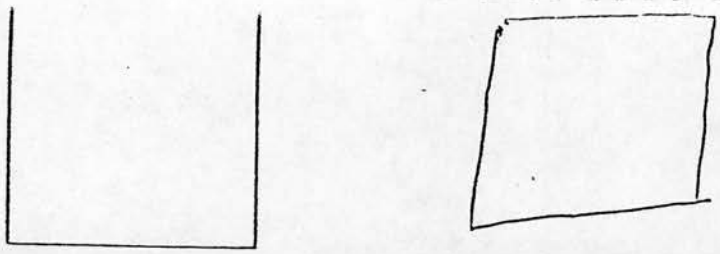
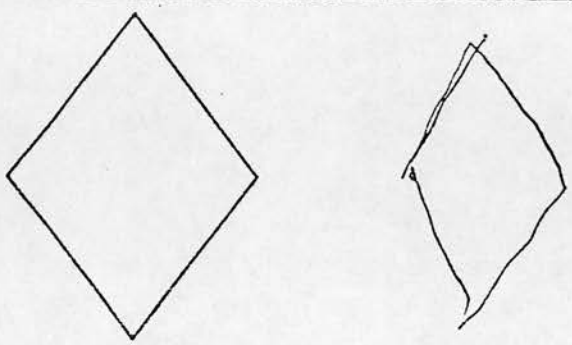
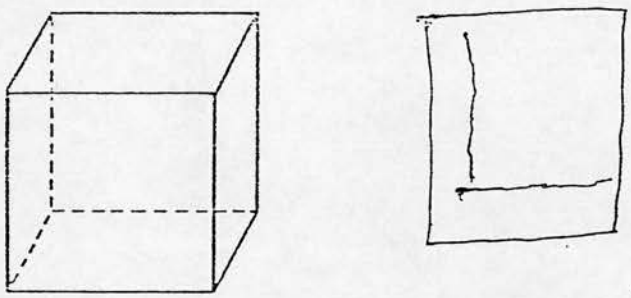
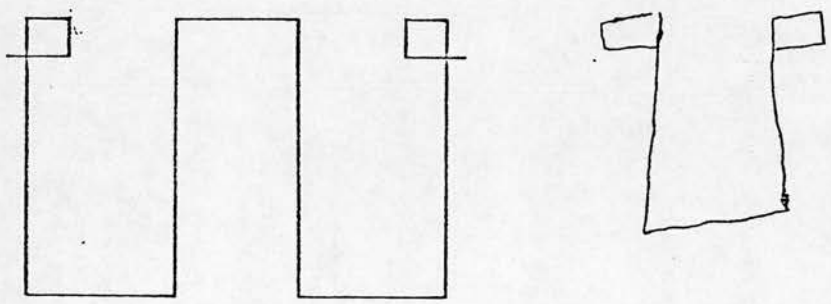
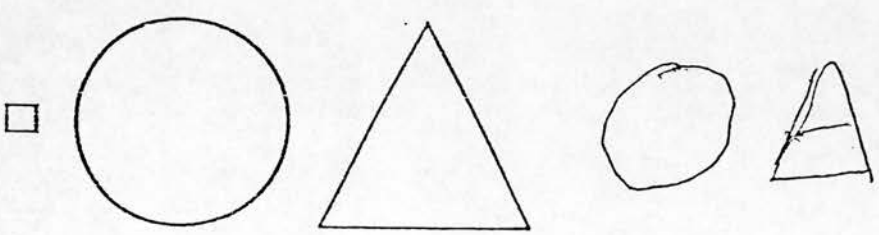


Figure 9b (cont.)









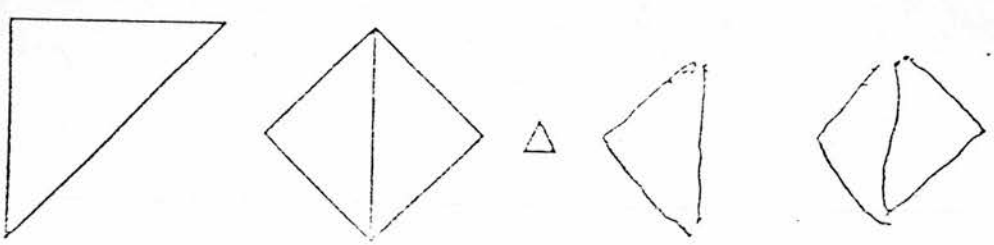


Figure 10a

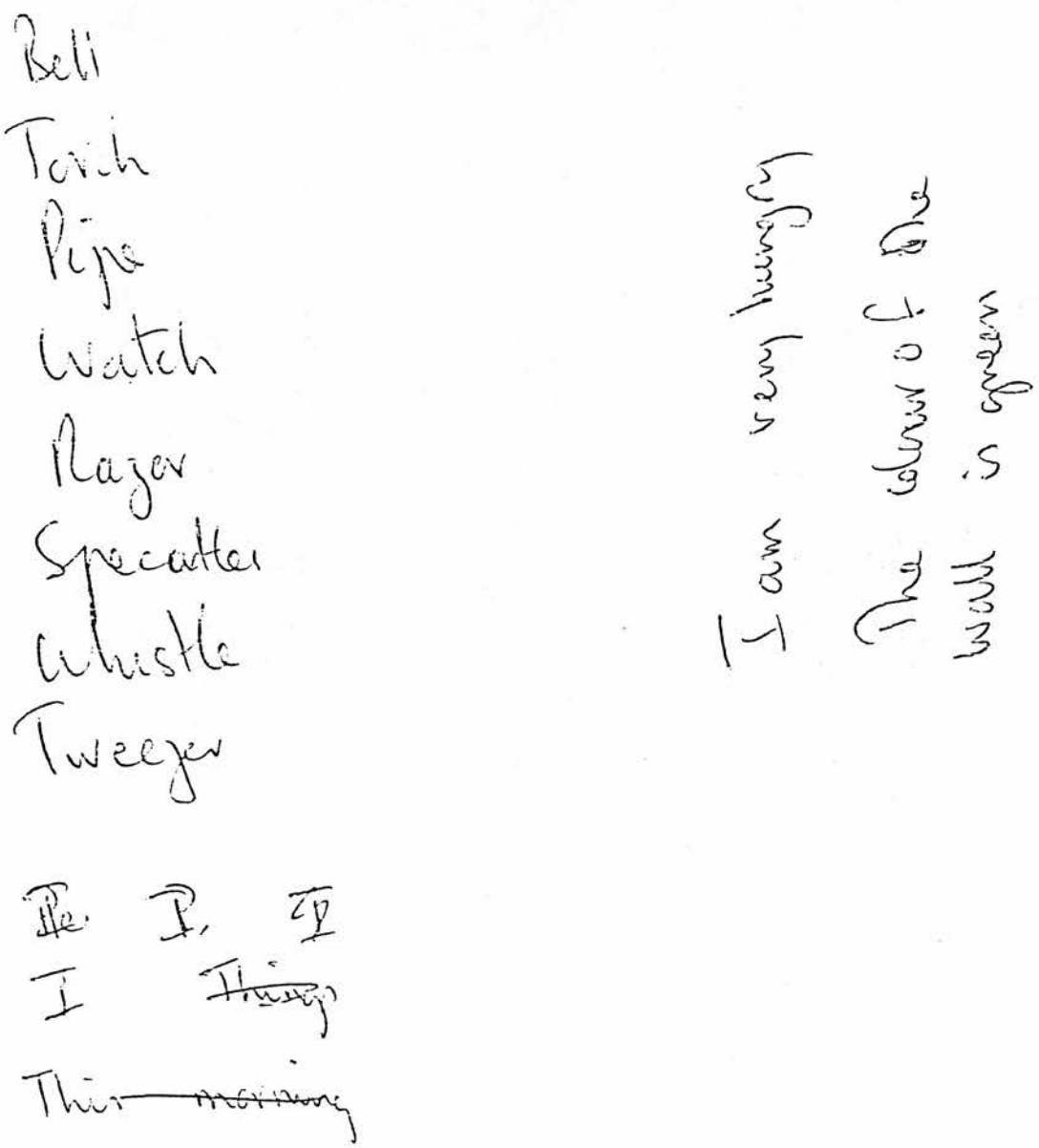
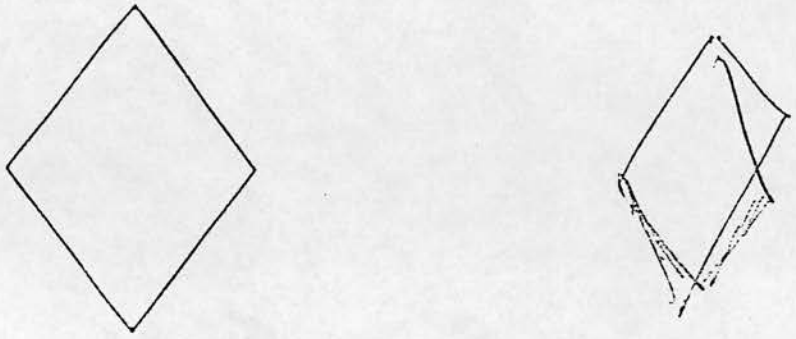
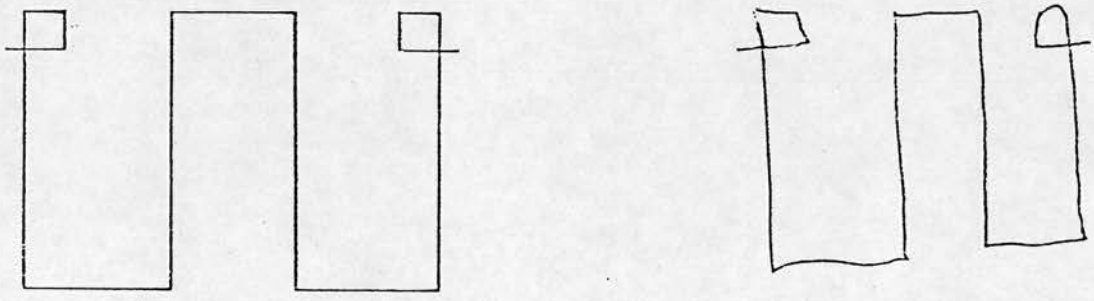
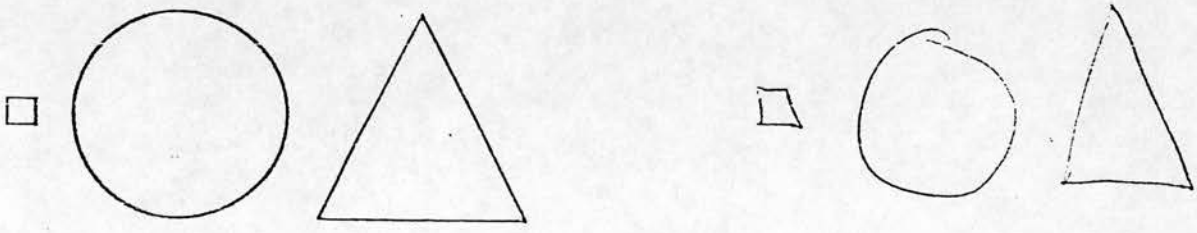
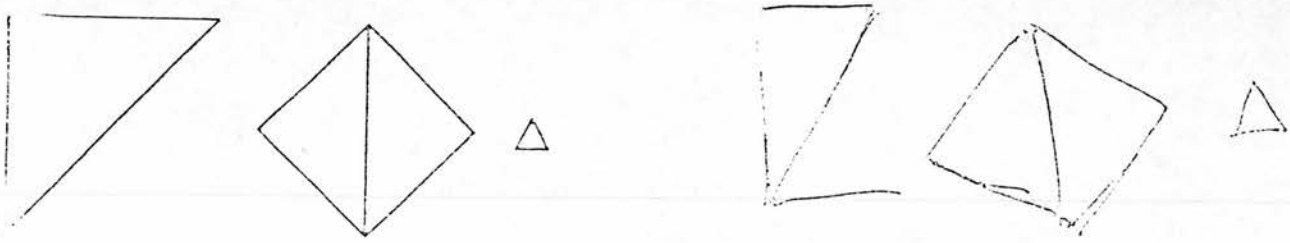

Bell
Torch
Pipe
Watch
Razer
Spectacles
Whistle
Tweezer
I am very hungry
The colour of the wall is green
The P. T.
I Hungry
This morning
This is nice day
This built building was This The
was

Figure 10a (cont.)









could be completed if a visual model was presented in copying sentences. Drawing errors are present. Although there is good perspective there is overdrawing of lines and perseveration. On the first design the patient perseverated the examiner's instruction to draw a square. Perseveration, repetition and corrections are regarded as a common symptomatology of Frontal lobe disorder (Walsh 1978).

In summary calligraphy in ATD degenerates with the progression of the disorder. Spatial agraphia appears less common when the patient is unable to draw geometric figures. Errors are related to phoneme-grapheme coding presenting as incomplete words, missed inflections, substituted letters and incomplete spelling more akin to left hemisphere lesion than to bilateral disturbance as in spatial dysgraphia. Moreover it appears that the ability to copy is more preserved being aided by the visual modality, because errors are present in writing to dictation. These errors appear to be a disturbance of the signal as it is perceived rather than generated neologistic jargon.

The ability to copy rather than write to dictation suggests that visual perception is less compromised in ATD. Neuroanatomical studies also note the greater sparing of the calcarine cortex than the inferior temporal lobes, although Stevens (1985) noted mis-perception of picture description in ATD patients.

The writing of ATD seems differentiated from Pick's disease which has specific frontal characteristics as false starts, corrections and perseveration.

5.5.2.10 Apraxia

The problem of apraxia in the dementias demands comment. Bucco facial apraxia affects speech fluency and at the level of the larynx it affects voice. Limb apraxia impairs kinesics and gait. Ideational apraxia reflects impairment in sequencing and constructional deficits cause distortion of visuo spatial analysis and synthesis. The clinical manifestations pervade daily living producing voicelessness, dysfluency, flattening of affect, disturbances of gait, dressing and spatial disorientation.

Dyspraxia is "the inability to perform certain subjectively purposive movements and movement complexes with the conservation of motility, sensation and co-ordination (Wilson 1908). Ideomotor, Ideational and Constructional forms of dyspraxia are apparent dementia. These, as visuo spatial deficits, affect the everyday demands of daily living from dressing to toileting, cleaning and cooking to making a phone call or a cup of tea.

In ideational apraxia engrams of individual movements are retained but lost in complex sequences. It was defined by Pick in 1905. The concept of ideational apraxia remains uncertain, it may be severe ideomotor apraxia (Sittig 1931, quoted De Renzie et al 1968).

Ideomotor apraxia is demonstrated by poor intransitive gestures that do not improve on imitation: spontaneous actions are not impaired. The condition appears to be a disorder of the programmer of motor engrams or memories: engrams are cortical traces established by habituation (Denny-Brown 1958). Ideomotor apraxia interferes with voluntary movement from gestural communication to walking.

Pick demonstrated ideational apraxia in degenerative dementia attributing it to bilateral lesions. Both Denny-Brown (1958) and De Renzie (1968) have argued for ideational apraxia being the inability to gesture the use of objects, whereas ideomotor apraxia is the inability to perform transitive gesture.

Movement apraxias are generally considered to be localised from lesions of the left hemisphere even in left handers (Hecaen et al 1969), but it needs to be differentiated from motor neglect impersistence and perseveration. Luria (1966) argued that post central lesions interrupt proprioreceptive analysis and lead to afferent apraxia, while posterior occipital - parietal lesions cause disruption of visuo-spatial relations. One would therefore expect ideomotor apraxia in diseases of the bilateral temporal and parietal cortex.

Apraxia as a verbal or speech disorder is defined by Rosenbek (1980):

> "apraxia of speech is a sensory motor speech disorder resulting from brain damage. Symptoms are impaired volitional production of normal articulation and prosody. The articulation and prosodic disturbances do not result from muscle weakness or slowness, or aphasia, confusion or general intellectual deficit or hearing loss. Rather they result from inhibition and impairment of CNS programming of skilled oral movements".

Apraxia of speech is not common in ATD, fluent speech is retained there is no articulatory groping. Ideomotor and ideational apraxia are a common feature of ATD (See below Chapter 5.7).

and disorders of visuospatial processing present constructional apraxia.

5.5.2.11 Constructional Apraxia

Warrington et al (1966) investigated drawing disability in relation to laterality of cerebral lesion and confirmed a difference in quality between left and right lesions: left hemisphere lesions produce poor analysis of the constituent parts of models while the right produce spatial disproportion. Neglect was related to right lesions confirming Arrigoni and De Renzie's study of 1964. Gainotti and Tiacci (1970) similarly compareddrawings of left and right hemisphere lesions and concluded left hemisphere lesions produced reduced figure size, simplification and a decrease in the number of right angles. Right hemisphere lesions produce unilateral spatial inattention, neglect, a piecemeal approach, poor orientation to the page, the inclusion of irrelevent material and poor spatial relations. Similar observations are reported by McFie and Zangwell (1960) and Piercy et al (1960).

Constructional apraxia is a common finding in ATD and early and late dementia. It represents either impairment of visuo spatial abilities (as a right hemisphere deficit), or failure of complex activity (as a left hemisphere deficit). It has been defined as "the difficulty of putting together one dimensional unit so as to form two dimensional figures and patterns" (Critchley 1953) or "a disturbance of formulative activity such as assembly, building or drawing in which the spatial form of the product proves to be insuccessful without there being an apraxia of single movements" by Kleist. Such deficits are usually demonstrated in drawing, copying geometric figures, mozaics, jigsaws, stick

assembly, brick construction and Block Design that appear to demand right hemisphere visuo spatial alignment and left hemisphere language mediation.

Constructional activities are deemed more sensitive to impairment in brain damaged patients than many WAIS subjects (Benton 1969) because they require spatial analysis, an ability of component assembly, sustained attention and a capacity for planned activity. Block Design has been included in intelligence scales for both children and adults. In 1923 Koh designed the test as one of non verbal intelligence rather than spatial ability believed it assessed broad and fundamental capacity of analysis of situation, discovery of problem solution and sythesis of details into a consistent unity.

Patients in this study were all asked to draw six original shapes taken from a study by Arrigoni and De Renzie (1964) that investigated constructional apraxia and hemispheric locus of lesion. Figure 11 is taken from Piercy et al (1960) and demonstrates a difference of performance of left and right hemispheric lesions.

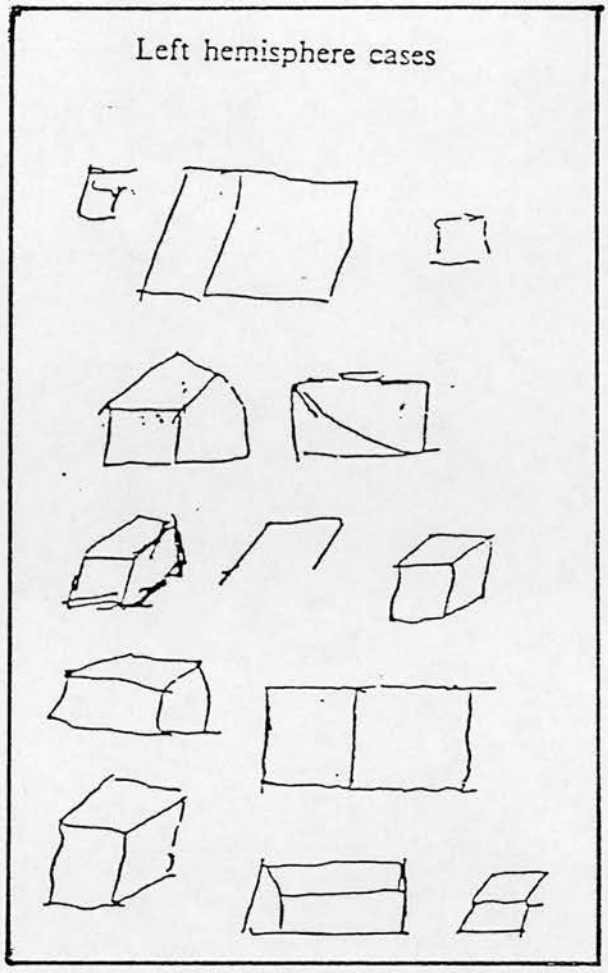
Constructional deficits impair abstract thought and performance on the RCPM regardless of the localisation of lesion (Benton and Fogel 1962, Perry and Smyth 1962, Arrigoni and De Renzie 1964)

Figures 12-15 present examples of copying of designs in the ATD cohort. Patients had little difficulty with the square and rhombus but errors of symmetry around a central axis were evidence of right orientation.

The figures include a cube of three dimensions represented in two dimensions and sequences of figures of different size and shape. Generally ATD patients tend to oversimplify (No. 2, 18) and neglect perspective (Nos. 5-7) when drawing the cube much like Piercy's left hemisphere cases.

Figure 13 demonstrates a closing in effect as the model overlaps the copy (No. 38). Neglect is often medial (Nos. 21, 25, 30, 34, 36) or bilateral (Nos. 26, 27, and 33). There is right neglect (No. 20 and 37) but no clear left neglect. Figures 14 and 15 show disturbances of sequencing. The rhombus usually has an error of its diagonal but there are not an overwhelming number of errors of size, scrawling or sequence. No. 61 represents perseveration of the preceding design, common in late dementia.

Figure 11
(After Piercy et al 1960)



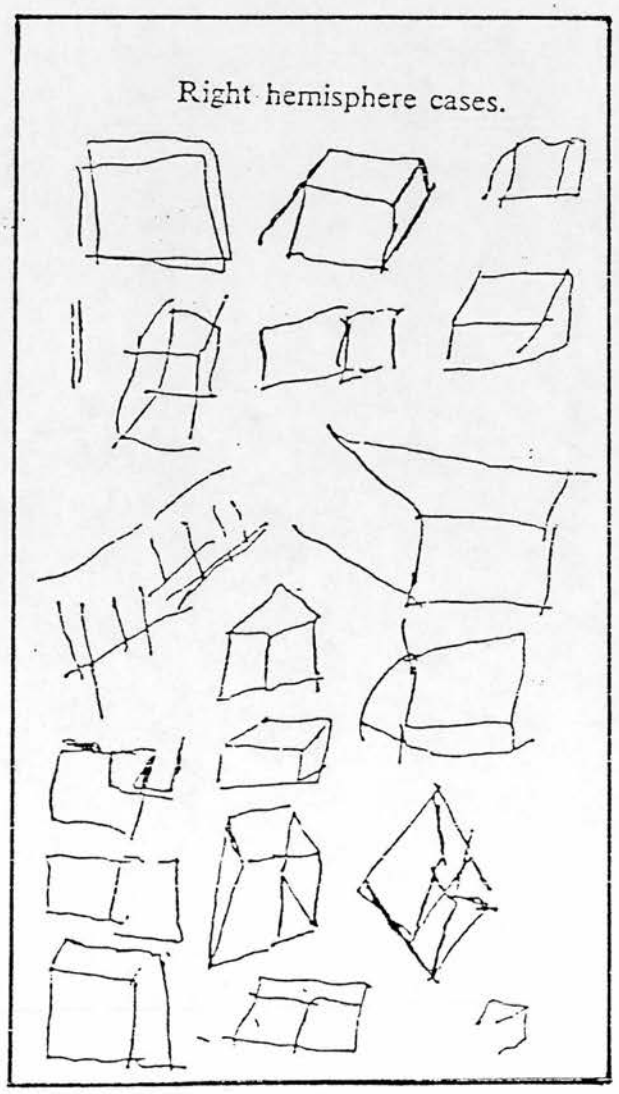
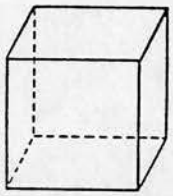
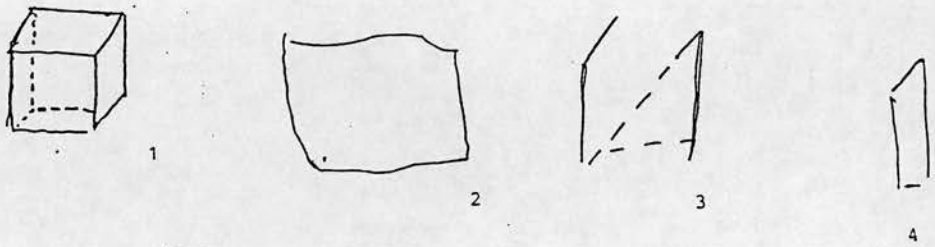
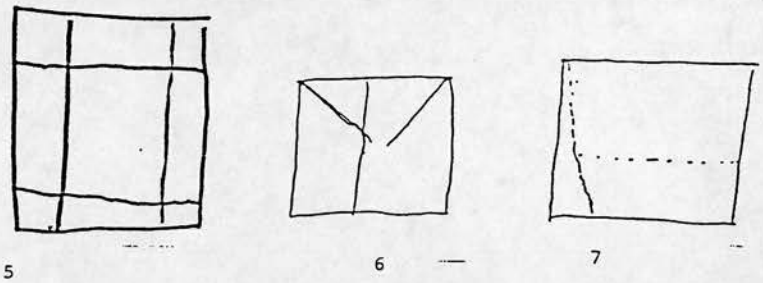
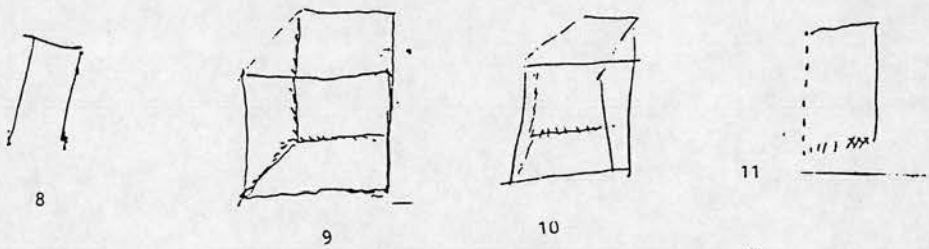


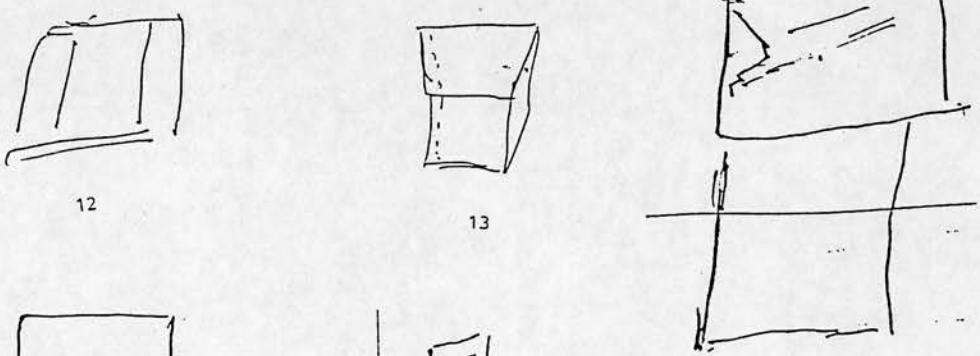
Figure 12

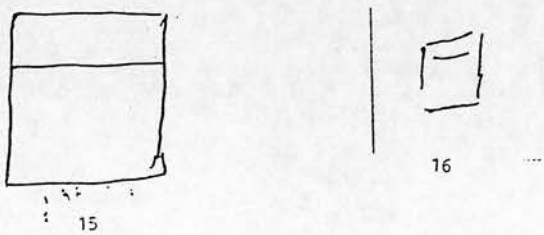












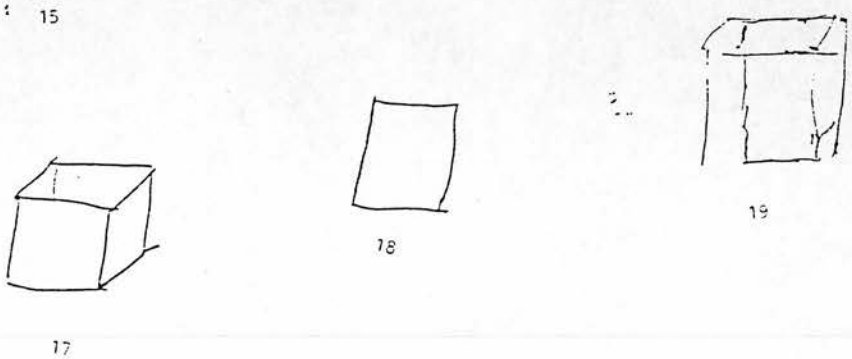


Figure 13

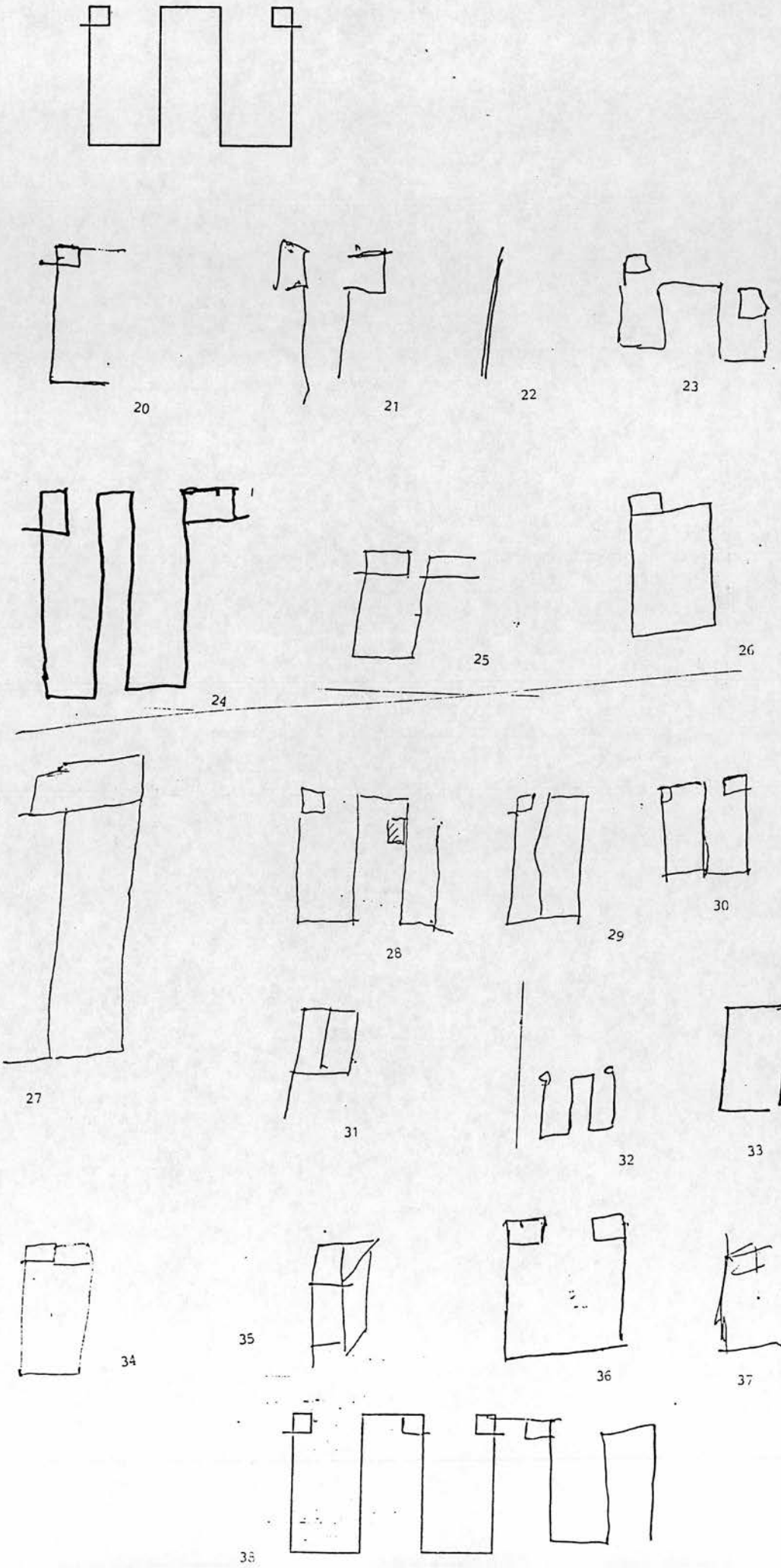


Figure 14

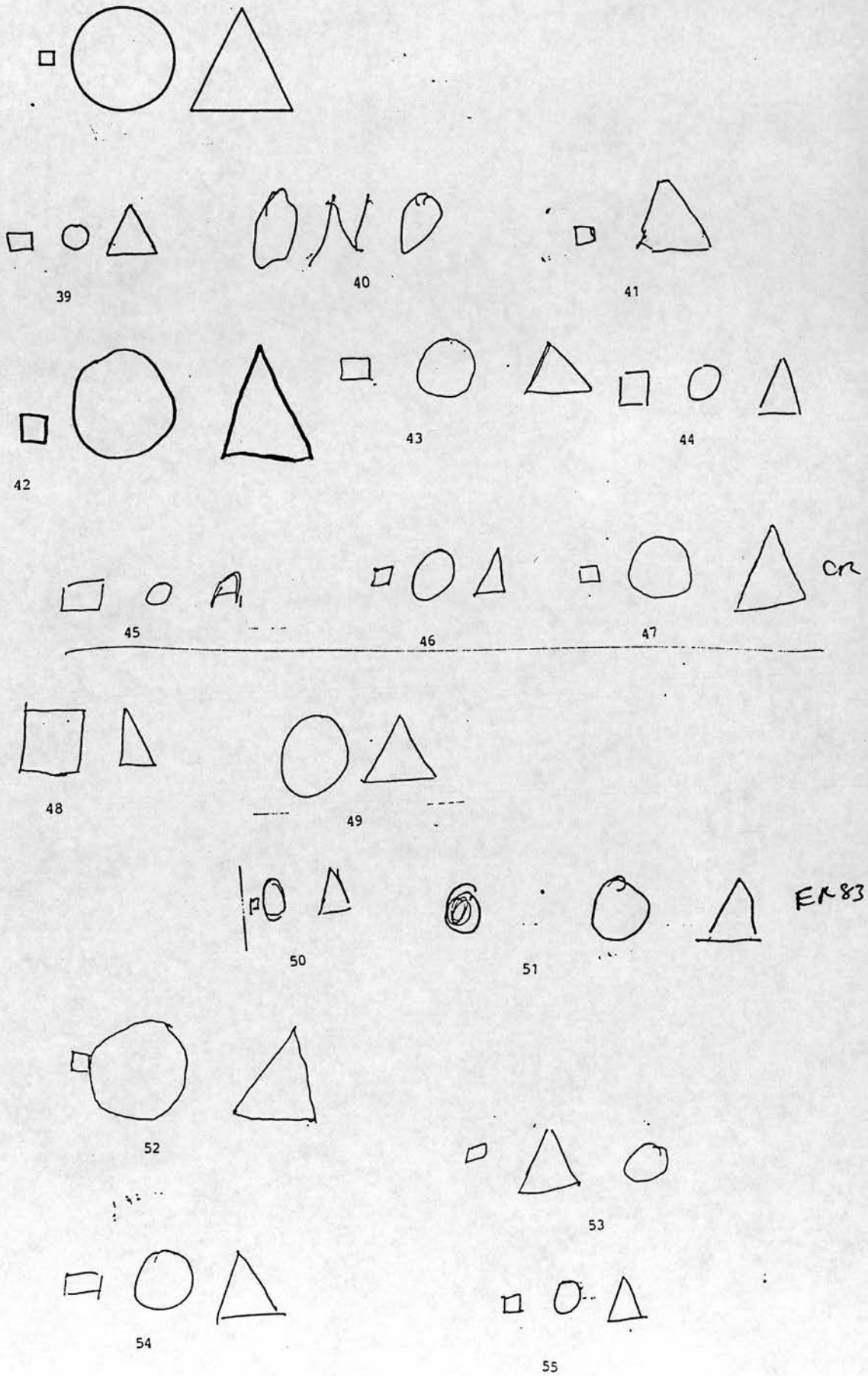
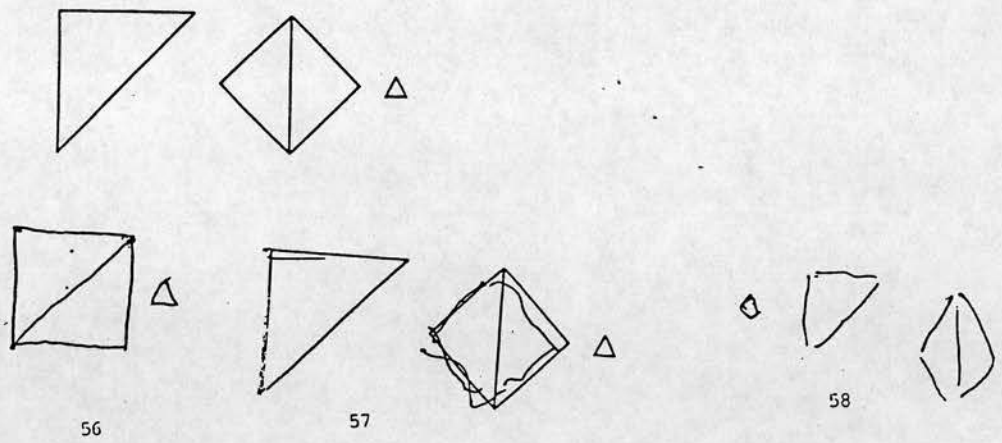
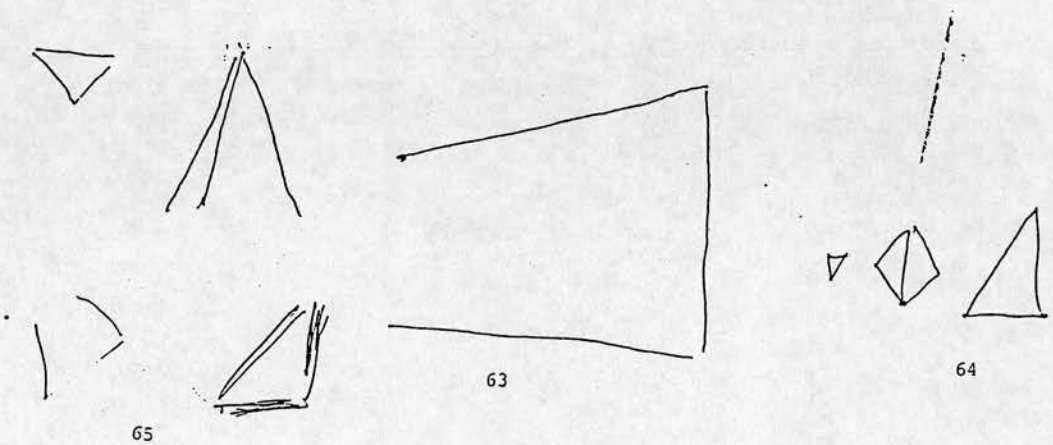
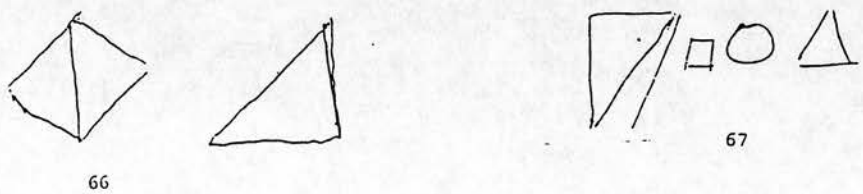
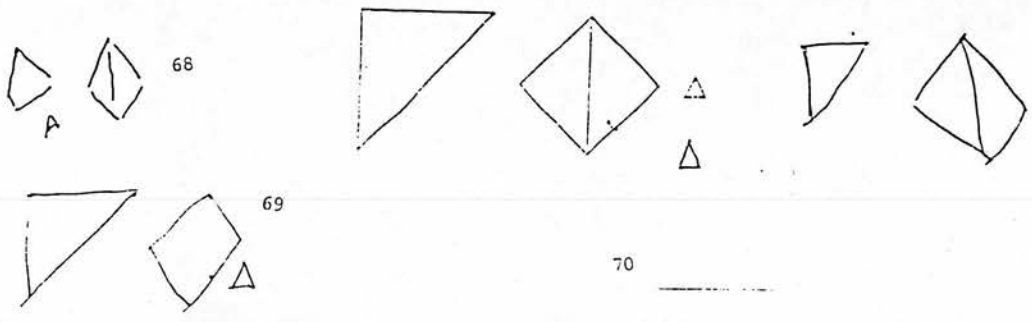


Figure 15









CHAPTER 5 (contd.)

RESULTS OF INVESTIGATION

5.6 LANGUAGE BREAKDOWN IN ALZHEIMER TYPE DEMENTIA OF PRESENILE ONSET

5.6.1 Introduction

Although cross sectional description of language in ATD exist longitudinal studies have not been reported.

5.6.2 Cross Sectional Analysis

Cross sectional analyses attempt to define language variables in terms of severity. Table 5.19 shows the range, median, mean and standard deviation of twenty diagnosed patients with ATD divided into "mild", "moderate" and "severe" dementia (see above Chapter 5.2.2). Table 5.20 gives a cross sectional analysis of the 8 patients tested for three years reclassified by severity. Table 5.21 describes the 20 patients with ATD graded by severity on initial assessment. The median values are profiled on Figure 5.16. Table 5.22 gives all test and retest scores graded by severity.

Table 5.19

ATD : CROSS SECTIONAL DATA

	MILD N = 9 Mean Age 60.3					MODERATE N = 8 Mean Age 63.6				SEVERE N = 3 Mean Age 57.0			
	Max	Range	Medn	Mean	S.D.	Range	Medn	Mean	S.D.	Range	Medn	Mean	S.D.
Rating Scale	40	21-39	28	28.8	5.9	15-20	20	20.9	3.1	7-11	8	9.0	2.7
Iden. Nam.	8	7-8	8	7.9	0.3	6-8	8	7.8	0.7	7-8	8	7.7	0.6
Iden. Funct.	8	8	8	8.0	0	6-8	8	7.6	0.7	4-8	8	6.7	2.3
Token T. A-E	67	65-67	67	64.4	0.9	15-65	60	54.1	17.0	30-55	33	40.0	13.9
Token T. F	96	74-96	92	88.7	7.9	45-90	70	71.2	16.6	35-65	45	48.7	14.8
Token T. Total	163	140-163	159	155.1	8.4	60-160	113	125.3	30.9	70-120	76	88.7	28.2
Sent. Rep.	22	10-15	12	12.0	1.7	9-13	12	11.9	1.6	6-10	7	7.7	2.1
Digit Rep.	14	4-8	6	5.8	1.3	3-7	5	4.9	1.3	2-6	7	4.0	2.0
Digit Rep. R	14	3-6	4	4.3	0.9	0-7	2	2.9	2.2	0	0	0	0
Auto. Speech	4	4	4	4.0	0	2-4	4	3.8	0.7	3	3	3.0	0
Con. Naming	16	15-16	16	15.7	0.5	1-16	14	12.6	4.9	8-9	8	8.3	0.6
Dest. Funct.	16	9	16	16.0	0	4-16	15	13.8	4.2	7-11	9	9.0	2.0
Tact. Nam. R	8	7-8	8	7.9	0.3	0-8	6	5.9	2.8	4-7	4	5.0	1.7
Tact. Nam. L	8	7-8	8	7.9	0.3	0-8	7	5.9	3.4	4-6	5	5.0	1.0
Gesture	16	16	16	16.0	0	12-16	16	15.4	1.4	13-16	16	15.0	1.7
Fluency	60	8-32	16	19.9	8.9	0-40	6	12.1	14.9	1-5	2	2.7	2.1
Sent. Constr.	25	9-25	20	19.2	5.4	0-24	13	13.2	9.5	0	0	0	0
Read Words	8	8	8	8.0	0	5-8	8	7.5	1.1	8	8	8.0	0
Word Recog.	8	8	8	8.0	0	3-8	8	7.4	1.8	6-8	6	6.7	1.2
Read Sent.	7	6-7	7	6.9	0.3	4-7	7	6.6	1.1	2-7	5	4.7	2.5
Sent. Comp.	32	22-32	32	29.4	3.8	5-30	26	24.1	9.2	8-18	12	12.7	5.0
Auto. Writ.	5	2-5	5	4.7	1.0	4-20	5	6.8	5.4	1-2	1	1.3	0.6
Spelling	24	13-24	23	21.2	3.7	2-24	22	18.0	8.3	0-11	9	6.7	5.9
Dictation	13	10-13	12	12.0	1.1	6-13	10	9.9	2.9	0-5	0	1.6	2.9
Copying	11	7-11	10	9.8	1.4	0-11	9	8.2	3.7	4-11	5	6.7	3.8
Calculation	10	4-10	8	7.9	2.2	0-10	7	6.1	3.2	0-2	1	1.0	1.0
Oral Aprax.	20	20	20	20.0	0	9-20	20	17.9	3.9	14-18	14	15.3	2.3
Ideom. Aprax.	20	20	20	20.0	0	14-20	18	17.6	2.6	13-17	17	15.7	2.3
Const. Aprax.	20	5-20	17	15.2	4.7	4-19	15	13.1	5.9	9-13	11	11.0	2.0
Block Design	48	0-40	8	12.9	11.3	0-30	12	11.1	10.8	0	0	0	0
RCPM	36	15-33	22	21.7	5.7	6-28	16	17.2	7.8	5-19	11	11.7	7

Table 5.21

GRADED CLASSIFICATION OF ATD:

INITIAL ASSESSMENT

MILD		MODERATE		SEVERE	
N = 9		N = 8		N = 3	
Digit Rep. R	.76**	RCPM	.69*	N: too small for significance	
Spelling	.63*				

Table 5.22

GRADED CLASSIFICATION OF ATD:

TOTAL TEST SCORES

MILD		MODERATE		SEVERE	
N = 13		N = 18		N = 8	
Sent. Rep.	.64**	Digit Rep. R	.63***	Token T. F	.89***
Sent. Constr.	.49*	Ideom. Aprax	.52**	Auto Speech	.87**
		Sent. Constr.	.49**	Word Recog.	.87**
		Fluency	.48**	Des. Funct	.85**
				Fluency	.79**
				Token T. Total	.78**
				Auto. Writ.	.75**
				Spelling	.75**
				Digit Rep. R	.73**
				Oral Aprax	.71**
				Gesture	.71**
				Sent. Constr.	.71**

For Tables 5.20, 5.21 and 5.22

* $p < .1$

** $p < .05$

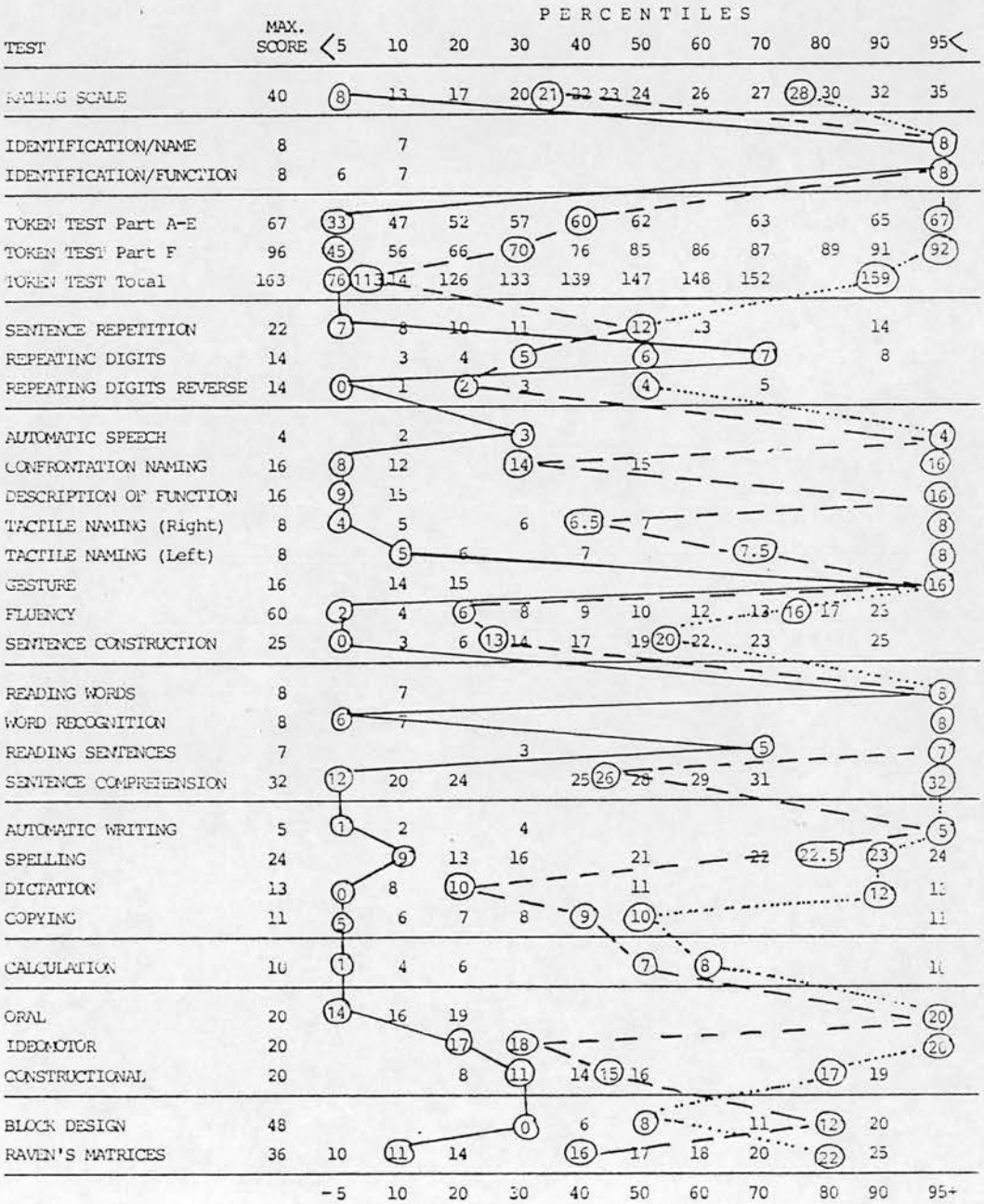
*** $p < .01$

Figure 5.16 CROSS SECTIONAL PROFILE : ATD

Mild (N = 9) .....

Moderate (N = 8) - - - - - - - - -

Severe (N = 3) _____



5.6.3. Discussion

Clinical impressions are that the presenting feature of ATD is a recent memory disturbance (Miller 1977, Roth 1980). The literature also suggests increasing aphasia, apraxia, and agnosia (Horenstein 1971). "Aphasic" disturbances are generally regarded as an impairment of semantics with late sparing of syntax and phonology (Bayles 1984).

Verbal and non verbal abilities described by the Language Scales suggest that memory in ATD is impaired when tested by Digit Repetition in Reverse. This task demands the registration of information, the holding of it, the manipulating of it and the recalling of it. Not only encoding stimuli but registration of the signal is affected. The argument that dements do not attend to the acoustic properties of a signal has been put forward by Miller (1971, 1972). Patients with ATD have difficulty repeating heard speech precisely, and this is reflected in dictation (see 5.6.2.9).

Digit Repetition Forward is not impaired in ATD but repeating sentences or grammatical word strings is affected in the mild stage of the disorder. Syntactic disturbance is reflected in the sentence construction task. ATD produces difficulty encoding speech grammatically but not because of a failure to recall the stimulus words. The belief that dementia does not impair syntax needs to be reviewed. It is possible that word order in sentences is disturbed cohesively rather than grammatically. Such an explanation would be in accord with concomitant memory disturbance.

Two linguistic transcoding processes are also impaired in mild dementia : coding phonemes into graphemes in written spelling tasks and coding written graphemes in copying tasks. Block Design is also impaired, it demands both visuo spatial constructional ability and sequential activity. This failure of sequencing may be reflected in ideational apraxia in dementia.

In moderate dementia there is a failure of memory, confirmed by reversed digit repetition tasks. There is also a failure of associational word fluency and another test of non verbal intelligence, the Coloured Matrices of Raven. There is evidence of an underlying failure of comprehension of written instructions by sentence complexity as well as the coding of words into syntax. Moreover ideomotor apraxia as shown in intransitive gesture may also reflect an underlying ideational apraxia.

In severe dementia there is widespread disruption of language systems with a loss of comprehension of spatial, temporal and logico grammatical complicated structures. Comprehension is not impaired because of the amount of information but because of its complexity. At sentence level the difficulty of sentence construction remains. There are also problems of word retrieval of verb forms rather than nouns, for verbs describethe abstract nature of objects. There is failure to derive meaning of written words although those words can be read. Finally, ideational apraxia is profound.

5.6.4 Longitudinal Analysis

The strength of a longitudinal analysis is that it describes which variables are more, or less, resistant to progressive ATD. Profiles of ATD patients in this study show individual rates and patterns of dysfunction. There is a trend of deterioration over neurolinguistic tasks and this trend is described in Table 5.23 and profiled in Figure 5.17. The significance of this trend has been described after Page (1963). Scores for the test retest paradigm were ranked and summed to give Page's "L" When N is equal to 8, three measures of significance are given:

$L = 104 \quad p < .05$

$L = 106 \quad p < .01$

$L = 109 \quad p < .001$

The language variables that diminished over the three years are given with these levels of significance below:

.001	Token Test
	Digit Repetition Reverse
	Fluency
	Constructional Apraxia
.01	Token Test Part F
	Tactile Naming (Right)
	Reading Comprehension
	Spelling
	Calculation
	Block Design
	RCPM
	Dictation
.05	Token Test A - E
	Digit Repetition
	Naming

Table 5.23

ATD : LONGITUDINAL DATA AND TREND

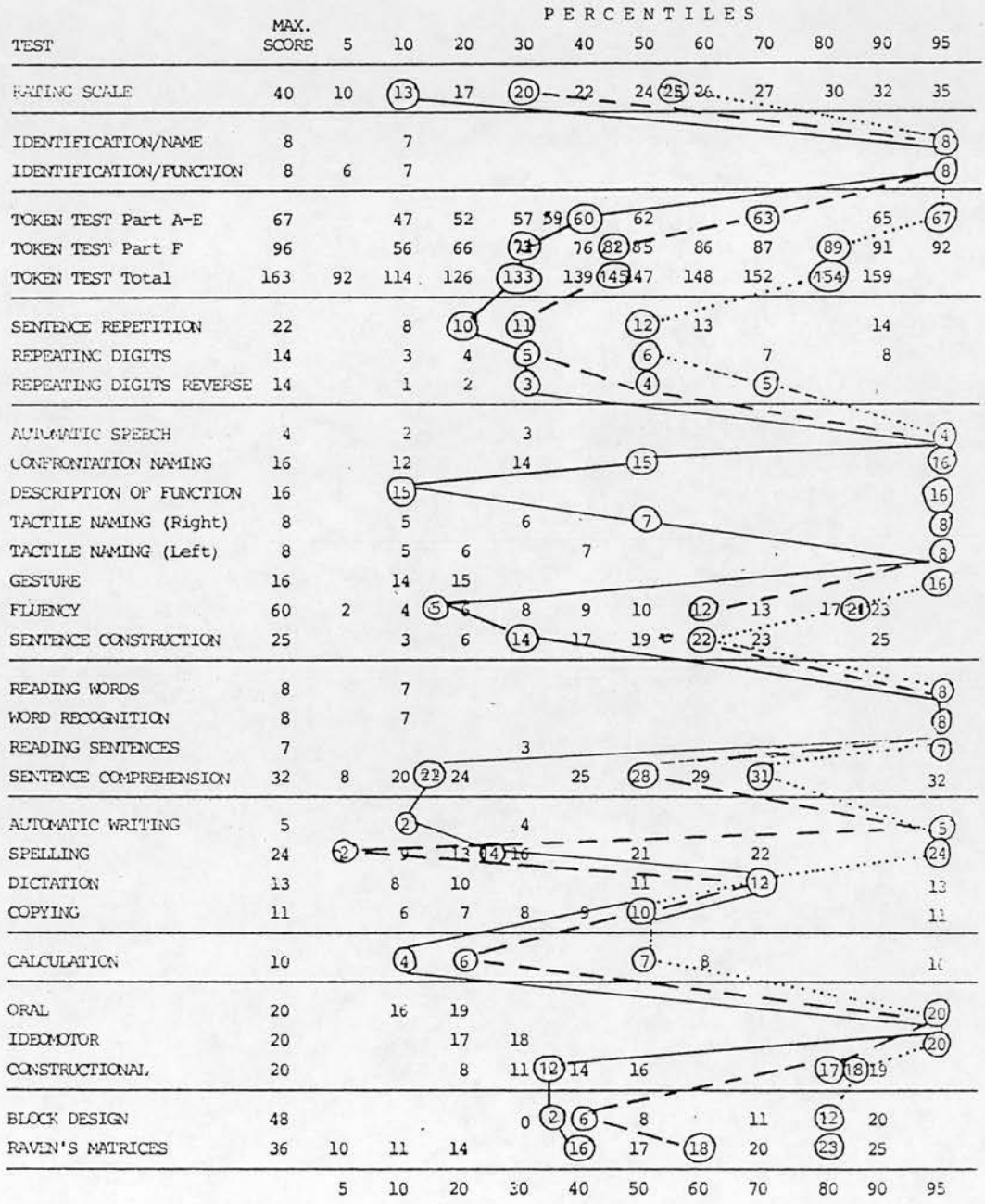
	Initial Assessment N = 8 Mean Age 60.3					Assessment after one Year N = 8 Mean Age 61.7				Assessment after two Years N = 8 Mean Age 62.7					
	Max	Range	Medn	Mean	S.D.	Range	Medn	Mean	S.D.	Range	Medn	Mean	S.D.	L*	p
Rating Scale	40	20-29	25	25.0	2.7	12-34	20	21.8	7.0	4-28	13	15.5	9.1	102	NS
Iden. Nam.	8	7-8	8	7.9	0.4	6-8	8	7.6	0.7	1-8	8	6.3	2.5	85	NS
Iden. Funct.	8	8	8	8.0	0	6-8	8	7.7	0.7	3-8	8	6.5	2.3	91	NS
Token T. A-E	67	50-63	67	64.1	5.9	35-65	63	59.9	10.9	25-63	60	53.0	16.7	104.5	.05
Token T. F	96	55-95	89	83.8	13.4	40-95	82	78.3	17.0	20-90	73	64.6	25.9	108.0	.01
Token T Total	163	100-158	154	147.9	18.9	75-160	145	138.1	27.5	40-153	133	117.6	41.9	110.1	.001
Sent. Rep.	22	9-13	12	11.5	1.5	9-12	11	10.9	1.1	5-14	10	10.3	2.9	102	NS
Digit Rep.	14	4-8	6	5.8	1.3	4-7	5	5.3	1.4	3-9	5	5.1	1.7	104.5	.05
Digit Rep. R	14	3-7	5	4.9	1.3	2-6	4	3.9	1.6	0-4	3	2.4	1.6	112	.001
Auto. Speech	4	4	4	4.0	0	3-4	4	3.9	0.4	0-4	4	3.1	1.5	101	NS
Con. Naming	16	1-16	16	13.9	5.2	1-16	16	13.9	5.1	1-16	15	12.3	5.4	105	.05
Dest. Funct.	16	4-16	16	14.5	4.2	3-16	16	14.1	4.6	0-16	15	12.9	5.4	103	NS
Tact. Nam. R	8	0-8	8	7.0	2.8	1-8	8	7.1	2.5	0-8	7	5.8	2.8	108	.01
Tact. Nam. L	8	0-8	8	6.9	2.8	0-8	8	7.0	2.8	0-8	8	6.0	3.1	98.5	NS
Gesture	16	15-16	16	16.1	0.8	13-16	16	15.6	1.1	6-16	16	14.8	3.5	99.5	NS
Flueecy	60	0-40	21	20.1	13.2	0-35	12	13.6	10.4	0-30	5	8.0	9.9	108.5	.001
Sent. Constr.	25	9-25	22	19.0	6.4	0-24	22	18.5	8.5	0-24	14	11.9	10.9	101.5	NS
Read Word	8	5-8	8	7.6	1.1	4-8	8	7.5	1.4	0-8	8	7.0	2.8	98	NS
Word Recog.	8	8	8	8.0	0	6-8	8	7.5	0.7	0-8	8	6.0	3.4	101	NS
Read Sent.	7	4-7	7	6.5	1.1	0-7	7	6.0	2.5	0-7	7	5.6	2.5	101.5	NS
Sent. Comp.	32	22-32	31	28.8	4.2	8-30	28	25.6	7.1	0-30	22	20.4	10.3	108	.01
Auto. Writ.	5	2-5	5	4.6	1.1	2-5	5	4.3	1.4	1-5	2	2.6	1.5	103.5	NS
Spelling	24	8-24	24	20.0	6.3	5-24	20	19.0	7.1	0-24	14	12.8	9.8	108	.01
Dictation	13	6-13	12	11.3	3.4	4-13	12	10.5	3.3	0-13	12	9.8	4.5	106.5	.01
Copying	11	7-11	10	10.0	1.3	8-11	10	9.5	1.2	0-11	10	8.1	4.2	101	NS
Calculation	10	4-10	7	7.3	2.1	1-9	6	5.6	3.0	0-8	4	3.6	2.9	107.5	.01
Oral Aprax.	20	16-20	20	19.5	1.4	11.20	20	18.9	3.2	9-20	20	17.5	4.7	99.5	NS
Ideom. Aprax.	20	14-20	20	19.2	2.1	7-20	20	18.1	4.6	8-20	20	16.9	5.2	102	NS
Constr. Aprax.	20	5-19	18	16.0	4.8	5-19	17	14.9	5.0	0-16	12	10.6	5.9	109	.001
Block Design	48	0-30	12	12.9	9.6	0-30	6	9.0	10.7	0-16	2	6.3	7.7	106	.01
RCPM	36	16-28	23	22.1	4.1	9-29	18	18.8	5.9	0-24	16	14.5	7.6	107	.01

* Ref. after Page (1963): See Chapter 5.6.4

Figure 5.17 LONGITUDINAL PROFILE : ATD

N = 8

YEAR I ..... YEAR II - - - - - YEAR III _____



The abilities that are most resistant to deterioration in progressive dementia are:

Identification of objects by name and function
Reading of single words
Left handed asteriognosis
Gesture
Oral apraxia
Automatic Speech

5.6.5. Discussion

The greatest diminuation in performance is in comprehension of instructions of complexity and length. Tasks that demand concentration and manipulation of abstract concepts such as word fluency, reverse digit span and complex visuo spatial manipulation and construction also show the most significant impairment in progressive ATD.

Less progressively impaired are comprehension of information that contains complex concepts whether they are presented verbally or graphically, orthography, dictation and tests of non verbal intelligence. Right hand tactile naming is also impaired.

The decline is less for tasks that demand memory as comprehension of instructions by length and digit recall, or the repeating of random words. The idea that loss of comprehension relates to the processing of concepts in instruction but not length of the instructions is confirmed by this differential progressive loss. Naming is also impaired.

Thus, although there is a general decline of most abilities tested in unremitting ATD the language variables that appear to deteriorate most are tasks that demand the processing of logical and spatial relationships, as well as written tasks and comprehension of verbal and written information. Abstract operations and phonemic-graphemic transcoding are also impaired. Results from Right and Left tactile naming tasks suggest hemispheric asymmetry.

These deficits are not dissimilar to language impairment produced by bilateral or left hemisphere temporo-parieto-occipital damage that affects grammatical instructions imbued with spatial or quasi-spatial significance and word meaning (Hier et al 1980).

5.6.6. The Dissociation of Abilities in Problem Solving

The relationship of visuo spatial and language abilities in tests of non verbal intelligence can be examined by the intercorrelation of verbal planning and graphic execution in Block Design and the RCPM.

Fluency may be regarded as a test of verbal divergent intelligence. It demands controlled word association with specific behavioural constraints. It has received particular investigation by neuro-physiologists who have been described its relation to functioning in the frontal lobe. The dorso lateral cortex of the left hemisphere is essential to the generation of word strings (Milner 1964). The left medial supplementary motor area has also been implicated in the task as it programmes language (Alexander and Schmitt 1980). Damage to the frontal areas affects verbal regulation of behaviour producing a dissociation of word and deed (Zangwell 1966): patients with dorso-lateral lesions divorce verbalisation from action.

In order to investigate the relationship of this task of verbal associative regulation and the RCPM as a test of non verbal reasoning, and the relationship between constructional abilities and Block Assembly, the four tasks were intercorrelated and the intercorrelations compared in mild, moderate and severe dementia.

In normals there is a strong relationship between language and visuo spatial ability in both the RCPM and Block Design. Language appears to be unrelated to design copying in normals (see Table 5.24).

Table 5.24

INTERCORRELATION MATRIX : NORMALS

N = 61

	Fluency	Constr. Apraxia	Block Design	RCPM
Fluency	1	.18	.37	.52
Constr. Apraxia		1	.40	.50
Block Design			1	.73
RCPM				1

$r_s = .27$ $p < .05$, $r_s = .35$ $p < .01$, $r_s = .44$ $p < .001$

In mild ATD the pattern of correlations is different. The language task correlates highly with Block Design while the visuo spatial task correlates at $p < 0.01$. Mild dements appear to be focussing language and visuo spatial ability on Block Design while losing perception of the RCPM as a visuo spatial problem (See Table 5.25).

Table 5.25

INTERCORRELATION MATRIX : MILD ATD

N = 13

	Fluency	Constr. Apraxia	Block Design	RCPM
Fluency	1	.66	.86	.73
Constr. Apraxia		1	.78	.51
Block Design			1	.88
RCPM				1

$r_s = .56$ $p < .05$, $r_s = .70$ $p < .01$, $r_s = .79$ $p < .001$

In moderate ATD the language components in the solution of Block Design are lost, the task is seen as a visuo spatial pattern, it is not seen as a problem. The Matrices similarly appear to be considered as a stimulus of patterns rather than a problem for verbal reasoning. However, in both mild and moderate ATD patients are using language in drawing tasks at .05. (See Table 5.26).

Table 5.26

INTERCORRELATION MATRIX : MODERATE ATD

N = 18

	Fluency	Constr. Apraxia	Block Design	RCPM
Fluency	1	.49	.29	.66
Constr. Apraxia		1	.80	.72
Block Design			1	.69
RCPM				1

$r_s = .47$ $p < .05$, $r_s = .60$ $p < .01$, $r_s = .69$ $p < .001$

In severe ATD the dissociation is complete. Block Design is regarded spatially and the Matrices considered verbally but there is no relationship between the abilities or non verbal tests.

Table 5.27

INTERCORRELATION MATRIX : SEVERE ATD

N = 8

	Fluency	Constr. Apraxia	Block Design	RCPM
Fluency	1	.21	.06	.72
Constr. Apraxia		1	.81	.35
Block Design			1	.24
RCPM				1

$r_s = .71$ $p < .05$, $r_s = .88$ $p < .01$, $r_s = .95$ $p < .001$

5.6.6.1 Discussion

The observations of dissociation of language from spatial abilities gives force to the findings of Ingvar et al (1975). Given two neuropsychological tests patients with ATD do not act normally in that in reasoning tests they do not see the problem - there is greater activation of the primary areas of the cortex rather than the association cortex as patients focus on the stimulus and not on the puzzle it presents (Ingvar et al 1975). Further, it appears that a dissociation occurs between approaches to problem solving in this case, language was continually compromised in Block Design and visuo spatial perceptions continually compromised in the Raven's Matrices.

5.6.7 Conclusions

ATD can be described cross-sectionally, in terms of severity, or longitudinally, in terms of its natural history. Symptoms that present clinically are similar to the deficits that are found statistically. Characteristics noted in cross-sectional studies are not always those observed in longitudinal ones although the overall pattern appears consistent.

In mild, moderate and severe stages, failures that are seen statistically are more precise but reflect the general abilities whose contamination impairs the activities of daily living. Thus patients who present memory loss do so because the verbal trace is not registered, the difficulty is not just of decoding audio verbal information but decoding it precisely. A failure of the ability to complete an everyday sequence of tasks is reflected in tests like Block Design that demand planning, executing and monitoring of behaviour as well as sub vocal regulation of that behaviour.

Memory is diminished when measured by tests that demand short term encoding rather than repetition of unrelated words. Syntactic encoding deficits are therefore not related to a failure to hold the stimulus words, but a failure to order them grammatically.

There is an increasing difficulty of word retrieval. Retrieval of verb forms which are more abstract, being concepts of the words they refer to, is more difficult than retrieval of words as labels. There is no evidence that the deficit of naming is a disorder of

perception or registration rather than lexical retrieval and selection: verbal agnosia is not a prominent feature of ATD and moreover patients circumlocute when they cannot retrieve a specific name.

Complex tasks that demand integration of several abilities and skills are progressively impaired. There is a steady decline of the ability to transcode from one modality to another: to read and understand a word or to interpret phonemes into graphemes.

There is a rapid dissociation of verbal abilities when problem solving.

Although aspects of verbal encoding and decoding appear as deficits in all stages of ATD, the progressive history is one of loss of comprehension, memory, verbal associative and visuo spatial abilities, while basic perception of words or sequences of learned non linguistic engrams and automaticisms is less impaired. ATD's progression is unremitting and the linguistic and cognitive disabilities than accompany it may be more profound than the disorders that accompany associated diseases and normal ageing.

5.6.8 Language Pathology in ATD and Associated Disorders

The linguistic and cognitive disorders of ATD may compare with symptoms of disease processes producing similar deficits or from similar lesion sites. To investigate this possibility thirteen variables that declined significantly in ATD were compared to scores produced in Korsakoff Psychosis, multi infarct dementia, temporal lobe epilepsy, transcortical aphasia and healthy controls. The reason for these comparisons is given below.

5.6 8.1 Korsakoff Psychosis

Korsakoff syndrome reflects disturbance of memory. Comparisons have been made between bilateral lesions of the hippocampus and Korsakoff syndrome producing memory deficits. Corsellis (1970) predicted that bilateral hippocampal lesions in Korsakoff Psychosis would produce a memory disturbance matched by ATD.

The clinical delineation of Korsakoff syndrome is still unclear but Walsh (1985) summarises the signs:

(1) Anterograde amnesia or the inability to acquire verbal and non verbal material distinguishing the condition from unilateral temporal lobe lesions

(2) Difficulty of recall of prior events, information decreasing as questions move closer in time to the present.

(3) Preserved audioverbal memory

(4) No deficits of speech, language or gesture

(5) Confabulation as necessary but not a consistent feature

(6) Frequent lack of initiative and sponteneity with blunting of affect

In Korsakoff syndrome the mammillary bodies are affected (Harper 1979) and there are further lesions to the hypothalamus, brain stem, cerebellum, frontal areas and the dorsal and medial nuclei of the thalamus (Victor 1976).

5.6.8.2 Multiple Infarct Dementia

Vascular or multi infarct dementia may be likened to a series of small strokes accompanying hypertension in extra cerebral vascular disease. No studies report the difference between ATD and MID on "language measures". Perez et al (1975) and Fuld (1983) have suggested a difference on measures of verbal intelligence. Bayles (1984) has commented that fluency and relevancy are impaired in dementia but notes precise differences between ATD and MID on these skills are not documented. Stevens (1984), using the Whurr Aphasia Screening Test was not able to differentiate the two populations. Fuld (1983) has suggested MID ought to be more easily demonstrated by strong focal signs but that vascular dements with focal signs are few. On tests of intelligence it is reported that ATD patients do worse than those with MID, the verbal-performance discrepancy being greater (Fuld 1983).

5.6.8.3. Transcortical Aphasia

Several authors have noted ATD to present as anomic aphasia (Constantinidis et al 1978, Kertesz 1979 and Appell et al 1982) and transcortical aphasia, or aphasias resulting from lesions posterior to Wernicke's area (Nicholas et al 1985). There are no comparisons with speech in fluent aphasia that have withstood analysis (Nicholas et al 1985).

The aphasias that may bear comparison with language of ATD are transcortical aphasias, that demonstrate intact repetition but disturbance of comprehension, naming, dynamism and fluency. The condition was described by Geschwind et al (1968) as an isolation of the speech area. More recently Hier et al (1980) has described semantic aphasia following lesions of the posterior dominant cortex that has striking similarities with the language of ATD. Semantic aphasia although defined by Luria in 1970 has been neglected in research papers.

Thus four groups presenting language and cognitive deficit as memory disorder, global cognitive disorder, temporal lobe disorder and language disorder may compare to ATD which similarly presents disorders of memory, cognition and language.

To investigate the possibility that these disorders may mirror ATD, the thirteen variables that deteriorated significantly in ATD were cross compared. Table 5.28 gives the median scores and probability measures of a Mann Whitney U Multiple Comparison. Only six of the sixty five measures of the five groups over thirteen variables were lower than those of ATD. Four were in the MID group (Digit Repetition (R), RCPM, Naming Tasks) and two were in the transcortical aphasic group (Digit Repetition (Reverse) and Spelling). Since only six measures are worse than those for ATD, the language of ATD may be considered different from other disease processes that may be associated with it.

Table 5.28

MANN WHITNEY U MULTIPLE COMPARISONS

ATD AND ASSOCIATED DISORDERS

Test	ATD	Korsakoff Psychosis	MID	Trans-cortical Aphasia	Controls
	N = 20	N = 14	N = 12	N = 10	N = 14
Rating Scale	24	30	24	34.5	40
		.01		.001	.001
		ATD = MID < K/S < TCAP = CONT.			
Token T. Total	141.5	159	142	151	162
					.001
		ATD = MID = K/S = TCAP < CONTD.			
Digit Rep. R	3.5	5	3	3	6.5
		.01			.001
		ATD = MID = TCAP < K/S < CONT.			
Conf. Naming	15	16	14.5	16	16
		ATD = MID = TCAP = K/S = CONT.			
Tactile Nam. R	8	8	7.5	8	8
		ATD = MID = TCAP = K/S = CONT.			
Fluency	10	22.5	12	16.5	41
					.001
		ATD = MID = TCAP = K/S < CONT.			
Sent. Constr.	15	25	23.5	18	25
		.001			.001
		ATD = MID = TCAP < K/S = CONT.			
Reading Comp.	27.5	32	28	30.5	.001
		ATD = MID = K/S = TCAP CONT.			

Table 5.28 (Cont.)

MANN WHITNEY U MULTIPLE COMPARISONS (contd.)

ATD AND ASSOCIATED DISORDERS

	ATD	Korsakoff Psychosis	MID	Trans-cortical Aphasia	Controls
	N = 20	N = 14	N = 12	N = 10	N = 14
Spelling	21.5	24	23.5	21	24
					.01
	ATD = MID = K/S = TCAP < CONT.				
Calculation	7	10	8	9.5	10
					.001
	ATD = MID = TCAP < K/S < CONT.				
Constr. Aprax.	14.5	18	17	20	20
				.01	.001
	ATD = MID + K/S < TCAP < CONT.				
Block Design	8	14	11	18	28
					.001
	ATD = MID = K/S = TCAP < CONT.				
RCPM	19	23	17	29	30
				.01	.001
	ATD = MID = K/S < TCAP < CONT.				

In order to determine the significance of the differences the Mann Whitney U Multiple Comparisons was computed. The significance level was raised to $p < .01$ so that less than one score would be obtained by chance. Table 5.28 also gives the significance of the difference of medians of ATD to controls and the four groups with neorological disease.

5.6.8.4. Discussion

Patients with ATD are more impaired on all 13 subtests that detect the disease's linguistic decline with the exception of, in MID, 'Digit Repetition, RCPM, Confrontation Naming and Tactile Naming (Right) and, in transcortical aphasia, Digit Repetition (Reverse) and Spelling. Naming ability did not significantly differentiate any group showing that anomia can present with non specific widespread lesions.

No tests discriminated ATD and MID. Korsakoff patients produced significantly diminished scores on the Rating Scale and memory. Like ATD they also demonstrated failure of tests of verbal fluency and non verbal intelligence. All pathological groups have significantly reduced comprehension of verbal and written instructions. Aphasia following lesions beyond the peri Sylvan borders causes memory impairment as well as disorders of syntax and lexical access.

5.6.8.5. Conclusions

A comparison of ATD with disorders that may mirror it psychologically, linguistically, cognitively and anatomically demonstrates that it differs in severity to all those disorders and it cannot be differentiated on those abilities that show progressive decline in ATD from MID.

CHAPTER 6

CONCLUSIONS, RECOMMENDATIONS AND REFLECTIONS

CONCLUSIONS

Communication is impaired in dementia. It is reduced linguistically and paralinguistically because of the general flattening of affect, impoverishment of thought and disorder of personality that accompanies the disease. It is associated with hospitalisation and mortality.

Dysphonia and dysarthria are described only in subcortical dementias (Parker 1985, Scott et al 1985).

Inner speech is impaired in early ATD and demonstrated by the failure of non verbal intelligence and problem solving (Bayles 1985). Comprehension is impaired through a reduced ability to register acoustic signals, a failure of auditory memory, and an inability to decode lexical and grammatical information. Expressively the language of dementia has comparatively preserved phonology and syntax but the semantic system is selectively impaired either because the lexical store is impoverished or because it can no longer be accessed (Nebes 1985).

Language tasks have been used to predict and diagnose ATD (Folstein and Breitner 1981, Bayles and Boone 1982). Language has been used to investigate the onset and management of dementia in both deep and screening measures (Isaacs and Kennie 1973, Gibson and Kendrick 1979). Language has been used to index premorbid intelligence (Nelson and O'Connell 1978) and diminished flexibility of thought in mild dementia (Stroop 1935, Henderson and Huppert 1984).

The literature asks whether language can diagnose dementia, whether demented language is aphasic, what is the relationship between normal language and that of mild dementia and what is the nature of language disorders in dementias with different underlying causes.

This investigation stated three hypotheses in its introduction:

Hypothesis 1 That aphasia is not a feature of language in dementia.

Specific neurological information about the diseases that produce aphasia and dementia is known: that the neuropathology differs in being focal or diffuse.

The natural history of the two disorders also differs. Dementia is a progressive and degenerative disorder but a test-retest paradigm in this study has demonstrated a trend to improvement in aphasic patients. There was a weak improvement of auditory comprehension, confrontation naming, sentence construction and calculation ($p < 0.1$). Fluency, sentence and digit repetition also improved ($p < 0.05$) as did comprehension of specific concepts when the memory load is held constant ($p < 0.02$).

If aphasic and demented language were similar then a factor analysis would produce a similar grouping of abilities with similar loading. These would align when ranked. In this study dementia yielded seven factors:

1. Visuo construction
2. Writing
3. Recognition
4. Naming
5. Comprehension
6. Reading
7 Motor-gesture

Aphasia yielded six factors:

1. Naming
2. Fluency
3. Visuo construction
4. Comprehension
5. Recognition
6. Reading

The first factor reflects task difficulty and complexity. In dementia these are of "intelligence", constructional apraxia, memory and calculation. In aphasia the first factor includes confrontation naming, tactile naming, verb retrieval, sentence construction, repetition, reading and writing ability. The percentage variance for the first factor in each disorder was similar.

Ranking of variables by their loading confirms the neurolinguistic dissimilarity of aphasia as a disturbance of semantics, fluency and grammar while demented language reflects a disruption of cognition. Whatever we may call the language of the demented it is not aphasia.

Hypothesis 2. That language pathology differs according to the diseases that cause it.

This begs a question that language can differentially diagnose disease processes from normal ageing.

The Language Scales devised for this investigation explore all communicative modalities, contain graded tasks and demonstrate internal consistency and compatability of scores. These reflect its face content validity and value. The Scales can predict demented from aphasic and normal language. Twentyone variables differentiated aphasics from controls, the two most powerful were semantic and syntactic measures. They correctly classified 90.6% of cases as aphasic or normal and 86.5% of cases as aphasic or demented. No controls were misclassified as demented but 13.6% of the dements yielded scores within normal limits. These were reclassified by Dementia Rating Scale, which has great discriminatory power (Bayles 1985), or language tests including confrontation naming, tactile naming, spelling, constructional apraxia and the RCPM. Controls in this study were carefully screened for the absence of neurological disease but a proportion of the senescent will contain a "grey area" of mild dements: Walker (1982) also found 14.6% of her population could be reclassified.

Some language skills do deteriorate with normal age change. The Language Scales were constructed to be as far as possible culturally neutral and unaffected by socio-economic background or educational opportunity. However, age correlates negatively with digit recall for both forward and reverse operation, comprehension of instructions by increased memory load (Token Test A-E), word search (Confrontation Naming, Word Fluency) and oral agility. More significantly ($p < 0.01$) correlations occur with spelling but the most significant correlations ($p < 0.001$) with age are the comprehension of complex concepts (Token Test Part F), Sentence Repetition, constructional abilities, Block Design and the RCPM. These observations are confirmed in the literature (Schow et al 1978, Wertz et al 1971, Vargo and Black 1984 and the normative data of Weschler 1958 and Raven 1977).

If normal ageing produces specific cognitive changes so do many diseases. Like ATD, Korsakoff Psychosis presents a memory disorder, MID presents a cognitive one, transcortical aphasia presents pathological language from lesions beyond the classical language zone. A Mann Whitney U Multiple Comparison between healthy controls and these groups, on 13 subtests that showed progressive and significant impairment in ATD, demonstrated Confrontation Naming and Tactile Naming (Right) produced no significant difference between groups. The other 11 variables showed variations of impairment according to diagnosis. Three (Token Test Total, Fluency and Spelling) produced patterns significantly different to normal ageing but of 65 correlations only 6 were worse than ATD. Two correlations were specific to transcortical aphasia (Digit Repetition (Reverse) and

Spelling) and 4 were specific to MID (Digit Repetition (Reverse), RCPM, Confrontation Naming and Tactile Naming (Right)). The pattern of disturbance of language differs between disease groups and controls but there is no real difference between the two dementias. On this group data and on these selective tests language tasks do not therefore differentiate between the major dementias.

Hypothesis 3. That language will deteriorate with progressive dementia.

ATD is progressive and its progress may be different to other dementias. The progression can be described both cross-sectionally and longitudinally.

For cross-sectional comparisons ATD has been divided according to the distribution of scores on the Dementia Rating Scale (Blessed et al 1968).

"Mild" dementia shows significant disturbance ($p < 0.1$) of Block Design, Spelling and Sentence Construction and a more significant disturbance ($p < 0.05$) of Copying, Digit Repetition and Sentence Repetition. In "Moderate" dementia Digit Repetition (Reverse) is most significantly impaired ($p < 0.05$) along with Sentence Construction and comprehension of written information as well as abstract word search (Word Fluency) and Ideomotor Apraxia. Less significantly impaired ($p < 0.1$) is the RCPM. In "severe" dementia language is more broadly impaired with a reduced ability to comprehend spatial, temporal and logico grammatical concepts ($p < 0.01$), Phoneme grapheme conversion, ideational loss, automaticisms, abstract word search and impaired reading comprehension are significant at $p < 0.05$.

In the progression from mild to severe dementia cognitive and linguistic abilities are selectively impaired. To investigate this a measure of verbal language (Word Fluency, or Thurstone's /w/ factor) and a test of visuo spatial drawing ability were compared to the RCPM and Block Design that demand sub vocal analogy, planning, monitoring and construction. Normals do not use language to aid design copying but they do use it, powerfully, along with visuo spatial abilities to solve the RCPM. They also use language and visual spatial ability to solve Block Design. In short they think verbally and think spatially. In ATD language initially becomes a tool in the drawing of patterns but it is gradually disconnected from spatial operation. It is increasingly compromised in Block Design and that task is eventually considered as no more than a series of patterns. Conversely visuo spatial analysis is continually compromised in tests of sub vocal reasoning. However dements may think to solve problems, interactive verbal and spatial thought is progressively dissociated.

To investigate the natural history of ATD 8 patients were tested for three consecutive years. The patterns of language breakdown were individual. Two patients deteriorated from normal limits to be untestable, 1 demonstrated pathological language as a presenting sign and remained impaired, 3 were tested into a fourth year and then suddenly showed accelerated deterioration and 2 demonstrated a gradual but incomplete deterioration. In spite of these differences a trend emerged. Least significantly impaired ($p < 0.05$) were comprehension of sentence length that increased memory load (Token Test A-E), Digit Repetition (Forward) and Confrontation Naming. More significantly

impaired ($p < 0.01$) were a specific measure of comprehension of sentence complexity (Token Test Part F), Tactile Naming (Right), comprehension of written instructions (Sentence Comprehension), Spelling, Dictation, Calculation, Block design and the RCPM. Most significantly impaired ($p < 0.001$) were a total measure of comprehension (Token Test Total), Digit Repetition (Reverse), Word Fluency and constructional ability.

Certain abilities are resistant to the progressive disease. They include automaticisms which are motor stereotypes and non linguistic, identification of single words that are presented verbally or in writing, Tactile Naming (Left), the gesture of objects and oral praxis. These tasks represent information stored as non linguistic engrams, reflect asymmetrical hemispheric involvement or preservation of cortical areas less compromised in Alzheimer's disease.

Some observations can be made from this investigation on opinions expressed in the literature. Constantinidis (1977) noted that language is more severely impaired in presenile forms of ATD. This study found no correlation between age and the thirty variables tested, all correlations were with severity of dementia. This confirms the observations of Hagberg and Ingvar (1976), Appell et al (1982) and Blackburn and Tyrer (1985).

There is evidence that changes that produce ATD may be bilateral but asymmetrical (Foster et al 1984, Haxby 1985). This study found asteriognosis of the right hand more severely impaired than that of the left suggesting Geschwind's (1965) disconnection syndrome.

Concepts of "fluid" and "crystalline" intelligence (Cattell 1943) may be valid for the investigation of language in dementia. Word Fluency, that demands an active search for words with abstract constraints, is more significantly impaired ($p < 0.001$) than confrontation naming or labelling ($p < 0.05$).

Although Alzheimer's disease spares the primary visual and auditory areas patients with ATD misperceive visual stimuli and so make errors on picture discription (Stevens 1985) and copying. They also misperceive auditory stimuli; the errors they make when writing reflect not neologism but approximations.

Observations that verbal language is retained rather than performance abilities (Roth and Hopkins 1953, Ron et al 1979) depend upon how language is interpreted. Performance abilities may fail, not only because of visuo spatial deficits but because language fails to monitor planning and motor acts. In dementia verbal abilities are seriously and significantly impaired but it is the verbal abilities of inner language and comprehension that most acutely reflect diminished language performance in mild disturbance. The complex interaction of cognitive abilities in problem solving is affected by selective dissociation.

The observation that phonology is unimpaired and syntax relatively unimpaired to semantics appears to hold true. The grammar of dementia may be impaired in terms of cohesion. The task of Sentence Construction is impaired throughout ATD. This may be an aspect of the task's demanding retrieval of stimuli from working memory or it may reflect syntax disturbance in the relationship of words and phrases.

Finally, Alzheimer's comments were pertinent for the language of the disorder that bears his name. There is a failure of naming, perseveration in writing, single syllable reading, circumlocution, paraphasia, ideational apraxia and a devastating loss of comprehension.

RECOMMENDATIONS AND IMPLICATIONS

This investigation has isolated specific areas that need further investigation in future studies.

1. The possibility that failure of comprehension may be attributed to defective registration of acoustic information lends itself to the science of experimental phonetics and speech perception. Instrumentation in the investigation of psychology by physiological measures has thus far confirmed classical theories of localisation and the dynamic functioning of the brain. Correlations of event related potentials to neurolinguistic acts remain speculative in terms of whether they reflect neural generators of the potential or the latency of stimulus processing. In all, physiological correlates of precise psychological tasks have been disappointing because added abilities are needed to react to the stimulus: the imageing of a pure mental event is very rare indeed. What is needed are more precise tests and greater precision of instrumentalisation to define cognitive processing. Instrumental measures of acoustic and verbal signals may clarify how those signals are registered, perceived and coded. Similarly, the use of electromyography may measure subvocalisation in comprehension and problem solving in dementia after the procedures of McGuigan (1970), Sokolov (1972) and Code and Ball (1984).

2. A grammatical analysis of speech samples of the language of the demented, using a similar procedure to that employed by Morice and Ingram (1982) with schizophrenic speech, would clarify the nature of any syntactic disturbance in dementia. This study has investigated language acts and not language usage or pragmatics.

3. Cohesion in language in dementia needs to be investigated using a similar procedure to that employed by Halliday and Hasan (1976) and Rochester and Martin (1979). The possibility that there is a lack of cohesion in the language of the demented is indicated in this study by the strong relationship between controlled fluency and memory impairment to sentence construction.

4. This study has been principally concerned with language and neurological dysfunction, tracing those abilities that progressively diminish in the progress of Alzheimer's Disease. Having mapped such deficits a new direction in psycholinguistics may clarify how those deficits affect communicative competence in psychiatrically impaired adults.

Some implications of the evaluation of progressive failure of aspects of language and non verbal intelligence in dementia in general and ATD in particular need to be discussed. This work has standardised linguistic acts, differentiated language pathology in global and diffuse lesions, mapped the breakdown of interrelated abilities and cross compared ATD with diseases that may mimic it.

Some authors have argued that language is impaired in dementia from onset with an inability to complete tasks of verbal intelligence, that is to judge language or verbal similarities and differences, to later stages of the disorder with impaired comprehension and expression.

These observations are verified experimentally. In mild dementia there is a failure to complete tasks of verbal reasoning or complete tasks that involve the solution of problems that demand ability to think both verbally and spatially, such as the skill brought to map reading and street direction. Two other tasks that also demand abstract spatial and verbal thought are also impaired in early dementia, calculation and the comprehension of complex grammatical structures. Auditory memory is reduced as is patient orientation, especially of time and place. The ability to recall non personal events that are not specifically related to patient's lives are also reduced.

As the disease progresses into moderate stages simple arithmetical problems and the ability to use money is lost, directional sense is impaired and patients tend to wander unaware of left and right orientation. Concentration is limited and there is a failure to understand instructions be they spoken or written. Spoken language begins to be rambling, without focus and the names of things or people that are particularly difficult are avoided, substituted or talked around. This loss of names, or anomia, which are the hooks on which we hand discourse, leads to empty speech filled with vague substitutions as 'thingme, what's its name and you know whose'.

In severe dementia language is in rapid decline. Patients are disorientated in place, unaware of relatives and carers and even themselves. They are inattentive, querilous but not always tearful. They understand by context and learned patterns and stereotyped behaviours. Language becomes fragmented into shortened clauses that do not relate to each other and may be meaningful only in unreal situations, so institutionalised patients will go shopping up and down a closed ward and return in their mind to the place where they were born. Indeed only birth places and dates may be retained as information that is over rehearsed and changeless. Comprehension is lost, language becomes a repetition of stereotypes, and wider aspects of communication are dampened with loss of volume and inflection in speech and loss of dynamism and interaction in behaviour. This is often combined with the disturbance of intention of purposeful movement in walking, eating, dressing or sequential actions. These gait apraxias and dressing apraxias are commonly observed in late stage dementia.

These features are mirrored by the ongoing progression of the disorder. Patients present to neurology wards with memory loss and ideational loss that appears to be forgetfulness or a disturbance of concentration. In the history of the disorder the ability to name which leads to empty speech is significantly impaired. More significantly impaired is the ability to understand written and spoken instructions and the ability to think and verbalise lucidly and flexibly. Most significantly impaired is the ability to understand everyday conversation with its complex spatial, temporal and logico grammatical concepts. This comprehension is further impaired by the difficulty of holding information in memory and acting on it appropriately.

So although dementia may be a disturbance of memory, personality and intelligence, we use language to reflect ourselves and define our needs. It is because of the awareness of the subtlety of language not just in communication but thought as well, that we need to pay greater attention to the role of language in dementing illness.

Language is a behaviour and, like many behaviours that are impaired in dementia it may be managed, indeed carers are rich in anecdotal suggestions from their experience. One clear example of language management is the work done in psuchogeriatric units where careful labelling, pictograms and coloured trails appear to assist the demented patient in the understanding of environment.

The study of language and behaviour is the science of psycholinguistics and the value of this neurolinguistic study is that it points the way to disease effects on language acts to language usage. Failure on the Token Test and memory subtests indicates the need to examine normal and abnormal ageing and the loss of comprehension. Cohen (1979) has suggested a review of the concept of crystalline abilities because of the failure of the elderly to infer, detect anomalies and get the "gist" of information. There is clear evidence of a significant loss of meaning at semantic level and a loss of language usage with a failure of cohesion or linkage both at inter and intra sentence level. This failure of cohesion may be responsible for syntactic errors rather than paragrammaticism.

The basis of comprehension disorder needs to be investigated. Loss of understanding of instructions relates to the length of utterance and, more significantly, the complexity of utterance. The literature reports phonology is generally spared, however the dysgraphic errors in ATD appear to reflect a breakdown in transcoding phonemic to graphemic features. Often it appears patients with ATD mishear and only approximate verbal information when they comprehend. This misperception of stimuli in one modality may be matched by misperception in another. Stevens (1984, 1985) has reported visual misperception in patients with ATD on picture description tasks. Speech perception and misperception has only recently been addressed at the Speech Science and Technology Conference, Canberra 1986.

Furthermore, failure of comprehension may reflect the loss of ability to register concurrently surface structure and meaning. Thus the Token Test Parts A-E are significantly impaired on operations of instructions by length where redundancy is controlled, but a greater failure exists with information demanding comprehension and manipulation of linguistic structures. The work of Cohen et al (1979) has identified loss of comprehension for unstated facts, true-false absurdities, anomalies and logical aspects in comprehension of text. Cohen and Faulkner (1981) have examined the loss of memory for surface structure in discourse.

Failure of monitoring is evident when there is a loss of cohesion in the discourse of the demented. It appears syntax is well preserved in the disease, and a syntactic filter may operate independently of cognition in patients who can correctly repeat sentences with errors. However, failure of cohesion in the speaker leaves the listner with

the task of comprehending and reflects the insensitivity of the speaker to a listner. Loss of cohesion in normal speakers may presuppose listner knowledge while in moderate dementia, where the degree of insight is reduced, may reflect an attempt by the speaker to camouflage memory failure by jargon, confabulation or circumlocution which is reported in the qualititive data on naming errors.

Specific deficits of language also need further investigation. Naming for example, is significantly impaired in ATD, the failure of word retrieval producing fluent "empty" speech. Memory for words may be regarded as "semantic" and memory for events as "episodic" knowledge. Deficits in one memory does not mean the other is similarly impaired. In ATD the ability to recognise word associations is retained suggesting that failure of naming may not be a deficit of semantic memory but access to it (Weingartner et al 1981).

Language and communication ability exist in social context and there is further need to investigate the failure of paralinguistic phenomena particularly in the institutionalised demented. This paralinguistic phenomena is an aspect of pragmatics; the relationship of language and behaviour in context. With the progressive loss of awareness of the regulators of communicative behaviour as gaze phenomena, kinesics and proxemics, situation and emotional regulation of language acts is compromised in turn taking, role and co-operation.

Thus the stage beyond this neurolinguistic study is to investigate how neurolinguistic deficits affect communication and behaviour. It lies in behavioural information that management is possible.

Can the language of dementia be managed? This investigation has primarily investigated the abilities that are vulnerable to change and the passage of those abilities in ATD. Those changes are unlike aphasia and strategies for the management of aphasia are inappropriate to the management of dementia. Language pathology needs to be identified, measured and subjected to ongoing evaluation. Thus the importance of standardised data on a format that provides easy reference to a broad range of language skills.

"Dementia" is a word that describes the behavioural effects of a disease process and as such may be managed behaviourally. Many skills are resistant to dementia and may provide the basis for reconsideration and relearning. Continence can be retrained in dementia for incontinence is the loss of a social skill not of neurological dysfunction in the demented (Malone-Lee 1986). There is no reason why the breakdown of language as a social act cannot be similarly managed.

Miller (1977) has reviewed management areas for the demented that include behaviour modification to reinforce communication, ergonomics to adapt the environment to patient's deficits, reality orientation and "normalisation" of giving patients goals that are as near to

reality even in institutionalised settings.

Pearce (1984), while holding out hope for pharmacological management of dementia, has noted that thus far only short term improvements have been demonstrated. There is however evidence that dements are sensitive to environmental change and Lindsley (1964) has suggested a theraputic or a "prosthenic environment". There have been reports of the need to change background stimulation to reduce disorientation (Cameron 1941), to rearrange seating for effective communication (Sommer and Ross 1958), occupational therapy (Cosin et al 1958), psychotheraputic discussion groups (Bowers et al 1967), the use of reinforcers in communication (Mueller and Atlas 1972), reality orientation therapy (Folsom 1983) and increased recreational activities (Bower 1967).

Studies of comparative language therapy have not been reported because of a lack of information about the progressive breakdown of language in dementia. Thus the use of resistant abilities to help deteriorating ones has not been explored. Unlike stroke rehabilitation the basic failure of memory and learning may hinder language therapy in dementia. However, knowing the patterns of the breakdown of language in dementia may allow professionals and the carers to modify verbal input to patients to improve their comprehension and ability to function.

Certain measures do appear more durable, dements in this study were able to recall personal histories better than non personal information. This comparative sparing of aspects of memory suggests the possibility of using reminiscence as a basis for language stimulation. There

are implications for early intervention where the recollections of one's life may be used to make the present more real. Haycox (1983) has commented "as the older person recounts stories of the past he tends to become again what he once was".

Studies in word retrieval indicate the patency of memory for words but a failure to retrieve them, patients in this study had difficulty recalling the names of objects they could identify. Theraputic strategies as "forced alternatives" may exploit this, the technique being the training of word recall by giving a correct alternative and a foil. Relearning is achieved by recognition rather than generation when semantic fields are blurred and produce paraphasia and circumlocution.

The demands of social expectations may be understood in specific deficits. Complex operations are hindered by a loss of ideation or the ability to sequence complicated activities. Ideational apraxia and neglect can produce spatial disorientation, dressing apraxia and reduced functioning in daily living, as in eating and grooming by ideomotor apraxia.

The role of communication and speech therapists is that of the role of councillor in the management of patients. Therapists may act as a resource in the psychotheraputic interchange and the environmental manipulation which allows demented patients the maximum of meaningful activity, communication and independence.

This study has provided a reliable examination for the investigation of the failure of language and cognition in aphasia and dementia. It

has also described the progressive breakdown of linguistic abilities in chronic brain failure. Further investigation by speech scientists should explore, more specifically, the nature of deficits with greater precision of testing. It is hoped that such further studies will investigate linguistic performance in the behavioural and medical management of the demented. It is also hoped that investigative techniques that describe the dynamic metabolism and functioning will be linked to language and so provide an understanding of the linguistic differences of the normal and abnormal brain.

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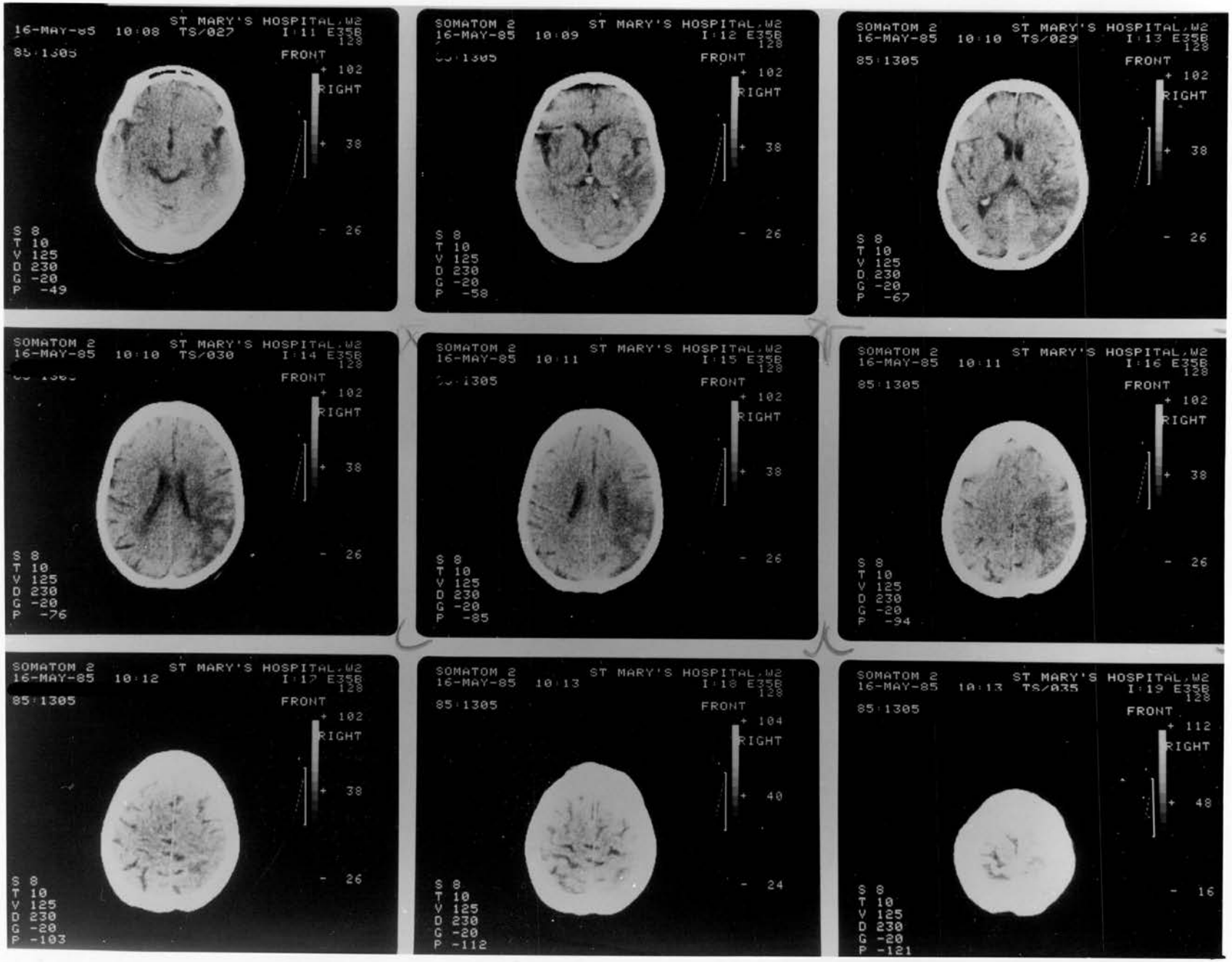
APPENDIX I

PROFILES

SUBTEST SUMMARY PROFILE (APHASIA)

NAME No. 1 DATE.....24.6.85.
SEX Female
D of B Age 77 years
DIAGNOSIS Right Hemisphere C.V.A.

TEST	MAX. SCORE	PERCENTILES										
		<5	10	20	30	40	50	60	70	80	90	95<
RATING SCALE	40											40
IDENTIFICATION/NAME	8	4	5	7								(8)
IDENTIFICATION/FUNCTION	8	4	5	7								(8)
TOKEN TEST Part A-E	67	2	5	12	20	30	45	52	60	62	(66)	67
TOKEN TEST Part F	96		0	20	35	50	61	65	67	75	85	(87) 96
TOKEN TEST Total	163	0	11	16	65	85	106	110	130	140	150	(153) 160
SENTENCE REPETITION	22			0	1	4	8	9	10	(12)	13	16
REPEATING DIGITS	14			0	1	2	3	4	5	6	7	(8) 9
REPEATING DIGITS REVERSE	14						1		2	(3)	4	7
AUTOMATIC SPEECH	4		0		2							(4)
CONFRONTATION NAMING	16	0	1	4	6	9	12	13	15			(16)
DESCRIPTION OF FUNCTION	16		0	3	7	9	13	14	15			(16)
TACTILE NAMING (Right)	8						0	2	4	6	7	(8)
TACTILE NAMING (Left)	8		1	2	3	5	6	7				(8)
GESTURE	16	1	12	13		15						(16)
FLUENCY	60					0	2	3	5	10	(12) 26	32
SENTENCE CONSTRUCTION	25							0	14	16	20	(25)
READING WORDS	8				1							(8)
WORD RECOGNITION	8	6	7									(8)
READING SENTENCES	7						3		6			(7)
SENTENCE COMPREHENSION	32		2	5	20	23	26	27	29	30	31	(32)
AUTOMATIC WRITING	5		0				3	4				(5)
SPELLING	24					0	3	14	18	19	22	(24)
DICTATION	13					0	4	9	11			(13)
COPYING	11		2	8	9	(10)						11
CALCULATION	10			1		3	5	6	(7)			10
ORAL	20	10		14	15	16	18		19			(20)
IDEOMOTOR	20	12	15	17								(20)
CONSTRUCTIONAL	20	(17)	18		19							20
BLOCK DESIGN	48		0	2	(4) 6	11	18	20	22	25		30
RAVEN'S MATRICES	36	10	15	(16) 20	22		25	27		30		35
		<5	10	20	30	40	50	60	70	80	90	95<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85:1305
FRONT
RIGHT
+ 102
+ 38
- 26
S 8
T 10
V 125
D 230
G -20
P -49
SOMATOM 2
ST MARY'S HOSPITAL,W2
16-MAY-85 10:09 I:12 E35B
85:1305
FRONT
RIGHT
+ 102
+ 38
- 26
S 8
T 10
V 125
D 230
G -20
P -58
SOMATOM 2
ST MARY'S HOSPITAL,W2
16-MAY-85 10:10 TS/029 I:13 E35B
85:1305
FRONT
RIGHT
+ 102
+ 38
- 26
S 8
T 10
V 125
D 230
G -20
P -67
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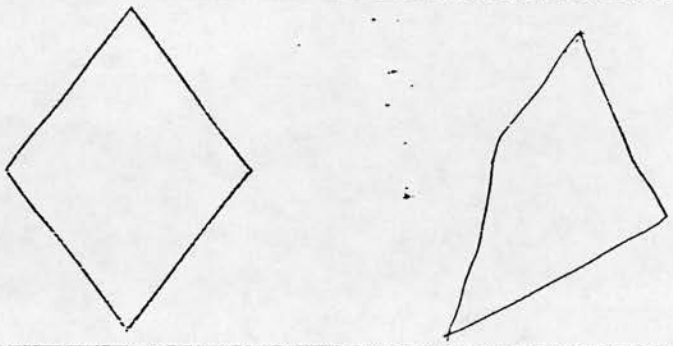
ST MARY'S HOSPITAL,W2
16-MAY-85 10:10 TS/030 I:14 E35B
FRONT
RIGHT
+ 102
+ 38
- 26
S 8
T 10
V 125
D 230
G -20
P -76
SOMATOM 2
ST MARY'S HOSPITAL,W2
16-MAY-85 10:11 I:15 E35B
85:1305
FRONT
RIGHT
+ 102
+ 38
- 26
S 8
T 10
V 125
D 230
G -20
P -85
SOMATOM 2
ST MARY'S HOSPITAL,W2
16-MAY-85 10:11 I:16 E35B
85:1305
FRONT
RIGHT
+ 102
+ 38
- 26
S 8
T 10
V 125
D 230
G -20
P -94
SOMATOM 2
ST MARY'S HOSPITAL,W2
16-MAY-85 10:12 I:17 E35B
85:1305
FRONT
RIGHT
+ 102
+ 38
- 26
S 8
T 10
V 125
D 230
G -20
P -103
SOMATOM 2
ST MARY'S HOSPITAL,W2
16-MAY-85 10:13 I:18 E35B
85:1305
FRONT
RIGHT
+ 104
+ 40
- 24
S 8
T 10
V 125
D 230
G -20
P -112
SOMATOM 2
ST MARY'S HOSPITAL,W2
16-MAY-85 10:13 TS/035 I:19 E35B
85:1305
FRONT
RIGHT
+ 112
+ 48
- 16
S 8
T 10
V 125
D 230
G -20
P -121

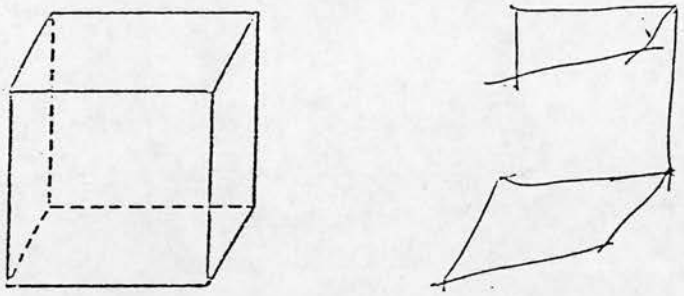
CT (18.4.85) There is a large area of low attenuation in the right posteriorly. It is not as far medial as the occipital region. There is no space occupying effect and the appearances are those of an infarct.

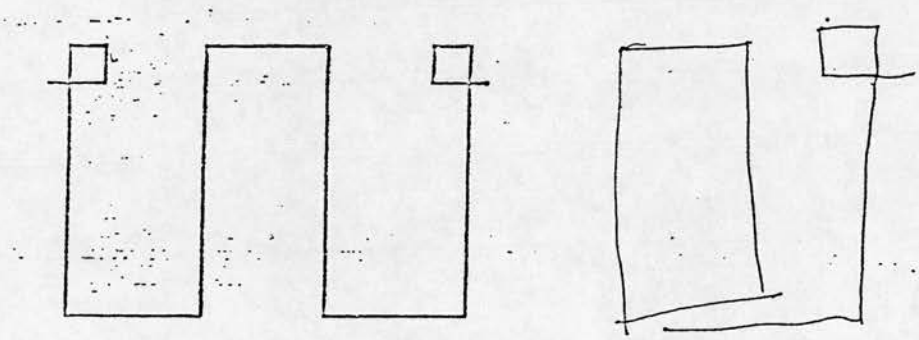
There was no evidence of dysarthria, verbal agnosia, anomia, reading or writing disturbance, ideational, oral or ideomotor apraxia. Auditory memory was reduced by Sentence Repetition to the tenth percentile normal, and Digit Repetition (Forward) to the sixtieth percentile normal. Reversed operations were reduced to the fifth percentile of normal. Digit repetition scores yielded a scaled score of 10. Constructional apraxia demonstrated a left neglect while both tests of non verval intelligence yielded scores below normal limits. Block Design gave a scaled score of 6.

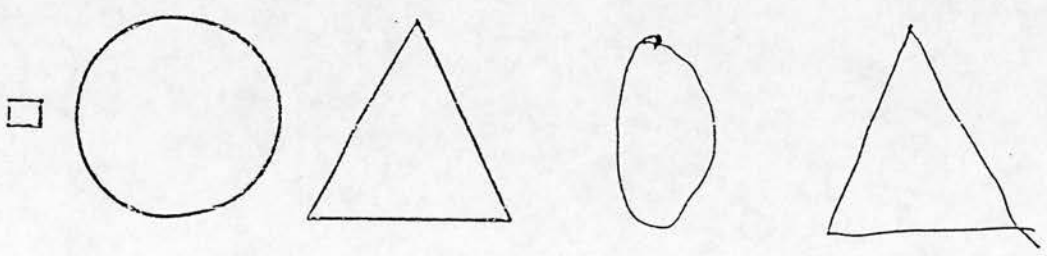
Comments:

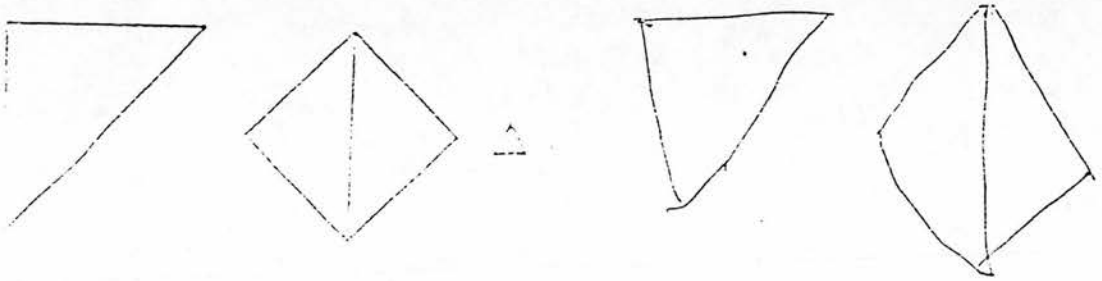
The profile demonstrates diminished memory scores that may relate to concentration difficulty as demonstrated by the inability to repeat automatic sequences in reverse. It may also explain the decreased word fluency score that nevertheless remained within normal limits. Comprehension errors in Part F of the Token Test appear to relate to memory rather than to the processing of spatial concepts. Comprehension of written instructions, where the trace is held stable, is normal. Failure of constructional tasks reflects contralateral neglect in parietal lesions (Brain 1941 , Battersby et al 1956). Such failure of visuo spatial perception would account for failure of tests that demand such parietal and occipital abilities as demonstrated by Risberg and Ingvar (1973).











SUBTEST SUMMARY PROFILE (APHASIA)

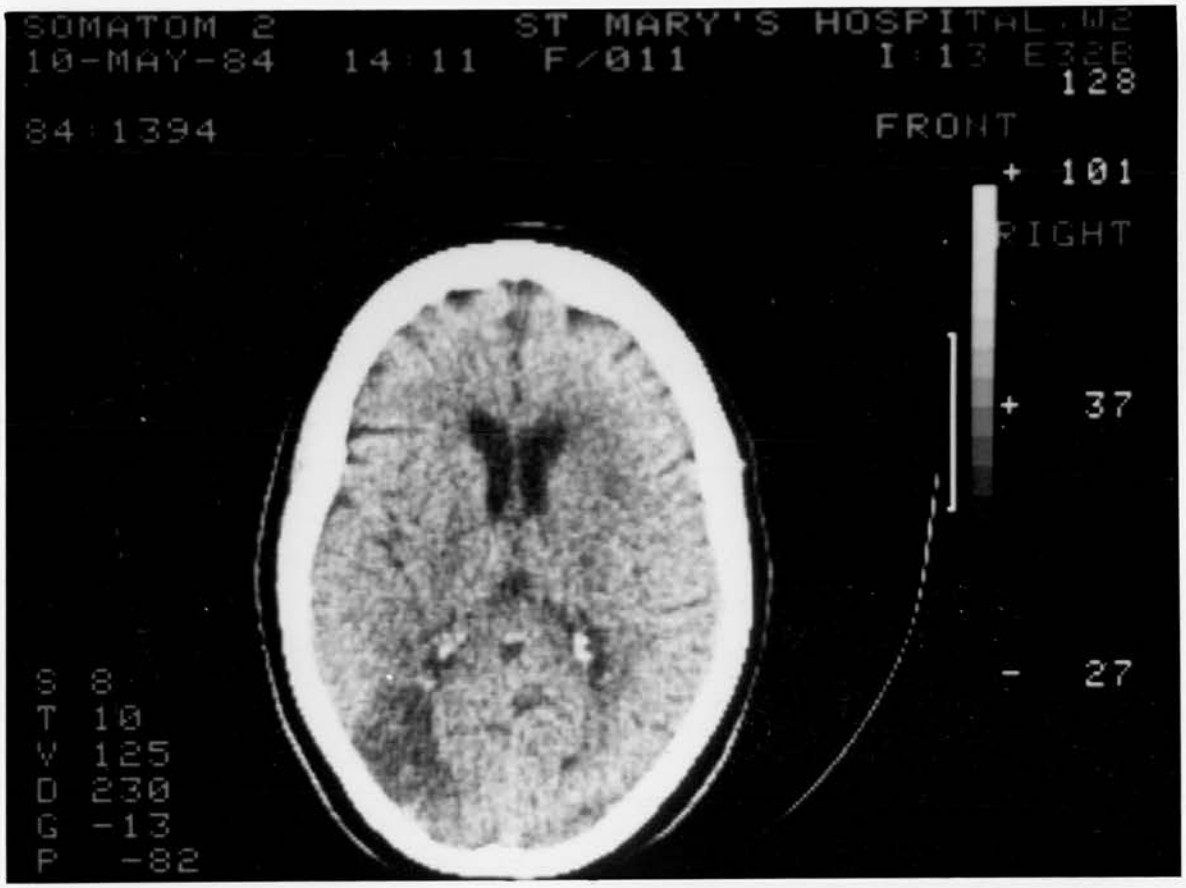
NAME No. 2 DATE 9.5.84

SEX Female ---------11.7.85

D of B Age 70 years

DIAGNOSIS Left Hempsphere C.V.A.

TEST	MAX. SCORE	PERCENTILES 5	10	20	30	40	50	60	70	80	90	95
RATING SCALE	40											40
IDENTIFICATION/NAME	8	4	5	7								8
IDENTIFICATION/FUNCTION	8	4	5	7								8
TOKEN TEST Part A-E	67	2	5	12	20	30	45	50	52	60	62	65	67
TOKEN TEST Part F	96		0	20	35	50	61	65	67	77	83	85	96
TOKEN TEST Total	163	0	11	16	65	85	106	110	127	130	140	148	150	160
SENTENCE REPETITION	22			0	1	4	6	8	9	10	12	13	16
REPEATING DIGITS	14			0	1	2	3	4	5	6	7	9
REPEATING DIGITS REVERSE	14						1		2	3	4	7
AUTOMATIC SPEECH	4		0		2							4
CONFRONTATION NAMING	16	0	1	4	6	9	12	13	15			16
DESCRIPTION OF FUNCTION	16		0	3	7	9	13	14	15			16
TACTILE NAMING (Right)	8						0	2	4	6	7	8
TACTILE NAMING (Left)	8		1	2	3	5	6	7				8
GESTURE	16	1	12	13		15						16
FLUENCY	60					0	2	3	5	10	14	24	26	32
SENTENCE CONSTRUCTION	25							0	14	16	19	20	21	25
READING WORDS	8				1							8
WORD RECOGNITION	8	6	7									8
READING SENTENCES	7						3		6			7
SENTENCE COMPREHENSION	32		2	5	20	23	26	27	29	30	31	32
AUTOMATIC WRITING	5		0				3	4				5
SPELLING	24					0	3	14	18	19	21	22	23	24
DICTATION	13					0	4	9	11					13
COPYING	11		2	8	9	10								11
CALCULATION	10			1		3	5	6	7	8				10
ORAL	20	10		14	15	16	18		19					20
IDEOMOTOR	20	12	15	17										20
CONSTRUCTIONAL	20	12	17	18	19									20
BLOCK DESIGN	48		0	2	6	11	16	18	20	22	25			30
RAVEN'S MATRICES	36	10	15	20	22		25	27		30	32			35
		5	10	20	30	40	50	60	70	80	90	95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I:13 E32B
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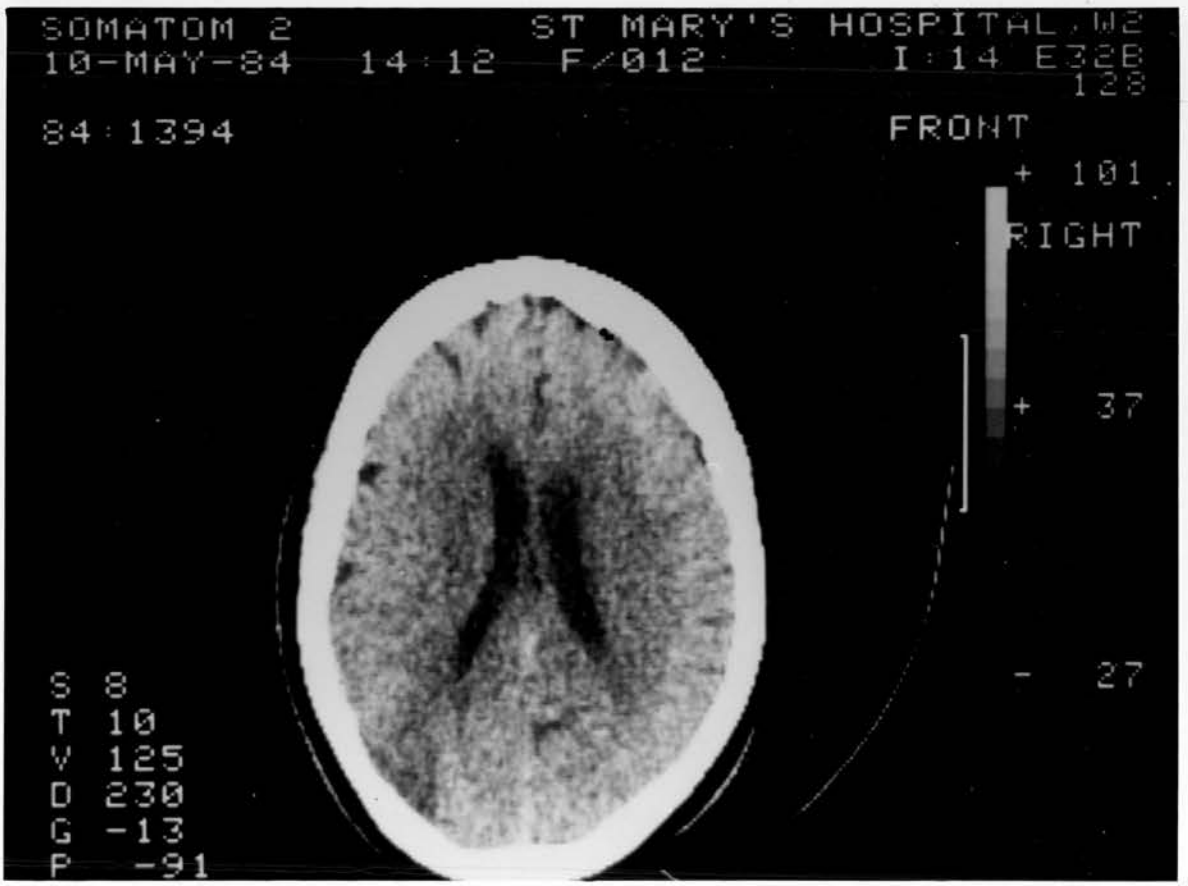
84:1394
FRONT
+ 101
RIGHT
+ 37
- 27
S 8
T 10
V 125
D 230
G -13
P -82


SOMATOM 2
ST MARY'S HOSPITAL.W2
10-MAY-84 14:12 F/012
I:14 E32B
128
84:1394
FRONT
+ 101
RIGHT
+ 37
- 27
S 8
T 10
V 125
D 230
G -13
P -91

This patient sustained a left temporo-parieto infarct. She was alert and a good historian with impaired concentration: automaticisms were intact but she was not able to recite in reverse.

Recent memory was reduced and immediate memory yielded a scaled score of 8. Sentence repetition was marked with sound substitution errors.

Impaired auditory memory dampened comprehension.

Naming produced phonemic paraphasia which is also reflected in writing (see attached). The errors are rule governed being substitutions of features ("porch" for torch is an error of the initial consonant by placement, where voice and manner are appropriate). There was also a word search producing mixed paraphasia (the search for tweezers produced "pluckers-pliers-peezers").

Syntax was normal but time constraints were violated. Word fluency was reduced by phonemic instability.

Reading and reading comprehension was normal, writing demonstrated orthographic errors, there was evidence of dyscalculia but none of disturbance of praxis.

Block Design gave a scaled score of 8, scores for the RCPM were evenly distributed and at the twentieth percentile of normal.

Comment:

Conduction aphasia is fluent and meaningful with phonemic paraphasia in verbal repetition tasks. Concentration is minimally impaired. Anatomically it results from damage to the arcuate fasciculus (Damasio and Damasio 1980).

On reassessment a year later all language abilities had improved. Concentration was mildly impaired. Sound substitution errors ("cut" for cup) persisted as did temporo parietal features described above.

BELL Z' (Bell)

RATEN (Razor)

PIPE (Pipe)

WHISTLE (Whistle)

TORCH (Torch)

WASTCH (Watch)

CLASSES (Glasses)

TWEEZERS (Tweezers)

THIS IS A VERY R' NICE TAG (Dictation: This is a very nice day

THIS BRICK BUILDE WAS BUILTER LAST YEAR

(Dictation: This brick building was built last year

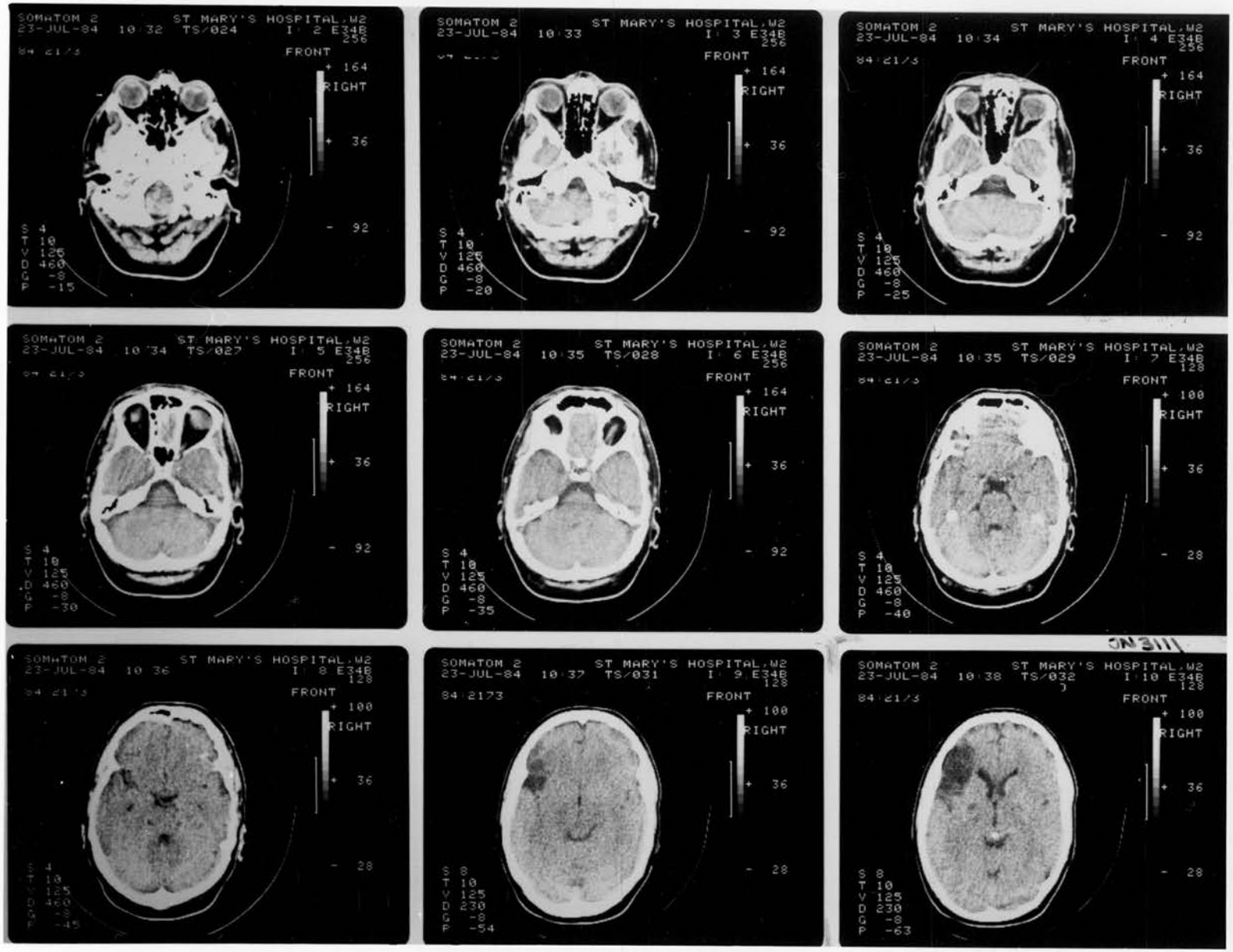
I AM VERY HANDREY (Copy: I am very hungry)

THE COLOAR OF THE WALLS IS GREEN (Copy: The colour of the walls is green

SUBTEST SUMMARY PROFILE (APHASIA)

NAME No. 3 DATE.....27.7.84
SEX Male
D of B Age 38 years
DIAGNOSIS Left Hemisphere C.V.A.

TEST	MAX. SCORE	PERCENTILES 5	10	20	30	40	50	60	70	80	90	95
RATING SCALE	40											40
IDENTIFICATION/NAME	8	4	5	7								(8)
IDENTIFICATION/FUNCTION	8	4	5	7								(8)
TOKEN TEST Part A-E	67	2	5	12	20	30	45	52	(60)	62		67
TOKEN TEST Part F	96		0	20	35	50	61	65	67	75	85 (92)	96
TOKEN TEST Total	163	0	11	16	65	85	106	110	130	140	150 (152)	160
SENTENCE REPETITION	22			0	1	4	8	9	10 (11)	12	13	16
REPEATING DIGITS	14			0	1	2	3	4	(5)	6	7	9
REPEATING DIGITS REVERSE	14						1		(2)		4	7
AUTOMATIC SPEECH	4		0		2							(4)
CONFRONTATION NAMING	16	0	1	4	6	9	12	13	15			(16)
DESCRIPTION OF FUNCTION	16		0	3	7	9	13	14	15			(16)
TACTILE NAMING (Right)	8						0	2	4	6	7	(8)
TACTILE NAMING (Left)	8		1	2	3	5	6	7				(8)
GESTURE	16	1	12	13		15						(16)
FLUENCY	60					0	2	3	(5)	10	26	32
SENTENCE CONSTRUCTION	25							0 (13)	14	16	20	25
READING WORDS	8				1							(8)
WORD RECOGNITION	8	6	7									(8)
READING SENTENCES	7						(3)		6			7
SENTENCE COMPREHENSION	32		2	5	20	23	26	27	29	30	(31)	32
AUTOMATIC WRITING	5		0				3	4				(5)
SPELLING	24					0	3	14	18	19 (20)	22	24
DICTATION	13					0	4	9	(11)			13
COPYING	11		2	8	9	10						(11)
CALCULATION	10			1		3	5	6	(7)			10
ORAL	20	10		14	15	16	(18)		19			20
IDEOMOTOR	20	12	15	17								(20)
CONSTRUCTIONAL	20	12	18		19							(20)
BLOCK DESIGN	48		0	2	6	11	18	20	22	25		(35)
RAVEN'S MATRICES	36	10	15	20	22		25	27		30	(34)	35
		5	10	20	30	40	50	60	70	80	90	95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FRONT
+ 164
RIGHT
+ 36
- 92
S 4
T 10
V 125
D 460
G -8
P -15
SOMATOM 2 ST MARY'S HOSPITAL,W2
23-JUL-84 10:33 I: 3 E34B
256
FRONT
+ 164
RIGHT
+ 36
- 92
S 4
T 10
V 125
D 460
G -8
P -20
SOMATOM 2 ST MARY'S HOSPITAL,W2
23-JUL-84 10:34 I: 4 E34B
256
84:21/3
FRONT
+ 164
RIGHT
+ 36
- 92
S 4
T 10
V 125
D 460
G -8
P -25
SOMATOM 2 ST MARY'S HOSPITAL,W2
23-JUL-84 10:34 TS/027 I: 5 E34B
256
FRONT
+ 164
RIGHT
+ 36
- 92
S 4
T 10
V 125
D 460
G -8
P -30
SOMATOM 2 ST MARY'S HOSPITAL,W2
23-JUL-84 10:35 TS/028 I: 6 E34B
256
FRONT
+ 164
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+ 36
- 92
S 4
T 10
V 125
D 460
G -8
P -35
SOMATOM 2 ST MARY'S HOSPITAL,W2
23-JUL-84 10:35 TS/029 I: 7 E34B
128
84:21/3
FRONT
+ 100
RIGHT
+ 36
- 28
S 4
T 10
V 125
D 460
G -8
P -40
SOMATOM 2 ST MARY'S HOSPITAL,W2
23-JUL-84 10:36 I: 8 E34B
128
84:21/3
FRONT
+ 100
RIGHT
+ 36
- 28
S 4
T 10
V 125
D 460
G -8
P -45
SOMATOM 2 ST MARY'S HOSPITAL,W2
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FRONT
+ 100
RIGHT
+ 36
- 28
S 8
T 10
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D 230
G -8
P -54
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23-JUL-84 10:38 TS/032 I:10 E34B
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RIGHT
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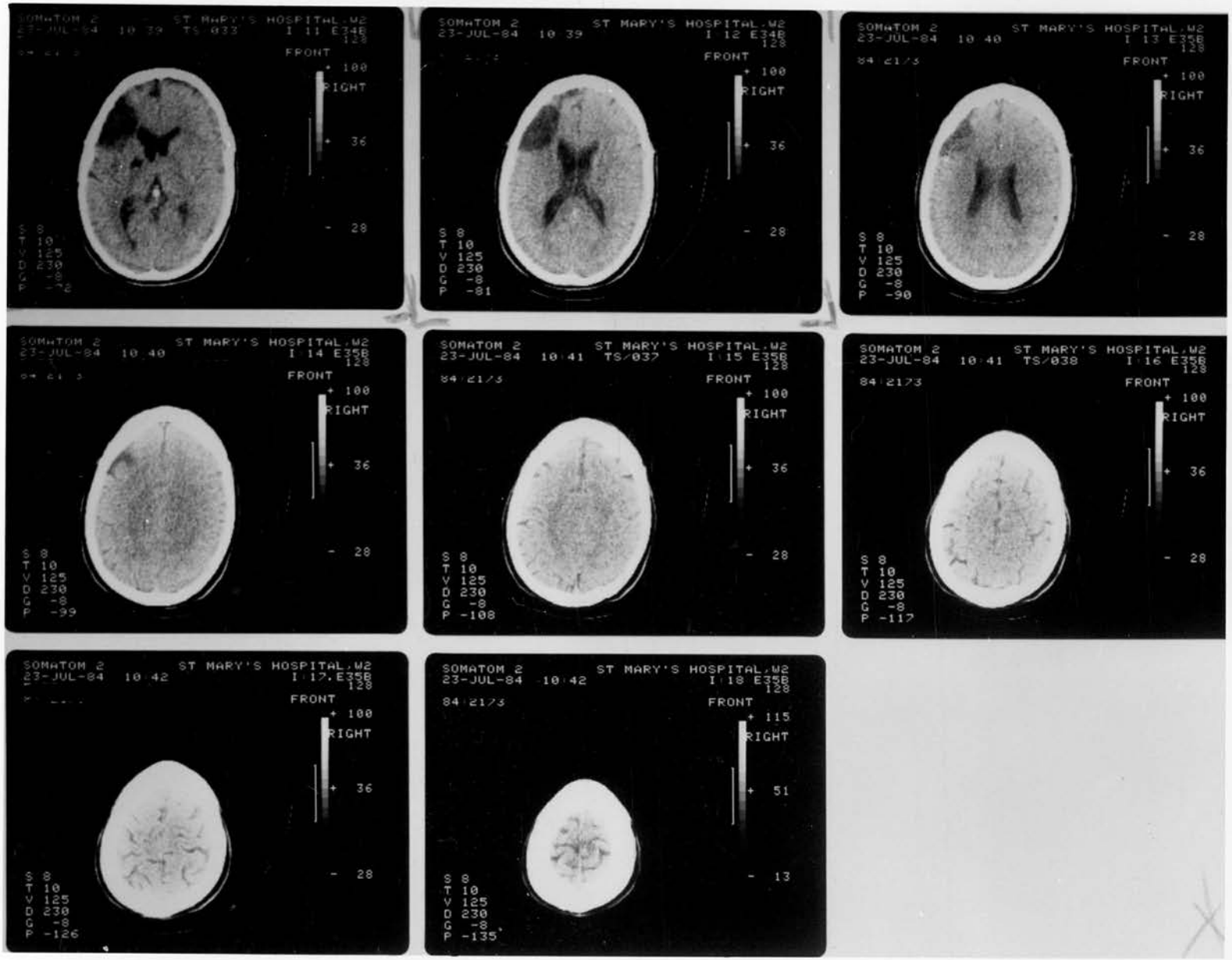
P -63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RIGHT
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T 10
V 125
D 230
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23-JUL-84 10:39 I 12 E348
128
FRONT
+ 100
RIGHT
+ 36
- 28
S 8
T 10
V 125
D 230
G -8
P -81
SOMATOM 2 ST MARY'S HOSPITAL,W2
23-JUL-84 10:40 I 13 E358
128
84:21:3
FRONT
+ 100
RIGHT
+ 36
- 28
S 8
T 10
V 125
D 230
G -8
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23-JUL-84 10:40 I 14 E358
128
84:21:3
FRONT
+ 100
RIGHT
+ 36
- 28
S 8
T 10
V 125
D 230
G -8
P -99
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128
84:21:3
FRONT
+ 100
RIGHT
+ 36
- 28
S 8
T 10
V 125
D 230
G -8
P -108
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23-JUL-84 10:41 TS/038 I 16 E358
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84:21:3
FRONT
+ 100
RIGHT
+ 36
- 28
S 8
T 10
V 125
D 230
G -8
P -117
SOMATOM 2 ST MARY'S HOSPITAL,W2
23-JUL-84 10:42 I 17 E358
128
FRONT
+ 100
RIGHT
+ 36
- 28
S 8
T 10
V 125
D 230
G -8
P -126
SOMATOM 2 ST MARY'S HOSPITAL,W2
23-JUL-84 10:42 I 18 E358
128
84:21:3
FRONT
+ 115
RIGHT
+ 51
- 13
S 8
T 10
V 125
D 230
G -8
P -135

CT (20.7.84) Large low attenuation in the left fronto parietal region affecting grey and white matter.

This young mechanical engineer was well oriented and a good historian. Recent memory was impaired and digit recall gave a scaled score of 4. This memory disturbance affected comprehension for the Token Test was reduced below the fifth percentile for normals. Written comprehension was less impaired.

Automaticisms were intact but concentration was impaired in reverse operations when reciting or repeating digits.

There was no evidence of anomia but Fluency was reduced using the FAS stimulus. Tests of categorical association, as in the recall of animal names, were slightly better. This differential between the two measures of fluency is described by Struss and Benson (1983). Ideational fluency by animal naming was at the ninetieth percentile for aphasics by the standardised data of Goodglass and Kaplan (1982).

Sentence Construction tasks were reduced because of inertia.

Single word reading and comprehension was intact but again inertia dampened sentence reading although comprehension of the instructions was normal.

Writing produced perseverative errors and corrections as well as orthographic errors (See attached). Errors of calculation were reflected in complex arithmetical problems of addition and subtraction.

There was evidence of oral apraxia, which is associated with anterior aphasia, but none of ideation, ideomotor or constructional performance. Drawing did however reflect perseveration of designs and overscoring (See attached).

Block Design yielded a scaled score of 11 and the RCPM was at the eightieth percentile.

Comment:

The syndrome of Transcortial Motor Aphasia, or Dynamic Aphasia has been described by Luria (1970) and reviewed by Rubens and Kertesz (1983) as localised anterior and superior to Broca's area. The syndrome reflects an emptiness of speech and a loss of the quality and quantity of speech.

Attached is a five minute written language sample that demonstrates this paucity of output, the inability to solve logical problems that are appropriate to this man's occupation and an inability to describe situations which was premorbidly intact. After stroke he was no longer able to "trouble shoot" engines for their faults.

The transcription is described according to phrase or clause boundaries, usually by conjunctions or where the utterance broke naturally. One dash represents a normal pause, two are longer than normal and three is an abnormal pause after Crystal (1982). Prosodic and intonation features are not marked, they were normal.

I am very hungry.

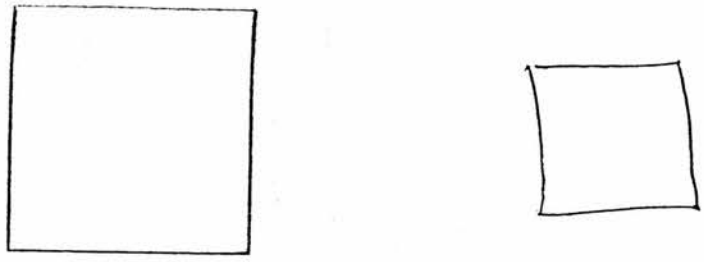
The colour OF THE WALLS IS green.

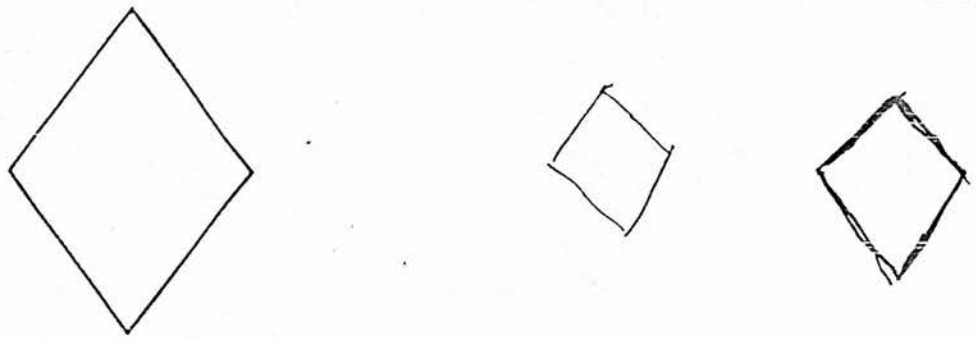
THIS A VERY NICE DAY.

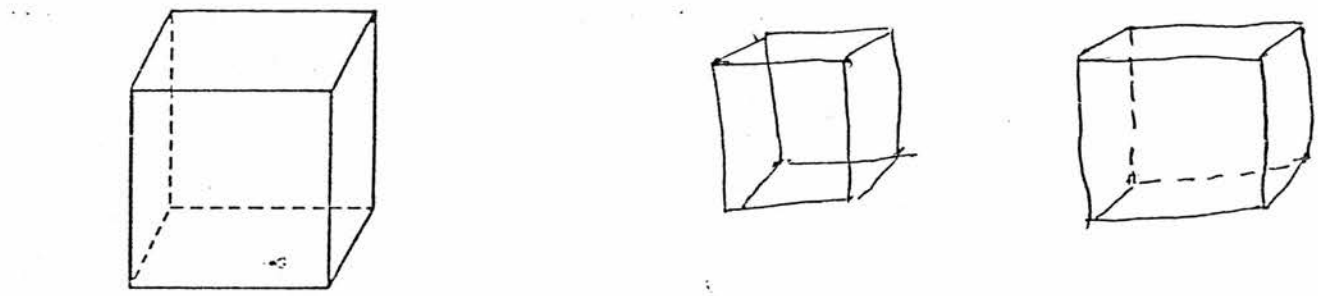
THE ~~RED~~ BRICK BUILDING WAS ~~BU~~ BUILT LAST YEAR.

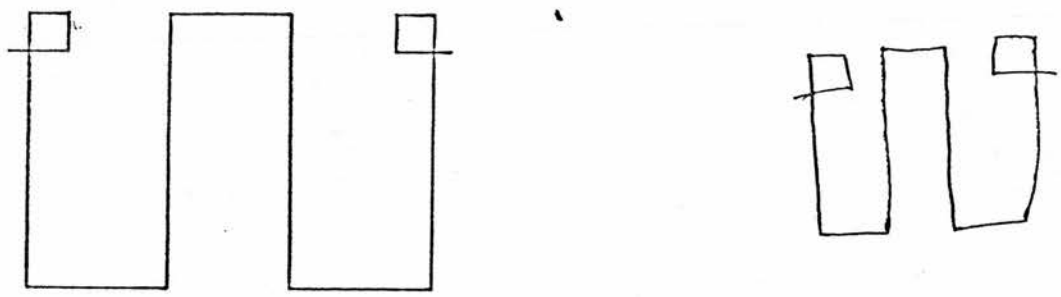
WHITE. WATCH GLASSES. TWISTER PIPE TORCH RAZOR.

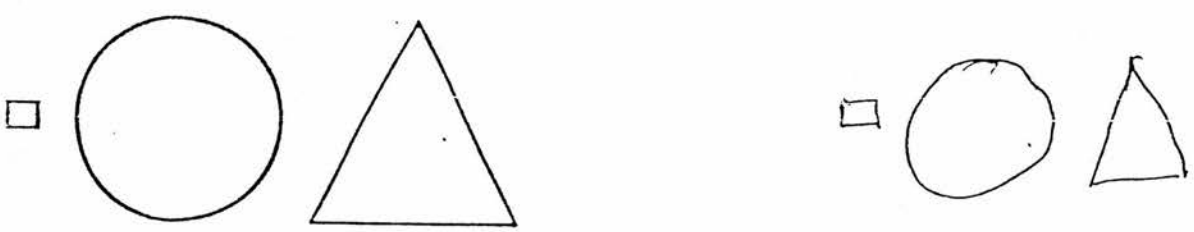
BELL

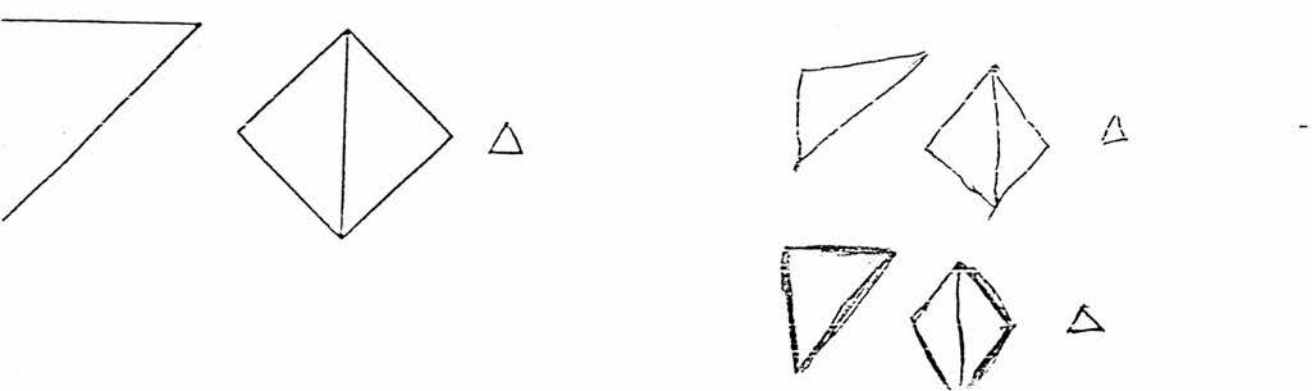












No. 18 Age 38 yrs

LANGUAGE SAMPLE

Therapist: What have you been doing today?

Patient: I went down to Bristol - -
five o'clock actually when I left here
examined an engine that developed an oil leak -
made a report -
that was it - - -

Therapist: Tell me about the engine

Patient: a big Rolls Royce engine - V12 - - - (laughed)

Therapist Tell me about this particular engine in Bristol.

Patient: erm (filled pause) - - -

Therapist What was it for?

Patient: its a generating set
for if the mains goes off
that can start up
and run off a load for the building

Therapist: What's a load?

Patient: - - - power - - -

Therapist: Tell me about the building.

Patient: its a big office actually - - -

1 min.
Therapist: Tell me the principles that make engines work.

Patient: mainly they're deisels - - -

Therapist How do they work?

Patient by running at certain revs - - -

Therapist: How does it give power?

Patient - - -

Therapist: What happens when you turn on a car engine?
What does the ignition do?

2.00 mins.

Patient: it produced power to - -
or takes from the battery the power to the distributor
going to the spark plugs - - er (filled pause) - -
produces a spark

Therapist: What does the spark do?

Patient: there's fuel coming in from the carburettor - - -

Therapist: What does the carburettor do?

Patient: it - - er (filled pause) - -
distributes (stammered, word change follows)
gives out certain amounts of fuel

Therapist: Then what happens?

Patient: that goes into the manifold the - -

Therapist: What's a manifold

Patient: a length of pipe

3.00 mins.

Therapist: Is the fuel ignited by a spark?

Patient: when it gets into - - - et (filled pause) - - pistons
cylinders actually
pistons go up and down - - -

4.00 mins

Therapist: What do they do?

Patient: there are four systems actually - - er (filled pause) - -
inlet - - ah (filled pause) - - compression explosion exhaust
that's a system

5.00 mins.

SUBTEST SUMMARY PROFILE (APHASIA)

NAME No. 4 DATE.....5.11.82

SEX Male _ _ _ _ _ _ _ _ 5.2.85

D of B Age 56 years

DIAGNOSIS Left Hemisphere C.V.A.

TEST	MAX. SCORE	PERCENTILES										
		<5	10	20	30	40	50	60	70	80	90	95<
RATING SCALE	40											40
IDENTIFICATION/NAME	8	4	5	7								8
IDENTIFICATION/FUNCTION	8	4	5	7								8
TOKEN TEST Part A-E	67	2	5	12	20	30	45	52	57	60	62	66	67
TOKEN TEST Part F	96		0	20	35	50	61	65	67	71	75	85	91	96
TOKEN TEST Total	163	0	11	16	65	85	106	110	128	130	140	150	157	160
SENTENCE REPETITION	22			0	1	4	8	9	10	12	13	14	16
REPEATING DIGITS	14			0	1	2	3	4	5	6	7	8	9
REPEATING DIGITS REVERSE	14	0					1		2		4	6	7
AUTOMATIC SPEECH	4		0		2								4
CONFRONTATION NAMING	16	0	1	4	6	9	12	13	15				16
DESCRIPTION OF FUNCTION	16		0	3	7	9	13	14	15				16
TACTILE NAMING (Right)	8						0	2	4	6	7		8
TACTILE NAMING (Left)	8		1	2	3	5	6	7					8
GESTURE	16	1	12	13		15							16
FLUENCY	60					0	2	3	5	10	11	26	32
SENTENCE CONSTRUCTION	25							0	12	14	16	20	25
READING WORDS	8				1								8
WORD RECOGNITION	8	6	7										8
READING SENTENCES	7						3	4		6			7
SENTENCE COMPREHENSION	32		2	5	20	23	26	27	28	29	30	31	32
AUTOMATIC WRITING	5		0				3	4					5
SPELLING	24					0	3	14	18	19	21	22	24
DICTATION	13					0	4	9	11				13
COPYING	11		2	8	9	10							11
CALCULATION	10			1		3	5	6	7				10
ORAL	20	10		14	15	16	17	18		19			20
IDEOMOTOR	20	12	15	17					19				20
CONSTRUCTIONAL	20	12	18		19								20
BLOCK DESIGN	48		0	2	6	11	18	20	22	24	25	28	30
RAVEN'S MATRICES	36	10	15	20	22		25	27	28		30		35
		<5	10	20	30	40	50	60	70	80	90	95<

CT (1.11.82) There is an extensive infarct in the middle and anterior cerebral artery territory.

This professor emeritus demonstrated reduced comprehension on the Token Test with an auditory memory span of three units. It was confirmed by "point" digit span, since he was speechless for propositional language.

Automatic speech was preserved as was the ability to utter single words. Articulatory apraxia contaminated Fluency and Sentence Construction.

The ability to read and recognise single words was retained but he could neither read nor act appropriately on whole instructions.

Writing was poor being completed with the non dominant hand, but there was also evidence of orthographic errors of reversal and perseveration which were not present when copying.

There was a failure of complex arithmetical processes and evidence of oral, ideomotor and constructional apraxia.

Block Design gave a scaled score of 10, the RCPM was at the thirtieth percentile of normal.

Two years later there was overall improvement. Comprehension was within normal limits as was the ability to repeat sentences and digits. Digit span gave a scaled score of 15.

Speech remains dysfluent with articulatory groping. Fluency was reduced and sentences paragrammatic and laboured.

Reading and reading comprehension were within normal range, dyscalculia had resolved and apraxic difficulties had improved. The skill of writing with a non dominant hand had been acquired and there were no real dysgraphic errors.

Tests of verbal intelligence remain below the fiftieth percentile of normal, although Block Design gave a scaled score of 11.

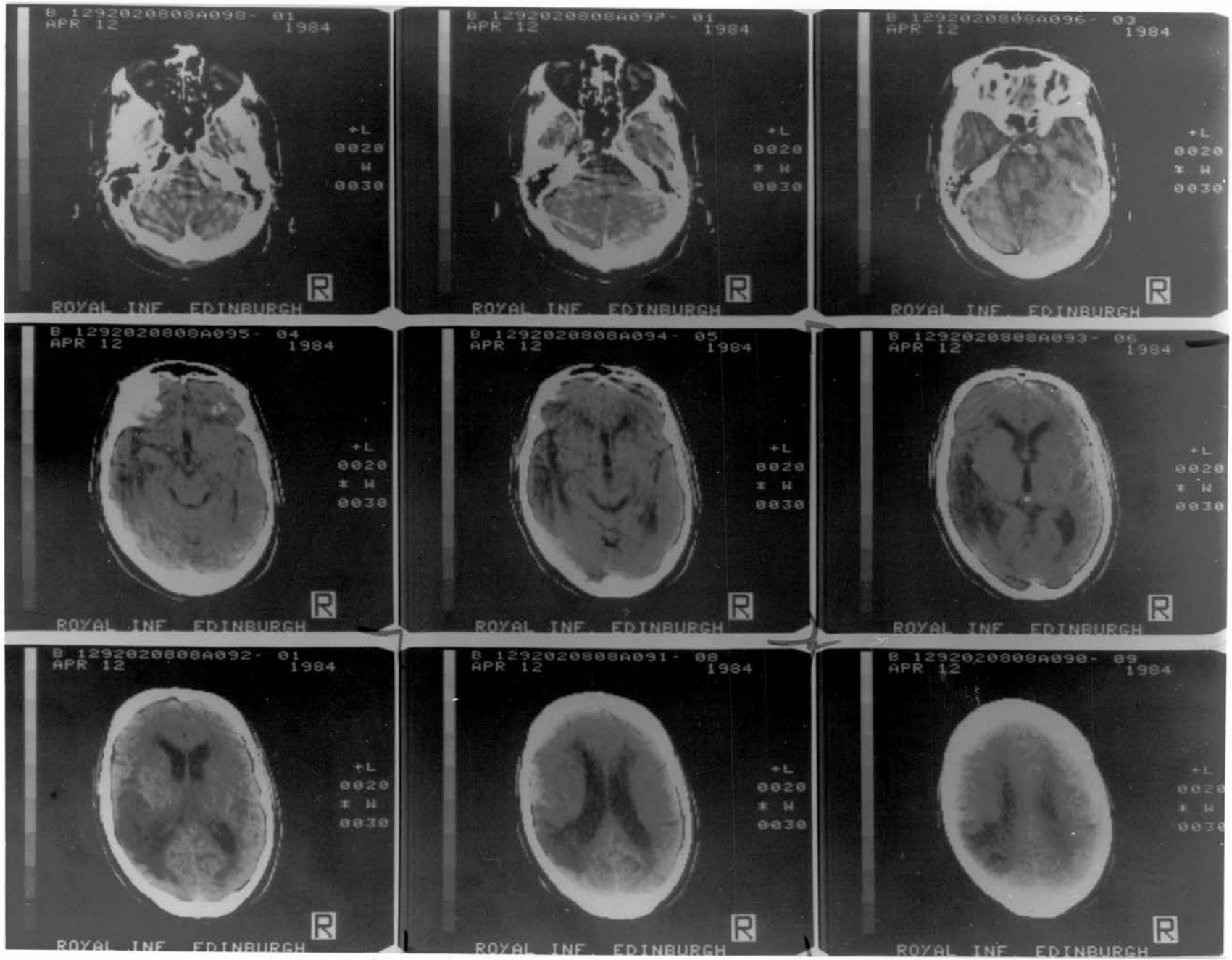
Comment:

Restoration of function is evident in a dramatic improvement of comprehension while expressive language functions improve semantically and syntactically. Articulatory skill, while still laboured, was established and reading and writing had also improved. The ability to write and speak were facilitated with regular speech and communication therapy.

SUBTEST SUMMARY PROFILE (APHASIA)

NAME No. 5 DATE.....13.1.84
SEX Male
D of B Age 76 years
DIAGNOSIS Left Hemisphere C.V.A.

TEST	MAX. SCORE	PERCENTILES 5	10	20	30	40	50	60	70	80	90	95
RATING SCALE	40											40
IDENTIFICATION/NAME	8	4	5	7								(8)
IDENTIFICATION/FUNCTION	8	4	5	(7)								8
TOKEN TEST Part A-E	67	(2)	5	12	20	30	45	52	60	62		67
TOKEN TEST Part F	96		(0)	20	35	50	61	65	67	75	85	96
TOKEN TEST Total	163	0	(2)	11	16	65	85	106	110	130	140	150	160
SENTENCE REPETITION	22				0	(1)	4	8	9	10	12	13	16
REPEATING DIGITS	14				0	1	2	(3)	4	5	6	7	9
REPEATING DIGITS REVERSE	14	(0)						1		2		4	7
AUTOMATIC SPEECH	4		(0)			2							4
CONFRONTATION NAMING	16	0	1	(2)	4	6	9	12	13	15			16
DESCRIPTION OF FUNCTION	16		(0)		3	7	9	13	14	15			16
TACTILE NAMING (Right)	8						(0)		2	4	6	7	8
TACTILE NAMING (Left)	8	(0)		1	2	3	5	6	7				8
GESTURE	16	1	12	13			15						(16)
FLUENCY	60						0	2	(3)	5	10	26	32
SENTENCE CONSTRUCTION	25								(0)	14	16	20	25
READING WORDS	8					1							(8)
WORD RECOGNITION	8	6	7										(8)
READING SENTENCES	7							3		6			(7)
SENTENCE COMPREHENSION	32			2	5	20	(21)	23	26	27	29	30	31	32
AUTOMATIC WRITING	5			0			(2)		3	4				5
SPELLING	24						0		(3)	14	18	19	22	24
DICTATION	13						(0)		4	9	11			13
COPYING	11			2	8	(9)		10						11
CALCULATION	10				1			3	(5)		6	7			10
ORAL	20	10			14	15	16	18			19			(20)
IDEOMOTOR	20	12	15	17										(20)
CONSTRUCTIONAL	20	12	18			19								(20)
BLOCK DESIGN	48			0	2	6	11	(18)		20	22	25		30
RAVEN'S MATRICES	36	10	15	20	22			25		27	(29)	30		35
		5	10	20	30	40	50	60	70	80	90	95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CT (12.4.84) Temporo parietal lesions.

This 76 year old banker was admitted as demented to a day unit for psychogeriatrics. He was assessed by the speech therapist and cross referred to a neurologist for further investigation. He was rediagnosed as aphasic.

Comprehension was severely impaired and audioverbal memory inaccessible.

Naming tasks produced fluent neologisms and jargon and tasks of Fluency and Sentence Construction were abandoned.

The patient was able to read single words and sentences. He was able to demonstrate the meaning of the words but not the sentences.

Dysgraphic errors included phonemic disturbance and neologism while Dictation was abandoned and two errors were evident in Copying.

There was no evidence of apraxia. Block Design gave a scaled score of 10 and the RCPM was at the fiftieth percentile of normal.

Comment:

The performance reflects fluent aphasia producing reduced comprehension and meaningless jargon. He provides an example of how stroke patients may be misdiagnosed as dements without formal language assessment.

An accompanying language sample illustrates output with normal prosody and rate.

Bill
Wife
Watch
Jack
Bisong
Glassoes
Missole
Jweller

Wife

I am very thankful for
The collier of the trouble
of the

No.20 Age 76 yrs

LANGUAGE SAMPLE

Therapist:	Talk to me
Patient:	Friday the 4th Friday the 24th
Therapist:	I want you to talk
Patient:	Australia - horses - dogs - (jargon)
	horsemen - horses - dogs and coffee
	(jargon) one
	Ralph one
	(jargon) and doctors alone
1 min.	the doctors might interest doctors
Therapist:	What about doctors?
Patient:	the doctor the doctor the doctor (jargon)
	one I can see it
	what's the chappie with the beard
	I don't ken him much
	But he runs up to him in London
	(begins to sing Waltzing Matilda)
2 mins.	
Therapist:	Rolph Harris
Patient:	rattle Ralph
	I see it
	I see it
	I just can't (jargon) it
Therapist:	Rolph Harris
Patient:	Ras (jargon)
Therapist:	Don't you like him?
Patient	and these Americans

Patient: if I sometimes say to myself

now I've got to leave - learn letter

and I've got to letter

and I've got to linger

and I've got to think or batsman

and think a man touching to me

and I think the man's standing there looking at me

and he's batching with his teeth

and he's batching with a cheek

and he's batting with a teach

and the (jargon) scar

(jargon) the glass

for the horses

3 mins. what the bloody hell

dishes dishes dishes dishes horses horses

now I see them all standing

if I could stand myself

and fall to all myself

and I just say anything

I like (jargon)

I like (jargon)

(jargon) were around

what's that g o l d

4 mins. g o l d (spelt repeatedly

Therapist: What about gold?

Patient: oh yes (jargon)

I made a lot of money

Australia Australia

SUBTEST SUMMARY PROFILE (APHASIA)

NAME No. 6 DATE.....17.9.84
SEX Male
D of B Age 70 years
DIAGNOSIS Left Hemisphere Posterior Neoplasm

TEST	MAX. SCORE	PERCENTILES 5	10	20	30	40	50	60	70	80	90	95
RATING SCALE	40											40
IDENTIFICATION/NAME	8	4	5	7								8
IDENTIFICATION/FUNCTION	8	4	5	7								8
TOKEN TEST Part A-E	67	2	5	12	20	30	40	45	52	60	62	67
TOKEN TEST Part F	96		0	20	35	44	50	61	65	67	75	85	96
TOKEN TEST Total	163	0	11	16	65	84	85	106	110	130	140	150	160
SENTENCE REPETITION	22			0	1	4	8	9	10	12	13	16
REPEATING DIGITS	14			0	1	2	3	4	5	6	7	9
REPEATING DIGITS REVERSE	14						1		2		4	7
AUTOMATIC SPEECH	4		0		2							4
CONFRONTATION NAMING	16	0	1	4	6	9	12	13	15			16
DESCRIPTION OF FUNCTION	16		0	3	4	7	9	13	14	15			16
TACTILE NAMING (Right)	8						0	1	2	4	6	7	8
TACTILE NAMING (Left)	8		1	2	3	5	6	7					8
GESTURE	16	1	8	12	13		15						16
FLUENCY	60					0	2	3	5	10	15	26	32
SENTENCE CONSTRUCTION	25							0	14	16	20		25
READING WORDS	8				1		3						8
WORD RECOGNITION	8	6	7										8
READING SENTENCES	7	0					3		6				7
SENTENCE COMPREHENSION	32	0	2	5	20	23	26	27	29	30	31		32
AUTOMATIC WRITING	5		0				3	4					5
SPELLING	24					0	3	10	14	18	19	22	24
DICTATION	13					0	4	5	9	11			13
COPYING	11		2	7	8	9	10						11
CALCULATION	10			1	2	3	5	6	7				10
ORAL	20	10		14	15	16	18		19				20
IDEOMOTOR	20	12	15	16	17								20
CONSTRUCTIONAL	20	12	14	18		19							20
BLOCK DESIGN	48		0	2	4	8	11	18	20	22	25		30
RAVEN'S MATRICES	36	10	15	17	20	22		25	27		30		35
		5	10	20	30	40	50	60	70	80	90	95

CT (15.9.84) Hyperdense mass in the region of the trigon of the left lateral ventricle obstructing the occipital horn, body and temporal horn of the left lateral ventricle.

This man's comprehension was fairly preserved. He was disoriented in everything but person but able to give a good personal history but not able to recall non personal events. Concentration and recent memory were all reduced but automaticisms preserved.

There was evidence of agnosia and a loss of comprehension on all parts of the Token Test. Sentence Repetition was at the lower limits of normal and digit recall gave a scaled score of 9. The gap between Repeating Digits forward and in reverse confirms concentration deficit. Naming tasks were marked with circumlocution. Fluency was barely within normal limits but Sentence Construction tasks demonstrated an inability to recall stimulus words.

Reading was limited and dissociated from comprehension while writing tasks demonstrated a failure of phoneme - grapheme transcoding. Copying of geometric figures demonstrated scrawling, loss of perspective, direction and shape recognition.

There was evidence of ideomotor apraxia and constructional apraxia. Comprehension impaired testing of praxis. Gesture was impoverished suggesting ideational apraxia.

Block Design gave a scaled score of 6 and the RCPM was also below normal limits.

Comment:

The syndrome of Transcortical Sensory Aphasia is described as fluent aphasia with reduced comprehension and preserved repetition. Rubens and Kertesz (1983) have published localisation studies implicating the temporo occipital areas and have noted its easy confusion with ATD.

Prince messrs. Clarence
Bateh

Mahike

has one a very nice race.

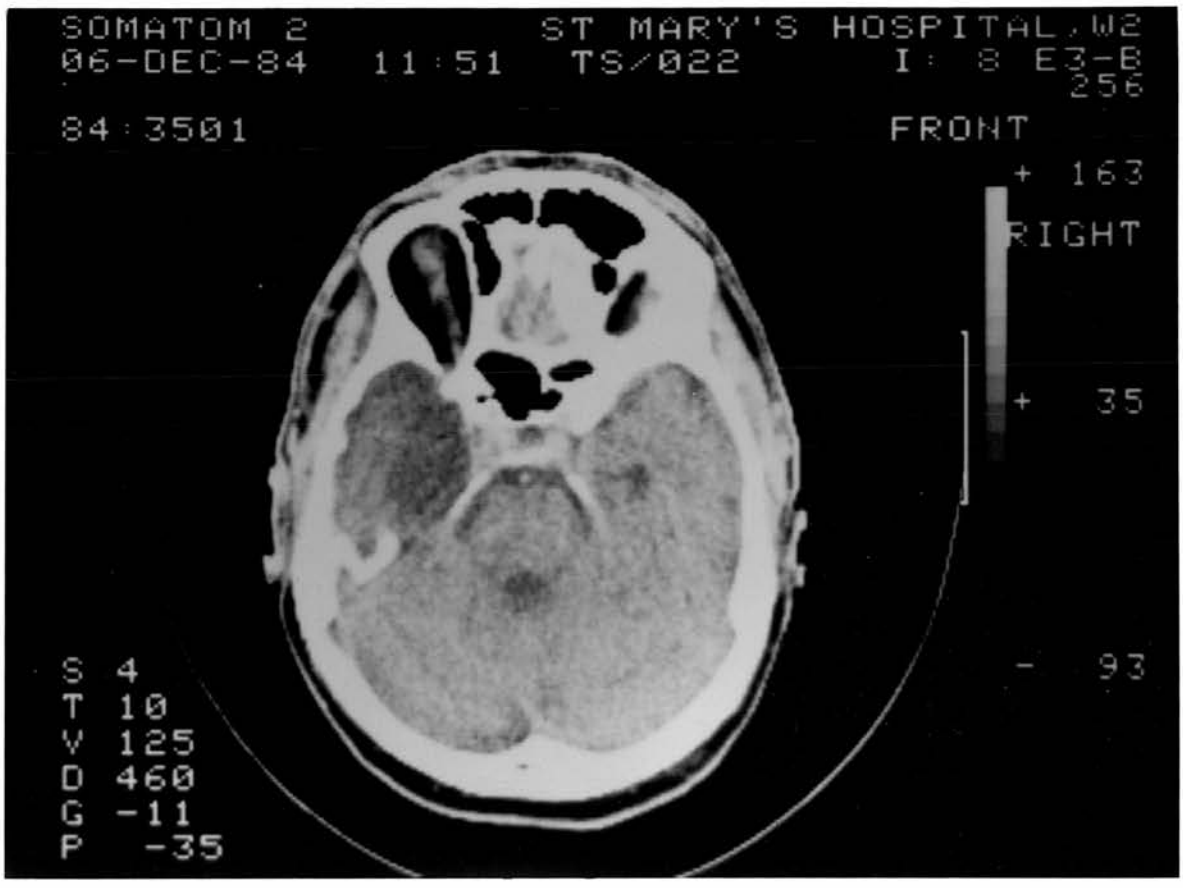
Miss muddy was welth Last Rain.
I am very Lonely.
He ellis of the well is crack.

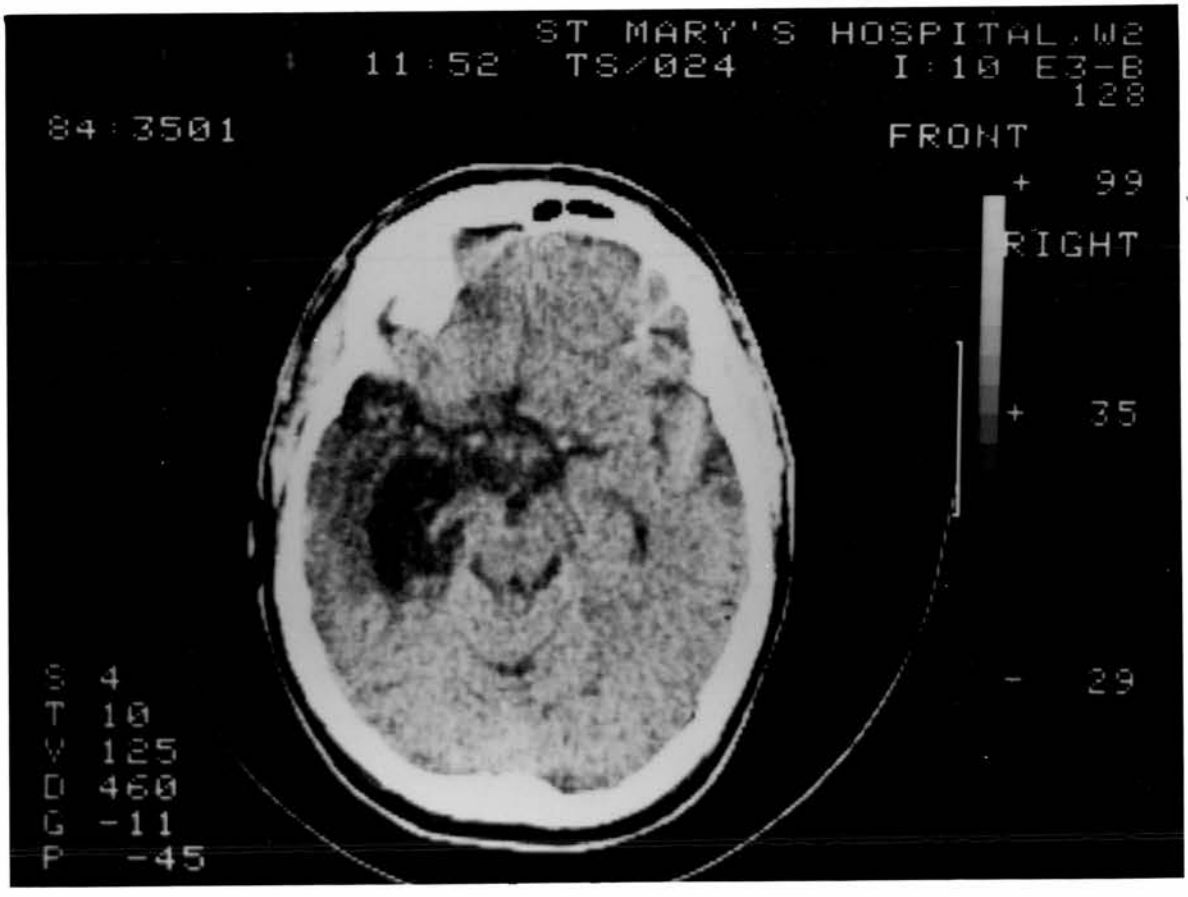
I Master 76 carted Lot apples
Bell. Corsh. Russia inserted
B Bruce

SUBTEST SUMMARY PROFILE (APHASIA)

NAME No. 7 DATE 29.12.84
SEX Male
D of B Age 63 years
DIAGNOSIS Herpes Simplex Encephalitis

TEST	MAX. SCORE	PERCENTILES 5	10	20	30	40	50	60	70	80	90	95
RATING SCALE	40											40
IDENTIFICATION/NAME	8	4	5	7								(8)
IDENTIFICATION/FUNCTION	8	4	5	7								(8)
TOKEN TEST Part A-E	67	2	5	12	20	30	45	(47) 52	60	62		67
TOKEN TEST Part F	96		0	20	35	50	61	65	(69) 75	85		96
TOKEN TEST Total	163	0	11	16	65	85	106	110	(116) 130	140	150	160
SENTENCE REPETITION	22			0	1	4	8	9	(10) 12	13		16
REPEATING DIGITS	14			0	1	2	3	(4) 5	6	7		9
REPEATING DIGITS REVERSE	14						1	2	(3) 4			7
AUTOMATIC SPEECH	4		0		2							(4)
CONFRONTATION NAMING	16	0	1	4	(5) 6	9	12	13	15			16
DESCRIPTION OF FUNCTION	16		0	3	7	(6) 9	13	14	15			16
TACTILE NAMING (Right)	8						(0)	2	4	6	7	8
TACTILE NAMING (Left)	8	(0)	1	2	3	5	6	7				8
GESTURE	16	1	12	13		15						(16)
FLUENCY	60					0	(1) 2	3	5	10	26	32
SENTENCE CONSTRUCTION	25							(0)	14	16	20	25
READING WORDS	8				1							(8)
WORD RECOGNITION	8	6	7									(8)
READING SENTENCES	7						3		6			(7)
SENTENCE COMPREHENSION	32		2	5	20	23	26	27	29	(30)	31	32
AUTOMATIC WRITING	5		0				3	4				(5)
SPELLING	24					0	3	14	18	19	22	(24)
DICTATION	13					0	4	9	11			(13)
COPYING	11		2	8	9	10						(11)
CALCULATION	10			1		3	5	6	7	(8)		10
ORAL	20	(9)	12	14	15	16	18		19			20
IDEOMOTOR	20	12	15	17								(20)
CONSTRUCTIONAL	20	12	18		19							(20)
BLOCK DESIGN	48		0	2	6	11	18	20	(21) 22	25		30
RAVEN'S MATRICES	36	10	15	20	22		25	27	(29) 30			35
		5	10	20	30	40	50	60	70	80	90	95


SOMATOM 2
ST MARY'S HOSPITAL,W2
06-DEC-84 11:51 TS/022
I: 8 E3-B
256
84:3501
FRONT
+ 163
RIGHT
+ 35
- 93
S 4
T 10
V 125
D 460
G -11
P -35


ST MARY'S HOSPITAL,W2
11:52 TS/024
I:10 E3-B
128
84:3501
FRONT
+ 99
RIGHT
+ 35
- 29
S 4
T 10
V 125
D 460
G -11
P -45

CT (6.12.84) Marked atrophy of the left temporal lobe and expansion of the whole of the lateral ventricle but particularly the temporal horn. Conclusion: post focal encephalic atrophy.

EEG (15.12.84) Low to moderate disorganised theta rhythm, maximal amplitude of moderate to high delta activity in left posterior temporal region suggestive of herpes encephalitis.

Psychometry (16.12.84) Verbal IQ 94 Performance IQ 119

Comprehension was diminished by loss of auditory memory to a scaled score of 7. Relatively preserved written comprehension suggests understanding was impaired by memory deficit.

Expressive speech reflected perseverative "tags" of "American" followed by a verbal perseverative response to target names (bell was given as "American bell",whistle was given as "American whistle", razor was given as "American razor"). Responses were usually neologisms. Verbal forms were also perseverated neologisms.

Fluency produced neologisms but sentences were syntactically acceptable but meaningless.

Oral reading at word and sentence level was preserved as was the ability to act on written instructions. There was evidence of dyscalculia and oral but not ideational, ideomotor or constructional apraxia.

Block Design gave a scaled score of 9 and the RCPM also gave a score at the fiftieth percentile of normal.

Comment:

Walsh (1978) described amnestic failure following unilateral temporal lobe lesions with herpes encephalitis and Kellet (1982) noted that it may masquerade as dementia.

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 8 DATE 20.1.86

SEX Male

D of B Age 70 years

DIAGNOSIS Huntington's Chorea

TEST	MAX. SCORE	<5	10	20	30	40	50	60	70	80	90	95<
		PERCENTILES										
RATING SCALE	40	10	13	17	20	(22)	24	26	27	30	32	35
IDENTIFICATION/NAME	8		7									(8)
IDENTIFICATION/FUNCTION	8	6	(7)									8
TOKEN TEST Part A-E	67		47	52	57	(60)	62		63		65	67
TOKEN TEST Part F	96		56	66		76	(79) 85	86	87	89	91	92
TOKEN TEST Total	163	92	114	126	133	(139)	147	148	152		159	
SENTENCE REPETITION	22		8	(10)	11		12	13			14	
REPEATING DIGITS	14		3	4	5		6		(7)		8	
REPEATING DIGITS REVERSE	14		1	2	3		4		5		(6)	
AUTOMATIC SPEECH	4		2		3							(4)
CONFRONTATION NAMING	16		12		14		(15)					16
DESCRIPTION OF FUNCTION	16	(14)	15									16
TACTILE NAMING (Right)	8		(5)		6		7					8
TACTILE NAMING (Left)	8		5	(6)		7						8
GESTURE	16		14	15								(16)
FLUENCY	60	2	4	6	8	9	10	12	13	17	23	(20)
SENTENCE CONSTRUCTION	25		3	6	14	17	19	22	(23)		25	
READING WORDS	8		7									(8)
WORD RECOGNITION	8		7									(8)
READING SENTENCES	7				3							(7)
SENTENCE COMPREHENSION	32	8	20	24		25	28	(29)	31			32
AUTOMATIC WRITING	5		2		4							(5)
SPELLING	24		9	13	16		21		22			(24)
DICTATION	13		8	10			11					(13)
COPYING	11		6	7	8	9	10					(11)
CALCULATION	10		4	6			(7)	8				10
ORAL	20		16	19								(20)
IDEOMOTOR	20			17	18							(20)
CONSTRUCTIONAL	20			8	11	14	16			17	(18) 19	
BLOCK DESIGN	48				(0)	6	8		11		20	
RAVEN'S MATRICES	36	10	(11)	14		16	17	18	20		25	
		<5	10	20	30	40	50	60	70	80	90	95<

CT (17.1.86) Minor changes in deep parietal matter of both hemispheres otherwise normal.

This retired headmaster, with a family history of Huntington's Chorea that had caused the death of his father and his father's brothers and sisters, was moderately demented. He was oriented in person but not place or time. He was a good historian for personal and non personal events. There was no disturbance of concentration but recent memory was impaired.

There was some evidence of failure of recognition as verbal agnosia and memory. Digit recall gave a scaled score of 10. Comprehension was reduced globally. Sentence Repetition appeared more impaired than digit repetition.

Naming produced nominal paraphasia and verbal paraphasia and retrieval of verb forms produced circumlocution. There was evidence of bilateral asteriognosis. A razor in the right hand was miscalled "a sharp point" and in the left hand a gun was called "a firing implement". Fluency was at the tenth percentile of normal and sentence construction produced logorrhoea, but no evidence of disturbance of syntax.

Reading of words and sentences was normal. There was no evidence of loss of comprehension of written instructions, but some of deficits of calculation and drawing ability. There was no oral, ideomotor or ideational apraxia.

Block Design was not completed and the RCPM was at the lower range for a demented population.

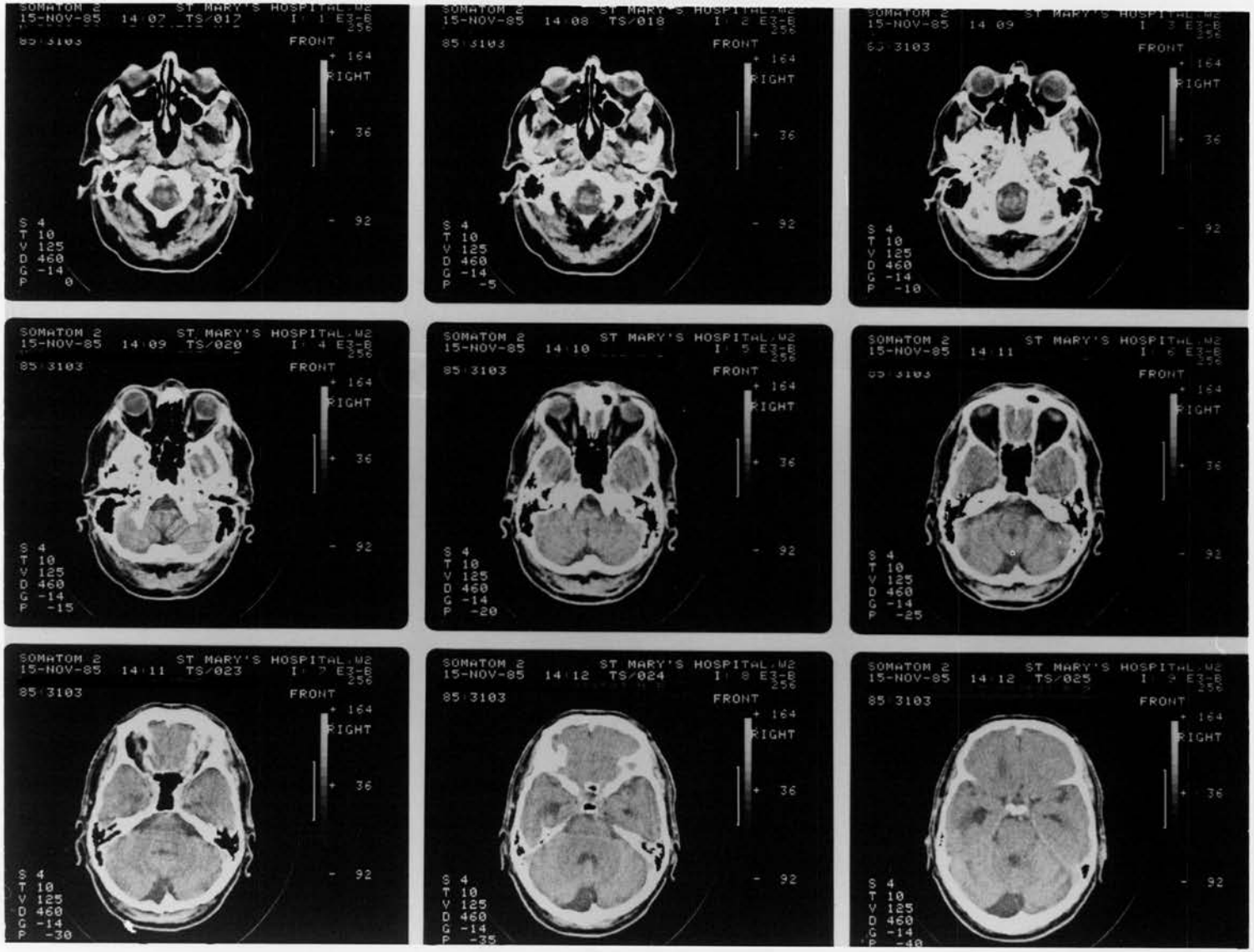
Comment:

Huntington's Chorea as a subcortical dementia has been described by Albert (1978) and Fedio et al (1979) with deficits similar to this case.

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 9 DATE 21.11.85
SEX Male
D of B Age 60 years
DIAGNOSIS Third Ventricular Hydrocephalus

TEST	MAX. SCORE	PERCENTILES <5	10	20	30	40	50	60	70	80	90	95<
RATING SCALE	40	10	13	17	20	22	24	(26)	27	30	32	35
IDENTIFICATION/NAME	8	(6)	7									8
IDENTIFICATION/FUNCTION	8	6	7									(8)
TOKEN TEST Part A-E	67		47	(57)	52	57	60	62	63		65	67
TOKEN TEST Part F	96		56	(59)	66		76	85	86	87	89	91	92
TOKEN TEST Total	163	92	(110)	114	126	133	139	147	148	152		159	
SENTENCE REPETITION	22		8	10	11		12	13			14	(16)
REPEATING DIGITS	14		3	4	5		(6)		7		8	
REPEATING DIGITS REVERSE	14		1	2	(3)		4		5			
AUTOMATIC SPEECH	4		2		3							(4)
CONFRONTATION NAMING	16		(12)		14		15					16
DESCRIPTION OF FUNCTION	16		15									(16)
TACTILE NAMING (Right)	8		5		6		(7)					8
TACTILE NAMING (Left)	8		5	6		7						(8)
GESTURE	16		14	15								(16)
FLUENCY	60	2	4	6	(8)	9	10	12	13	17	23	
SENTENCE CONSTRUCTION	25		3	6	(13)	14	17	19	22	23		25	
READING WORDS	8		7									(8)
WORD RECOGNITION	8		7									(8)
READING SENTENCES	7				3							(7)
SENTENCE COMPREHENSION	32	8	20	24		25	28	29	(31)			32
AUTOMATIC WRITING	5		2		4				(7)			5
SPELLING	24		9	13	16	(19)	21		22			24
DICTATION	13		8	(9)	10		11					13
COPYING	11		6	7	8	9	10					(11)
CALCULATION	10		4	(6)			7	8				10
ORAL	20		16	19								(20)
IDEOMOTOR	20			17	18							(20)
CONSTRUCTIONAL	20			8	11	14	16			17	19	(20)
BLOCK DESIGN	48				0	6	8		11		20	(34)
RAVEN'S MATRICES	36	10	11	14		16	17	18	20		25	(30)
		<5	10	20	30	40	50	60	70	80	90	95<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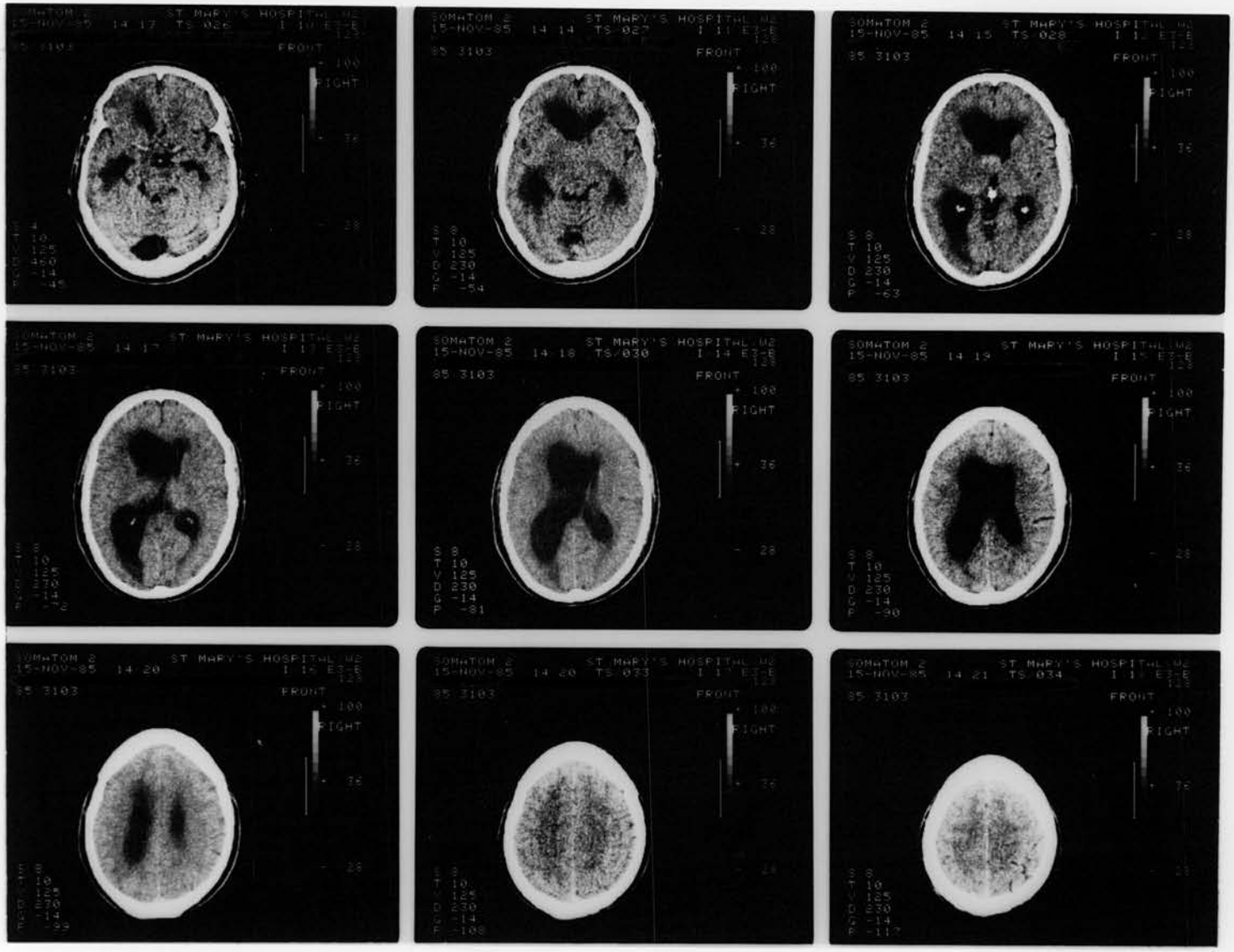
ST MARY'S HOSPITAL W2
15-NOV-85 14:07 TS/012
85 3103
FRONT
RIGHT
+ 164
+ 36
- 92
S 4
T 10
V 125
D 460
G -14
P 0
SOMATOM 2
ST MARY'S HOSPITAL W2
15-NOV-85 14:08 TS/018
85 3103
FRONT
RIGHT
+ 164
+ 36
- 92
S 4
T 10
V 125
D 460
G -14
P -5
SOMATOM 2
ST MARY'S HOSPITAL W2
15-NOV-85 14:09
85 3103
FRONT
RIGHT
+ 164
+ 36
- 92
S 4
T 10
V 125
D 460
G -14
P -10
SOMATOM 2
ST MARY'S HOSPITAL W2
15-NOV-85 14:09 TS/020
85 3103
FRONT
RIGHT
+ 164
+ 36
- 92
S 4
T 10
V 125
D 460
G -14
P -15
SOMATOM 2
ST MARY'S HOSPITAL W2
15-NOV-85 14:10
85 3103
FRONT
RIGHT
+ 164
+ 36
- 92
S 4
T 10
V 125
D 460
G -14
P -20
SOMATOM 2
ST MARY'S HOSPITAL W2
15-NOV-85 14:11
85 3103
FRONT
RIGHT
+ 164
+ 36
- 92
S 4
T 10
V 125
D 460
G -14
P -25
SOMATOM 2
ST MARY'S HOSPITAL W2
15-NOV-85 14:11 TS/023
85 3103
FRONT
RIGHT
+ 164
+ 36
- 92
S 4
T 10
V 125
D 460
G -14
P -30
SOMATOM 2
ST MARY'S HOSPITAL W2
15-NOV-85 14:12 TS/024
85 3103
FRONT
RIGHT
+ 164
+ 36
- 92
S 4
T 10
V 125
D 460
G -14
SOMATOM 2
ST MARY'S HOSPITAL W2
15-NOV-85 14:12 TS/025
85 3103
FRONT
RIGHT
+ 164
+ 36
- 92
S 4
T 10
V 125
D 460
G -14



CT (15.11.85) Hyperdense mass within the anterior part of the third ventricle. Its boundary is causing marked obstruction of the left lateral ventricle. There is mild hydrocephalus of the right lateral ventricle.

This man was oriented in person and time but not place. He was a good historian for personal but not non personal events. Concentration was unimpaired but recent memory and immediate memory were reduced giving a digit recall scaled score of 8. Sentence repetition was less impaired.

There was evidence of verbal agnosia, comprehension was globally reduced although written comprehension was spared.

Naming produced nominal paraphasia and right hand tactile naming produced semantic confusion. Fluency was reduced to the lower limits of normal and Sentence Construction failed because one response was perseverated on to the next test item.

Reading and Reading Comprehension were virtually unimpaired but writing was marked with cancellations (see attached). There was dyscalculia but no disturbance of praxis.

Block Design gave a scaled score of 13 and the RCPM was at the sixtieth percentile of normal.

Comment:

Communicating hydrocephalus has been mentioned by Trimble (1981) but its effect on language is unreported. This case produced mild to moderate dementia affecting recognition and memory which dampened comprehension.

BELL

FORCHORCH

RAZOR

GLASSES

PIPE

WHISTLE

TWEEZERS

WATCH

THIS IS A VERY NICE DAY

THIS BOOK BUILDING HAS BUILDT LAST YEAR

I AM VERY HUNGRY

THE COLOUR OF THESE WALLS IS GREEN

SUBTEST SUMMARY PROFILE (APHASIA)

NAME No. 10 DATE.....27.7.84
SEX Male
D of B Age 54 years
DIAGNOSIS Pre-Frontal Leucotomy (1949)

TEST	MAX. SCORE	PERCENTILES <5	10	20	30	40	50	60	70	80	90	95<
RATING SCALE	40											40
IDENTIFICATION/NAME	8	4	5	7								(8)
IDENTIFICATION/FUNCTION	8	4	5	7								(8)
TOKEN TEST Part A-E	67	2	5	12	20	30	45	52	(58)	60	62	67
TOKEN TEST Part F	96		0	20	35	50	61	65	67	75	(78) 85	96
TOKEN TEST Total	163	0	11	16	65	85	106	110	130	(136) 140	150	160
SENTENCE REPETITION	22			(0)	1	4	8	9	10	12	13	16
REPEATING DIGITS	14			(0)	1	2	3	4	5	6	7	9
REPEATING DIGITS REVERSE	14	(0)					1		2		4	7
AUTOMATIC SPEECH	4		(0)		2							4
CONFRONTATION NAMING	16	(0)	1	4	6	9	12	13	15			16
DESCRIPTION OF FUNCTION	16		(0)	3	7	9	13	14	15			16
TACTILE NAMING (Right)	8						(0)	2	4	6	7	8
TACTILE NAMING (Left)	8	(0)	1	2	3	5	6	7				8
GESTURE	16	1	12	13		15						(16)
FLUENCY	60					(0)	2	3	5	10	26	32
SENTENCE CONSTRUCTION	25							(0)	14	16	20	25
READING WORDS	8	(0)			1							8
WORD RECOGNITION	8	6	7									(8)
READING SENTENCES	7	(0)					3		6			7
SENTENCE COMPREHENSION	32		2	5	20	(23)	26	27	29	30	31	32
AUTOMATIC WRITING	5		0				(3)	4				5
SPELLING	24					0	3	14	18	19	22	(24)
DICTATION	13					0	4	9	11	(12)		13
COPYING	11		2	8	9	10						(11)
CALCULATION	10	(0)		1		3	5	6	7			10
ORAL	20	10	(11)	14	15	16	18		19			20
IDEOMOTOR	20	12	15	17		(19)						20
CONSTRUCTIONAL	20	12	18		(19)							20
BLOCK DESIGN	48		0	2	(4) 6	11	18	20	22	25		30
RAVEN'S MATRICES	36	10	15	(16) 20	22		25	27		30		35
		<5	10	20	30	40	50	60	70	80	90	95<

CT (14.8.85) Bilateral frontal scars extending to the tips of the anterior horns of the lateral ventricles to the periphery. Bi-frontal surgical bone defects consistent with the history of previous pre-frontal leucotomy.

History Acute psychotic illness at 16 years, pre-frontal leucotomy 1949, patient became mute, detached withdrawn, self absorbed and manneristic.

This patient was mute with no vocalisation but no deficit of ideational apraxia, spelling or writing to dictation or copy. Auditory memory was reduced to three units and there was global comprehension loss on the Token Test. Written instructions did not improve comprehension. Both tests of non verbal intelligence were reduced. Block Design gave a scaled score of 3 and the RCPM was below normal limits.

Comment:

This patient has preserved abilities represented in the posterior cortex of basic comprehension, gestural and spatial skills, subvocal reading and writing ability and a preserved phonemic system that can be transcoded graphically. He has frontal signs including oral apraxia that may lead to speechlessness although disintegration of motor speech disorganisation or elected mutism may account for his silence. Basomedial regions of the frontal lobe are involved in alerting states while the lateral convexity of the posterior and motor cortex allow for the organisation and execution of purposive activity. Lesions for these areas produce perseveration and movement disturbance and lesions to the orbitomedial regions, linked to the reticular formation and limbic system can produce "pseudo depressed" or "pseudo-psychopathic" states respectively (Blumer and Benson 1975). Disturbances of the pre-frontal cortex produce memory and cognitive deficit (Luria 1970).

SUBTEST SUMMARY PROFILE (NORMALS)

NAME No. 11 DATE.....29.4.84
SEX Male 1.2.85
D of B Age 68 years
DIAGNOSIS Pseudo-dementia

TEST	MAX. SCORE	PERCENTILES 5	10	20	30	40	50	60	70	80	90	95
RATING SCALE	40	(36)	(38)									40
IDENTIFICATION/NAME	8		7									(8)
IDENTIFICATION/FUNCTION	8		7									(8)
TOKEN TEST Part A-E	67	64	65		(66)							(67)
TOKEN TEST Part F	96		90	(92)			95					(96)
TOKEN TEST Total	163		154	(159)	160		(162)					163
SENTENCE REPETITION	22		12			14	15	16	17	(18)	(19)	20
REPEATING DIGITS	14		5		6		7	(8)	9	(10)	11	12
REPEATING DIGITS REVERSE	14	3	4	5	(6)		(7)	8	9	10		12
AUTOMATIC SPEECH	4											(4)
CONFRONTATION NAMING	16	14				15						(16)
DESCRIPTION OF FUNCTION	16		10									(16)
TACTILE NAMING (Right)	8				7							(8)
TACTILE NAMING (Left)	8				7							(8)
GESTURE	16	16										(16)
FLUENCY	60	(8)	(12)	20	27	31	33	35	36	40	44	50	60
SENTENCE CONSTRUCTION	25	23		24								(25)
READING WORDS	8											(8)
WORD RECOGNITION	8											(8)
READING SENTENCES	7											(7)
SENTENCE COMPREHENSION	32		(31)									(32)
AUTOMATIC WRITING	5											(5)
SPELLING	24	(21)				(23)						24
DICTATION	13	11		12								(13)
COPYING	11	9		10								(11)
CALCULATION	10	8	9									(10)
ORAL	20											(20)
IDEOMOTOR	20											(20)
CONSTRUCTIONAL	20		18		19							(20)
BLOCK DESIGN	48	15	(20)	(24)	25	27	28	30	35	40		42
RAVEN'S MATRICES	36	19	20	23	(25)	27	28	(29)	31	33	34	35	36
		5	10	20	30	40	50	60	70	80	90	95

CT (4.6.84) No major infarcts seen.

EEG (5.6.84) Medium to low amplitude throughout, a little slowing of the left temporal region is the only abnormality seen.

This farmer had no real disturbance of comprehension or expressive language. There was no disturbance of reading, writing, calculation or praxis. He was a good historian and well oriented. Digit recall gave a scaled score of 12 and scores for non verbal intelligence gave a scaled score of 10 for Block Design and the thirtieth percentile for the RCPM. Fluency was reduced to the fifth percentile for normal.

On reassessment a year later there were no real errors of comprehension or orientation. Expressive language, calculation, and praxis were also free of error. Digit recall gave a scaled score of 40 and Block Design a scaled score of 9 while the RCPM was at the fiftyfifth percentile.

Comment:

Depression affecting cognitive performance and masquerading as dementia is well recognised (Roth 1955, Miller 1977, Trimble 1981, Pearce 1984). Wells (1979) discussed the condition "Pseudodementia". A differential diagnosis is critical, the conditions have different treatments and different outcomes (Roth 1955, Miller 1977). At least 15% of the depressed elderly will present cognitive deficits (Roth 1978) that can include memory and language disturbance (Golper and Binder 1981). Affective disorders are also described in dementing populations (Roth 1955). Aphasias, apraxias and agnosias are rare in depression (Post 1975). Depressed patients do not perseverate but are subject to lability (Post 1975), and many cognitive deficits resolve with antidepressants. Many of this man's deficits improved after a years treatment with antidepressants as profiled here.Generally scores have remained stable unlike degenerative dementias.

SUBTEST SUMMARY PROFILE (NORMALS)

NAME No. 12 DATE.....7.8.84
SEX Female
D of B Age 45 years
DIAGNOSIS Vertebro-basilar Ischaemic Disorder

TEST	MAX. SCORE	PERCENTILES										
		<5	10	20	30	40	50	60	70	80	90	95<
RATING SCALE	40	(37)	38									40
IDENTIFICATION/NAME	8		7									(8)
IDENTIFICATION/FUNCTION	8		7									(8)
TOKEN TEST Part A-E	67	64	65		66							(67)
TOKEN TEST Part F	96		90	92			95					(96)
TOKEN TEST Total	163		154	159	160		162					(163)
SENTENCE REPETITION	22		12			(14)	15	16	17	18		20
REPEATING DIGITS	14		5		(6)		7	8	9	10	11	12
REPEATING DIGITS REVERSE	14	3	4	5	(6)		7	8	9	10		12
AUTOMATIC SPEECH	4											(4)
CONFRONTATION NAMING	16	14				15						(16)
DESCRIPTION OF FUNCTION	16		10									(16)
TACTILE NAMING (Right)	8				7							(8)
TACTILE NAMING (Left)	8				7							(8)
GESTURE	16	16										(16)
FLUENCY	60	10	20	27	31	33	35	36	(39)	40	44	50 60
SENTENCE CONSTRUCTION	25	23		24								(25)
READING WORDS	8											(8)
WORD RECOGNITION	8											(8)
READING SENTENCES	7											(7)
SENTENCE COMPREHENSION	32		31									(32)
AUTOMATIC WRITING	5											(5)
SPELLING	24					23						(24)
DICTATION	13	11		12								(13)
COPYING	11	9		10								(11)
CALCULATION	10	8	(9)									10
ORAL	20											(20)
IDEOMOTOR	20											(20)
CONSTRUCTIONAL	20		18		19							(20)
BLOCK DESIGN	48	15	20		25	27	28	30	35	40		(42)
RAVEN'S MATRICES	36	19	20	23	25	27	28	31	33	34	35	(36)
		<5	10	20	30	40	50	60	70	80	90	95<

CT Normal. Duplex Dopplers of carotids and vertebral arteries all normal. Normal haemotological, biochemical and CSF screening

There was no evidence of dysarthria, verbal agnosic, receptive or expressive language disorder, nor any disorder of reading, writing or praxis. Scores for Block Design yielded a scaled score of 15 while the RCPM were at the ninetififth percentile of normal. Word Fluency as a cognitive measure was at the sixtieth to seventieth percentile of normal.

All scores for auditory memory were selectively reduced, Sentence Repetition to the fortieth percentile of normal and Digit Repetition to the thirtieth percentile of normal yielding a scaled score of 8.

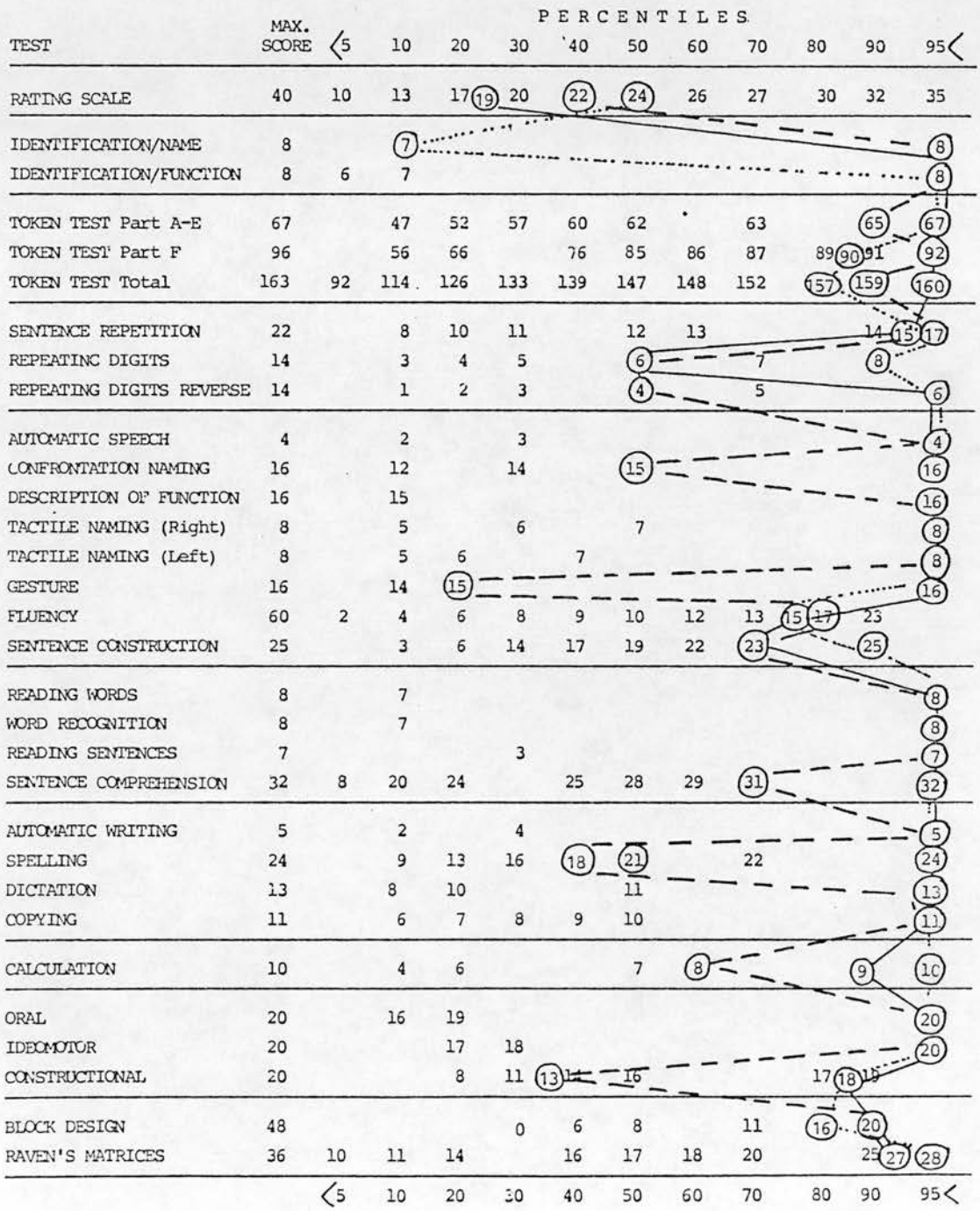
Comment:

This is a specific disorder of auditory memory when material is briefly held in storage. Luria (1970) described the syndrome of Acoustic Aphasia in lesions to the inferior temporal lobes and Penfield and Matthieson (1974) described memory loss following lesions of the hippocampal gyri. Rivera et al (1974) described amnesia following vertebro basilar arterial insufficiency after ischaemia of the para amygdaloid areas and other limbic and cortical structures irrigated by the posterior cerebral circulation. Patients with VBI may be considered as part of the natural history of dementia (Perez et al 1975).

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 13
SEX Female
D of B Age 70 years
DIAGNOSIS Multi Infarct Dementia

DATE.....15.1.82
- - - - - - - - - - 20.5.83
_____ 2.7.84



CT (25.1.82) Numerous CSF lakes are evident particularly in the right parietal region. These suggestions are of degenerative vascular changes rather than Alzheimer's disease.

This lady was moderately demented but gave a good personal history with unimpaired concentration.

There was evidence of agnosia in identification tasks but comprehension was within normal range. Digit Repetition gave a scaled score of 13. There were no errors of expressive language or reading and writing comprehension, or calculation or praxis however both Fluency and constructional abilities were diminished. Nevertheless, Block Design gave a scaled score of 8 and the RCPM was at the fiftieth percentile of normal.

On second assessment the lady was oriented in all but time with reduced comprehension and poor non personal history. Comprehension was within normal range and digit recall gave a scalled score of 8.

Expressive language gave one semantic paraphasia, Fluency was marginally diminished and there was no real evidence of syntactic disturbance. Reading and writing produced no real errors. There was no disturbance of praxis but constructional Apraxia became evident and Block Design gave a scaled score of 9. The RCPM was marginally worse.

On third assessment the patient remained disoriented in time, demonstrated impaired recent memory and was unable to give a non personal history. Comprehension was within normal limits and digit repetition gave a scaled score of 9.

Expressive language produced one anomic error but Fluency and Sentence Construction remained intact. Diminished scores were violations of time constraint.

Reading and writing were unimpaired, there was one error of complex calculation and none of praxis apart from the inability to gesture the use of one object.

Drawing ability improved, Block Design gave a scaled score of 9 and the RCPM score lay at the fortieth percentile of normal.

Comment:

Despite a moderate dementia this patient's language abilities appeared diminished on the second assessment but on the first and third assessment generally produced complete scores. It appears to be the picture of a non progressive disorder.

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 14 DATE.....12.1.83
SEX Male
D of B Age 71 years
DIAGNOSIS Multi Infarct Dementia

TEST	MAX. SCORE	PERCENTILES 5	10	20	30	40	50	60	70	80	90	95
RATING SCALE	40	10	(12) 13	17	20	22	24	26	27	30	32	35
IDENTIFICATION/NAME	8	(6)	7									8
IDENTIFICATION/FUNCTION	8	(5)	7									8
TOKEN TEST Part A-E	67		(47)	52	57	60	62		63		65	67
TOKEN TEST Part F	96		56	(60) 66		76	85	86	87	89	91	92
TOKEN TEST Total	163	92	(107) 114	126	133	139	147	148	152		159	
SENTENCE REPETITION	22	(2)	8	10	11		12	13			14	
REPEATING DIGITS	14	(2)	3	4	5		6		7		8	
REPEATING DIGITS REVERSE	14		(1)	2	3		4		5			
AUTOMATIC SPEECH	4		2		(3)							4
CONFRONTATION NAMING	16	(5)	12		14		15					16
DESCRIPTION OF FUNCTION	16	(6)	15									16
TACTILE NAMING (Right)	8	(4)	5		6		7					8
TACTILE NAMING (Left)	8	(3)	5	6		7						8
GESTURE	16		14	15								(16)
FLUENCY	60	2	4	(5) 6	8	9	10	12	13	17	23	
SENTENCE CONSTRUCTION	25	(0)	3	6	14	17	19	22	23		25	
READING WORDS	8		7									(8)
WORD RECOGNITION	8		7									(8)
READING SENTENCES	7				3							(7)
SENTENCE COMPREHENSION	32	8	(14) 20	24		25	28	29	31			32
AUTOMATIC WRITING	5	(1)	2		4							5
SPELLING	24	(3)	9	13	16		21		22			24
DICTATION	13	(0)	8	10			11					13
COPYING	11	(1)	6	7	8	9	10					11
CALCULATION	10	(2)	4	6			7	8				10
ORAL	20		16	(17) 19								20
IDEOMOTOR	20	(15)		17	18							20
CONSTRUCTIONAL	20			8	11	(13) 14	16			17	19	
BLOCK DESIGN	48				(0)	6	8		11		20	
RAVEN'S MATRICES	36	10	11	14		16	(17)	18	20		25	
		5	10	20	30	40	50	60	70	80	90	95

CT (2.2.83) There is moderate enlargement of the lateral ventricles There is shrinkage in the sylvian regions, more markedly on the left. The appearances are of a generalised cerebral atrophy more marked on the left probably with a degenerative vascular basis.

This man was disoriented in place and time and unable to give a non personal history. Recent memory was reduced as was the ability to concentrate and recite automaticisms.

Comprehension was reduced at single word level and globally for instructions that were presented verbally or in writing. Auditory memory gave a scaled score of 3 for digit recall and was severely impaired when repeating sentences.

Expressive language produced phonemic paraphasia, verbal paraphasia and nominal paraphasia as well as circumlocution.

Fluency was below normal range and syntax was impaired. The ability to read was retained but dissociated from understanding.

There was no evidence of an ideational apraxia but there were deficits of oral and ideomotor programming. There were also deficits of drawing ability.

Writing ability was virtually non existent and calculation almost extinct. No score was achieved for Block Design and the RCPM was at the fiftieth percentile for a dementing population.

This man was reassessed a year later. He was only able to read single words, repeat his name and gesture one item.

Comment:

This is severe dementia with errors reflecting disturbances of the periSylvian borders. A language sample is attached which reflects a lack of cohesion and a paraphasia.

No. 36 Age 71 years

LANGUAGE SAMPLE

Therapist: Tell me about your days in the navy

Patient: oh don't tell me

I had forty

eh ah no, it was three, three lots

and I went out with the Navy

and was there for nearly four years, four years

and then I got in all right with the Navy

so then all of a sudden it was a matter of either you do or you don't

so that was it back again

they said the same thing

but I said

they were going out for the game anyway

I wasn't going to do any more in the navy

because I'd got put out

well that's what it is

you know for year

she, he says

he says

well that'll be fine then Peter

he says

he used to put me to the bus

and then just before that the other ship went

she went right down with the whole ship

I mean eight percent

and the rest were worn

I was left with not a few of them

there were seventy back there

but then there was eight hundred in all or eighty at the other end

and it just went down like that

and there was a German thingme a German thingme car at the door

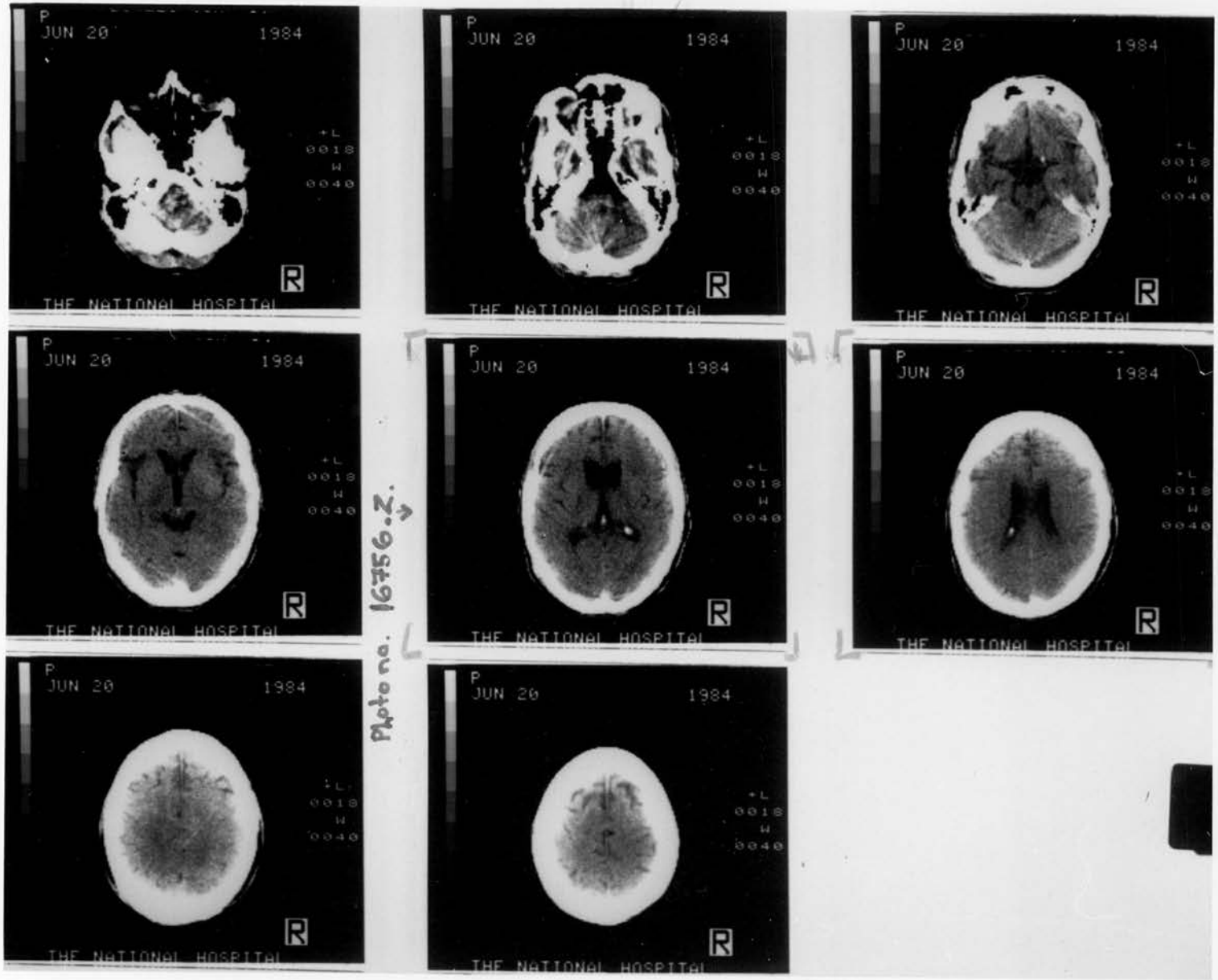
Note:

This man's ship was sunk off Malta in the second world war with the loss of 800 lives. The "thingme" and "car" refer as paraphasias to the ship. The "8%"is a paraphasia for 800. The discourse reflects poor cohesion of the relationship of one phrase to another.

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 15 DATE.....3.5.84
SEX Male
D of B Age 43 years
DIAGNOSIS Pick's Disease

TEST	MAX. SCORE	PERCENTILES <5	10	20	30	40	50	60	70	80	90	95<
RATING SCALE	40	10	13	17	20	22	24	26	27	30	33	35
IDENTIFICATION/NAME	8		7									8
IDENTIFICATION/FUNCTION	8	6	7									8
TOKEN TEST Part A-E	67		47	52	54	57	60	62		63		65	67
TOKEN TEST Part F	96		56	66	70	76	85	86	87	89	91	92
TOKEN TEST Total	163	92	114	124	126	133	139	147	148	152	159	
SENTENCE REPETITION	22	7	8	10	11		12	13			14	
REPEATING DIGITS	14		3	4	5		6		7		8	
REPEATING DIGITS REVERSE	14		1	2	3		4		5			
AUTOMATIC SPEECH	4		2		3							4
CONFRONTATION NAMING	16		12		14		15					16
DESCRIPTION OF FUNCTION	16	0	15									16
TACTILE NAMING (Right)	8		5		6		7					8
TACTILE NAMING (Left)	8		5	6		7						8
GESTURE	16		14	15								16
FLUENCY	60	2	4	6	8	9	10	12	13	17	23	
SENTENCE CONSTRUCTION	25	0	3	6	14	17	19	22	23		25	
READING WORDS	8		7									8
WORD RECOGNITION	8		7									8
READING SENTENCES	7				3				4			7
SENTENCE COMPREHENSION	32	8	20	24		25	28	29	31			32
AUTOMATIC WRITING	5		2		4							5
SPELLING	24		9	13	16		21		22			24
DICTATION	13	7	8	10			11					13
COPYING	11		6	7	8	9	10					11
CALCULATION	10		4	6			7	8				10
ORAL	20	8	16	19								20
IDEOMOTOR	20			17	18							20
CONSTRUCTIONAL	20			8	11	14	16			17	19	20
BLOCK DESIGN	48				0	6	8		11		20	38
RAVEN'S MATRICES	36	10	11	14		16	17	18	20		25	26
		<5	10	20	30	40	50	60	70	80	90	95<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THE NATIONAL HOSPITAL
Photo no. 16756.Z.

CT (20.7.84) Mild Atrophy.

EEG (25.1.83) Low voltage, normal frequency. No focal signs. No indication of temporal cortex dysfunction.

Psychometry Verbal IQ 70, performance IQ 95.

Medical History Depression, anxiety, withdrawal impulsiveness, intellectual deterioration, violence to own children.

This 43 year old accountant was oriented, a good historian with automaticisms intact but a loss of concentration and recent memory. Digit recall gave a scaled score of 6.

Comprehension was reduced for verbal and written information. Responses on Part F of the Token Test were perseverated.

Naming was intact but there was an inability to recall verb forms.

Word Fluency was at the fifth percentile for normals when measured by the FAS Test, the responses being perseverative or obscenities. Sentence construction was impaired by a failure to transfer the stimulus words into the task. Reading and comprehension of words was unimpaired but there was a failure to read or act appropriately on written instructions.

There was evidence of dysgraphia and disturbed ability to copy. There was no disturbance of calculation. Examples are given as Figure 10a in Chapter 5.6.

There was evidence of oral apraxia. Ideomotor and ideational praxis were preserved but drawing ability showed an overscoring of lines and perseveration of the instruction on to the task.

Block Design yielded a scaled score of 12 and the RCPM was at the thirtyfifth percentile for normals.

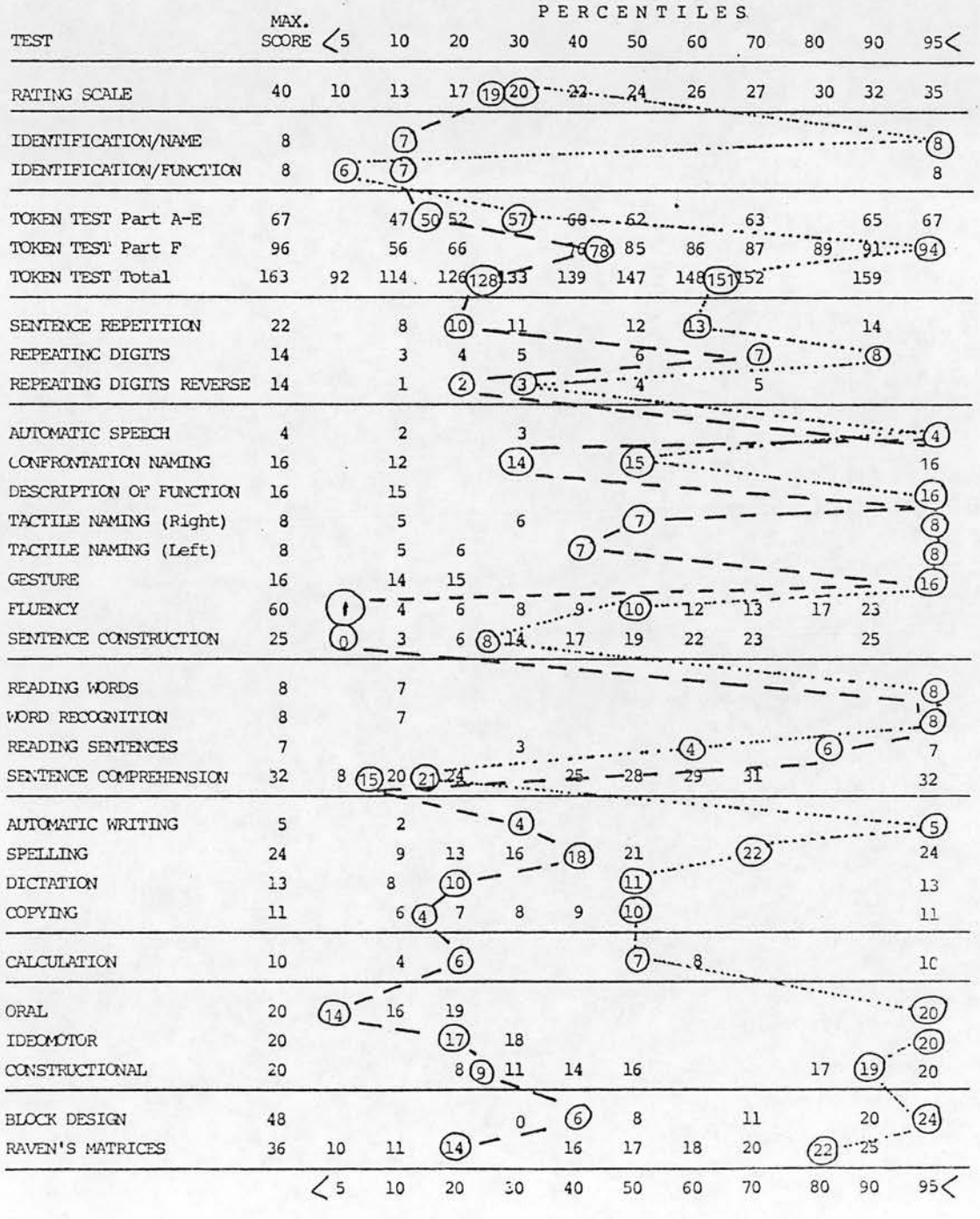
Comments:

The performance reflects difficulties of initiation, appropriateness and quality. Speech was preceded by motor steriotypes used as starters to overcome a lack of dynamism. There was an inability to inhibit inappropriate responses and a failure of fluency. The symptoms of rigidity of thought, perseveration and impairment of abstract attitude are all associated with frontal lobe pathology. The accompanying CT scan indicated selected atrophy of the anterior superior frontal area corresponding to the tertiary supplementary motor area.

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 16
SEX Male
D of B Age 64 years
DIAGNOSIS Pick's Disease

DATE 30.5.83
_____9.8.84



CT (10.2.83) Slight enlargement of lateral ventricles minimally asymmetrical, the left being larger. Moderate generalised atrophy.

This retired nautical engineer was moderately demented. On first assessment there was evidence of attention disorder on digit repetition which gave a scaled score of 9 but was dampened by reverse operations.

Comprehension was diminished by failure to operate on length of instruction and concepts in written instructions.

Expressive language tasks produced one paraphasia but Fluency tasks produced perseveration and violation of the behavioural constraints the test demands. These violations included perseveration. Sentence Construction also produced perseverative responses.

There were errors in oral reading, complex calculation and non verbal intelligence. Block Design gave a scaled score of 10 and the RCPM was at the twentieth percentile of normal.

On second assessment there was evidence of verbal agnosia and increased memory loss as digit recall gave a scaled score of 8 again with a gap between forward and reversed operations indicating concentration deficit.

Comprehension was globally reduced and expressive speech marked with mild nominal paraphasia. Fluency yielded one response which was an obscenity and there was no response to Sentence Construction.

There was evidence of oral and ideomotor apraxia and tests of constructional apraxia showed loss of perspective and detail with perseveration. Block Design gave a scaled score of 5 and fell with the RCPM.

Comments:

These results can be interpreted qualitively as Pick's disease. There is a failure of concentration and a dramatic test retest gap.

BELL

RAZOR

WHISTLE

PIPE

TORCH

WATCH

GLASSES.

TWISTERS.

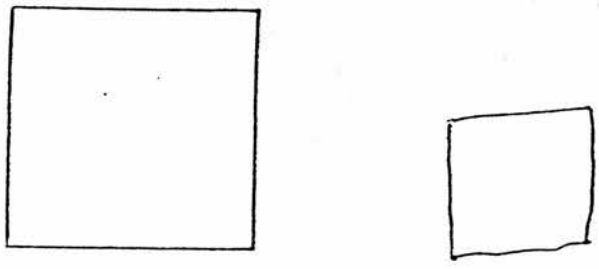
THIS IS VERY NICE DAY

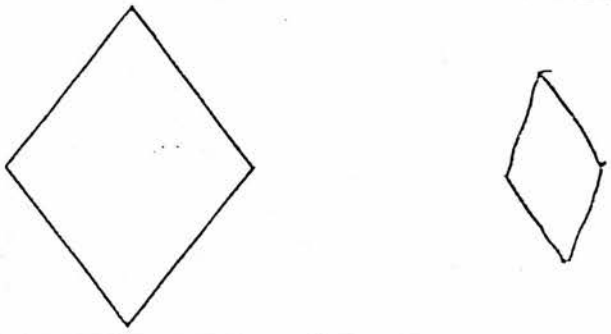
THIS BRICK BUILDING WAS BUILD LAST YEAR.

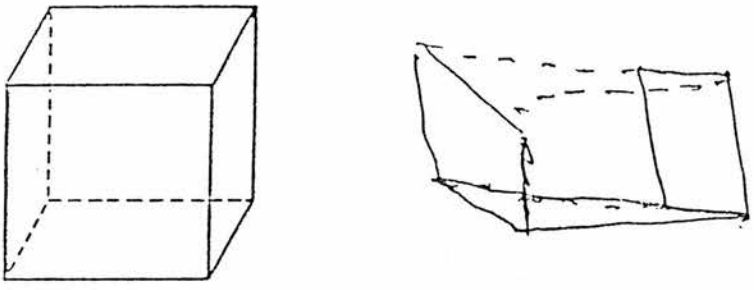
I'M AM VERY HUNGRY

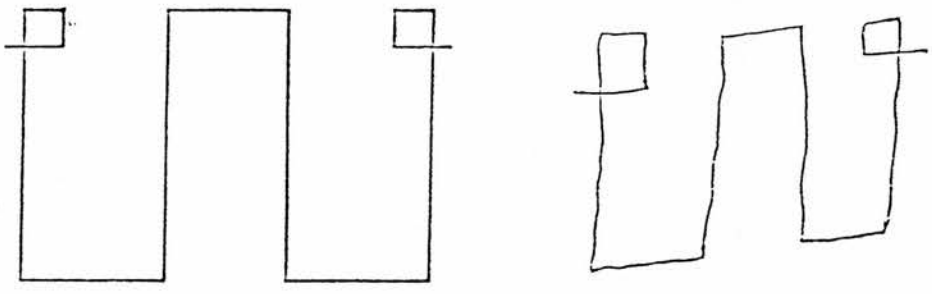
THE COLOUR OF THE

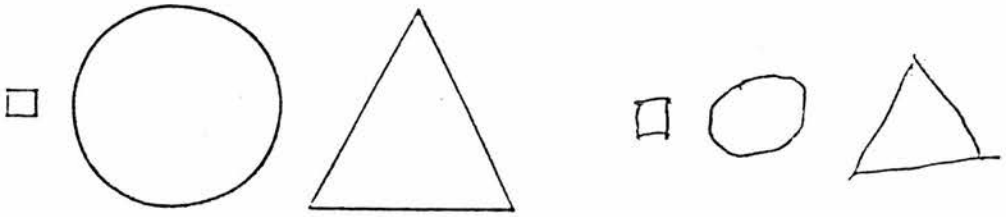
WALLS IS GREEN.

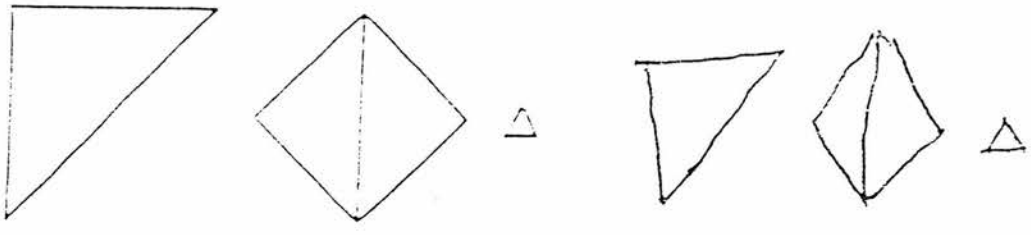












Test 2

BELL

TORCH

RASOR

WHISTE

PIPE

WATCH

GLASSES

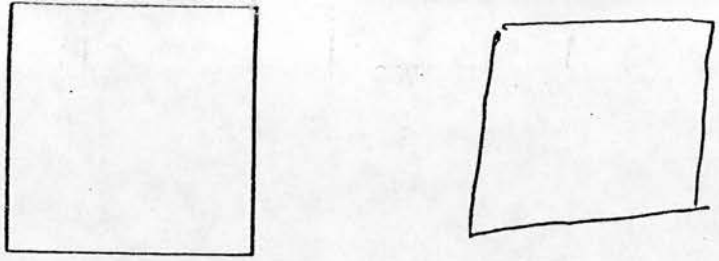
TEWST

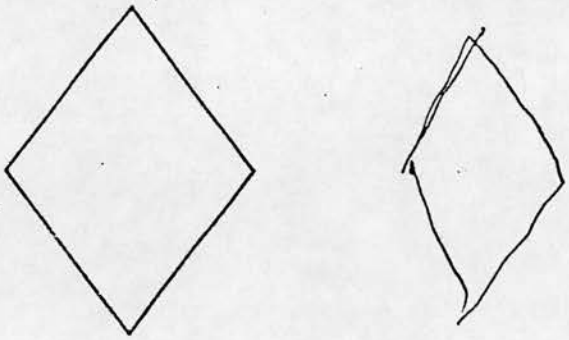
THIS RS VERY RS TOBS

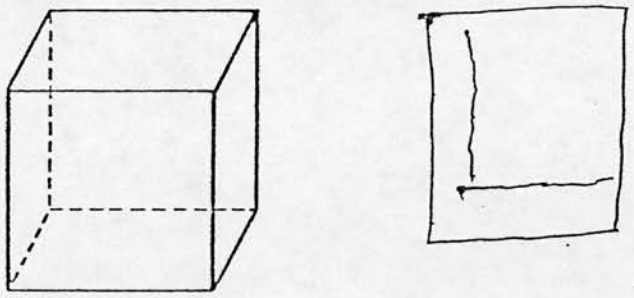
THIS BRICK BUILDING WAS BUILT

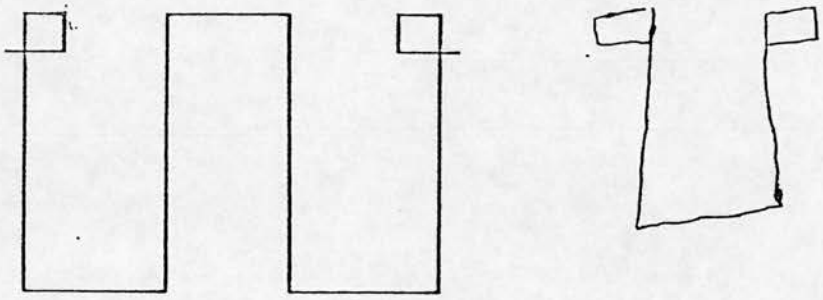
LAST YEAR

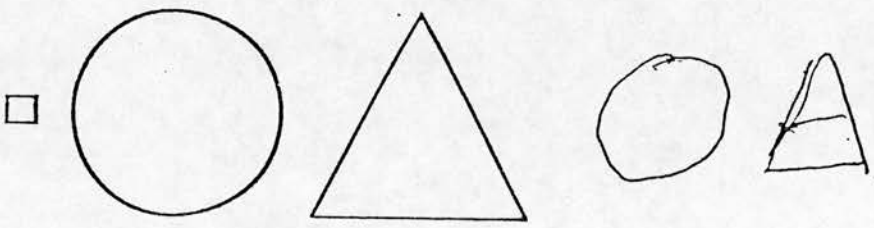
& BUILD

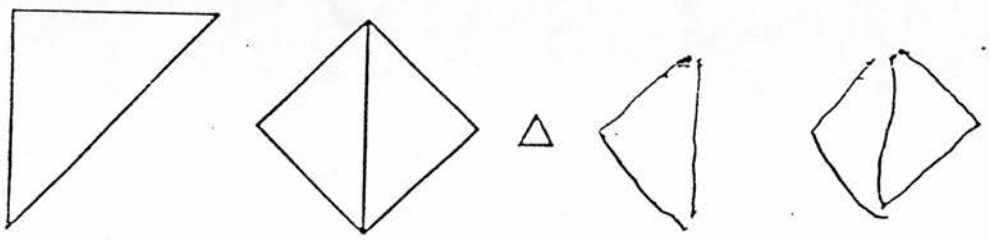












SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 17 DATE 14.5.85
SEX Female
D of B Age 60 years
DIAGNOSIS Alzheimer Type Dementia

TEST	MAX. SCORE	<5	10	20	30	40	50	60	70	80	90	95<
		PERCENTILES										
RATING SCALE	40	10	13	(16)	17	20	22	24	26	27	30	32	35
IDENTIFICATION/NAME	8		7										(8)
IDENTIFICATION/FUNCTION	8	6	(7)										8
TOKEN TEST Part A-E	67		47	52	57	60	(61)	62		63		65	67
TOKEN TEST Part F	96		56	(64)	66		76	85	86	87	89	91	92
TOKEN TEST Total	163	92	114	(125)	126	133	139	147	148	152		159	
SENTENCE REPETITION	22		8	10	11		(12)	13				14	
REPEATING DIGITS	14		3	4	5		6		(7)			8	
REPEATING DIGITS REVERSE	14		(1)	2	3		4		5				
AUTOMATIC SPEECH	4		2		3								(4)
CONFRONTATION NAMING	16	(10)	12		14		15						16
DESCRIPTION OF FUNCTION	16	(13)	15										16
TACTILE NAMING (Right)	8		5		6		7						(8)
TACTILE NAMING (Left)	8		5	6		(7)							8
GESTURE	16	(13)	14	15									16
FLUENCY	60	2	4	6	8	(9)	10	12	13	17	23		
SENTENCE CONSTRUCTION	25		(3)	6	14	17	19	22	23		25		
READING WORDS	8		7										(8)
WORD RECOGNITION	8		7										(8)
READING SENTENCES	7				3								(7)
SENTENCE COMPREHENSION	32	8	20	(24)		25	28	29	31				32
AUTOMATIC WRITING	5		(2)		4								5
SPELLING	24	(3)	9	13	16		21		22				24
DICTATION	13		(8)	10			11						13
COPYING	11		6	7	8	9	10						(11)
CALCULATION	10	(3)	4	6			7	8					10
ORAL	20		16	19									(20)
IDEOMOTOR	20	(14)		17	18								20
CONSTRUCTIONAL	20			8	(11)	14	16				17	19	
BLOCK DESIGN	48				0	(2)	6	8		11		20	
RAVEN'S MATRICES	36	10	11	(14)		16	17	18	20			25	
		<5	10	20	30	40	50	60	70	80	90	95<

CT (13.5.85) No abnormality noted.

EEG (4.8.85) There is a fairly diffuse irregular low to moderate amplitude seen throughout the record. There is a striking bilateral increase in slow rhythm with little alpha rhythm. The record is consistent with Alzheimer's disease.

The patient was oriented in person but not place nor time. She gave an adequate personal history but a poor non personal history and demonstrated reduced recent memory and concentration.

The Token Test was below normal limits for comprehension of concepts while the ability to comprehend written instructions was also diminished. Digit recall gave a scaled score of 9 with a difference in the scores reflecting concentration loss.

Expressive language contained circumlocution (tweezers were "for picking up small things", a brush was "for painting walls" and a torch was "a flash thing"). There was nominal paraphasia (matches were "cigarettes" and a fountain pen was a "nib"). Verb forms were also circumlocated and this drive to associate paraphasically was evident in tasks of sentence construction. Asked to produce a sentence from the words "hot" and "summer", the response was "bikini",or "drive", "street" and "car" was "crossing the road". Fluency tasks gave similar results, a word stream from F produced "fish, skate fish, frying".

Reading demonstrated a dissociation from comprehension beyond one word level, writing produced orthographic errors, dictation was marked with substitution but the ability to copy was preserved.

There was no oral apraxia but there was evidence of ideational apraxia and ideomotor apraxia. Constructional tasks had "closing in" effects on figures demanding left right representation. The last figure

was perseverated (see attached).

Block Design gave a scaled score of 3 and with the RCPM was below the thirtyfifth percentile for dementia.

Comments:

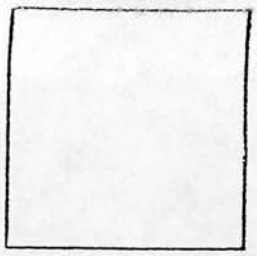
The results demonstrate the language pathology of moderate to severe ATD with receptive and expressive language loss, perseveration, paraphasia and a dissociation between reading and comprehension. There is also a memory disorder and reduced measures of verbal and non verbal intelligence.

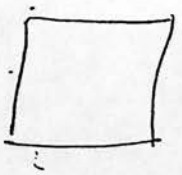
For I
Bell
P.P.
Wistst.
Wat
To
Glasses.
Rasor
Twesers.

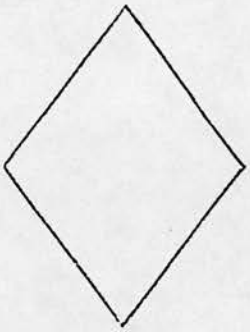
This is a very nice 9.

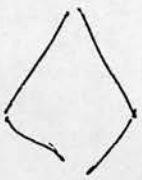
This Brick Building was bulet
this year.

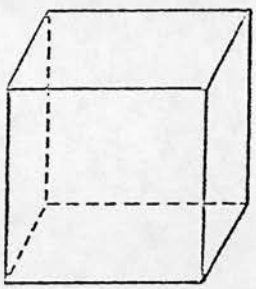
I am very Hungry
The colour of the walls is green.

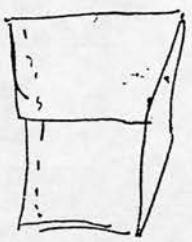


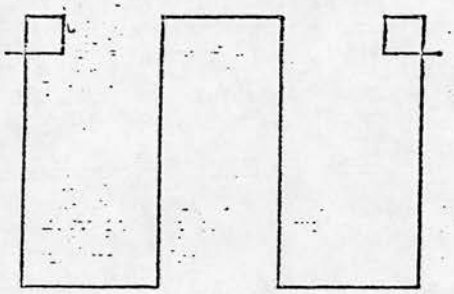


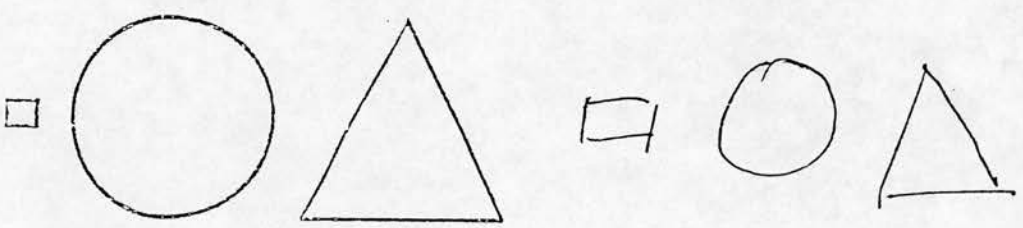


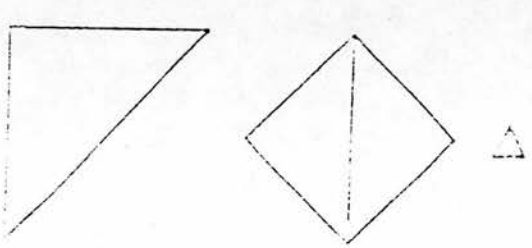


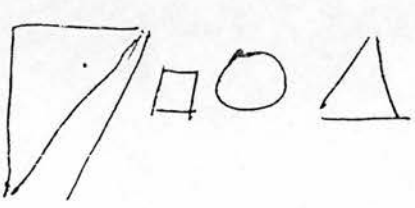








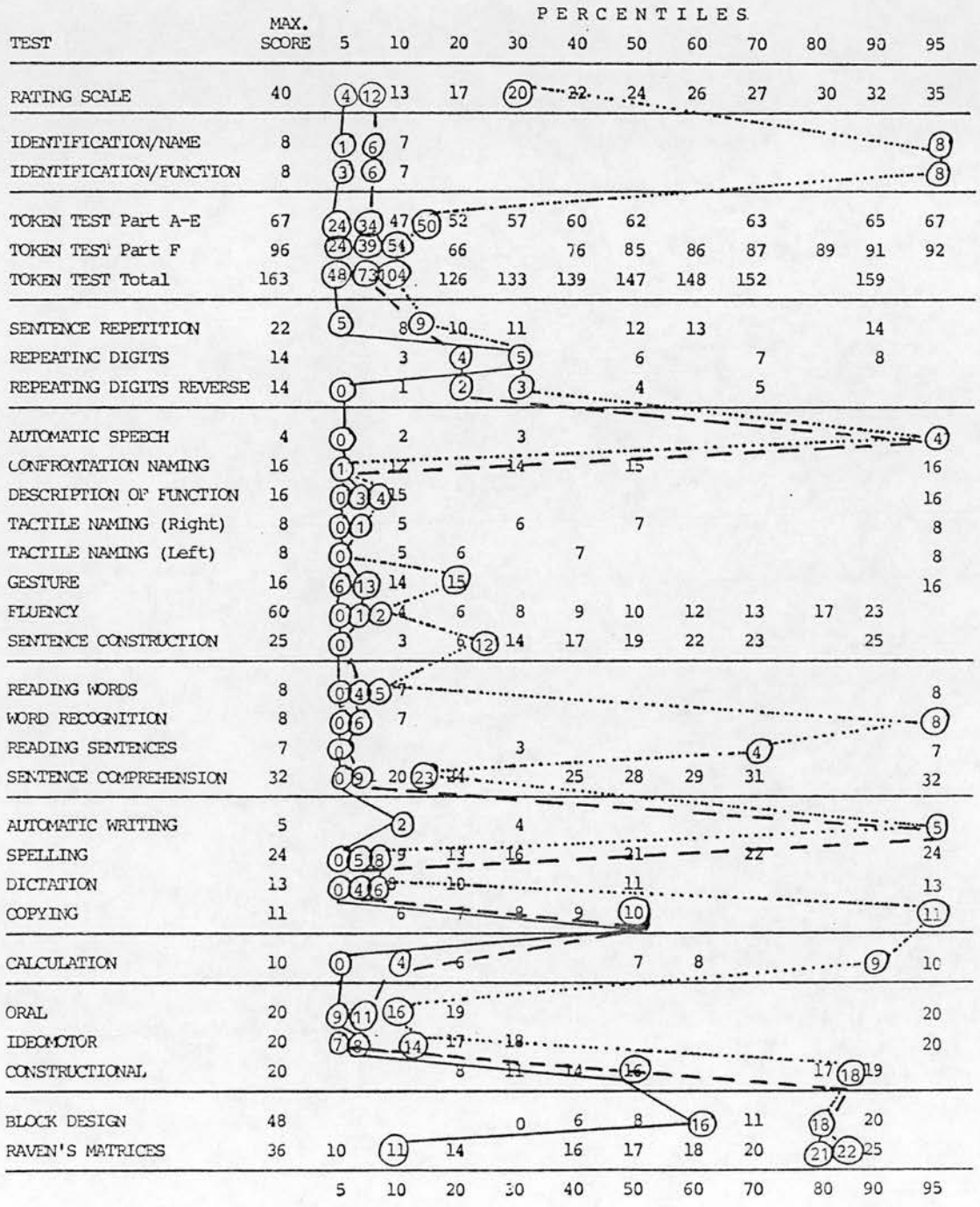




SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 18
SEX Female
D of B Age 56 years
DIAGNOSIS Alzheimer Type Dementia

DATE.....1.8.83
_ _ _ _ _ _ _ _ 6.8.84
13 9 85



CT (14.1.83) The ventricular system is well defined with no displacements. No real abnormalities, no focal lesion.

This patient was assessed for three consecutive years. Her presenting signs were disturbance of language.

She was oriented in time and place but not person and retained this sense of time into the third year. She was unable to give a non personal history, recall information from recent memory and, at the second assessment, concentrate. At the end of the third year automaticisms were lost.

Auditory memory by digit span was reduced giving scaled scores of 7, 8 and 3 consecutively. Repetition fell from her being able to repeat nine syllables after two years to only five in the third.

Verbal instructions were just diminished on the first assessment and comprehension was marginally improved by having instructions written. This suggests that initially memory was a major component in the loss of comprehension since she was able to process prepositional concepts and other "parietal" abilities as visuo spatial orientation and calculation.

Expressive language was marked with anomia, circumlocution, gesture and steriotypes. The patency of gesture indicates the concept of the word was retained the difficulty was the retrieval of the label from lexical store.

Initial attempts at sentence construction indicate a reduced ability to generate sentences that was lost in subsequent years. Fluency was extinct.

Reading was dissociated from word comprehension and attempts to read were reduced to letter spelling or jargoned responses. The patient was able to match words for meaning on the first assessment thus demonstrating initial representation of the word and concept although she was unable to say the words. This disruption between the lexical store and the phonemic realisation was reinforced by sentences that were partially read and instructions that were partially understood. These abilities were all lost on subsequent testing.

The progressive deterioration of writing is illustrated in Figures 8 a, b and c in Chapter 5.6. Initially automatic writing was preserved as was the ability to copy but not write to dictation. Visuo graphic transcoding was intact but not phonemic graphemic transcoding, indeed poor phonemic coding is demonstrated in writing where consonant graphemes are presented as approximations, so "pipe" becomes "bide". The feature of articulatory stops is maintained but articulatory placement and voicing is confused.

Tests of praxis demonstrate increasing oral and ideomotor apraxia as well as an ideational apraxia. Tasks for apraxia were not able to be imitated indicating the disorder more than one of comprehension. Gesture, when spontaneous as an adjunct to naming, was preserved. It was impaired when volitional.

Visuo spatial abilities as copying were diminished but with no real disturbance of line or shape. Gradually her perspective was lost. This patency of graphic skills was supported by preserved ability to copy writing than write to dictation. There was increasing dyscalculia.

Block Design gave a scaled score of 7, 7 and 6 while the RCPM fell to the lower limits of normal in the first two years, and into dementing range on the third.

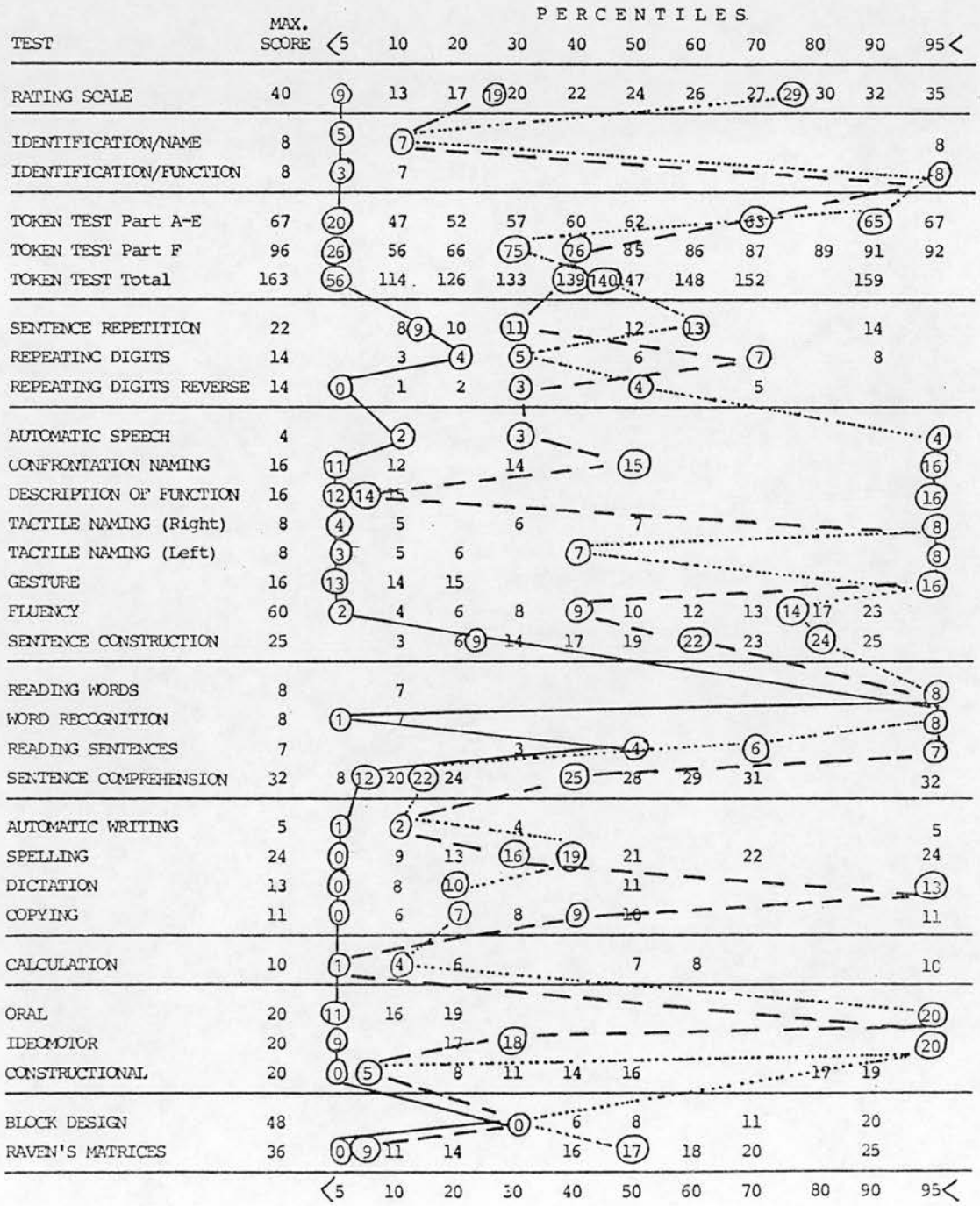
Comment:

The pattern is one of three years deterioration of language with a marked drop in the first to second year and almost complete alienation of word meaning both verbally and graphically. Automaticisms were more resistant to deterioration as were quasi spatial abilities as drawing and calculation.

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 19
SEX Female
D of B Age 67 years
DIAGNOSIS Alzheimer Type Dementia

DATE 8.7.82
- - - - - - - - - - 4.8.83
_____ 8.8.84



This retired nurse was assessed for three consecutive years. She was initially oriented and gave a complete personal and non personal history. Concentration and recent memory were impaired.

There was no real verbal agnosia but she did misidentify some objects in the same semantic class.

Digit recall gave a scaled score of 8 but loss of comprehension appeared to relate more to a failure of understanding concepts.

Expressive language contained anomia and semantic paraphasia while writing reflected orthographic errors in dictation and copying. Examples of writing and drawing ability are given in Chapter 5.6 Figures 6 a and b. Syntax was preserved.

There was no evidence of disturbance of praxis but drawing ability was fragmented, uncertain and demonstrated poor quality of line and lack of perspective.

Block Design failed to produce a score and the RCPM was below normal range.

On second assessment the patient was disoriented in time and place. Recent memory was affected as was concentration and the ability to recite. Confusion in recognition persisted and comprehension scores were below normal limits. Digit recall gave a scaled score of 9, again the scores reflected loss of concentration.

Expressive language contained verbal paraphasia (razor was "for shaving") or circumlocution (a bell was "you call with it" and tweezers "to cut eyebrows"). Syntax remained relatively preserved and reading ability was dissociated from comprehension.

Block Design gave no score and the RCPM fell below the tenth percentile for dements.

On the third assessment the patient was virtually mute but could still say her name. The agnosic confusion persisted and worsened, auditory memory had failed with a digit recall of 2 and repetition produced substitution of acoustically similar sounds.

Expressive language was sparse and without initiative and produced semantic paraphasia. Attempts to describe functions of objects were unrelated (a pen was "to comb hair" and a fork was "for cooking"). These errors contain the agnosic quality that Rochford (1971) described, for the patient was unable to gesture the use of the objects she could not describe or label.

Sentence construction was fragmentary, perseverative and palalalic. Word recognition remained preserved but sentence reading and sentence comprehension were impaired.

Writing and calculation were extinct and there was global apraxia.

Comments:

This description is the passage of three years ATD from diagnosis to agnosia. Initially moderate dementia reflected reduced comprehension and verbal expression with preserved syntax which deteriorated to disorientation, impaired comprehension and a divorce of reading ability to understand. Final assessment showed the only resistant abilities were engrams as reading single words with even automaticisms impaired.

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 20
SEX Male
D of B Age 64 years
DIAGNOSIS Alzheimer Type Dementia

DATE 30.12.83
28.10.84
17.09.85

TEST	MAX. SCORE	<5	10	20	30	40	50	60	70	80	90	95<
		PERCENTILES										
RATING SCALE	40	10	13	17	20	22	24	26	27	30	32	35
IDENTIFICATION/NAME	8		7									8
IDENTIFICATION/FUNCTION	8	6	7									8
TOKEN TEST Part A-E	67		47	52	57	60	62		63	64	65	67
TOKEN TEST Part F	96		56	66		76	85	86	87	88	89	91	92
TOKEN TEST Total	163	92	114	126	133	139	141	147	148	152		159	
SENTENCE REPETITION	22		8	10	11		12	13			14		
REPEATING DIGITS	14		3	4	5		6		7		8		9
REPEATING DIGITS REVERSE	14		1	2	3		4		5	6			
AUTOMATIC SPEECH	4		2		3								4
CONFRONTATION NAMING	16		12		14		15						16
DESCRIPTION OF FUNCTION	16		15										16
TACTILE NAMING (Right)	8		5		6		7						8
TACTILE NAMING (Left)	8		5	6		7							8
GESTURE	16		14	15									16
FLUENCY	60	2	4	6	8	9	10	12	13	17	19	23	42
SENTENCE CONSTRUCTION	25		3	6	14	17	19	20	22	23		25	
READING WORDS	8		7										8
WORD RECOGNITION	8		7										8
READING SENTENCES	7				3								7
SENTENCE COMPREHENSION	32	8	20	24		25	28	29	30	31			32
AUTOMATIC WRITING	5		2		4								5
SPELLING	24		9	13	16		21		22		23		24
DICTATION	13		8	10			11						13
COPYING	11		6	7	8	9	10						11
CALCULATION	10		4	6			7	8					10
ORAL	20		16	19									20
IDEOMOTOR	20			17	18								20
CONSTRUCTIONAL	20			8	11	14	16			17	19		
BLOCK DESIGN	48				0	6	8	11	12	14	16	20	
RAVEN'S MATRICES	36	10	11	14	15	16	17	18	19	20		25	26
		<5	10	20	30	40	50	60	70	80	90	95<	

This railwayman was assessed for three consecutive years. He was hospitalised for the third year.

The patient was moderately demented on initial assessment being disorientated in time and place but gave a preserved and accurate personal history. Memory for non personal events was impaired as was recent memory.

Comprehension as measured by the Token Test was below normal limits but Digit Repetition gave a scaled score of 11.

Expressive tasks were normal, Word Fluency being particularly impressive. There was no evidence of a disturbance of reading or writing nor of oral or limb apraxia, but there was evidence of constructional apraxia and dyscalculia.

Block Design yielded a scaled score of 7 and the RCPM was at the 35th percentile for normals.

On second assessment the patient remained moderately demented with an impaired ability to concentrate.

Comprehension remained reduced but stable as did auditory memory. Expressively verbal skills remained normal but Word Fluency dropped dramatically and there was increased difficulty in constructing sentences. Comprehension of written instructions diminished, Block Design yielded a scaled score of 6 and the RCPM fell below normal limits.

On third assessment the patient was severely demented and disoriented in all but person. Both personal history and concentration were impaired.

Overall comprehension continued to fall particularly because of a failure to process the concept within instructions. Digit span rose to a scaled score of 14 but the gap between repetition forward and in reverse reflected increasing loss of concentration.

Expressive abilities remained uncontaminated but Word Fluency fell four more decile ranks.

Reading comprehension at sentence level continued to diminish as did automatic writing, writing to copy and spelling.

Constructional apraxia became more evident and while the RCPM maintained its ranking, Block Design rose to a scaled score of 7.

Comment:

This is a three year pattern of the progression of ATD. Comprehension diminished in the third year and there was a progressive loss of Word Fluency, Calculation, Constructional Apraxia and non verbal intelligence. Auditory memory as measured by straight repetition was always high but scores dampened when digit reverse scores were added to the total. There was no real disturbance of expressive language although Sentence Construction diminished.

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 21
SEX Female
D of B Age 57 years
DIAGNOSIS Alzheimer Type Dementia

DATE 6.2.82
- - - - - - - - - - 2.9.83
13 1 84

TEST	MAX. SCORE	PERCENTILES <5	10	20	30	40	50	60	70	80	90	95<
RATING SCALE	40	9	13	17	20	22	25	24	26	27	30	32	35
IDENTIFICATION/NAME	8		7										8
IDENTIFICATION/FUNCTION	8	6	7										8
TOKEN TEST Part A-E	67		47	52	57	60		62		63		65 66	67
TOKEN TEST Part F	96		56	66	69	76	81	85	86	87	89	91	95
TOKEN TEST Total	163	92	114	126	129	133	139	147	148	152		159	162
SENTENCE REPETITION	22		8	9	10	11		12	13			14	
REPEATING DIGITS	14		3		4	5		6		7		8	
REPEATING DIGITS REVERSE	14		1		2	3		4		5			
AUTOMATIC SPEECH	4		2			3							4
CONFRONTATION NAMING	16		12			14		15					16
DESCRIPTION OF FUNCTION	16		15					14					16
TACTILE NAMING (Right)	8		5			6		7					8
TACTILE NAMING (Left)	8		5		6		7						8
GESTURE	16		14		15								16
FLUENCY	60	0	4		6	7	8	9	10	11	12	13	17	23
SENTENCE CONSTRUCTION	25	0	3		6	9	14	17	19	22	23			25
READING WORDS	8		7										8
WORD RECOGNITION	8		7										8
READING SENTENCES	7					3							7
SENTENCE COMPREHENSION	32	8	20	21	24			25	27	28	29	31		32
AUTOMATIC WRITING	5		2			4							5
SPELLING	24		9	13	16			21		22			24
DICTATION	13		8	10				11			12		13
COPYING	11		6	7	8	9		10					11
CALCULATION	10		4	6				7		8			10
ORAL	20		16	19									20
IDEOMOTOR	20			17	18			19					20
CONSTRUCTIONAL	20			8	10	11	14	16				17	19
BLOCK DESIGN	48				0	4	6	8		11			20
RAVEN'S MATRICES	36	10	11		14		16	17	18	20	23		25
		<5	10	20	30	40	50	60	70	80	90	95<	

This clerkess was assessed for three consecutive years. She was initially well oriented with a deficit of recent memory and auditory memory where digit recall gave a scaled score of 8. Sentence Repetition was also impaired. Comprehension was normal and there were no semantic or syntactic errors. Fluency was diminished, as was Block Design which gave a scaled score of 6 while the RCPM was at the twentieth percentile of normal.

Although conversation appeared normal medical notes comment on increased dressing apraxia and avoidance of speech by use of such phrases as "you've caught me on a bad day today" or "I've been very busy with the children today and I don't feel like talking just now".

On second assessment she was disoriented but remained a good historian. Sentence Repetition diminished and digit recall gave a scaled score of 4.

Disorders of comprehension could be attributed to both failure of memory and failure of the ability to process constructions in both written and verbal commands.

Expressive language remained free of naming errors but Fluency continued to be reduced and impairment of sentence encoding remained.

Reading ability was dissociated from comprehension and writing demonstrated intrusion errors.

Praxis was retained but constructional abilities demonstrated directional confusion, sequencing disorder and poor perspective.

Block Design gave a scaled score of 4 and the Matrices fell below normal range.

On third assessment the patient was disoriented and unable to give a history. Recent memory, automaticisms and concentration were impaired. Digit recall gave a scaled score of 4. Comprehension fell over all sections of the Token Test and was matched by a failure to understand written instruction.

Naming ability remained normal but for one paraphasic error. Writing ability became more impaired both orthographically and calligraphically (see Chapter 5.6 Figures 5 a, b and c.)

Block Design gave a scaled score of 3 and the RCPM, with an even distribution of scores fell to the tenth percentile for dements.

Comment:

The three assessments show increasing disintegration of cognitive ability. There is an early loss of memory contaminating auditory comprehension. Semantic skills were retained but intrusion errors noted as dementia fell from mild to moderate. Reading was dissociated from understanding and writing ability deteriorated as did the ability of complex calculation. Constructional abilities were progressively lost and measures of intelligence progressively diminished.

The accompanying language sample demonstrates avoidance, intrusion and lack of cohesion in terms of the relationship between phrases and clauses and the relationship of responses to questions. There is evidence of word finding difficulty in discourse but the better preservation of simple syntax.

No. 29 Age 67 yrs

LANGUAGE SAMPLE

Therapist: Do you ever have trouble finding what it is you want to say?

Patient: Something different

Therapist: Yes or do you forget words?

Patient: No
I wouldn't like to swear on that
now you've given me something to think about

Therapist: Were you in the war

Patient: Yes I was

Therapist: What did you do in the war?

Patient I was an Inspectress

Therapist What did you have to do?

Patient: Oh just check everything that was going there
I went through training at em (filled pause)
Oh, oh isn't that annoying - -
No it's gone
but I've got something to sit and think about now

Therapist: What sort of things were you an Inspectress of?

Patient: guns
small pieces and things like that
I was lucky
I wasn't put into a big dirty place or anything like that
but I still had to
and when I did this
and I was quite pleased

Patient: bashing

and when I saw this that's fine

but many night I had to do it again

because I've been too tired

Therapist: What did you have to do again?

Patient: Well this thing you had to clean it all

the material you're working on

even the

when you are in the living room - -

I keep saying living room

this keeps bugging me this

where was I

I got this started

Therapist: What were you doing in the war?

Patient: I now I just

rather got lost missed that

even the bombing

and things like that

It wasn't very pleasant

It gives everybody was underneath the table

Therapist: What do you mean when you keep saying living room?

Patient: Its the main room

it's one of these things

you know when you've got company coming

you strip everything off

put the white cover you know for the tablecloth

I thoroughly enjoyed it

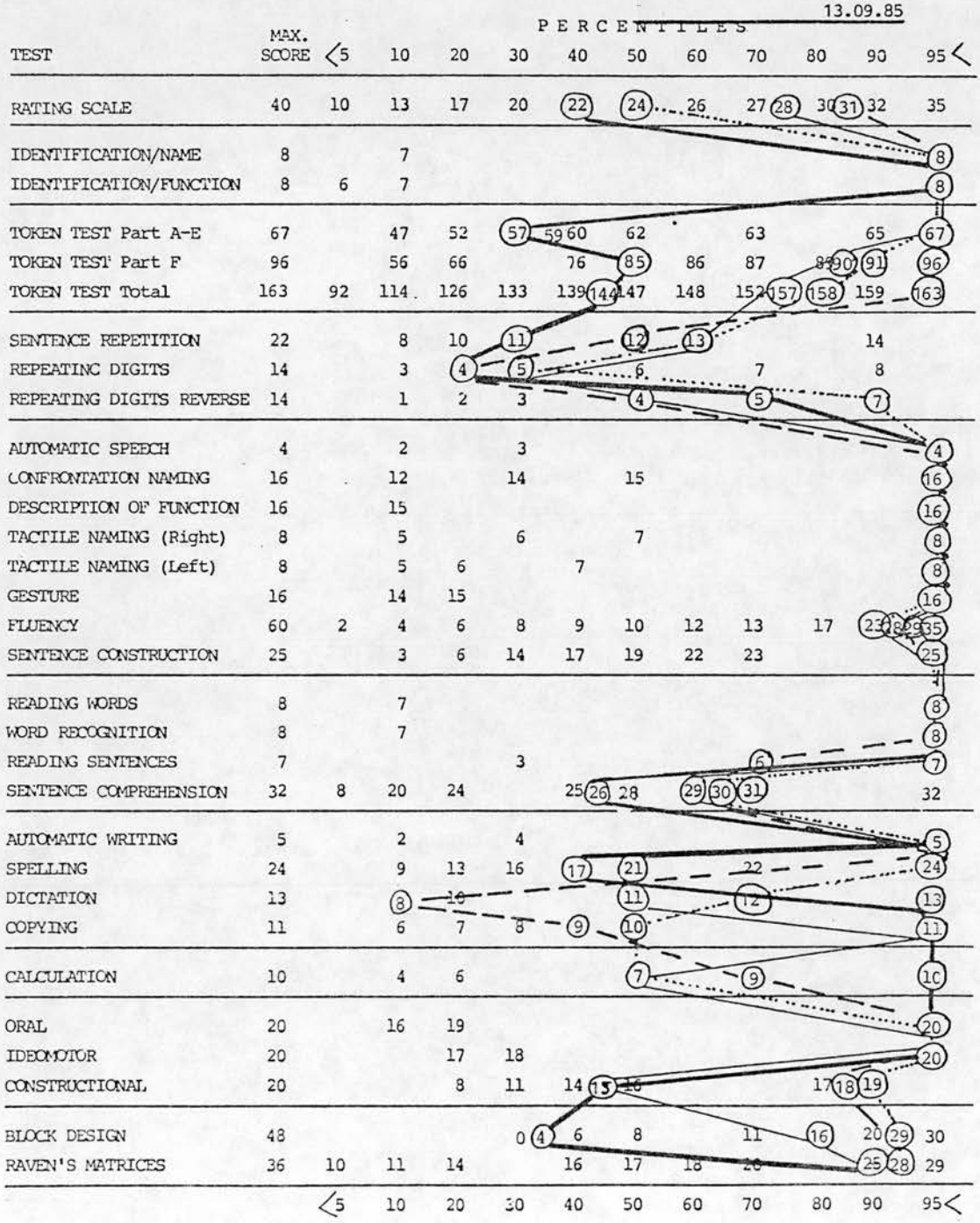
and then suddently

this wee things coming up

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 22
SEX Male
D of B Age 56 years
DIAGNOSIS Alzheimer Type Dementia

DATE ..... 7.7.82.
- - - - - - - - - - 1.9.83
26.10.84
13.09.85



This storeman was assessed for four consecutive years. In the first year he was oriented only in person but able to give a complete personal history. Recent memory was reduced as was concentration. There was no real loss of comprehension and memory scores were within normal limits. Digit recall gave a scaled score of 11 with reverse operations better than those of forward operation.

Naming and syntax were unimpaired, Fluency was at the twentieth percentile of normal. Reading and Reading Comprehension were also normal but calculation was not. There was no disturbance of praxis and the Block Design and the RCPM were within normal range. A scaled score of 12 was achieved for the constructional task.

On second assessment the man was totally oriented but auditory memory had deteriorated, digit recall giving a scaled score of 7. All expressive language tasks had improved. There was a tendency to over define terms, so bell became "hand bell" and torch "hand torch" while razor and watch were "safety razor" and "wrist watch". There were no real writing errors, or errors of calculation, or errors of praxis but there were marginal errors in drawing.

Block Design gave a scaled score of 12 and the RCPM remained within normal limits.

On third assessment the man was mildly demented. He gave a poor non personal history and recent memory was impaired.

Digit recall gave a scaled score of 4, and comprehension showed errors on Part F of the Token Test.

Expressive language produced one paraphasia, Fluency decreased marginally and there was no syntactic disturbance. Reading was preserved but written comprehension diminished. There was growing dyscalculia.

Oral, ideomotor and ideational praxis were maintained but copying ability showed medial neglect and disturbances of sequencing (see attached). Block Design fell to a scaled score of 7 and the RCPM to the thirtieth percentile for normals.

The man was assessed a fourth consecutive year and was moderately demented being disoriented in time and place. He was unable to recall his personal past, recall information from recent memory or concentrate.

Failure of comprehension was evident over all sections of the Token Test but particularly comprehension of instructions by length.

There were naming errors, a marginal decrease of fluency and preserved syntax.

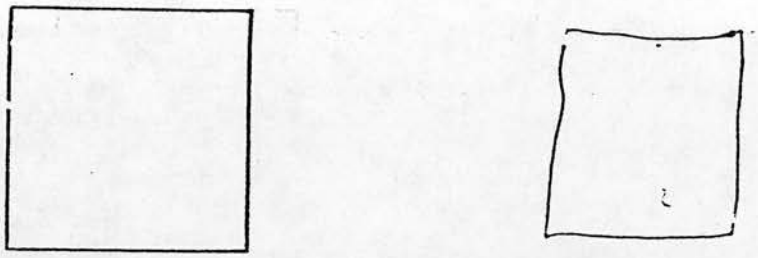
Reading was in tact but reading comprehension continued to be impaired. Writing confused upper and lower case and print and script. There was no dyscalculia.

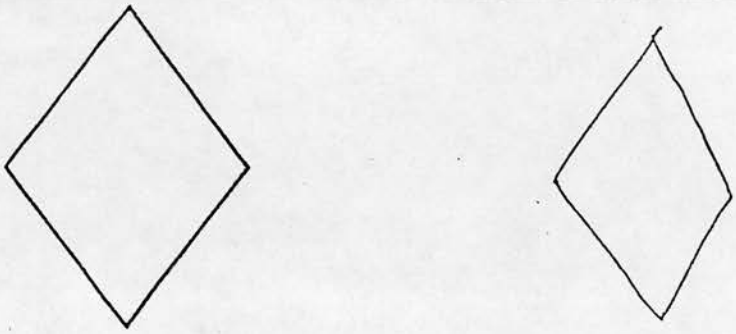
There was also no oral or ideomotor or ideational apraxia but drawing deficits continued to be apparent.

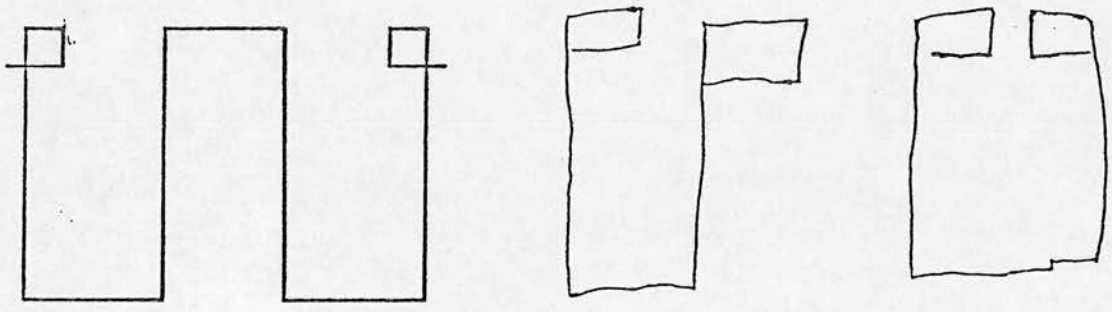
Block Design gave a scaled score of 4 and the RCPM reduced to the twentififth percentile of normal.

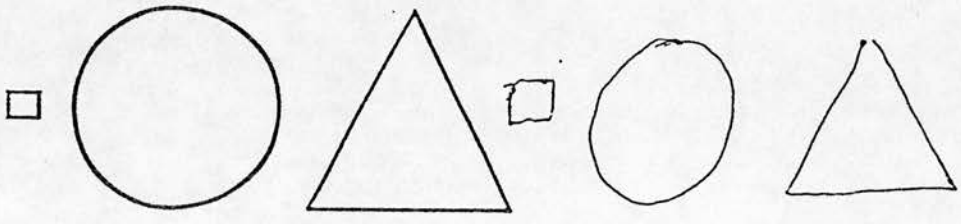
Comment:

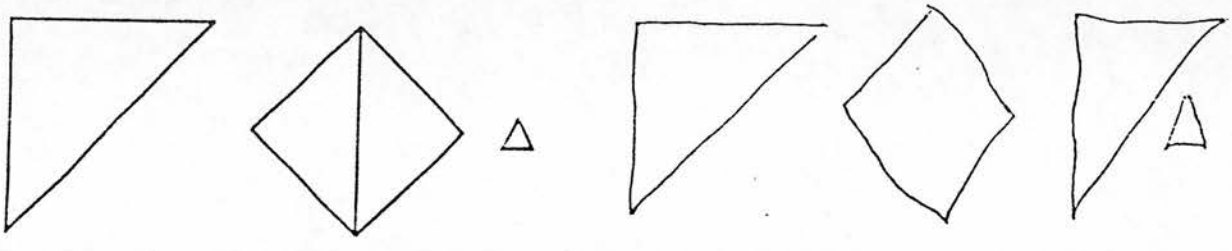
This man's history shows progressive and often fluctuating orientation with recent memory increasingly impaired. Comprehension was impaired by understanding of concepts as much as disturbance of memory. Expressive language skills remained strong, reading was intact while writing lost consistency and became perseverative. Calculation was preserved as was apraxia but drawing ability was progressively lost. Both tests of non verbal intelligence fell dramatically after the fourth year.







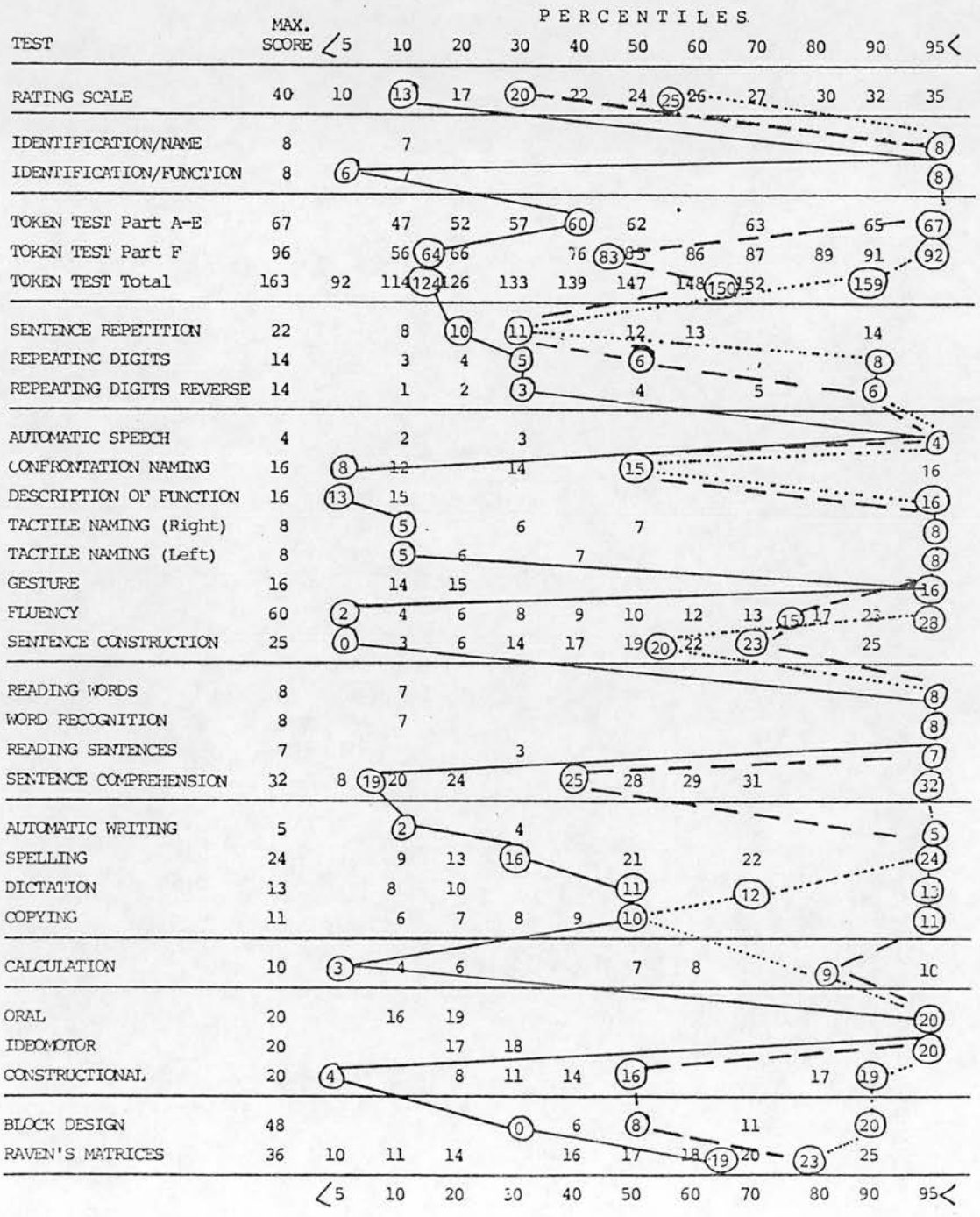




SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 23
SEX Female
D of B Age 62 years
DIAGNOSIS Alzheimer Type Dementia

DATE 7.6.83
- - - - - - - 10.8.84
——— 18.9.85



CT (2.4.80) Slight to moderate dilation of both lateral ventricles. Cerebral tissue absorbtion pattern is normal and there is no evidence of cortical shrinkage.

This lady was assessed for three consecutive years. She was initially mildly demented, disoriented in place and time but a good historian with impaired concentration and recent memory.

Comprehension was within normal range, digit recall gave a scaled score of 10. There were no errors of expressive language. Fluency was within normal limits and syntax preserved.

There was one error of calculation and one error of copying and no dyspraxia. Block Design gave a scaled score of 8 and with the RCPM was at lower range of normal.

On second assessment the patient remained disoriented but was no longer able to give a history or recall information from recent memory.

Digit recall gave a scaled score of 9 and there were increasing errors of comprehension of written and verbal complex concepts.

Expressive language contained nominal paraphasia, a loss of fluency and difficulty formulating sentences while reading was preserved but reading comprehension impaired.

Praxis was maintained but there were errors of calculation and drawing while Block Design fell to a scaled score of 6 and was matched by the RCPM.

The third assessment indicated severe dementia, disorientation and loss of remote and recent memory.

Comprehension for written and verbal instructions was globally impaired and digit recall gave a scaled score of 6. Sentence Repetition

was always more impaired than digit recall.

Expressive tasks produced semantic paraphasia and circumlocution (tweezers were "nippers" and a razor was "a shaving thing"). Recall of verb forms was affected and there was bilateral asteriognosis. Fluency was reduced to the base line for dements and tests of sentence construction had to be curtailed.

The ability to read and understand single words was retained but the ability to read and understand sentences was impaired to the base line for dementing data.

Oral, ideomotor and ideational praxis were retained but constructional skills, and the ability to calculate, were lost. There was no attempt at Block Design and the RCPM continued to diminish.

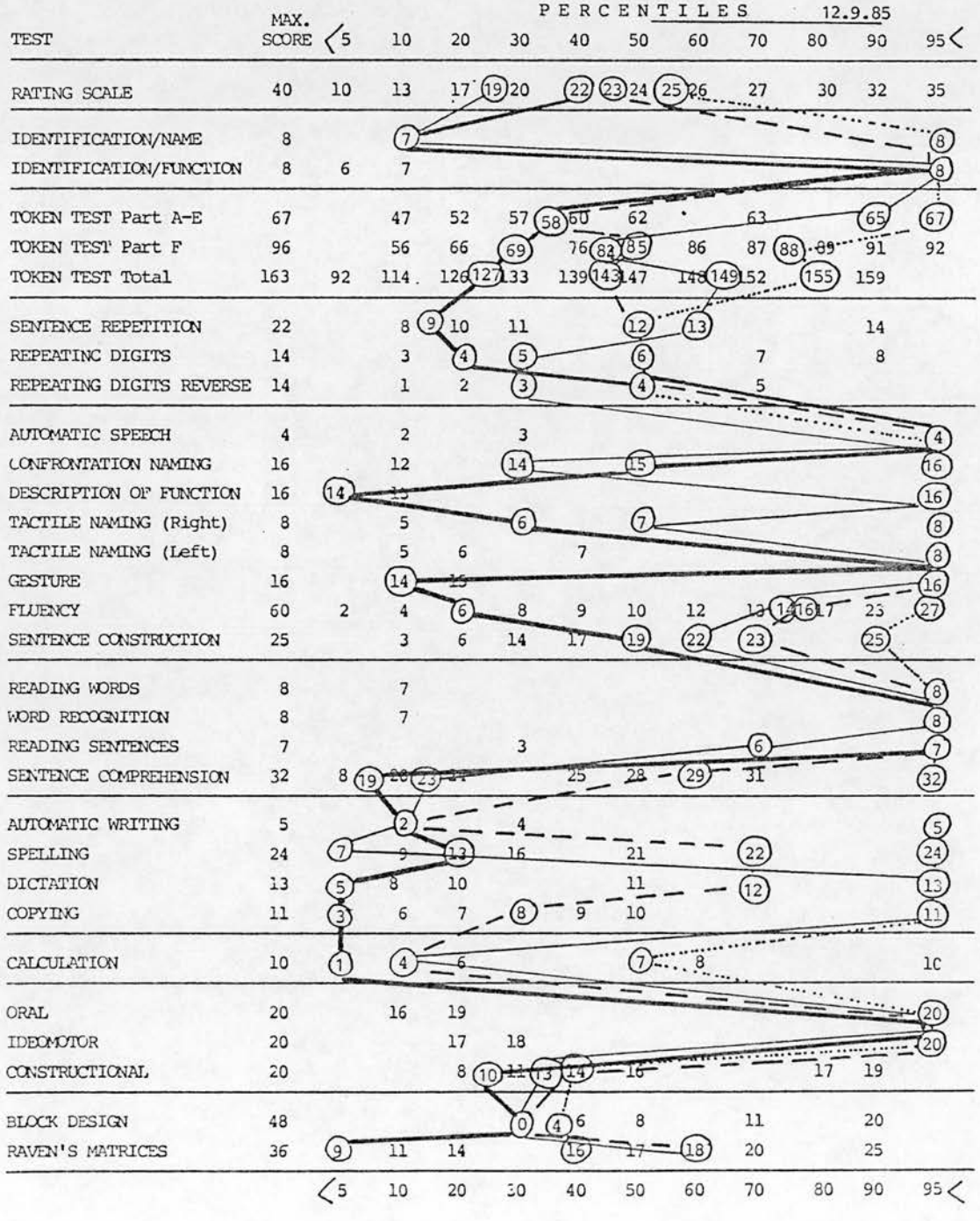
Comment:

This three year assessment depicts the unremitting affect of ATD on language from mild to severe impairment. Memory was initially affected, particularly by the registration of sentences. Non verbal intelligence was also initially impaired. Memory deficits soon affected comprehension of verbal material and a failure of the ability to process basic concepts in instructions fell with calculation and visuo spatial ability. Later, expressive language was contaminated, both semantics and fluency were impaired. Syntax also declined. Word recognition and automaticisms and simple gesture were spared. Examples of the progressive writing disorder and drawing disability are given in Chapter 5.6 Figures 7 a, b and c.

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME No. 24
SEX Female
D of B Age 64 years
DIAGNOSIS Alzheimer Type Dementia

DATE 25.1.82
- - - - - - - - 31.8.83
_____ 16.3.84
12.9.85



This patient was assessed over four consecutive years. She was initially disoriented in place and time but a good historian with poor recent memory and impaired concentration.

Auditory memory gave a digit recall scaled score of 8. Comprehension was reduced similarly to the lower range of normal but there were no errors in expressive language.

Reading ability was preserved but there were errors of calculation, construction and non verbal intelligence. Block Design gave a scaled score of 4.

On second assessment the woman was moderately demented being disoriented and with impaired recent memory but a good historian who was able to concentrate.

Digit recall gave a scaled score of 8 and comprehension failed as instructions increased by length and complexity. However there were no errors of expressive language. Fluency was within normal limits and syntax preserved. Reading ability was retained but comprehension for written instructions worsened.

Mild errors appeared in writing but greater errors appeared in calculation and drawing tasks where there was a loss of perspective and sequencing.

Block Designs were not completed, the RCPM improved slightly.

On third assessment the patient remained moderately demented and disoriented in anything but name. She was unable to give a non personal history and recent memory was impaired.

Comprehension of the concepts in written and verbal instructions was lost although digit recall, with a scaled score of 8, suggests loss of comprehension by length of instruction.

Expressive language produced semantic paraphasia ("lantern" for torch and "pincers" for tweezers) while Fluency was at the lower range of normal syntax was preserved but processing slow.

Reading and word recognition were unimpaired but comprehension of written sentences reduced. Writing was increasingly poor calligraphically and it was orthographically uncertain. Drawing showed evidence of directional errors, sequencing errors and neglect. Tasks of non verbal intelligence gave consistant scores.

On the fourth assessment orientation slightly improved but comprehension was globally diminished.

Digit recall gave a scaled score of 8 but sentence repetition was more impaired than digit repetition, moreover reversed operations produced the better scores.

Naming produced semantic paraphasia ("prong" for tweezers) and circumlocution (a torch was "a thing for seeing"). Fluency was diminished as was sentence construction.

Reading ability was preserved but comprehension of written instructions lost. Writing became illegible with orthographic, calligraphic and alignment errors.

The woman was dyscalculic and, while oral and ideomotor praxis was maintained, ideational and constructional praxis was impaired. Drawing ability demonstrated right neglect.

Tests of non verbal intelligence fell to the lower limits of the scale.

Comment:

This performance reflects the gradual loss of language abilities despite fluctuating scores on the Rating Scale and early loss of memory, constructional apraxia and non verbal intelligence.

Comprehension diminished with loss of memory and quasi spatial processing including calculation and visuo spatial ability. Memory scores were always depressed but could be raised in effort.

Expressive language ability fell on the third and fourth assessment although writing ability appeared to slip on the second.

Engrams as word reading and word recognition as well as oral and ideomotor praxis and automaticisms were preserved.

Examples of the loss of writing and drawing ability on second to final testing are attached.

Test 1.

Bell Rope

Bike

Whistle Watch.

Jumpers

Torch

Glasses

This is a very nice day

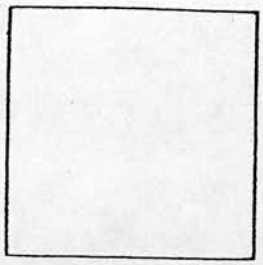
This brick building was

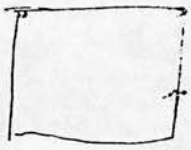
built last year.

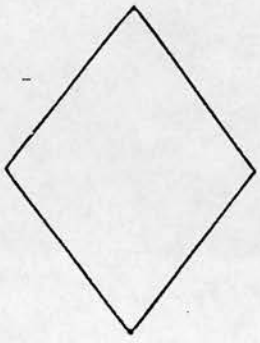
I am very hungry

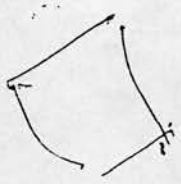
The colour of the

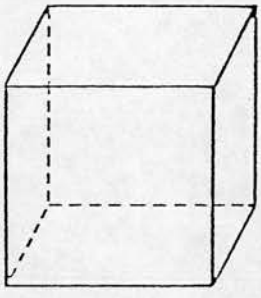
walls is green

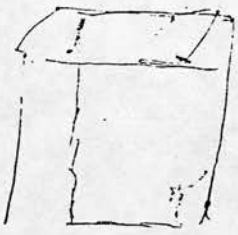


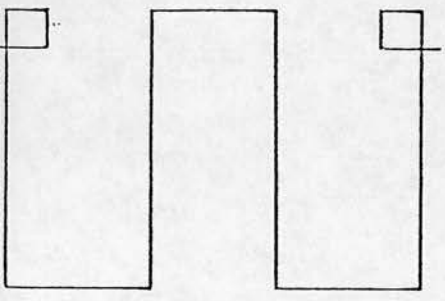


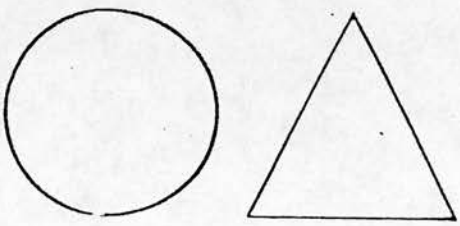


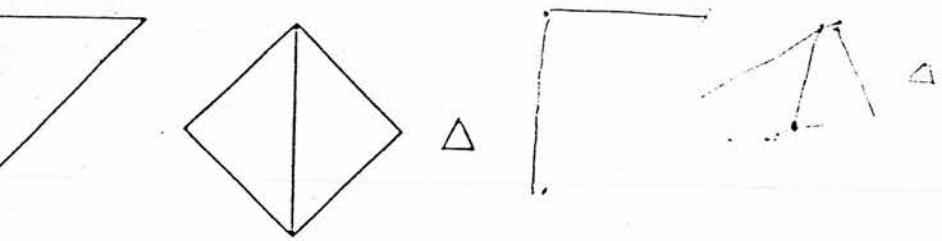












Torch

Razor Bell

Whistle

Smeeser

Pebe

Glasses.

Watch

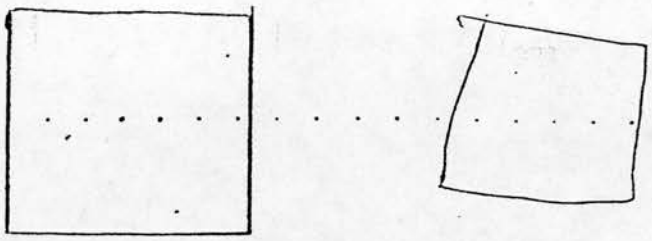
I am very running

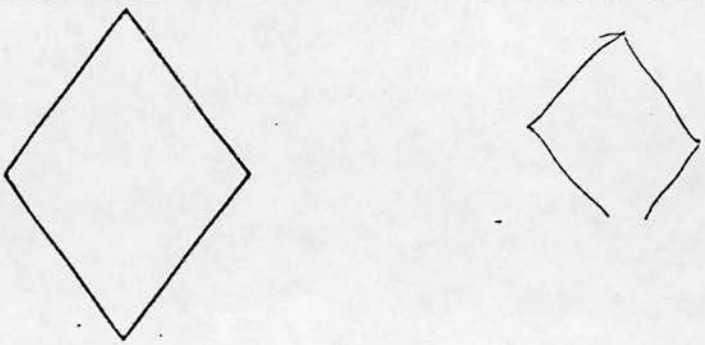
The corner of the two walls is your

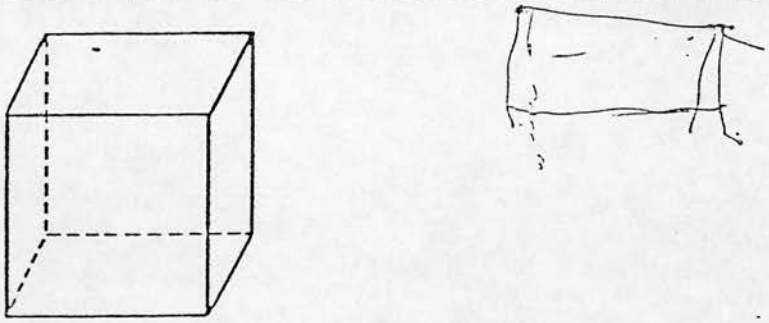
This is a very nice day

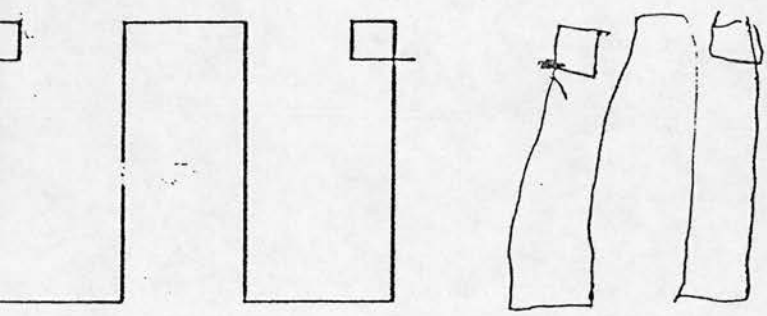
This Buck Burbanks was built

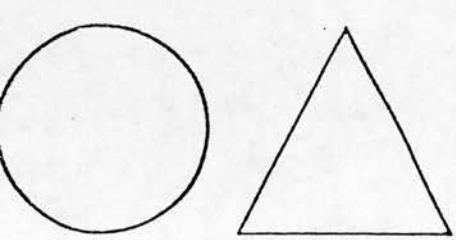
last year

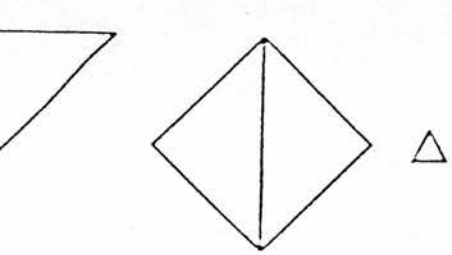


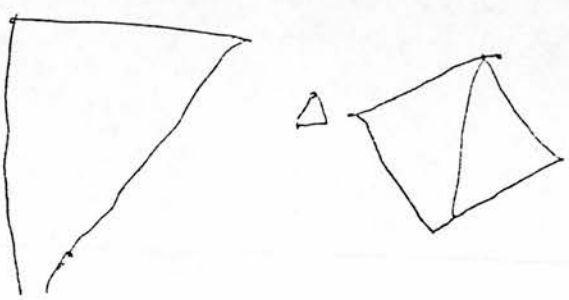












Bell

Pencil

Razor

Torch

Whistle

Glasses

Watch

Tree

Thumb a few Days

F. Bonelli, Burglar was Burnt

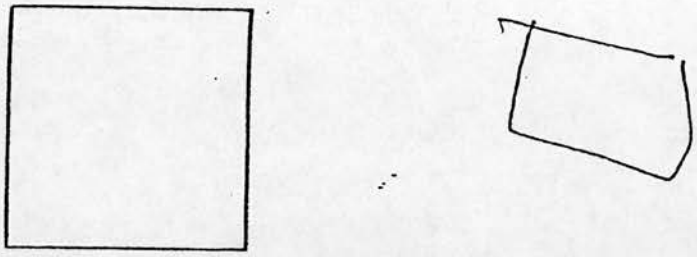
last Year

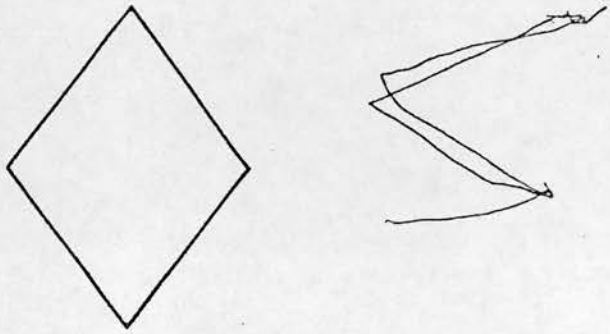
T. Coleman

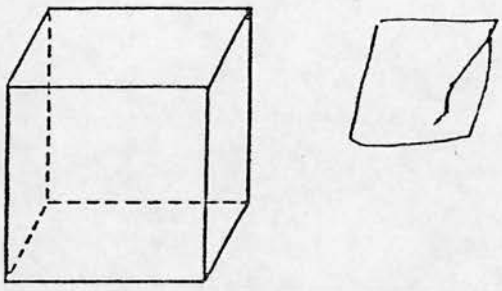
T. Coleman of Tivs

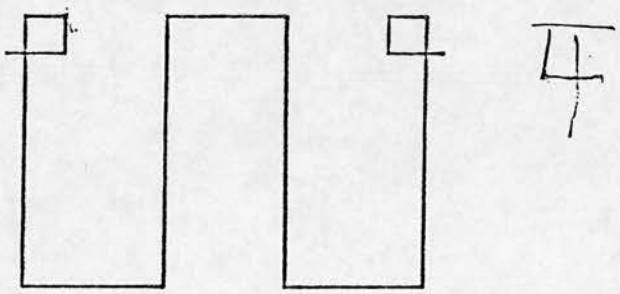
Wales

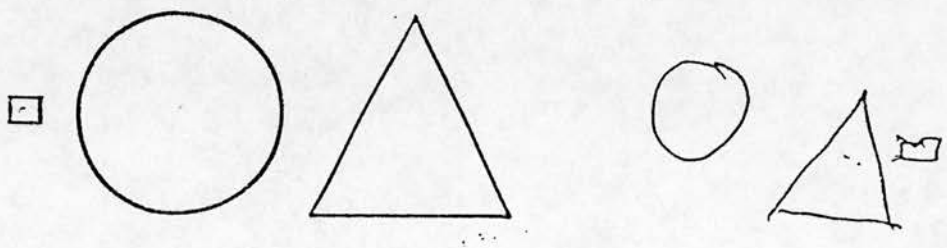
I. A. V. by Kuna - 4

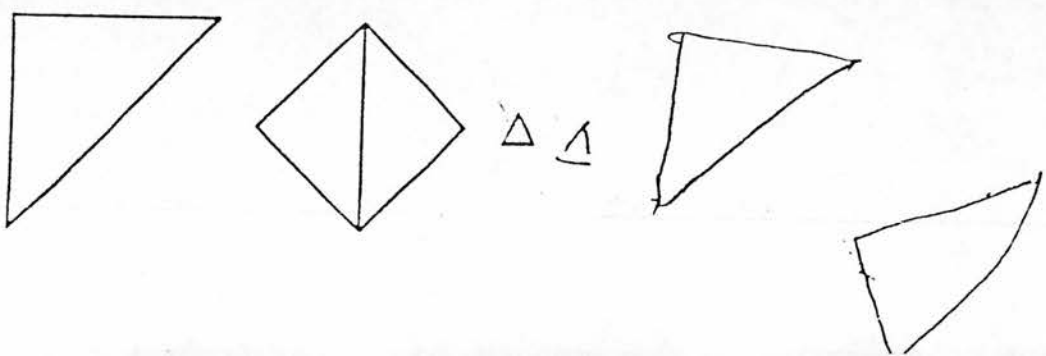












APPENDIX II

TEST BOOKLET AND PROFILE FORMS

B.M.U. LANGUAGE SCALES (M.R.C. Edin.)

For Research Purposes Only

This aphasia battery represents a set of language scales taken from acknowledged sources for the description of language performance in normal and abnormal ageing.

IAN THOMPSON, B.App. Sci.(Speech Pathology) B.A. (Melb.)

NAME _____

ADDRESS _____

D of B _____ HANDEDNESS _____ FILE _____

DIAGNOSIS _____ DATE _____

EXAMINER _____

MRC BRAIN METABOLISM UNIT : ROYAL EDINBURGH HOSPITAL : SCOTLAND

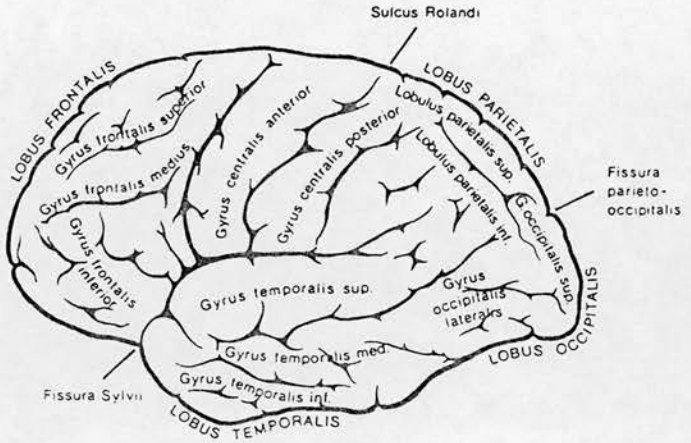
ALZHEIMER STUDY

NAME ..... Unit no. .....

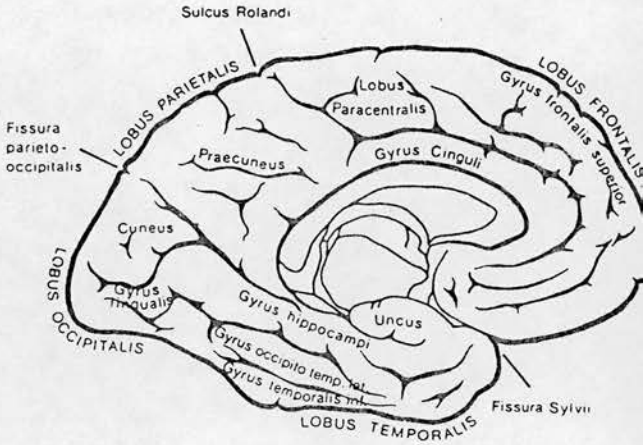
Date of admission .....

	? to be done	date completed
Nursing		
Admission weight		
Urinalysis		
24 hour urine		
Stockton rating scale		
Medical		
Bloods:		
Urea and electrolytes and creatinine		
T4		
LFTs		
Chromosomes		
Investigations		
CAT scan		
EEG		
Skull and chest x-ray		
ECG		
Lumbar puncture		
Neuroendocrine day		
Neuroendocrine challenges		
Apomorphine		
Clonidine		
Dexamethasone suppression test		
Psychological testing		
Speech therapist assessment		

Suggested localization of lesion according to conclusion

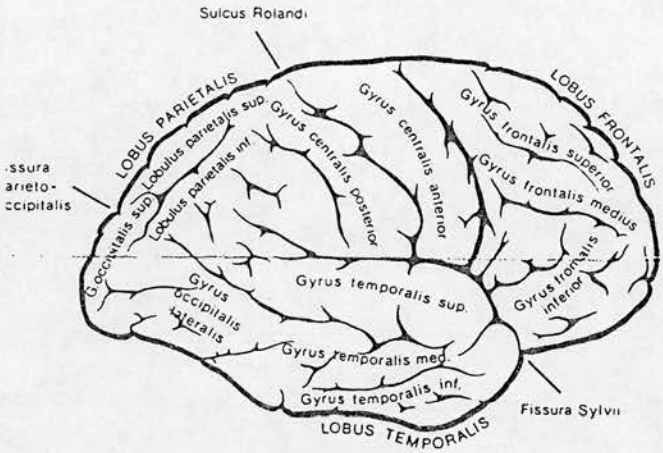


Lateral



Mesial

LEFT



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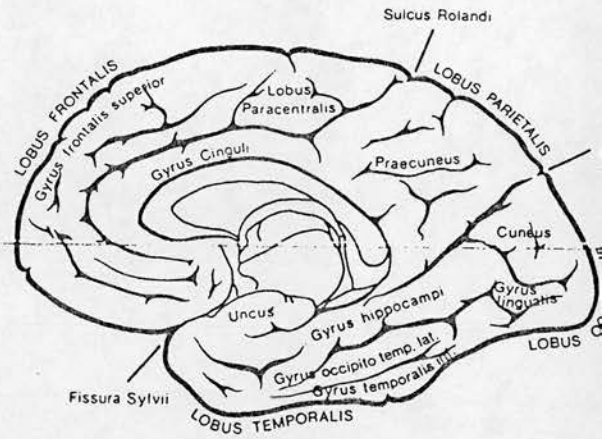
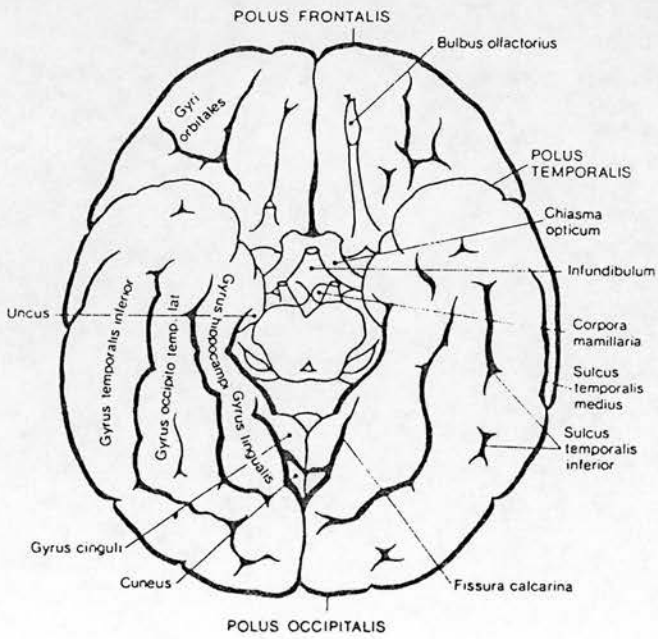


Mesial

RIGHT



Basis

Conclusion:

Orientation/Memory/Concentration Test

After Blessed et al - B.J.Psychiat. (1968) 114.

Information	Score
Name	1
Address	1
Time (hour)	1
Time of Day	1
Day of Week	1
Date	1
Month	1
Season	1
Year	1
Place Name	1
Street/Suburb	1
Town/City	1
Type of Place	1
Recognition of one person	1

Memory

1. Personal

Date of Birth	1
Place of Birth	1
Name of wife/sibling/parent	1
School attended	1
Occupation	1
Name of town where patient worked	1
Name of employers	1

2. Non-personal

Date of World War I	1
Date of World War II	1
Monarch	1
Prime Minister	1

3. Name and address (5 minute recall)

Ian Thompson	2
11 Fowler Terrace	3
Edinburgh	1
Nationality	1

4. Automatic Speech/Concentration

Counting 1 - 20	2	1	0
Counting 20 - 1	2	1	0
Weekdays forward	2	1	0
Weekdays reverse	2	1	0

Total 10

IDENTIFICATION BY NAME

TRAY A	RESPONSE	ERROR TYPE	SCORE
RAZOR			
PIPE			
TORCH			
BELL			
GLASSES			
TWEEZERS			
WHISTLE			
WATCH			
		TOTAL	

IDENTIFICATION BY FUNCTION

TRAY B	RESPONSE	ERROR TYPE	SCORE
GUN			
SCISSORS			
PEN			
SPONGE			
FORK			
CUP			
BRUSH			
MATCHES			
		TOTAL	

IDENTIFICATION BY SENTENCE (TOKEN TEST) (After Spreen O & Benton A - N.C.C.E.A. 1969)

A. Present tokens as in Fig. 4.	Instructions may be repeated once	
1.	Show me a circle	
2.	Show me a square	
3.	Show me a yellow one	
4.	Show me a red one	
5.	Show me a blue one	
6.	Show me a green one	
7.	Show me a white one	
	TOTAL A(7)	

B. Present only large tokens.	Instructions may be repeated once	
8.	Show me the yellow square	
9.	Show me the blue circle	
10.	Show me the green circle	
11.	Show me the white square	
	TOTAL B(8)	

C. Present all tokens as in Fig. 4	Do not repeat instructions	
12.	Show me the small white circle	
13.	Show me the large yellow square	
14.	Show me the large green square	
15.	Show me the small blue square	
	TOTAL C(12)	

D. Present large tokens only.	Do not repeat instructions	
16.	Take the red circle and the green square	
17.	Take the yellow square and the blue square	
18.	Take the white square and the green circle	
19.	Take the white circle and the red circle	
	TOTAL D(16)	

E. Present all tokens as in Fig. 4.	Do not repeat instructions	
20.	Take the large white circle and the small green square	
21.	Take the small blue circle and the large yellow square	
22.	Take the large green square and the large red square	
23.	Take the large white square and the small green circle	
	TOTAL E(24)	

F. Present large tokens only. Do not repeat instructions.	
24. Put the red circle on the green square.	
25. Put the white square behind the yellow circle.	
26. Touch the blue circle with the red square.	
27. Touch the blue circle and the red square.	
28. Pick up the blue circle OR the red square.	
29. Move the green square away from the yellow square.	
30. Put the white circle in front of the blue square.	
31. If there is a black circle, pick up the red square.	
32. Pick up all squares except the yellow one.	
33. Put the green square beside the red circle.	
34. Touch the squares slowly and the circles quickly.	
35. Put the red circle between the yellow square and the green square.	
36. Touch all circles, except the green one.	
37. Pick up the red circle —no— the white square.	
38. Instead of the white square, pick up the yellow circle.	
39. Together with the yellow circle, pick up the blue circle.	
TOTAL F(96)	
TOTAL A-F (163)	

NTENCE REPETITION (A) - (After Spreen O & Benton A - N.C.C.E.A. 1969)

1.	Look	
2.	Come here.	
3.	Help yourself.	
4.	Bring the table.	
5.	Summer is coming.	
6.	The iron was quite hot.	
7.	The birds were singing all day.	
8.	The paper was under the chair.	
9.	The sun was shining throughout the day.	
10.	He entered about eight o'clock that night.	
11.	The pretty house on the mountain seemed empty.	
12.	The lady followed the path down the hill toward home.	
13.	The island in the ocean was first noticed by the young boy.	
14.	The distance between these two cities is too far to travel by car.	
15.	A judge here knows the law better than those people who must appear before him.	
16.	There is a new method in making steel which is far better than that used before.	
17.	This nation has a good government which gives us many freedoms not known in times past.	
18.	The friendly man told us the directions to the modern building where we could find the club.	
19.	The king knew how to rule his country so that his people would show respect for his government.	
20.	Yesterday he said he would be near the village station before it was time for the train to come.	
21.	His interest in the problem increased each time that he looked at the report which lay on the table.	
22.	Riding his black horse, the general came to the scene of the battle and began shouting at his brave men.	
	TOTAL SCORE	

REPETITION OF DIGITS (After W.A.I.S. Ed 1955)

(Record actual performance)

Digits forward	Score
5–8–2	3
6–9–4	3
6–4–3–9	4
7–2–8–6	4
4–2–7–3–1	5
7–5–8–3–6	5
6–1–9–4–7–3	6
3–9–2–4–8–7	6
5–9–1–7–4–2–8	7
4–1–7–9–3–8–6	7
5–8–1–9–2–6–4–7	8
3–8–2–9–5–1–7–4	8
2–7–5–8–6–2–5–8–4	9
7–1–3–9–4–2–5–6–8	9
SCORE	

REVERSAL OF DIGITS (After W.A.I.S. Ed 1955)

(Record actual performance)

Digits backward	Score
2–4	2
5–8	2
6–2–9	3
4–1–5	3
3–2–7–9	4
4–9–6–8	4
1–5–2–8–6	5
6–1–8–4–3	5
5–3–9–4–1–8	6
7–2–4–8–5–6	6
8–1–2–9–3–6–5	7
4–7–3–9–1–2–8	7
9–4–3–7–6–2–5–8	8
7–2–8–1–9–6–5–3	8
SCORE	

CONFRONTATION NAMING

TRAY A	RESPONSE	ERROR TYPE	SCORE
RAZOR			
PIPE			
TORCH			
BELL			
GLASSES			
TWEEZERS			
WHISTLE			
WATCH			
		TOTAL A	

TRAY B	RESPONSE	ERROR TYPE	SCORE
GUN			
SCISSORS			
PEN			
SPONGE			
FORK			
CUP			
BRUSH			
MATCHES			
		TOTAL B	
		TOTAL A & B	

DESCRIPTION OF FUNCTION

TRAY A	RESPONSE	ERROR TYPE	SCORE
RAZOR			
PIPE			
TORCH			
BELL			
GLASSES			
TWEEZERS			
WHISTLE			
WATCH			
		TOTAL A	

TRAY B	RESPONSE	ERROR TYPE	SCORE
GUN			
SCISSORS			
PEN			
SPONGE			
FORK			
CUP			
BRUSH			
MATCHES			
		TOTAL B	
		TOTAL A & B	

TACTILE NAMING (RIGHT)

TRAY A	RESPONSE	ERROR TYPE	SCORE
RAZOR			
PIPE			
TORCH			
BELL			
GLASSES			
TWEEZERS			
WHISTLE			
WATCH			
		TOTAL A	

TACTILE NAMING (LEFT)

TRAY B	RESPONSE	ERROR TYPE	SCORE
GUN			
SCISSORS			
PEN			
SPONGE			
FORK			
CUP			
BRUSH			
MATCHES			
		TOTAL B	
		TOTAL A & B	

GESTURE

TRAY A	RESPONSE	ERROR TYPE	SCORE
RAZOR			
PIPE			
TORCH			
BELL			
GLASSES			
TWEEZERS			
WHISTLE			
WATCH			
		TOTAL A	

TRAY B	RESPONSE	ERROR TYPE	SCORE
GUN			
SCISSORS			
PEN			
SPONGE			
FORK			
CUP			
BRUSH			
MATCHES			
		TOTAL B	
		TOTAL A & B	

WORD FLUENCY (After Spreen 0 & Benton A - N.C.C.E.A. 1969)

	F	A	S
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			
15			
16			
17			
18			
19			
20			

Sum F _____ Sum A _____ Sum S _____

TOTAL _____

TENCE CONSTRUCTION (After Spreen O & Benton A - N.C.C.E.A. 1969)

	Circle Obtained Score			
	greater than 20"		within 20"	within 10"
ow – boy	0	3	4	5
t – summer	0	3	4	5
idge – walk – man	0	3	4	5
ir – water – girl	0	3	4	5
ive – street – car	0	3	4	5
			TOTAL	

READING NAMES

READING NAMES FOR MEANING

TRAY B	ORAL READING	MEANING	
GUN			
SCISSORS			
PEN			
SPONGE			
FORK			
CUP			
BRUSH			
MATCHES			

ORAL READING SENTENCES

READING SENTENCES FOR MEANING (POINTING)

	TEST 13	TEST 15
1. Show me a CIRCLE		
2. Show me a SMALL SQUARE		
3. Show me a SMALL WHITE CIRCLE		
4. Show me a YELLOW SQUARE and a RED CIRCLE		
5. Put the RED CIRCLE BETWEEN the YELLOW SQUARE and the GREEN SQUARE		
6. Put the YELLOW CIRCLE BEHIND the WHITE SQUARE		
7. MOVE the GREEN SQUARE AWAY FROM the YELLOW SQUARE		

AUTOMATIC WRITING

VISUO GRAPHIC NAMING
WRITING NAMES

TRAY A	SUPPLIED NAME	
RAZOR		
PIPE		
TORCH		
BELL		
GLASSES		
TWEEZERS		
WHISTLE		
WATCH		
	TOTAL	

DICTATION

THIS IS A VERY NICE DAY	
THE BRICK BUILDING WAS BUILT LAST YEAR	
TOTAL	

WRITING (COPYING)

I AM VERY HUNGRY	
THE COLOUR OF THE WALLS IS GREEN	
TOTAL	

CALCULATION

$5 \times 4 =$

$7 \times 8 =$

$3 + 4 =$

$6 + 7 =$

$7 - 4 =$

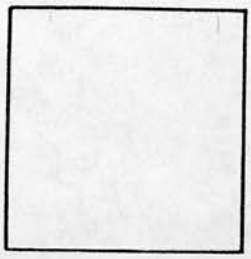
$8 - 5 =$

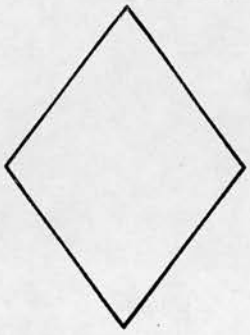
$$\begin{array}{r} 27 \\ 8+ \\ \hline \\ \hline \end{array} \qquad \begin{array}{r} 44 \\ 57+ \\ \hline \\ \hline \end{array}$$

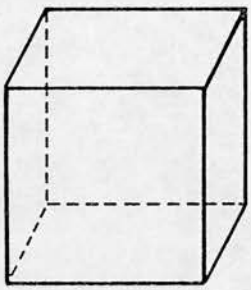
$$\begin{array}{r} 31 \\ 7- \\ \hline \\ \hline \end{array} \qquad \begin{array}{r} 41 \\ 14- \\ \hline \\ \hline \end{array}$$

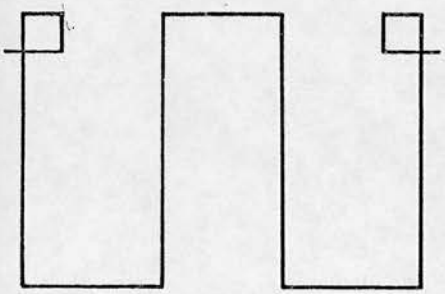
RAL APRAXIA (After Mayo Clinic)	
. STICK OUT YOUR TONGUE	
. BLOW	
. CLICK YOUR TONGUE	
. CLEAR YOUR THROAT	
. LICK YOUR LIPS	
. BLOW UP YOUR CHEEKS	
. TOUCH YOUR NOSE WITH YOUR TONGUE	
. MOVE YOUR TONGUE IN AND OUT	
. MOVE YOUR TONGUE FROM SIDE TO SIDE	
0. SMILE	
TOTAL	

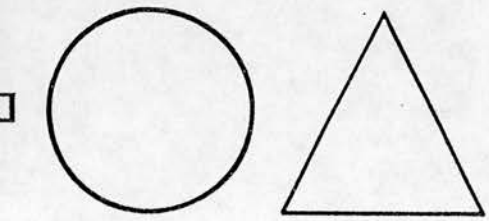
)EOMOTOR APRAXIA (After de Renzie et al - Neuropsychologia 1968)	
. SALUTE	
. WAVE	
. SNAP YOUR FINGERS	
. MAKE A FIST	
. MAKE THE LETTER 'O' WITH YOUR FINGERS	
. GIVE THE VICTORY SIGN	
. DRUM YOUR FINGERS ON THE TABLE	
. PRETEND YOU ARE SHAKING HANDS	
. SHOW ME HOW YOU CLEAN YOUR TEETH	
). WIPE YOUR BROW	
TOTAL	

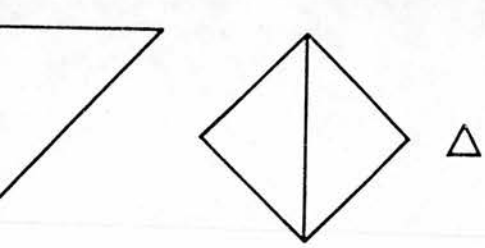












BLOCK DESIGN (Ref: WAIS 1955)			
	Time	Score	
1. 60''	1. 2.	0 2 4	
2. 60''	1. 2.	0 2 4	
3. 60''		0 4	
4. 60''		0 4	
5. 60''		0 4	
6. 60''		0 4	
7. 120''		0 4	31 - 40 5 / 1 - 30 6
8. 120''		0 4	40 - 70 5 / 1 - 45 6
9. 120''		0 4	61 - 80 5 / 1 - 60 6
10. 120''		0 4	61 - 80 5 / 1 - 60 6

STANDARD PROGRESSIVE MATRICES

Test begun_____ Test ended_____

A		B		C		D		E	
1		1		1		1		1	
2		2		2		2		2	
3		3		3		3		3	
4		4		4		4		4	
5		5		5		5		5	
6		6		6		6		6	
7		7		7		7		7	
8		8		8		8		8	
9		9		9		9		9	
10		10		10		10		10	
11		11		11		11		11	
12		12		12		12		12	

Time	Total	Grade

Notes

Tested by_____

W.G./73/100,000

B.M.U. Language Scales (M.R.C.Edin.)

		Raw Score	Scaled Score
RECEPTIVE LANGUAGE			
1.	Orientation		
2.	Identification by Name		
3.	Identification by Function		
4.	Token Test - Parts A-E		
5.	- Part F		
6.	- Total		
7.	Sentence Repetition		
8.	Repetition of Digits		
9.	Reversal of Digits		
EXPRESSIVE LANGUAGE			
10.	Automatic Speech		
11.	Confrontation Naming		
12.	Description of Function		
13.	Tactile Naming (Right)		
14.	Tactile Naming (Left)		
15.	Gesture		
16.	Word Fluency		
17.	Sentence Construction		
SYMBOLIC LANGUAGE			
18.	Reading Names		
19.	Reading Names for Meaning		
20.	Reading Sentences		
21.	Reading Sentences for Meaning		
22.	Automatic Writing		
23.	Visuo-graphic Naming		
24.	Dictation		
25.	Copying		
26.	Calculation		
APRAXIA			
27.	Oral Apraxia		
28.	Ideomotor Apraxia		
29.	Constructional Apraxia		
NON-VERBAL INTELLIGENCE			
30.	KOH Block Design		
31.	Coloured Progressive Matrices		
ADDITIONAL TESTS			
32.	Sentence Repetition		
33.	Visual Memory		
34.	Learning		

SUBTEST SUMMARY PROFILE (NORMALS)

NAME DATE

SEX

D of B

DIAGNOSIS

TEST	MAX. SCORE	PERCENTILES 5	10	20	30	40	50	60	70	80	90	95
RATING SCALE	40	37	38									40
IDENTIFICATION/NAME	8		7									8
IDENTIFICATION/FUNCTION	8		7									8
TOKEN TEST Part A-E	67	64	65		66							67
TOKEN TEST Part F	96		90	92			95					96
TOKEN TEST Total	163		154	159	160		162					163
SENTENCE REPETITION	22		12			14	15	16	17	18		20
REPEATING DIGITS	14		5		6		7	8	9	10	11	12
REPEATING DIGITS REVERSE	14	3	4	5	6		7	8	9	10		12
AUTOMATIC SPEECH	4											4
CONFRONTATION NAMING	16	14				15						16
DESCRIPTION OF FUNCTION	16		10									16
TACTILE NAMING (Right)	8				7							8
TACTILE NAMING (Left)	8				7							8
GESTURE	16	16										16
FLUENCY	60	10	20	27	31	33	35	36	40	44	50	60
SENTENCE CONSTRUCTION	25	23		24								
READING WORDS	8											8
WORD RECOGNITION	8											8
READING SENTENCES	7											7
SENTENCE COMPREHENSION	32		31									32
AUTOMATIC WRITING	5											5
SPELLING	24					23						24
DICTATION	13	11		12								13
COPYING	11	9		10								11
CALCULATION	10	8	9									10
ORAL	20											20
IDEOMOTOR	20											20
CONSTRUCTIONAL	20		18		19							20
BLOCK DESIGN	48	15	20		25	27	28	30	35	40		42
RAVEN'S MATRICES	36	19	20	23	25	27	28	31	33	34	35	36
		5	10	20	30	40	50	60	70	80	90	95

SUBTEST SUMMARY PROFILE (APHASIA)

NAME DATE

SEX

D of B

DIAGNOSIS

TEST	MAX. SCORE	PERCENTILES 5	10	20	30	40	50	60	70	80	90	95
RATING SCALE	40											40
IDENTIFICATION/NAME	8	4	5	7								8
IDENTIFICATION/FUNCTION	8	4	5	7								8
TOKEN TEST Part A-E	67	2	5	12	20	30	45	52	60	62		67
TOKEN TEST Part F	96		0	20	35	50	61	65	67	75	85	96
TOKEN TEST Total	163	0	11	16	65	85	106	110	130	140	150	160
SENTENCE REPETITION	22			0	1	4	8	9	10	12	13	16
REPEATING DIGITS	14			0	1	2	3	4	5	6	7	9
REPEATING DIGITS REVERSE	14						1		2		4	7
AUTOMATIC SPEECH	4		0		2							4
CONFRONTATION NAMING	16	0	1	4	6	9	12	13	15			16
DESCRIPTION OF FUNCTION	16		0	3	7	9	13	14	15			16
TACTILE NAMING (Right)	8						0	2	4	6	7	8
TACTILE NAMING (Left)	8		1	2	3	5	6	7				8
GESTURE	16	1	12	13		15						16
FLUENCY	60					0	2	3	5	10	26	32
SENTENCE CONSTRUCTION	25							0	14	16	20	25
READING WORDS	8				1							8
WORD RECOGNITION	8	6	7									8
READING SENTENCES	7						3		6			7
SENTENCE COMPREHENSION	32		2	5	20	23	26	27	29	30	31	32
AUTOMATIC WRITING	5		0				3	4				5
SPELLING	24					0	3	14	18	19	22	24
DICTATION	13					0	4	9	11			13
COPYING	11		2	8	9	10						11
CALCULATION	10			1		3	5	6	7			10
ORAL	20	10		14	15	16	18		19			20
IDEOMOTOR	20	12	15	17								20
CONSTRUCTIONAL	20	12	18		19							20
BLOCK DESIGN	48		0	2	6	11	18	20	22	25		30
RAVEN'S MATRICES	36	10	15	20	22		25	27		30		35
		5	10	20	30	40	50	60	70	80	90	95

SUBTEST SUMMARY PROFILE (DEMENTIA)

NAME DATE

SEX

D of B

DIAGNOSIS

TEST	MAX. SCORE	PERCENTILES 5	10	20	30	40	50	60	70	80	90	95
RATING SCALE	40	10	13	17	20	22	24	26	27	30	32	35
IDENTIFICATION/NAME	8		7									8
IDENTIFICATION/FUNCTION	8	6	7									8
TOKEN TEST Part A-E	67		47	52	57	60	62		63		65	67
TOKEN TEST Part F	96		56	66		76	85	86	87	89	91	92
TOKEN TEST Total	163	92	114	126	133	139	147	148	152		159	
SENTENCE REPETITION	22		8	10	11		12	13			14	
REPEATING DIGITS	14		3	4	5		6		7		8	
REPEATING DIGITS REVERSE	14		1	2	3		4		5			
AUTOMATIC SPEECH	4		2		3							4
CONFRONTATION NAMING	16		12		14		15					16
DESCRIPTION OF FUNCTION	16		15									16
TACTILE NAMING (Right)	8		5		6		7					8
TACTILE NAMING (Left)	8		5	6		7						8
GESTURE	16		14	15								16
FLUENCY	60	2	4	6	8	9	10	12	13	17	23	
SENTENCE CONSTRUCTION	25		3	6	14	17	19	22	23			25
READING WORDS	8		7									8
WORD RECOGNITION	8		7									8
READING SENTENCES	7				3							7
SENTENCE COMPREHENSION	32	8	20	24		25	28	29	31			32
AUTOMATIC WRITING	5		2		4							5
SPELLING	24		9	13	16		21		22			24
DICTATION	13			10			11					13
COPYING	11		6	7	8	9	10					11
CALCULATION	10		4	6			7	8				10
ORAL	20		16	19								20
IDEOMOTOR	20			17	18							20
CONSTRUCTIONAL	20			8	11	14	16			17	19	20
BLOCK DESIGN	48				0	6	8		11		20	
RAVEN'S MATRICES	36	10	11	14		16	17	18	20		25	
		5	10	20	30	40	50	60	70	80	90	95