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Alzheimer’s Disease and the Utilisation of Procedural Learning.

Kathryn A. Russell

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Abstract

This thesis investigated the utilisation of procedural learning to improve self-care tasks and activities of daily living (ADLs) in mild-to-moderate Alzheimer's disease (AD). Procedural learning is relatively preserved in AD, presumably due to relatively little degeneration of critical neural circuits. Current interventions for AD (medical and non-medical) have limited effects on these skills, and attempts to enhance memory with explicit learning methods have been largely ineffective.

Two studies assessed the effectiveness of procedural training for ADLs in AD. Study one involved both a group and single subject design (multiple baseline across tasks) with 12 pre-selected tasks. Eight AD participants were trained on six tasks (counterbalanced across subjects) for 10 days, six tasks remained untrained. The group showed significant improvement at post-test in performance time and number of errors for trained tasks, whereas untrained tasks showed no significant change. At post-test no significant difference was found between the AD and control groups on trained tasks. Seven of the 8 participants showed a significant decrease in overall performance time (binomial analyses) and errors (visual analyses) for trained tasks. Significant training effects were maintained at 12-week follow-up. There was variability in training effectiveness between individuals and tasks.

Study two assessed the effectiveness of individually-tailored training programmes, in which tasks were selected for each individual according to task impairment and relevance. A single subject design, multiple baseline across tasks, was repeated across 6 participants. Change in task performance (time or errors) coincided with the onset of training. At post-test all participants showed some training benefits in either performance time or number of errors: 4 showed training effects on 77 to 100% of their tasks and 1 participant showed training effects in 60% of tasks. The final participant showed training effects in 75% of tasks, although a truncated follow-up period limited analyses. Training benefits endured throughout follow-up for 3 participants (2 to 4 months), and for at least 5 months for the 2 participants who had longer follow-up periods (some tasks endured up to 10 (KW) and 13 months (PB)).

Procedural learning methods can assist individuals with mild-to-moderate AD maintain self-care skills and ADLs. Additionally, this method could be adapted to provide a model of effective communication for caregivers, for example, breaking tasks into components and presenting requests one at a time.
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The most important contributors to this thesis have been the participants and their families. Without their willingness to participate in such a long-term study the research would not have moved forward from the original ideas. My participants, many of whom have now died, will remain in my memory. Several of them inspired me with their courage in the face of an often devastating and unrelenting disease.

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Preface

Thesis Rationale

This thesis will investigate the premise that individuals with AD may be able to improve their self-care and activity of daily living skills with the use of procedural retraining. This premise is based on findings that individuals with AD may be capable of a particular type of new learning. The findings of a considerable body of experimental research indicate that procedural learning (a type of implicit learning) is relatively well maintained in the mild-to-moderate stages of AD (e.g., Deweer, Ergis, Fossati, et al., 1994; Dick, Kean & Sannds, 1988; Eslinger & Damasio, 1986; Gabrieli, Keane, Stanger, Kjelgaard, Corkin & Growdon, 1994; Heindel, Salmon, Shults, Walicke & Butters, 1989). This retained learning ability provides a potential means by which some aspects of impaired functional skills in individuals with AD might be relearnt.

Research focusing on this idea has been carried out by the Dementia Research and Care Unit and Geriatric Research Group (Zanetti et al., 1997) from Bascia, Italy. Their research involved an exploratory investigation of the effectiveness of procedural learning on relearning functional skills. The research was designed and analysed using a simple group design. The study involved three weeks procedural training of 10 self-care tasks (10 tasks remained untrained). The results showed a decrease in the time taken by participants with AD to perform the trained tasks over a period of three weeks. However, they did not describe the accuracy of the participants on the tasks nor quantify the actual individual improvements in time taken. They did not investigate the duration of the effects of training in this study, although recently they repeated training with a new group of participants (and a control group) and published a 4 month single point follow-up. The results of this second study suggested the training effects were maintained by the active group for 4 months, whilst the control group showed no change (Zanetti et al., 2001).

The research in this thesis involves a more sophisticated design, which is not only capable of replicating the general findings of Zanetti and colleagues (Zanetti et al., 1997, Zanetti et al. 2001), but is also able to identify specific individuals who show gains from training. It also investigates, in greater detail, the duration of
improvements in performance of daily living skills. The research is based on strong experimental evidence of relatively spared procedural learning in the AD population. This study provides a test of the application of this spared ability to daily tasks and may potentially enhance our understanding of specific subsystems of memory and learning.

AD is one of the most pervasive and devastating disorders of older age which, as will be examined in chapter one, leads to an extensive loss of memory and of the ability to function independently. As of yet there are no effective treatments for AD. If it were possible to retrain and maintain specific self-care and daily-living skills, this would both improve the quality of life for individuals with AD and also improve the quality of life for caregivers. If the application of procedural learning techniques is effective, this could form the basis for the development of training programs for people with AD. These training programmes could be carried out by nurses, occupational therapists, trained AD caregivers and potentially even family members.

**Aims of Thesis**

The general aims of this thesis are:

1. To investigate whether self-care tasks can be either re-taught or improved in individuals with mild-to-moderate AD using procedural learning methods.
2. To determine the duration of any gains from procedural training. This will assist in judging the effectiveness of training, and indicate appropriate timing for when refresher training would be required.
3. To investigate the effectiveness of an individually tailored needs-based procedural training programme targeting self-care skills, which could improve the quality of life for people with AD.
4. During the course of the research a fourth aim was developed: To review the research on AD communication and distress, and discuss suggestions for assisting these areas of difficulty based on the findings of this thesis.
Outline of Thesis

Chapter 1 will provide a general overview of AD, including an outline of symptoms and current treatment trends, thereby providing the context for the intervention investigated in this thesis. Chapter 2 contains a review of the literature on procedural learning, including theories of procedural learning and how it is relatively preserved in AD. Chapter 3 is a research paper reporting the first study, which investigates the potential to utilise procedural learning to retrain a set of 12 self-care tasks in 8 individuals with AD, using a mixed group and single subject design. Chapter 4 describes study two, which investigates the potential to utilise procedural learning to retrain self-care and activity of daily living tasks based on the individual needs of 6 participants using a multiple baseline across tasks design. Chapter 5 is a discussion paper, which summarises literature and addresses issues of concern for people with AD and their caregivers in relation to the causes of distress in AD, and the role of communication. This chapter also draws upon findings from study one and two to suggest that the procedural learning method contains elements which model effective communication with AD individuals. The final chapter provides a general discussion of the findings of this thesis.
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1 Chapter One
General Introduction

1.1 Introduction

Alzheimer’s disease (AD) is a slow, progressive disease involving the degeneration of brain tissue, which leads to memory loss, confusion and the inability to function in many day-to-day tasks (Butters, Delis, & Lucas, 1995). In the final stages of the disease, individuals can lose the capacity to communicate both verbally and non-verbally, and can become bedridden and completely dependent on others for care. Researchers in fields as diverse as genetics, pharmacology, neurology, epidemiology, psychology and occupational therapy have studied different aspects of this disease. This thesis addresses one area of research, namely, self-care tasks and activities of daily living, which become severely disrupted during the progression of AD. More specifically it focuses on procedural learning in the mild-to-moderate stages of AD and the utility of these processes for improving impaired functional abilities.

Before reviewing current literature related to procedural learning and memory (chapter 2), an outline of the prevalence, neuropathology, and clinical presentation of AD (including symptomology, effects on functionality and the course of the disease) will be presented in this chapter. In addition, a brief review of the interventions or treatment approaches, both pharmacological and non-pharmacological, will be provided focussing particularly on approaches that attempt to improve functional self-care skills. This introduction will provide the context for the intervention investigated in the empirical studies contained in this thesis.

1.2 Prevalence, Incidence and Etiology of Alzheimer’s Disease

AD is the most common form of dementia with approximately 55% of all adult dementia’s being of the Alzheimer’s type (Rocca, Amaducci, & Schoenberg, 1986). A person with AD typically lives between 3 and 20 years from disease onset
(Reisberg, 1990). Many epidemiological studies have found the median survival time after diagnosis to be approximately 6 years (Wolfson et al., 2001). Katzman and Kawas (1994) found that approximately 10% of all people over 65, and 33% of people over 85 years, have AD. For people aged between 65 and 84 Anderson, Nielsen, Lolk, Andersen, Becker and Kragh-Sorensen (1999) found the incidence rate for very mild-to-severe AD in the Danish population to be 20.9 per 1,000 people per year. Bachman, Wolf, Linn, Knoefel, Cobb, Belanger, et al. (1993) conducted a 40-year longitudinal study with 2117 older adults (aged 60-94 years) and found the incidence of probable AD doubled with every 5 years, from 3.5 per 1000 at ages 65-69 to 72.8 per 1000 at ages 85-89. A meta-analysis of 23 published studies reporting age-specific incidence data of AD showed that incidence of AD rose exponentially up to 90 years of age, with no sign of levelling off. Women tended to have a higher incidence of AD in very old age and East Asian countries had a lower incidence of AD than did Europe (Jorm & Jolley, 1998).

There is currently no identified single or absolute cause of AD, although some risk factors have been identified, and the current view is that a variety of factors contribute to the development of the disease. Age is the greatest single risk factor for AD, although the mechanism in which ageing influences or contributes to the development is unknown (Katzman & Kawas, 1994). Another risk factor is being female (Rocca et al., 1990). This may relate to the women living, on average, longer than men do, although after adjusting for the statistically different length of life between the genders, Rocca et al (1990) showed that the ratio of women to men with AD is still 2:1. The possible reasons proposed for this difference include unrecognised environmental factors, hormonal effects (including menopause), the presence of one or more AD-related genes on the X chromosome, and the higher incidence of the apoE4 allele in women (Poirier et al., 1993).

The genetic component of AD has been said to have a variable role in the causation of AD across individuals. Although some families may have specific genes which predispose them to the development of AD, this development may still be dependent on environmental factors (Poirier, Danik, & Blass, 1999). A genetic predisposition to AD has been identified with between 10-40% of AD cases due to abnormalities on the chromosomes 1, 14, 19, 21 and possibly chromosome 12. Most individuals with Down’s syndrome develop AD by the time they reach 40 years of
age, suggesting a chromosome 21 link (Heston, Mastri, Anderson, & White, 1981; Wisniewski, Dalton, McLachlan, Wen, & Wisniewski, 1985). It is now thought that early-onset AD is linked to chromosomes 14 (presenilin 1) and 21 (amyloid precursor protein (β-APP)) whilst familial AD is linked to the chromosome 1 gene known as presenilin 2 (Post & Whitehouse, 1998). People with more apolipoprotein E ε4 (ApoE4) allele copies (chromosome 19) have increased risk of AD, as well as decreased age of onset, increased plaque density and decreased cholinergic neuron density and activity (Mayeux et al., 1995; Schmechel et al., 1993; Soininen et al., 1995). In recent years the relation between AD and genes has been increasingly researched, with genetic links being made to the course, severity, and nature of individual disease process, in addition to onset (Post & Whitehouse, 1998).

Head injuries increase the likelihood of AD, thought to be a result of the diffuse plaques (caused by head injury) turning into amyloid plaques (Mayeux et al., 1995). One population-based study showed that head injury was only a risk factor for apoE4 carriers, showing a possible interaction between genetic and environmental triggers (Mayeux et al., 1995). Theories focused upon toxic effects of contaminants suggest that exposure to ionic aluminium increases the risk for AD, although the evidence is contradictory (Birchall & Chappell, 1988). AD can develop without aluminium toxicity but a laser probe of analysis of brain aluminium in AD reported small aluminium accumulation in neurons with and without neurofibrillary tangles (NFTs) (Lovell, Ehmann, & Markesbery, 1993). Other risk factors include cerebrovascular amyloidosis, inflammation of the neurons (acute phase reactants), metabolic lesions (decreased metabolism/mitochondria), infections (slow acting viruses) and lower education levels (Stern et al., 1994b). The latter may be because the functional reserve of skills (such as cognitive skills) is more developed in individuals with a better education, thus providing added resilience to the effects of damage caused by AD (Stern et al., 1994b).

Some authors have attempted to create a more unified hypothesis of the creation of AD pathology, which acknowledges that there is probably not any one cause or even one single cascade. For example, Emmerling, Gracon, and Roher (1999) suggest that there are multiple pathologies and these collectively contribute to the overall pathology. As an example Emmerling et al. discuss the interplay between “the cardiovascular, immune and nervous systems, and the genetic background
against which the pathological processes occur” p41. They speculate that the presence of β-amyloid peptide amplifies any existing brain injury by any noxious mechanism (such as ischaemia), which in turn creates an ongoing immunological response (and therefore inflammation) to clear the protein and the related plaques. It is hypothesised that this inflammation leads to synaptic and neuronal loss (animal models show sustained inflammation causes such damage). Any difficulty with blood supply, resulting in subtle hypoxia and ischaemia, would exacerbate the condition. Age is an additional factor, although genetic background modifies the effects, e.g., the genetic effects on cardiovascular health, amyloid accumulation, and nerve growth and repair. They also hypothesise that genetic factors are more likely to affect the process, onset or duration of the disease, rather than cause the disease itself (with the exceptions of familial or early-onset AD) (Emmerling et al.).

A debate on the importance of inflammation on the AD process has developed over the last few years. It began with the epidemiological findings from several studies that Non-Steroidal Anti-inflammatory medications (NSAIs) reduced the prevalence of AD (McGeer, Schulzer, & McGeer, 1996). It is possible that inflammation is a secondary response to AD neuropathology; an immunological response to damaged neurons, amyloid deposits and tangles which over many years may significantly exacerbate the process that caused it (Akiyama et al., 2000). The effects of inflammation toxicity, its influence on protein synthesis or the interference it may exert with APP-metabolism are yet to be assessed (Fassbender, Masters, & Beyreuther, 2000). It is interesting to note that while some improvements with neuropsychological testing have been found for those with AD on long term NSAIs, no visible difference is found at autopsy (Halliday et al., 2000). Some researchers believe inflammation is far less important in the disease process and point to the metabolic factors, gene effects (APOE-4 allele and APP717 mutations), and neurobiological pathology as more likely primary influences (Roses, 2000).

### 1.3 Pathology of Alzheimer’s Disease

Several types of changes occur in the brain during AD including neurotransmitter loss, synaptic loss, nerve cell death, oxidative damage, inflammation, and excessive protein accumulation (Emmerling et al., 1999). This
section attempts to summarise some of the more simple, fundamental changes in structure and process.

1.3.1 Neuropathological Changes in Alzheimer’s Disease

Although changes are widespread throughout the brain (with exceptions of structures involved in procedural learning (see chapter 2), and primary visual and sensorimotor cortex) the most severe degeneration occurs, firstly, in the entorhinal cortex (input region to the hippocampus) (Juottonen et al., 1997) and then the CA1 region of the hippocampus (Squire & Kandel, 1999). The hippocampus is involved in making new memories. During the course of the disease the association areas of cerebral cortex are also affected by cell loss, especially in the frontal, medial limbic and temporal lobe regions (Damasio, Van Hoesen, & Hyman, 1990). Imaging techniques, such as CT scanning, show an apparent shrinking of the brain with a corresponding increase in size of the fluid-filled ventricles. In addition the fissures and sulci (the folds in the cortex) appear to become wider (Price, 2000; Terry, Masliah, & Hansen, 1994).

One of the long identified changes that occurs in AD is a degeneration and cell death of neurons that secrete acetylcholine during neurotransmission, especially neurons in the nucleus basalis (Damasio et al., 1990; Price, 2000). Along with changes in the number of neurons (due to cell death), there can also be changes in the cell structure as well as in the spaces between the cells. Affected neurons lose most of their dendrites (Kolb & Wishaw, 1995), thereby impairing their communication with other cells (Berg & Morris, 1990; Kolb & Wishaw, 1995). Recently it has been proposed that the lack of neuronal plasticity (differentiation control) is a critical factor in AD pathology. This theory suggests that normal cells are constantly changing synaptic contacts and making new connections. This type of structural plasticity is proposed to be lost in AD (Arendt, 2001). However, the two principle cortical changes which are characteristic of AD are neurofibrillary tangles (NFTs) and neuritic amyloid plaques (NP) (Price, 2000).

Plaques and tangles are hypothesised to be triggered by mutations in the amyloid precursor protein (APP) encoding gene (or by the binding of the amyloid protein with apo E4 (Beffert et al., 1998)). The cascade has been described by Hardy (1993) as follows:
1. APP mutations in the gene (mutations in presenilin 1 & 2 also lead to increased amyloid deposits (Gomez-Isla et al., 1999; Price, 2000)).

2. Alterations in metabolism

3. β-amyloid protein formation

4. Apo E4 binding to β-amyloid protein

5. β-amyloid deposits

6. Formation of plaques and tangles

7. Neuronal destruction

8. Dementia

Apo E4 is one form of the protein\(^1\). The other forms of the protein seem to be less related to AD. For example, if a person has the gene combination of APOE2/3 (as one type is inherited from each parent) the average age of onset for AD is 90 years. In contrast for those with APOE4/4 the average age of onset is early to mid 70s (Roses, 1995).

### 1.3.1.1 Neurofibrillary Tangles

The cytoplasm of affected neurons can have bundles of NFTs, which are abnormally twisted, paired helical protein filaments. The major component of these tangles is tau protein, which has changed shape because of increased phosphorylation (Roush, 1995), while the other major component of tangles is glycolipids (Goux, Rodriguez, & D.R., 1996). In a healthy neuron, the filaments support a transport network, with the tau proteins acting as structural cross-piece support for the tubules (creating part of the cytoskeleton). When the tau become twisted into helical filaments they no longer support the tubules causing the tubules to collapse, and clog the neuron so that nutrients and chemical messenger-filled organelles are unable to travel down the cell (National-Institute-of-Aging, 1995; Price, 2000).

Higher quantities of tangles are thought to be related to cognitive decline in AD (Damasio et al., 1990; Price, 2000). These tangles are predominately found in layers III and V of the entorhinal cortex, then in the CA1 region of the hippocampus and the subiculum (Boller & Duyckaerts, 1997; Squire & Kandel, 1999), possibly contributing to deficits in new learning. As the disease progresses NFTs may also

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\(^1\) Apo E is the protein notation, APOE is the gene notation.
become present in the association areas, but not the primary cortex (Arnold, Hyman, Flory, Damasio, & Van Hoesen, 1991). It has been found that higher concentrations of NFTs are more associated with intellectual deficits than higher concentrations of amyloid complex (Arends, Duyckaerts, Rozemuller, Eikelenboom, & Hauw, 2000).

### 1.3.1.2 Neuritic Plaques

The other major change in AD occurs in the areas between neurons. Senile (or neuritic) plaques form in these areas, consisting mostly of toxic β-amyloid$1-42$ (made during dysfunctional protein synthesis). β-amyloid binds to degenerated neuron endings that have become swollen due to neurofibrillary tangles. Apo E4 protein also tends to bind to β-amyloid (Stephenson, 1997).

The plaques that form between the neurons tend to make the surrounding neurons become twisted (Hof & Morrison, 1994; Roush, 1995) and they also limit many of the neuron’s processes (Hof & Morrison, 1994; Price, 2000). Links have been identified between β-amyloid dependent activation of microglia (latent phagocytes) and several other markers of neuronal cell death in AD (Combs, Karlo, Kao, & Landreth, 2001). There has been a clear correlation between the occurrence of AD and plaque concentration (Perry, 1986). As AD develops the density of plaques in Layer II of the neocortex increases (Arnold et al., 1991).

Plaques and tangles are not unique to AD. All healthy aged brains have some degree of plaques and tangles and excessive numbers have been noted in several other conditions such as Pick’s disease and Down syndrome (Terry et al., 1994). It is the specific pattern of distribution and density of plaques and tangles that is related to a diagnosis (and the symptoms) of AD (Damasio et al., 1990). The areas that are most affected by the plaques and tangles in AD are the hippocampal and amygdala areas, temporolimbic regions (those within the limbic system and temporal lobes), posterior cingulate gyrus, and the temoro-parieo-occipital junction area (the border region between the three regions) (Mann & Yates, 1986; Price, 2000). The areas that are relatively spared from the plaques and tangles are the primary motor cortex, primary somatosensory cortex, occipital lobe, as well as the anterior cingulate gyrus (Price, 2000). This corresponds with the functional decline in memory, emotional and cognitive functioning, and also explains why sight, hearing and perceptions of touch are not so affected (Squire & Kandel, 1999).
In addition to plaques and tangles, two other neuropathological changes are found in AD although these are not included in the diagnostic criteria. The first of these is granulovacuolar degeneration which occurs in the pyramidal neurons of the hippocampus (Mena, Robitaille, & Cuello, 1992; Price et al., 1986; Reisberg, 1985). The second neuropathological change is the presence of Hirano bodies; these are rod like structures that are seen in or near the cytoplasm of pyramidal cells. These tend to be seen primarily in the hippocampus and are more common in people with AD, although there is some overlap with healthy populations (Terry et al., 1994).

**1.3.2 Neurotransmitter Changes in Alzheimer’s Disease**

In addition to the physical changes to the brain structure in AD, there are also changes in the neurotransmitters systems (Beffert & Poirier, 1996). The most consistent finding has been a reduction of up to 95% in the level of cholinergic activity in the brains of individuals with AD compared with age-matched controls (Beffert & Poirier, 1996; Bondareff, Mountjoy, & Roth, 1981; Bowen, Palmer, Francis, Procter, & Lowe, 1988; Geula & Mesulam, 1994). This depletion appears mostly in the temporal lobe, including limbic, paralimbic and areas of association cortex (Geula & Mesulam, 1994). Some research suggests that it is the loss of cholinergic cells in the nucleus basilus, thereby reducing the cholinergic input to hippocampal structures, which underpins the impairments of new learning in AD (Broks et al., 1988; Christensen, Matlby, Jorm, Creasy, & Broe, 1992). The lack of acetylcholine not only affects the functioning of the hippocampus, but also causes widespread cortical dysfunction (Squire & Kandel, 1999).

There are also changes in the levels of the neurotransmitters norepinephrine, serotonin, dopamine and somatostatin (Damasio et al., 1990; Feldman & Grundman, 1999). The abnormalities of norepinephrine and serotonin reflect degeneration in the dorsal raphe nucleus and locus coeruleus in the brain stem (Gottfreis, 1994; Rossor, 1987). These neurotransmitters are necessary for communication between nerve cells and when the neurotransmitter levels are reduced, it may contribute to impaired neural functioning and consequently contribute to impaired cognitive functioning (Damasio et al., 1990).

AD is not a disease of a single neurotransmitter, it has many related, and interrelated, neurotransmitter and neuropeptide deficiencies (Feldman & Grundman,
For a detailed review of the increases and decreases of 12 neurotransmitters and 6 neuropeptides in six different regions of the brain refer to Gottfries (1994).

1.4 Clinical Presentation of Alzheimer’s Disease

1.4.1 Diagnostic Criteria

Diagnosis of AD is usually based on the criteria of the DSM IV (American-Psychiatric-Association, 1994) and the NINCDS-ADRDA (National Institute of Neurological and Communicative Disorders and Stroke, and the Alzheimer’s Disease and Related Disorders Association), refer Table 1 (McKhann, Drachman, & Folstein, 1984). To receive a diagnosis of AD from the DSM IV criterion the symptoms must include impairments of memory and at least one other cognitive impairment (e.g., aphasia, apraxia, agnosia, or difficulties with executive functioning). There must also be a gradual onset of the symptoms with progressive impairment. The deficits must exhibit a decline from how the person previously functioned and they must cause impairment in their social or professional activities. In addition, these symptoms must not be explained through any other condition that would create similar symptoms (e.g., psychiatric symptoms or substance abuse), and not be due to delirium (American-Psychiatric-Association, 1994).

The NINCDS-ADRDA (McKhann et al., 1984) (refer Table 1) diagnosis system has three levels of classification of AD: definite AD, probable AD and possible AD. The definite AD diagnosis is a probable or possible AD diagnosis with confirmation by biopsy or autopsy of brain tissue, which displays specific structural changes. The diagnosis of probable AD (which has an accuracy of 85% when confirmed by autopsy (Joachim, Morris, & Selkoe, 1988)) is established by clinical examination, as well as medical and neuropsychological tests. The individual must display deficits in two or more areas of cognition, have progressive memory difficulties (and other cognitive functions), be conscious and have no other brain disease to cause the deficits. The person must not have displayed the first symptoms of the disease before the age of 40 or after the age of 90. Possible AD is diagnosed if the clinician judges the cognitive decline to be caused by AD but there are other
Table 1
NINCDS-ADRDA Criteria for the Diagnosis of Definite, Probable, and Possible AD (McKhann et al., 1984)

1) Definite AD
   a) Patient meets clinical criteria for Probable AD.
   b) There is biopsy or autopsy evidence compatible with AD

2) Probable AD
   a) Dementia is established by a standardised mental status questionnaire and confirmed by
      neuropsychological testing.
   b) There are deficits in two or more areas of cognition.
   c) There is progressive worsening of memory and other cognitive functions.
   d) There is no disturbance of consciousness (delirium).
   e) The onset of the illness is between 40 years and 90 years of age.
   f) There is no systemic illness or brain disease that could account for the progressive mental
      status changes.
   g) The diagnosis of Probable AD is supported by the following:
      i) Progressive deterioration of specific skills such as language (aphasia), motorskills
         (apraxia), or perceptual recognition (agnosia).
      ii) Impaired activities of daily living and altered patterns of behaviour.
      iii) Family history of similar disorders, particularly if confirmed to be AD
           neuropathologically.
      iv) Laboratory results such as:
          (1) Normal standard lumbar puncture.
          (2) Normal or non-specific slowing on EEG.
          (3) Cerebral atrophy on neuro-imaging procedures with progression on serial
              documentation.
   h) Other features that are consistent with Probable AD diagnosis, once other causes are excluded
      include:
      i) Plateaus in the course of the illness
      ii) Associated symptoms such as: depression, insomnia, incontinence, delusions, illusions,
          hallucinations, emotional/verbal or physical outbursts, sexual disorders and weight loss.
      iii) Other neurological abnormalities such as increased muscle tone, myoclonus or gait
           disorder in advanced stages.
      iv) Seizures in advanced stages.
   i) Features that make Probable AD unlikely or uncertain include:
      i) Sudden onset.
      ii) Focal neurological findings such as hemiparesis, sensory loss, visual field deficits, and
          lack of coordination at early stages on the disease.
      iii) Seizures or gait disturbances at onset or early stages.

3) Possible AD
   a) There are variations in the onset, presentation, or clinical course of a dementing illness that
      are unusual in AD but for which there is no alternative explanation.
   b) There is a second systemic or brain disease capable of producing a dementia syndrome that is
      not considered to be the cause of the dementia in this case.
   c) There is a single gradually progressive deficit.

possible explanations for the decline. These secondary factors should not be significant enough to cause the deficits, but may contribute to the presentation. For example, the decline may be of a more variable than constant rate, or other significant events may have occurred such as a stroke (McKhann et al., 1984).
To date, no single test confirms the presence of AD in living people. However, one study showed that MRI hippocampal volume measurements performed by a blind assessor rated the presence of AD with 86% accuracy; and a delayed recall test (Russel’s adaptation of the Visual Reproduction Test) correctly classified the presence of AD with 94% accuracy (Laakso, Hallikainen, Haenninen, Partanen, & Soininen, 2000). In these cases the participants were identified with probable AD in accordance with the NINCDS-ADRDA criteria.

1.4.2 Symptomatology and Disease Course

1.4.2.1 Symptomatology

1.4.2.1.1 Memory Deficits

‘Memory’ and ‘learning’ are related terms. At this point they will simply be defined in lay language, a more detailed explanation of the differences will be found in chapter 2. Learning is the process of acquiring knowledge or skills of any kind. Memory, in contrast, is the ability to retain or store that information (Kupfermann, 2000).

An essential feature of AD is the loss of ability to store new memories. Consequently, the first symptoms noticed by families often occur when people with AD cannot remember recent events or names of once familiar acquaintances. With progression of the disease, general knowledge is lost along with more remote memories. Disorientation in both time and place is very common in people with AD, with questions about location, day, year, month or season causing much difficulty (Kertesz & Mohs, 1999). Memory itself, is a topic of much research and theoretical hypothesising. Since a specific kind of learning and memory is central to the research within this thesis, a summary of the types of memory and how they relate to each other is provided in Figure 1 and its accompanying key. This is based upon the model of memory proposed by Squire (1992b) for the explanation of memory systems. The process of how a memory is made is conceptualised at a basic level in Figure 2. A brief description of how each component/ type or conceptualisation of memory is affected by AD is presented below.
Key for Figure 1:

1) Explicit memory (interchangeable with declarative): this requires deliberate effort to recall or remember e.g., conscious reflection shown in tests of recall or recognition. This uses medial temporal lobe and diencephalon structures (Squire, 1992b; Squire, 1993; Tulving, 1985).
2) Declarative memory: this can be declared or brought to mind visually, in conscious awareness. (In animals such as rats, this type of memory is called relational or configural (Squire, 1992b)). Declarative is divided into three types of learning by some theorists:
   a) Automatic declarative learning e.g., repeating a word aloud many times.
   b) Eventful declarative learning e.g., a type of learning requiring effort, such as reversing a list of numbers.
   c) Contextual declarative learning e.g., learning that is related to other information (Lezak, 1995).
3) Episodic memory: is the memory for events, which contains both temporal and contextual information. Episodic memory uses bits of information to reconstruct memories e.g., an earthquake. This type of memory can be for one event (as no repetition is required), or for a sequence of events. Recall for this type of memory is active (as it is explicit) and changes because of differences in perception (Squire, 1992b).
4) Autobiographical memory: is memory for events in one’s own life (personal episodes) and for personal semantic knowledge also (Squire, 1992b).
5) Implicit memory: occurs without conscious recall or recognition (this was once termed procedural, but this has a different meaning at present) (Squire, 1992b).
6) Non-declarative memory: this is difficult to differentiate from the definition of implicit memory.
7) Reflexive memory: this is the automatic quality of the memory. This is not dependent on awareness or consciousness or even cognitive processes such as comparison or evaluation. It accumulates slowly (over repetition), and relies on performance of tasks. It is difficult to express in declarative sentences (Kupfermann, 2000).
8) Non-associative memory: is formed by learning from exposure to single stimulus, without related stimuli. This learning uses reflex pathways (Kupfermann, 2000).
9) Childhood imitation: this type of learning is thought to have elements of non-associative learning during the acquisition of language e.g., learning the grammatical rules of speech (Kupfermann, 2000).
10) Habituation: is the reduction of response level to repeated benign stimuli due to depression of synaptic transmission (Kupfermann, 2000).
11) Sensitisation: is the increased response to many stimuli following an intense or noxious stimulus due to enhancement of synaptic transmission (Kupfermann, 2000).
12) Procedural memory: is evidenced by an improved performance without conscious recall of performing the skill. This type of memory is dependent on striatal structures (Squire, 1992b).
13) Associative memory: this is based on what an experimenter does e.g., classical conditioning and operant conditioning (Kupfermann, 2000).
14) Classical conditioning: this type of learning relates one stimulus to another. The amygdala and medial dentate are important for emotional responses along with parallel circuits in the lateral interpositus nuclei of the cerebellum for skeletal musculature responses (Kupfermann, 2000).
15) Operant conditioning: this type of learning relates stimuli to an organism’s behaviour (Kupfermann, 2000).
16) Priming: this involves improved recall of previously experienced stimuli e.g., improved recall of previously seen words if presented with first letters of those words; even without conscious recall of ever having seen those words. This type of learning is dependent on the neocortex (Kupfermann, 2000).
Figure 1. Memory Systems.

Memory: verbal, non-verbal and spatial.

Refer figure 2

Short term/working memory

Explicit memory (1)
Declarative memory (2)

Episodic (events) (3)
Semantic (facts/general information).

Non-Autobiographical.
Autobiographical (4)

Implicit memory (5)
Non-declarative (6)
Reflexive (7)

Non-associative (8)

Imitative childhood
Language acquisition (9)

Habituation (10)

Sensitisation (11)

Procedural (12)

Dispositions
Associative (13)

Classical conditioning (14)

Operant Conditioning (15)

Priming (16)

Perceptual priming

Semantic priming

In addition to direct implicit learning: declarative memory, by repetition, may become reflexive and procedural (e.g., learning to drive).

# Both declarative and reflexive elements may contribute to conditioned responses.

1. Based on concepts and diagrams by Squire 1992. Numbering denotes explanation to be provided in the key on the facing page. If the concepts/terms have similar meanings, they appear in the same box.
1. **Registration/Sensory memory.**
   Lasts milliseconds. One component is iconic memory involving retinal or auditory after-imaging.

2. "Short term” storage.
   a. **Primary/immediate memory.**
      Lasts approximately 30 seconds without rehearsal, 7 items average, attention dependent.
   b. **Active/Working memory.**
      Simple rehearsal by overlaying same neural pathways e.g., excitatory feedback loop. Information can be manipulated.

3. **Rehearsal**
   Can last hours.

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1. **Recent memory.**
   Part of the consolidation process. This is more easily disrupted than remote memory until it is converted to Long term storage.

2. **Long Term Storage/Remote memory.**
   This is stored in the neocortex by long term potentiation. It is insensitive to disruption but is eventually lost from Long term storage.

3. **Retrieval**
   This process involves finding the information once it has been stored. This enables the recall of the information stored in Long term memory.

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**Short Term Memory:** Electrochemical activation of synapses.

**Long term memory:** Semi-permanent in cell structure or protein synthesis to connections between neurons and physical changes such as new synaptic connections.

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**Consolidation.** (Hippocampus, Temporal lobes, Diencephalon and Neocortex).

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Figure 2. Explicit memory and storage.
(Kupfermann, 2000; Squire, 1992).
The categories described below often overlap and in no way represent a complete explanation of memory in AD.

1.4.2.1.1 Short-Term, Working and Long Term Memory

Short-term memory (about 30-60 seconds in duration without rehearsal) can be disturbed in early AD, and declines with the progression of the disease, in contrast to amnesia where it is usually preserved (Corkin, 1982). “Working memory” (holding and manipulating information (Kertesz & Mohs, 1999)) shows an increased rate of forgetting between 15 seconds and 10 minutes for AD individuals compared to controls (Hart, Kwentus, Taylor, & Harkins, 1987; Moss, Albert, Butters, & Payne, 1986). For explicit knowledge to enter long-term memory it must first be held intact in working memory and/or rehearsed (Kertesz & Mohs, 1999), consolidation of the knowledge is then required (see Figure 2). Long-term memory refers to the relatively long-term storage of information, which a person can recall following distraction (Squire, 1993). The rate of storage into long-term memory is severely impaired in AD, as found in verbal learning tests (Petersen, Smith, Ivnik, Kokmen, & Tangalos, 1994), and visual learning tests (Freedman & Oscar-Berman, 1989; Kato, Knopman, & Liu, 2001) of mild AD participants.

In the earlier stages of AD it may be possible that the prominent memory deficit is caused by impairment in acquisition and encoding rather than the forgetting or destruction of the memories once they are acquired (Petersen et al., 1994). In the mild-to-moderate stages of AD it is likely, firstly, that memories are not acquired, and secondly, those that are acquired into long-term memory are lost by the degeneration of neurological tissue that contained those memories (Petersen et al., 1994).

1.4.2.1.2 Implicit and Explicit Memory

One conceptualisation of memory distinguishes two categories of memory, implicit and explicit memory. Explicit memory depends on a person’s conscious recollection of an experienced event. Explicit memory includes semantic and episodic memory, and involves a deliberate effort to remember, including both recall and recognition abilities. Implicit memory, in contrast, does not rely on conscious awareness but is reflected in changes in behaviour or knowledge of a skill despite lack of conscious recollection (Graf, 1994; Squire, 1993). Implicit memory includes classical conditioning, priming and procedural skills learning and memory. Implicit
memory can be described as knowledge that is expressed in performance without an individual necessarily being aware that they even possess it.

In AD, forming explicit memories is impaired possibly due to problems with encoding, attention and retrieval (Kertesz & Mohs, 1999). Although explicit memory of distant memories can often be recalled, these memories too become affected over the course of the disease. In contrast, while some aspects of implicit memory (including classical conditioning, and lexical and semantic priming) show some deficits in early AD, other types of implicit learning, such as procedural learning, remain relatively intact (refer chapter 2) (Butters, Heindel, & Salmon, 1990).

1.4.2.1.2.1 Episodic Memory

Episodic memory is memory for events (recent or remote) that contain both temporal and contextual information. New learning of this type of memory is impaired in individuals with AD from very early on in the course of the disease (Kertesz & Mohs, 1999). From the early stages of AD there is development of a progressive retrograde gradient amnesia, which, in the severe stages of the disease, affects remote memories for all the decades of the person’s life (Bondi, Salmon, & Butters, 1994; Carlesimo & Oscar-Berman, 1992; Price, 2000; Squire & Kandel, 1999). This pattern was established in a variety of studies using historical cues such as pictures of famous people or news events (Cohen & Squire, 1981; Sagar, Cohen, Sullivan, Corkin, & Growdon, 1988; Squire & Cohen, 1982). Autobiographical memory is the memory of the events and knowledge in one’s own life. The temporal gradient decline is also present in AD for events in people’s own lives, with those events in their childhood being retained for the longest time (Sagar et al., 1988).

1.4.2.1.2.2 Semantic Memory

Semantic memory refers to a component of long term memory which represents knowledge of objects, facts, and concepts, as well as words and their meanings (Tulving & Donaldson, 1972). Dependent on language, semantic memory begins to be acquired at a young age and it is not bound to particular times (Kertesz & Mohs, 1999). In comparison with episodic memory, semantic memory may be relatively preserved in the early stages of AD, although semantic memory impairments are thought to develop soon after episodic impairments in many individuals with AD (Hodges & Patterson, 1995). Word fluency is often used as a
measure of semantic memory in AD. Both letter-prompted fluency and category fluency are impaired in AD (although there are factors other than semantic memory required for these tasks, such as attention and initiation) (Ober, 1986, Butters, 1987). AD individuals may still retain awareness of semantic categories once they have lost knowledge of individual exemplars (Warrington, 1975); for example they may say a carrot is a vegetable, but cannot name it as a carrot.

1.4.2.1.2 Language Deficits

Language deficits occur in many individuals during the early stages of AD, and are found to be universal in the moderate-to-severe stages (Faber-Langendoen et al., 1988). There is some overlap between these difficulties and semantic memory impairments. As AD advances in to the moderate stages, semantic impairments (e.g., verbal fluency, naming tasks) and complex verbal comprehension difficulties are common, although day-to-day conversation is moderately well maintained (Bayles & Kaszniak, 1987). In the moderate stages of AD, there are increasingly fewer intact sentences, although sentence meanings may still be communicated. There are increasing numbers of incorrect word substitutions e.g., ball for bag (most likely related to semantic memory problems). Comprehension difficulties also increase and the classification of Wernicke’s aphasia may be applied. Repetition and perseveration of phrases may also be common (Appell, Kertesz, & Fisman, 1982; Cummings, Benson, Hill, & Read, 1985; Murdoch, Chenery, Wilks, & Boyle, 1987). Writing ability and reading comprehension are also affected in the moderate stages (Murdoch et al., 1987), whilst reading aloud and the ability to repeat a sentence or word that is spoken to them may still be relatively spared (Appell et al., 1982). In the advanced stages of AD fluency decreases, perseveration of words or phrases increases, and non-speech utterances (noises) become more common. The communication pattern is severely disturbed, with sentences getting progressively shorter until one word utterances (often yes or no) are all that occur. Eventually no verbal or non-verbal expression remain. In addition to the ceasing of verbal expression there is little apparent comprehension (although the exact level is often impossible to assess) (Appell et al., 1982).
1.4.2.1.3 Visuospatial Deficits

Visuospatial problems can occur in the early stages of AD. These usually appear as route-finding difficulties, getting lost in familiar areas, and the inability to use machinery (Kertesz & Mohs, 1999). Neuropsychological tests, which assess visuospatial abilities, include drawing patterns, copying figures, line orientation, and block design (Kertesz & Mohs, 1999). Spatial disorientation is considered to be a combination of loss of visuospatial memory, and visuoperceptual dysfunction (Flicker, Ferris, Crook, & Reisberg, 1988). This impairment is related to bilateral parietal degeneration (Kertesz & Mohs, 1999) and typically worsens during the course of the disease (Kertesz & Mohs, 1999).

1.4.2.1.4 Executive Functioning Deficits

Deficits of executive functions (reliant on the frontal regions of the brain) such as difficulties with planning, sequential organisation, attention and problem solving begin to appear during the course of the disease (Butters et al., 1995). Impairments of judgement, insight, motivation, flexibility (hence perseveration) and inhibition may also be seen. In addition, apathy may be seen as a clinical feature in some individuals with AD (Kertesz & Mohs, 1999). In a practical sense, these deficits are evident as impaired problem-solving in day-to-day living. There is often impaired judgement in areas such as social conduct, resulting in inappropriate social behaviour and lack of inhibition (including sexual disinhibition) affecting a variety of behaviours (Eastwood & Reisberg, 1999). The attentional and information-sorting functions of the frontal lobes may exacerbate the experience of memory deficits as this region contributes to working memory and storage. Deficits in the frontal region may also make the utilisation of strategies, which facilitate learning, difficult (Kopelman, 1991; Miller et al., 1991).

AD is generally viewed as a parietal/temporal dementia, and contrasted with frontal lobe dementia. However, it has been found that frontal lobe (and related executive functioning) impairments are present in AD, which, although less prominent than memory problems, become more pronounced as the disease progresses (Duke & Kaszniak, 2000). In contrast, in frontal lobe dementia executive dysfunction occurs earlier in the disease process and is more severe (Duke & Kaszniak).
1.4.2.1.5 Mood and Behaviour Changes

Changes in mood can include depression, anxiety, agitation and irritability, as well as symptoms of psychosis such as hallucinations and delusions. Some individuals may display a flat affect. The behavioural changes may include: wandering, sundowning patterns\(^3\), increased compulsive behaviours, screaming, restlessness, and verbal or physical violence or aggression (Eastwood & Reisberg, 1999; Folstein & Byslma, 1994). Other changes may have alternative explanations. Sundowning, for example, may be related to melatonin levels (Björeksten, Basun, & Wetterberg, 1995; Mirmiran, Swaab, Witting, & Honnebier, 1989). In addition to mood and behavioural difficulties, eating patterns and eating behaviours may also change (Eastwood & Reisberg, 1999).

1.4.2.2 Disease Course

The symptoms described above come together to form a progressive and potentially terminal disease. The initial signs of AD usually involve difficulties with forming new memories and simple semantic difficulties such as problems with naming objects (Bayles & Tomoeda, 1991; Chenoweth & Spencer, 1986). The subtle behavioural and mood changes (such as anxiety, mild depression, lowered motivation, and changes in sleep or eating patterns) are often confused with a major depressive episode, which can present in a very similar manner to early AD (Lezak, 1995; Rabins, 1998).

There is some research describing a ‘preclinical’ phase in AD. It is suggested that this phase preceeds AD by many years. In one population-based study over 22 years with 1076 people, neuropsychological testing showed a difference between the individuals that did and did not develop AD (Elias et al., 2000). Elias et al. concluded that although this difference was present in the groups, it would be difficult to use this information to predict which individuals would develop AD due to the overlapping ranges of performance for those that developed AD and those that did not.

Each individual with AD has a distinct, individual course and pattern of cognitive decline and not all symptoms are present in all individuals (Kertesz &

\(^3\) Sundowning is increased activity and/or agitation in the late afternoon and night and decreased activity in the morning (Folstein & Byslma, 1994).
The time span of decline is variable, with some individuals showing rapid decline whilst others show only one or two types of symptoms for several years. Those with only one copy of apoE4 have a slower rate of decline in comparison to those with multiple copies (Stern et al., 1996). Occasionally people with AD can go into a period of remission, also known as a plateau (Kertesz & Mohs, 1999). Huff, Growdon, Corkin, and Rosen (1987) have suggested that this variability in disease course could be due to age of onset, however, several studies have found that age of onset, duration of disease and family history of disease do not predict the speed of deterioration (Ortof & Crystal, 1989; Salmon, Thal, Butters, & Heindel, 1990; Stern et al., 1994a). In terms of the disease course for people with AD, after 8 years of symptoms 50% of a sample population with AD are still alive (Barclay, Zemcov, Blass, & Sansone, 1985).

Thomas, McGonigal, McQuade, Starr, and Whalley (1997a) found the 5 year survival rate for 451 individuals with early-onset AD (aged in 40s or 50s) was 32% for men and 43% for women. Several studies have found that there is no difference in the disease course (i.e., speed of the onset of difficulties) of early-onset AD compared to late-onset AD (Bracco et al., 1994; Haupt, Kurz, & Pollmann, 1992; Haupt, Pollmann, & Kurz, 1993). People with early-onset AD may, nevertheless, reach more advanced stages of the disease, because they may live longer with AD (Brandt, Mellts, Rovner, Gordon, & et al., 1989).

At the advanced stages symptoms increase and people are typically unable to dress, feed or bathe themselves. In addition they may experience aphasia, agnosia, apraxia, a parkinsonism-like gait and even convulsive seizures (Butler & Lewis, 1977). In practical terms, these deficits result in changes in walking speed or gait, and eventually in the loss of mobility. Vocabulary diminishes continuously until it is reduced to one word; this word may be persistent, depending on the individual, and is commonly “yes” or “no”. The word may be used in response to all stimuli and once that last word goes the only communication is with incoherent noises such as grunts. In the final stages of the disease, individuals are frequently unable to sit, smile or hold up the head (Bergener, 1987). Sulkava, Haltia, Paetau, Wilkstrem and Palo (1983, cited by Bergener, 1987) found that 14% of AD autopsies show no immediate cause of death other than the disease itself. In these cases, the central regulation of vital functions had been damaged enough by AD to cause death.
1.4.3 Functionality

The term functionality has been used when talking about ‘quality of life’ and ‘active’ life expectancy as opposed to actual life expectancy and mortality (Katz et al., 1983). Functionality may be influenced by symptoms of disease, such as cognitive function, but is not defined by them. Examples of functional ability include the ability to dress, or use a toilet. These are commonly referred to as activities of daily living (ADL’s) (Gallo, Reichel, & Andersen, 1995).

The deterioration of functional abilities in daily life has a significant impact on individuals with AD and their families or caregivers (Ferm, 1974; Teunisse, Derix, & Van Crevel, 1991). A decline in functionality is an essential feature of AD and is included in the criterion for probable dementia in the NINCDS-ADRDA (McKhann et al., 1984) and the DSM-IV (American-Psychiatric-Association, 1994). Importantly the deterioration of functional abilities has been found to be a predictor of institutionalisation (Mittelman et al., 1993; Riter & Fries, 1992).

There is a significant relationship between cognitive losses and functional losses. Auer, Sclan, Yaffee, and Reisberg (1994) reported a significant correlation, \( r = -0.77 \) (\( p < .05 \)), between the Modified Ordinal Scales of Psychological Development and the Functional Assessment Staging (FAST) scale. The functional deficits in AD probably have multiple causes related to deficits in cognition (e.g., memory and attention), perception (e.g., objects and place recognition, and spatial orientation), motor skills (praxis), and executive functions (e.g., judgement, planning, motivation, and initiation) (Carswell & Eastwood, 1993). The decline of executive functions may also impact on the ability to sequence activities correctly, as well as decrease spontaneity and increase difficulty completing tasks once they are started (Skurla, Rogers, & Sunderland, 1988).

The specific course of the functional deterioration varies for each individual. Several other studies have shown weak positive correlations between cognitive ability and functional ability (e.g., Reed, Jagust, & Seab, 1989; Teri, Borson, Kiyak, & Yamagishi, 1989), although they did not assess every area of cognitive ability focusing primarily on memory and attention. This suggests, however, that functional decline may not be solely dependent on memory decline. Other factors which may further complicate functional ability, include those that may also impact on neurologically intact older people, such as physical limitations (e.g., immobility due
to hip replacement), non-neurological medical conditions (e.g., arthritis, angina), medication (e.g., benzodiazepines), and psychosocial factors (e.g., mental health and social support).

Functional decline in AD can result in the experience of emotional and behavioural reactions such as frustration or humiliation (Carswell & Eastwood, 1993). These in turn will impact on social functioning (Carswell & Eastwood, 1993) and will eventually lead to increased demands on caregivers. Additionally, as a person with AD becomes less able to function independently there may be safety issues for them staying at home alone.

Functional decline typically progresses in a hierarchical pattern (Carswell & Eastwood, 1993; Green et al., 1993). In the early stages this may be noticed as difficulties with complex occupational tasks (Reisberg, 1984). For example, an electrician may have increasing difficulties wiring up circuit boards, and will probably have to give up work, as it becomes too difficult and dangerous. As AD progresses the individual may have trouble organising their finances or finding their way around unfamiliar environments (Reisberg, 1984). Further down the track the individual may have difficulty driving, or operating household appliances such as ovens. Even in moderate stages of the disease, activities such as dressing, grooming and bathing, or toileting become more difficult (Reisberg, 1984). Previously simple tasks become increasingly difficult. For example, an individual might make a cup of tea but rip off the end of the tea bag and pour the leaves into the cup. Alternatively, they may forget to use toothpaste when brushing their teeth. Each of these apparently simple daily tasks is comprised of several components which the person must perform in the correct order. In addition, the individual must correctly identify objects (e.g., toothbrush) and use them in the appropriate way.

As noted previously, in the more severe stages of the disease incontinence occurs and eventually communication and ability to walk are lost (Reisberg, 1984). At the extreme, the ability to smile or even to hold up the head are lost (Sclan & Reisberg, 1992).

1.5 Treatment Approaches

There are several interventions or treatment approaches for AD (pharmacological and non-pharmacological), which attempt to improve, slow the
decline of, or prevent the decline of functioning. These interventions focus on many
different aspects of the disease process such as cognitive ability, functional ability,
mood, or quality of life.

1.5.1 Pharmacological Approaches

Effective pharmacological treatments of AD are difficult to develop for three
reasons. Firstly, there are multiple neurotransmitter changes that occur within the
disease process. Secondly, it is very difficult to replace lost neurotransmitters in a
way that mimics normal neuro-chemical release (Francis, Cross, & Bowen, 1994).
Thirdly, in addition to neuro-chemical loss, there is also a loss of neurons in some key
areas such as the hippocampus. Feldman (1999) has commented that it is unlikely
that the correction of a single neurotransmitter abnormality would counter the
widespread pathology of AD. Nevertheless, many types of medication have been
investigated with varying results. At this point there is only evidence for
symptomological benefits, although some of the therapies described below may have
mild protective properties.

New pharmacological treatments for AD are continually being trialed, and the
literature is constantly changing. In a review by Bayer (1998) 67 pharmaceutical
companies were investigating 180 potential anti-dementia agents in 1991. Since then
many more companies have attempted even greater numbers of studies. The most
researched mechanism involves the cholinergic system, but other commonly
investigated approaches include: neuro-endocrine systems (oestrogen), anti-
inflammatory agents, antioxidants, and neurotrophins (nerve growth factor). In
addition, adrenergic agents, serotonergic agents, glutaminergic agents, neuropeptides,
opiate antagonists, angiotensin-converting enzyme inhibitors, aluminium chelators,
anti-platelet agents (aspirin) and hyperbaric oxygen are also being investigated
(Bayer, 1998). More recently four concepts have shown promise: protein kinase
inhibitors, which protect against programmed cell death (Maroney et al., 1999);
nicotinic acetylcholine receptor subunit agonists, which mediate cell survival,
trophism and plasticity (Belluardo, Mudo, Blum, & Fuxe, 2000); nootropic
medications, which affect metabolism and enhance blood flow (Bae et al., 2000); and
amyloid deposit modulators, which block enzymes involved in plaque formation (e.g.,
a vaccine) (Schenk et al., 1999).
The major types of pharmacological interventions for AD have been conceptualised into three broad categories, those that act to alter symptoms, those that stabilise AD, and those that prevent or delay the onset of the disease (Gray & Gauthier, 1999). Feldman and Grundman (1999) consider symptomatic treatments to be interventions that result in measurable improvement in the cognition, behaviour or function of the person with AD. Stabilising treatments are those that slow the clinical decline by modifying the disease process (probably at the earlier stages of the disease) and prevention therapies delay the onset of symptoms in people with pre-diagnosis memory problems, or even healthy populations. Gray and Gauthier (1999) described the theoretical differences between the symptomatic and stabilising approaches: symptomatic agents create short-term improvements without having any impact on the underlying deterioration (masking the problem). They show an initial improvement, then a parallel decline to a placebo intervention, and regression to placebo level if the treatment is withdrawn. In contrast the stabilising agents (or disease modifying agents) slow the disease progression itself. The decline is at a slower rate, and only becomes parallel to placebo when the treatment is withdrawn, thereby maintaining the advantage over intervention with placebo (Gray & Gauthier).

1.5.1.1 Cholinergic Therapies (Symptomatic)

The major movement of research and currently available treatment medications are ‘cholinergic therapies’ (Feldman & Grundman, 1999). Cholinergic therapy was developed to address the degeneration of cholinergic neurons in AD, the role of acetylcholine in attention and memory in AD, and the hypothesis that increasing cholinergic activity enhances cognitive function (Feldman & Grundman). There are three mechanisms within the cholinergic system which have been targeted for intervention (Bayer, 1998). The first of these is a presynaptic approach; these medications include precursors and acetylcholine release enhancers. The second of these acts at the synaptic cleft i.e., cholinesterase inhibitors. The last of these is a post-synaptic approach; these medications include muscarinic agonists, nicotinic agonists (Bayer, 1998) and uptake enhancers (Huff et al., 1996).

At this point, effects of presynaptic interventions appear to be inconclusive, although there is some reduction of the rate of decline for early onset individuals (Bayer, 1998; Brooks, Yesavage, Carta, & Bravi, 1998; Feldman & Grundman, 1999).
The postsynaptic interventions are also inconclusive, although they have shown potential for further research (Feldman & Grundman, 1999). For example, intravenous nicotine showed some benefits (Bayer, 1998).

The synaptic gap medications (acetylcholinesterase inhibitors) have had the most research success (Bayer, 1998; Feldman & Grundman, 1999). These include four currently available medications which ‘enhance’ cholinergic activity (cholinesterase inhibitors): tacrine (Cognex), donepezil hydrochloride (Aricept), rivastigmine tartrate (Excelon and Prometax) (Feldman & Grundman, 1999) and galantamine (Nivalin and Reminyl) (Blesa, 2000). Mild benefits have been demonstrated in clinical trials for anticholinesterase inhibitors in comparison to placebo controls (Bayer, 1998). The acetylcholinesterase inhibitors results vary, and reports of symptomatic response rates vary from 5-40% over many studies of the various medications (Feldman & Grundman, 1999). There has been some suggestion that certain APOE genotypes may be less responsive to treatment (Bayer, 1998). The long term affects of cholinesterase inhibitors are now beginning to be analysed. A follow-up study of several cholinesterase inhibitors showed that there were two long term benefits, increased cognitive functioning at one year and decreased nursing home admissions over 3 years (Lopez et al., 2002). However, cholinesterase inhibitors had no effect on MMSE scores, activity of daily living (ADL) scores or rate of deaths over 3 years (Lopez et al.). In contrast, an evaluation of galantamine (which also modulates nicotinic receptors) found improvements on ADL tasks, behavioural features and caregiver burden, in addition less supervision time was required (Blesa, 2000). A review of galantamine investigations confirmed those findings, and also reported that donepezil and rivastigmine also improved ADL tasks (Winblad, 2001).

**1.5.1.2 Neuro-endocrine Therapies (Stabilising, Preventative)**

A variety of neuro-endocrine abnormalities have been found in AD, including lowered oestrogen levels (Feldman & Grundman, 1999). Post-menopausal women not on Hormone Replacement Therapy have a significantly increased incidence of AD (Baldereschi et al., 1998; Giacobini & Michel, 1998; Henderson, 1997; Kawas et al., 1997; Paganini-Hill & Henderson, 1994). Once AD has been diagnosed, however, there is no significant cognitive or functioning enhancement if oestrogen is taken (Feldman & Grundman, 1999; Mulnard et al., 2000; Weiss, 1987), although there is
some suggestion that oestrogen may enhance the affect of the cholinergic therapies (Schneider, 1995). It has been hypothesised that oestrogen affects the brain as a result of stimulating nerve growth factor production, acetylcholine synthesis and inhibits the release of amyloid precursor protein (Gray & Gauthier, 1999). It has also been found that oestrogen promotes synaptic plasticity (Foy, Henderson, Berger, & Thompson, 2000). Future research into the influence of oestrogen in AD will probably focus on prevention.

1.5.1.3 Anti-inflammatory Therapies (Stabilising, Preventative)

Patients with rheumatoid arthritis on anti-inflammatory medication have demonstrated a relatively low incidence of AD in more than 17 epidemiological studies (McGeer et al., 1996). Anti-inflammatories may lessen the histological response to plaques and tangles thereby slowing the disease progression (Aisen & Davis, 1997). Several studies with anti-inflammatories have shown promise (Broe et al., 2000; MacKenzie & Munoz, 1998; Mackenzie, 2000; Stewart, Kawas, Corrada, & Metter, 1997). An improvement on the ADAS cognitive assessment scores was seen when AD participants were taking indomethacin versus placebo (Rogers et al., 1993). Propentofylline has produced promising results: 901 AD participants took Propentofylline for 24-56 weeks and showed improvements when compared to the placebo group, while a withdrawal phase showed that Propentofylline had relieved symptoms (symptomatic) and slowed progression of AD (stabilisation) (Rother et al., 1998). It has been proposed that once inflammation is established in the AD brain, anti-inflammatories are unlikely to significantly affect the disease process, and the best use of NSAIs is related to their protective properties if taken before the onset of AD (Thal, 2000).

1.5.1.4 Antioxidants (Stabilising/Preventative)

Vitamins E, C, and beta-carotene, coenzyme Q-10, idebenone and monoamine oxidase B (MAO-B) inhibitors have anti-free radical actions (Gray & Gauthier, 1999). It has been argued that the antioxidant action may prevent the aggregation of amyloid proteins and prevent neuron damage through oxidation (Gray & Gauthier, 1999). In-vitro studies of primary cultures of certain neurons have increased survival when vitamin E was added (Nakajima et al., 1991). Vitamin E also prevented the loss of cholinergic function in rats with neurotoxic lesions (Maneesub, Sanvarinda, &
Vitamin E was also found to protect sympathetic nerve cells from amyloid-beta protein-induced cell death (Behl, Davis, Cole, & Schubert, 1992). The application of these vitamins in human AD studies has only begun to be investigated (Gray & Gauthier, 1999), although there have been suggestions that these vitamins may be protective against AD (Gray & Gauthier, 1999). A randomised, double blind placebo controlled trial with 314 AD participants tested the effect of both vitamin E and MAO inhibitor on AD. The findings suggested that both of these agents slowed the progression of AD (Sano, Ernesto, Thomas, & Klauber, 1997). Several studies on MAO inhibitors have shown evidence for the slowing of the disease progression (Lawlor, Aisen, Green, Fine, & Schmeidler, 1997; Sano et al., 1997), although there is some discrepancy in the research as to symptomatic benefits (Gray & Gauthier, 1999).

1.5.1.5 Nerve Growth Factors (Stabilising)

In animal studies nerve growth factors (NGF) enhanced the size and activity of cholinergic neurons (Tappen, 1997; Thal, 1994). Once cells die, neurotransmitter stimulation has no effect. NGF stimulates cells to make new connections and protects cells from damage (Lahiri, Ge, & Farlow, 2000). Some minor benefits have been reported from intraventricular infusion of NGF for a person with advanced AD, but it is difficult to deliver effectively, and adverse reactions may outweigh the benefits (Bayer, 1998). An intravenous NGF and several oral agents are currently under investigation (Gray & Gauthier, 1999).

1.5.1.6 Neuroleptic Medication (Symptomatic)

Behavioural difficulties and psychosis can be treated in AD with neuroleptic medication, just as they are in people without AD. These may include a range of antidepressant, antipsychotic, anxiolitic (Benzodiazepine), anti-manic (lithium), and anticonvulsant medication. A meta-analysis of neuroleptic therapy with AD showed only 18% of patients received any benefit (Schneider, Pollock, & Lyness, 1990). Double blind placebo controlled trials of neuroleptic medication withdrawal found no differences in behavioural disturbances or staff distress between active group and placebo, concluding that any beneficial effects of neuroleptics would only be short-term (Bayer, 1998). The final query with neuroleptic medications is that many have an anti-cholinergic action, which may increase some of the problematic cognitive
symptoms (Bayer, 1998). In contrast, selective serotonin reuptake inhibitors (SSRIs) are non-sedating, have no anticholinergic effects (Bayer, 1998), and have been found to improve the mood of mildly depressed people with AD (Sultzer, Gray, Gunay, Wheatley, & Mahler, 2001).

### 1.5.2 Non-pharmacological Approaches

There are many non-pharmacological techniques used to assist people with AD. These range from music therapy, art or occupational activities, to therapies directly aimed to improve cognitive function (Tappen, 1997). This section will focus on some of the more common and well-researched, therapies.

#### 1.5.2.1 Memory Rehabilitation

This approach targets memory management in people with memory deficits, including AD (Wilson, 1987). The primary principles are the systematic use of memory aids such as diaries, which may be useful in the early stages of AD. More recently, electronic aids have shown promise as prompts, with auditory reminders set at certain times (De Vreese, Neri, Fioravanti, Belloi, & Zanetti, 2001). Individuals with more advanced AD, however, typically do not remember to use memory aids (Miller & Morris, 1993). A familiar and secure environment with predictable routine, which avoids excessive demands on memory is thought to assist individuals in the moderate-to-severe stages of AD (Woods, 1996).

Certain cognitive strategies attempt to change the level of functioning of a person with AD. The basic concept is to identify areas of functioning that are relatively unimpaired, and maximise their use, whilst putting minimal strain on the processes suffering greater impairment (Woods, 1996). One of these strategies specifically involves the reduction of cognitive load. Individuals with AD have an attention problem that is evident when they are required to do two or more tasks simultaneously, compared to control participants (Nebes & Brady, 1989). This reduced ability to attend to a primary task when there are other ‘items’ to be processed translates into real life difficulties. For example when an individual with AD is doing a primary task, any background activity (e.g., music playing, two people talking at the same time, or a lot of movement) may significantly reduce their performance of the primary task (Woods, 1996). To reduce the cognitive load on
memory it has been suggested that the environment be reorganised so that clutter is reduced, important items be placed in prominent places and items be labelled and signposted (Holden & Woods, 1995). In terms of communication, one-to-one conversations were found to be easier for AD individuals to understand than group conversations. Furthermore, the larger the group was, the harder the communication became for them (Alberoni, Baddeley, Della Sala, & Logie, 1992). Other concepts found to be useful in reducing cognitive load for communication included using short, simple sentences starting with the major point and cutting out unnecessary pronouns. In addition to this, closed questions were found be helpful to those who have difficulty generating sentences (expressive aphasia) (Hart & Semple, 1990). In the latter stages of the disease the use of pictures (as prompts or accessors of semantic knowledge), and non-verbal communication assist with communication, as these abilities are slower to decline in AD (Woods, 1996).

Tappen (1997) summarises some of the central concepts for successful memory or cognitive rehabilitation from the work of Backman (1992) and Arkin (1991). These were as follows:

1. Acknowledgement of the cognitive deficit.
2. Basing the intervention on relatively well preserved skills.
3. Enhancing the encoding and retrieval processes of memory with aids or prompts.
4. Selection of relevant tasks that are meaningful to the individual.
5. Involvement of the family or caregivers in any intervention when possible (Tappen, 1997).

Over the last 5 years attempts at specific AD memory rehabilitation have developed. These are summarised in a review by De Vreese, Neri, Fioravanit, Belloi and Zanetti (2001). These memory rehabilitation methods have focused on both preserved explicit memory in the early stages of AD, and preserved implicit memory (including procedural learning).

Despite extensive memory difficulties, it has been found that some early phase AD individuals have benefited from types of explicit memory training if extensive support is given at the time of acquisition and retrieval (De Vreese et al., 2001). Some examples of this are seen in several studies looking at remembering personal autobiographical memories (Arkin, 2000a; Arkin, 2000b; Arkin, 2001), face-name learning (Davis, Massman, & Doody, 2001), and remembering internally generated
words through repetition (although these were only remembered when the participants were not specifically trying to remember them) (Barrett, Crucian, Schwartz, & Heilman, 2000). However, as Davis et al (2001) commented, the improvements were small, and did not generalise to neuropsychological tests, functioning or quality of life measures.

It has been found that memory training for personal semantic facts and specific events, when combined with cholinesterase inhibitors, improves scores on ADAS cog more than cholinesterase inhibitors alone (De Vreese & Neri, 1999). Therefore memory training aimed at preserved explicit memory may be an effective adjunct to medication in the early stages of AD (De Vreese et al., 2001).

It appears that greater effects of memory training are found if the training utilises preserved implicit memory (De Vreese et al., 2001). De Vreese (2001) reviewed four methods: expanding rehearsal, vanishing cues, errorless learning and procedural learning (the latter will be reviewed in chapter 2).

The expanding rehearsal technique involves repeated recall of information with a gradual increase of the time interval; this technique relies on priming (De Vreese et al., 2001). It has shown effectiveness for learning face-name pairs (Camp & Foss, 1997), object location (Bird & Kinsella, 1996) and object naming (Camp, Bird, & Cherry, 2000). However, there is no evidence of spontaneous generalisation of this skill to non-target material (Camp et al., 2000).

The vanishing cues technique involves using letters of the target to prompt memory, letters are progressively added until the correct answer is achieved. The cue is then presented with one less letter after each correct response (De Vreese et al., 2001). This technique has been used for learning names, objects and addresses and the effects can last several weeks. However, the vanishing cues method in isolation is less useful for relearning face-name connections than simple repetition (De Vreese et al., 2001).

The errorless learning principle involves minimising errors during learning, as errors interfere with learning and are not distinguished from correct responses. It has been speculated that people with AD may learn this way through intact implicit learning (Wilson & Evans, 1996). Clare, Wilson, Carter, Breen, Gosses and Hodges (2000) demonstrated the usefulness of this technique for learning personally relevant information for 5 of 6 participants with mild AD; and this benefit lasted up to 6
months. However, in this study the errorless learning principle was applied via various methods including vanishing cues, expanded rehearsal, verbal elaboration, forward cueing, instructional audio-tapes and memory aids. The vanishing cues were found to be the least useful, and also difficult to use in an everyday setting. All of the methods used the errorless learning principle, however the results could not be directly attributed to any one method.

### 1.5.2.2 Reality Orientation Therapy

Reality orientation therapy (RO) is a method, which aims to orientate an individual to their environment by means of continuous stimulation. There are two main varieties of RO, which have been extensively explained and evaluated (Holden & Woods, 1995; Miller & Morris, 1993). The first of these is Classroom RO. This involves 3 to 5 AD participants, meeting for two sessions a week for at least half an hour. The group tries to learn each others’ names through the use of verbal prompts and name badges. The facilitator provides information on day, date, weather, and corrects incorrect information. During discussions with more mild participants, the group members talk about personal information and histories. Facilitators use newspapers, slides or films to attempt to orientate people to the present, and food prices to orientate participants to current costs of living (Holden & Woods, 1995).

The second type of RO is 24-hour RO. The purpose of this therapy is to communicate information to the person with AD throughout all their waking hours. This approach comprises of two elements: first, to change the environment by providing orientating information (e.g., weather boards, labels, signs, clocks, pictures). Second, to directly communicate with the individual in everyday conversation, in which they are reminded of their name, where they are, time, date, and current occurrences. Incorrect information is corrected (Hanley, 1984). More recently a more reactive method has been emphasised; in this method the staff only respond to requests from the individuals with AD, rather than providing them with unsolicited information (Reeve & Ivison, 1985; Woods, 1996).

A review of 21 RO studies was described by Holden et al (1995). The studies varied in terms of the type of RO evaluated, the setting, the measures used, the length of intervention and methodology. The conclusions, however, are consistent: they showed support for RO for increasing verbal orientation in the mild-to-moderate
stages of AD. Some studies suggest that other improvements in new learning ability may follow cognitive stimulation by RO (Breuil et al., 1994), however this remains controversial (Woods, 1996). Holden et al (1995) also mention that changes in functionality and behaviour are much more elusive and were not found to be changed by RO. One important example shown to be successful involved the use of signs for toilets, which helped participants to find the toilets without assistance (Holden & Woods, 1995). Other elements of RO, however, are less effective and sometimes harmful to the person with AD, especially when used insensitively (Dietch, Hewett, & Jones, 1989).

1.5.2.3 Validation Therapy

Validation therapy was developed as a countermeasure to the insensitive use of RO (Feil, 1993). For example, repeatedly informing an elderly individual with AD that her mother is dead when she asks where they are (this may cause repeated distress, as the knowledge is not retained). Another example would be confronting a person with the severity of their illness or condition on a daily basis (Miller & Morris, 1993). Validation therapy aims not to correct the incorrect information, but to ‘validate’ whatever it is that the person with AD is experiencing (Miller & Morris, 1993). For example, if a woman talked of an imminent visit from her mother (who was no longer alive), the response would involve empathy towards any feelings of loss and insecurity; rather than correcting the information about her mother (Miller & Morris, 1993). This method is very reliant on the therapist correctly identifying the AD person’s feelings. It is claimed that this therapy enhances communication with AD individuals (Feil, 1993), although the results are not consistent, as some individuals show improvement in communication and others show decline (Morton & Bleathman, 1991).

1.5.2.4 Reminiscent Therapy

In reminiscent therapy groups, memories from the past are evoked to increase positive discussions between group members (Woods, 1996). This type of therapy relies on preserved autobiographical memories from childhood and early adult life in people with AD (Miller & Morris, 1993). Photographs, music and cues from their families stimulate personal memories in the person with AD; they are then encouraged to talk about these memories to the group. This may be an enjoyable
activity for people with AD, and it increases their interaction within the group, but there is little evidence that cognitive function or functioning ability outside the group changes (Woods & McKeirnan, 1995). This approach is not appropriate if an individual’s memories are predominantly negative (Miller & Morris, 1993).

Interestingly in a comparison with RO, reminiscent therapy tended to increase life satisfaction of the individual with AD, whilst RO tended to decrease life satisfaction by confronting the AD individual with current unfavourable life situations (Baines, Saxby, & Ehlert, 1987).

1.5.2.5 Environmental Modification

A number of environmental factors influence people with AD, this is especially notable when there is a change in the environment (Tappen, 1997). It has been reported that the environment can be an important part of an intervention for AD, especially in institutions (Haitt, 1991). One example of an environmental modification technique that has been shown to be effective involves putting people’s names and large photos of them when they were in their early twenties on the doors of their rest-home rooms. After 3 days the participants were finding their rooms successfully (Nolan, Mathews, & Harrison, 2001).

Some of the elements of environmental modification include a ‘home-like’ setting, privacy, allowance of individual differences, tolerable amounts of stimulation (i.e., not to much noise or too many people), ordinary looking items, and safe environments (e.g., no sharp knives) (Tappen, 1997).

1.6 Summary

This chapter has reviewed the background of AD, by outlining prevalence, incidence, etiology, pathology, clinical presentation, symptomatology, disease course and functionality. This has provided an overview of the disease and the way it affects cognition, functioning and quality of life. In the last two decades there has been an increasing focus on approaches to improve quality of life for individuals with AD. Medications have been developed which attempt to improve or slow the decline of cognitive difficulties, behaviour problems, daily functional difficulties, and quality of life. In terms of non-pharmacological interventions several approaches have been tried; environmental modification appears useful, as does the sensitive use of RO and...
validation, along with some memory enhancing techniques. However, on the whole, attempts to assist memory have shown limited results. One exception to this finding occurs in the area of procedural learning, which is the focus of chapter 2.
2 Chapter Two

Theoretical Review of Procedural Learning and Alzheimer’s Disease

2.1 Introduction

In many laboratory-based assessments procedural learning has been shown to be well preserved in people with Alzheimer’s disease (AD) (Deweer et al., 1994; Dick, Kean, & Sands, 1988; Eslinger & Damasio, 1986; Gabrieli et al., 1994; Heindel, Salmon, Shults, Walicke, & Butters, 1989). Several of these researchers concluded that this preserved ability could be potentially used in the everyday lives of individuals with AD, but this has seldom been investigated further. As individuals progress into moderate stages of AD tasks that were once automatic, and performed without conscious thought, become difficult, frustrating, and often confusing. Errors begin to occur in all areas of functioning, from using soap instead of toothpaste to ripping the corner of a tea bag and pouring the tea leaves into a cup. This leads to the question posed in this thesis: Can preserved procedural learning, as demonstrated in laboratory-based studies, be used to retrain lost ADL abilities in those with AD? This research question raises two theoretical issues of importance. Firstly, can learning reinstate tasks that are no longer correctly stored in procedural memory? Secondly, if procedural learning is maintained in people with AD then why are these tasks lost from procedural memory in the first place?

2.2 Theoretical Review of Procedural Learning

Historically there have been many developments in theories of learning and memory, and these will continue to evolve. For almost two decades procedural learning, along with classical conditioning, priming, habituation, and adaptation effects, have together been viewed as a class of learning labelled ‘implicit’ (Roediger, 1990; Schacter, Chiu, & Ochsner, 1993). Graf and Schacter (1985) first used the terms implicit and explicit to distinguish between two forms of memory. The key distinction between these types of memory is the individual’s experience at the time of memory retrieval. More specifically, it is argued that explicit memory requires conscious recollection of previous experiences or information, whereas implicit
memory is revealed when previous experience facilitates performance on a task, without requiring conscious recollection (e.g., Graf & Schacter, 1985). All types of implicit learning can be performed without conscious awareness or memory of the learning, that is, the ‘learning’ effect can be demonstrated without the person recalling ever having previously performed the task (Squire, 1992). Prior to this, the term ‘procedural memory’ was defined as skills learning, and contrasted with ‘declarative memory’, as the two major memory systems (Winograd, 1975). However, this ‘procedural memory’ category did not incorporate other types of non-declarative memory (such as classical conditioning etc.), hence the origin of the term ‘non-declarative memory’. In a very comprehensive article Squire (1992a) described a model of memory comprised of two subsystems, namely declarative and non-declarative memory. Within non-declarative memory he defined four categories; skills, priming, dispositions and non-associative learning. Squire stated that non-declarative memory had a meaning similar to implicit memory (implicit memory and non-declarative memory have since been used interchangeably by researchers). Non-declarative memory is characterised by a change in skilled performance, which is difficult to describe in words. Squire comments, however, that it is difficult to know when declarative (explicit) learning is playing a role in the acquisition of non-declarative knowledge. Consequently, amnesia studies have commonly been used to investigate the differences between these memory systems (Squire).

The products of procedural learning (i.e. the acquisition of skills) are stored as procedural knowledge or memory, a type of non-declarative (or implicit) memory (Squire, 1992a). Procedural knowledge then enables skilled performance (Tulving, 1985; Tulving, 1987; Tulving & Donaldson, 1972). Squire suggested that procedural memory is more automatic and less conscious than explicit memory, and is not broken into short or long-term memory components (Squire, 1992a). Procedural learning is the acquisition of this knowledge via correct repetition rather than conscious attempts to remember. For example, learning that a certain condition or action should occur before the next action is performed (e.g., the clutch in a car needs to be pressed before the gear stick is moved) can become ‘automatic’ i.e., stored in procedural memory through the process of correct repetition. Additionally, with correct repetition a single action or movement can also be learnt (e.g., turning a door handle). When a correct action or series of actions (movements/thoughts/perceptions) is performed, the neural
representation of that action is strengthened through activation of the neural circuitry involved in procedural learning. As a task is successfully repeated that particular neural pathway is continually strengthened until the task can be successfully performed without prompts or instruction. Well practised habits, such as walking, dressing, and eating can be described as activities that are part of procedural knowledge. Other activities that involve procedural memory include writing, typing, solving rule-based puzzles or even riding a bicycle (Moscovitch, 1992).

Tulving (1985) suggested that procedural memory evolves as the earliest of all memory systems, developing as young as 3 months of age. This enables the development of skills which are acquired throughout the early developmental stages, such as grabbing objects and putting things into the mouth. Nelson (1995) also proposes that procedural memory in children appears in the first few months of life. This suggestion is supported by studies which examine infants’ abilities to form expectations about future events by recording eye movements as they fixate on lights that create certain patterns (Haith, Hazan, & Goodman, 1998). Infants as young as 3.5 months can learn the sequence of light, showing reduced reaction times from baseline. When the light patterns change the reaction times become slower. Interestingly, a similar study originally evaluated procedural learning in people with AD (Knopman & Nissen, 1987) and they also showed learning trends.

Digiulio, Seidenberg, O’Leary and Raz, (1994) argued that procedural memory is dissociated from declarative (explicit) memory, and is seen during child development. Based on the performance of different aged children on perceptual skills learning (Gollin figures and degraded word task) and explicit learning (word and figure recall) they reported that the level of implicit learning stabilises in childhood while explicit learning continues to improve.

Procedural learning is conceptualised, or subdivided, into a collection of motor, perceptual and cognitive skills that make up skilful behaviours or habits (Squire, 1992a). Hirono et al. (1997) investigated motor, perceptual and cognitive procedural learning in individuals with mild-to-moderate AD, using three tasks which isolated the three types of procedural learning. Acquisition of tactile Hiragana character reading (i.e., reading letters using fingers) was used to isolate perceptual learning, bi-manual motor co-ordinated tracing (i.e., tracing shapes using both hands) was used to isolate motor learning, and cognitive learning was isolated through
individuals’ repeated attempts to solve a puzzle. The AD group showed all three types of procedural learning.

Knopman (1991) posed the very important question, how long is procedurally learnt material maintained in the procedural memory system in AD? Knopman hypothesised that even when procedural learning is relatively intact in AD, the retention of the procedural knowledge itself could still be impaired. After some experimental work Knopman concluded that at least some AD participants (6 of 8) showed long term retention of implicitly acquired knowledge over 2 weeks. This article pointed out an important distinction; that procedural learning (the acquisition of new procedural knowledge) could be relatively normal, whilst the maintenance, or retention of that knowledge might be impaired.

2.3 Anatomical Structures Implicated in Procedural Learning

Neurological disorders involving degeneration of the striatum (e.g., Parkinson’s disease and Huntington’s disease) commonly result in deficits in procedural learning. In contrast, disorders with lesions in cortical structures typically leave procedural learning intact (Soliveri, Brown, Jahanshahi, & Marsden, 1992). These findings have led to theories that the areas affected in Parkinson’s disease and Huntington’s disease (e.g., the striatum) are critical for procedural learning. Lesion studies in animals and humans have suggested the striatum plays a primary role in procedural learning and the cerebellum is also involved. More recently imaging studies have added to this knowledge, confirming that the striatum and cerebellum are involved in procedural learning and also revealing frontal lobe involvement, specifically the supplementary motor cortex.

Comparison of cortical (e.g., AD) and subcortical dementias (e.g., PD) has added support to the neural distinction between memory systems (i.e., explicit and implicit). These studies reveal a double dissociation between Parkinson’s disease or Huntington’s disease individuals (who demonstrate normal explicit memory and impaired procedural learning) and AD individuals (who demonstrate the opposite association). Bondi and Kasznick (1991) compared an AD group and a PD group on
stem completion priming, rotary pursuit, the fragmented pictures test and several explicit memory tasks. The AD group were impaired on the explicit memory tasks, and stem completion, but intact on rotary pursuit and the skills learning component of the fragmented pictures test. In contrast, the PD individuals performed better on all explicit tests but performed poorly on the fragmented picture tests and rotary pursuit task. It is interesting to note that studies comparing AD to PD often exclude AD individuals when they have Parkinsonian features. It has been demonstrated that when Parkinsonian features are present in AD, the striatum is affected in a different way. In AD (with Parkinsonian features) there is a dopamine 2 receptor decline, rather than presynaptic nigrostriatal alterations that are present in Parkinson’s. However, the overall effect in both cases is a decrease in striatal dopamine levels (Pizzolato et al., 1996). Presumably procedural learning is affected by this decline in striatal dopamine levels in AD with PD features, although this has not been investigated.

Individuals with PD have impairments on the rotary pursuit task (e.g., Heindel et al., 1989) and the serial reaction time task (e.g., Ferraro, Balota, & Connor, 1993). Pascual-Leone (1993) reported that both the basal ganglia and the cerebellum are involved in procedural learning, but that the two structures have different functions. Comparing controls to those with cerebellum degeneration and those with PD, Pascual Leone concluded that the cerebellum indexed and ordered events and was therefore essential for functions involving sequences, while the basal ganglia was involved in the actual learning in a procedural manner. Jordan (1994) also concluded that the striatum is involved in procedural learning following research with PD participants, although acknowledges the difficulty in interpreting results because of extra-striate pathology and other non-procedural cognitive deficits in PD.

Hikosaka et al. (1996) suggest that the supplementary motor cortex is one of the key structures responsible for learned sequential movements, in combination with the dorsolateral frontal cortex, basal ganglia (specifically the striatum) and cerebellum. They studied these ideas using a sequential button press task with both monkeys and human participants. Once these were mastered the monkeys could perform tasks with a break of up to six months. They then blocked the neural function of different parts of the striatum by injecting muscimol (a gamma-aminobutyric acid (GABA) agonist). The results suggested that the anterior part of
the striatum (both caudate nucleus and putamen) was related to learning new sequences and the posterior part of the striatum related to execution of learned sequences. In the human participants they used MRI scans to test the same hypothesis. They compared “learn” to “watch” and found that the pre-supplementary motor area is related to learning of new sequences, not movements, while the supplementary motor area is related to performing these movements. The pre-supplementary motor area is thought to be linked to the anterior striatum in neural circuits and the posterior striatum to the supplementary motor area. The authors point out that the location of the procedural memories themselves is still unknown (Hikosaka et al., 1996). The results from other MRI scanning research with procedural tasks suggest the cortico-caudate system (the caudate nucleus being part of the striatum) underlies motor procedural learning (Granholm, Bartzokis, Asarnow, & Marder, 1993).

In support of the link between the supplementary motor area and procedural learning Ackerman, Daum, Schugens & Grodd (1996) reported impaired procedural learning (tested by serial reaction time and mirror reversed tracking tasks) for a patient with damage to the left supplementary motor area. They too suggested that supplementary motor area as well as the striatum and cerebellum are important for procedural learning.

The frontal cortex has been implicated in learning a visuomotor procedural skill (serial reaction time task) by impaired procedural learning of this task (for the hand contralateral to the foci) by adults and children with frontal epileptic foci. In contrast individuals with temporal epileptic foci had no effect on procedural learning (De Guise, Jambaque, Dulac, & Lassonde, 1999). The effect of frontal epileptic foci on procedural learning may be due to disruption of supplementary motor cortex functioning, as outlined above, or it may relate to disruption of two frontal-striatal circuits (as outlined below, section 2.3.1).

Although it is mentioned in several studies that intact cerebellar function is important for procedural learning, lesion studies show that participants with just cerebellum lesions have no impairment in procedural learning. However, participants with both cerebellar and brain stem lesions show impaired performance on learning procedural tasks (Daum et al., 1993). These results do not support theories of procedural learning being primarily dependent on cerebellar function. It has been
proposed that the cerebellum may play more of a role in conditioning (Gabrieli, 1998). An alternative hypothesis is that while the learning of repetitive motor sequences depends on the striatum, the learning of mapping between perceptual cues and motor responses (e.g., learning the relationship between the movement of a cursor on a computer screen and moving a joystick with your hand) is dependent upon the cerebellum (Willingham, Koroshetz, & Peterson, 1996).

In a review of memory and functional neuroimaging Gabrieli (1998) noted the importance of the basal ganglia and cerebellum in procedural motor skill learning, but also acknowledged the role of the neocortex. Learning of the rotary pursuit task and serial reaction time task increased activation of the primary and secondary motor cortex as well as the basal ganglia. It was also apparent that cerebellar activity decreases as the skill of a task increases for perceptual and motor tasks. Most imaging studies report complex patterns of activation during learning, with changes in activation patterns once learning has occurred, indicating that procedural learning involves complex interactive neural networks (Gabrieli, 1998).

Petersen, Van Mier, and Raichle (1998) used PET studies to identify the sequence of structures activated during motor procedural learning. In addition to the motor cortex, in the early stages of task performance the prefrontal cortex, parietal cortex, and the cerebellum show increased levels of activation. These areas are probably involved to ensure that the correct movements are assembled together (requiring attention, working memory and coordinated movements) (Squire & Kandel, 1999). Then, after practice, these three areas show less activation and the striatum, motor cortex and supplementary motor cortex show increased activation (Petersen et al.).

In summary, a variety of different studies have added to knowledge about the nature of procedural learning and the anatomical structures important for this type of learning. It appears that cognitive and motor learning are supported by the dorsal striatum and cerebellum, although perceptual procedural learning appears to be related both to the striatum and to the relevant sensory cortex (Gabrieli, 1998). The various types of learning probably reflect distinct striatal-thalamic-cortical loops with distinct cortical inputs, however, the loops are likely to be parallel and share similar neural properties (Alexander, DeLong, & Strick, 1986; Gabrieli, 1998).
2.3.1 The Striatum

The striatum is the cortical area which has been most consistently researched in relation to procedural learning. It is also considered to be the most essential anatomical component of the system underlying procedural learning. Given the significance of this neural area, this section includes a brief outline of the main striatal anatomy and functional circuitry. The striatum consists of three connected anatomical components; the putamen, the caudate and the ventral striatum (the nucleus accumbens) (Delong, 2000). The ventral striatum is ventral to the caudate nucleus and has connections to the limbic system (Delong, 2000). Both the caudate and putamen are formed from the same telecephalic structure and therefore have the same types of cells; they are also fused anteriorly (Delong, 2000). They are the input nuclei for the basal ganglia, meaning that most afferent connections terminate in the striatum (Delong, 2000).

The striatum is part of the basal ganglia, through which run five parallel feedback loops of the following general form: cortex-striatum-globus pallidus-thalamus-cortex (Cote & Crutcher, 1991; Delong, 2000). The skeleto-motor circuit begins and ends in the motor fields (premotor cortex, supplementary motor area and motor cortex). This circuit controls a range of movements and also has input from the somatosensory area. The oculo-motor circuit begins and ends in the frontal and supplementary eye fields and controls eye movements. The limbic circuit projects from/to the anterior cingulate area and medial orbitofrontal cortex and mediates the limbic system (primarily emotions and motivation). Finally there are two prefrontal circuits. The first is the lateral orbitofrontal circuit, which originates in lateral and orbitofrontal regions of the frontal lobes and also has temporal cortex input. This circuit is thought to be involved in the ability to change behavioural set. The second is the dorsolateral prefrontal circuit, which originates in the dorsolateral prefrontal cortex and also has parietal cortex input. This circuit is involved in aspects of memory concerned with spatial orientation (Delong, 2000). It has been proposed that the skeleto-motor circuit and the frontal circuits are commonly involved in procedural learning (Schmidtke, Handschu, & Vollmer, 1996). Support for this comes from PET studies which show an increase in metabolic activity in the prefrontal associative cortices during procedural tasks (Perani et al., 1993), and from frontal epilepsy.
studies which show contralateral impairment in procedural learning with frontal epileptic foci (De Guise et al., 1999).

Each of the five circuits engage different portions of the striatum. The association areas project to the caudate and rostral putamen; sensorimotor areas project to most of the central and caudal putamen; and limbic areas project to the ventral striatum (Delong, 2000). Studies involving acid-induced local lesions in rats suggest that the ventral striatum (nucleus accumbens) is not involved in procedural learning (as the caudate and putamen was found to be) but does have a role in attention and flexibility for changing set (Reading, Dunnett, & Robbins, 1991).

In summary, the five main circuits receive input to the striatum from the motor, sensory, and association corticies (to the dorsal striatum) and limbic system (to the ventral striatum) (Cote & Crutcher, 1991). Thus the functions of the striatum are not direct but are mediated by the cortex (Alexander et al., 1986; Delong & Georgopoulos, 1981).

### 2.4 Implicit Learning and AD

Several studies have shown that individuals with AD fail to benefit from training that relies on explicit memory to improve cognitive ability (Deweer et al., 1994), although some temporary benefit has been found with simple tasks such as learning specific face-name pairs in mild AD (De Vreese et al., 2001). In contrast preserved implicit memory has been seen in AD with intact classical conditioning and skills learning. Repetition priming has shown conflicting results with some researchers reporting preserved ability (Schacter, 1987) and others reporting impairment (Gabrieli et al., 1994). Most research demonstrating intact implicit memory, however, has focused on two areas: perceptual priming and procedural learning (De Vreese et al., 2001). Note that perceptual priming can occur with a single exposure to a stimulus, while the development of procedural knowledge of a skill is incremental with practice (Soliveri et al., 1992). Mild-to-moderate AD participants show normal magnitudes of short and long term perceptual priming (Fleischman & Gabrieli, 1998) for such things as single words (Keane, Gabrieli, Fennema, Growdon, & Corkin, 1991), dot patterns (Postle, Corkin, & Growdon, 1996), and objects (Fleischman, Gabrieli, Reminger, Vaidya, & Bennett, 1998). This may reflect the lack of AD-related degeneration to primary motor, somatosensory,
auditory and visual cortical areas (Gabrieli et al., 1994; Keane, Gabrieli, Growdon, & Corkin, 1994).

Individuals with mild AD, whose pathology primarily affects association areas in the neocortex rather than the basal ganglia, perform well on general skill acquisition through procedural learning (e.g., the rotary pursuit task), even though their performance on comparable explicit tests is severely impaired (Huberman, Moscovitch, & Freedman, 1994).

Experimental studies of procedural learning have focused on all three types: motor, perceptual and cognitive skills. Examples of the successful training of motor learning in individuals with AD include the finger maze (Grosse & Wilson, 1991; Kuzis et al., 1999), mirror tracing (Deweer et al., 1994; Gabrieli, Corkin, Mickel, & Growdon, 1993) and rotary pursuit (Deweer et al., 1994; Dick, Nielson, Beth, Shankle, & Cotman, 1995; Eslinger & Damasio, 1986; Heindel et al., 1989). Successful cognitive skill learning has included puzzle assembly (Hirono et al., 1997; Poe & Seifert, 1997), and repeated performances of the digit symbol test (Carlesimo et al., 1998). Successfully learnt perceptual skills include visual serial rotation (Knopman & Nissen, 1987), mirror reading (Deweer, Pillon, Michon, & Dubois, 1993), tactile recognition (Hirono et al., 1997) and perceptual verbal skills with verbal material (Cohen & Squire, 1980; Martone, Butters, Payne, Becker, & Sax, 1984).

Deweer et al. (1994) designed a study to test the different aspects of memory functions in mild AD participants (learning a list of words (explicit memory), the rotary pursuit task, a priming task, and a perceptual skill task with verbal material). The AD participants exhibited normal procedural learning for the rotary pursuit task, learning at the same rate as the elderly controls. They were also able to retain what they had learned over a 4 week period. For the implicit part of lexical priming the participants were as able as the controls to produce real words when provided with the first three letters but showed severe deficits on the explicit learning part of the study. Deweer et al. argued that, in people with AD, the successful procedural learning performance reflected the integrity of the basal ganglia, particularly the caudate nucleus.

Gabrieli, Keane, Stanger, Kjelgaard, Corkin & Growdon (1994) reported that people with AD have preserved skill learning on rotary-pursuit, mirror tracing and serial reaction time tasks but impaired repetition priming on word completion and
word association tasks. They noted that the most consistent sites of dense neuropathology in people with AD are the limbic and new cortical association areas, which support repetition priming, word completion and word association. This explains why there are impairments on these types of implicit learning tasks whereas perceptual priming and procedural learning remain relatively intact.

Dick (1995) investigated the duration of procedural learning effects for the rotory pursuit task with 12 moderate-to-severe AD participants. They had 40, 80 or 120 trials of training (40 trials per day), followed by a 15-trial retention test at 20 minutes, 2 days, seven days and 37 days after training. Training resulted in improved performance for both groups in the first 40 trials but additional training had no effect. The AD participants showed minimal forgetting across the four retention tests demonstrating that they can effectively learn and retain a motor skill for at least 37 days.

Willingham, Peterson, Manning, and Brahear (1997) reported that dementia ratings predicted the ability to perform a variety of laboratory-based procedural learning tasks, but not the ability to learn those same tasks. Therefore, they suggest it is acceptable for researchers to claim AD patients can learn a motor skill even if other participants of the same dementia severity can not perform the task (i.e., lack of ability to perform a task does not mean that the task is not able to be learned). They also reported that an ability to learn one task did not predict the ability to perform another task, showing that some tasks can be learnt by some AD individuals whilst others cannot. Unfortunately, no explanations for these diverse patterns were provided.

AD individuals in the later stages of the disease who are more severely affected by the disease process may show learning deficits on all tasks, as it is assumed that as AD progresses procedural learning will eventually be impaired (Corkin, 1982). However, at least in the early and middle stages, the retained procedural learning ability provides a potential means by which some aspects of impaired functional skills in individuals with AD might be relearned.
2.5 How are the Structures Implicated in Procedural Learning Affected in AD?

As already noted, the three subtypes of procedural learning appear relatively preserved in AD. How are the neural structures implicated in procedural learning (primarily the supplementary motor cortex, striatum and cerebellum) affected by AD pathology? How consistent are the pathology findings and the results on laboratory-based procedural learning tasks?

AD pathology typically begins in the medial temporal structures, then progresses throughout the association cortices, leaving the areas of primary cortex (motor and sensory, including the supplementary motor cortex) relatively unaffected (Almkvist, 1996), as well as those implicated in procedural learning, specifically the striatum and cerebellum. Despite these general findings, several studies outlined below show that neurochemical and structural changes do occur in the striatum and cerebellum, although these changes are different (in nature and degree of change) from those in the cortex.

It appears that large neurons in the striatum are affected by AD pathology and are often damaged (Oyanagi, Takahashi, Wakabayashi, & Ikuta, 1987), or contain NFTs (Braak & Braak, 1990; Cullen & Halliday, 1998). Small and medium sized neurons, however, are hardly affected (Braak & Braak, 1990; Oyanagi et al., 1987). In addition to this, cerebral blood flow to the basal ganglia remains good throughout the course of AD, whereas hippocampal and some cortical areas have reduced flow (Okuda, Tachibana, Kawabata, Takeda, & Sugita, 2001).

The Neuritic plaques (or senile plaques), which are common in the cortex of AD individuals, are rare in the striatum, although some increases in diffuse amyloid deposits or plaques are seen (Braak & Braak, 1990; Brilliant, Elble, Ghobrial, & Struble, 1997; Gearing et al., 1993; Love, Wilcock, & Matthews, 1996). In addition, there are fewer NFTs in the striatum than in the cortex (Love et al., 1996), although it has been reported that the presence of diffuse plaques and increased levels of lysosomes in the striatum indicate the beginning signs of metabolic dysfunction (Cataldo, Barnett, Mann, & Nixon, 1996). However, electron microscopy of the diffuse plaques of the dorsal striatum found only rare degenerating neurites and no apparent fibrillar amyloid. In addition, the amyloid protein precursor in the cortex
and the striatal tissues appears to be processed differently (Gearing et al., 1993). These findings imply that while there are increases in diffuse plaques in the striatum, they are not equivalent to those in the cortex, as they do not seem to be as associated with neuronal degeneration or tangles.

The ventral and dorsal striatum appear to be affected differently by the AD process. The ventral striatum (nucleus accumbens), which is closely affiliated with the limbic system, is frequently affected by amyloid deposits with dystrophic neurites, suggesting that the ventral striatum is more vulnerable to AD than the dorsal striatum (where most of the diffuse plaques did not have dystrophic neurites) (Suenaga, Hirano, Llena, Yen, & Dickson, 1990). The dorsal striatum has less tangles and only some diffuse plaques when compared to the ventral striatum (Mak, Yang, Vinters, Frautschy, & Cole, 1994; Selden, Mesulam, & Geula, 1994b) and it appears that the latter is more affected by the decline of the acetylcholine system (Kasa, Rakonczay, & Gulya, 1997). Overall, the ventral striatum is more vulnerable to plaques and tangles (Selden et al., 1994b). Given the findings of the lesion study with rats (Reading et al., 1991), and the fact that procedural learning is preserved in laboratory-based tasks in AD, it is likely that ventral striatum integrity is not essential for procedural learning. In contrast, the intact dorsal striatum is likely to relate to the preserved procedural learning in AD.

In the cholinergic system the density of M2 muscarinic and nicotinic sites are reduced in the cortex and hippocampus, but this is generally not found in subcortical areas such as the striatum (Araujo, Lapchak, Robitaille, Gauthier, & Quirion, 1988). The size of cholinergic neurons in the dorsal striatum show no difference between AD individuals and controls (Geula, Tokuno, Hersh, & Mesulam, 1990). However, there are several changes in the neurochemistry in the striatum in AD. The ventral striatum loses 75% of its choline acetyltransferase (at postmortem) and if cholinergic neurons are not lost then they are smaller (Selden, Geula, Hersh, & Mesulam, 1994a), which is consistent with limbic psychopathology (Selden et al., 1994a). In contrast, the dorsal striatum is no different from controls in terms of choline acetyltransferase (Selden et al., 1994a), although another study did find some dorsal striatum reduction of choline acetyltransferase activity (Boissiere, Faucheux, Ruberg, Agid, & Hirsch, 1997).

In terms of the dopamine system, Kemppainen (2001) found that striatal dopamine uptake was reduced by 30% in AD, however Pizzolato (1996) found no
change in the binding of dopamine to the uptake site in the caudate (whereas in Parkinson’s it was reduced by 75%), and even a 13% increase of dopamine 3 receptors in the dorsal stratum (compared to a 13% reduction in PD). There appears to be a reduction of the levels of nerve growth factor in both the dorsal and ventral striatum in AD (Boissiere et al., 1997).

Although the striatum may be relatively spared in AD, some of the areas of projection (e.g., the posterior parietal cortex) may be damaged as the disease progresses to the later stages, and therefore interfere with procedural learning (Willingham et al., 1997).

The cerebellum does not appear to be the central element in procedural learning (Gabrieli, 1998). Nevertheless it has been implicated as part of the system for procedural learning due to its role in sequencing (Hikosaka et al., 1996). The cerebellum shows a number of pathological changes in AD including deposits of diffuse amyloid (although not in the same numbers as other areas of the brain)(Cataldo et al., 1996). However, Tau proteins and neurofibrillary tangles have not been seen (Larner, 1997). It has been hypothesised that the pathological processes of AD do not commence in the cerebellum until later in the course of the disease (Larner, 1997). This would imply that during the earlier stages of the disease the cerebellum is relatively spared from the degenerative processes of AD.

Given the pathology findings (both in terms of structure and neurochemistry) it appears that the striatum (with the exception of the ventral striatum) and the cerebellum are relatively preserved in AD. This is consistent with the laboratory findings of relatively preserved procedural learning ability in AD, and current theories about the neurological underpinnings of procedural learning.

2.6 Self-care Tasks and Daily Living Skills in AD:

Why does the Toast go in the Teapot?

Why is procedural learning preserved whilst procedural knowledge is lost? Essentially, they are different processes. Learning is mediated by the striatum, and knowledge is probably stored in a distributed manner similar to other types of knowledge. In the explicit memory system, certain types of memories are stored in specific areas in the cerebral cortex (Squire, 1999a). For example, damage to the left
temporo-parietal region (in right-handed people) can cause selective losses of category-specific knowledge, i.e., a person may not be able to identify small inanimate objects but retain knowledge about living things (Squire). It is thought that for explicit memory, the memories are stored in cortical areas close to where the information (in the memory) was originally processed (Squire). In other words, the particular sensory and motor systems used to learn information influence where that information is stored. For example, information learned through visual processing is stored within the inferior temporal lobe while information about inanimate objects involving manual interactions are located within the parietal lobe etc (Squire). If this is also the case for procedural learning, areas near the motor cortex may be crucial. Although no specific location for procedural knowledge has been located (Hikosaka et al., 1996), an MRI study has found increased motor cortex activation after prolonged exposure to a motor task. This change persisted for many weeks, along with the improvement in the task (Karni, Meyer, Jezzard, & Adams, 1995). Squire (1999b) hypothesised that although the location of the ‘memory trace’ of procedurally-learned skills is not precisely known, it probably occurs within the areas of motor or sensory cortex engaged during the performance of the skill. Furthermore, he proposes two alternative locations for that storage. The first possibility was that memory storage occurs in the areas engaged during practice, or secondly, that the synaptic changes occur in the connections from the cortex to the putamen and caudate nucleus.

Figure 3 shows a schematic representation of how the explicit memories are lost, and not replaced, in moderate AD. In contrast, procedural knowledge is lost, however, new learning can still occur to regain some of that lost knowledge. Procedural learning does not appear to be dependent on the explicit memory system. In AD the structures responsible for procedural learning (mainly the striatum, the cerebellum and supplementary motor cortex) are relatively preserved. Even though procedural knowledge is lost in the moderate stages of the disease, that knowledge can be relearned. If tasks are relearned, however, it is likely that these will again be lost from procedural knowledge over time due to AD pathology, although the durability of newly learned procedures is poorly understood.
A person without AD has normal procedural and explicit memory and learning systems. A large number of memories go in, most are stored, and some are lost.

However, as a person progresses through AD, both memory systems are affected by neural degeneration, although in different ways. The figures below show a representation of both explicit and procedural learning and memory, as it may be when a person has moderate AD. As procedural learning can continue to occur in mild-to-moderate AD, the amount of procedural knowledge maintained in procedural memory has the potential to be greater than that in explicit memory.

Figure 1. Procedural memory and learning, in contrast to explicit memory and learning in AD.
There are several explanations for why errors occur in everyday tasks involving procedural knowledge during the course of AD. The first is simply that the disease process may disrupt the memory/knowledge itself. The second relates to errors which may occur in task performance. It is likely that healthy individuals occasionally make errors, but other methods and abilities are used to correct these errors, such as problem solving and explicit memory. In the AD individual, however, these errors may go uncorrected, the procedural memory is then not strengthened, and the tasks are then increasingly performed incorrectly. If this continues, in the manner of procedural learning, the task could be learnt incorrectly. In other words, the repeated errors of incorrect performance might ‘over-ride’ or ‘over-write’ previously correct memory/knowledge. In addition to the disruption of well-learned procedures, many self-care tasks may also have elements of explicit memory use, and planning (both of which are impaired as AD progresses).

Despite the reasons for the loss of procedural knowledge, it remains clear that procedural learning is relatively preserved. The implications for this are that procedural learning may be able to be used to retrain useful tasks for people with AD, such as daily living skills. There may be difficulties that emerge, however, when trying to transfer from laboratory-based tasks to daily living tasks. Firstly, the environment in laboratory-based tasks is very consistent and controlled, and the tasks are usually very simple, with limited elements. In contrast to this, tasks of everyday living can be very complex. Tasks often involve elements other than the motor ability such as motivation, specific environmental factors, and various tools or objects.

This leads to two questions of interest. Can self-care skills and activities of daily living be re-taught? And, if so, how long will the training last? Three small studies have begun to address these questions (Josephsson, Baeckman, Borell, Nygard, & Bernspang, 1995; Zanetti et al., 1997; Zanetti et al., 2001); these will be reviewed in the introduction of chapter 3.

When developing a training programme to answer these two research questions it is essential that the method of training follows the requirements of procedural learning, and that tasks are chosen for training that can, in fact, be trained using this method. A task must be able to be taught in a procedural manner, i.e. not requiring any explicit memory. This method would require the correct repetition of tasks. Some other aspects of the training that may be important for the training to be
successful may include: task simplicity; that they are performed exactly the same way each time; and that no decision making is required at any stage (other than the decision to do the task). If a task contains planning elements it would not be purely procedural in nature.

The research in this thesis will examine how well AD individuals improve their self-care/daily activity skills after procedural retraining, and determine the duration of training effects. If results indicate learning is successful and the effects last for a period of time after training, then this may form the basis for the development of a procedural self-care skills training programme to improve the quality of life for people with AD.
3 Chapter Three

Procedural Learning of Daily Tasks in Alzheimer’s Disease: Study One

3.1 Introduction

Alzheimer’s disease (AD) is a progressive neurodegenerative disease, which typically involves a loss of the ability to form new explicit memories (Butters et al., 1995), visuospatial deficits (Caramelli et al., 1998; Franceschi et al., 1995; Price et al., 1993; Thal, 1998), difficulties with semantic memory (Bentham, Jones, & Hodges, 1997; Daum, Riesch, Sartori, & Birbaumer, 1996; Graham, Simons, Pratt, Patterson, & Hodges, 2000; Tippett, Grossman, & Farah, 1996), and word-finding difficulties (Appell et al., 1982; Bayles, Salmon, Tomoeda, & Jacobs, 1989). In addition to this, there are often problems with other aspects of language (Butters et al., 1995), impairments in frontal lobe functioning (Binetti, Magni, Padovani, & Cappa, 1996; Lamar et al., 1997; Starkstein, Vazquez, Petracca, & Sabe, 1997), and an increasing retrograde episodic amnesia (Beatty, Salmon, Butters, Heindel, & Granholm, 1988; Kopelman, 1989; Nebes, 1992; Price, 2000). These cognitive deficits, in turn, underlie a progressive decline of functional ability in many day-to-day tasks (Reisberg, Ferris, Borenstein, Franssen, & Sinaiko, 1990; Teri, Larson, & Reifler, 1988). Decline of functional ability can severely reduce the quality of life both for those with AD and for their families.

One of the most debilitating of the wide ranging deficits accompanying AD is the profound dysfunction in aspects of memory and learning. There are some circumstances however, in which individuals with AD may be capable of new learning. Laboratory-based findings suggest that procedural learning (a type of implicit learning) is relatively well maintained in the mild-to-moderate stages of AD (Deweer et al., 1994; Dick et al., 1988; Eslinger & Damasio, 1986; Gabrieli et al., 1994; Heindel et al., 1989). Retained procedural learning ability provides a potential means by which some aspects of impaired functional skills in individuals with AD might be re-learned.

Procedural learning is a type of implicit learning that does not require explicit memory for the learning or recall of information (Mayes, 1988; Squire, 1992a). It is
conceptualized, or subdivided, into three potentially overlapping domains: motor, perceptual and cognitive. Procedural learning in these three domains, either singly or in combination, can produce skillful behaviours such as those that make up habits of everyday life (Hirono et al., 1997; Squire, 1992a). For example, consider the components of putting on a seat belt. This task involves motor and perceptual procedural learning. After repetition, the feel of the buckle is learnt through perceptual procedural learning, which may then help with holding the correct part of the buckle in the correct orientation. The motor skill component might include automatically reaching back over one shoulder and pulling the belt around the body, then pushing down firmly until the belt clicks into the catch. Procedurally-learned skills are generally performed without conscious recollection of when, or how, they were learned. These skills are also performed without information about how to execute the procedures involved (Squire, 1992a). Different neural circuits appear to underlie procedural learning and explicit learning (Hirono et al., 1997; Paulsen, Butters, Salmon, & Heindel, 1993; Postle et al., 1996; Squire, 1992a; Squire, 1993; Squire, 1999a; Squire, 1999b). Advocates of models which differentiate explicit and implicit memory systems postulate a neural circuit involving the cortical-striatal system for the acquisition and performance of motor procedural skills, and circuits including the medial temporal lobe, diencephalic structures and frontal regions for explicit memory (Mayes, 1988; Squire, 1992a; Squire & Kandel, 1999; Tulving, 1985). These distinctions are supported by research focused on neurodegenerative conditions that have relative sparing of the striatum (such as AD), and also neurodegenerative conditions that do involve the striatum (such as Parkinson’s disease). AD individuals have relatively intact skill-learning and memory for procedurally-learned skills regardless of whether they have conscious recall of previous performance of the skills (Hirono et al., 1997). Similarly, HM (who underwent bilateral removal of the medial temporal structures including the hippocampus due to epilepsy) and other amnesic patients with lesions in the medial temporal circuit have demonstrated preserved procedural learning (e.g., Cohen & Corkin, 1981; Corkin, 1968). This contrasts with impaired procedural learning in Parkinson’s Disease (Deweer & Ergis, 1998; Haaland, Harrington, O’Brien, & Hermanowicz, 1997; Roncacci, Troisi, Carlesimo, & Nocentini, 1996) as discussed in chapter 2.
There is considerable evidence that procedural learning is relatively well preserved in individuals with mild-to-moderate AD. Performance on a range of tasks shows improvement with training. For example, AD individuals show normal learning on the rotor pursuit task (Eslinger & Damasio, 1986), a serial rotation task (Knopman, 1991; Knopman & Nissen, 1987) and a transformed text task (Moscovitch, Winocur, & McLachlan, 1986). Hirono et al. (1997) investigated motor, perceptual and cognitive procedural learning in individuals with mild-to-moderate AD using three tasks which isolated the three types of procedural learning. Acquisition of tactile Hiragana character reading (i.e., reading letters using fingers) was used to isolate perceptual learning, bi-manual motor co-ordinated tracing (i.e., tracing shapes using both hands) was used to isolate motor learning, and cognitive learning was isolated through individuals’ repeated attempts to solve a puzzle. The AD group showed all three types of procedural learning although not to the same level as the control participants. Deweer et al. (1994) tested a number of different aspects of memory functioning in an AD sample: explicit memory (tested with the Wechsler Memory Scale - Revised and the California Verbal Learning Test), motor-skill learning (tested with a rotor-pursuit task), and perceptual skill learning (tested by a task involving reading words aloud from a mirror). While none of the participants had explicit recall of the initial learning episode on the rotor pursuit task, they all demonstrated long-term retention of the task after 4 weeks without any evidence of procedural ‘forgetting’. In other words, 4 weeks after training they performed at the same level on the rotor pursuit task despite not remembering ever having performed the task. Thus the AD participants in this study displayed motor skill learning (although not at the same level as the control participants) but had severe deficits in explicit memory for the training itself. They also demonstrated perceptual procedural learning on the mirror reading task but did poorly on the explicit memory tests. Gabrieli, Keane, Stanger, Kjelgaard, Corkin & Growdon (1994) found that AD participants had preserved skill learning on rotary-pursuit, mirror tracing and serial reaction time tasks but impaired repetition priming on word completion and word association tasks. These results once again indicate relatively preserved procedural memory in AD, but not other types of implicit memory such as priming, with the exception of perceptual priming (Gabrieli, 1998).
In summary, any activity that requires explicit memory (recognition or recall) is typically very poorly learnt by someone with AD (Butters et al., 1995). Healthy adults without memory difficulties can learn from things they are told or shown (in addition to procedural learning), as they are able to use explicit memory to remember what they have learnt. The learning can then be put into practice (Craik & Lockhart, 1972). A person with AD, however, can no longer rely on this method to learn new facts or tasks. Not surprisingly, several studies have shown that AD individuals fail to benefit from training that relies on explicit memory to improve performance (Backman, 1992; Backman, 1996; Breuil et al., 1994; Dick & Kean, 1989; Martin, Brouwers, Cox, & Fedio, 1985), including attempts to train functional skills directly using explicit methods (Breuil et al., 1994; Dick & Kean, 1989). Overall these findings indicate that a more logical approach to remedial training of individuals with AD would incorporate procedural learning, a type of memory that appears to be relatively intact in this group.

Findings from three preliminary studies, which used procedural learning methods to retrain functional skills, have demonstrated the potential of this approach. A small study using procedural learning for retraining a single simple task found intervention-related gains for the 3 AD participants but no gains for the participant with vascular dementia (Josephsson et al., 1995). Unfortunately the conclusions in this study were limited as only one task per participant was trained using procedural methods. The researchers concluded that further research should be conducted in the use of procedural learning. They suggested that any such research should take into account each individuals’ established habits, and noted that participants need to be motivated to perform the tasks in order for the training methods to be useful without prompting.

Zanetti et al. (1997) investigated whether a procedural learning training programme would improve performance on daily living skills in individuals with AD, targeting 20 self-care tasks. Each participant received three weeks of procedural training on half of these tasks, while the other 10 tasks remained untrained as a control measure. Baseline level of performance for all tasks was assessed for each participant at a treatment clinic, with independent raters used for training and testing. The dependent variable was the time taken to perform the tasks (error rates were not reported). AD participants showed a decrease in the time taken to perform the trained
tasks after training. The untrained tasks also showed a significant improvement, though of smaller magnitude. No direct comparison was made between performance on the trained and untrained tasks. Recently Zanetti and colleagues published a similar study with a single point baseline, but this time including a single point follow-up measure at 4 months post-training (Zanetti et al., 2001). The results suggested some durability of the training effects in comparison with a wait-list control group that showed no change. There are some limitations associated with this study. It was unclear whether the one-hour training periods enabled repetition of tasks during training. Additionally, there was a comparison of only two time points (baseline and 4-month follow-up) and once again they assessed only time to perform tasks (combined across 13 tasks) with no statistical report on task accuracy for the groups. The group design also meant that it was not possible to determine how many individuals showed significant benefits from the training or on how many tasks. Furthermore, during these investigations measurements were not conducted at the participants’ homes therefore the researchers could not assess if the skills learnt at the hospital generalised to the home settings. Although there are limitations associated with all three of these studies their results suggest that the development of training programmes for self-care skills to improve the quality of life for people with AD may be viable.

The application of procedural learning to retraining of daily living skills in individuals with AD is explored further in the present study. Tasks related to daily functioning such as self-care tasks and activities of daily living (which typically become severely disrupted during the progression of AD) were selected. A training system based on principles of procedural learning was conducted in participants’ homes, both because of the ecological validity of the setting and also because it removed the need to generalise learning across settings. Follow-up measures over a 12 week time period were conducted to assess the duration of the effects of procedural training. A key feature of this study is the use of both a single subject and group design, which enables the identification of individual gains as well as allowing the observation and analysis of group trends. A multiple baseline design across tasks is used, which enables assessment of the direct effect of the intervention (i.e., the procedural learning paradigm) for each individual subject, by looking at the co-occurrence of behavioural changes and training-onset for each task. The staggered
timing of the intervention across tasks (i.e., the multiple baseline) makes it possible to eliminate the influence of ‘extraneous’ factors, which may otherwise be responsible for observed changes in the individual at the time the intervention is introduced (Barlow & Hersen, 1984).

In summary, the aim of this study is to investigate the effectiveness of using procedural methods to retrain performance on impaired tasks in individuals with mild-to-moderate AD, and also to determine the duration of the training effects over time. It was predicted that tasks subjected to procedural training would show statistical improvements (as measured by the time taken to perform the task and the number of errors made), whereas untrained tasks would not. In addition we predict that these improvements for the trained tasks would be maintained following the completion of training.

### 3.2 Methods

#### 3.2.1 Participants

##### 3.2.1.1 Participants with Alzheimer's Disease

Eight individuals (seven female and one male) between the ages of 61 and 87 (M = 76.75, SD = 8.46) participated in this study. They were recruited from the Memory Clinic of North Shore Hospital, Auckland, New Zealand. All AD group participants had a diagnosis of probable AD according to NINCDS-ADRDA criteria (McKhann et al., 1984). They had all received this diagnosis following a full medical and clinical history (refer Appendix VII), and completion of a neurological examination, standard blood and urine analysis, and CT scans to exclude other dementias.

Participants were required to be in the mild-to-moderate stages of AD as assessed by scores on the Mini Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975). The lower cut-off used for moderate impairment in this study was 10 out of 30, as a score below 10 is considered to be in the severe range of AD (Reisberg, 1984). The mean score on the MMSE for AD participants was 17.75 (SD = 5.50), with a range from 10 to 25. Potential participants were excluded if they had a history of substance abuse, head injury, major cardiovascular accident,
psychiatric or other neurological diseases, or if they were taking neuroleptic medications, as all of these events could potentially cause neurological impairment instead of, or in addition to, AD. They were also excluded if they had significant immobility, hearing loss or sight loss, due to the difficulties these would create during training. Potential participants were also excluded if they had comprehension difficulties (a score of less than 28/36 on the Short Token Test (De Renzi & Faglioni, 1978)) and if they were taking any medications for the treatment of AD (e.g. Tacrine or Aricept). This was due to the potential for these medications to create improvements, which could not be distinguished from those resulting from training. All participants were required to be living at home, since living in a residential care facility setting may limit the daily performance of self-care tasks. One participant lived alone (this participant had an MMSE score of 25), the other seven lived with either a spouse or other family. All 8 participants identified themselves as Pakeha/New Zealand European, although participants from any ethnic group could have participated.

Twenty individuals with AD were invited to participate in the study. Nine individuals or their families declined, one individual failed the screening requirements and two individuals withdrew from the study for personal reasons before completion of the training phase.

3.2.1.2 Control Participants

Eight control participants were approached from a list of older people who had previously participated in research projects and had consented to their involvement in further studies. These control participants were matched pair-wise with the AD individuals for age (M = 75.63, SD = 8.42), ethnicity (all New Zealand European) and gender (one male, seven female), and as closely as possible for educational level. The same exclusion and inclusion criteria used for the AD participants were applied to the healthy elderly control participants, with the important addition that the control participants were to have an MMSE score of 26 or above, and have no history of memory problems. The MMSE scores of the control participants ranged from 27 to 30, which is within the normal range for healthy elderly (Bleecker, Bolla-Wilson, Kawas, & Agnew, 1988).
Demographic characteristics of both groups are presented in Table 2. As described above the two groups were identical for composition of gender and ethnicity. There was no significant difference between the two groups for age ($t(14) = .267, p = .794$), number of years education ($t(14) = 0.588, p = .588$), or estimates of pre-morbid intelligence ($t(14) = 0.407, p = .690$).

### Table 1
**Demographic Characteristics of AD and Control Participants**

<table>
<thead>
<tr>
<th>Characteristic or test name</th>
<th>AD group (n=8)</th>
<th>Control group (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (M/F)</td>
<td>1/7</td>
<td>1/7</td>
</tr>
<tr>
<td>Mean age (SD)</td>
<td>76.75 (8.46)</td>
<td>75.63 (8.42)</td>
</tr>
<tr>
<td>Mean years of education (SD)</td>
<td>12.13 (4.05)</td>
<td>13.13 (2.59)</td>
</tr>
<tr>
<td>Mean years since first symptom reported (SD)</td>
<td>4.69 (1.85)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Mean MMSE score (SD)</td>
<td>17.75 (5.50)</td>
<td>28.25 (1.28)</td>
</tr>
<tr>
<td></td>
<td>range: 10-25</td>
<td>range: 27-30</td>
</tr>
<tr>
<td>Mean Nart-R Full Scale IQ (SD)</td>
<td>104.69 (12.43)</td>
<td>112.2 (10.51)</td>
</tr>
</tbody>
</table>

### 3.2.2 Materials, Apparatus and Measures

#### 3.2.2.1 Screening Tasks

##### 3.2.2.1.1 Explicit Memory Tasks

Two tests were used to assess explicit learning. The Verbal Paired Associates sub-test of the Wechsler Memory Scale-Revised (WMS-R) (Wechsler, 1987) was used to assess verbal learning ability. In this test the participant is required to learn eight pairs of words over three trials. Half of the word pairs have a logical semantic connection (e.g., Fruit – Apple) and the other half have no such connection (e.g., Cabbage – Pen). The Visual Paired Associates sub-test of the WMS-R (Wechsler) was used to assess visual learning. In this test the participant must learn six pairs of unrelated colours and nonsense designs over three trials.

##### 3.2.2.1.2 New Adult Reading Test – Revised (NART-R)

The NART-R (Blair & Spreen, 1989), based on the NART (Nelson & O’Connell, 1978), was used as an estimate of premorbid intelligence. This test, which involves reading aloud 60 irregular words, has been shown to predict 72% of the
variance in the WAIS-R Verbal IQ section (Crawford, Stewart, Cochrane, & Parker, 1989). This test is correlated positively with education and negatively with socio-economic class (Crawford, 1992).

3.2.2.1.3 Short Token Test

The Short Token Test (De Renzi & Faglioni, 1978) was used to assess language comprehension (refer Appendix VIII). A total of thirty-six instructions are read out loud, one at a time to the participant, who must perform each one. These involve comprehending characteristics of the tokens such as colour, size and shape (e.g., small red circle), as well as the verbs and prepositions in each instruction. Adult scores between 32 and 36 are considered normal, scores between 28 and 31 are considered mildly impaired, between 20 and 27 moderately impaired, between 12 and 19 severely impaired, and scores of 11 or less are considered very severely impaired (De Renzi & Faglioni, 1978).

3.2.2.2 Assessment of Functionality

Caregivers completed two questionnaires each assessing different aspects of participants’ functional abilities.

3.2.2.2.1 Physical Self-Maintenance Scale

The Physical Self-Maintenance Scale (Lawton & Brody, 1969) was used to assess independence for various self-care tasks (refer Appendix IX). The scale is a questionnaire in which caregivers rate the abilities of the individual in the areas of toileting, feeding, dressing, grooming, locomotion, and bathing. A scale of 1 to 5 is used, where 1 indicates no difficulty, and 5 indicates intrusive impairment. A total score of 6 indicates no difficulties and a score of 30 reflects severe self-maintenance difficulties, or a total inability to carry out self-maintenance.

3.2.2.2.2 Instrumental Activities of Daily Living Scale

The Instrumental Activities of Daily Living Scale (Lawton & Brody, 1969) was used to assess independence in performing activities of daily living (ADLs) (refer Appendix X). This test requires caregivers to rate the participant on telephone use, shopping, food preparation, housekeeping, laundering, use of transportation, correct compliance with medication, and financial operation. A scale of 1 to 4 (one item uses a scale from 1 to 3) is used, where a score of 1 indicates no
difficulty and a score of 4 (or 3) indicates severe impairment. A total score of 8 reflects no difficulties and a score of 31 reflects severe difficulties with ADL’s. Three of the tasks (food preparation, laundry and housekeeping) are strongly gender linked in older people; consequently, validation is provided for an eight component questionnaire for women and a five component questionnaire for men (Lawton & Brody, 1969).

3.2.2.3 Laboratory-Based Procedural Learning Tasks

3.2.2.3.1 Mirror Drawing Task

On the mirror drawing task (Milner, 1965) individuals were required to trace around a star shape (refer Appendix XI), keeping within the boundaries of two lines, while watching their hands and the star in a mirror. Participants completed as many stars as possible in 30 minutes. The AD group repeated this task on three consecutive days to assess their rate of learning and retention. The control participants only completed this task on one occasion for 30 minutes.

3.2.2.3.2 Rotory Pursuit Task

On the rotory pursuit task (or motor pursuit task)(Corkin, 1968) participants held a stylus in their dominant hand, placed it on a small metal target, and tried to maintain contact with the target as it rotated at 30 revolutions per minute. A counter measured the amount of target contact time. Each learning trial lasted 30 s with a 30 s delay between each trial. Participants were required to perform five consecutive learning trials and, after a 20 minute delay, five additional consecutive trials.

3.2.2.4 Procedural Learning Training Programme

3.2.2.4.1 Self-care and Daily Living Tasks for Procedural Training

Twelve self-care and/or daily living skill tasks were selected for procedural training. These tasks were selected from the twenty tasks used in the original study by Zanetti et al. (1997), and from a number of other tasks which are known to cause difficulty for people with AD. The 12 tasks comprised two sets of six tasks, which were coarsely matched pair-wise for the number of elements and degree of fine motor co-ordination required for completion (see Table 3). In practice, the precise form of each task reflected not only the sequence of elements necessary to complete the task, but also the previously well-learnt method used by each participant and the specific
living requirements of each person. For example, for the training task ‘making a cup of tea or coffee’, if an individual’s usual hot drink was instant coffee made in a single cup, then training was based on that procedure. In contrast, if another individual preferred tea made in a pot, with a hot water pot, milk jug and sugar bowl on a tray, then training was based on this more complex procedure. Similarly, for the task ‘setting the table for a meal’, if a participant lived alone they were asked to set the table, for dinner, for one person, whereas a participant who lived with their spouse would be asked to set the table for two people.

Table 2
Self-care and Daily Living Tasks used for Procedural Training

<table>
<thead>
<tr>
<th>Set 1</th>
<th>Set 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Washing face and hands</td>
<td>Brushing teeth or cleaning dentures</td>
</tr>
<tr>
<td>Making a cup of tea/coffee</td>
<td>Making a simple sandwich</td>
</tr>
<tr>
<td>Putting on an item of clothing with buttons e.g., shirt or cardigan</td>
<td>Putting on shoes and doing them up (laces or buckles)</td>
</tr>
<tr>
<td>Setting the table for dinner</td>
<td>Making the bed</td>
</tr>
<tr>
<td>Locating four rooms in the house: Kitchen, bedroom, bathroom, and lounge</td>
<td>Using the TV: Turn on, channels, adjust volume, turn off</td>
</tr>
<tr>
<td>Locking the door of house and taking key</td>
<td>Making a phone call (number provided on card)</td>
</tr>
</tbody>
</table>

Approximately half of the participants were trained on the tasks in Set 1, the other half were trained on the tasks in Set 2 in order to reduce the chance of task-specific problems or benefits influencing the trained-untrained comparison. There were four deviations from this arrangement, which resulted in four tasks being trained with five individuals. This was primarily due to constraints with the last participant, who should have been trained on Set 2. Her caregiver, however, requested she not be trained on making sandwiches (due to her confusion in the kitchen and fears regarding the use of a sharp knife to cut her sandwiches) or on making the bed (the caregiver used bed-making as an excuse to clear some of the hoarded items of food from her mother’s room). A third deviation was also made in error for this participant with training on the cardigan task instead of the shoe task. The final deviation was also a result of error, which resulted in a participant being trained on the TV task instead of the room finding task.
3.2.2.4.2 Procedural Training Method

The aim of the procedural training method was to allow repeated successful performance on a skill or task. The repetition of accurate task completion may then result in a gradual, incremental increase of skill level. During procedural training on the self-care tasks, participants performed the tasks without demands on explicit memory. In other words, participants were not required to recall any instructions or remember that they had ever previously performed the task. In order to facilitate accurate completion of the tasks, a number of techniques were used: (1) Verbal support in the form of prompts, cues and answers to questions; (2) Verbal correction of mistakes (when mistakes were made, participants were stopped and instructions to achieve the correct procedure were provided step-by-step); (3) If necessary, physical demonstration of tasks, or portions of them, were carried out in order to model correct performance to the participants. The purpose of these three elements was to ensure that each task was performed correctly from beginning to completion on each trial, even if this meant providing a large number of corrections and cues (see Appendix XIa for a detailed description and examples of implementing the intervention). Participants were not asked to remember explicitly the order in which task components should be done, but they were corrected (by the methods mentioned above) if errors were made. Procedural training is attempted simply by the repetition of correct procedures. The difference between an approach requiring explicit memory and the procedural learning approach described is illustrated in the following example of ‘cleaning teeth’. If a participant forgot to put toothpaste on the toothbrush, the trainer in this procedural paradigm would say “Could you put some toothpaste (pointing) on the brush” at the time the error occurred. The same thing would be said if the participant forgot to put toothpaste on the brush a second time. In contrast, if the trainer watched the entire incorrectly performed task and then, at the end, said “You forgot the toothpaste, I would like you to remember to use it next time”, this instruction would require explicit memory and would not be consistent with a procedural learning method.

3.2.3 Procedure

Ethics approval was gained from the North Health Ethics Committee of New Zealand. Once consent was gained (refer Appendix I, II, III, IV) the AD participants
underwent a screening session comprising of an interview to obtain demographic and biographical information, and the screening tests. Following this session the experimental component of the study began, which included a baseline phase, a training phase and follow-up measures. The mirror-drawing test and motor pursuit test were performed during the last 4 days of training when the workload on the participants was decreasing.

The procedural training programme was implemented using a multiple baseline across tasks design, in which the duration of baseline measurement of the tasks varied. The first two pairs of tasks were measured at baseline for 5 days (one task of each pair was to be trained, the other task of each pair remained untrained) e.g., washing face and hands (trained), and cleaning dentures or teeth (untrained). The length of the baseline for the next two pairs of tasks was 7 days, and the baseline length for the final two pairs of tasks was 9 days. Baseline observations were followed by a training period of 10 daily sessions for each of the six trained tasks, with the matched tasks not trained. The training period took a total of 14 days because of the staggered baseline. Following training, post-test measures of all 12 tasks (six trained, six untrained) were taken at several time points, up to at least three months after the last day of training. The first post-test measure followed completion of the last day of training for each task. Each matched non-trained task was also tested at this time. Subsequent follow-up testing sessions then occurred at 1 week after the completion of all training, 2 weeks, 4 weeks and 12 weeks post-training. One of the 8 participants did not have a follow-up visit at 12 weeks post-training due to health reasons. Some participants had additional follow-up visits to those listed, at 8 weeks and monthly beyond the 12 week follow-up point.

Two dependent measures were recorded for all tasks: Time taken to completion of the task; and accuracy (number of errors). Time was measured from the commencement of the task until the completion of the task (time was deducted if the participant spent time waiting for the kettle to boil, or was interrupted in some way). Accuracy was measured by a count of the number of errors made until the completion point of the task. Written notes were kept about the qualitative nature of the errors, and of any other factors that may have affected the measurement (e.g., giving up, or refusing to attempt or complete a task).
3.2.3.1 Baseline Phase

During the baseline phase, all tasks were attempted once. As this research did not aim to test explicit memory, clear and simple instructions (e.g., I would like you to make a cup of tea) were repeated as many times as necessary, on any one occasion, before tasks were initiated. In some cases the instructions were repeated during the task.

3.2.3.2 Training Phase

The participants received 10 days of training sessions for each task. These training sessions were a maximum of 2 hours in duration, for a total of up to six tasks. On every training day, each of the tasks to be trained was repeated five times with corrections carried out in accordance with the procedural training method (no data was collected during this training). Following this, each task was carried out a sixth time with no correction of errors or prompting of any sort, during which the participants were timed and marked for accuracy.

The training and the testing were conducted in the appropriate rooms of each participant’s home. For example, making a cup of tea was done in the kitchen, and washing face and hands was done in the bathroom. If participants became tired, breaks were incorporated into the training programme. The researcher intervened if a participant was going to harm themselves (e.g., cut themselves while making a sandwich) or property (e.g., boiling an empty kettle) during the sessions.

3.2.3.3 Follow-up Phase

On each follow-up visit, all 12 tasks were tested for time and accuracy. At the completion of the follow-up phase, caregivers of the AD participants were asked questions on a range of issues related to any changes they had noticed in the participant’s mood or behaviour (refer Appendix XII).

3.2.3.4 Control Group Procedure

Control participants did not undergo procedural training and were tested on only one occasion. Following the gaining of consent (refer Appendix IV & V) the control participants received the same initial interview and screening tasks (including the rotary pursuit task) as the AD participants. In contrast, the mirror drawing was only performed on 1 day (30 minutes). Spouses or family members completed the
PSM and IADL scales for the control group. In addition, a single baseline observation of the 12 tasks was taken, with time and accuracy recorded. This was to provide a guide as to the range of performance on these tasks for normal elderly individuals without AD.

### 3.3 Results

In the results section exact $p$-values will be given to 3 decimal places. If smaller than .001 the $p$-value will be shown as $p < .001$.

#### 3.3.1 Screening tests

Table 4 provides a summary of the performance of AD and control groups on the explicit learning tasks from the WMS-R, the comprehension test, and the caregiver questionnaires which assess current functional abilities, and Table 5 shows the

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Performance of AD and Control Groups on Screening Measures</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AD Group</td>
</tr>
<tr>
<td>WMS-R Verbal Paired Associates, (Mean, [SD]), Max=24</td>
<td>8.13 (3.98)</td>
</tr>
<tr>
<td>Range: 1 to 14</td>
<td>Range: 14 to 21</td>
</tr>
<tr>
<td>WMS-R Visual Paired Associates, (Mean, [SD]), Max=18</td>
<td>4 (1.31)</td>
</tr>
<tr>
<td>Range: 2 to 6</td>
<td>Range: 7 to 18</td>
</tr>
<tr>
<td>Physical Self-Maintenance Scale, (Median, [Interquartile range])</td>
<td>9 (3.5)</td>
</tr>
<tr>
<td>Range: 6 to 13</td>
<td>Range: 6 to 6</td>
</tr>
<tr>
<td>Instrumental ADL Scale, (Median, [Interquartile range])</td>
<td>20 (13)</td>
</tr>
<tr>
<td>Range: 9 to 26</td>
<td>Range: 8 to 8</td>
</tr>
<tr>
<td>Short-token test, (Mean, [SD])</td>
<td>32.13 (2.75)</td>
</tr>
<tr>
<td>Range: 28 to 36</td>
<td>Range: 33-36</td>
</tr>
</tbody>
</table>
### Table 4
Demographic Characteristics and Screening Test Scores of Individual AD and Control Participants

<table>
<thead>
<tr>
<th>Characteristic or test name</th>
<th>Participants 1-8 (Matched control results in brackets)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (M/F)</td>
<td>F(F)  M(M)  F(F)  F(F)  F(F)  F(F)  F(F)  F(F)</td>
</tr>
<tr>
<td>Age</td>
<td>77(76)  61(60)  69(66)  76(78)  85(82)  78(78)  81(80)  87(85)</td>
</tr>
<tr>
<td>Years of education</td>
<td>14(12)  10(13)  13(14)  14(12)  9(13)  10(11)  7(11)  20(19)</td>
</tr>
<tr>
<td>Years of symptom length</td>
<td>5(0)  5(0)  2.5(0)  8(0)  5(0)  6(0)  3.5(0)  2.5(0)</td>
</tr>
<tr>
<td>MMSE score (max=30)</td>
<td>25(29)  10(30)  25(30)  14(28)  14(27)  15(28)  18(27)  21(27)</td>
</tr>
<tr>
<td>Nart-R (max=60)</td>
<td>34(56)  11(41)  51(45)  19(38)  40(37)  26(40)  12(11)  50(56)</td>
</tr>
<tr>
<td>WMS verbal pairs (max=24)</td>
<td>10(17)  1(18)  5(21)  9(14)  6(20)  10(17)  10(15)  14(21)</td>
</tr>
<tr>
<td>WMS visual pairs (max=18)</td>
<td>3(11)  2(18)  5(11)  6(9)  5(14)  3(7)  4(8)  4(12)</td>
</tr>
<tr>
<td>Physical Self-Maintenance Scale</td>
<td>9(6)  9(6)  6(6)  13(6)  10(6)  9(6)  6(6)  6(6)</td>
</tr>
<tr>
<td>Instrumental ADL Scale</td>
<td>19(8)  26(8)  9(8)  28(8)  21(8)  25(8)  13(8)  12(8)</td>
</tr>
<tr>
<td>Short-token test (max=36)</td>
<td>34(36)  30(36)  36(36)  32(36)  30(35)  32(36)  28(33)  35(36)</td>
</tr>
</tbody>
</table>

performance of individual participants on these measures. To compare the performance of the AD and control participants on these tasks independent sample t-tests and Wilcoxon signed ranks test were used, as appropriate. There was a significant difference between the performance of the two groups on the Verbal Paired Associates task ($t(14) = 5.773$, $p < .001$) and the Visual Paired Associates task ($t(14) = 5.439$, $p < .001$) of the WMS-R. As expected, the AD participants performed significantly more poorly than the control participants on both tasks, indicating impaired new learning of visual and verbal material. Each individual’s score was then converted to age-scaled scores using norms of the Mayo’s older
American normative studies (MOANS) (Ivnik et al., 1992). The scores of all of the control participants fell within 1 standard deviation from the mean for their age group, on both the paired associates tasks. Seven of the AD participants had scores that fell more than 1 standard deviation below the mean on the verbal task, while participant AD8 had a MOANS scaled score of 9 (within 1 standard deviation from the mean).

On the Visual Paired Associates task, 6 AD participants had scores more than 1 standard deviation below the mean, with the scores of the other 2 participants (AD8, AD4) falling within 1 standard deviation of the mean. The participant whose MOANS scores fell within 1 standard deviation from the mean for both tests was 87 years of age. For persons in the age range 83 and above (midpoint age 88) there is a tendency towards floor effects in the normative data for these tasks. In this case a MOANS scaled score of 9 (mean = 10, SD = 3) was obtained when correctly remembering only 14 out of 24 possible Verbal Paired Associates, with recall of only 2 out of 12 on the difficult word pairs. This participant also had a MOANS scaled score of 8 when remembering only 4 out of 18 of the Visual Paired Associates items. As a score of 3 out of 18 is chance performance, this suggests that a real difficulty with new learning may be masked by the floor effect on the normative data for this task. This account seems very likely for this individual, particularly considering her high NART-R pre-morbid IQ estimation of 120, and her high level of educational attainment with completion of a double degree. One could reasonably estimate her premorbid abilities as in at least the ‘high average range’, if not ‘superior’. Given this, we would expect her to be performing significantly above the mean for her age group on most kinds of tasks, including memory.

There was a significant difference between AD and control groups on the Short Token Test, \( t(14) = 3.237, p = .010 \), with the AD participants performing at a lower level than the control participants on this measure of comprehension. However, the scores of all 8 AD participants were above the cut-off for mildly impaired performance (28/36).

The data from the caregiver rater scales (see Table 4) were analysed using Wilcoxon-Mann-Whitney tests due to the ordinal nature of the measures. There were significant differences between the AD and control groups on the Physical Self Maintenance Scale \( W_x \geq 88, n = 8, m = 8, 2\text{-tailed } p = .038 \) and the Instrumental Activities of Daily Living Scale \( W_x \geq 100, n = 8, m = 8, 2\text{-tailed } p < .001 \). All
members of the control group scored the minimum possible score on the two caregiver-rated scales, indicating that the control participants were completely independent on all the physical self-maintenance and daily living skills examined in the questionnaires. Five of the 8 AD group participants needed assistance with physical self-maintenance skills and all 8 participants needed assistance with activities of daily living.

3.3.1.1 Laboratory Based Procedural Learning Tasks

3.3.1.1.1 Mirror Drawing Task

Six of the 8 AD participants had data from the mirror drawing task which could not be analysed. After 3 days (30 minutes per day) of training the attempts at tracing the star by these 6 participants either did not resemble a star, or had more than 100 exits from the star shape. Of the 2 participants with measurable results, the first went from a mean of 68.5 (SD = 37.48) exits from the star on Day 1, to a mean of 34.5 (SD = 3.5) exits from the star on Day 3, and consistently took at least 15 minutes to complete each star. The second participant improved from an average of 49.67 (SD = 9.71) to 19.67 (SD = 4.51) exits on Day 3 and consistently took at least 10 minutes to trace each star for all 3 days.

The data of the control group were also difficult to analyse. Two participants were not willing to attempt this task, and a third gave up almost immediately. Of the remaining 5 participants, 4 immediately performed the task well, with a range of 0-9 exits from the star on the first trial. Because of the high level of initial performance, there was no real change over the 30 minutes of training, and these individuals made between 0 and 7 exits on the final attempt. All of these participants took 5 minutes or less to trace each star. The eighth control participant made over 100 exits from the star on the first trial and improved to 56 on their third and final attempt. This participant took more than 10 minutes to draw the first two stars, and under 10 minutes for her third star.

3.3.1.1.2 Rotory Pursuit Task

Figure 4 shows the mean contact time on the rotory pursuit target for each trial for the two groups. The control group began with a higher amount of contact time and this difference was maintained across trials. However, both groups displayed an increase in contact time over the trials and maintained this improvement.
between Trials 5 and 6. This was confirmed by a repeated measures analysis of variance (ANOVA) with Group as a between-subjects factor, and Learning trials as a within-subjects factor. There was a significant main effect of Group (\(F(1,9) = 10.825, p = .005\)) with control participants achieving a higher amount of contact time overall than AD participants. There was also a significant main effect of Learning trial (\(F(1,9) = 18.474, p = .001\)). Pairwise comparisons revealed significant differences in contact time between Trial 1 and Trials 3 to 10; Trial 2 and Trials 9 and 10; and Trial 4 and Trials 8 and 10. In other words, all significant increases in contact time occurred during the first four learning trials.

![Figure 1. The mean time on target on the rotary pursuit task for the AD group and the control group. Maximum time on target was 30 s during two sets of five observations 30 s apart with a 20 minutes break between Trials 5 and 6.](image)

There was no significant trial by group interaction (\(F(1,9) = 0.449, p = .514\)). In other words, although the control group achieved more contact time overall, the learning gradient (rate of change over learning trials) of the two groups was not different. When a visual analysis of the individual plots was conducted, all AD participants showed improvement, but only 2 of 8 participants reached high levels of performance (i.e., with contact time close to maximum possible performance on the
final trials). In contrast, all but 2 control participants reached near maximum performance (i.e., very close to 30 s, out of a possible 30 s of contact time with the target) after just nine attempts. It is possible that these ceiling effects could have masked differences in the learning gradients of the two groups.

There was no significant difference between Trials 5 and 6, i.e., the trials before and after the 20 minute gap ($p = 1.00$). To look directly at whether level of performance was maintained after a delay in each of the groups, paired t-tests comparing contact time on Trial 5 and Trial 6 were conducted. There was no significant difference in mean contact time on these two trials in the AD group ($t(7) = 1.268$, $p = .245$) or the control group ($t(7) = 0.644$, $p = .540$). In other words, the improved performance achieved at the end of five learning trials was maintained over a 20-minute delay in both groups.

### 3.3.2 Procedural Learning of Self-care Skills and Activities of Daily Living

#### 3.3.2.1 Group Analyses

Due to the small number of participants and the non-normal distribution of the data, non-parametric analyses were used to analyse the effectiveness of the procedural training programme. First, change in performance during baseline was assessed comparing first and last baseline observations. Following this, baseline performances of the tasks were compared to task performances at post-test. Finally, the raw response time for each task, for each participant, was converted into a measure of percent improvement from baseline to post-training, using the following formula:

$$\frac{\text{baseline value} - \text{post-test value}}{\text{baseline value}} \times 100 = \text{percent change}.$$ 

This reduced some of the variability in the time taken to complete different tasks, and also made it easier to compare training effectiveness across different participants. Note, however, that this transformation did not normalise the data sufficiently to perform parametric statistical analyses.
3.3.2.1 Baseline Phase

To assess whether there was overall improvement in performance on tasks during the baseline period, the average time taken for AD participants to complete the 12 tasks during the first baseline observation was compared to the average time taken during the last baseline observation. A Wilcoxon signed ranks test showed no significant difference in the mean time to complete the tasks between these two time points ($T_- = 21, n = 8, p = .371$). There was also no significant difference in the average numbers of errors committed between the two baseline time points ($T_+ = 15, n = 6, p = .469$). Given this result, the last baseline measurement was used for all subsequent comparisons.

3.3.2.1.2 Effectiveness of Training

To investigate whether there was a decrease in time taken to perform tasks during training, a Wilcoxon signed ranks test was used to compare the mean raw baseline scores of the trained tasks for each participant with the mean scores after training had been completed. Trained tasks were performed significantly faster at post-test than at baseline, ($T_+ = 35, n = 8, p = .008$). In contrast, the time taken to perform the untrained tasks after the 3-week training interval was not significantly different from baseline, ($T_- = 21, n = 8, p = .371$).

Analyses were also conducted to evaluate whether the percentage change in time taken to complete trained and untrained tasks at post-test (relative to baseline) was significantly different from zero. For the trained tasks there was a significant percentage decrease in mean time taken to perform the tasks after training ($T_+ = 35, n = 8, p = .008$), whereas there was no significant percentage change for untrained tasks ($T_- = 19, n = 8, p = .473$). Figure 5 illustrates that the reduction in time taken to complete trained tasks was consistent across all 8 AD participants.

In order to assess the effectiveness of procedural training in reducing the number of errors made during task performance, the mean number of errors made on the six trained tasks at baseline were compared to the mean number of errors made on the six tasks at post-test.
Figure 2. Mean percent change in task performance time for individual AD participants from the last baseline to the post-test measure for the trained and untrained tasks. Note the horizontal line at zero corresponds to the last baseline point.

Figure 3. Mean percent change in number of errors for individual AD participants from the last baseline to the post-test measure for trained and untrained tasks. Note the horizontal line at zero corresponds to the last baseline point.
A Wilcoxon signed ranks test revealed that significantly fewer errors were made at the post-training test \((T^+ = 28, n = 7, p = .008)\). In contrast, when the same analysis was conducted on the error data for untrained tasks, there was no significant difference in the mean number of errors made at baseline and post-test \((T^- = 15.5, n = 7, p = .438)\). Figure 6 shows the percent change in mean numbers of errors after training (at post-test) for both trained and untrained tasks for each participant. Fewer errors were made on trained tasks for 7 participants (all except AD2). Four AD participants showed non-significant reductions in the number of errors on untrained tasks.

### 3.3.2.1.3 Comparison of Alzheimer’s and Control Groups

The performance of the AD group participants was compared to that of the healthy elderly participants at baseline and post-test. Time taken to perform all tasks and number of errors were averaged for the control group and compared to AD group performance, firstly, on the last baseline and, secondly, to post-test performances on trained tasks only, using Wilcoxon-Mann-Whitney tests. AD participants performed tasks slower than the control group before training \((W \chi \geq 99, n = 8, m = 8, \text{2 tailed } p = .000)\). However, at post-test there was no significant difference in task performance time between the two groups \((W \chi \geq 83, n = 8, m = 8, \text{2-tailed } p = .130)\). Figure 7 shows the mean task time for the control group, indicated as a horizontal line (mean = 30.718, Std error = 2.037). The AD group baseline and the post-test mean task times (for both the trained and untrained tasks) are shown in Figure 7, with the tasks that were to be trained or untrained separated at baseline as well as post-test.

At baseline the AD group made significantly more errors than the control group \((W \chi \geq 88, n = 8, m = 8, \text{2-tailed } p = .038)\). To test whether training of the AD group improved performance to the same level as the control group, the post-test average number of errors on trained tasks was compared between the AD group and the control group. There was no significant difference in the number of errors between the two groups at post-test \((W \chi \geq 100, n = 8, m = 8, \text{2 tailed } p = .000)\). Mean number of errors committed by the AD group during task performance at baseline and post-test are shown in Figure 8, with the tasks that were to be trained or untrained separated at baseline as well as post-test. Note that control participants made no errors.
Figure 4. AD group mean task performance times (trained and untrained) at baseline and post-test, compared to control group mean (horizontal line). Baseline tasks were divided into the tasks that were subsequently either trained or untrained to give an accurate comparison.

Figure 5. AD group mean number of errors per task for trained and untrained tasks at baseline and post-test compared to control group (zero errors). Baseline tasks were divided into the tasks that were subsequently either trained or untrained, to give an accurate comparison.
3.3.2.1.4 Did Training Effects Last?

To determine whether improvement in performance on trained tasks was maintained, the mean last baseline performance time on trained tasks for each AD participant was compared to mean performance times at three follow-up test points (namely 2 weeks, 4 weeks and 12 weeks post-training) using Wilcoxon Signed Ranks tests. As can be seen in Table 6, performance on the trained tasks at the three follow-up points was significantly faster than at baseline. The same analyses were undertaken with the untrained tasks, which revealed no significant difference between baseline and any of the three follow-up time points in time taken to perform the untrained tasks.

Table 5
Wilcoxon Signed Ranks Test Results for AD Participants Between the Final Baseline and Post-test Follow-ups for Time Taken (Both Raw Data and Percent Change) and Errors Made

<table>
<thead>
<tr>
<th></th>
<th>BL-1st PT</th>
<th>BL-wk2</th>
<th>BL-wk4</th>
<th>BL-wk12</th>
</tr>
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<tr>
<td><strong>Time</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trained (raw)</td>
<td>$T_{+}=35$, $n=8$, $p=0.008$</td>
<td>$T_{+}=34$, $n=8$, $p=0.020$</td>
<td>$T_{+}=37$, $n=8$, $p=0.004$</td>
<td>$T_{+}=28$, $n=7$, $p=0.008$</td>
</tr>
<tr>
<td>Not trained (raw)</td>
<td>$T_{-}=21$, $n=8$, NS</td>
<td>$T_{+}=28.5$, $n=8$, NS</td>
<td>$T_{+}=18$, $n=7$, NS</td>
<td>$T_{-}=23$, $n=7$, NS</td>
</tr>
<tr>
<td>Trained (% change)</td>
<td>$T_{+}=35$, $n=8$, $p=0.008$</td>
<td>$T_{+}=33$, $n=8$, $p=0.020$</td>
<td>$T_{+}=37$, $n=8$, $p=0.004$</td>
<td>$T_{+}=28$, $n=7$, $p=0.008$</td>
</tr>
<tr>
<td>Not trained (% change)</td>
<td>$T_{-}=19$, $n=8$, NS</td>
<td>$T_{+}=29$, $n=8$, NS</td>
<td>$T_{+}=26$, $n=8$, NS</td>
<td>$T_{-}=18$, $n=7$, NS</td>
</tr>
<tr>
<td><strong>Errors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trained</td>
<td>$T_{+}=28$, $n=7$, $p=0.008$</td>
<td>$T_{+}=28$, $n=7$, $p=0.008$</td>
<td>$T_{+}=28$, $n=7$, $p=0.008$</td>
<td>$T_{+}=27$, $n=7$, $p=0.017$</td>
</tr>
<tr>
<td>Not trained</td>
<td>$T_{-}=15.5$, $n=7$, NS</td>
<td>$T_{+}=11$, $n=6$, NS</td>
<td>$T_{+}=8.5$, $n=5$, NS</td>
<td>$T_{+}=15$, $n=7$, NS</td>
</tr>
</tbody>
</table>

First and last baseline comparison of time $T_{-}=21$, $n=8$, NS (non-significant increase in time (raw data)). First and last baseline comparison of errors $T_{+}=15$, $n=6$, NS (non-significant decrease in errors).

Figure 9 shows percentage change in time taken to perform tasks for both trained and untrained tasks for the AD group. The AD group performed trained tasks
a mean of 28% faster at post-test compared to final baseline. Although the size of the improvement declined over the follow-up period, all comparisons between percentage change in time taken between baseline and the three follow-up time points were significant (see Table 6). In other words even the 15% reduction in mean time taken to perform trained tasks at 12 weeks post-training represented a statistically significant decrease in time taken to perform the tasks. In contrast performance time increased for untrained tasks over the follow-up phase, although this increase was not significant (see Table 6 for the details of the comparisons between baseline and the three follow-up points for the untrained tasks).

![Graph showing mean percent change in task performance time between last baseline and three follow-up points.](image)

**Figure 6.** AD group mean percent change in task performance time between last baseline and three follow-up points.

The mean error rates on the six trained tasks for each participant were also compared at baseline and at the three follow-up test points to determine whether error reduction following training was maintained over time. As can be seen in Table 6, significantly fewer errors were made at all three post-training follow-ups (2 weeks, 4 weeks and 12 weeks) than at baseline. In contrast, for untrained tasks there was no
significant difference in the mean number of errors made at baseline and the three follow-up measures.

### 3.3.2.2 Single Subject Analyses

The data for each task for each participant are presented in Figures 10-20, including expanded graphs for instances when tasks took in excess of 200 s to complete during data collection. The single-subject design incorporated in this study enabled analyses of training effectiveness at the level of individual tasks for each participant. In order to make consistent decisions about the degree of success or failure of training across individuals and across tasks, the following procedure was adopted. Using the sigma-plot statistical package, the baseline and post-training performance times for each participant and each task were plotted against real time (days). A linear-regression analysis was conducted on each set of follow-up data, with 95% confidence intervals (CI) calculated and projected back in time to the baseline period. This made it possible to see, for each task, how many baseline observations were “worse”, at the level of 97.5% confidence (i.e., those points falling above the projected upper 95% CI), than expected given performance during the follow-up period. If training was effective there should be significantly more points above the upper 95% CI in baseline for the trained tasks than for the untrained tasks, as performance in the follow-up period for the trained tasks should be faster and less variable.
Figure 10 continued. The subsequent figures for each participant follow the same format. The performance time data comprises the time taken to complete a task (points), the error data (bars) show the number of errors made (maximum 10). If there is no point shown for time data the task was either not completed, or not attempted. Baseline points are presented as a line plot (if single points occur in baseline they are represented with a cross). The post-test points are represented with circles and the follow-up points are represented with squares. The vertical lines through the graphs show the length of the baseline, and training phases for each task. Each task name is written alongside the data for that task. If the time taken to complete the task exceeds 200 seconds an extended graph is included on the facing page.
Figure 7. Participant AD1: All 12 tasks, either trained (T) or untrained (UT) (see facing page).
Figure 8. The expanded data for participant AD2.
Figure 9. Participant AD2: All 12 tasks, either trained (T) or untrained (UT). These graphs show both time data (points), and error data (bars).
Figure 10. Participant AD3: All 12 tasks, either trained (T) or untrained (UT). These graphs show both time data (points), and error data (bars).
Figure 11. Participant AD4: All 12 tasks, either trained (T) or untrained (UT). These graphs show both time data (points), and error data (bars).
Figure 12. The expanded data for participant AD5.
Figure 13. Participant AD5: All 12 tasks, either trained (T) or untrained (UT). These graphs show both time data (points), and error data (bars).
Figure 14. The expanded data for participant AD6.
Figure 15. Participant AD6: All 12 tasks, either trained (T) or untrained (UT). These graphs show both time data (points), and error data (bars).
Figure 16. Participant AD7: All 12 tasks, either trained (T) or untrained (UT). These graphs show both time data (points), and error data (bars).
Figure 17. Participant AD8: All 12 tasks, either trained (T) or untrained (UT). These graphs show both time data (points), and error data (bars).
To illustrate this method the data comprising a complete set of tasks (trained and untrained respectively) and analyses for 1 participant (AD7) are presented in Figures 21 and 22. The six trained tasks in Figure 21, have the following proportion of baseline observations which are slower than expected at a 97.5% level of confidence, based on AD7’s performance in the follow-up period: 5/5, 4/5, 3/7, 2/7, 9/9 and 9/9. When a task can not be completed, the time taken to complete the task is judged to be above the upper 95% CI. In contrast, on the untrained tasks shown in Figure 22, AD7 has the following proportion of baseline observations which are slower: 1/5, 2/5, 0/7, 1/7, 0/9, 3/9. Note that tasks with no improvement in time taken to completion may still have shown a reduction in the number of errors that were made.

Given the relatively sparse nature of the error data, it was decided to use time data as the primary measure of training effectiveness for single subjects. Binomial tests were used to determine whether each AD participant showed an overall effect of training on performance. For each binomial test, for each participant, the total number of baseline points that fell above the upper 95% CI for all untrained tasks was the ‘expected’ value and the number that fell above the upper 95% CI for all trained tasks was the ‘actual’ value. The binomial Z score values and corresponding p-values are shown in Table 7. All but 1 AD participant showed a statistically significant overall training effect.

In addition, a way to decide which of the individual tasks were successfully trained for individual participants was required. A figure of 70% was selected, that is, if 70% of the baseline points fell above the upper 95% CI generated by performance in the post-training and follow-up period, then the task would be considered successfully trained. Although this is a somewhat arbitrary figure, it is clearly well outside of the range of performance for untrained tasks in this study. On average 14.9% of the points at baseline for untrained tasks fell above the upper 95% CI. Only 2 participants had values higher than this, with the highest at 23.8% above the upper 95% CI. Given this, requiring 70% of the points at baseline to be above the upper 95% CI is a conservative figure with which to judge training success.
Figure 18. The baseline and follow-up data points for six trained tasks for participant AD7. The graphs show the regression line for the follow-up points and the 95% Confidence Intervals associated with it. If the participant was unable to perform the task there is no point shown for that attempt.
Figure 19. The baseline and follow-up data points for six untrained tasks for participant AD7. The graphs show the regression line for the follow-up points and the 95% Confidence Intervals associated with it. If the participant was unable to perform the task there is no point shown for that attempt.
In Table 7 the number of points at baseline falling above the upper 95% CI for each trained task are shown, as well as the number of baseline observations which fell below the upper 95% CI. For example, task one for AD1 on Table 7 shows the figures ‘1/8’. This indicates that one baseline point fell above the upper 95% CI and 8 points fell below the upper 95% CI, i.e., this task showed no training effect. In the table there is, additionally, an indication of whether each task met the criterion for showing a training effect by time (“Y” if it did). Any tasks performed at ‘normal levels’ in baseline (with normal defined as falling within the control group range of performance) are marked as “norm” in Table 7. The summed total of the points at baseline falling above the 95% CI for the untrained tasks (UT, 7-12) is also shown. Note that only one untrained task for 1 participant met the criteria for a trained effect out of a possible 48 (i.e., six tasks for 8 participants). This was the ‘finding rooms’ task for participant AD6.

Error data were used as a second judgement criterion regarding judgement of training effectiveness. The error data were visually assessed and considered successful when there was a reduction in the frequency of errors between baseline and follow-up. In addition to the judgements of the researcher, an independent rater, experienced in single subject design and visual analysis, was provided with the criterion for the EI judgement and independently classified the error data for each task and each participant. The inter-rater reliability for judgement of error improvement was $r = .910$. Given the high level of inter-rater reliability the initial ratings were used. If the error rate in the follow-up period was lower than errors committed at baseline then ‘error improvement’ (EI) is indicated on Table 7. Two untrained tasks (out of 48) showed an error improvement: The ‘TV’ task for AD1 (AD); and the ‘putting on shoes’ task for AD2 (BH). In summary, if a task did not show either a time (Y) or error improvement (EI), then no training effect was seen for that task. Note, however, that those tasks performed at normal levels throughout the research period would not be expected to show training benefits.
### Table 6

**Overall Effectiveness of Training of Each Participant, and Task by Task**

**Judgement of Time and/or Error Improvement**

<table>
<thead>
<tr>
<th>Participant</th>
<th>Binomial score</th>
<th>p-value</th>
<th>Task 1</th>
<th>Task 2</th>
<th>Task 3</th>
<th>Task 4</th>
<th>Task 5</th>
<th>Task 6</th>
<th>UT 7-12</th>
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<tbody>
<tr>
<td>AD1</td>
<td>-0.222</td>
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<td>0.0/9</td>
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<td>2/3</td>
<td>1/6</td>
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<td>7/30</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>(EI)</td>
<td>(EI)</td>
<td>(norm)</td>
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<td></td>
<td></td>
</tr>
<tr>
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<tr>
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<td></td>
<td></td>
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<td>(EI)</td>
<td>(EI)</td>
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<td>(Y,EI)</td>
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</table>

EI = error improvement; Y = Yes, there was a time improvement; Norm = normal performance at baseline; UT = untrained tasks

Overall, of the 8 participants, 3 (AD2, AD5, and AD7) showed a training effect for all six trained tasks (100% success). A fourth participant, AD3, showed a training effect for four tasks and was normal for the remaining two, thereby also showing a 100% training success rate for impaired tasks. AD6 and AD4 showed a training effect for five tasks (83.3%). AD8 showed a normal performance in two tasks, and a training effect for three of the remaining four tasks (75%). Finally, participant AD1, who was the only participant who did not show an overall training
effect based upon the binomial results, had two training tasks in the normal range at baseline. Of the remaining four, two showed a training effect for error improvement (50%).

To determine whether there were particular tasks that contributed to the training effect, and others that did not, the results from Table 7 were reordered by task (rather than by participant) and are presented in Table 8. The binomial evaluation used to assess improvement in performance time was repeated for each task using the untrained data for that task for the ‘predicted’ value and the trained data for that task for the ‘actual’ value. As before, the key data was the number of baseline points falling above the upper 95% CI associated with the regression line for follow-up data and projected back in time. The binomial results (see Table 8) showed that, using time as the dependent variable, 10 of the 12 tasks showed a training effect. The second level of analysis, looking at the results for each participants’ performance of each task, showed whether the task was at a normal level at baseline (norm), showed a time improvement (Y), or an error improvement (EI). The two tasks which showed no significant training effect based on response time, were the ‘putting on cardigan’ task and the ‘putting on shoes’ task. For the ‘putting on cardigan’ task, 2 of the 5 participants receiving training performed normally in baseline, 1 participant showed an error improvement, and 2 participants showed no training effect. For the ‘putting on shoes’ task, 1 of 3 participants performed this task normally during baseline, and the remaining 2 participants showed error improvements. Therefore, with the ‘putting on shoes’ task the error improvements indicate training effectiveness despite the ‘no effect’ indicated by the binomial analyses on performance time.

### 3.3.2.3 Individual Differences in Effectiveness of Training

An important issue regarding this type of training programme is whether it is possible to accurately predict who will benefit from training. More specifically, are there any factors or aspects of an individual’s pre-existing abilities which indicate whether procedural training is likely to be effective? To investigate this, performance in a number of screening test areas (e.g., cognitive level) were correlated with results of the training procedure using Spearman’s correlation (See Table 9).
Table 7
Overall Training Effectiveness for Each Task, and Task by Participant
Judgement of Time and/or Error Improvement

<table>
<thead>
<tr>
<th>Task</th>
<th>Binomial score</th>
<th>p-value</th>
<th>R1</th>
<th>R2</th>
<th>R3</th>
<th>R4</th>
<th>R5</th>
<th>UT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sandwich</td>
<td>-13.385</td>
<td>.000</td>
<td>8/1</td>
<td>6/3</td>
<td>5/0</td>
<td></td>
<td></td>
<td>3/38</td>
</tr>
<tr>
<td>Tea/Coffee</td>
<td>-5.984</td>
<td>.000</td>
<td>2/7</td>
<td>2/7</td>
<td>5/0</td>
<td>9/0</td>
<td>1/8</td>
<td>3/20</td>
</tr>
<tr>
<td>Teeth Cleaning</td>
<td>-6.641</td>
<td>.000</td>
<td>0/9</td>
<td>2/7</td>
<td>2/7</td>
<td>4/1</td>
<td></td>
<td>1/31</td>
</tr>
<tr>
<td>Wash Face &amp; Hands</td>
<td>-3.326</td>
<td>.001</td>
<td>7/2</td>
<td>1/4</td>
<td>1/8</td>
<td>0/9</td>
<td></td>
<td>3/29</td>
</tr>
<tr>
<td>Make Bed</td>
<td>-3.674</td>
<td>.000</td>
<td>5/2</td>
<td>1/4</td>
<td>3/4</td>
<td></td>
<td></td>
<td>4/23</td>
</tr>
<tr>
<td>Set Table</td>
<td>-3.796</td>
<td>.000</td>
<td>3/4</td>
<td>3/4</td>
<td>4/3</td>
<td>6/1</td>
<td>2/3</td>
<td>6/13</td>
</tr>
<tr>
<td>Shoes</td>
<td>.0340</td>
<td>.488 (NS)</td>
<td>0/7</td>
<td>0/5</td>
<td>2/5</td>
<td></td>
<td></td>
<td>4/27</td>
</tr>
<tr>
<td>Cardigan/Shirt</td>
<td>.2148</td>
<td>.216 (NS)</td>
<td>3/2</td>
<td>0/7</td>
<td>0/7</td>
<td>2/5</td>
<td>2/3</td>
<td>3/16</td>
</tr>
<tr>
<td>Lock Door</td>
<td>-4.368</td>
<td>.000</td>
<td>3/2</td>
<td>5/0</td>
<td>1/6</td>
<td>9/0</td>
<td></td>
<td>6/15</td>
</tr>
<tr>
<td>Phone</td>
<td>-7.725</td>
<td>.000</td>
<td>0/5</td>
<td>7/2</td>
<td>5/0</td>
<td>1/6</td>
<td></td>
<td>2/24</td>
</tr>
<tr>
<td>TV</td>
<td>-13.383</td>
<td>.000</td>
<td>4/1</td>
<td>7/0</td>
<td>7/0</td>
<td>5/0</td>
<td>9/0</td>
<td>3/18</td>
</tr>
<tr>
<td>Rooms</td>
<td>-3.475</td>
<td>.000</td>
<td>5/0</td>
<td>7/2</td>
<td>3/4</td>
<td></td>
<td></td>
<td>11/ 22</td>
</tr>
</tbody>
</table>

EI = error improvement; Y = Yes, there was a time improvement; norm = normal performance at baseline, UT = Untrained tasks (summed results)

More specifically, correlations were calculated between results of the training procedure and MMSE scores, scores on the WMS-R tests, ADL caregiver scale scores, ability to learn the rotary pursuit task (difference between 1st and 10th trial), and overall ability to perform the rotary pursuit task (mean contact time over 10
trials). Two measures of procedural training success were used as indicators of benefit from procedural task retraining. The first measure was mean percent improvement between baseline time and the mean follow-up time for each participant.

The second measure involved the same method with error data (mean percent improvement from baseline to mean follow-up).

**Table 8**

**Correlation Analysis of the Individual Screening Test and Training Results**

<table>
<thead>
<tr>
<th>Participant</th>
<th>MMSE Score</th>
<th>WMS-R Verbal memory</th>
<th>WMS-R Visual memory</th>
<th>PSM</th>
<th>IADL</th>
<th>Rotor pursuit (RP) difference from 1st to 10th trial in sec.</th>
<th>Mean total RP over 10 trials</th>
<th>Time % improvement mean BL to average FU</th>
<th>Error % improvement mean BL to average FU</th>
</tr>
</thead>
<tbody>
<tr>
<td>AD1</td>
<td>25</td>
<td>10</td>
<td>3</td>
<td>9</td>
<td>19</td>
<td>24.7</td>
<td>21.3</td>
<td>25.5</td>
<td>88</td>
</tr>
<tr>
<td>AD2</td>
<td>10</td>
<td>1</td>
<td>2</td>
<td>9</td>
<td>26</td>
<td>22.7</td>
<td>15.3</td>
<td>48.9</td>
<td>87.9</td>
</tr>
<tr>
<td>AD3</td>
<td>25</td>
<td>5</td>
<td>5</td>
<td>6</td>
<td>9</td>
<td>11.8</td>
<td>26</td>
<td>29.7</td>
<td>100</td>
</tr>
<tr>
<td>AD4</td>
<td>14</td>
<td>9</td>
<td>6</td>
<td>13</td>
<td>28</td>
<td>5.2</td>
<td>16.7</td>
<td>7.4</td>
<td>58.5</td>
</tr>
<tr>
<td>AD5</td>
<td>14</td>
<td>6</td>
<td>5</td>
<td>10</td>
<td>21</td>
<td>7.1</td>
<td>3.5</td>
<td>25.6</td>
<td>72.6</td>
</tr>
<tr>
<td>AD6</td>
<td>15</td>
<td>10</td>
<td>3</td>
<td>9</td>
<td>25</td>
<td>8.5</td>
<td>14.2</td>
<td>26.1</td>
<td>90.1</td>
</tr>
<tr>
<td>AD7</td>
<td>18</td>
<td>10</td>
<td>4</td>
<td>6</td>
<td>13</td>
<td>4.2</td>
<td>2.8</td>
<td>45.2</td>
<td>66.4</td>
</tr>
<tr>
<td>AD8</td>
<td>21</td>
<td>14</td>
<td>4</td>
<td>6</td>
<td>12</td>
<td>12.5</td>
<td>9</td>
<td>25.8</td>
<td>87.5</td>
</tr>
</tbody>
</table>

**MMSE correlations**

| MMSE correlations | 0.426 | 0.139 | -0.541 | **-0.762** * | 0.204 | 0.445 | -0.159 | 0.452 |

**Verbal memory correlations**

| 0.192 | -0.183 | -0.283 | -0.304 | -0.257 | -0.434 | -0.178 |

**Visual memory correlations**

| 0.267 | -0.153 | -0.695 | -0.043 | -0.670 | -0.501 |

**PSM correlations**

| **0.881** * | -0.070 | 0.081 | -0.586 | -0.540 |

**IADL correlations**

| 0.073 | -0.009 | -0.251 | -0.340 |

**Rotor pursuit improvement correlations**

| 0.502 | 0.306 | 0.578 |

**Rotor attainment correlations**

| -0.243 | 0.563 |

**Time % improvement BL to FU, correlations**

| 0.281 |

*Significant correlations are indicated in bold*

MMSE was significantly negatively correlated with IADL scale indicating the higher the MMSE score the better the self-care abilities. As expected the PSM and IADL measures were also significantly correlated. No other significant correlations were found. In particular there were no significant correlations between the laboratory-based procedural learning task (rotor pursuit) and the measures of success of procedural training for the ADL tasks.

The difficulties with the two measures of training success used in the correlations are that they are coarse estimates i.e., means of means. It was not useful or meaningful to use the ‘Y’ and ‘EI’ results from Table 7 for the correlation analysis, as the participants did not show enough difference between them to be ranked. For example, 5 of the 8 participants showed an 100% training effect. In addition, 7 of 8 participants showed a training effect from the binomial calculations. Even given
these measurement difficulties is should be noted that 2 participants showed unexpected results on training given their rotory pursuit ability. Participant AD1 showed excellent rotory pursuit ability but no procedural training effect on task performance time of daily tasks (additionally, only two of four possible tasks showed an error improvement). In contrast, participant AD7 showed poor rotory pursuit learning and yet showed a significant training effect with all six tasks demonstrating an effect of training. While some AD individuals may show a relation between self-care skill learning abilities and their rotory pursuit learning abilities, the unexpected results of these 2 participants suggest it is not possible to predict who will gain significant benefit from procedural training based on performance on the rotory pursuit task.

3.3.2.4 Single Case Example (AD7)

Case AD7 is presented to illustrate in detail the research process and depth of information gathered during this study. AD7, an 81-year-old New Zealand European female, lived in her own home with her husband of 60 years. She had a four-year history of gradual, increasing forgetfulness. Her spouse reported that her memory difficulties had been evident in several areas of functioning including forgetting recent events, contents of conversations, the location of objects around the house, and also difficulties with word finding. She also had difficulty with some ADLs (e.g., ripping the end of the tea bag and pouring the leaves into a cup) and could no longer handle money or do the shopping.

Two years before screening AD7 was diagnosed with probable AD according to NINDAS criteria (McKhann et al., 1984) at the Memory Clinic at North Shore Hospital, Auckland. AD7’s CT scan showed a pattern of cortical atrophy consistent with a diagnosis of AD, with no signs of focal lesions or other abnormalities. There was no known history of dementia in her family. She did not have any history of drug or alcohol abuse and she consumed on average seven standard drinks per week.

3.3.2.4.1 Results for AD7

AD7 had progressed into the moderate stages of AD, as determined by her functional difficulties and tests on neuropsychological measures, at the time of screening. AD7 scored 18 on the MMSE (orientation 8/10, registration 2/3, recall 0/3, serial sevens 1/5, naming 2/2, repetition 0/1, three stage command 3/3, reading
She scored 10/24 on Verbal Paired Associates of the WMS-R (MOANS age scaled score of 6 (Ivnik et al., 1992)) obtaining 10/12 for the easy pairs and 0/12 for the difficult pairs. Similarly, her score on Visual Paired Associates of the WMS-R was 4/18 (MOANS age scaled score of 8). AD7 scored 28/36 for the short token test with all but one error made in the set of more complex instructions. Her NART-R estimated IQ was 90.36, which is consistent with her educational and work history. AD7 left school at the age of 12 and worked as a waitress in her family’s hotel, and later as a shop assistant.

According to her husband’s ratings on the Physical Self-Maintenance Scale AD7 has no functional impairment in the area of physical self-maintenance (score of 6/30). In real terms this would imply that AD7 could, without assistance, use the toilet, eat, dress, wash herself, and walk freely. The researcher’s observations were consistent with this, with the exception of the area of personal hygiene such as cleaning teeth, washing her hair, and hand washing. Her performance on these tasks was below the level described by her spouse.

The Instrumental Activities of Daily Living scale was also completed by her husband. Her score of 13/31 indicated that AD7 was able to operate the telephone fully, maintain the housework independently, do all the laundry, and accurately administer her medication at the correct time. Her husband reported that she had some difficulty with preparing meals, shopping, and using public transport. She did not contribute to major financing but managed day-to-day purchases. The researcher’s observations were not consistent with the scores given by her husband, which possibly indicated that the caregiver overestimated AD7’s abilities. She could not look up telephone numbers, and occasionally made dialling errors (IADL score = 2). She could not prepare meals if supplied with ingredients, nor use the microwave to heat meals; although she could occasionally make a simple sandwich (IADL score = 4). Her ability to maintain the house was greatly exaggerated by her spouse. They had a housekeeper once a week, as AD7 could no longer do any housework (IADL score = 4). AD7 did not travel alone or arrange transport (IADL score = 3). AD7 administered her own asthma medication but often forgot her daily morning and nightly dose of Becotide (preventative) resulting in her regularly needing additional doses of Ventolin when she felt breathless (IADL score = 2). AD7 had no occasion to handle money as her husband did all the shopping but she had made mistakes with
change in the months prior to screening (IADL score = 3). The IADL score would have been 22/31 if completed by the researcher in contrast to a score of 13/31 when completed by her spouse. This demonstrates the potential difficulty with caregiver interview scales, namely that caregivers may underestimate or overestimate difficulty to a greater degree than another observer.

3.3.2.4.1.1 Laboratory Based Procedural Learning Tasks

Over the 3 day test period for the mirror drawing task AD7 showed some minor improvement. However, on the final attempt after 90 minutes (over 3 days) she made upward of 50 errors. When required to draw the shape inside the star without the mirror, AD7 could do this without any errors, indicating that apraxia was not the reason for her difficulties. AD7 showed some small levels of improvement on the rotary pursuit task indicating some procedural learning skills. However, her rate of learning and overall performance of this task was lower than any other participant in the study.

3.3.2.4.1.2 Experimental Tasks

Figure 19 shows AD7’s performance on all 12 experimental tasks. Figures 23 and 24 show the comparison of a coarsely matched task pair (trained and untrained) for time taken to perform the tasks and errors respectively. Figure 23 shows that the improvements (in performance time) from training are maintained throughout the follow-up period for the trained task, whereas the untrained task shows no clear trend. This pattern is also present in Figure 24, which clearly shows that whilst the trained task was performed without errors during follow-up, the untrained task was performed with errors throughout the follow up period. AD7 showed an overall training effect using the binomial procedural (see Table 7). All six trained tasks showed a reduction in error scores, and four of those also showed a training effect for performance time.
Figure 20. The performance time data for participant AD7 for two tasks: making a phone call (untrained task) and locking the back door behind her (trained task).

Figure 21. The error data for participant AD7 for two tasks: making a phone call (untrained task) and locking the backdoor behind her (trained task).
**Table 9**

**Caregiver Feedback Interview Summary Results**

<table>
<thead>
<tr>
<th>Participant</th>
<th>Behavioural changes</th>
<th>Physical changes</th>
<th>Mood changes</th>
<th>Anxiety about training</th>
<th>Disliked elements</th>
<th>Liked elements</th>
<th>Overall changes during training</th>
<th>Overall changes during follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>AD1</td>
<td>Agitated</td>
<td>None</td>
<td>Irritated on the day</td>
<td>None</td>
<td>AD1 thinks unnecessary</td>
<td>Company</td>
<td>None</td>
<td>Decline is continual</td>
</tr>
<tr>
<td>AD2</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Why doing this?</td>
<td>Unknown</td>
<td>None</td>
<td>Doing more around house, vacuuming, showering and dressing quicker.</td>
</tr>
<tr>
<td>AD3*</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Why doing this?</td>
<td>Interest &amp; company</td>
<td>Improved</td>
<td>Improved</td>
</tr>
<tr>
<td>AD4</td>
<td>C1: None</td>
<td>C1: None</td>
<td>C1: None</td>
<td>C1: Unknown</td>
<td>C1: Unknown</td>
<td>C2: None</td>
<td>C1: None</td>
<td>C2: None</td>
</tr>
<tr>
<td>AD5</td>
<td>None</td>
<td>A bit tired</td>
<td>Improved</td>
<td>None</td>
<td>None</td>
<td>Company</td>
<td>None</td>
<td>OK since caregiver training</td>
</tr>
<tr>
<td>AD6</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Unknown</td>
<td>Doing OK</td>
</tr>
<tr>
<td>AD7</td>
<td>None</td>
<td>None</td>
<td>Cheered up</td>
<td>Slight, temporary</td>
<td>None</td>
<td>Company</td>
<td>Makes bed, locks door</td>
<td>None</td>
</tr>
<tr>
<td>AD8</td>
<td>None</td>
<td>None</td>
<td>Improved, enjoyed sessions</td>
<td>None</td>
<td>None</td>
<td>Sense of purpose</td>
<td>None</td>
<td>Stabilising</td>
</tr>
</tbody>
</table>

* AD3 lived on her own and gave her own feedback.

**Additional comments:**

AD1: Caregiver reported that her agitation related to her personality. That she was never wrong and nothing was wrong. He felt that any intervention challenged her beliefs and this irritated her.

AD2: Caregiver reported that he initially enjoyed training but he got a bit tired after several months.

AD3: Feedback is self-report, no additional comments were made.

AD4: Two caregivers participated in the interview independently. Caregiver one reported that the training was excellent and would be good if it could be ongoing like a caregiver coach. Caregiver two reported that AD4 needed prompting and that she couldn’t initiate tasks.

AD5: Caregiver reported that at first the training seemed inconvenient and frustrating for the caregiver (although not for AD5). This improved as he got used to the training.

AD6: Caregiver reported that AD6 is sometimes OK, sometimes not.

AD7: This caregiver did not have any further comments.

AD8: Caregiver reported that AD8’s memory had stabilised, she seemed calmer now and had begun to tell people she had difficulties with her memory.
3.3.2.5 Caregiver Feedback Interview (refer Appendix XII)

The caregivers reported no adverse events or outcomes from the procedural training, although 1 participant reportedly became more tired in the evening, when she had training that day. The training reportedly irritated another participant (AD1) as she was sure nothing was wrong with her. Although she did not enjoy the training, she did not want to discontinue participation when asked each training day. A variety of positive outcomes were reported by all caregivers ranging from improvements in trained tasks, to enjoyment of the social contact. Table 10 summarises caregiver’s views of outcome.

3.4 Discussion

One of the numerous symptoms accompanying AD is a progressive decline of functional ability in many day-to-day tasks (Reisberg et al., 1990; Teri et al., 1988). The present study asked the following questions: can procedural learning methods be used to effectively retrain impaired tasks of this sort in mild-to-moderate AD? If so, how long do these training effects last?

AD participants performed the self-care and ADL tasks both faster and with fewer errors following procedural training, but showed no such improvement on comparable untrained tasks. Thus, not only was there no speed-accuracy trade off during performance of trained tasks, but the significant findings on both dependent measures provided consistent evidence of improvement following training. Two factors indicated that the improvement did not relate merely to the ongoing presence or attention of the investigator, nor to any other extraneous events. Firstly, the lack of improvement on the untrained tasks and secondly, the absence of significant change in performance between first and last baseline times at any of the multiple baseline lengths. It can therefore be assumed that any subsequent improvement in tasks was due to the intervention. These findings provide strong support for the claim that procedural retraining can be effective in mild-to-moderate AD.

One possible alternative account for the training effect is that the participants were using residual explicit learning abilities to improve performance on the trained tasks rather than procedural learning. While it is not possible to rule out the use of explicit memory for the learning of trained tasks, this is unlikely given the participants’ difficulties in this area. Nearly all participants scored very poorly on the
explicit memory measures used (WMS-R scores) and certainly the WMS-R scores did not correlate with any measure of training success. Consistent with this interpretation was the finding that, when asked, the participants were not able to remember what tasks they were doing that day, nor describe how to complete them. Finally, previous research has found only very limited success with explicit memory training in mild-to-moderate AD (De Vreese et al., 2001). A second explanation of the training results is the placebo effect from receiving an intervention as both participants and caregivers/family members were aware of the training programme. However, as the untrained tasks did not improve, and the participants did not remember which tasks were trained or untrained, the possibility of a placebo effect differentially affecting the trained tasks (and not the untrained tasks) seems unlikely. In summary, the weight of evidence in this study indicates improvement in performance most likely reflects procedural learning.

One limitation of this research is that the researcher carried out the baseline, training and follow-up tests, so measurements were not made blind. It is possible therefore that there was an expectation bias. The researcher attempted to counter this by using outcome measures that were designed to reduce bias as much as possible. The tasks were timed and the numbers of errors were recorded with written description of the nature of errors and any unusual occurrences during the task. For measures of task performance time, there were set criteria for the start of the task (the participant touched the object involved) and the end of the task (the task was complete, or the participant thought the task was complete and stopped working on the task). Judgement as to what constituted an error was made in consultation with an experienced neuropsychologist; who also reviewed a random selection of the decisions for validity. Additionally, random selections of error descriptions were compared to the number of errors by the researcher to encourage reliability. Nevertheless, the data collection still included elements of subjective decision making and relied on researcher judgement and this remains a weakness of the research.

One question of interest was whether, prior to training, the AD participants were performing at a level that was worse than the matched, control participants. If this was a case then did the procedural retraining improve their performance to the level of healthy elderly? Not surprisingly caregivers’ reports on the PSM and IADL scales indicated that the AD group, before training, had more difficulties with self-
care and daily activity tasks, compared to the control group, who scored at ceiling levels. The caregiver-rated scales confirmed that the AD group had functional difficulties, which were easily identified by caregivers and caused clear difficulties in everyday life. In terms of the daily living tasks used in this study, the control group took significantly less time to complete the tasks and made no errors when compared to the AD group at baseline, indicating clearly that the AD participants performed at a lower level. When the control group performance was compared to the AD group performance on trained tasks at post-training, there was no statistical difference between the two groups suggesting the AD performance was not significantly different to normal performance. However, it should be acknowledged that the non-parametric test used is conservative, and ‘no difference’ will be found if just 2 of 8 AD participants perform at the same level as controls. Certainly, the AD group error rate remained higher than the control group (see Figure 8), although numbers of AD group errors on the trained tasks at post-test was not significantly different to the control level. Given these factors we would be hesitant to make the claim that after training the AD group performance on tasks was in the normal range for their age group, although clearly it had improved.

Having demonstrated that training was successful, the next important question was whether these training effects were long lasting, as training would only be clinically useful if the effects endure. The AD participants continued to show the benefits of training for at least 3 months, with improved performance on trained tasks compared to baseline performance, both in terms of time taken and number of errors made. In contrast, the untrained tasks took longer to complete during the follow-up period, although they were not significantly different from baseline at any follow-up point. The mean percent improvement in time taken to complete trained tasks from baseline to post-test was 28%. At 3 months post-training the percent improvement in time taken to perform tasks had reduced to 15%, indicating that some decline in performance had occurred during this follow-up period. Nevertheless, the duration of significant training effects provides strong support for the application of this approach to retraining self-care tasks. This is consistent with the findings of Zanetti et al. (2001), who reported that the benefits of procedural training in AD persist until at least 4 months. They measured a single follow-up point at 4 months post-training, which showed maintenance of the training effect, compared to ‘no change’ for task
performance by a wait-list control group. This study only compared two time points, which does not provide information about the change or rate of change occurring during the 4 month follow-up period. The group design makes it difficult to determine if there was any individual variation between the participants. In addition, the lack of information on change in task accuracy also limits full analysis of training success. Zanetti et al. suggest further research should investigate ‘ability to execute an action’, i.e., making note of error levels, clinical relevance, personal needs, and the maintenance of autonomy in AD patients own homes. These recommendations are addressed in this study and in study two (chapter 4). To date, no other research has examined the issue of training effect duration in an AD group sample.

The group analyses in the present study demonstrated the overall usefulness of the procedural learning method in a group of mild-to-moderate AD participants. In order to evaluate the utility of this approach for clinical application, however, it is important to know how effective training was for individual participants. In other words, how many of the AD participants benefited from the training? And of those that benefited generally, how many tasks were successfully trained?

The binomial analysis of overall training effectiveness for each individual showed that 7 of the 8 participants benefited from training. Note that this statistical method of determining effectiveness contrasts with the predominant method of analysis of single-subject data, which typically involves solely a visual judgement of graphed raw data. The finding that 7 of 8 participants showed significant training effects indicate that the procedural learning method is effective. The 8 members of the AD group varied in terms of premorbid functioning (as assessed by educational/work history and the NART-R) and range of AD severity. That 7 of 8 AD participants benefited from training is all the more notable in light of this variability.

When a more specific evaluation was used to determine how many tasks were successfully trained for each participant, performance ranged from improvement on two of four possible tasks (see participant AD1) to improvement on all impaired tasks (in 4 participants). Judgement of task improvement was based on either performance time improvement, or, error improvement, in tasks not performed at a ‘normal’ level throughout baseline. Notably, when looking at the individual results, performance on
only one of the untrained tasks for one person showed a performance time improvement, and only 2 of the 48 untrained tasks showed a mild error improvement.

Of interest was the 1 AD participant (AD1) who showed no general benefit for training. AD1 was also the only participant who was at times reluctant to do the training, although when asked about her participation each training day, she did not want to withdraw from the study. AD1 denied any functional or memory difficulties (but other more successful participants also had limited insight) and was less willing to do the tasks repeatedly (she complained about tasks and appeared to do the tasks with minimal effort). Although, according to her caregiver she had always complained about not liking or wanting to do various tasks during task performance, even before the onset of AD. It is important to note that the successful repetition of the tasks is a key requirement for the acquisition of skills by procedural learning. Despite participant AD1’s objections to the training, she would not cease the training programme possibly because of the social aspect of regular visits. It is possible that the unwillingness of this participant, at the level of repeated task performance, in some way interfered with procedural learning. It should be noted, however, that despite the lack of general benefit in terms of reduced time taken to perform trained tasks, AD1 did show improvement in terms of error reduction on two of four tasks. The remaining two tasks were performed at a normal level (within control range) during baseline. Both of these factors are likely to contribute to the lack of overall training effect for time taken to perform the trained tasks.

Although 7 of the 8 participants showed a significant training effect overall, some participants nevertheless did better than others. This provides an opportunity to consider an important question: Are there any identifiable predictors regarding whether an individual will benefit from training or not, and if so, how much will they benefit? This is especially relevant given the time consuming nature of the training. The laboratory-based procedural learning tasks (mirror drawing task, and rotary pursuit task) were included in this study in part to ascertain whether or not the AD participants had relatively spared procedural learning skills, and also to ascertain whether performance on these tasks could be used to predict who would benefit from the training.

The AD group (with the exception of two individuals) in this study was completely unable to learn the mirror drawing task. Interestingly, some of the control
participants may also have had difficulty, although this was difficult to assess as 3 refused, 1 showed moderate procedural learning, and the 4 remaining control participants performed well, even on their early attempts. These results contrast with the findings of Gabrieli et al. (1993) who demonstrated intact mirror tracing in mild-to-moderate AD participants. Notably, however, half of the participants that entered that study could not commence the mirror tracing and were not included in the study. The determination that participants ‘could not’ commence training were not clear, did they refuse or could they not begin the task? Gabrieli et al. suggested the reasons for participant failure to learn this task related to apraxia, spatial disorientation or lack of frustration tolerance. Given the findings that only 1 participant showed clear learning trends (a second showed moderate learning) in the present study and half of the participants in Gabrieli et al. did not commence learning, it is questionable whether claims that AD individuals show procedural learning on this task are accurate.

One hypothesis regarding successful learning of the mirror drawing task is that it involves abilities other than procedural learning, such as inhibition or selective attention. These functions can be impaired in AD (Binetti et al., 1996; Lamar et al., 1997). This hypothesis, that inhibition and/or selective attention are involved in mirror drawing, is based on the following analysis: when a mirror guides drawing, the natural impulse or prepotent response is to move your hand in the opposite direction to that required. If a person has difficulty inhibiting this prepotent response they will make many errors. If they are unable to perform at least some errorless sequences then it is not possible for procedural learning to occur, as this requires successful repetitions of the motor skill. One way to test this account of mirror drawing would be to test individuals with frontal lesions and non-frontal (e.g., temporal lobe) damaged stroke participants. If the individuals with damage to prefrontal cortex could not perform this task (despite intact striatal structures), and the other participants could, this would suggest the mirror drawing task is not simply a task of procedural learning.

In contrast to the mirror drawing task, the AD participants showed learning on the rotory pursuit task with increased time on the target over the training, and maintenance of this improvement after a 20-minute break. The results from this AD sample are consistent with the findings of Eslinger et al. (1986) and show that procedural learning for this motor task is relatively preserved in AD. Controls and
those with AD learned at a similar rate, however the overall ability for those with AD was much lower both before, and after, learning this task. In other words, throughout the training of this task the control group was consistently better than the AD group, however, the AD group improved by the same amount. This would suggest that even when ability to perform a motor skill task is impaired, the ability to learn the task procedurally remains. It should be acknowledged, however, that the control group reached ceiling levels of performance before the end of the training period, thus it was not possible to evaluate their full learning potential.

A study involving participants with Huntington’s disease has demonstrated the opposite findings to the current study; the Huntington’s participants had intact ability to perform the mirror drawing task, however they were impaired on the motor pursuit task (Gabrieli, Stebbins, Singh, Willingham, & Goetz, 1997). The authors suggested that different neural circuits might mediate these two types of procedural learning tasks.

Did procedural learning on the rotory pursuit task predict training success? Unfortunately, in these data no such trend was observed. With only 8 participants there needs to be a very consistent relation between the two variables to give a statistically significant correlation. In this study two participants showed the opposite extremes: AD1 was poor at self-care/ADL task training, but good at rotory pursuit learning and performance, and AD7 was good at self-care/ADL training but poor at rotory pursuit learning and performance. One aspect of AD1’s performance which may be important is that she ‘wanted’ to perform the rotory pursuit task (and other screening tasks) to prove that she did not have any memory problems, which may have resulted in her performing to her best ability. She was not motivated to demonstrate her maximum performance on the daily living tasks as she did not believe she had any problems. While this account could be presented to explain AD1’s pattern (i.e., lack of training success appeared to relate to her motivation) it is difficult to develop a ‘logical’ explanation for the results of AD7. Given these examples, the usefulness of the rotary pursuit to predict who may benefit from self-care task training appears limited.

No other significant correlations, which may have indicated potential predictors of success on a procedural retraining programme, were found. However, the expected association between MMSE and the caregiver rated IADL and PSMS
was found to be significant. Lower MMSE scores were associated with increased difficulty with self-care tasks or daily activities, suggesting that as individuals decline (generally indicated by lower MMSE scores) they also lose the ability to do tasks that were previously performed effortlessly. According to the literature, procedural learning is ‘relatively preserved’ in AD, however the assumption in the literature seems to be that this also declines over the course of the disease, just at a slower rate. Note, however, that in this study MMSE scores were not significantly correlated with ability to do the rotary pursuit task or with ability to benefit from procedural retraining of self-care tasks, which is not consistent with this view. Of course the success of training may rely on procedural learning ability, but which is not measured with enough sensitivity by the rotary pursuit task. Alternatively, the ability to detect such a relation may simply not be possible with such a small sample, particularly when some participants were performing normally on some of the tasks to be trained (which may have masked the true level of potential gain from the procedure to relearn impaired tasks). The relationship between the rotary pursuit task (and other procedural learning tasks) and retraining of daily tasks needs further investigation with a larger sample and a training programme more tailored to the needs (and thus the deficits) of individuals.

Overall, procedural retraining was effective in this study using 12 selected tasks. Of interest were issues regarding the nature of the different tasks and their differing potential for successful training. In the binomial analysis of task effectiveness, 10 of the 12 tasks were performed more rapidly following training, while two tasks (putting on shoes and cardigan) took longer to execute after they had been trained. Performance on these two tasks during the baseline phase appeared to be conducted with a speed-accuracy trade off. In other words, very rapid performance was often associated with very inaccurate performance (all participants performed these tasks at baseline). If this observation was correct, accurate performance might be expected to take longer and thus a slower performance of these tasks at post-training may reflect a reduction in task errors. However, this does not appear to be the case for the cardigan task, as there is only 1 participant of 5 who showed an error improvement. On the shoe task, however, 2 participants showed error improvement (1 had normal performance at baseline) while not improving in the time taken to perform the task. This does suggest a speed-accuracy trade off for this task,
prior to training, possibly because during baseline several participants did not do up their shoelaces (some of these participants were not trained for this task). This resulted in higher errors, but also in faster (but incorrect) performance of the task. At follow-up 2 participants showed error improvements by doing up their shoes correctly after training, however, this took longer.

Overall most tasks did not show the effects of such a speed-accuracy trade off, as shown by the binomial results for the effectiveness of training by task. However, one clear example of speed-accuracy trade off occurred for participant AD8 on the task ‘cleaning teeth’, which took her longer to perform following training. During baseline she did not use toothpaste, and only rinsed her mouth. Following training, she correctly included all elements of the task, which consequently took longer to complete. In other words, while in most cases training success can be seen with a reduction in performance time, occasionally an improvement is seen in task performance when it takes longer to perform, as indicated by error improvement. Of course error improvement without any change in time taken also indicates training success. These unique outcomes show different patterns of training success and need to be isolated from the general trends.

One task bought up an issue during training, for some participants. Although the task ‘Setting the table’ showed overall time and error improvements, it was nevertheless a difficult task from the point of view of applying the method of retraining. For some participants, (e.g., AD2, AD7) this task showed improvements but still presented problems during training, and errors were still occurring during follow-up. For the participants who were more successfully trained in this task, their method of setting the table appeared simpler, and they had fewer objects involved when they set the table. Planning is an executive function, which has been shown to be often impaired in people with AD (Binetti et al., 1996; Lamar et al., 1997; Starkstein et al., 1997). In the participants who showed only limited improvement, the task involved a method that was complex and involved planning for what was needed and number of people present (AD participants’ questions included “How many people”, “What’s for dinner”, “What do I need” etc.). The method had to be adjusted to prompt performance during training; participants were told to set the table for the appropriate number of people (usually one or two depending on whether they were living with a caregiver or alone). When they asked what they needed, prompts
were given such as: “You are setting the table for two people for dinner”. The
application of this method is only likely to be successful with the task when it is
simple and does not have a significant planning component.

3.4.1.1 Single Case Example (AD7)

AD7 showed gains from procedural learning for all trained tasks; four showed
improved time and two showed reduced errors, whereas on the untrained tasks there
was generally increased errors and no improvement in performance time.

One of the issues raised in this case study is that of the variability between the
investigator ratings for the ADL scales and the caregiver ratings. For most of the
participants in this study, the researcher was in closer agreement with the caregiver’s
ratings, so in general this may not present a major problem. However, this example
shows the potential for caregiver overestimation of functioning. This not only has
implications for this type of research, but also for caregiver expectations of the
abilities of those with AD. If a caregiver is overestimating the abilities of the person
with AD this may lead to frustration and anger when tasks are not performed
accurately nor to the caregiver’s expectations. These issues are discussed further in
chapter 5.

3.4.1.2 Caregiver Feedback

The caregivers generally reported small or no changes in the behaviour of
their family member. The caregivers did not report any change in initiation of tasks,
or change of motivation levels (with the exception of AD4 who began to ring her
sister again). There was no evidence that the training generalised to other ADL tasks;
the training, therefore, appears to be task specific, although this was not directly
evaluated.

The participants improved on three to six tasks, but these tasks were not
necessarily the ones most relevant for each individual. Therefore it is not surprising
that caregivers did not mention any major improvements in the quality of life for their
family member, especially given the many other issues and difficulties these families
face. However no harm was done to any of these participants and therefore an ethical
basis has been created to continue with further research with a method more focused
on individual needs. Additionally, the majority of family members reported that the
visits were a positive experience for the person with AD, primarily because it provided company and someone for the AD individual to talk to.

### 3.4.2 Assumptions for a Successful Training Programme

During the development of the training programme, three assumptions were made about how training could be conducted in a procedural manner so as to be consistent with current knowledge and theory. These were that the tasks: would be done in the same way each time; would have a limited number of elements; and would have no planning requirements. How well did these assumptions hold up within the context of the research results and relevant literature?

One important component of the procedural training method is exact repetition, with the outcome that the task will be performed in that same way. The generalisability of tasks is theoretically limited i.e., learning to put on a blouse may not generalise to improvements in putting on a dress; however, this was not directly assessed in this study. This study contributes evidence that sameness (i.e., repetitive training of the same task) is effective, however there was no evaluation to see if tasks that are performed variably could be trained. Dick, Hsieh, Dick-Muehlke, Davis, and Cotman (2000) showed that a motor task (tossing a small beanbag at a target) was successfully trained in people with mild-to-moderate AD only when it was trained constantly and repeatedly. This task did not, however, generalise to either a similar task (throwing horse shoes at a peg), or to the same task when throwing sideways, or when the weight of the beanbag was changed for over-arm throws (although underarm throws showed some generalisability). In contrast, healthy controls showed learning with less constant training, and the training effects generalised to both the similar and closely related tasks. This provides evidence supporting the requirement of sameness and the lack of procedural learning generalisability in those with AD.

It was observed during training that participants often do the same task in different ways. One example of this is the setting the table task. For some people this was a simple task. They would set the table for one or two people with the appropriate selection of knives, forks, place-mats, salt and pepper. However, other participants set the table for varying numbers of people, with any range of cutlery, crockery, and condiments. Participants who had a more complex method for this task had more difficulty learning or improving their performance. Therefore the more
simple and constant the task was, the more likely that training would be effective. This implies that simple tasks are better for training, presumably related to the assumption that tasks need to be performed the same way each time, and additionally that the same home environment, position of objects, and daily routine may be beneficial. It has been found that consistent routines and environments help with the orientation of people with AD and reduce levels of confusion (Haitt, 1991; Tappen, 1997). This would suggest that training in people’s own homes is preferable to training in a clinic.

The assumption that simple tasks with limited elements are preferable for training is presumably because they can be performed in an automatic run. This idea is based on literature which found that the complexity of a task played a part in whether it could be performed using only implicit learning (Milner, 1965). If tasks become too long then the potential for interruption is increased. With interruption, it may be that explicit memory would be required to remember what the task was and the point at which the interruption occurred. The present study did not address the issue of simplicity, as no complex tasks were included. However, for some participants on the task ‘setting the table’ the complexity in their method may have limited their ability to learn this task procedurally.

The simplicity of a task also relates to the third assumption (no planning), as simple tasks are less likely to require planning. As previously mentioned, executive function is often affected in AD, therefore the task must be able to be performed without different options or planning requirements. For example, participants should not need to think “How many people will be having dinner with us? What things do I need?” Some evidence for this assumption was seen in the results for individual tasks and participants, and in the literature on impaired executive functioning in AD (Binetti et al., 1996; Lamar et al., 1997; Starkstein et al., 1997).

### 3.4.3 Directions for Future Research

In this study a small number of specific tasks were chosen. It was necessary to have consistency over participants, since we wished to ascertain whether the training method was useful. The tasks were not matched to the needs of the participants and, consequently, some participants had no difficulty with the tasks that were going to be trained (performing at ceiling level, which in this case was a similar
level to controls). Occasionally, the task to be trained was not appropriate to the individual. For example, a 61-year-old male participant (AD2) had never set the table in his life and objected to being trained on this task saying, “This is women’s work”. In this case it was not surprising that the training for this task had a limited effect (that is some error reduction, although errors continued to occur during follow-up). This study, despite the limitation of tasks selection and task inappropriateness, nevertheless showed that the training method is effective. In future studies, it will be possible to select tasks according to the needs of the individual, which will reflect both relevance and level of impairment. If the tasks are important to the person with AD and/or the caregiver then it is more likely that they will be useful if successfully trained. The inclusion of longer follow-up periods would also provide more useful clinical information about the duration of training and the progression of the loss of daily skills in AD.

Although procedural learning improves the ability to perform tasks, this does not necessarily change the ability to initiate tasks. However, given that most individuals with AD live with caregivers, it is likely that prompts will be provided during the course of daily life. During the research period it was not possible to observe the spontaneous performance of these tasks. Interesting questions remain. How were these tasks performed when not observed? Was there any increase in spontaneous performance? How did the participants react if caregivers asked them to do these tasks? Was there any procedural learning from doing these tasks correctly when the researcher was not present?

3.4.4 Conclusion

This study provides evidence supporting the hypothesis that daily living skills can be successfully trained in people with mild-to-moderate AD using the procedural learning method and, furthermore, that these benefits can be maintained over time. The reduction in time taken to perform tasks and the reduction of errors in every-day tasks and self-care skills will have beneficial impacts on people’s lives, as well as on the lives of their caregivers. The results confirm those of Zanetti et al (1997), and previous studies that suggest implicit methods (specifically procedural) are an effective form of learning in people with AD (Butters et al., 1995; Deweer et al., 1994; Gabrieli et al., 1994). Participants with AD showed impaired performance on
memory tasks involving recall and recognition, but showed preserved procedural learning of daily living skills. These findings support theories which propose that at least two memory systems are in operation.

In conclusion, this study suggests that a rehabilitation programme for people with mild-to-moderate AD could be worthwhile. Such a programme would lengthen some self-care abilities and temporarily maintain quality of life. In addition to re-learning some of the tasks that have become difficult for people with AD, these benefits remain for some time, due to the relatively spared nature of procedural learning in AD.
4 Chapter Four

Procedural Learning of Daily Tasks in Alzheimer’s Disease: Study Two

4.1 Introduction

The results of study one indicate that there is potential significant benefit in the use of procedural training to relearn daily living skills for people with mild-to-moderate AD. In the first experimental study the efficacy of the training method was tested using both a group and single subject design in which a sample of AD participants were assigned counter-balanced sets of trained and untrained tasks. Inevitably such a design meant participants were assigned training tasks whether or not they were relevant to their lives, even if they performed within the normal range for any of these particular tasks before training. Despite these strict design features, 7 out of 8 participants showed benefits from training, with improvement in performance maintained over several months. Given these findings one might expect the procedural learning methods to produce significant gains when applied to tasks identified as problematic for an individual.

The following study was designed to test this prediction and, in so doing, simulate a functional rehabilitation programme that would be useful to individuals diagnosed with probable AD. To do this a flexible tailored retraining programme was required, which related directly to the needs of each individual with AD whilst conforming to constraints of the procedural training methods. In so doing this study assesses, more directly, the utility of procedural training whilst continuing to use quantifiable measures of performance, namely time taken to perform a task and the number of errors made. Findings from the previous study demonstrated clearly that untrained tasks did not change as a consequence of procedural training of other tasks, therefore no untrained tasks were included in this study. Instead a series of individuals were trained on those tasks identified as problematic in their lives that conformed to the constraints of the training technique, using a single subject design, multiple baseline across tasks.

In summary, the first aim of this study was to investigate the effectiveness of procedural training methods to train a series of individuals, each on a unique set of
selected tasks showing impairment. The second aim was to determine the duration of the training effects over time. It was predicted that there would be a statistical improvement in task performance when compared to baseline performance and that this improvement would be maintained after training was completed.

4.2 Methods

4.2.1 Participants

Six individuals (five female and one male) between the ages of 69 and 87 (M = 78.333, SD = 6.022) participated in this study. All 6 participants were recruited from the Memory Clinic of North Shore Hospital, Auckland, New Zealand. They all had a diagnosis of probable AD according to NINCDS-ADRDA criteria (McKhann et al., 1984), following a full medical and clinical history, completion of a neurological examination, standard blood and urine analysis and CT scans to exclude other dementias. All 6 participants identified themselves as New Zealand European, although participants from any ethnic group could have participated.

Participants were required to be in the mild-to-moderate stages of AD as assessed by scores on the Mini-Mental State Examination (MMSE) (Folstein et al., 1975). The lower cut-off used for moderate impairment in this study was an MMSE score of 10 out of 30 (Reisberg, 1984). Potential participants were excluded if they had a history of substance abuse, head injury, major cardiovascular accident, psychiatric history or other neurological diseases, or were currently taking neuroleptic medications, as all of these events could potentially cause neurological impairment instead of, or in addition to, AD. Participants were also excluded if they had significant immobility, or hearing or sight loss, due to the difficulties these would create during training. In addition, participants were excluded if, at screening, they were taking any medications for the treatment of AD (e.g., Aricept or Excelon), as these medications could create improvements which would not be distinguished from those resulting from training.

All participants lived at home at the commencement of their training, as living in a residential care facility setting may have limited the performance of self-care tasks (e.g., not being able to access the kitchen, or laundry). One participant lived
independently, 2 participants lived with family and 3 participants lived with their spouse. The demographic characteristics of the participants are presented in Table 11.

Four potential participants were excluded because they were taking Excelon, and another potential participant was excluded due to extensive difficulties with physical mobility.

**Table 1**

**Demographics and Performance on Pre-training Measures of Functioning**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>GA</th>
<th>PB</th>
<th>KW</th>
<th>EW</th>
<th>HM</th>
<th>BP</th>
</tr>
</thead>
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<td>M</td>
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<td>78</td>
<td>87</td>
<td>69</td>
<td>78</td>
<td>82</td>
</tr>
<tr>
<td>Years of Education</td>
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<td>10</td>
<td>11</td>
<td>11</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Years since first symptom reported</td>
<td>2</td>
<td>6</td>
<td>5</td>
<td>4</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>MMSE</td>
<td>16</td>
<td>10</td>
<td>11</td>
<td>14</td>
<td>24</td>
<td>23</td>
</tr>
<tr>
<td>Physical self maintenance scale</td>
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<td>17</td>
<td>17</td>
<td>9</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>Instrumental ADL scale</td>
<td>19</td>
<td>28</td>
<td>29</td>
<td>24</td>
<td>25</td>
<td>11</td>
</tr>
<tr>
<td>Short Token Test</td>
<td>30</td>
<td>&lt;16</td>
<td>30</td>
<td>31.5</td>
<td>25.5</td>
<td>36</td>
</tr>
</tbody>
</table>

**4.2.2 Materials, Apparatus and Measures**

**4.2.2.1 Tests to Evaluate Pre-training Functional Levels**

A brief history of each participant was gained from the participants and their families (see Appendix VII). Pre-training measures included the MMSE, Short-Token Test (Appendix VIII), Physical Self-Maintenance Scale (Appendix IX), Instrumental Activities of Daily Living Scale (Appendix X), and rotary pursuit task (see task descriptions in the methods section of chapter 3). The speed of rotation on the rotary pursuit task remained at 30 RPA as in study one.
An additional pre-training task employed in this study was the Canadian Occupational Performance Measure (COPM) (Law et al., 1991) (see Appendix XV). This involves the researcher asking questions of the AD participant and the caregiver, in the form of a semi-structured interview. This measure elicits descriptions of difficulties in daily living skills, leisure activities, self-care, and employment, with prompting questions for both the participant and their caregiver. The questions focus on identifying daily activities that the person with AD wants to do, needs to do, or is expected to do, in a typical day. Then the researcher asks which of these tasks are difficult for them to do to their satisfaction. When problematic tasks are identified a rating scale is used to determine which tasks are most important (1-10 on importance) to the individual, in terms of impact on their life. Then the same 1-10 scale is used to estimate how well the task is performed and the individuals level of satisfaction with the task (Bodiam, Mew, Fossey, & Chan, 1999; Law et al., 1990; Law et al., 1991; Law et al., 1994).

4.2.3 Procedure

Ethics approval was gained from the Auckland Ethics Committee (Health Funding Authority). Following the gaining of consent (refer Appendix XIII & XIV), AD participants underwent a pre-training session comprising an interview to obtain demographic and biographical information (refer Appendix VII), tests of functional levels (PSM, IADL, MMSE, Short-token test, rotory pursuit task), and the clinical interview for task selection. The COPM measure was used as a starting point for a clinical assessment involving both the AD participant and the caregiver. Some AD participants were unable to provide information on the difficulties that they were experiencing. In these cases information from the family member was used more extensively to decide which tasks might be chosen for training. Other participants, with more insight, suggested tasks themselves. All tasks were chosen with the consent of the participant, and tasks that were important to the participant were given preference. Tasks were only chosen if they were assessed as suitable for procedural training according to the criteria outlined in study one (i.e., tasks must be simple, be performed in the same way each time and contain limited planning elements). Although the COPM produces a ranking of tasks in order of priority, this ranking was not needed to limit the number of tasks trained, as the number of tasks requiring
training did not exceed 13. As a consequence all tasks that were considered problematic and important were trained (if they were suitable for training).

Two participants and their caregivers requested the inclusion of tasks that were not ultimately used for training. The first was relearning to knit (participant GA), which was excluded because of the difficulty and complexity of pattern reading, and potential frontal/planning involved in performance. (In addition the researcher lacked appropriate levels of knitting knowledge.) The second task excluded was ‘setting the table’ (participant KW), due to the nature of impairments of the participant and the potential complexity of the task. The researcher observed the participant’s attempt at setting the table (to decide whether the task could be trained), noticed difficulties with the spatial arrangement of items (visuospatial difficulties), and problems with decisions about the number of items and which items were needed (likely to reflect deficits of executive functioning). It was judged, therefore, that this task did not meet the criteria for a potentially suitable task based upon experience in the first study (see discussion chapter 3).

Table 12 shows the final selection of tasks that were trained for each participant, with the number of tasks selected for trained ranging between 6 and 13.

Following this initial session, the experimental component of the study began (namely implementation of the procedural training programme), using the single subject design, multiple baseline across tasks. This included a baseline phase, a training phase, and a follow-up phase.
Table 2
Tasks Trained for Each of the Six Participants

<table>
<thead>
<tr>
<th>Participant</th>
<th>Tasks</th>
</tr>
</thead>
<tbody>
<tr>
<td>GA</td>
<td>Using labels, answering phone/taking messages, using washing machine, using drier, making tea, making a phone call.</td>
</tr>
<tr>
<td>PB</td>
<td>Washing face and hands, cleaning teeth, putting on a dressing gown with buttons, putting glasses on, turning TV on, closing curtains, turning lights on, opening screen door, answering phone, pegging up a towel on the clothes line, putting away cutlery, putting on car seat belt, opening car door.</td>
</tr>
<tr>
<td>KW</td>
<td>Writing name, making a phone call, tying shoe laces, cleaning dentures, brushing hair, washing face and hands, making bed, putting on a cardigan, doing up buttons on cardigan.</td>
</tr>
<tr>
<td>EW</td>
<td>Doing up jacket zip, making a phone call, using microwave, using washing machine, using drier, cleaning teeth, making a cup of tea.</td>
</tr>
<tr>
<td>HM</td>
<td>Heating wheat-bag in microwave, putting down bath mat, using electric razor, pouring breakfast into bowl, putting video in machine and pressing play, making coffee, putting glasses on.</td>
</tr>
<tr>
<td>BP</td>
<td>Using washing machine, using microwave, using oven, using griller, using heater, using TV (on, off, channel change).</td>
</tr>
</tbody>
</table>

4.2.3.1 Baseline Phase

In accordance with the design, the duration of baseline measurement of tasks varied in length (5 days, 10 days, and 15 days). For individuals doing six tasks, performance on two tasks was measured for each of the three baseline lengths. For those doing more than six tasks, the tasks were spread over the different baseline lengths as evenly as possible. For example, participant PB was trained on 13 tasks. Four tasks were observed for each of the first two baseline lengths (5 days and 10 days), and five tasks were observed for the third baseline length (15 days). The participants attempted the tasks once at each baseline measure. The instructions were repeated as many times as necessary in baseline, but no prompts or help with the tasks was provided.
### 4.2.3.2 Training Phase

Baseline observations for all tasks were followed by a training period of 10 days. The baseline and training period spanned a total of 25 weekdays because of the staggered baseline.

The procedural training method used was the same as that described in study one (see methods section 3.2.2.4.2 for details). Each participant was trained on each task for 10 days, five times each day. The sixth attempt at each task was unassisted with two dependent measures recorded for all tasks: time taken to completion of the task, and accuracy (number of errors). Time was measured from the commencement of the task until its completion. Written notes were kept about the qualitative nature of the errors, or any other factors that may have affected the measurement.

### 4.2.3.3 Post-test and Follow-up Phase

Following the training phase, post-test measures of all tasks were taken at several time points. The first post-test measure followed completion of the last day of training for each task. Follow-up recordings were taken every day the researcher visited, so the first tasks trained have follow-up measures for an extra 10 days compared to the last tasks trained. Follow-up measures were then attempted weekly, or fortnightly after training for as long as possible. The length of follow-up varied for each participant, ranging from 2 weeks to 13 months. The circumstances surrounding duration of follow-up will be described for each individual in the results section.

### 4.3 Results

#### 4.3.1 Tests to Evaluate Pre-training Level of Functioning

Table 11 (see methods section) provides a summary of the performance of each participant on the caregiver questionnaires (PSM, IADL), the MMSE and the Short Token comprehension test. Functional levels varied across the participants, reflecting the stage and severity of the disease in each case.

#### 4.3.1.1 Rotory Pursuit Task

Figure 25 shows the performance of each participant on the rotary pursuit task over 10 trials. All participants increased contact time on target, but to differing
degrees. Three participants showed improvements of at least 10 s contact time over 10 trials for the rotory pursuit task: GA, EW and HM. GA showed steady improvement over the first five trials and this was maintained after a 20 minute break. This improvement continued in the second set but then declined suddenly at Trial 8, although Trials 9 and 10 improved and were above the level of the first set. GA’s sudden decline in performance at Trial 8 may have been due to fatigue or distractibility (before Trial 8 she looked around the room, began to move away and the researcher directed her attention back to the task). EW showed clear improvement over the first five trials and further improvement on Trial 6, after the 20 minute break. Performance stabilised over the second set of trials (6-10), with contact time remaining between 20 s and 23 s. HM showed steady improvement over the first five trials. On the Trial 6 (after the 20 minute break) performance dropped to about the level of the Trial 3 but he continued to improve on Trials 7-9.

![Graph showing contact times for each participant](image)

**Figure 1.** Individual participant contact times with the rotory pursuit task target. Observations occurred over two sets of five trials for each participant (with a 20 minute break between Trials 5 and 6). Each trial lasted 30 s and trials occurred 30 s apart.

The remaining 3 participants had markedly lesser gains on the rotory pursuit task, achieving no more than 6 s contact time after 10 trials. PB showed a low level
of ability and learning on this task with no improvement in the first five trials. There 
was a trend towards improvement in Trials 6-9 (with a maximum contact time of 5 s), 
although this dropped back to zero on Trial 10. KW showed a very small 
 improvement on the first set (improving from 0 s to 3 s) and continued this gradual 
learning trend in the second set of trials (up to 6 s contact time). BP showed little 
improvement in the first five trials and only reached a maximum of 3 s contact time 
on the final attempt of this task.

4.3.2 Procedural Learning of Self-care Skills and Activities of 
Daily Living

4.3.2.1 Analysis of Training Success

Figures 26 - 35 show the data for each participant on each task at each visit, 
including expanded graphs for instances when tasks took in excess of 200 s to 
complete during data collection. These graphs enable the visual evaluation of when 
change occurred in relation to the onset of training for both dependent variables 
(performance time and number of error).

The following procedure (developed in study one, chapter 2) was used to 
provide a method for consistent and objective decisions about the success or failure of 
training at the level of individual tasks for each participant. Using the Sigma-Plot 
package baseline and post-training follow-up data (for the ‘variable task performance 
time’) for each task and each participant were plotted against real time. A linear 
regression procedure was applied to each set of follow-up data with 95% confidence 
intervals (CI) plotted and projected back in time to the baseline period. Figure 21 
(refer chapter 3) shows an example of the 95% CI levels projected back into the 
baseline period. Once this was performed the number of points, in baseline, falling 
above the upper 95% CI was counted and compared to the number that fell below this 
level. Note that if the task could not be performed or competed, it was classified as 
falling above the upper 95% CI. If there was no effect of training it would be 
expected that 97.5% of baseline points would fall below the upper 95% CI level and 
only 2.5% above. If there was an effect of training, baseline points would be more 
likely to fall above the upper 95% CI, reflecting the level and variance of performance 
time after training.
Figure 26 continued. Task 1 involved reading labels out loud to find four items located in four different bedroom drawers. The part of the task which required learning was the reading of the labels to find the items, rather than random searching. Task 2 involved answering a ringing phone and recording a simple phone message on paper. Task 3 involved starting the washing machine. Task 4 involved closing the drier door and turning it on. Task 5 involved boiling the kettle and making a cup of tea with milk. Task 6 was making a phone call from a card with phone numbers and names on it. This involved reading the name then dialing the phone number.
Figure 2. Case GA: Six trained tasks, showing time data (points), and error data (bars). The subsequent figures for each participant follow the same format. The time data comprises the time taken to complete each task, and the error data indicates the number of errors made (maximum 10). If there is no point shown for time data the task was either not completed, or not attempted. The baseline time data are represented with a line plot (if single points occur in the baseline they are represented with a cross). The post-test data point is represented with a circle and the follow-up points are represented with squares. The vertical lines through the graphs show the length of the baseline, and training phases for each task. If time taken to complete a task is greater than 200 s an extended graph is included on the facing page (cont’d on facing page).
Figure 27 continued. Task 1 involved washing of face and hands with soap and tap water. Task 2 involved cleaning teeth with a toothbrush and toothpaste. The toothbrush was prepared for PB, so she was only trained on the actual brushing of her teeth. Task 3 involved putting on her dressing gown and doing up the buttons. Task 4 involved putting on her glasses the correct way up. Task 5 involved turning the TV on/off, with the button on the TV. Task 6 involved closing the curtains using a drawstring.
Figure 3. Case PB: The first six trained tasks, showing time data (points), and error data (bars) (continued on facing page).
Figure 28 continued. Task 7 involved turning on a light at the switch. Task 8 involved opening a sliding screen door. Task 9 involved picking up a ringing phone and saying hello into the correct end of the phone. Task 10 involved hanging a towel on the line using pegs. Task 11 involved putting a knife, fork and spoon away in the cutlery draw in the correct places. Task 12 involved putting on a seat belt in the car. Task 13 involved opening the car door.
Figure 4. Case PB: The second seven tasks (7-13), showing time data (points), and error data (bars) (continued on facing page).
Figure 5. Expanded data for participant KW.
Figure 6. Case KW: Nine trained tasks, showing time data (points), and error data (bars). Task 1 involved writing her name. Task 2 involved making a phone call. Task 3 involved tying shoelaces. Task 4 involved cleaning dentures with toothpaste and toothbrush, and rinsing mouth. Task 5 involved brushing her hair. Task 6 involved washing her face and hands using soap, water and a flannel. Task 7 involved making her bed. Task 8 involved putting on her cardigan. Task 9 involved doing up four buttons.
Figure 7. Expanded data for participant EW.
Figure 8. Case EW: Seven trained tasks, showing time data (points), and error data (bars). Task 1 involved doing up a jacket zip. Task 2 involved making a phone call. Task 3 involved putting on the microwave for 1 minute on high. Task 4 involved turning on the washing machine. Task 5 involved turning on the drier. Task 6 involved cleaning her teeth, and partial denture with toothpaste and brush. Task 7 involved making a simple cup of tea with milk.
Figure 9. Expanded data for participant HM.
Figure 10. Case HM: Seven trained tasks for HM, showing time data (points), and error data (bars). Task 1 involved putting a wheat-bag in the microwave and heating it for two minutes. Task 2 involved placing the bathmat on the floor in front of the shower. Task 3 involved taking the cap off the top of the electric razor, turning it on, applying it to the face, and beginning shaving. Task 4 involved pouring out breakfast cereal into a bowl and then pouring milk in the bowl. Task 5 involved making a black cup of coffee. Task 6 involved putting glasses on.
Figure 11. Case BP: Six trained tasks, showing time data (points), and error data (bars). Task 1 involved turning the washing machine on at the wall, turning it on at the machine and pressing start. Task 2 involved putting the microwave on for one minute on high. Task 3 involved putting the oven on bake, at 250 degrees Celsius. Task 4 involved using a griller oven, turning it on to a set time. Task 5 involved turning on a gas heater. Task 6 involved turning the TV on, changing the channel and turning it off.
As outlined in chapter 3, an arbitrary figure of 70% baseline points above the upper 95% CI was chosen as the criterion for successful training for an individual task. For example, if five of the five baseline measures fell above the upper 95% CI projected back from the follow-up data for time taken to complete the task, then this task would be classified as successfully trained. In contrast, if three of the five baseline measures fell above the upper 95% CI then this task would be classified as unsuccessfully trained. Although arbitrary, the figure of 70% is a conservative criterion for training effectiveness given the results from study one, which found that a mean of 14.9% of baseline points of untrained tasks fell above the upper 95% CI. If the task could not be performed at all during baseline (i.e., worst possibly performance), but was performed in follow-up (even with errors present), the task was judged as successfully trained.

As in study one, error reduction in task performance could also indicate training success. Presence or absence of an error reduction between the baseline and follow-up phases was judged visually using the graphs presented in Figures 26-35. In addition to the judgements of the researcher, an independent rater, experienced in single subject design and visual analysis, was provided with the criteria for the error improvement judgement and classified the error data for each participant and each task. The inter-rater reliability judgement of error improvement was $r = .938$. Given the high level of inter-rater reliability, the initial ratings were used.

Table 13 provides a summary of training outcome for each participant’s performance of each task. It includes the number of observations in baseline that fell above or below the upper 95% CI (projected back from performance in follow-up) for each participant and each task. The first of the two numbers is the number of observations the fell above the upper 95% CI, the second is the number of baseline observations that fell below the upper 95% CI. Tasks judged as successfully trained (70% of the baseline points fell above the upper 95% CI) are denoted with a Y. If there was no improvement (less that 70% of baseline points fell above the upper 95% CI) this is denoted with an N. Normal performance at baseline is denoted with a ‘Norm’. (In this case the researcher suspected ‘making a phone-call’ for participant GA was being performed at a normal level at baseline and when a comparison was made with control group performance in study one GA’s performance did fall within the normal range.) If there was judged to be a reduction in the number of errors made
# Table 3
## Classification of Training Success or Failure for Performance Time and Error Data

<table>
<thead>
<tr>
<th>Task</th>
<th>Participant</th>
<th>GA</th>
<th>PB</th>
<th>KW</th>
<th>EW</th>
<th>HM</th>
<th>BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6/0 (Y, EI)</td>
<td>5/0 (Y, EI)</td>
<td>3/2</td>
<td>4/1 (Y, EI)</td>
<td>4/1 (Y)</td>
<td>5/0 (Y)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>3/3</td>
<td>0/5 (EI)</td>
<td>5/0 (Y)</td>
<td>5/0 (Y)</td>
<td>1/4 (EI)</td>
<td>2/3</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>9/2 (Y, EI)</td>
<td>5/0 (Y, EI)</td>
<td>5/0 (Y)</td>
<td>5/0 (Y)</td>
<td>5/0 (Y, EI)</td>
<td>10/0 (Y)</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>7/4 (EI)</td>
<td>5/0 (Y, EI)</td>
<td>10/0 (Y, EI)</td>
<td>10/0 (Y, EI)</td>
<td>N/A</td>
<td>10/0 (Y)</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>11/6</td>
<td>10/0 (Y, EI)</td>
<td>2/8 (EI)</td>
<td>10/0 (Y)</td>
<td>10/0 (Y)</td>
<td>15/0 (Y)</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>7/9 (Norm)</td>
<td>8/2 (Y, EI)</td>
<td>1/9 (EI)</td>
<td>15/0 (Y)</td>
<td>N/A</td>
<td>15/0 (Y, EI)</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>6/4</td>
<td>12/3 (Y)</td>
<td>5/10 (EI)</td>
<td>N/A</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>9/1 (Y, EI)</td>
<td>1/14 (EI)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>11/4 (Y, EI)</td>
<td>10/5 (EI)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>10/5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>5/10</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>15/0 (Y, EI)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>13/2 (Y)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Total number (across tasks) of baseline observations above/below the upper 95% CI**

43/24

102/28

49/41

54/11

20/5

57/3

**Summary**

<table>
<thead>
<tr>
<th>Task successes:</th>
<th>3 (2Y/EI; 1EI)</th>
<th>10 (8Y/ EI; 1Y; 1EI)</th>
<th>8 (1Y/EI; 3Y; 4EI)</th>
<th>7 (2Y/EI; 4Y; 1 EI)</th>
<th>4 (1Y/EI; 2Y; 1EI)</th>
<th>5 (1Y/EI; 4Y)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Task failures:</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>3 N/A</td>
<td>1</td>
</tr>
</tbody>
</table>

**Task Normal tasks:**

<table>
<thead>
<tr>
<th>Task successes:</th>
<th>3 (2Y/EI; 1EI)</th>
<th>10 (8Y/ EI; 1Y; 1EI)</th>
<th>8 (1Y/EI; 3Y; 4EI)</th>
<th>7 (2Y/EI; 4Y; 1 EI)</th>
<th>4 (1Y/EI; 2Y; 1EI)</th>
<th>5 (1Y/EI; 4Y)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Task failures:</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>3 N/A</td>
<td>1</td>
</tr>
</tbody>
</table>

**Key:** EI = errors improvement. Y = training success with time. N = no training effect. Norm = within normal range.
in the follow-up phase, success is denoted with an EI (error improvement). Table 13 also includes the total number of tasks that showed either improvement in performance time (Y), and/or number of errors (EI) for each participant.

4.3.2.2 Summary of Each Single Case

4.3.2.2.1 Participant GA

GA was a 76-year-old Scottish immigrant, who lived with her daughter’s family. GA had been diagnosed with AD for 2 years, however, her memory problems may have been present earlier. Her MMSE score was 16/30 at baseline. GA appeared to have good comprehension skills (Short Token Test score of 30). Although GA scored well on the PSM (6), she had some difficulties with daily tasks as reflected by the IADL score (19). Following administration of the COPM, six tasks were jointly agreed upon for training by the researcher, GA and her daughter (see Table 12).

Figure 26 shows GA’s results on each of the six tasks. Visual analyses of the graphs indicate that three tasks (using labels to find objects, using the washing machine and using the drier) show clear changes coinciding with the onset of the training intervention, which occurred following different baseline lengths. The changes evident at intervention occur for both time taken and errors made. Note, however, that the task ‘using the drier’ did not meet either criterion for training success.

Overall, as can be seen in Table 13, GA showed an improvement in three out of five (60%) tasks for either time or error improvement (Figure 26, Graphs 1, 3 and 4) when comparing her performance at baseline and follow-up. The sixth task ‘making a phone call’ was performed at a normal level in baseline and therefore not included in this calculation. Only two tasks (using labels and using the washing machine), however, showed a training effect for performance time when using the criterion 70% of the baseline points falling above the upper 95% CI (derived from follow-up performance). Both of these tasks also showed an error improvement, as did one additional task (using the drier). Two other tasks (Graphs 2 and 5) showed no real change in time or errors. The task ‘Making tea’ showed a trend towards a decrease in time taken at follow-up, but this change was not of a magnitude to meet the criterion for successful training.
The changes that occurred in the three successfully trained tasks were maintained throughout the 7 month follow-up period. The follow-up period ended when GA was placed in day-care and prescribed haloperidol to decrease her distress while there. Although this improved GA’s mood, she appeared to become less coordinated and had greater memory and reality orientation difficulties. Her daughter enrolled GA in day-care because of her mother’s loneliness during the day (when the family was at work), which was resulting in GA becoming regularly tearful. In addition GA’s family had increasing worries about her safety, for example, opening the door to anyone or leaving a stove element on.

4.3.2.2.2 Participant PB

PB was a 79-year-old female New Zealander of European decent, who lived with her husband. She had a six-year history of increasing forgetfulness. PB was the lowest functioning participant at baseline. She was in the moderate stages of AD at the commencement of training (October 1999), as defined by her MMSE score (10/30), level of functioning (PSM, IADL), and comprehension difficulties. PB scored very poorly on the Short Token Test (16/36) suggesting she had considerable difficulty with comprehension. She could follow the simple instructions such as “touch a red square”, but when the third instructional element of size was added she could no longer follow the instructions.

Both PB and her husband judged 13 tasks as problematic and important; all of these tasks met the criteria for retraining (see Table 12). As can be seen in Figures 27 and 28, nine tasks show clear changes (five in number of errors, one in performance time and three in both performance time and errors) coinciding with the onset of the intervention, which occurred following different baseline lengths. Tasks that did not change at the onset of intervention included ‘closing curtains’, ‘turning on lights’, ‘answering phone’, and ‘opening the car door’.

As shown in Table 13, when PB’s performance at follow-up was compared with baseline performance, she showed an improvement in 10 of 13 (77%) tasks. Nine tasks showed an effect of training on performance time when using the criterion for training success of 70% of baseline time points falling above the upper 95% CI level derived from follow-up performance. Of these nine tasks, eight also showed a decrease in the number of errors made during performance, with one additional task also showing an error improvement. These improvements were noticed and
commented upon by PB’s husband and a home-help worker who assisted PB with showering and personal hygiene.

Three tasks did not meet the criteria for training success for either performance time or error improvements. Although time taken to perform the task ‘turning on the light’ reduced, this reduction was not large enough to show a significant result. ‘Hanging out a towel on the line’ did not reach the criterion for training success, but for several months the pegs went the right way up (although other errors continued to occur); this task declined over follow-up and could not be completed by the participant at the last six follow-up visits. The task, ‘sorting cutlery’, also did not reach criteria for training success for either performance time or error improvement. However, over the first 7 weeks of follow-up visits the cutlery was sorted in the correct compartments (i.e., for 7 weeks after training the task could be performed well), however, this task declined over follow-up and could not be performed at all by the participant in the last four follow-up visits.

In summary, results for this participant showed that the procedural retraining methods increased the ability of the performance of most trained tasks, and that these improvements, whilst declining slightly in some tasks, remained in other tasks over an extensive follow-up period.

Participant PB was followed up weekly (although some weeks were missed) for 13 months; this was ceased at the end of the research collection period. At baseline PB could not open her sliding screen door to go outside, she would push or pull rather than slide the door. After training she could perform this task at an almost ‘normal’ level even 13 months after training. Most of the task improvements were maintained for many months, several tasks showed a decline towards the end of the 13 month follow-up period (‘cleaning her teeth’ and ‘putting on dressing gown’ were maintained during follow-up but began to decline at the very end of the follow-up period). In contrast, ‘putting on a seat belt’, was originally successfully trained, but declined over the follow-up period and could not be completed during the last three follow-up visits. This task could not be performed at all during baseline, but the task was completed several months into the follow-up period (variably with and without errors).

PB showed a clear decline in functioning during the last few months of follow-up, three tasks could no longer be performed, and daily living skills in general
were becoming difficult. After the follow-up visits concluded her husband managed her care for a further 6 months and then she was moved into a dementia care facility.

4.3.2.2.3 Participant KW

KW was an 87-year-old New Zealand European woman who lived with her husband. She had a 5 year history of memory problems, and her MMSE score was 11/30 at baseline. KW had good comprehension skills (Short Token Test score of 30), although she had difficulty with self-care tasks (PSM score of 17), and tasks of daily living (IADL score of 29). After the administration of the COPM, nine tasks were jointly agreed upon for training by the researcher, KW and her husband (see Table 12). It was KW’s husband who described the tasks KW was having difficulty with, as KW had limited insight.

Figure 30 shows KW’s results for each of the nine tasks. Six tasks showed clear changes coinciding with the onset of the training intervention, which occurred following different baseline lengths. Two tasks ‘putting on shoes’ and ‘doing up buttons’ showed a change in performance time, whilst three tasks ‘brushing hair’, ‘washing face and hands’, and ‘putting on a cardigan’ showed change in the number of errors, and ‘cleaning dentures’ showed a change on both measures. Two tasks, ‘making the bed’ and ‘doing up buttons’ did not show a clear improvement with the onset of the intervention, and a third task ‘writing name’ did not show change at any point.

As shown in Table 13, when KW’s performance at follow-up was compared with baseline performance, she showed a training effect for eight out of nine (89%) tasks. Four tasks showed improvement in performance time (applying the criterion of 70% baseline points falling above the upper 95% CI derived from follow-up performance), one of which also showed an error improvement. A further four tasks showed an error improvement only. The task that did not show any improvement with either of these two measures, ‘writing her name’, improved in quality during training (writing was more even, letters clearer), but declined over the follow-up period.

KW was followed up for a period of 10 months. The changes that were made in seven of the eight successfully trained tasks were maintained throughout this 10 month period. KW was unable to perform the eighth task ‘making the bed’ after 5 months. The tasks ‘tying shoes’, ‘making a phone call’ and ‘cleaning her dentures’
also showed some decline in the last 3 months of follow-up, but still were performed better than baseline levels.

Follow-up assessment of KW ceased at the end of the research collection period. Following this KW continued to be cared for at home, although some home help was sought to help wash and put KW to bed, as she had begun to have difficulty with task initiation, which proved difficult for her husband. KW died several months after the conclusion of follow-up visits.

4.3.2.2.4 Participant EW

EW was a 69-year-old New Zealand European woman who lived with her stepson. EW had a 4 year history of memory problems, and her MMSE score was 14/30 at baseline. EW had good comprehension skills (Short Token Test score of 31.5), although she had minor difficulty with self-care tasks (PSM score of 9), and difficulties with daily living tasks (IADL score of 24). After the administration of the COPM, seven tasks were jointly agreed upon for training by the researcher, EW and her niece (one of her two caregivers) (see Table 12). EW’s niece provided most of the information about tasks that were difficult for EW, as EW had limited insight.

Figure 32 shows EW’s result on each of the seven tasks. Visual analyses of the graphs indicate that all tasks showed clear changes coinciding with the onset of the training intervention across all baseline lengths. Three tasks ‘making a cup of tea’, ‘zipping up jacket’, and ‘using the washing machine’ showed a change in both performance time and error data, the other four tasks showed a change in performance time only.

As shown in Table 13, when EW’s performance at follow-up was compared with baseline performance, she showed a training effect for all seven (100%) of her tasks. Six tasks showed improvement in performance time (according to the 70% criteria based on baseline points above the upper 95% CI), two of these six tasks also showed an error improvement. The final task showed an error improvement. Three of the tasks were not performed at all during baseline; EW would look at the items but did not commence the tasks.

Participant EW was followed up for 2 months, the training effects for all seven tasks were maintained throughout the follow-up period. EW was doing well with the study throughout the research period, however, she was found wandering on three occasions, two of these times on the motorway verge. This prompted her family to
accommodate EW in a full time dementia care unit. As this unit did not allow residents access to the kitchen, laundry or phone facilities, the follow-up of this participant was ceased at this point. EW adjusted to her new environment, after an initial period of distress.

4.3.2.2.5 Participant HM

HM was a 78-year-old New Zealand European male who lived with his wife. HM had a 5 year history of memory problems, and his MMSE score was 24/30 at baseline. HM had some difficulties with comprehension skills (Short Token Test score of 25.5), although no major difficulties with self-care tasks (PSM score of 7). He did, however, have many difficulties with ADLs (IADL score of 25). After the administration of the COPM, seven tasks were jointly agreed upon for training by the researcher, HM and his wife (see Table 12). Tasks were mostly chosen by HM, although his wife also had several suggestions.

Figure 34 shows HM’s results on each of the seven tasks. Visual analyses of the graphs indicate that six tasks showed clear changes coinciding with the onset of training across the different baseline lengths. Two tasks ‘heating wheat-bag in microwave’, ‘putting video in machine and pressing play’ showed a change in performance time, and four tasks ‘putting down bathmat’, ‘using electric razor’, ‘pouring out breakfast cereal and milk’, and ‘putting on glasses’ showed a change in both performance time and error data.

Unfortunately HM had a truncated follow-up period (no more than 2 weeks). As shown in Table 13, he showed an improvement on the three tasks that were evaluated into the follow-up phase. Two of these three tasks showed an improvement in performance time based on the criterion of 70% of baseline points falling above the upper 95% CI, one of these tasks also showed an error improvement. The third task showed an error improvement. In addition, the task ‘putting on a video’ also met the criterion for successful training as the task was completed at two follow-up points, but not at all during baseline (therefore the baseline points were judged as falling above the upper 95% CI). However, overall even these results are inconclusive due to the short follow-up period of no more than 2 weeks. Although training effects were seen, it is not possible to predict whether these would have been maintained into a longer follow-up period. Three tasks could not be evaluated in the follow-up phase due to lack of data.
Three tasks were followed up for two weeks: the ‘wheat-bag’ task showed improvements over training, but declined in the final two visits. The ‘electric razor’ task was only completed three out of five times in baseline but was consistently performed well in the follow-up phase. ‘Putting the bathmat on the floor correctly’ could be performed well by HM during follow-up testing i.e., it appeared to have improved due to training. However, when doing this task independently his wife reported he created elaborate towel formations covering the entire bathroom floor (a new behaviour). Two tasks had only two follow-up points. The task ‘putting on the video’ was completed during these two follow-up points. The task ‘pouring breakfast cereal and milk into bowl’ improved over training, but insufficient follow-up measures were conducted to determine an outcome.

Participant HM did not have follow-up data collected for as long as was planned. Follow-up measures of tasks where taken daily whilst training continued on other tasks, so while there are seven follow-up points (for three tasks), these only spanned 2 weeks (3 days were missed due to HM feeling unwell). HM completed training on two further tasks and had two days of follow-up (Monday and the following Friday). Training was not completed for the final two tasks. HM declined during the last two days of the training/follow-up testing, after being prescribed anti-androgens for a recurrence of testicular cancer (which was believed to be controlled some years earlier). His cognitive decline continued when he was placed in a dementia care unit whilst his wife underwent surgery. He was admitted to hospital 2 weeks later due to declining health, and the presence of blood in his urine. Aggressive behaviour and confusion facilitated his continued care at the hospital. HM went into a vegetative state and died after 5 weeks in hospital. His CT scan did not show any abnormalities further than those associated with AD, and his cause of death was thought to be cancer related.

4.3.2.2.6 Participant BP

BP was an 82-year-old New Zealand European woman who lived independently in a cottage in a retirement village. The village management provided some cleaning support, and nearby health services. BP had a 1 year history of memory problems, and her MMSE score was 23/30 at baseline. BP had good comprehension skills (Short Token Test score of 36), and had no difficulty with self-care tasks (PSM score of 6, completed by her daughter). She did have some
difficulties with ADLs (IADL score of 11). After the administration of the COPM, six tasks were agreed upon by the researcher, BP and her daughter (see Table 12). These tasks all involved the use of appliances, which she was finding increasingly difficult to use.

Figure 35 shows BP’s results for each of the six tasks. Visual analyses of the graphs indicate that five of the six tasks showed clear changes coinciding with the onset of training at each of the different baseline lengths. All five showed a change in performance time, one task ‘TV on/off’ also showed a change in number of errors.

As seen on Table 13, when BP’s performance at follow-up was compared with baseline performance, she showed a training effect for five of six (83%) tasks. These five tasks showed an improvement on performance time applying the criterion of 70% baseline points falling above the upper 95% CI level derived from follow-up performance. One of these tasks also showed an improvement in the number of errors. The microwave task was the task with the least difficulties at baseline; the results showed an error in baseline, but no errors in follow-up, this change is too minor to be considered significant. In four of the other tasks, BP did not physically attempt the tasks during baseline. She reported that she did not know what to do, and did not know where to start. She would occasionally touch the appliance, then remove her hand and not attempt the task. This occurred on twelve occasions for the TV task, although on three occasions during baseline she did attempt the task, with several errors on each occasion.

Participant BP was followed up after the official end of the research period to increase the length of the follow-up to 5 months. For the five tasks that showed improvements in either performance time or number of errors, the improvements were maintained throughout the 5 month follow-up period, although occasional errors occurred with the task ‘turning on the heater’. At the time of writing BP continued to live independently, with home support for housework, and family support with shopping.

4.4 Discussion

Four of the 6 participants showed training effects for at least 77% of the tasks they were trained on. GA showed an effect of training for 60% of her trained tasks. Additionally, HM may have shown training effects however his results were inclusive
due to the short length of follow-up. For the 5 participants that did continue follow-up testing, the tasks that showed an effect of training maintained that improvement throughout follow-up for at least 2 months and in four cases for substantially longer. For example, PB initially showed a training effect for 10 of her 13 tasks. Seven of these tasks were maintained throughout the long follow-up period.

The results of this study provide an evaluation of a training programme tailored to individual needs. These results showed improvements for impaired tasks for all participants (to varying extents). The tasks that improved showed measurable improvements in both performance time and/or error data. Some tasks that were not able to be performed at all in baseline were successfully learned by these participants (turning on the TV and putting on a seatbelt for PB; using the phone, microwave and cleaning her teeth for EW; using the microwave and putting a video in the machine for HM; and using the washing machine, oven, a new griller, and gas heater for BP). Given the extent of pre-training impairment demonstrated by these participants on these tasks, the successful outcomes provide overwhelming evidence that people with AD can learn using this procedural method. It also provides support for the hypothesis of Knopman (1991) that even when tasks are lost from procedural knowledge they can be relearned using a procedural learning method. Furthermore, this learning can be retained in procedural knowledge for several months.

Two extraneous variables may have impacted upon the results: firstly, any additional attempts at training by caregivers; and secondly, ordinary daily practice of tasks by the AD participants (unknowingly). As far as the researcher was aware, no additional training by caregivers took place, however, if this did occur then additional training may have strengthened any training effects. In terms of participants performing the tasks outside of the follow-up visits, this is highly likely as the tasks were a normal part of daily life and the effects of these additional performances can not be measured. The multiple baseline design clearly showed, however, that if there was change (in performance time or number of errors), this change occurred, or began to occur, at the point coinciding with the onset of training, regardless of the baseline length (5 days, 10 days or 15 days). This finding was true for all six participants. In total 37 of the 40 tasks that showed a change, did so at the onset of training (including the three tasks for HM that were not able to be evaluated at follow-up). This indicates strongly that the changes were a result of the training, and not extraneous variables.
The use of the methodology for classifying training success provided an objective alternative to judging training effectiveness by visual analysis of graphed data alone. In study one we compared AD participants to control participants, as well as trained to untrained tasks within a participant, which enabled additional analyses not possible in this study. The criterion for judging training effectiveness for individual tasks in this study was the same as that used in study one, namely more than 70% of baseline points falling above the projected upper 95% CI (generated from the linear regression of follow-up data points and projected back in time to the baseline). This figure is considerably higher than the mean of 14.9% of baseline points of the untrained tasks that were above this CI in study one.

PB’s results provide an excellent illustration of what is possible for an individual with moderate AD. PB’s level of functioning was very low compared to the other participants at the beginning of training yet she showed considerable training effects. Although some of the tasks selected for training were simple, the improvements impacted positively on her quality of life and her caregiver noticed the difference. These improvements gradually declined and some of the trained abilities were lost over the course of the year, although some of the simple tasks remained better than baseline even after a year. For example, the task ‘opening a sliding door’ proved to be the most successfully trained task in this study. After training PB could perform this task at an almost ‘normal’ level, which was maintained at 13 month follow-up. That tasks can be maintained in procedural knowledge over a 1 year period has significant implications for the application of procedural learning in AD, i.e., procedural training is worth undertaking as it can last for a considerable length of time.

The observation of the loss of some tasks from procedural knowledge over the course of time contributes knowledge to theories of procedural memory. Two participants (PB and KW) showed some decline in their tasks over long follow-up phases (13 months and 10 months respectively). For PB, tasks that declined over the year, not surprisingly, were more complex, for example, tasks that required spatial orientation (hanging out a towel with the pegs up the wrong way, answering the phone but holding the receiver upside down, attempting to put the seat belt clip in the buckle sideways) or cognitive procedural ability (inability to sort cutlery by category). In contrast, the simple motor actions (sliding a door, turning on a light...
switch, pulling the curtains, pushing the TV on/off button, opening the car door) were retained. KW was the only other participant who showed decline in her tasks, only one of these tasks showed extreme decline: making the bed. This task may have possibly became difficult due to the elements of spatial orientation, as KW pulled sheets down, put pillows in strange orientations, and six months into the follow-up period she no longer attempted the task.

Although there were a small number of participants in this study, the results of this single subject design replicated over 6 individuals in combination with the findings in study one, demonstrate that people with mild-to-moderate AD are capable of learning daily living skills with procedural learning methods. The range of the MMSE, IADL and PSM scores of participants in this study are similar to those of participants in study one. The Short Token Test scores were lower for 2 participants in this study, which may potentially have presented difficulties during training, especially with understanding simple instructions. This doesn’t appear to have been the case, however, as PB, the most impaired individual on all pre-training function tests, including the Short Token Test was still successfully trained. In this case, a score indicating moderate impairment on the Short Token Test did not predict poor response to this training method.

Task selection was based on a structured clinical measure the COPM, complimented with a clinical interview which helped elicit more tasks, and identify which of these were important. The COPM acted as a useful starting point for identifying tasks, although additional questioning and discussion about difficult tasks with both the caregiver and person with AD was found to be necessary.

The rotory pursuit task was included for the purpose of assessing motor procedural learning in the participants and to investigate whether this would predict level of benefit from procedural training. The participants showed a range of abilities on the rotory pursuit task. Three participants showed clear procedural learning effects, but 3 participants (PB, KW and BP) showed very limited procedural learning on this task. These three participants, however, demonstrated a clear benefit from procedural training on daily living tasks despite their poor performance on the rotor pursuit task. The findings of these 3 participants once again indicate that failure to show a clear learning trend on the rotory pursuit task does not predict inability to learn daily living skills using the procedural training method.
The overall outcome of the participants in this study (with both improved task performance time and/or error improvements) provides strong support for the usefulness of a training programme. The post follow-up outcomes, however, also reflect the progressive nature of AD, and the multiple difficulties that families face (in addition to daily self care issues), as even the most positive of outcomes declined over the 18 month course of the degenerative disease. The results suggest, however, that within a 2-12 month time period the affects of training are relatively well maintained. It is possible that ‘refresher’ or ‘booster’ training sessions may extend the effects of training, although this was not evaluated in this study.

4.4.1 Conclusion

This study was designed to simulate a functional rehabilitation programme that would be useful to AD individuals by testing a tailored retraining programme, which related directly to the needs of each individual with AD. In doing this the potential utility of procedural training can be seen in the ability of participants to learn tasks that were specifically problematic for them, with the procedural learning method. The single subject design, multiple baseline across tasks, was able to provide clear patterns of improvement seen both visually and with judgement of training success (performance time or error improvement). The improvements in task performance were maintained after training was completed, and in some cases for some tasks this was maintained at a level higher than baseline for many months. Given the degenerative nature of AD this provides strong evidence for the effectiveness of this intervention.
5 Chapter Five

Reducing Distress and Aiding Communication in the Care of People with Alzheimer’s Disease: Procedural Learning as a Model

5.1 Introduction

As people with AD progress from the mild to the moderate stages of the disease process there are often increased behavioural problems, such as aggression (Haupt, Kurz, & Jaenner, 2000), increased cognitive difficulties, such as language difficulties (Bayles, Tomoeda, Cruz, & Mahendra, 2000), and an increased occurrence of emotional distress (intense acute sadness, fear, or anxiety (Lee, Strauss, & Dawson, 2000)). Emotional distress and behavioural problems have often been attributed to precipitating factors such as misunderstandings with caregivers, frustration from failure to perform tasks, or fear related to confusion about the environment (Richter, Roberto, & Bottenberg, 1995; Somboontanont, 2001).

There are many contributing factors to emotional distress, including factors within the person with AD, factors within the caregivers, and factors relating to interactions between them, specifically the communication difficulties between the caregiver and the person with AD. Factors related to the person with AD include disease-related problems (such as mood changes, memory problems, language problems, behavioural problems, and problems with ADL’s), and resultant communication problems with their caregiver. The caregiver factors cited in the literature include caregiver burden, grief, depression, ineffective interventions (due to lack of effective knowledge about AD), and communication problems due to the changing needs of the person with AD and the caregivers difficulty detecting and responding to those changes. The factors associated with both AD and caregivers contribute to interactional difficulties particularly with communication, which are frequently listed among the top stressors in surveys of caregiver concerns (Gitlin et al., 2002; Ripich, Ziol, & Lee, 1998; Vetter, Steiner, Kraus, Kropp, & Moeller, 1997; Williamson & Schulz, 1993).

Several interventions have been found helpful for reducing AD distress. Interventions involving people with AD usually focus on environmental modification,
medication, and reassurance. Interventions involving the caregiver include support groups, counselling, education, and learning effective methods of communication. The focus of this chapter is on interventions involving the interaction between the caregiver and person with AD to reduce distress; these interventions include communication and behavioural management techniques.

The procedural learning method, outlined in chapter 3, is a method used to retrain self-care tasks. This method involves a number of components which may also assist effective communication between caregivers and individuals with AD. For example, the procedural method involves breaking tasks into manageable steps, giving only one instruction at a time, and not putting demands on the explicit memory of the person with AD. Using this method, individuals with AD can follow instructions without becoming confused or distressed.

This chapter will review some of the factors relating to distress in AD, and the methods used to assist distress and improve communication. The procedural learning method will then be presented as a model for effective communication for caregivers. It is argued that the procedural learning method may be useful to reduce AD distress and caregiver burden, and to improve quality of life (both in terms of improving daily living skills, and providing an example of effective communication). It will be suggested, in this chapter, that a multifaceted approach to care be applied when addressing the many different needs of individuals with AD and their families.

**5.2 AD Factors Contributing to Distress and Communication Difficulties**

Many cognitive and behavioural changes occur in AD and these can result in lower functional abilities. Changes in behaviour and mood can appear to create a new personality in the individual with AD (Jacomb & Jorm, 1996). This can be very difficult for family to accept, as they often feel the changes equate to the loss of the person they once knew.

There are many behavioural and emotional problems in AD, some of which may relate to levels of distress, for example, aggression (Deutsch & Rovner, 1991; Devanand et al., 1992; Eker & Ertan, 2000; Harwood, Ownby, Barker, & Duara, 1998; Hwang, Yang, Tsai, & Liu, 1997; Savorani, Vulcano, Boni, Sarti, & Ravaglia,
Incidents of verbal and physical aggression by people with AD often have clear precipitants. Somboontanont (2001) found that during showering agitation and assaultive behaviour were not uncommon amongst individuals with AD at a residential care facility. Negative communications by staff, invalidation, disrespectful speaking, restraint, or being hurried etc. were reliable precipitants of verbal aggression or threats. Physical assaults were precipitated most commonly when staff members sprayed individuals with AD with water without warning, tried to wash their hair or wash their genital areas. In almost all instances, aggressive outbursts had a clear precipitating event.

In a number of studies depression in AD was found to be highly correlated with reduced ability to perform ADLs (e.g., Lyketsos et al., 1997; Reifler, 1996). Decline in the performance of ADLs also resulted in an increase in the amount of care needed for the person with AD (Galasko, 1998). Activities that appear simple to the fully functional person can be infinitely complex for a person with AD, and consequently frustrating, as often if just one step in a series is not completed then a task cannot be completed (for example, a hot cup of tea cannot be made unless you boil the kettle).

In one longitudinal study a group of 109 individuals, at approximately the same stage of AD, were followed over a five-year period (Newens, Forster, & Kay, 1995). Five years after diagnosis 53% remained in their own homes, and the others were in residential care. These two groups had equivalent MMSE scores, however, the group in permanent residential care showed a decline in their ability to perform ADLs (prior to residential care placement) relative to the group who remained at home. In addition to decline in ADL ability, the onset of incontinence and poor relationships with caregivers were the two greatest predictors of who was placed in residential care (Newens et al., 1995). This demonstrates not only the importance of maintaining ADL ability but also the importance of maintaining positive interactions between caregivers and their family member with AD.

Communication difficulties between the person with AD and their caregiver commonly lead to distress for both individuals. These difficulties are in part due to the language deficits and semantic knowledge impairments that commonly occur in AD, sometimes during the early stages, but almost universally in the moderate-to-severe stages of AD (Faber-Langendoen et al., 1988). In the moderate stages, there
are fewer intact sentences used in speech, although sentence meanings may still be communicated (Harper, 2001). There are also an increasing number of incorrect word substitutions (e.g., ball for bag) and comprehension difficulties. Unsure statements such as ‘I don’t know’ increase over time, as questions become more common, and AD speech contains less and less information units (Ripich, Carpenter, & Ziol, 2000). In addition, repetition of phrases may also be common (Appell et al., 1982; Cummings et al., 1985; Murdoch et al., 1987). These repetitive sentences and questions can become very frustrating for caregivers.

5.3 Caregiver Factors Contributing to Distress and Communication Difficulties

In addition to deficits associated with AD itself, caregiver factors also contribute to AD distress and communication problems, reflecting the intensive nature of the relationship between the caregiver and the person with AD. Caregivers commonly experience disturbances in both their physical and psychological health (Bell, Araki, & Neumann, 2001; Burns & Rabins, 2000), which may impact on the provision of care for the person with AD, and the quality of their interactions. Stressful interactions between people with AD and caregivers may be, in part, due to caregivers’ failures to understand the nature of communication difficulties, and unrealistic expectations of the person with AD in many areas (self-cares, behaviour, memory, communication etc.) (Ripich et al., 1998). Rose, Stauss, Neundorfer, Smyth and Stuckey (1997) found that caregivers with low distress ratings were more likely to use coping strategies, such as ‘acceptance’ of problematic AD behaviours, and problem-solving techniques. The problem-solving techniques involved efforts to manage or alter stressful situations in a practical manner. In contrast, caregivers with higher stress levels (as judged by self-report and higher cortisol levels), and lower mood, were more likely to make personal attributions of negative events to the individual with AD, and more commonly believed that these events were within the control of the person with AD. In addition, caregivers that were more actively critical toward the individual with AD (i.e., used high levels of criticism during interactions) experienced higher cortisol and self-reported stress levels. In contrast, caregivers
with greater warmth towards their family member with AD had the opposite stress association, and reported fewer conflictual interactions (Rose et al., 1997).

Professional caregivers have been found to accept aggression and distress as part of the disease process, whereas family caregivers are more prone to feelings of helplessness and despair (Richter et al., 1995). If a caregiver is experiencing high levels of depression, grief or burden from caring for a family member with AD it is likely that communication skills will be negatively impacted, and the quality of interaction with the person with AD will decline. Caregivers often experience decline in their physical health, financial security, marital status/relationship, social interaction/support and satisfaction with life (Guerriero Austrom & Hendrie, 1992). These factors are important to consider, as Coen et al. (1997) found that the quality of the relationship between the caregiver and the person with AD related to the caregiver’s mental health.

Caregivers experience multiple losses throughout the course of AD. Feelings of immediate grief are common due to the loss of intimacy and the mutual aspects of the relationship (Garner, 1997). The caregiver experiences a shift in the status of their relationship with the person with AD, moving from a role as spouse or child to one where they are responsible for the AD person’s wellbeing. This caregiver responsibility changes the role and nature of a once intimate relationship (Garner, 1997). Caregivers often feel that their lives now centre on the needs of the person with AD (Loos & Bowd, 1997).

### 5.3.1 Caregiver-related Interventions

Caregiver knowledge of stressors, positive coping strategies, use and respite care and higher levels of social support are all predictive of positive outcomes on measures of quality of life for caregivers (Goode, Haley, Roth, & Ford, 1998; Jivanjee, 1994).

Education and support groups may contain elements that reduce isolation and stigma, and increase social networks and support, and could therefore be a very important element in the welfare of the caregiver, and consequently impact on the quality of caregiver interaction with the person with AD. Support groups focusing on education and emotional support for caregivers has been shown to be effective in reducing both caregiver burden and depression (Mittelman, Ferris, Shulman, &
Steinberg, 1995). Chiverton and Caine (1989) evaluated a brief education programme, finding that the caregivers felt more competent after the training, but also described some negative consequences of the training (feeling overwhelmed by the large amount of material presented and unresolved feelings about the AD diagnosis).

Family or individual counselling for caregivers can provide an essential resource to ease feelings of anger or frustration, which may otherwise be directed towards the individual with AD. This type of support for caregivers enables them to better care for the person with AD, and manage that AD person’s distress. The specific needs of families, their relationships, roles and communication patterns all impact family dynamics. Understanding these needs will help provide appropriate care and support for people with AD and their families (Bonjean, 1989).

Families that attended a minimum of six family therapy sessions (in addition to a support group) were found to delay institutional care for their family members with AD on average 329 days longer than those in a control group (Mittelman, Ferris, Shulman, & Steinberg, 1996). The impact on the quality of care for those with AD was not evaluated; however, it was speculated that improved caregiver wellbeing might result in better care for the person with AD.

Adult day-care facilities for people with AD can benefit caregivers, giving them time away from caregiving and possibly reducing the frequency of problematic interactions between the caregiver and the person with AD. Day-care can provide an alternative to full time institutionalisation (Beisecker, Wright, Chrisman, & Ashworth, 1996). Home-care support, such as assistance showering, is another alternative that provides practical support for caregivers thereby reducing caregiver burden (Vetter et al., 1997).

5.4 The Importance of Communication

Communication is an important aspect of relationships, involving many verbal and non-verbal interactions. However, once those abilities are disrupted in AD, caregivers are under greater pressure to compensate with more flexible communication styles. This may be especially difficult for spouses, who have a shared history and many years of developing their own ways of communicating within the dyad, which are rendered ineffective by the disease process (Richter et al., 1995). As previously mentioned, stressful interactions have been hypothesised to be
attributable to caregivers’ failure to understand the nature of the communication problems (Ripich et al., 1998). Ripich et al. (1998) suggested that caregivers who lack appropriate communication strategies might develop misconceptions about communication changes, develop unrealistic expectations and develop non-productive patterns of communication (such as arguing or criticism).

5.4.1 Ineffective Interventions and Distress

“Ineffective communication strategies only exacerbate the caregiving problems. Examining the effectiveness of a variety of intervention activities can assist family and formal caregivers in reducing the burden of caregiving” (Richter, 1995, p. 285). Richter (1995) found that arguing, restraints and reality orientation (e.g., when an 85 year old women is looking for her mother, being told “your mother is dead”) were some of the more common ineffective behavioural interventions. Many ineffective behavioural and communication techniques are tried by caregivers to change the behaviour of a person with AD, often resulting in distress, without changing the behaviour. It can be difficult for family members to accept that poor performance on a task is not simply due to lack of effort or a personality flaw. It can be difficult for them to extend their knowledge of the diagnosis of AD into an understanding, or integration, of exactly what the memory impairments and other cognitive impairments of AD mean in day-to-day life.

To illustrate common ineffective caregiver patterns of interaction, two specific examples observed by the researcher during the two studies in this thesis will be described. In both cases there are explanations (in terms of the deficits accompanying AD) as to why specific caregiver interventions did not work. In the first situation, a husband became upset with his wife (with AD) and criticised her for not making the bed as neatly as she used to. Her husband told her she was getting worse and should try harder to make the beds properly. This was an ineffective intervention because her declining functional ability was not related to her level of effort, but instead to her disease progression. She was greatly distressed by the criticism, as she had always prided herself on her housekeeping. Nevertheless, despite her efforts, she was unable to make an improvement. Consequently she became increasingly distressed at her shortcomings and her failure as a wife and homemaker. In this case there are a number of potentially successful alternatives for handling the situation. Firstly, the
husband could make the beds (although this may also upset his wife, as it was her job), or they could do the task together. Secondly he could accept her new level of functional ability and have a slightly less perfect bed. These first two options could potentially improve the quality of their interaction, if instead of criticism the caregiver attempted a more co-operative and supportive approach. Thirdly, procedural retraining to retain bed-making skills could be conducted, although, given the level of perfectionism required by this caregiver, retraining may never reach his standards.

In the second example, a man with AD had been shoplifting small items. His wife became angry with him and asked the community constable to discuss the problem with him. At the time this was distressing for the man, but did not change his behaviour, as he did not recall the event beyond the day it occurred. Unlike a child, people with AD cannot learn from being told about their mistakes. In other words, emotional distress does not mean they will behave differently next time. Angry responses, threats, and explanations are ineffective behavioural interventions, because individuals with AD have poor explicit memory, and fail to store the lesson. In this second case example there are few possible effective interventions. Supervision and gentle removal of stolen items with explanation or distraction could be used when stealing occurs.

5.4.2 Review of Effective Communication for Distress Reduction

Effective communication helps manage difficult situations and thus reduces distress in AD, however, to enable effective communication caregivers need information, support, and time away from caregiving (Gitlin et al., 2002). Caregivers that used task management (e.g. simplification of tasks, help with difficult aspects) and used effective communication strategies were found to be less upset over AD behaviours, than caregivers that used criticism-based strategies for the same AD behaviours (Gitlin et al., 2002).

Communication studies between caregivers and people with AD show certain types of communication to be more effective, and suggest techniques that may be useful. Fredericks (2001) found that in mild-to-moderate AD, participants could continue conversations with nurses/caregivers if the nurses/caregivers help the
conversation with corrections, word finding and by making an attempt to follow along with the individual’s content.

An education and communication training group (eight hours in duration) developed by Ripich et al. (1998) was effective for caregivers in the active group (no change for the control group) as it increased their knowledge of AD and communication techniques, and reduced the amount of communication difficulties they experienced. This effect remained even at 12-month follow-up. This programme provided information about AD and communication, to correct misconceptions and offer techniques to enhance communication. The group programme included discussion, videotapes, role-plays and written material. Specific communication techniques related to the title of the programme (FOCUSED), i.e., Face to face, Orient to topic, Continue topic, Unstick communication blocks, Structure questions, Exchange communication, Direct, short sentences (Ripich et al., 1998).

Bohling (1991) showed that sensitive listening and partial entry into the AD individual’s reality (even if it is incorrect) can be an effective communication technique to prevent behavioural problems and anxiety related outbursts. Professional caregivers have found certain techniques improved communication, such as the use of short verbal cues, breaking activities into components and presenting them one at a time, and continual verbal and non-verbal reassurance (Richter et al., 1995). Many reassuring phrases can be useful to diffuse situations e.g. ‘It’s going to be OK’ or ‘You’re going to be fine’. Using easily understandable gestures and gentle calm tones will probably get better results than an angry or frustrated response, which may contribute to aggressive behaviour. For example, in study two (refer chapter 4) participant HM, who could not communicate very easily but appeared to comprehend well, became agitated and frustrated at not being able to express himself. The researcher stated ‘It must all be very frustrating’, to which he replied ‘yes’ and began crying and smiling. There is a relationship between reduced ability to produce, repeat or comprehend emotions and higher levels of agitated behaviours and/or distress. This implies that if individuals with AD have difficulty understanding caregiver’s emotions or expressing their own emotions, then this may result in agitation, in the form of restlessness, anxiety, fearfulness, or being emotionally labile (Roberts, Ingram, Lamar, & Green, 1996). As a result of this, communication
strategies have to be suited to the individual’s abilities to participate in
communication, and their limitations in terms of emotional understanding accounted
for. For caregivers, being prepared, and somewhat willing, to answer the same
question many times may make it easier for a caregiver to be patient. It may be useful
if caregivers/family members do not expect the person with AD to remember anything
that he or she is told during interactions, and that may help reduce caregivers’ feelings
of frustration and disappointment.

Non-verbal communication (gestures, facial expressions, nods, noises etc)
remains long after verbal communication has become too poor to be effective. Some
literature suggests training caregivers in non-verbal communication may assist in
prolonging relationships and care in the home. Sabat and Cagigas (1997) reported a
case-study involving a woman with AD who used mime and facial expressions to
communicate her message to a willing listener when she could not communicate with
words. When she was able to get her message across she appeared satisfied with the
interaction. Often non-verbal communications can be utilised to prompt caregivers to
ask questions. For example, anxious facial expressions or pacing may prompt
questions such as ‘Do you need to go to the toilet?’ ‘Are you cold?’ Asking closed
questions that only require a yes or no answer, or a nod or a shake of the head,
reduces the language requirements of people with more advanced AD (Sabat &
Cagigas, 1997). Other non-verbal cues used by the person with AD may indicate a
need for attention, for example, putting a hand out. A caregiver may also use non-
verbal cues. A pat on the arm, for example, may indicate affection, and smiling is
universal. Even during the terminal stage of AD there is some evidence that severely
ill people with AD are able to communicate with facial expressions, vocalisations and
single words (Jansson, Norberg, Sandman, & Athlin, 1992).

The success of various communication training programmes suggests that the
care of people with AD could be improved by enhancing carer communication skills
(Ripich et al., 1998). New skills and knowledge could potentially reduce the stress
and frustration of caregivers, and also assist in reducing distress in the person with
AD, thereby improving the quality of their relationship.
5.5 Procedural Learning Retraining as a Model for Communication

In chapters 3 and 4, procedural learning was shown to be an effective method of new learning in individuals with AD. Declining ability to perform ADLs can be frustrating for individuals with AD, and the lack of ability to do tasks can also become a source of frustration for caregivers, which can lead to negative interactions (Lyketsos et al., 1997; Reifler, 1996). The procedural learning method may result in reducing distress in people with AD and their caregivers simply by improving ADLs. However, it may also involve development of a style of communication that reduces distress. In the two experimental studies, the researcher interacted and communicated with the participants. This involved simple explanations, answers to repetitive questions, reassurance and distraction when needed, and simple communication skills such as listening, being reciprocal, smiling, eye-contact, talking about topics of interest to the participant, and suggesting words or options when communication was limited. None of these communication techniques were part of the research itself, and they are not included in the procedural learning method. However, they were an essential component of the research process to build rapport and facilitate the participants’ engagement in the training process. Behavioural research with healthy participants often requires a ‘detached’ style with limited interaction between the researcher and the participant. This style is not appropriate with the AD population, as it would add to their confusion or distress. Note that the communication skills did not affect the outcome of the research as both trained and untrained tasks were performed under the same supportive conditions.

In addition to the communication skills used to engage with the participants, the method of procedural learning training for self-care tasks may model several key features of effective communication with this population and demonstrate a new way to communicate with individuals with AD. The basis of the procedural retraining of daily tasks is the repetition of tasks and step-by-step guidance. This method models a simple approach to activities that could be learned by caregivers, i.e., breaking tasks into components, slowly giving one instruction at a time accompanied by pointing or using demonstration if necessary (similar to the recommendations made by Richter et al., 1995). This could also provide a medium for educating caregivers about the
limitations of memory in AD. If taught by a proficient health professional, several other approaches for reducing distress (including communication techniques) could be incorporated (such as not arguing, distraction, reassurance, calm tones, smiling, holding hands, giving advanced warning of action and gently correcting or redirecting mistakes). In this way caregiver support and education could be included whilst learning the procedural training method.

In summary, it is possible that the procedural retraining method, when taught to caregivers, may also improve communication within the AD individual - caregiver relationship. Additionally it may allow people with AD to perform tasks for themselves and consequently reduce caregiver burden. This method may also improve the general health of people with AD by keeping them busy, and reducing their frustration. If provided in a supportive way this method may reduce incidents of aggression or depression in AD, therefore the method has implications for not only improving caregiver communication but also reducing distress in both the individual with AD and their caregiver.

5.6 A Multifaceted Approach to Care

Supporting good health and quality of life for people with AD and their caregivers is probably best achieved with an integrated approach to the care. This could include any of a range of services, such as access to family education, support groups, counselling, home help, social support, and on-going health professional monitoring and advice. In addition to this, it may be useful to implement both procedural learning training for self-care tasks and caregiver communication training.

Kenowsky (1996) outlined how a caregiver’s attitude can have a profound affect on the quality of life and the behavioural disturbances of people with AD. The author suggests not only supporting activities of daily living but also respecting the wishes of the person with AD as much as possible and trying to understand the triggers behind behavioural difficulties.

Several strategies can be used by caregivers (both family and professional) to effectively diminish distress in people with AD, including reducing or changing environmental stimulation (e.g., moving to a quiet place), avoiding reality orientation techniques and arguing, and providing verbal and non-verbal reassurances (Richter et al., 1995).
Increasing simple leisure activities has been shown to improve the relationship between the caregiver and the person with AD, and it also increases the quality of life for both (Dupuis & Pedlar, 1995; Mobily & Hoeft, 1985). Frequent (but non-routine disrupting) activity has been shown to have a positive effect on mood for people with AD (Dupuis & Pedlar, 1995). Unsurprisingly the frequency of leisure activities declines with the increasing severity of the disease, and there is a corresponding decrease in quality of life (Albert, Del Castillo-Castaneda, Sano, & Jacobs, 1996). However, people with moderate AD can still laugh and have fun. Some research suggests that music can delay the onset of agitation (Thomas, Heitman, & Alexander, 1997b). Other enjoyable activities may include walking, dancing, and watching sport (no story line), wild life programmes, gardening, receiving hugs from loved ones, interactions with grandchildren etc.

When communicating with people with AD, it is important to understand the potential fears, frustrations, and confusion, and for caregivers to try and maintain a relationship with skills such as reassurance and affection, so that the experience of interaction can be enjoyable for both the caregiver and the person with AD. Poor communication is just one of the many causes of caregiver burden, and distress in individuals with AD. Improving communication is one method, which has been shown to improve the AD individual - caregiver relationship, and the procedural learning method is a potential way to teach and improve communication in this context. The procedural training method may become a model for communication, as it requires no explicit memory demands, and it endorses moment-by-moment living and communication. It also emphasises the necessity for simple sentences and basic step by step instructions. When these methods are practised they may help caregivers and family to avoid placing unrealistic demands on the person with AD in their daily lives or their communications.
Chapter Six

General Discussion and Conclusions

Alzheimer’s disease (AD) is one of the most pervasive and devastating disorders of older age, which leads to an extensive loss of memory and ability to function independently. The recent trends in research have primarily focused on medication development to slow disease progression, with an emphasis on improving cognitive functions such as memory and learning; although more recently improvements in ADL ability and quality of life have also received some attention (Gauthier, Thal, & Rossor, 1999). Most of the more successful medications show some symptomatic effects or have been shown to have a mild preventative effect in epidemiological studies (Gauthier et al., 1999). Some of the newer medications such as galantamine have been shown to help ADLs and quality of life, but on the whole the effects of these medications decline as the disease progresses (Blesa, 2000). The non-pharmacological interventions for AD have been primarily limited to environmental modification, reality orientation, reminiscence (refer chapter 1, section 1.5.2), and caregiver training and education (refer chapter 5, section 5.4.2 & 5.5.3). Therapies that modify the environment to suit the person with AD are helpful in behaviour management (Tappen, 1997), as is caregiver communication training (Ripich et al., 1998), but such interventions do not change functional levels within the individual with AD.

Direct interventions to improve memory function in AD have achieved little success. Memory training has only achieved temporary benefit in individuals with mild AD for tasks such as learning specific face-name pairs (Davis et al., 2001; De Vreese et al., 2001). The premise of this thesis is based on demonstrations of relatively preserved procedural learning ability in mild-to-moderate AD (e.g., Deweer, Ergis, Fossati, et al., 1994; Dick, Kean & Sannds, 1988; Eslinger & Damasio, 1986; Gabrieli, Keane, Stanger, Kjelgaard, Corkin & Growdon, 1994; Heindel, Salmon, Shults, Walicke & Butters, 1989). These findings led to the hypothesis that this type of learning could be utilised to retrain and improve performance of self-care tasks and ADLs. The present research, therefore, investigated whether individuals with AD are able to improve their self-care and ADL
skills following procedural retraining, given findings that individuals with AD are capable of this particular type of new learning in laboratory-based tasks.

When reviewing non-pharmacological interventions for AD, Zeisel (2000) commented that approaches towards the treatment of AD should improve quality of life, slow disease progression, support the environment to maximise potential, and reduce the need for medication. Many of these elements are addressed in the training method outlined in this thesis, as this approach aims to improve quality of life through slowing the loss of daily life skills, or reinstating skills that have been lost. This involves specific training of individuals in their home environments, aimed at individualised needs and utilising a preserved function, which may allow individuals to do tasks for themselves.

The present research had four general aims. Firstly, to investigate whether self-care tasks could be either re-taught or improved in people with mild-to-moderate AD, using procedural learning methods. This aim was achieved using a combined group and single subject design in study one (chapter 3). The findings from this study were that self-care tasks can be re-taught and/or improved in individuals with mild-to-moderate AD using a procedural training method.

The second aim was to determine the duration of any gains from procedural training, an important component in judging training effectiveness, and also providing an indicator of appropriate timing for refresher training sessions. Individuals in both experimental studies showed a variable pattern of duration of training effects across tasks and across participants, suggesting that there is no specific length of time that procedural-training effects last. The gains that were made, however, often lasted 3 months or more. Additionally, the results from the second experimental study demonstrated that for some individuals the gains on some tasks (the more simple ones) could last up to 13 months. The overall between-subject and between-task variability indicates that refresher training sessions would need to be tailored to individual needs, as the duration of the training effects can not be generalised across individuals, or tasks.

The third aim was to investigate the effectiveness of a needs-based procedural training programme for self-care skills, which could improve the quality of life for people with AD. This was addressed in the second experimental study. The results suggested that the procedural-training method indeed could be a useful basis for a
training programme if based on the specific needs of the individuals (i.e., targeting daily skills that show functional impairment).

The final aim was to review the research on communication and care in AD, and to use the findings of the present research as a basis for suggesting areas of development for caregiver support or education. The current literature on distress in people with AD and the role of communication was reviewed in chapter 5. It was argued that the procedural training method modelled some of the important elements for effective communication with people with AD. In addition to this, caregiver support and education (to reduce distress in caregivers and those with AD) was seen as vital for improving the quality of life of both the individual with AD and their caregivers or family. In other words, the procedural-training method can only address some of the needs of people with AD, and a multifaceted approach to care is probably best.

6.1 Experimental Studies One and Two

As the results of the two experimental studies have been discussed in some detail in chapters 3 and 4 respectively, only the main conclusions will be reviewed here.

The findings of study one demonstrated that procedural learning is relatively preserved in AD, and this can be applied in a practical way to ADLs. Furthermore, the benefit of training was still evident, relative to baseline performance, 3 months after training. This suggests retention of the trained skills in procedural knowledge. The use of multiple baselines and untrained tasks showed that it was the task-specific training, and not general effects of experimenter presence or other extraneous variables, which caused the improvements. Additionally, there was an absence of significant change in task performance between first and last baseline times at any of the multiple baseline lengths. Taken together, therefore, it can be assumed that any subsequent improvement in tasks was due solely to the intervention. The data analyses, both at the group and individual levels, made it possible to conclude that procedural training is effective for these participants (although not for every participant or every task). AD participants performed the selected self-care and ADL tasks faster and with fewer errors following procedural training, but showed no such improvement on comparable untrained tasks. Thus, no speed-accuracy trade-off was
apparent during performance of the trained tasks, and the significant findings on both performance time, and number of errors, provide consistent evidence of improvement following training.

The group analyses not only compared AD group and control group results, but also analysed the group results comparing each individuals’ performance at post-test, 2 week, 4 week and 12 week follow-ups to their own baseline performance (for performance time and number of errors). These analyses showed positive training effects, and duration of these effects for 12 weeks. The single subject analyses employed a statistical criterion for the judgement of training success (i.e., training was judged to be successful when 70% of baseline points fell above the upper 95% CI projected back in time from the follow-up period) and provided an objective judgement of training success for performance time data. This contrasts with the more commonly used method of visual analysis of single subject design (which involves making a judgement about training success by reviewing plotted data for a change in performance). Error data was used as a second level of analyses. The visual analysis method was used for evaluating improvement in the number of errors because some participants performed tasks with a small numbers of errors overall.

The finding that 7 out of 8 participants in the first study showed benefit from training on a fixed set of tasks (regardless of whether these tasks were appropriate to each individual) led to the hypothesis that a personally-tailored training programme would provide even greater benefits due to a better match between trained tasks and the needs of the individual. Therefore, in the second study an assessment to determine trainable tasks was undertaken, which chose tasks that were important to AD participants (and their caregivers), and which were currently causing some difficulty. Selecting tasks by this method assisted with participant motivation and gave purpose to the training activity (i.e., the AD participants could understand why they were being trained on these tasks). The findings of this study demonstrated that an individually tailored programme produced positive individual results, as all participants showed procedural learning in at least three of their tasks. Additionally, more of the tasks (across the participants) showed training effects in either performance time or error improvement than in the first experimental study.

In study two, the design (multiple baseline across tasks) was able to demonstrate clear patterns of improvement coinciding with the onset of training,
either by reduced performance time or number of errors. For the 5 participants observed in the follow-up phase, the benefits were shown to last for a considerable length of time. In one case the impact of training was still seen for some relatively simple tasks at the 13 month follow-up point (although some of the other more complex tasks had declined to baseline levels). Overall, these results clearly demonstrate the potential of this training method to make a considerable contribution to ADL and self-care ability of people with AD.

No predictors of who would benefit from training were found in either experimental study. The rotory pursuit task did not show the expected correlations with measures of success of task training. In both studies one participant performed poorly on the rotory pursuit task (AD7 change of 4.2 seconds, and BP change of 3 seconds, over the 10 trials), whilst showing success from procedural retraining of daily tasks. This suggests that even if a group with a larger sample size showed significant correlations between rotory pursuit performance and success in daily skills retraining, this may not be predictive at an individual level. The question remains as to why performance on the rotory pursuit task was unable to predict success in procedural retraining of daily tasks? It is possible that different procedural learning circuitry underlies performance on the rotory pursuit task and performance of daily living skills. Alternatively, it is possible that changes in the parameters of the rotory pursuit task might affect how well AD participants acquire the skills. For example, if the rotory pursuit task had been administered at a slower rotation, the participants less able to learn the rotory pursuit task might have shown greater evidence of learning trends. Alternatively, the number of trials could account for the different patterns. For the rotory pursuit task the participants performed a total of 10 trials (during 1 day). In contrast, the trained daily living skills were performed 50 times (over ten days). In other words, learning trends on the rotory pursuit task may have become more apparent with higher numbers of trials.

One of the expectations of the research was that there would be a coarse association between procedural learning ability and dementia severity. However, no obvious relationship between the severity of dementia (as measured by the MMSE) and effects of procedural training on experimental tasks (daily living skills) was found. This was also true for the relation between dementia severity and the rotory pursuit task. It is possible that these trends would be seen if there was a larger sample
size, however individual variations suggest that there would always be exceptions to
that trend, so any results from a large sample size would not generalise to the
individual level. Perhaps the ability to learn procedurally continues relatively intact
in AD, but the tasks that can be successfully trained may need to be simpler as AD
severity increases (e.g., the rotary pursuit task may still be able to be learnt by
individuals with more severe AD if set at a slower speed). In terms of daily living
skills this may mean that more simple tasks need to be chosen for procedural
retraining as a person progresses through the course of AD. For example, learning to
make a cup of tea may be able to be trained for a person with mild AD, and turning on
a light may be able to be trained for a person with severe AD.

In summary, the results from the two experimental studies contradict
conventional wisdom that people with AD can not learn, or cannot learn tasks that
they have lost the ability to perform. The successful application of the procedural
learning method has shown this to be one of the first effective techniques for working
with people with AD to improve their quality of life directly (rather than through
caregiver training alone). The detailed study at the individual level has provided
unique evidence, which supports and extends the earlier group findings of Zanetti et
al. (1997, 2001) as well as the findings of Josephsson (1995). This research also
enhances our understanding of the procedural learning subsystems of memory and
learning by demonstrating that even when procedural knowledge is lost in AD, the
ability to relearn that knowledge can be relatively intact.

6.2 The Relationship of the Results to Theories of
Procedural Learning

In chapter 2 theories regarding procedural learning were reviewed, including a
discussion of the difference between procedural learning and procedural memory.
Although no clear evidence exists regarding specific areas of the brain and procedural
learning and memory, several structures are currently hypothesised to be critical
(primary sensory/motor areas, the dorsal striatum and the cerebellum), based on
experimental and functional imaging evidence of their involvement in this type of
learning (e.g., Ackermann, Daum, Schugens, & Grodd, 1996; Gabrieli, 1998;
Hikosaka et al., 1996). These critical structures are relatively preserved in AD, which is thought to explain why procedural learning is also relatively preserved.

Three frontal-striatal circuits have been proposed to relate to procedural learning: the lateral orbitofrontal circuit, dorsolateral prefrontal circuit and the motor-skeletal circuit. It has been found that individuals with epilepsy resulting from frontal loci have contralaterally impaired motor procedural learning, although it is unknown which specific frontal area or circuit may cause this impairment (De Guise et al., 1999). While the motor/skeletal circuit is relatively preserved in AD, it is unclear if the other two frontal-striatal circuits are affected, as AD pathology does affect the frontal lobes later in the disease course (Butters et al., 1995; Duke & Kaszniak, 2000; Eastwood & Reisberg, 1999). However, given the preserved procedural learning ability of most AD individuals in the mild-to-moderate stages it seems likely that these critical frontal-striatal circuits are not primarily affected by AD pathology.

It is proposed in this thesis that although procedural knowledge is lost in AD, procedural skills can, nevertheless, be relearned. However, the duration of procedural knowledge in AD and the pattern in which procedural knowledge is lost from memory in AD is unknown. Can any conclusions about these questions be drawn from the results of the two experimental studies? One participant (PB in study two) clearly showed that procedural knowledge can be lost in a variable manner, that is, in her case different tasks were lost at different rates. Those tasks that did decline over the 13 month research period appeared to do so gradually with increased errors and longer performance time as the tasks slowly reverted to their baseline level. In her case it appears that the more complex tasks (i.e., those with more elements) and those tasks requiring visual spatial abilities (e.g., pegging up clothes) or cognitive procedural knowledge (e.g., sorting cutlery into categories in the draw, knives with knives etc.) were lost more quickly than more simple motor tasks (e.g., opening a sliding door), which were performed well at the 13 month follow-up. The variable decline in different tasks could be a result of their level of difficulty. Simple tasks may be more easily maintained, and therefore continue to be repeated correctly. In contrast complex tasks allow more opportunity for error, and those errors may interfere with the procedural knowledge of that task. No other participant showed such a clear pattern, most likely because of the shorter follow-up lengths.
The findings of the two experimental studies demonstrate that procedural learning can add to procedural knowledge in individuals with mild-to-moderate AD, whereas explicit learning in these same individuals does not add to explicit knowledge. Additionally, both forms of knowledge are continually eroded in AD, although these results indicate that in some instances any newly learned procedural knowledge may last for up to a year. This pattern of spared and impaired learning and memory indicates that Figure 3 (chapter 2) has some validity, and is consistent with previous laboratory-based research findings of preserved procedural learning in AD. The implication of both sorts of findings is that, in AD, neurological areas underpinning procedural learning are relatively spared, and that this neural circuitry is distinct from that involved in explicit learning, as proposed by Squire (1992).

6.3 The Importance of Distress Reduction and Clear Communication in Alzheimer’s Disease: Procedural Learning as a Model

Chapter 5 provided a review of the literature on communication techniques for distress reduction in individuals with AD. Based on the work in this thesis, it is proposed that procedural learning techniques are a type of methodology that could be used to demonstrate effective communication (i.e., presenting one request at a time, breaking these requests into small components etc.). The procedural learning methodology is consistent with the recommendations of Richter (1998), namely due to, the use of short verbal cues, breaking activities into components and presenting them one at a time, and the use of verbal reassurance. It may be possible to teach caregivers to use the procedural learning method, although, not all caregivers would be suitable because of caregiver factors such as frustration, lack of patience, perfectionism etc. As reviewed in chapter 5, distress is caused by many different factors, some within the AD person due to the disease process, some related to caregiver issues, and still more due to poor communication between the person with AD and their caregiver. In order to help with the latter two factors, support and education of the caregiver about AD and effective communication strategies are essential. Additionally, interventions for the person with AD, such as environmental modification (Tappen, 1997), reassurance (Richter et al., 1995), medication (Gauthier
et al., 1999) and procedural learning retraining have all been shown to be beneficial, implying that a multifaceted approach to AD care may be most effective.

The procedural learning method may become a model of communication that provides caregivers with something practical to do. Caregivers often give instructions that are complex and easily forgotten, causing distress to both parties. Important principles of communication may be included when training caregivers the procedural training method, such as the use of prompts to encourage task initiation and breaking tasks into components (giving one instruction at a time) to encourage task continuation and completion. This method provides an opportunity for health workers to educate caregivers about loss of function (e.g., the person with AD can not use explicit memory to learn tasks or remember information) and changing levels of ability (e.g., the caregiver would be informed that the person with AD could not be trained to cook the family meals again, but they could be taught to make a sandwich). Seeing the results of this method may reduce high caregiver expectations on explicit memory, and change caregiver beliefs that the person with AD is somehow responsible for their forgetfulness, or being naughty or lazy.

It is ethical practice for health professionals to consider distress reduction when working with people with AD, and this method provides no exception. Caregiver education related to communication, and changing levels of ability is extremely important so that anger is not directed at the individual with AD. The pressure of caring for people with AD can result in those with AD being recipients of physical abuse (Comptom, Flanagan, & Gregg, 1997; Pot, van Dyck, Jonker, & Deeg, 1996). A method that can reduce the burden on the caregiver and improve the quality of the relationship between the caregiver and the individual with AD has clear benefits. The difficulty with this method is that it will not be suitable for all caregivers, as it requires patience and a willingness to try something new. In some cases these requirements may increase the level of stress the caregiver is experiencing. In these situations it is probably preferable that a trained person such as a nurse or support worker conducts the procedural learning retraining, rather than the caregiver. In these situations the caregiver could continue to be involved (e.g., by receiving education about AD, communication training, or be trained to use prompts).
6.4 Who will do the Training: Problems and Logistics

The procedural training method is time consuming and involves many hours of one-to-one training. There are several barriers to the implementation of the findings of this research into the wider community. Firstly there is the question of who will implement the training programme. Secondly, will the time-consuming nature of training limit its implementation? In dementia care facilities staff could be trained in this method, although staff enthusiasm and patience may fluctuate and impact on the quality of the training.

Caregivers, nurses or other health professionals may be trained to use this method, but a key component of successful procedural training is selecting tasks appropriate for this approach. For example relearning to bake a cake is too complex as it involves many elements: initiation, motivation, intact spatial organisation, and planning. Some of these elements may interfere with the ability of the task to be learned in a procedural manner. Judgements of task appropriateness are essential for training success. Some trainers (especially caregivers) may not understand the theoretical foundation of procedural learning, and this, in combination with personal expectations of the caregiver, may lead to inappropriate tasks being chosen. Some nurses/health professionals could possibly be trained to decide viability of tasks to be trained (for example, using criteria such as that listed in chapter 3) or, alternatively, an appropriately skilled person could assist in those decisions prior to the commencement of the training. Another possible solution to these difficulties might be to prescribe a detailed list of tasks that can or can’t be trained (although in reality this would be highly variable between AD individuals). This may limit the potential flexibility of training and limit some tasks that may be trainable; also it is likely that many possible tasks would be missed. However, if a list were to be developed it may be of the following form:

**Moderately severe AD** (MMSE score 0-9)
Cleaning teeth; washing face and hands; opening doors; putting on specific items of clothing (jersey, a specific pair of shoes); turning on/off lights; closing/opening curtains; turning on/off TV; brushing hair.

**Moderate AD** (MMSE score 10-16 (Reisberg, 1984))
As with moderately severe, plus:
Making phone-call with phone numbers and names printed on a large card (put 4 or 5 on a card only and have by the phone); writing name; putting on seat belt in car; putting something in the microwave and starting (microwaves with instant cook button only); shaving with electric shaver; washing self in shower; drying dishes (not putting them away); answering a ringing phone; pulling up the sheets on a bed.

Mild AD. (MMSE score 17-24 (Reisberg, 1984))

As with moderate and moderately severe plus:

Making a simple sandwich; making a cup of tea/coffee; using microwave; using radio or TV remote (on and off, volume and channel); using oven; using stove top; using washing machine/drier; using garden hose; putting on more complex clothing (stockings, ties); vacuuming; simple cleaning jobs; setting a simple table for a meal; locking door and taking a key (best to have a door that you can only lock with a key – to avoid getting locked out). Putting things in labelled draws (the learning is reading labels before putting things away, and reading the labels to find them again); hanging out washing; drying self after a shower; making a bed.

In future research the validity of the recommendations at each level could be evaluated. Can these judgements be made accurately for the AD population? Does there need to be a detailed set list? The suggested tasks arise from observations of levels of functioning (the MMSE scores provide a coarse guide) and learning abilities observed in the two experimental studies in this thesis, although clearly they are only a guide. Extensive research with large numbers of participants would be needed before these lists could be considered reliable or valid, and there are probably many more tasks that would benefit from training.

It is important to note that whilst tasks may be successfully trained by procedural learning methods, prompting may be required for task initiation. For example, a person may learn how to turn off an electric stove element, but the ability to do this task does not signify that they will remember to do that task without prompting. This training teaches people how to do activities; it does not teach the person to perform these tasks spontaneously. However, given that these individuals usually live with a caregiver, the provision of prompts is often easily provided.
6.5 Concluding Statement

The research in this thesis suggests it is possible to retrain and maintain specific self-care and daily living skills in AD. Therefore, the application of procedural learning techniques could form the basis of a specific training programme. As we improve our understanding of the nature and variability of AD our intervention practices can become more focused on the needs of individuals and families. The procedural learning method is one way to assist with the daily functioning of a person with AD and potentially improve their, and their caregiver’s, quality of life. Utilising a relatively preserved ability in AD to achieve manageable daily living skills may potentially lessen feelings of frustration or failure. This research has shown that tasks can be retaught in AD and the even though these tasks are lost from procedural knowledge this research has demonstrated that they can be reinstated.
References


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deficits in a selected group of patients with Alzheimer's disease.  


## Appendix

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PARTICIPANT INFORMATION SHEET

Relearning of impaired self care skills in Alzheimer’s Disease

PRINCIPAL INVESTIGATOR

The study is being carried out by Kathryn Russell, Dr. Lynette Tippett and Dr. Phil Wood. Kathryn Russell is a Master’s student in the Department of Psychology at the University of Auckland, Ph 2676659 (home), 4861491 extn 2868 (work), Dr. Lynette Tippett, a Lecturer in the Department of Psychology, University of Auckland, Ph 3737599 extn 8551, and Dr. Phil Wood is a Senior Lecturer and Consultant Physician, Health Service of Older People/Section of Geriatric Medicine, North Shore Hospital.

INTRODUCTION

You are invited to take part in a research study which will examine how well impaired self care skills can be relearned in individuals with Alzheimer’s Disease, following a training programme. You are not in any way obliged to participate in this study. If you would like to participate, however, we would be grateful if you could let us know (by phone or letter). We would like you to make a decision about your participation within the next 2 weeks, but you may take longer if necessary.

ABOUT THE STUDY

- The aim of the study is to see if people with Alzheimer’s Disease can relearn self care tasks that are causing them some difficulty. These tasks range from making a cup of tea or a sandwich to brushing your hair. We wish to see if a particular way of retraining is effective and if it is, how long the training effect lasts.
- Participants in this study either have memory problems and a diagnosis of probable Alzheimer’s Disease (active study group, 10 members) or are healthy elderly people who are not experiencing memory difficulties (control group, 10 members). All participants will be fluent in English.
• Individuals on any treatment drug for Alzheimer's disease will not be able to participate in this study because if there is an improvement in self care skills we will not know if it is the medication or the training that has caused an improvement.

• You are being invited to participate in this study because you have memory problems and are eligible for the active study group; or are a family member/caregiver of that person.

• During this study, the first visit will take place either at North Shore Hospital or at your home, depending on which your prefer. This visit will take about one and a half hours. The remainder of the training and testing will occur in your home.

• You will be involved in the study for about 3 months. For 3 weeks there will be daily sessions from Monday to Friday, and for the rest of the time you will be seen 1 session per week. Family members will only be involved in the first session.

• The first visit will involve answering some questions about your background, completing six short tasks involving memory and language. Family members will only be asked some questions about how you are managing at home.

• The sessions that follow will be in your home; for one week you will be asked to perform 12 daily tasks such as making a cup of tea. You will then receive training on six of the tasks for 2 weeks; during this time each visit may be up to 2 hours long. Training will involve being shown how to do the activities and practicing the activities; you will be corrected when you make mistakes.

• After this training period, of two weeks, there will be several short visits (20 minutes every week for 2 months) to see how well you can perform the tasks after you have been taught them.

RISKS AND BENEFITS.

1. There are no expected risks although the training sessions will take up to 2 hours per day, and the visits after that will take 20 minutes once a week. Training may be a little tiring but you will be able to stop and have breaks when you need them.
2. The benefits you may receive from taking part in the study is that you may relearn one or more of the self care tasks that you are having difficulty with. The study will also help increase our understanding of memory processes in Alzheimer's disease.

3. Taking part in this study will not cost you anything. There is no payment or reimbursement for your time.

4. There are no alternate training programmes currently available to improve self care skills for individuals with Alzheimer's disease.

PARTICIPATION

1. Your participation is entirely voluntary (your choice). You do not have to take part in this study, and if you choose not to take part you will receive the usual healthcare.

2. If you do agree to take part you are free to withdraw from the study at any time, without having to give a reason and this will in no way affect your future health care. Participation in this study will be stopped should any harmful effects appear or if any health professional, such as your GP, feels it is not your best interests to continue.

GENERAL

- There will be no additional training at the conclusion of the study, but you and your family will be able to discuss your results with your health professionals if you wish.
- If you, or a relative or friend, have any questions or wish to know more about the study please phone Kathryn Work: (09) 486 1491 (Extn. 2868) Home: (09)267 6659.
  Alternatively you may contact her at: The Department of Psychology, University of Auckland, Private Bag, 92019, Auckland.

You are free to withdrawal from the training or the study at any time if you wish.

If you have any queries or concerns about your rights as a participant in this study you may wish to contact the Health Advocates Trust, telephone (09) 623 5799.

CONFIDENTIALITY

No material which could personally identify you will be used in any reports on this study.
All data will be kept strictly confidential and your privacy will be protected. Your performance will remain confidential to the investigators during the study. At the completion of the study all records will be locked away in filing cabinets for up to 10 years.

RESULTS

The results of your performance will be available to you at the conclusion of the testing, and a summary of the results from the thesis will be forwarded to all participants who request them.

STATEMENT OF APPROVAL

This study has received ethical approval from the North Health Ethics Committee.

Please feel free to contact the researcher if you have any questions about this study.

COMPENSATION

If you suffer physical injury as a result of your participation in this clinical trial, you may be covered by ARCIC. You should note, however, that eligibility for cover is not automatic.

Your claim for cover may be accepted by ARCIC but your entitlement to compensation will depend on a number of factors such as whether you are an earner or a non-earner. You should note that in most cases ARCIC provides only partial reimbursement of costs and expenses and there is no lump sum compensation payable under the current ARCIC legislation.

If you have suffered only mental injury, there will be no ARCIC compensation available.

You should also be aware that if you have cover under the ARCIC legislation your right to sue the researcher(s) or anyone else involved in the clinical trial is extremely limited.

If you have any questions about cover or entitlements under the ARCIC scheme you should contact your nearest ARCIC branch office for further information before you consent to participate in this trial.
CONSENT FORM
(Activity study group).
Relearning of impaired self care skills in Alzheimer’s Disease

Principal Investigator: Dr Lynette Tippett, Kathryn Russell

Name: ___________________________ Age: ___ years

REQUEST FOR INTERPRETER

<table>
<thead>
<tr>
<th>Language</th>
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<th>No.</th>
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<tbody>
<tr>
<td>English</td>
<td>I wish to have an interpreter</td>
<td>Yes</td>
</tr>
<tr>
<td>Maori</td>
<td>E kia hiia mea ahau ki tetahi tangata hei korero Maori ki ahau.</td>
<td>Ae</td>
</tr>
<tr>
<td>Samoan</td>
<td>O te mano o e nei se fumuamatu upe.</td>
<td>Joe</td>
</tr>
<tr>
<td>Tongan</td>
<td>O ku fiamea ha fakatonu a.</td>
<td>Io</td>
</tr>
<tr>
<td>Cook Island</td>
<td>Ka inauaro sa i tetai tangata uri reo.</td>
<td>Ae</td>
</tr>
<tr>
<td>Niuean</td>
<td>Fia manako sa ko fakaanga e tagata fakohoko ho vauhau.</td>
<td>E</td>
</tr>
</tbody>
</table>

I have read and I understand the information sheet dated ______ for volunteers invited to take part in the study designed to re-teach self care skills. I have had the opportunity to discuss this study and I am satisfied with the answers I have been given. I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time and this will in no way affect my future health care.

I understand that my participation in this study is confidential and that no material which could identify me will be used in any reports on this study. I understand that the investigation, will be stopped if it should appear harmful to me. I understand the compensation provisions for this study. I have had time to consider whether to take part. I know who to contact if I have any questions about the study.

I wish to receive a copy of the results. YES/NO

I ___________________________ (Print name) hereby consent to take part as a participant in this research.

Signed ___________________________ (participant). Date ______.

and ___________________________ Family member/caregiver.

In my opinion, consent was given freely and with understanding.

Witness name: ______________. Witness Signature________________________.

If you have any queries or concerns about your rights as a participant in this study you may wish to contact Health Advocates Trust, telephone (09) 623 5799.

Signature ____________ Date ______

Researchers: Kathryn Russell, Ph 2676659, 4861491 extn 2868.
Lynette Tippett, Ph 3737599 extn 8551.
Appendix III

CONSENT FORM

(Family member/caregiver of participant).

Relearning of impaired self care skills in Alzheimer’s Disease
Principal Investigator: Dr Lynette Tippett, Kathryn Russell

Name: __________________________________________________________________________.Age: ___ years

REQUEST FOR INTERPRETER

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<td>English</td>
<td>I wish to have an interpreter.</td>
<td>Yes</td>
<td></td>
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<tr>
<td>Maori</td>
<td>E biahi aua ahu ki teai i mero ki korero Maori ki ahu.</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>Samban</td>
<td>Oste manavu e ia te i anu kura.</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>Tanganyan</td>
<td>Otu tetu ha tetu tetu.</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>Cook Island</td>
<td>Ka tangaro a i tetu tangato uri reo.</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>Niasan</td>
<td>Fiinamanu a ku fakaoa e taga tawakoko tawakoko vauhu.</td>
<td>Yes</td>
<td></td>
</tr>
</tbody>
</table>

I have read and I understand the information sheet dated ___ for volunteers invited to take part in the study designed to re-teach self care skills. I have had the opportunity to discuss this study and I am satisfied with the answers I have been given. I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time and this will in no way affect my future health care.

I understand that my participation in this study is confidential and that no material which could identify me will be used in any reports on this study. I understand that the investigation, will be stopped if it should appear harmful to me. I understand the compensation provisions for this study. I have had time to consider whether to take part. I know who to contact if I have any questions about the study.

I wish to receive a copy of the results. YES/NO

I _____________ (Print name) hereby consent to take part in this study.

Signed _____________________ (participant) date ______

In my opinion, consent was given freely and with understanding.

Witness name: ___________ Witness Signature __________________________

If you have any queries or concerns about your rights as a participant in this study you may wish to contact Health Advocates Trust, telephone (09) 623 5799.

Signature __________ Date __________

Researchers: Kathryn Russell, Ph 2676659, 4861491 exm 2868.
Lynette Tippett, Ph 3737599 exm 8551.
Appendix IV

CONSENT FORM
(Caregiver Interview).

Relearning of impaired self care skills in Alzheimer’s Disease
Principal Investigator: Dr. Lynette Tippett, Kathryn Russell

Name: ____________

REQUEST FOR INTERPRETER

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<tbody>
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<td>English</td>
<td>I wish to have an interpreter.</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Maori</td>
<td>E hiahia ana aha ki tetahi tangata hei kore o Maori ki ahaa.</td>
<td>Ae</td>
<td>Kao</td>
</tr>
<tr>
<td>Samoan</td>
<td>Oute manao e lai se f'amatapua upe.</td>
<td>Io</td>
<td>Leai</td>
</tr>
<tr>
<td>Tongan</td>
<td>Yoka flemea ha fa'akatatese.</td>
<td>Io</td>
<td>Ilaai</td>
</tr>
<tr>
<td>Cook Island</td>
<td>Ka manganu au i tetai tangata uri reo.</td>
<td>Ae</td>
<td>Kare</td>
</tr>
<tr>
<td>Niuean</td>
<td>Fia manako au i tea fakanoga e tagata faakiokohoko vagauna.</td>
<td>E</td>
<td>Nukai</td>
</tr>
</tbody>
</table>

I wish to take part in an a 10–20 minute interview about the effects of the procedural learning training programme on my family member. I have had the interview explained to me. I have had the opportunity to discuss the interview and I am satisfied with the answers I have been given. I understand that taking part in this interview is voluntary (my choice) and that I may withdraw from the interview at any time and this will in no way affect my, or my family members future health care.

I understand that my participation in this study is confidential and that no material which could identify me will be used in any reports on this study. I understand that the investigation, will be stopped if it should appear harmful to me. I understand the compensation provisions for this study. I have had time to consider whether to take part.

I know who to contact if I have any questions about the study. I wish to receive a copy of the results. YES/NO.

I consent to having the interview taped for ease of transcribing. YES/NO.

I ____________ (Print name) hereby consent to take part in this study.

Signed ____________________________ (participant). date ________.

In my opinion, consent was given freely and with understanding.

Witness name: ____________ . Witness Signature. ____________

If you have any concerns about the study, you may contact: Health and Disability Services Consumer Advocate, telephone (09) 623 5799

Signature
Researchers: Kathryn Russell, Ph 2676659, 4861491 extn 2868.
            Lynette Tippett, Ph 3737599 extn 8551.
Appendix V

PARTICIPANT INFORMATION SHEET
Control participants
(Healthy elderly without Alzheimer’s Disease).
Relearning of impaired self care skills in Alzheimer’s Disease

PRINCIPAL INVESTIGATOR.
The study is being carried out by Kathryn Russell, Dr. Lynette Tippett and Dr. Phil Wood. Kathryn Russell is a Master’s student in the Department of Psychology at the University of Auckland, ph 2676659 (home), 4861491 extn 2868 (work), Dr. Lynette Tippett, a Lecturer in the Department of Psychology, University of Auckland, Ph 3737599 extn 8551, and Dr. Phil Wood is a Senior Lecturer and Consultant Physician, Health Service of Older People/Section of Geriatric Medicine, North Shore Hospital.

INTRODUCTION
You are invited to take part in a research study which will examine how well impaired self care skills can be relearned in individuals with Alzheimer’s Disease, following a training programme. You are not in any way obliged to participate in this study. If you would like to participate, however, we would be grateful if you could let us know (by phone or letter). We would like you to make a decision about your participation within the next 2 weeks, but you may take longer if necessary.

ABOUT THE STUDY
• The aim of the study is to see if people with Alzheimer’s Disease can relearn self care tasks that are causing them some difficulty. These tasks range from making a cup of tea or a sandwich to brushing your hair. We wish to see if a particular way of retraining is effective and if it is, how long the training effect lasts.

• Participants in this study either have memory problems and a diagnosis of probable Alzheimer’s Disease (active study group, 10 members), or, are elderly people who are not experiencing memory difficulties (control group, 10 members). All participants will be fluent in English.

• You are being invited to participate because you are a normal healthy elderly individual not experiencing bad memory difficulties.
Your involvement in this study will involve one session, conducted in your home, and will take about 2 hours of your time.

You will be asked to answer some questions about your background and to complete six short tasks involving memory and language. You will also be asked to perform 12 common daily tasks such as making a cup of tea.

We need normal elderly individuals to participate, to provide us with information about normal performance on these tasks.

**RISKS AND BENEFITS.**

1. There are no expected risks in this study although the testing will take up to 2 hours. You will be able to stop and have breaks when you need them.

2. The benefits of this study are to help research on retraining self care tasks in individuals with Alzheimer’s Disease, and therefore possibly improve the quality of life of these individuals.

3. There will be no therapeutic effect, or remedial benefit for control participants.

4. Taking part in this study will not cost you anything. There is no payment or reimbursement for your time.

**PARTICIPATION**

1. Your participation is entirely voluntary (your choice). You do not have to take part in this study, and if you choose not to take part this will not affect any future health-care.

2. If you do agree to take part you are free to withdraw from the study at any time, without having to give a reason and this will in no way affect your future health care.

**GENERAL**

- If you, or a relative or friend, have any questions or wish to know more about the study please phone Kathryn Work: (09) 486 1491 (Extn. 2868) Home: (09)267 6659.

Alternatively you may contact her at: The Department of Psychology, University of Auckland, Private Bag, 92019, Auckland.
You are free to stop the interview or testing at any time if you wish.

If you have any queries or concerns about your rights as a participant in this study you may wish to contact Health Advocates Trust, telephone (09) 623 5799.

CONFIDENTIALITY
No material which could personally identify you will be used in any reports on this study. All data will be kept strictly confidential and your privacy will be protected. Your performance will remain confidential to the investigators during the study. At the completion of the study all records will be locked away in filing cabinets for up to 10 years.

RESULTS
The results of your performance will be available to you at the conclusion of the testing, and a summary of the results from the thesis will be forwarded to all participants who request them.

STATEMENT OF APPROVAL
This study has received ethical approval from the North Health Ethics Committee.

Please feel free to contact the researcher if you have any questions about this study.

COMPENSATION

If you suffer physical injury as a result of your participation in this clinical trial, you may be covered by ARCIC. You should note, however, that eligibility for cover is not automatic.

Your claim for cover may be accepted by ARCIC but your entitlement to compensation will depend on a number of factors such as whether you are an earner or a non-earner. You should note that in most cases ARCIC provides only partial reimbursement of costs and expenses and there is no lump sum compensation payable under the current ARCIC legislation.

If you have suffered only mental injury, there will be no ARCIC compensation available.
Appendix VI

CONSENT FORM
(Active study group).

Relearning of impaired self care skills in Alzheimer’s Disease

Principal Investigator: Dr Lynette Tippett, Kathryn Russell

Name:_________________________ Age: ____ years

REQUEST FOR INTERPRETER

<table>
<thead>
<tr>
<th>Language</th>
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<td>No</td>
</tr>
<tr>
<td>Maori</td>
<td>E kaiaia ana ahu ki tonahi tangata he Korero Maori ki ahu.</td>
<td>Ao</td>
<td>Ka</td>
</tr>
<tr>
<td>Samoan</td>
<td>Ota manu o e iasi se fiasamata upa.</td>
<td>Joe</td>
<td>Lea</td>
</tr>
<tr>
<td>Tongan</td>
<td>Oka fuemau ha fakatsonula.</td>
<td>Io</td>
<td>Kel</td>
</tr>
<tr>
<td>Cook island</td>
<td>Ka inaepe aai i tai tangata uri reo.</td>
<td>Ao</td>
<td>Kae</td>
</tr>
<tr>
<td>Niuean</td>
<td>Fia mokako au he fakaeaga e tagata fakahoko ho vasauna.</td>
<td>E</td>
<td>Nokai</td>
</tr>
</tbody>
</table>

I have read and I understand the information sheet dated ______ for volunteers invited to take part in the study designed to re-teach self care skills. I have had the opportunity to discuss this study and I am satisfied with the answers I have been given. I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time and this will in no way affect my future health care.

I understand that my participation in this study is confidential and that no material which could identify me will be used in any reports on this study. I understand that the investigation, will be stopped if it should appear harmful to me. I understand the compensation provisions for this study. I have had time to consider whether to take part. I know who to contact if I have any questions about the study.

I wish to receive a copy of the results. YES/NO

I ____________________ (Print name) hereby consent to take part as a participant in this research.

Signed ____________________ (participant). date_____.

and ____________________ Family member/caregiver.

In my opinion, consent was given freely and with understanding.

Witness name: ____________. Witness Signature ____________________

If you have any queries or concerns about your rights as a participant in this study you may wish to contact Health Advocates Trust, telephone (09) 623 5799.

Signature ____________________ Date ____________________

Researchers: Kathryn Russell, Ph 2676659, 4861491 extn 2868. Lynette Tippett, Ph 3737599 extn 8551.
Appendix VII

History Questionnaire.
Participant:
Current Age:
DOB:

Educational attainment:
What age did they leave school?
Did they gain any further education?
Where?
How long?
Occupational History:

Drug and alcohol use:
What is the alcohol intake in units per week, what is the maximum on any one occasion?
Were they ever considered a heavy drinker?
Did they ever have a drink related problem?
Did they every have a drug dependence problem?

Past psychiatric history?
Have they ever required treatment for an emotional, nervous of psychiatric illness?

Relevant medical history?
Head injuries?
Strokes?
Any major heart problems?
Any major surgery?
Seizures/Fits?

Medications:
What medications are they currently on?

History of memory problems:
When did the memory problems begin?
When were they diagnosed?
Appendix VIII

Short Token Test (De Renzi & Faglioni, 1978).
Score: 36.

Part I (LARGE TOKENS ONLY)
1. Touch a circle
2. Touch a square
3. Touch a yellow token
4. Touch a red one
5. Touch a black one
6. Touch a green one
7. Touch a white one

Part II
1. Touch the yellow square
2. Touch the black circle
3. Touch the green circle
4. Touch the white square

Part III (ADD SMALL TOKENS)
1. Touch the small white circle
2. Touch the large yellow square
3. Touch the large green square
4. Touch the small black circle

Part IV (REMOVE SMALL TOKENS)
1. Touch the red circle and the green square
2. Touch the yellow square and the black square
3. Touch the white square and the green circle
4. Touch the white circle and the red circle

Part V (ADD SMALL TOKENS)
1. Touch the large white circle and the small green square
2. Touch the small black circle and the large yellow square
3. Touch the large green square and the large red square
4. Touch the large white square and the small green circle

Part VI (REMOVE SMALL TOKENS)
1. Put the red circle on the green square
2. Touch the black circle with the red square
3. Touch the black circle and the red square
4. Touch the black circle or the red square
5. Put the green square away from the yellow square
6. If there is a blue circle, touch the red square
7. Put the green square next to the red circle
8. Touch the squares slowly and the circles quickly
9. Put the red circle between the yellow square and the green square
10. Touch all the circles, except the green one
11. Touch the red circle – no – the white square
12. Instead of the white square, touch the yellow circle
13. In addition to touching the yellow circle, touch the black circle
Appendix IX


A. Toilet
   1. Cares for self at toilet completely, no incontinence
   2. Needs to be reminded, or needs help in cleaning self, or has rare (weekly at most) accidents.
   3. Soiling or wetting while asleep more than once a week.
   4. Soiling or wetting while awake more than once a week.
   5. No control of bowels or bladder.

B. Feeding
   1. Eats without assistance.
   2. Eats with minor assistance at meal times and/or with special preparation of food, or help in cleaning up after meals.
   3. Feeds self with moderate assistance and is untidy.
   4. Requires extensive assistance for all meals.
   5. Does not feed self at all and resists efforts of others to assist in feeding.

C. Dressing
   1. Dresses, undresses and selects clothes from own wardrobe.
   2. Dresses and undresses self, with minor assistance.
   3. Needs moderate assistance in dressing or selection of clothes.
   4. Needs major assistance in dressing, but co-operates with efforts of others to help.
   5. Completely unable to dress self and resists efforts of others to help.

D. Grooming (neatness, hair, nails, hands, face, clothing.)
   1. Always neatly dressed, well-groomed, without assistance.
   2. Grooms self adequately with occasional minor assistance, e.g., shaving.
   3. Needs moderate and regular assistance or supervision in grooming.
   4. Needs total grooming care, but can remain well-groomed after help form others.
   5. Actively negates all efforts of others to maintain grooming.

E. Physical Ambulation (Movement)
   1. Goes about grounds or city.
   2. Ambulates within residence or about one block distance.
   3. Ambulates with assistance of (check one) a) another person, b) railing, c) cane, d) walker, e) wheelchair.
   4. Sits unsupported in chair or wheelchair, but cannot propel self without help.
   5. Bedridden more than half the time.

F. Bathing
   1. Bathes self (tub, shower, sponge bath) without help.
   2. Bathes self with help in getting in and out of tub.
   3. Washes face and hands only, but cannot bathe rest of body.
   4. Does not wash self but is co-operative with those who assist in bathing.
   5. Does not try to wash self and resists efforts of assistance to keep clean.
Appendix X


A. Ability to use Telephone.
   1. Operates telephone on own initiative, looks up and dials numbers, etc.
   2. Dials a few well-known numbers.
   3. Answers telephone but does not dial.
   4. Does not use telephone at all.

B. Shopping.
   1. Takes care of all shopping needs independently.
   2. Shops independently for small purchases.
   3. Needs to be accompanied on any shopping trip.
   4. Completely unable to shop.

C. Food Preparation.
   1. Plans, prepares and serves adequate meals independently.
   2. Prepares adequate meals if supplied with ingredients.
   3. Heats and serves prepared meals, or prepares meals but does not maintain adequate diet.
   4. Needs to have meals prepared and served.

D. Housekeeping.
   1. Maintains house alone or with occasional assistance (e.g., heavy work-domestic help)
   2. Performs light daily tasks such as dishwashing, bedmaking.
   3. Performs light daily tasks but cannot maintain acceptable level of cleanliness.
   5. Does not participate in any housekeeping tasks.

E. Laundry.
   1. Does personal laundry completely.
   2. Launders small items, rinses socks, stockings, etc.
   3. All laundry must be done by others.

F. Mode of Transportation.
   1. Travels independently on public transport or drives own car.
   2. Arranges own travel via taxi, but does not otherwise use public transport.
   3. Travels on public transportation when assisted or accompanied by another.
   4. Travel limited to taxi or automobile with assistance of another.
   5. Does not travel at all.

G. Responsibility for own Medications.
   1. Is responsible for taking medication in correct dosages at correct time.
   2. Takes responsibility if medication is prepared in advance in separate dosages.
   3. Is not capable of dispensing own medication.

H. Ability to Handle Finances.
   1. Manages financial matters independently (budgets, writes checks, pays rent, bills, goes to bank), collects and keeps track of income.
   2. Manages day-to-day purchases, but needs help with banking, major purchases, etc.
   3. Incapable of handling money.
Appendix XI
Appendix XIa

Procedural Training Method: Description and Examples

In order to replicate the procedural training method researchers need to be aware not only of the key features as described on page 64, but also of the practical details of implementing the method. In both of the studies described in this thesis the precise structure of tasks varied across subjects according to their living situation, habits and specific life demands. Despite this variability in the tasks themselves there are elements of the training method that were consistent across tasks and across participants.

As mentioned on page 64 the aim of training was repeated successful performance on a skill or task, in other words performance without errors. The most important element of the method was that errors were immediately noticed and corrected, i.e., there was a synchronicity between the onset of an error and the correction. This was achieved via verbal prompts, cues, answers to questions and verbal corrections of mistakes (given to the participant step-by-step). When these tasks were repeated it was important that they were performed the same way each time to achieve the repetition necessary for procedural learning.

Tasks were based on participants habits (e.g., making tea with a teapot verses putting the teabag in the cup) and also their likes and dislikes (e.g., jam sandwiches verses cheese sandwiches). So the first step before implementing the training was to establish the task requirements.

During the training period the researcher and participant would move to the appropriate location (e.g., kitchen, lounge, bedroom). The researcher would check that there was nothing unusual present in the environment that would interfere with successful completion of the task (e.g., tidying away extra knives on a kitchen based task). This checking rarely resulted in changes to the testing environment. Usually testing was done with the environment as it was found and objects were kept in their usual places (e.g., toothbrush and paste in the holder by the sink in the bathroom).

Before training a task, a further decision must be made. That is whether objects relating to a task are found for the participant, or whether finding the objects is part of the task. If a specific piece of equipment is required then it may be necessary for the trainer to find that item, for example if training an AD individual how to put on and do up a specific pair of shoes. In contrast for some tasks finding
the items may be an important component of the task, for example making a sandwich. A mild AD individual may be able to find the objects needed for making a sandwich however struggle putting these together to make a sandwich; a situation ideal for procedural training. As AD progresses some of the ‘finding’ and ‘identifying’ elements of these tasks may make training them difficult. For example a more moderate AD individual may not be able to find or decide on the items necessary, or they may not be able to identify the necessary objects. Training could then involve making a sandwich, but not finding the ingredients (the items would be put out on the kitchen bench ready for them). However this may not be useful for their daily lives unless caregivers are willing to prepare task items for them. If caregivers can not perform this function, it is probably more useful in moderate and moderately severe AD to train tasks that have the necessary objects present. For a detailed list of tasks in relation to level of impairment see the guidelines in the General Discussion, pages 177 & 178.

Once task requirements are established they are incorporated into the directions, which are stated before the task began. For example: I would like you to make a jam sandwich, I would like you to make a cup of tea with milk or I would like you to put on this cardigan.

The best way to illustrate the procedural learning method is with detailed examples.

**Example One: Mild-Moderate AD**

In Baseline this participant would put soap on her toothbrush instead of toothpaste.

**Trainer:** I’d like you to clean your teeth

*Participant reaches for toothbrush*

**Trainer:** Good

*Participant holds toothbrush in right hand and reaches for soap*

**Trainer:** You need the toothpaste (*Trainer points at toothpaste*)

*Participant reaches for toothpaste in left hand and pauses*

**Trainer:** Now put down the toothbrush

*Participant puts down the toothbrush*

**Trainer:** OK. Take the lid off the toothpaste

*Participant takes the lid off the toothpaste and pauses*
Trainer: Good. Pick up the toothbrush, … that’s right, and squeeze some toothpaste onto the brush, … right

Participant then puts water on the brush and proceeds to brush her teeth correctly, including rinsing her mouth and putting the brush and toothpaste back in the jar.

Trainer: That was good. Then they have a break – talk about something else and begin the process again (5x per day, over 10 days)

Note that the participant only received instructions when she started making an error. These instructions were given step-by-step, with one component at a time and continued until she started doing the task without hesitation and without error.

In the above example reassurance and positive statements were used, in addition to the corrections, to make the experience less unpleasant. When errors occur, terms such as ‘wait’, ‘hold on’, ‘OK now I would like you to ….’ can be used. It is not important for participants to know what they are doing wrong. The trainer just needs to prevent the errors from happening and make sure the correct procedure is repeated.

**Example Two: Moderate-Severe AD**

In Baseline this participant would walk up to the sliding screen door, put her hands on the mesh and push, eventually she would put her finger through the hole in the mesh and either shake the screen or slide the door open.

Trainer: I would like you to open the screen door

Participant stands at the screen door and puts both hands up onto the mesh

Trainer: Put your hand on that metal handle (pointing)

Participant: No response

Trainer: Move over here (beckons) … that’s right

Participant moves closer

Trainer: Put your hand on this, like this (trainer demonstrates)

Participant puts her hand out and touches the handle with a pointed finger

Trainer: Good! Put your hand on the handle again, I’m going to show you how to hold it

Participant touches handle again
**Trainer** moves participant’s hand around the correct way, curls her fingers onto the handle and slides the door open still holding her hand

**Trainer:** That’s it! Good!

**Trainer** closes door

**Trainer:** Lets do that again…. I would like you to open the screen door…. (e.t.c)

Training must relate to individuals’ abilities and needs in regard to task selection. Some participants may have deficits that interfere with the procedural learning method success for some tasks. For example an individual in the moderate stage of AD may struggle to set the table because she can not decide on the number or type of objects required and when she does put objects on the table she may display clear visual spatial difficulties.

**Summary**

There are several key elements to the training:

1. Do the task the same way each time – limit decisions in the tasks and in the directions
2. Prevent errors occurring in order to facilitate errorless performance
3. The corrections/prompts/guidance/demonstration needs to occur at the time of the error (preferably at the start of the error)
4. Various methods can be used to achieve correct performance such as cues, pointing, verbal instructions, and even physically moving their hand.
5. Give guidance step-by-step in small, simple sentences
6. Use reassurance and positive encouragement
7. The AD individual does not need to know what they are doing wrong, they only need to repeat the task correctly
8. Keep objects in the same place, preferably visible
9. Be aware of the AD individual’s limitations e.g., visual spatial problems
Appendix XII

Caregiver Follow-up Interview.

I would like to get a general picture of how the training programme affected your family member, so that I can see if there are effects of training other than those I measured in the follow-up visits. I’m going to ask you some questions about anything you noticed during the training period.

**Question One**
Over the three week training period did you notice any changes in _____’s behaviour? By behaviour I mean, for example, being more or less active, or being more fidgety etc.?

**Question Two**
Over the training period did you notice any physical changes, for example: was _____ more tired than usual, or did they have headaches etc.?

**Question Three**
Where there any changes in _____’s mood, for example was he/she more sad, happy, or angry than usual?

**Question Four**
Was _____ nervous or anxious about the training programme (if yes, on the first few days only or throughout the entire time?)

**Question five**
What (if anything) did _____ dislike most about training?

**Question six**
What (if anything) did _____ like most about training?

**Question seven**
In general how do you think _____ has changed over the three week training period?

**Question eight**
How has _____ been since the training was completed?

**Question nine**
Is there anything else you would like to add?
Information Sheet

Title: Retraining or improving living skills in people with memory problems.

Principal Investigator:

This study is being carried out by Kathryn Russell and Dr. Lynette Tippett.

Kathryn Russell is a PhD student in the Department of Psychology at the University of Auckland. Phone: 529-4040 (Hm), 021-2159719 (Mobile).

Dr. Lynette Tippett is a Lecturer in the Department of Psychology at the University of Auckland. Ph 373-7599 extn: 8551.

Introduction:

You are invited to take part in a research study which will examine how well tasks that are causing some difficulty can be relearned in individuals with memory problems, following a training programme. You are not obliged to participate in this study. If you would like to participate, however, we would be grateful if you could let us know by phone or letter. We would like you to make a decision about your participation within the next fortnight, but you may take longer if necessary.

About the Study:

The aim of the study is to see if people with memory problems can relearn daily living tasks that are causing them some difficulty. These tasks will be chosen according to the particular needs of each individual in the study. We wish to see if a particular way of training is effective and if it is, how long the beneficial effects of training last.

Ten people will be included in this study.

To take part in this study you (or your family member) must have memory problems, speak English and not be on medications for Alzheimer's Disease. You must not have a history of heart disease or any other neurological disorders. The assessment phase of the research may find that some people will not be able to benefit from the training. Please do not be offended if this is the case, it only means your personal needs can not be met by this type of training.
The study will take place at your own home. The first visit involves identifying which areas of daily living skills are causing you difficulty by talking to you and your family member, so we can decide which tasks might get better with training. You and your caregiver/family member will answer some questions about your background, you will complete a short task involving memory and language, a short comprehension test and a test involving a simple motor skills test. This assessment will take between one and two hours to complete. The caregiver is only required at the initial assessment and for an interview at the end of the research. You do not have to answer all the questions, and you may stop the interview at any time.

One the next visit you will be observed performing the tasks that you are having difficulty with. The time it takes, and any errors made will be recorded by Kathryn Russell, this will be done over 5 days.

The next phase of the research is training. This will take up to two hours every weekday for 14 days. Training will involve being shown how to do the activities and practising them in a special way.

After training there will be four follow up visits to see if the training is still working. On the follow up visits the tasks will be timed and Kathryn Russell will observe how you are doing the tasks. These will take no more than one hour and will occur 2 weeks, 4 weeks, 8 weeks and 12 weeks after the training has finished. At this time the family member/caregiver will be asked some questions about what they thought of the training. The caregiver/family member does not have to answer all the questions, and may stop the interview at any time.

Information about you will be kept confidential, and secure.

Benefits, Risks and Safety.

The benefits you may receive from taking part in the study is that you may relearn one or more daily tasks that are currently frustrating for you. This study will also help increase our understanding of memory and learning.

There are no expected risks, although the training sessions will take up to 2 hours per day and you may become a little tired. You will be able to stop and have breaks when you need them.

Taking part in this study will not cost you anything. There is no payment or reimbursement for your time.

There are no alternate training programmes currently available to improve self care skills for individuals with memory difficulties.
Compensation:

In the unlikely event of a physical injury as a result of your participation in this study, you will be covered by the accident compensation legislation with its limitations. If you have any questions about ACC please feel free to ask the researcher for more information before you agree to take part in this trial.

Participation.

Your participation is entirely voluntary (your choice). You do not have to take part in this study, and if you choose not to take part, this will not affect any future care or treatment.

If you do agree to take part, you are free to withdraw from the study at any time, without having to give a reason and this will in no way affect your future health care.

Participation in this study will be stopped should any harmful effects appear.

General information.

There will be no additional training at the conclusion of the study, but you and your family will be able to discuss your results with your health professionals if you wish.

If you, or a relative or friend, have any questions or wish to know more about the study please phone Kathryn on 529-4040 (Home) or 021-215-9719 (Mobile). Alternatively you may contact her by letter at The Professional Psychology Unit, Department of Psychology, University of Auckland, Private Bag 92019, Auckland.

If you have any queries or concerns about your rights as a participant in this study you may wish to contact Health Advocacy Trust, telephone: 0800-205-555 Northland to Franklin.

Confidentiality.

No material which could personally identify you will be used in any reports on this study. The notes and results will be checked by Kathryn Russell and Dr. Lynette Tippett only. After that all results will be related to a participant number with no identifying details.

All data will be kept strictly confidential and your privacy will be protected. Your performance will remain confidential to the investigators during the study. At the completion of the study all records will be locked away in filing cabinets for up to 10 years. It will then be destroyed.
Results.

The results of your performance will be available to you at the conclusion of testing, these will be explained to you upon request. Overall findings from this study will be published in an international journal, no identifying details about you will be included. There can be a delay between research and publication of over 1 year.

This study has received ethical approval from the NFA Auckland Ethics Committee.

Please feel free to contact the researcher if you have any questions about this study.
CONSENT FORM

Retraining or improving living skills in people with memory problems.

1. I have read and I understand the information sheet dated ..........99 for volunteer taking part in the study designed to retrain or improve daily living skills. I have had the opportunity to discuss this study. I am satisfied with the answers I have been given.

2. I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time and this will in no way affect my future health care.

3. I understand that my participation in this study is confidential and that no material that could identify me will be used in any reports on this study.

4. I understand that the training will be stopped if it should appear harmful to me.

5. I understand the compensation provisions for this study.

6. I have had time to consider whether to take part.

7. I know whom to contact if I have any negative effects to the study.

8. I know whom to contact if I have any questions about the study.

9. I would like the researcher to discuss the outcomes of the study with me.

YES/NO

I __________________________ (full name) hereby consent to take part in this study.

__________________________ (full name) hereby consent to take part in this study, and

__________________________ (Full name) hereby consent to take part in this study, and

to give consent for my family member with Alzheimer’s Disease to take part in this study also.

__________________________ (Signature)

Researchers: Kathryn Russell & Dr. Lynette Tippett.
Contact Kathryn Russell: Home: 529-4040, Mobile: 021-215-9719
Project explained by Kathryn Russell (Principal investigator).
Signature: __________________________

Date: ______________

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STEP 1: PROBLEM DEFINITION
To identify occupational performance problems interview the client using these questions as guidelines.
(i.e. self-care, productivity and leisure).

If yes to either a, b, or c
a. Do you need to do _____________________________ ?

b. Do you want to do ____________________________ ?

c. Are you expected to do _________________________ ?

And no to either d, e, or f

d. Can you do _____________________________ ?

e. Do you do _____________________________ ?
f. Are you satisfied with the way you do _______________________ ?

Then go on to identify specific problems.
If no to a, b, or c OR yes to d, e, or f then proceed to the next area.

STEP 1A: Self-Care

Personal Care (e.g. dressing, bathing, feeding, hygiene)

Functional Mobility (e.g. transfers, indoor, outdoor)

Community Management
(e.g. transportation, shopping, finances)

1B: Productivity

Paid/Unpaid Work (e.g. finding/keeping a job, volunteering)

Household Management
(e.g. cleaning, laundry, cooking)

Play/School (e.g. play skills, homework)

1C: Leisure

Quiet Recreation (e.g. hobbies, crafts, reading)

Active Recreation (e.g. sports, outings, travel)

Socialization (e.g. visiting, phone calls, parties, correspondence)

STEP 2: PROBLEM WEIGHTING
Using the scoring cards provided, ask the client to rate, on a scale of 1 to 10, the importance of each activity.
Place the ratings in the corresponding boxes.

STEP 3: SCORING
Ask the client to choose the 5 most important problems and record them below.
Using the scoring cards, have the client rate each problem on performance and satisfaction.
Then calculate the weighted scores.

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<th>SATISFACTION</th>
<th>IMP x PERF</th>
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TOTALS:
TOTAL IMP x PERF 1/# of problems = Performance Score 1
TOTAL IMP x SAT 1/# of problems = Satisfaction Score 1

STEP 4: REASSESSMENT
Re-evaluate each problem at a suitable interval in terms of performance and satisfaction.
Calculate the new weighted scores and the change.

| PROBLEMS | IMP x PERF | IMP x SAT | IMP x PERF | IMP x SAT | IMP x PERF | IMP x SAT | IMP x PERF | IMP x SAT | IMP x PERF | IMP x SAT | IMP x PERF | IMP x SAT | IMP x PERF | IMP x SAT | IMP x PERF | IMP x SAT |
|----------|------------|-----------|------------|-----------|-----------|-----------|------------|-----------|-----------|------------|-----------|-----------|-----------|------------|-----------|------------|-----------|
|          |            |           |            |           |           |            |            |           |           |            |           |           |            |            |           |            |           |
|          |            |           |            |           |           |            |            |           |           |            |           |           |            |            |           |            |           |
|          |            |           |            |           |           |            |            |           |           |            |           |           |            |            |           |            |           |

CHANGE IN PERFORMANCE = Performance Score 2 - Performance Score 1
CHANGE IN SATISFACTION = Satisfaction Score 2 - Satisfaction Score 1

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IMPORTANCE

1 2 3 4 5 6 7 8 9 10
not important at all extremely important

PERFORMANCE

1 2 3 4 5 6 7 8 9 10
not able to do it able to do it extremely well

SATISFACTION

1 2 3 4 5 6 7 8 9 10
not satisfied at all extremely satisfied