CHAPTER ONE
INTRODUCTION

1.1 PRELIMINARY PERSPECTIVES

This study examines the impact of stroke on Executive Functions (EF) and explores some dimensions of assessment and therapy. As a speech language pathologist, the complexity of conversation and its disruption in the wake of neurological damage has always held interest. Like general intelligence, the skill with which a person manages everyday interactions is multifaceted and represents a complex, nuanced process, reflecting what this study is going to term “conversational intelligence”. Neurological damage has pervasive effects on conversational ability with the ultimate consequence being the alienation of individuals from their social contexts. Despite significant interest in the application of ecologically valid treatment approaches that target communication in the real world, satisfactory levels of remediation or compensation are often difficult to achieve. New developments in the fields of neurology, pharmacology, neuropsychology and neuroimaging hold promise for offering potential solutions to the dilemmas faced when treating people with stroke induced communication deficits.

Stroke is the most common neurological disorder and is the third most common medical condition after heart attacks and cancer in the developed world (Mlcoch & Metter, 1994). In sub-Saharan Africa, stroke and other vascular diseases increasingly contribute to the burden of disease (Connor, Walker, Modi & Warlow, 2007). In fact Thorogood, Connor, Tollman, Hundt, Fowkes and Marsh (2007) have stated that South Africa is facing the challenge of an emerging epidemic of vascular disease. However, as Connor et al. (2007) point out, there are no systematic reviews of stroke mortality, prevalence, and incidence and case fatality.

Among the most disabling of all the sequelae of stroke are communication deficits. These impairments impact on daily activities and quality of life in ways that profoundly affect both the stroke patient and significant others in their lives. While language and communication therapies have advanced, there are still many clients who do not appear to benefit optimally
from therapy. More specifically, there is a considerable sub-group of patients who master a number of communication strategies in the clinical setting yet fail to transfer them successfully to every day environments (Purdy, 1992). In addition, there are groups of clients who appear to reach a plateau and regardless of the approach used, stop benefiting from intervention. Strikingly, many of these clients perform well on formal language assessments, which is misleading when taking into account the significant difficulties they have communicating in conversational settings.

In order to understand this discrepancy between clinical assessment and real world functioning, speech language pathologists have begun to turn to other disciplines such as neuropsychology, neurology and pharmacology. One of the reasons recently proposed to account for these clinical observations is the presence of co-existing executive function deficiency in individuals with strokes. There is increasing recognition that the attribution of communication problems in this population to deficits of language or symbolic representation in the case of aphasia and pragmatic deficits in the case of right hemisphere disorders, are no longer sufficiently inclusive. Communicative success may depend on the integrity of executive functions (EF) which are called into play when we are involved with complex, novel activity. The availability of organisational, planning and learning processes has been shown to be associated with faster recovery (Bailey, Powell & Clark, 1981). These functions along with generating, selecting and applying strategies are frequently disrupted in individuals with brain damage resulting from stroke (Burgess & Shallice, 1996; Lawson & Rice, 1989; Owen, Downes, Sahakian, Polkey & Robbins, 1990). The ultimate goal of therapy for clients with communication difficulties arising from stroke is to improve communication in everyday settings where unpredictable demands and fluctuating conditions are inherent. To achieve success, goal oriented behaviour and flexible problem solving is needed – skills that are intrinsically executive.

Therefore the lack of ability to generalise treatment gains may be attributed to impaired strategy generation where patients would be less likely to make use of trained methods for circumventing language difficulties (Keil & Kaszniak, 2002). Purdy (2002) hypothesised that individuals with aphasia ‘lack the ability to initiate, plan, monitor and correct their communicative performance and thus are unable to use their available verbal and nonverbal abilities to achieve their goals’ (Purdy, 2002, p.5). In this way EF deficit may further interact with or amplify limitations due to language disorders (Keil & Kaszniak, 2002). Language
treatments themselves may be limited if the patient has impairments in strategy application or abstract reasoning (Reitan, 1988).

Despite increasing recognition of the importance of EF to communication, existing research has not yet produced comprehensive assessments of the EF deficits associated with strokes. Isolated, although complex dimensions of EF have been investigated, with particular emphasis on attention and working memory capacities but these areas are by no means inclusive of all constructs considered executive. What is needed is a theoretically sound framework upon which to base the construction and administration of a battery designed to tap a broad array of executive constructs. Neuropsychological profiling can be of particular use here to define and delineate the executive features of individuals with stroke related brain damage.

In terms of incorporating neuropsychological findings with communication assessment, the domain most reflective of the complex integration of language and executive skills is conversation. The ability to analyse the communicative environment to which the stroke patient needs to adapt through conversation, allows an in-depth assessment of the structure of the interaction, providing insight into the individual’s conversational flexibility in real time and real life settings – essentially a reflection of novel and complex adaptive behaviours - the domain of executive functioning (Penn, 2000). The ability to participate meaningfully in conversational discourse requires paying attention to a communication partner, sustaining attention, appropriately sequencing pieces of information, monitoring ongoing communication processes and flexible shifting strategies (Miyake, Emerson & Friedman, 2000). In their study of individuals with aphasia completing dual tasks, Miyake et al. (2000) found that those who did not evidence performance decrement were also those who had been rated as being effective communicators, whereas, those who were rated as being ineffective communicators showed marked deterioration, even though the degree of language impairment was relatively comparable for both groups. These findings were interpreted to reflect crucial aspects of EF.

It appears that formal language tests are not sufficiently taxing to stress the executive system to a degree that they uncover deficits of executive function, nor do they appear able to predict whether or not individuals will cope with the demands of conversation (Hardin & Ramsberger, 2004; Hayashi, Ramsberger & Menn, 2000). There is an implication that EF may therefore be more observable in conversation than on some formal language tests.
A precedent for the relationship between executive characteristics and conversational features was established in a previous study by the author (Frankel & Penn, 2007). Two individuals who had sustained Traumatic Brain Injury (TBI) following motor vehicle accidents participated in a study with a similar design to this one. They were assessed on a specifically designed executive battery and recorded having naturally occurring conversations with familiar interlocutors over the course of a four phase drug trial. Two results were particularly relevant to the current discussion. The first was that for both participants with TBI, unique executive profiles showed strong relationships to verbal phenomena (different manifestations of perseveration) at a conversational level. These preliminary results suggested that EF may have a significant impact on verbal communication and that these influences were discernible in conversation. More than any other form of communication assessment, the inclusion of an assessment of participation in conversation appears to be critical.

The second important result was that both individuals showed positive responses to pharmacological intervention (Ritalin), which was measurable on fundamental aspects of the EF battery as well as in conversation. The findings are also relevant because advanced knowledge regarding mechanisms of stroke and brain recovery suggest that the neuroanatomical and neurochemical environment of the brain, particularly the penumbra (the area surrounding the infarct), needs to be stabilised in order to facilitate recovery and enhance therapeutic endeavours (Weiller, 2000). This understanding has contributed to numerous drug trials, examining the impact of various pharmacological agents on brain and subsequently functional recovery. Research studies have not convincingly demonstrated overwhelming evidence to support the routine use of drugs as either a complementary or alternative treatment in stroke patients, especially as an adjunct to aphasia therapy. Rigorous research is still needed to determine which patients would benefit from which types of drug therapy. Relative to the preceding discussion, pharmacological agents of particular interest are those whose features include protection against brain insults, low toxicity, potential for enhancing learning and importantly alleviation of impaired executive dysfunction thus enhancing the efficacy of higher integrative mechanisms of the brain (Neyens, Alpherts & Aldenkamp, 1995).

In its most severe form, the onset of focal neurological deficit is so severe as to render the patient comatose or lead to immediate death. In its mildest form the effects are so subtle as to go unnoticed. Yet the long term consequences of stroke are far from subtle and disrupt every
aspect of daily life. The effects of stroke have been studied for well over a century and with constantly evolving technology and the opportunities to study the brain in ever more sophisticated ways, interest in brain-behaviour relationships has never been more intense. Despite the growing interest in aspects of EF and their relevance to communication, there is a conspicuous gap in the literature related to the comprehensive assessment of EF deficit in stroke related communication problems. This research presents a study of EF as a central component of conversational intelligence and its impact on conversation in individuals with stroke. The response to a carefully chosen pharmacological agent (Leviteracetam) is also determined.

1.2 OUTLINE OF THE FOLLOWING CHAPTERS

There are three introductory chapters, each detailing critical components that form the focus of this study. Chapter 2 provides a contextual orientation in terms of stroke in South Africa. The context is important to understand in terms of the social and economic repercussions for areas of sub-Saharan Africa at early stages of economic and health transition (Thorogood et al., 2007). In the absence of ready access to sophisticated neuroimaging and hemodynamic apparatus the marrying of two diverse assessment and research paradigms (Conversation Analysis and neuropsychology) may therefore provide useful yet cost effective avenues for meaningful assessment within this context. The mechanisms of stroke and factors affecting recovery are described, establishing neuroanatomical integrity and stability of the neurochemical systems as crucial aspects that contribute to conversational intelligence. Against this background, the communication disorders following stroke are discussed with reference to aphasia and Right Hemisphere Disorders (RHD). This chapter concludes with an evaluation of both traditional as well as modern approaches to the treatment of stroke related communication disorders, highlighting conversation and pharmacotherapy as two more recent developments.

Chapter 3 presents a description of executive function (EF) in terms of its definitions, its neuro-anatomical representation in the brain and its development throughout the lifespan. Intricate neurochemical and behavioural relationships are discussed in depth to highlight the complexity of the constructs under discussion and the challenges of choosing an appropriate drug for investigation. Disclosure of the nature of EF renders its potential involvement in conversational intelligence clear. The chapter concludes with a presentation of current theories of EF culminating with Barkley’s hybrid model (1997) which forms the framework for this research.
Chapter 4 relates the previous discussion to specific EF deficits in the presence of a variety of neurological communication disorders. This chapter highlights the fact that perhaps counter-intuitively, some of the EF deficits previously associated with more diffuse lesions or disease processes, are found as a consequence of stroke as well. The chapter concludes with a summary of the areas that would benefit from further investigation as well as the inherent challenges in such an undertaking.

Chapter 5 provides the aims and general methodology of this study, together with the aims, procedures and findings for preliminary investigations that applied to this work. Chapter 6 presents the results of this study, integrating the findings with existing literature where applicable and demonstrating the significance of EF as a critical component of conversational intelligence. With reference to the previous chapters on EF and various manifestations of deficit in neurogenic communication disorders, the chapter concludes with an analysis of the profiles obtained with regard to site of lesion and communication disorder classification.

Finally Chapter 7 summarises the investigation, highlighting general findings and critiquing methodological issues. This work is concluded with a discussion of the clinical and research implications derived.
Stroke is a devastating condition with pervasive influences on the lives and psychosocial wellbeing of the stroke survivor and their significant others. Within a South African context, stroke is low on the list of priorities relative to communicable and poverty-related diseases with regards prevention and management. Within this context, it is critical to examine aspects of disability associated with stroke that provide insight into every day experiences and which suggest treatment interventions that will impact in an ecologically valid manner. This chapter explores stroke within the South African context, in terms of mortality, prevalence and incidence. The mechanisms of stroke with data highlighting the stroke types and subtypes in South Africa are described as well as current approaches to the processes facilitating recovery. The chapter provides a description of the communication disorders resulting from stroke relative to aphasia and right hemisphere communication disorders as well as historical and modern approaches to treatment. In this way this chapter presents some of the critical contributors to conversational intelligence relevant to the current study, namely neurochemical stability, neuroanatomical integrity and neurolinguistic processing. Conversation and pharmacology emerge as recent and valuable therapeutic developments.

2.1 THE SOUTH AFRICAN CONTEXT

Stroke is the second most common cause of death worldwide (Murray & Lopez, 1997) with two-thirds of these deaths occurring in developing regions of the world, such as sub-Saharan Africa (SSA) (Connor & Bryer, 2006). The incidence of stroke in developing countries is expected to rise in the future as the populations undergo what has been called the ‘health transition’ (Reed, 1990). Although the major health burden in SSA are infectious diseases and diseases related to poverty and malnutrition, urbanisation is predicted to increase the risk factors for vascular disease and hence lead to a sharp increase in stroke, such as is found in developed countries.

Prevention and management of chronic diseases and the unhealthy lifestyles and risk factors that precede them is heavily influenced by the quadruple burden of disease within South Africa
Disease patterns in this country are characterised by a combination of poverty-related illnesses together with the emerging chronic diseases associated with urbanisation, industrialisation and a westernised lifestyle. This double burden of diseases is exacerbated by high injury rates associated with high levels of crime and by the epidemic of HIV/AIDS across the African continent. This multiple burden represents a demand on the health services of South Africa far beyond those experienced in developed countries and what the limited resources can accommodate. Because little recognition is given to the magnitude of the burden of chronic disease of lifestyle (CDL) in South Africa and prevention of unhealthy lifestyles, early diagnosis and cost-effective management of CDL risk factors are low on the list of priorities compared to other competing groups of diseases (Steyn, 2006).

The burden of stroke includes mortality, prevalence and incidence (Warlow, 1998). Burden also refers to the high morbidity rate which leaves up to 50% of survivors chronically disabled (Wilkinson, Wolfe, Warburton, Rudd, Ross Russell & Beech, 1997). Although it would also be helpful to know what the economic burden of the disease is in terms of its impact on the health service directly, and on the individual, family and community, both directly and indirectly, financially and psychosocially, the truth is that very little known is about socio-economic impact of stroke in South Africa (Connor & Bryer, 2006). More significantly, accurately understanding the burden and nature of stroke in this population allows insight into where the population is along the health transition and allows for appropriate health service planning for acute services and primary and secondary prevention. However in South Africa this task is complicated by different population groups and socio-economic structures. Not only does the relative importance of risk factors for stroke and cardiovascular disease differ between population groups globally (Warlow, 1998), but as a result of socio-economic and past political influences we have population groups that in general reflect different stages of the health transition (Connor & Bryer, 2006).

### 2.1.1 Mortality of stroke

Traditionally, mortality figures are derived from the vital registration data. However, the HIV/AIDS epidemic has caused dramatic changes in demographic features and mortality profiles (Bradshaw, Groenewald. Laubscher, Nannan, Nojilana et al., 2003a) and therefore more complex models have been devised to estimate cause of death, including stroke. From these ‘Initial burden of disease estimates for South Africa, 2000’, stroke was found to be the
fourth most common cause of death, accounting for 6% of all deaths in 2000, with estimations showing more female than male deaths. Stroke is therefore the most important non-communicable disease which causes death in females, compared to ischaemic heart disease in males (Bradshaw, Groenewald, Laubscher, Nannan, Nojilana et al., 2000b). The MRC/Wits (Agincourt) Rural Public Health and Health Transition Research Unit found stroke caused 5.5% of all deaths in a rural population of 63 000 in Limpopo Province between 1992 and 1995. In this study the sensitivity and specificity for stroke was 87% and 97% respectively (Kahn & Tollman, 1999). Stroke was the most common cause of death in the 55-74-year-old age group. While in the 35-54-year-old and ≥ 75-year-old age groups, it was the second commonest cause of death after assault and congestive heart failure respectively.

2.1.2 Prevalence of stroke
In high-income countries, stroke prevalence studies are usually conducted telephonically or using postal surveys. However in South Africa identifying people with stroke at home, particularly outside urban areas, is fraught with difficulty (Connor & Bryer, 2006). The Southern African Stroke Prevention Initiative (SASPI) recently published the first stroke prevalence study from South Africa based on the Agincourt demographic and health surveillance site in Limpopo. The crude prevalence was 300/100 000 after a correction was made by sex and 10-year age band for those not examined who screened positive. A significant finding of this study was the prevalence of stroke survivors’ needing help with at least one activity of daily living (200/100 000) (Connor, Thorogood, Casserly, Dobson & Warlow, 2004). These individuals require the most help and place the greatest burden on the family, community and health services. As yet there is no related data on the prevalence of communication disorders arising from stroke, although this is clearly an area of research needing attention.

2.1.3 Stroke incidence and case fatality
Neither stroke mortality, nor stroke prevalence studies provide accurate data on the nature of stroke types and the exact cause of the stroke, all of which are best determined within hours or days after the onset of a first-ever stroke. The best epidemiological studies of stroke are therefore community-based incidence studies of first-ever-in-a-lifetime stroke with long term follow up of case fatality (Warlow, 1998). As yet no community-based stroke incidence studies have been done in Southern Africa because of the difficulty of performing them (Connor, Warlow & Fritz, 2000). There have been urban hospital-based studies from South Africa and
Zimbabwe that have attempted to estimate stroke incidence in these regions. The same is true for case fatality data. According to hospital-based urban stroke registers, fatality ranging between 33% and 35% at one month post stroke has been recorded (Joubert, 1991; Matenga, 1997; Rosman, 1986). However, stroke is a heterogeneous condition and stroke types have significant influence on incidence data. As an example Rosman (1986) found case fatality to be 22% at one month for cerebral infarction but 58% for cerebral haemorrhage in Pretoria.

2.1.4 Stroke types and subtypes in South Africa
There are three types of pathological stroke conditions: cerebral infarction, cerebral haemorrhage and subarachnoid haemorrhage. Cerebral infarction or ischaemic stroke is further divided into various subtypes including intracranial small vessel disease, large-vessel atherosclerotic disease and embolism from the heart (Warlow, 1998). These types and sub-types differ in terms of outcome and treatment (Warlow, Dennis, van Gijn, Hankey, Sandercock & Bamford, 2001). The proportion of various stroke types also changes within the population depending on their various stages along the health transition. As an example, early in the transition when the prevalence of hypertension is high but smoking, blood cholesterol and atherosclerotic disease are low, cerebral haemorrhage forms a greater proportion of all strokes (Howson, Reddy, Ryan & Bale, 1996). However, later as other risk factors become more common and cerebral atherosclerosis increases, there is a decrease in the rate of cerebral haemorrhage and an increase in the rate of cerebral infarction.

In order to accurately understand the true nature of stroke, community-based incidence studies are needed with early brain imaging and investigations of risk factors and cause. Needless to say, this data does not yet exist in South Africa. However hospital-based stroke registers can add to our knowledge of the nature of stroke within the population. Table 2.1 on the following page, presents the findings of stroke type and subtype from the published studies that used brain imaging in South Africa and Zimbabwe taken from Connor and Bryer (2006).
Table 2.1: Comparison of pathological stroke types and subtypes from hospital-based studies
2.1.5 Stroke risk factors in South Africa

Stroke risk factors are divided into those that are modifiable (like smoking) and those that are not, such as increasing age. Hospital-based studies suggest that age-specific stroke incidence is higher in younger (35-54-year old) age groups in South Africa than in high income regions (Matenga, 1997; Rosman, 1986). In terms of the prevalence of modifiable stroke risk factors, the following have been found.

- Hypertension in patients with cerebral infarction 32-76%
- Hypertension in patients with cerebral haemorrhage 76-93%
- Diabetes mellitus 3-10%
- Hypercholesterolaemia <2-10%
- Atrial fibrillation 1-7%
- Cigarette smoking 15-28%
- Previous stroke or transient ischaemic attack 2-7%

In the SASPI study of stroke prevalence in rural South Africa, the following was found: hypertension 71%, diabetes mellitus 12%, cigarette smoking 9%, and current alcohol use 20%.

It would be impossible to discuss stroke within a South African context without mentioning the impact of HIV/AIDS. Anecdotally many clinicians mention increasing numbers of young HIV-positive stroke patients in our hospitals. There are many reasons why someone with HIV may present with a stroke including tuberculous meningitis, toxoplasmosis affecting cerebral blood vessels or even cardiac disease (Connor, 2007; Connor, Lammie, Bell, Warlow, Simmonds & Brettle, 2000). The question about whether or not HIV actually causes or independently increases the risk of stroke is not easily answered. It certainly causes small vessel vasculopathy (Connor et al., 2000) and ‘an extra-cranial large artery ‘vasculitis’ of sorts’ (Connor & Bryer, 2006, p. 200). However, only one study has convincingly found HIV to be an independent risk factor for stroke (Cole, Pinto, Hebel, Buchholz, Earley et al., 2004).

The Durban Stroke Register found 20% of young black stroke patients to be HIV positive and have HIV-related stroke (Hoffmann, 2000). On the other hand, the older rural SASPI stroke prevalence study found only 2% of stroke patients were thought to be HIV positive (Connor et al., 2004). In both cases, these figures are likely to reflect the HIV prevalence in the general
population; what we need are case-controlled studies to answer the question (Connor & Bryer, 2006).

2.2. MECHANISMS OF STROKE

The brain is exquisitely reliant on minute to minute supply of oxygenated blood. Brain tissue deprived of blood supply undergoes ischaemic necrosis also termed infarction. The most common cause of this interruption of blood flow is obstruction of a cerebral artery by a thrombus or embolus although circulatory failure following heart attack can also underlie the condition. Stroke can be also caused by intracranial haemorrhage due to rupture of an arterial aneurysm or arteriovenous malformation. Following a recent infarction there is an area of brain destined to die and the surrounding area (penumbra) is at risk. Interventions to reduce cell death in the penumbra have been the focus of much pharmacological research (Fawcett, Rosser & Dunnett, 2001).

Ischaemia occurs when blood supply is cut off to the brain. This primary event combines several different toxic processes including excitotoxicity, metabolic toxicity and oxidative stress, which converge to produce central nervous system (CNS) degeneration following stroke. The release of excitotoxic neurotransmitters includes glutamate directly from neurons and the supporting glial cells (Klein & Albert, 2004). Binding to specialised receptors, NMDA (N-methyl-D-aspartate), AMPA (α-amino-3-hydroxy-5-methylisoxazole-4-proprionic acid) and kainite causes release of calcium leading to an influx of sodium into the brain, resulting in increased intracellular water, neuronal swelling and membrane disruption. Such injury to the CNS will damage cells so badly that they will be disrupted and die rapidly leading to an area of necrosis (see below) at the centre of the lesion that is not rescuable. However in the penumbra, cell death takes time to develop and may take two to three days to occur. Cell death here is not due to rapid primary necrosis but largely apoptotic processes (see below) and due to a cascade of secondary changes induced by the initial ischaemic event. Free radicals are also released at the time of the initial injury, leading to destruction of the cell membrane fatty acids. As neurons and glial cells die, they activate macrophage scavenger cells. The destructive cascade continues resulting in not only immediate cell death but also secondary degeneration over the next few weeks to months. Surviving cells could remain weak and vulnerable permanently (Klein & Albert, 2004). An understanding of these mechanisms can lead to therapeutic intervention to stop the destructive cascade and prevent this secondary cell death. Cells can die by two
processes. The first - necrosis - follows a complete overwhelming of the homeostatic processes in the cell and is associated with swelling and then disruption of the nuclear endoplasmic reticular and cell surface membranes. The cell splits open, spilling its contents into the extracellular space. The second cell death process is called apoptosis which is an active suicide in which the cell uses its own cellular mechanisms to initiate a series of molecular events that lead to the cell digesting away many of its components from the inside (Fawcett et al., 2001). These two processes are depicted diagrammatically in Figure 2.1 below.

Figure 2.1: The appearance of necrosis and apoptosis (Fawcett, Rosser & Dunnett, 2001)

2.3 MECHANISMS OF RECOVERY IN STROKE
Recovery can be divided roughly into two stages. During first stage recovery the acute effects of metabolic and membrane failure, ionic and transmitter imbalance, haemorrhage, cellular reaction and oedema need to be stabilised. The re-establishment of circulation in areas of partial ischemia or ischemic penumbra and reperfusion after thrombolysis are possible early mechanisms of recovery (Wise, 2003). Damage can be reversed if blood flow can be elevated beyond anoxic values and many of the neuroprotective agents try minimising damage by protecting cells in the penumbra until oxygenation can be restored. The first few days and
weeks following the event are critical when partially damaged neurons regenerate injured parts and others form new connections to compensate for ones that have been lost (Fawcett et al., 2001).

In second stage recovery, which can occur months or even years after injury, recovery mechanisms remain largely unknown. Physiological and functional recovery at this stage is probably attributable to compensation by intact structures for functional loss (Kertesz & Gold, 2003). There are essentially three hypotheses regarding what accounts for recovery following stroke and they are discussed briefly below.

2.3.1 Recovery of the penumbra
In the first view, the recovery of tissue around the edge of the lesion is considered critical to recovery (Warburton, Price, Swinburn & Wise, 1999). The residual tissue is able to support the impaired behaviour more or less because of redundancy within the local neural system. This viewpoint encompasses the theories of equipotentiality, diaschisis and redundancy and vicariation. Proponents of the theory of equipotentiality liken the plasticity of the cerebral cortex to embryogenetic capacity so that recovery represents the continuation of growth capacity of the organism to develop fully. The fact that there is recovery at all is interpreted as proof that rigid localization of functions does not exist. Recovery therefore has more to do with remaining intact cortical tissue than with the extent of the lesion (Warburton et al., 1999). Those favouring diaschisis maintain that when there is acute damage, the surrounding functionally connected tissues are deprived of enervation and become inactivated. As enervation is regained from uninjured areas, function returns to these undamaged structures (Warburton et al., 1999). Supporters of the theory of redundancy state that there is a biological protective mechanism which anticipates injury. Redundancy is provided by structures than can substitute for damaged functions. This idea that some structures can take over functions they were not previously associated with, is called vicarious functioning. Function is also re-represented at several levels so that damage to higher levels releases low levels of activation from inhibition and leads to compensation (Warburton et al., 1999).

2.3.2 Transfer of lost function to the homotopic cortex in the right cerebral hemisphere
According to this view of hemispheric substitution the right hemisphere is capable of taking over some speech functions like comprehension and automatic non-propositional speech. There
is however a wide range of individual variation in people’s ability to make use of commissural connections and some can activate cortical mechanisms in the right hemisphere more than others. A number of studies in fact suggest that good recovery of language functions in non-fluent aphasia is accompanied by greater perilesional than right hemisphere reorganisation (Cao, Vikingstad, George, Johnson, & Welch, 1999; Heiss, Karbe, Weber-Luxenburger, Herholz, Kessler, et al., 1997; Karbe, Thiel, Weber-Luxenburger, Herholz, Kessler, & Heiss, 1998; Rosen, Petersen, Linenweber, Snyder, White, et al., 2000). To this end, Crosson, Moore, Gopinath, White, Wierenga, et al., (2005) have proposed that persistent non fluent aphasia is representative of an intentional disorder and would benefit from treatments that specifically prime a right medial frontal intention mechanism to enhance word production.

2.3.3 Adoption of strategies that circumvent the lost function

The underlying tenet of this theory relies on regeneration and plasticity of the brain. Changes in cortical maps occur with training or experience related to changes in membrane excitability and growth of new connections (Weiller, 2000). Increases in dendritic spines can result from an enriched environment and training, lending support to the importance of early and appropriate intervention. According to functional compensation theories there is a behavioural rather than a neural model for recovery. Instead of rerouting connections, brain damaged organisms develop new solutions to problems using residual structures (Wise, 2003).

These theories of recovery are essentially what underlie attempts at rehabilitation (Wise, 2003). As will be discussed below, the majority of research related to drug treatments of aphasia and stroke has attempted to salvage the penumbra of potentially viable tissue around the edge of the recent infarct. This has been attempted through thrombolytic therapy to encourage reperfusion or by use of one of many, but so far unlicensed, neuroprotective agents. Early reperfusion has been shown to reverse aphasic deficit. Behavioural treatment may work by retraining neural systems ipsilateral or contralateral to the lesion or direct the patient to develop strategies to overcome deficit. But the types of behavioural treatments attempted in clinical settings are rarely informed by any direct evidence of the changes in brain function that they are supposed to induce. The potential value of functional activation studies in aphasia is therefore evident (Wise, 2003).
According to Weiller (2000) the brain must upregulate, best focally, its excitability to make use of sparse remaining connections. Once some function has been regained, repetitive use and learning will increase the effective connectivity of this feasible pathway and as a consequence fewer neurons will be needed for the same effect, resulting in a concomitant normalization of activation. This understanding of mechanisms of stroke and recovery, inform the section which follows at the end of the chapter, related to pharmacological treatment of stroke and related communication deficits.

2.4 FACTORS AFFECTING RECOVERY

There are several factors that play a part in the degree of recovery from stroke. Although reliable predictors of measuring prognoses are still limited, some guidelines exist (Murray & Chapey, 2001). These can be arranged in terms of a number of variables including language, biographical, medical as well as social variables. It is particularly noteworthy that the presence of executive dysfunction has not made its way into mainstream literature in speech pathology as a potential factor affecting recovery. Yet, it is becoming increasingly apparent that the presence of EF deficits may hold some important clues as to why some of our clients are not responding satisfactorily; even to well designed, theoretically supported and competently administered therapeutic intervention. Below is a summary of the existing factors affecting recovery.

2.4.1 Language variables

In general type of aphasia is often predictive of level of recovery. Individuals with severe (global) aphasia often remain severely impaired, particularly when they continue to present with global aphasia beyond three months post onset, despite some improvement in comprehension (Paolucci, Antonucci, Pratesi, Traballesi, Lubich, & Grasso, 1998). Broca’s, anomic, conduction, Wernicke’s and transcortical aphasias often have good prognosis with most showing excellent spontaneous recovery and resolving towards anomic aphasia over time (Goodglass, 1993) and half of the individuals with anomic aphasia making a complete recovery (Kertesz, 1979 in Murray & Chapey, 2001).

Severity is often tied to type of aphasia and considered to be highly predictive of outcome i.e. individuals most severely affected have poorer outcomes and mildly affected people tend to have more complete recoveries (Pedersen, Jorgensen, Nakayama, Raaschou, & Olsen, 1995).
Conversely, patients with lower scores initially have more room for improvement as opposed to people with higher scores who have reached ceiling (Weiller, 2000).

2.4.2 Cognitive variables

Rates of cognitive recovery in the stroke population are not readily available, although a 1996 study by Desmond, Moroney, Seno and Stern found long term improvement may be evident in association with left hemisphere infarct (approximately 54% of the time). On the other hand, severe hemispheral syndromes were particularly compromised when co-occurring with diabetes, and only an 11.9% recovery rate was recorded when these patients were followed up three months and then annually post stroke. Riepe, Riss, Bittner and Huber (2003) found that cognitive malfunctions occurred frequently in acute stroke (up to two thirds of people assessed) with only a 15% rate of recovery after a year. Clinical accounts and studies such as this one suggest that cognitive deficits are often persistent among those who have suffered strokes. With increased recognition that such deficits impede progress in therapy, the consideration of these cognitive and specifically executive functions must become part of the speech language pathologists’ areas of expertise.

2.4.3 Biographical variables

Age is a significant factor. Recovery in children before 10 – 12 is excellent, as maturation of the left hemisphere inhibits language abilities of the right. Lesions before this maturation can facilitate more complete transfer of function. Also functional plasticity of younger patients may depend on adaptability of Golgi type II cells which remain adaptive whereas cells with long axons responsible for major transmission of information in and out of the central nervous system are under early and exacting genetic specification and control. Flexibility of these neurons may be terminated in teenagers by hormonal changes (Fawcett et al, 2001).

Estimates of premorbid functioning which weigh standard demographic data including such variables as age, education, gender, occupational category as examples, into specifically developed equations may have more predictive power than educational level alone except in the cases where the patient had been illiterate (Lecours, Mehler, Parente, Beltrami, Detolipan, et al., 1988). Handedness and gender have little predictive value as to recovery outcome (Pedersen et al., 1995).
2.4.4 Medical variables

Traumatic etiology is associated with better prognosis than vascular etiology (Basso, Capitani, Laiacona, & Luzzatti, 1980) and survival after hemorrhagic stroke is likely to result in better recovery than following an ischemic event (Holland, 1984).

There is usually a great deal of recovery during the first two weeks, and the greatest amount in the first two to three months after onset. After six months, rate of improvement significantly drops and in the majority of cases spontaneous recovery stops after about a year. Still many patients report improvement for many years after the event with therapy (Weiller, 2000). Lesion size is an important factor in determining the extent of recovery but there are some exceptions where in the left hemisphere other factors have proved more important for prognosis (Murray & Chapey, 2001). Not only lesion size but location of the lesion is important in recovery. Significantly, site of lesion has usually been considered relative to aphasia type, although these areas are also critical for the support and performance of EF, a fact which has largely gone unrecognised within the speech pathology literature. As an example, it has been noted that left hemisphere structures connected to opercular and anterior insular regions play crucial roles in recovery from Broca’s. Patients with damage to the inferior portion of the precentral gyrus and anterior parietal region have less recovery than those where those areas are spared (Weiller, 2000). While this data suggests the role of these structures in compensation for language disturbance, the contribution of these structures to EF has been under-acknowledged in the past.

The presence of other physical and mental health difficulties in addition to aphasia often results in poorer prognosis (Jorgensen, Nakayama, Raaschou, & Olsen, 1995). Depression and anxiety can have a negative impact on rehabilitation and are associated with increased mortality rates (Linden, Blomstrand & Skoog, 2007). Similarly, stroke patients with fewer concomitant medical problems tend to have a shorter length of stay in hospital which is also associated with better recovery (Murray & Chapey, 2001). This is particularly significant within the South African context when considering the impact of TB, HIV/AIDS and other poverty-related diseases including malnutrition, on general health and wellbeing. Finally certain medications such as antidepressants and anticonvulsants can result in dysarthria or stuttering (Patsalos, 2000). Furthermore, antiepileptics can also produce confusion, fatigue and decreased levels of arousal which can affect rehabilitation activities (Patsalos, 2000).
2.4.5 Social variables

Research indicates that personality variables and social support are linked to health generally, to morbidity and mortality as well as to prognosis in treatment in a variety of health conditions including stroke (Tompkins, Jackson, & Schulz, 1990). A committed and healthy support network can have an influential impact on recovery. Stroke patients are found to do significantly better in therapy when the primary caregiver is not depressed, is married and is knowledgeable about stroke care (Tompkins et al, 1990). Once again, the existing South African context, particularly in rural areas, presents a challenging and not always optimistic picture of recovery with regard to these variables. Of particular concern is the impact of increasing burden of stroke on the country’s middle-aged and elderly population. The impact of HIV is felt when many families have lost their parents and the role of caregiver is taken on by grandparents, who themselves are more at risk for stroke and vascular disease (Connor & Bryer, 2006). Also, knowledge of stroke and related risk factors is poorly developed.

2.5 COMMUNICATION DISORDERS FOLLOWING STROKE

Communication difficulties resulting from stroke are common with as many as 25% of stroke patients having significant aphasia, when damage occurs in the cerebral cortex specialised for language, most commonly in the left hemisphere (Kertesz, 2007). In addition, there are a range of communicative disturbances associated with right hemisphere damage (RHD) which are often more subtle and difficult to diagnose. This is because despite increased interest and research in this area, we do not have a standard or well-accepted term for the communicative deficits associated with RHD (Lehman-Blake, 2005). The reviews below present the next contributors to conversational intelligence which rely to a great extent on neurolinguistic processing, supported by specific neuroanatomical sites and which are variably disrupted in different aphasia syndromes and RHD.

2.5.1 Aphasia

A number of aphasic syndromes have been described and their taxonomy has been widely debated. However the most commonly accepted classification and terminology are presented in Table 2.2 adapted from Kertesz (2007) on the following page. Articulated language is a complex cognitive function subserved by a neural network which offers a significant contribution towards conversational intelligence. Broca’s aphasia is characterised by speech that is effortful with hesitations, pauses, word-finding difficulty, phonemic errors and
agrammatism but relatively preserved comprehension (Goodglass & Kaplan, 1972; Kearns, 2005). Pure motor aphasia or verbal apraxia are terms used to denote hesitation, stuttering, dysprosody, initial consonant substitutions, deletions, repetitions, transpositions and anticipation that can occur as part of Broca’s aphasia or occasionally alone (Darley, Aronson & Brown, 1975). Transcortical motor aphasia is characterised by poor spontaneous speech but good repetition and comprehension (Kertesz, 2007). Global aphasia on the other hand is defined by the loss of speech output as well as comprehension usually associated with the destruction of both the anterior and posterior language areas (Kertesz, Harlock & Coates, 1979).

### Table 2.2 – Major aphasic stroke syndromes

<table>
<thead>
<tr>
<th></th>
<th>Output</th>
<th>Comprehension</th>
<th>Repetition</th>
<th>Naming</th>
<th>Other feature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Broca’s aphasia</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>Agrammatic</td>
</tr>
<tr>
<td>Pure motor aphasia</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>±</td>
<td>Better writing</td>
</tr>
<tr>
<td>Transcortical motor aphasia</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>±</td>
<td>Not paraphasic</td>
</tr>
<tr>
<td>Global aphasia</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td>Mute or stereotypy</td>
</tr>
<tr>
<td>Wernicke’s aphasia</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Paraphasic</td>
</tr>
<tr>
<td>Pure word deafness</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Better reading</td>
</tr>
<tr>
<td>Transcortical sensory aphasia</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>±</td>
<td>Semantic jargon</td>
</tr>
<tr>
<td>Anomic</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Only naming deficit</td>
</tr>
<tr>
<td>Conduction aphasia</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>Mainly repetition deficit</td>
</tr>
<tr>
<td>Mixed transcortical aphasia</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>Only repetition spared</td>
</tr>
</tbody>
</table>

Key: (+) relatively preserved; (-) impaired; (±) variable presentation

A different group of aphasia subtypes is classified according to sensory characteristics. Wernicke’s aphasia is characterised by fluent, paraphasic speech with impaired comprehension, repetition and naming. While syntax and morphology are relatively spared, substantive words are often substituted by both semantic and phonological paraphasias (Kertesz, 2007). “Pure word deafness” is a term used to describe a complaint of not understanding speech; but hearing, reading, and speech output, remain undisturbed (Kohn & Friedman, 1986). Neologistic jargon occurs in severe Wernicke’s aphasia when most substantive words are substituted with unintelligible phonological paraphasias (Kertesz & Benson, 1970). Conduction aphasia is distinguished by poor repetition with relatively fluent but phonologically paraphasic speech and
good comprehension (Kertesz & Phipps, 1977). Transcortical sensory aphasia presents with fluent, semantic jargon, poor comprehension and good repetition (Kertesz, 2007). In the mixed transcortical aphasia, features of both motor and sensory transcortical aphasia are present (Kertesz, 2007). Finally, anomic aphasia represents the mildest form of aphasia characterised by fluent output, good comprehension and only naming and word finding difficulty (Benson, 1979). However the cognitive and neural bases of naming breakdown vary across individuals with aphasia and characteristic patterns of impairment can be observed depending on whether semantic or phonological stages of naming are disrupted (Raymer, 2005).

Each syndrome is usually associated with its own particular site of lesion, summarised in Table 2.3 below (Arseni & Botez, 1961; Goldstein, 1948; Kertesz, 1983; Kertesz, 2007; Kertesz et al, 1979; Kertesz, Sheppard & MacKenzie, 1982; Levine & Sweet, 1983; Peterson, Fox, Snyder & Raichle, 1990) and depicted diagrammatically in Figure 2.2 on the following page (LaPointe, 2005).

Table 2.3 – Aphasia syndromes and related sites of lesion

<table>
<thead>
<tr>
<th>APHASIA SYNDROME</th>
<th>SITE OF LESION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Broca’s aphasia</td>
<td>“Foot” of the inferior frontal convolution of F3; rolandic operculum, anterior insula, subcortical area and periventricular lesions</td>
</tr>
<tr>
<td>Pure motor aphasia</td>
<td>Broca’s area anterior subcortical, inferior rolandic and insular cortical lesions</td>
</tr>
<tr>
<td>Transcortical motor aphasia</td>
<td>Superior mesial frontal region or supplementary speech area in the dominant hemisphere</td>
</tr>
<tr>
<td>Global aphasia</td>
<td>Anterior and posterior language areas</td>
</tr>
<tr>
<td>Wernicke’s aphasia</td>
<td>At or around the Sylvian fissure in the dominant first temporal gyrus</td>
</tr>
<tr>
<td>Pure word deafness</td>
<td>Right sided, bilateral temporal or left temporal lesions</td>
</tr>
<tr>
<td>Transcortical sensory aphasia</td>
<td>Posterior lesion in the watershed area between the middle cerebral and posterior cerebral circulation</td>
</tr>
<tr>
<td>Anomic</td>
<td>Anterior and central lesions, posterior temperoparietal lesions, but wide distribution can account for anomia</td>
</tr>
<tr>
<td>Conduction aphasia</td>
<td>Posterior temperoparietal region at the end of the Sylvian fissure, posterior insula, or arcuate fasciculus</td>
</tr>
<tr>
<td>Mixed transcortical aphasia</td>
<td>Lesions surround the middle cerebral artery territory often in watershed areas</td>
</tr>
</tbody>
</table>

Finally, subcortical strokes can also result in a range of language disturbances, although the role of the subcortical structures in language is widely debated. Motor impairments often dominate deficits in lesions of the basal ganglia ranging from dysarthria to hypophonia to severe global...
aphasia (Kertesz, 2007). Anomic aphasia is also often observed (Alexander & Loverme, 1980). Lesions in the putamen and anterior internal capsule produce slow, anomic, dysarthric speech and with posterior extension, comprehension can also be impaired with paraphasic speech and jargon (Damasio, Damais, Rizzo, Varney & Gersh, 1982).

Figure 2.2: Major lobes of the brain with location of important language areas.

In addition to the aphasia syndromes that result from stroke, there are a number of acquired dyslexias (reading disorders) as well as agraphias (writing disorders) that are associated with aphasia. Three major psycholinguistic classifications of reading disorders have been identified: deep dyslexia, surface dyslexia and phonologic alexia (Webb, 2005). In deep dyslexia, there is a disruption of semantic representations and impaired grapheme-to-phoneme conversion. Therefore, errors are semantically related to the target, the patient can not read pseudowords, there is little effect of length or spelling regularity but a pronounced effect of frequency (Webb, 2005). In surface dyslexia, the impairment rests in an inability to access the grapheme input lexicon or the representations within. Errors are phonologically related to the target and there is a pronounced effect of spelling regularity (Webb, 2005). Phonologic alexia represents an impairment of grapheme-to-phone conversion and patients exhibit an inability to read pseudowords and difficulty with low frequency words (Webb, 2005).

In a similar vein, McNeil and Tseng (2005) describe agraphia subtypes based on neuropsychological/psycholinguistic models. In lexical or surface agraphia, difficulty arises
from spelling words with irregular sound-to-letter conversion rules and writing to dictation often results in errors that approximate the phonologic shape of the target word (Hatfield & Patterson, 1985). In phonological agraphia a patient can write a word that has regular or irregular spelling but has difficulty in writing nonwords. Deep agraphia is a variant of phonologic agraphia in which the writing profiles are similar with an increase in semantic paraphasias in deep agraphia e.g. a patient may write ‘happy’ for ‘glad’ (McNeil & Tseng, 2005). A grapheme buffer impairment is a relatively rare disorder resulting in errors (letter additions, deletions, substitutions or transpositions) across a variety of tasks. Errors tend to cluster in the middle of words and are likely to increase with the length of the word (Jonsdottir, Shallice & Wise, 1996). Finally, peripheral agraphias are writing disorders that are not attributed to psycholinguistic deficits and include allographic impairment, apraxic agraphia and spatial agraphia (Rapcsak & Beeson, 2000; Roeltgen, 1993). There are a range of treatment strategies available to treat disorders of reading and writing at varying levels of impairment, but these are not discussed due to space constraints.

A number of aphasic syndromes result from stroke, usually depending on site of lesion. These syndromes can be accompanied by disorders of reading or writing in variable presentations, also depending on site of lesion and the psycholinguistic processes affected. Each syndrome is characterised by distinct verbal communication profiles related to speaking, comprehending, repeating and naming, all of which manifest during conversational interactions. Thus, language integrity forms a basic and important component of conversational intelligence.

2.5.2 Right hemisphere deficit
It has only been during the last 25 years that the importance of the nondominant hemisphere for efficient communication processes has been fully recognised (Lehman-Blake, 2005). These discoveries were made, according to Gardner (1994) when aphasiologists were using individuals with right hemisphere damage (RHD) as controls in aphasia studies, but soon realised that these individuals did not perform as well as non-brain-damaged adults on language tasks. In terms of communication, adults with RHD usually appear to have little difficulty with basic comprehension and expression. They may exhibit mild word finding difficulties, verbal fluency or auditory comprehension deficits but these are often not attributable to linguistic deficits per se, but rather cognitive impairments such as reduced attention (Myers, 1999).
A commonly described deficit is misinterpretation of nonliteral language with impaired ability to fully appreciate the abstract meaning of words and phrases (Myers & Linebaugh, 1981). Despite initial findings that patients with RHD had a tendency towards literal interpretations of metaphor expression, research has since shown that these individuals are usually not completely concrete (Lehman-Blake, 2005). Generation and interpretation of inferences may also be problematic for individuals with RHD. This may cause literal, disjointed interpretations of discourse and may also account for factual but disorganised verbal output which can often resemble a list of details rather than a coherent story (Beeman, 1993). Comprehension difficulties may result when multiple interpretations are possible. According to Tompkins, adults with RHD are able to generate multiple interpretations but have difficulty when they have to decide which of the possible interpretations is most appropriate to the context (Tompkins, Lehman-Blake, Baumgaertner & Fassbinder, 2001). Difficulties may also arise in attributing emotions to a story character that are different from the comprehender’s emotions (Lehman-Blake, 2005).

Discourse is frequently disrupted in RHD as patients may have difficulty identifying the main ideas or themes in a discourse and/or reduced coherence in discourse production. Therefore discourse may be characterised as off-target, disjointed and disorganised (Joannette, Goulet, Ska & Nespoulous, 1986). Humour may be affected with the patient with RHD being drawn to physical or slapstick humour with loss of sensitivity to various situations or communicative partners, making them prone to telling inappropriate jokes (Lehman-Blake, 2005).

In terms of conversation, studies have found individuals with RHD to take fewer turns and talk more about themselves than healthy older adults. They may also prolong conversations instead of terminating them upon cues from their interlocutors (Kennedy, 2000). They may have a tendency towards tangentiality and lack the capacity to repair conversational breakdown (Lehman-Blake, 2005). They may also have difficulty with both the comprehension and production of prosody making it difficult to determine the speaker’s emotional state and the interpretation of sarcasm can often be disrupted as a result. Findings from affective studies in this population indicate that both deficits of hypo- and hyperaffectivity can occur (Blake, Duffy, Myers & Tompkins, 2002).
Finally a range of cognitive deficits can commonly arise with RHD including attention, neglect, anosognosia, visuoperception, organizing, sequencing, reasoning and problem solving (Blake et al., 2002). These cognitive deficits impact on communication in various ways and interact with other deficits. Thus, although the right hemisphere is not localised for language, this brief review has demonstrated its significant contribution to conversational intelligence in terms of higher order, abstract, conceptual and emotive aspects of communication.

2.6 TREATMENT OF COMMUNICATION DISORDERS FOLLOWING STROKE

The traditional model in Western medicine has been termed a medical model, which considers problems as personal and residing within each patient. Treatment is provided to the patient by an “expert” with the ultimate goal of curing the disorder (Elman, 2005). In contrast, a social model of medicine has evolved, which sees problems as an interaction among personal, physical, environmental and societal factors. Treatment within this paradigm is collaborative and the ultimate outcome is to promote positive changes, even when a cure is not possible (Elman, 2005). Some historical approaches to aphasia align themselves clearly within the medical model. Assessment may be aimed at identifying relative strengths and deficits of the communication profile and intervention planned to specifically address the underlying psycholinguistic deficit. Social approaches on the other hand, focus on real life, functional approaches to the assessment and treatment of aphasia. Both approaches are extremely valuable. In 2002, LaPointe suggested that in the future we should be able to articulate a “sociology of aphasia” that could integrate the psychosocial ramifications of aphasia with neurolinguistic knowledge. Below is a brief review of some of the historical and more modern socially oriented approaches to the treatment of aphasia and RHD communication deficits.

2.6.1 Historical approaches to the treatment of aphasia and disorders related to RHD

According to Kearns (2005), language therapy for aphasia is an inexact science informed by the clinician’s training, their philosophy about the nature of aphasia and patient-specific considerations such as severity of language disturbance, time since onset, presence of associated impairments and patients’ communicative environments. Some generic treatment strategies exist, which refer to intervention approaches that do not target improvement in a single modality. As an example, intact functions can be used to “deblock” an impaired ability where patients can be taught a specific self-cuing strategy as a means of circumventing communicative difficulties (Luria, 1970). Prompting Aphasics’ Communicative Effectiveness (PACE) (Davis
Wilcox, 1985) is another general treatment approach which does not require high-level verbal skills and can be easily adapted to varying severity levels. While these approaches may be useful in some individuals and certainly encourage independent transfer of functions to more natural environments, they do not take into account some of the executive disorders that may impede the use of self-cuing or effective monitoring. They may in fact fail altogether to treat the underlying attention or working memory deficit in affected clients, which may further account for why some clients experience limited success despite considerable strengths in their language profiles and positive response to strategies within the therapy room context.

2.6.1.1 Broca’s aphasia

There is less availability of structured auditory comprehension treatment programmes than interventions designed for expressive language. However, the auditory stimulation treatment procedures developed by Schuell, Jenkins and Jimenz-Pabon (1964), elaborated below, have been used for decades with success. Mapping therapy addresses agrammatism by targeting the relationship between sentence structure and thematic roles (Marshall, 1995). The Cueing Verb Treatments (CVT) programme involves a sentence-production training programme based on the idea that the verb is the central constituent in sentence structure (Loverso, Prescott & Selinger, 1992). Thompson (2001) designed a novel, linguistically based therapy approach using Chomsky’s theory of government and binding as a conceptual framework. The Helm Elicited Language Program for Syntax Stimulation (HELPSS) uses a story completion framework to elicit multiple exemplars of 11 different syntactic structures, which are then trained at two different levels of difficulty until successful production is reached (Helm-Estabrooks, Fitzpatrick & Barresi, 1981; Helm-Estabrooks & Ramsberger, 1986). Melodic Intonation Therapy (MIT) uses intoned melodies as a means of improving verbal production, which operates on the notion that the intact right cerebral hemisphere, specialised for melodic functions, can be recruited to facilitate verbal responding (Helm-Estabrooks, Nicholas & Morgan, 1989). MIT appears to be most effective for highly motivated patients with unilateral left hemisphere lesions involving Broca’s area with poorly articulated, nonfluent or severely restricted verbal output in the presence of moderately preserved auditory comprehension and poor repetition (Kearns, 2005). Finally, Response Elaboration Training (RET) is a programme designed to increase the length and information content of verbal responses of nonfluent patients with aphasia (Kearns, 1985). This programme was designed as a loose training programme to counter the limiting effects of overly structured treatments that may inhibit the
use of language in more creative and flexible ways. Response parameters are loosened by using patient-initiated responses as the primary content of therapy. This approach has been successful as shown in a series of single-subject experimental studies (Wambaugh, Martinez & Alegre, 2001). However, most significantly, the success of these interventions rests to a large degree on the patient being able to self-activate the treatment strategy in naturally occurring contexts, something that can fail in patients with EF deficits, who are not always capable of using context to facilitate self-initiated adaptive responses.

2.6.1.2 Wernicke’s aphasia

Many treatments advocated for this group of individuals come from the social approaches to aphasia therapy intervention and rely to some degree on the participation of friends and significant others in the pursuit of formulating and achieving specific goals and facilitating conversation. Two other techniques include Schuell’s stimulation approach, which is one of the most effective in the treatment of comprehension deficits (Robey, 1998). This intervention employs strong, controlled, and intensive auditory stimulation in the hope that immersion in language through intensive sensory stimulation can increase neuronal firing, causing an increase in neural activation (Caspari, 2005). In addition, PACE (Davis & Wilcox, 1985) is widely used among individuals with Wernicke’s aphasia.

2.6.1.3 Conduction aphasia

Unlike many treatment approaches for other types of aphasia, verbal imitation is not used as a deblocking process as a result of the fact that repetition is the primary deficit, it becomes the target rather than the approach to treatment. When verbal imitation forms a treatment approach, it can reduce excessive self-correction behaviours during conversation by introducing sentences of increasing levels of difficulty to tax the patient’s verbal output and focus attention away from production accuracy (Kohn, Smith, Arsenault, 1990; Simmons-Mackie, 2005). However, repetition deficits can be resistant to therapy and this is not surprising when considering some underlying EF deficits (such as attention and working memory factors) as potential causes or contributors. Word retrieval strategies focusing on either semantic or phonological levels where appropriate are often utilised. Divergent word retrieval tasks have been used using visual imagery and RET has also been used as a format to facilitate divergent tasks to approximate requirements of natural communication (Simmons-Mackie, 2005). In some individuals with fluent aphasia, the goal of therapy is to impose a controlled approach to speech using “stop
strategies” to allow patients to structure and control sentences and then use more extended verbalisation. Other treatments cover deficits of reading and writing and may use facilitatory channels such as verbal, gestural and visual cues as well as rhythm and song (Simmons-Mackie, 2005). The “stop strategies” proposed for this group, are heavily reliant on intact self-monitoring systems, which may be less efficient in some individuals with EF deficits.

2.6.1.4 Transcortical aphasias

Equivocal research findings, limited information about treatment efficacy and conclusions drawn from single case studies, make the prognosis for recovery from transcortical motor aphasia unclear (Cimino-Knight, Hollingsworth & Rothi, 2005). Still restitutive strategies have been proposed to restore impaired function in this population. Johnson (1983) proposed remediation of motor function in general and verbal motor skill in particular, self-cuing strategies for naming have been emphasised (Kools, 1983). Expansion of restricted sentence structure and generativity of semantic category membership have also been proposed (Huntley & Rothi, 1988). However, according to Cimino-Knight et al. (2005), the pervasive communication deficit of verbal aspontaneity often remains relatively uninfluenced. Perhaps considering this aphasia subtype from the perspective of a dysexecutive syndrome, may therefore be useful in opening the door to previously untried intervention strategies. Some awareness of executive aspects being incorporated in therapy can be seen in a treatment method proposed by Raymer, Rowland, Haley and Crosson (2002) who paired nonsymbolic limb movements with the production of sentences in order to engage right frontal mechanisms to enhance verbal initiation.

There is virtually no literature regarding the treatment of transcortical sensory aphasia (Cimino-Knight et al., 2005). One hypothesis for this is the fact that anosognosia is a frequent accompanying symptom, which makes effective participation in a treatment programme unlikely (Cimino-Knight, et al, 2005). Compensatory strategies are advised for patients with more severe forms of aphasia that remain chronic. Robinson and Grossman (1997) have suggested that treatment should capitalise on preserved visual and perceptual processing components to facilitate naming ability.

Mixed transcortical aphasias are complicated to assess and treat. They can arise from pathological disease processes other than stroke, and in such cases like presenile dementia, the
etiology responsible may contribute to lack of syndrome evolution to milder forms of impairment (Cimino-Knight et al, 2005). Treatment programmes relying on repetition skill have been proposed, although their functional utility is undermined because they address a function that is preserved and performed almost automatically. Repetition may therefore serve as a starting point, but should move slowly to less automatic and more intentional verbalisations (Alexander & Schmitt, 1980).

2.6.1.5 Anomic aphasia

There are a number of treatments for anomic aphasia and word finding deficits in general. Some are restitutive in nature, devised to target specific levels of naming impairment and restore function to that mechanism (Rothi, 1995). Others are vicariative and are meant to engage cognitive mechanisms to support the process of word retrieval in a way that differs from the normal process (Rothi, 1995). Some restitutive approaches include cuing hierarchies, semantic and phonological treatments. Examples of vicariative approaches include reading, writing and gesture. One of the problems with some of the treatment approaches described above is that while improvement is often seen in structured lexical tasks, effects are not generalized in natural conversation settings (Raymer & Rothi, 2001). Social approaches that focus on conversational interaction and may include role-playing (Linebaugh, 1997), inclusion of caregivers or significant others (Boles, 1998) and participation in group therapy may assist with more effective carryover of treatment gains. The consideration of EF processes such as attention and working memory may further elucidate patient performance relative to these treatment strategies.

2.6.1.6 Global aphasia

Collins (2005) cautions that the need and desire to communicate as well as dependence on communication can vary among patients with severe aphasia and these differences should be recognised and respected by clinicians. Collins further lists goals that he construes as reasonable and attainable for individuals with severe aphasia. They include improving auditory comprehension supplemented with contextual cues, improving the production of unequivocal responses such as ‘yes’ and ‘no’ in controlled situations, improving the ability to produce several written responses or approximations of functional words of daily living spontaneously, improving production of several simple unambiguous gestures, improving drawing so that simple clear messages can be conveyed, ensuring that a small basic core of communicative
intentions can be transmitted and eliciting production of a few spoken words. Visual Action Therapy (VAT) is a programme where the patient is trained to associate ideographic forms with particular objects and actions and to carry out a series of tasks in association with these drawings (Helm-Estabrooks, Fitzpatrick & Barresi, 1982). Intersystemic reorganization involves the use of one communicative modality to facilitate the use of another (Rosenbek, Lapointe & Wertz, 1989), although this approach has met with variable results in individuals with global aphasia (Collins, 2005). Communication boards, group treatments (Elman & Bernstein-Ellis, 1999), intensive residential interventions, computer-assisted programmes (Steele, Weinrich, Kleczewska, Wertz & Carlson, 1987), PACE (Davis & Wilcox, 1985) and the training of communicative volunteer partners (Lyon cited in Collins 2005) have also been suggested for this group of individuals.

2.6.1.7 Right Hemisphere Disorders

As with the treatment of some of the transcortical aphasias, there are very few studies of treatment efficacy and outcomes for RHD with the exception of some treatments for attention and neglect (Lehman-Blake, 2005). With respect to communication disorders related to RHD, Myers (1999) has classified treatments as either task or process oriented. Task-oriented treatments focus on improving specific tasks or actions and are not expected to generalise to other activities. These approaches are often used to target a specific skill needed for daily functioning or increased independence (Lehman-Blake, 2005). Process-oriented treatments on the other hand address deficits that underlie various behavioural disorders, such as attention. Just as with aphasia, some approaches are facilitatory in that they aim to treat the level of impairment so that one brain area can take over for the damaged area, while others are compensatory and aim to teach the patient to accomplish a task in different ways to their pre-stroke approach (Robertson & Murre, 1999).

The challenge of treating RHD-related communication deficits highlights some of the difficulties with rehabilitation in cognitively compromised individuals. Specific strategies or rules may not be generalised, as patients may not to be able to analyse the component behaviours and realize application in other circumstances. Rules that are too general may also be difficult to teach, as patients may not be able to determine when and where they could be used and struggle with strategy selection. Some strategies specifically rely on self monitoring, problem solving or meta-linguistic capacity, which may be exactly those areas that are deficient...
in the brain injured patient. Patients may also be unable to reliably use strategies in attentionally demanding situations, a set of cognitive processes particularly vulnerable to brain injury.

There are a range of detailed and specific pragmatic treatments described by Myers (1999) and Tompkins (1995) for RHD-related difficulties. Hierarchical Discourse therapy (Penn, Jones & Joffe, 1997) may also be effective in allowing the development of various levels of text management to facilitate more complete comprehension of reading material.

2.6.2 Modern approaches to the treatment of communication difficulties

There are two main topics related to modern approaches of treatment for communication difficulties. The first has to do with social approaches to intervention, the other to do with pharmacology. Social approaches to treatment evolved fairly recently in response to the call for functional assessment and a consideration of the psychosocial impact of aphasia and other stroke related disorders (Holland, 1980; Sarno, 1981). The realm of pharmacological treatment remains in its infancy with more questions unanswered than not but which continues to evolve as our understanding of brain mechanisms involved in stroke and recovery advance. Although much of the literature presented below, was derived from aphasia-related research, it is no less applicable to individuals with RHD. Perhaps one of the reasons that social approaches have become so popular is not only for the obvious reason that they address the individual with a communication difficulty more holistically but that in effect because treatment strategies are by nature more collaborative, they remove the burden of interaction from the person with aphasia or RHD and instead offer support in the face of disrupted EF.

One social approach is the life participation approach to aphasia (LPPA). This is a philosophy emphasising the reengagement in life for all those affected by aphasia (LPPA Project Group, 2001). Within such an approach emphasis is placed on wellness and quality of life, which is not determined by absence of disease but rather as the presence of important factors such as leading a purposeful life (Ryff & Singer, 2000). Research from various fields has indicated that social connection is highly correlated with longevity, which places the emphasis of the communication specialist’s work on assisting our clients to maintain interpersonal connections (Elman, 2002).
Some specific treatment approaches aligned with LPPA are the following. Group treatment has demonstrated both efficacy and effectiveness (Elman & Bernstein-Ellis, 1999) in that it provides increased variety of communicative functions or speech acts to use among group members, a wider array of conversational partners, the potential for improved psychosocial functioning and finally an excellent environment for language improvisation (Elman, 2004). The training of communicative partners and volunteers provides added interpersonal support as well as facilitates increased participation in the community (Lyon, 2000). Couples and family training involves videotaping interactions between family members and individuals with aphasia. Specific therapist feedback of successful or unsuccessful interactions is provided and the family then collaborates with the therapist in order to optimise more effective strategies. Some specific techniques may include supported conversation (Kagan, 1998) which employs scaffolding techniques in which the communicative partner provides cues or facilitators within the natural flow of conversation. Self advocacy training groups are self-help groups initially started with support from a speech-language pathologist, who gradually retreats while group members hold their own meetings. Each group sets its own agenda and political advocacy projects are often undertaken (Coles & Eales, 1999). Raising awareness and understanding of aphasia and other neurogenic communication disorders in the work environment can also serve to reduce environmental barriers that patients may encounter when returning to work and ease transition back to the work place (Lyon & Shaddon, 2001). Other intervention strategies that are consistent with LPPA are the use of augmentative and alternative communication (Hux, Manasse, Weiss & Beukelman, 2001), internet training (Elman, 2001) and advocacy on behalf of individuals with stroke related communication difficulties (Elman, Ogar & Elman, 2000).

Within the realm of LPPA, conversation has a uniquely central role. Conversation cements relationships and allows us to connect to one another, it is also the way we interface with our communities. Below, is a thorough review of the role of conversation in our lives and the reasons for its centrality in the assessment and treatment of individuals with neurogenic communication disorders as evidenced by the recent surge in the use of Conversation Analysis (CA) as both a research and clinical tool.
2.6.3 CONVERSATION AND CONVERSATIONAL ANALYSIS (CA)

‘…talk in interaction is the place where the results of brain damage become visible and consequential for people’s lives’ (Goodwin, 1996) (p. 231).

The increasing frequency with which CA is being used in research related to communication disorders is attributable to a number of factors. A significant body of research already exists using CA as a methodology for the assessment and treatment of a range of communication disorders. These are discussed below embedded in a rationale for the use of CA in the current investigation.

2.6.3.1 Conversation as prototype of language use

The first reason that CA has increased in popularity as a research and clinical tool is that conversation has been viewed as the prototype of language use (Levinson, 1983) and is therefore pivotal to a field committed to the science of communication. Conversation Analysis (CA) itself derives from the ethnomethodology framework and was pioneered by sociologists (Levinson, 1983). This methodology originates from a context of research dedicated to true description of naturally occurring events. One of the primary contributions of this approach is the conviction that events are both context shaping and context renewing (Roger & Bull, 1989). This means essentially, that participants work together to negotiate meaning (Lesser & Perkins, 1999). Another principle of CA is that the analysis is participant driven. Phenomena are described as they exist using the ‘next turn proof procedure’ (Hutchby & Wooffitt, 1998). A major resource for the analyst in finding out about a speaker’s contribution is to look at how it was responded to by the recipient in the next turn in the conversation. A further assumption CA makes is that conversation is orderly. It is therefore possible to observe phenomena such as conversational repair, overlaps, pauses and turn taking because they are systematic and have rules, which when broken can be traced and analysed.

2.6.3.2 Research utilising CA as a methodology in relation to communication disorders

The fact that CA facilitates accurate description of conversational phenomena is primarily what lends validity to CA as a research and clinical tool. Social scientists and clinicians have believed that this approach has the ability to allow for the transition from description to application to be authentic, as analysis occurs at the level at which intervention is ultimately
targeted (Perkins, Whitworth & Lesser, 1998). It makes sense then that CA has been used and applied to everyday practice and research in fields as different as medicine and business to education and robotics. Within Speech Language Pathology, CA has been applied in a number of clinical settings, particularly, with adult neurogenic disorders such as dementia and aphasia, although more recently with children too. Radford and Tarplee (2000) studied topic management in a child with pragmatic difficulties and Tarling, Perkins and Stojanovik (2006) looked at conversational success in a child with William’s syndrome while Tetnowski and Damico (2001) applied CA to individuals who stutter.

Perkins et al., (1998) devised the ‘Conversation analysis profile for people with cognitive impairment’ (CAPPCI). This assessment tool evaluates the interaction between individuals with dementia and their conversational partners, the caregivers’ perceptions of current conversational abilities of the person with dementia, the strategies being employed in interaction and their success, changes from premorbid styles and opportunities for interaction and finally the relationship between the caregiver’s perceptions and what actually occurs in samples of conversation. The importance of interactional monitoring to facilitate order during conversations was investigated with two individuals with dementia of the Alzheimer’s type by Muller and Guendouzi, (2005). Orange, Lubinski and Higginbotham (1996) used CA to investigate the conversational repair in individuals with Alzheimer’s disease as did Watson, Chenery and Carter (1999). Friedland and Miller (1999) looked at conversational ability in individuals with dementia with a focus on bilingual aspects of language functioning over a period of a year. Friedland and Miller (1996) also used CA to describe conversational breakdown in patients with closed head injury.

In the area of aphasia Damico, Simmons-Mackie and Wilson (2006) used CA to investigate turn sequences in a therapeutic encounter between an individual with aphasia and dysarthria and a clinician. Hird and Kirsner (2003) researched the effect of right cerebral damage on the ability to share responsibility for the development of intentional structure of a conversation.

Lock, Wilkinson and Bryan (2001), embarked on the ‘Coping with Communication’ project for people with aphasia and their partners. This project drew on CA as a basis for assessment and treatment ideas. Treatment studies that have made use of CA have focused on providing advice to key conversational partners of people with aphasia (Booth & Perkins, 1999; Booth &
Swabey, 1999; Lesser & Algar, 1995, Wilkinson, Bryan, Lock, Bayley, Maxim, Bruce, Edmundson, & Moir, 1998). These studies provide evidence that supplying individualized advice based on analyses of conversation can effect change in conversational style and thereby improve the quality of interactions for individuals with aphasia and their significant others.

Perkins (1995) used CA to investigate distributions of turns at talk in aphasic participants’ conversations with a relative and herself. Her work highlighted the usefulness of CA in uncovering the relationship between linguistic impairment and conversational ability and subsequent guidance it provides in the selection of specific management strategies. Milroy and Perkins, (1992) used CA to investigate specific repair strategies in aphasic individuals. Crockford and Lesser, (1994) found by comparing a standard rating schedule completed by relatives, an analysis of speech elicited through role play and CA conducted on a sample of everyday conversation, that although more time-consuming, CA was a more sensitive measure of stability or change of communicative effectiveness over time than the other two measures. They also found that the CA provided the added advantage of highlighting avenues for intervention.

### 2.6.3.3 The study of conversation provides an assessment of language in context

As a result of the fact that language performance in individuals with communication difficulties is often assessed on contrived tasks that do not represent natural contexts, there is little known about the grammar of communicatively disabled individuals in interactional speech (Beeke, Wilkinson & Maxim, 2003a). Task based, elicited language such as picture naming, picture description or monologues are very different from conversation and the relationship between the two is complex (Wilkinson, Beeke & Maxim, 2003b). These authors further attribute the lack of improvement in spontaneous speech after therapy to the fact that therapy approaches often come from this decontextualised approach.

CA offers a different alternative. Sequentiality refers to the way in which an utterance is constructed so as to be oriented to the sequential context inherent in the turn-by-turn progression of conversation. There is an assumption that adjacent turns are related unless a special technique is employed to indicate that this is not the case. Hearers work to understand an utterance within the previous context and speakers work to be understood by paying heed to the preceding context when constructing a turn at talk. Recipient design refers to the way in
which speakers take into account what their co-participants know about one another and their circumstances (Beeke et al., 2003a).

Also, it is the contention of those who study disabled communication with this methodology that there are interactional motivations that account for some of the grammatical features noted and which are not, as traditionally believed, reflective of an underlying grammatical impairment. Eves (1999) further elaborates that the demands related to taking a turn can account for the nature of interactional grammar. The use of specific grammatical phenomena is used for example to buy time in which to complete grammatical processing for sentence or phrase formulation. Therefore demands of talk can influence the characteristics of aphasic grammar. Certain grammatical behaviours that have previously been interpreted as direct symptoms of an underlying linguistic deficit can actually arise from the interactional context in which they are produced and may represent an attempt to manage sequential demands of talk within the confines of aphasic limitations. More importantly, these phenomena are not necessarily visible in elicited language.

Goodwin (2003) in his work with a patient called Chil raises several interesting points which are also relevant to the following discussion about nonverbal behaviours. Chil presented with significantly limited verbal communication, however, he was able through adaptation of his gestural utterances to make himself understood. This was achieved by obtaining the orientation of a hearer to an emerging strip of talk – securing the gaze of the addressee with a preliminary version of the gesture and then redoing the gesture once mutual orientation had been established.

Goodwin’s argument is that though Chil’s abilities as an individual declined catastrophically, the social system within which he was embedded adapted to this crisis and evolved by creatively reshaping frameworks for the organisation of interaction so that he was able to continue to function as a viable and indeed central actor in the courses of collaborative action that made up their lives. The relevant unit for the analysis of Chil’s condition is not him as an individual but rather the framework for the production of meaning and action within interaction that links him to consequential partners who share his life with him. Thus context of this particular social context becomes integral to Chil’s ability to function as an enabled communicator.
In summary CA differs from other approaches to talk which see the mind/brain as the scene of all the action where the medium or the space of interaction seems almost to be beside the point. In CA the domain of interaction is seen as highly relevant and therefore may influence the form of the talk produced. In this way language adaptations to specific environments become discernible (Wilkinson et al., 2003).

2.6.3.4 CA provides access to nonverbal as well as verbal means of communication

Video-recordings used in CA capture the context of conversations and allows for the analysis of both verbal and nonverbal features which are often so critical in creating meaning (Cunningham & Ward, 2003). The fact that conversational information is often conveyed through mimicry, gesture, eye movement or posture, indicates that ‘conversation is certainly the linguistic context in which the relation between signs and users finds its fullest extension’ (Patry & Nespoulous, 1990, p.20). Therefore more studies that incorporate information about the use of conversational gesture, the deeper our insight into nonverbal aspects of conversation will be.

In this regard, Rose and Douglas (2003) looked at the relationship between limb apraxia and conversational gesture finding no significant relationships between limb apraxia and natural gesture use. Conversely Glosser, Wiener and Kaplan (1986) explored aphasics’ use of a set of hand and arm gestures which met designated criteria for communicative behaviours. Their findings supported the belief that aphasics are impaired in their gestural communicative competence in natural conditions of dyadic communication. They further suggest that in aphasia there is a central communication disorder which disrupts referential communicative behaviour in all channels in a parallel fashion. With increasing studies on conversation and aphasia the area of the interaction of conversational competence and nonverbal communication is likely to expand as well. Perhaps, the influence of executive functions will also come into play as the ability to engage nonverbal means of gestural communication may in fact represent a generative skill in corolling flexible and adaptive resources when verbal means fail.

Other studies have examined the use of writing to facilitate conversation (Clausen & Beeson, 2003) as well as the use of laughter as a conversational strategy (Madden, Oelschlaeger & Damico, 2002). Their study found that laughter was used in four ways: as a turn taking cue; a display of understanding; an orienting cue and an instruction to hear. Laughter was often
associated with trouble. As a non-linguistic behaviour, Madden et al (2002) felt that laughter can contribute to the reestablishment of social interaction and meaning-making in a conversation.

2.6.3.5 Conversation reflects a psychosocial process of defining the self in relation to others

The growing interest in conversational research in aphasia has been paralleled by increased awareness of the long term effects of aphasia on psychological wellbeing (Cunningham & Ward, 2003). With perceptions of disability changing all the time speech and language therapists have responded to the critique of therapists being ‘fixers’ rather than ‘enablers’ and are looking more at being the latter. Furthermore Lock, Wilkinson, Bryan, Maxim, Edmundson, Bruce and Moir (2001) have emphasised the fact that the psychosocial functioning of people with aphasia and their partners is closely linked, and therefore psychosocial adjustments for both may be needed. Lock et al., (2001) maintain that targeting aphasia therapy directly at conversation through CA can make a genuine functional difference to their clients because conversation is the vehicle through which we establish and maintain our social identities.

Conversation provides the most natural means by which to establish, maintain and strengthen social ties within the world. It is ‘a vehicle through which selves, relationships and situations are socially constructed’ (Schiffrin, 1988, p. 272). Furthermore, it is the deterioration of the functions associated with conversational ability and the loss of language as a social tool that is often the most distressing aspect of change in the discourse ability for people with brain damage and their caregivers (Kagan & Gailey, 1993; Perkins et al., 1998) and therefore often represents the area which requires the greatest need for intervention by the SLP.

Within the context of CA, several studies have highlighted the psychosocial adaptations appropriated by aphasic individuals as a way of preserving their appearance as a competent communicator and individual. As an example, Wilkinson, Beeke and Maxim (2003) discuss allusions to linguistic difficulties which are seen in response cries e.g. displays of annoyance such as clicks or other exclamatory interjections that are not fully fledged words (Goffman, 1981) and comments on their own performance. Goffman (1981) interprets these behaviours as displays by the aphasic person that overall he/she is a competent individual by showing awareness of the lapse.
Further work has been published in relation to the negotiation of repair between aphasic individuals and their partners. Oelschlaeger and Damico (2003) present the interaction between a husband and wife, where her guess sequences are framed as questions giving her husband the opportunity to continue his turn as competent communicator, rather than simply repairing the utterance with a declarative rendering him inferior in the exchange. These results have been replicated in similar studies (Simmons, 1993; Simmons-Mackie & Damico, 1996).

Beeke (2003) described a patient’s use of the phrase ‘I suppose’ a linguistic construction not otherwise present in his repertoire, for a similar purpose of presenting himself as a person who is essentially able. Under typical circumstances, the construction is preceded by a statement, and then followed by an opinion. There are therefore both linguistic and pragmatic expectations associated with the phrase which characterise the user as someone who a) still has something to say, thus securing the floor space for his turn and b) as someone with something of consequence to impart in the elucidation of an opinion.

It therefore seems natural that the two streams of interest - conversation and psychological wellbeing – have culminated in an increase in research that looks at communication as an interactive process, not just as a transaction of messages. There is increased awareness for the need for individuals with aphasia to be treated as a social unit whose conversation needs support from their partners so that satisfactory exchange can take place.

2.6.3.6 Conversation is sensitive to underlying neural and neuropsychological factors

A third motivation for studying conversation rests on the global perception articulated by Chafe (1997) that 'conversational language reflects the natural workings of the mind more closely than language of any other kind' (p. 52). The complex, adaptive and coordinated system needed for successful participation in conversation is susceptible to breakdown from numerous sources in individuals with aphasia (Penn, 2000). These disruptive influences have formed an important focus in aphasia intervention over the last decade. The intricate contributions of the neural system in conversation suggest that the analysis of conversation would be a sensitive research tool, providing more informative and comprehensive hypotheses of underlying neural functioning than other observational devices, leading to significant clinical and theoretical implications. As examples, Snow, Douglas and Ponsford (1998) found modest correlations between executive function disorder and conversational skill in individuals in chronic stages of
recovery of TBI. Frankel and Penn (2007) used an executive functioning framework to assess
europsychological underpinnings of perseveration as they manifested uniquely in the
conversation of two individuals with TBI.

2.6.3.7 Results of formal language testing do not anticipate conversational performance

Beeke, Wilkinson and Maxim (2003b) conducted a study in which they asked whether language
testing and conversation provide similar results. Their participants completed story retell and
formal language testing and also participated in naturally occurring conversations. They found
conclusively that task based grammar does not mirror conversational grammar. Conversational
grammatical phenomena constitute adaptations to aphasia that occur in the context of real-life,
real-time interaction aiding the production of relatively unproblematic turns at talk despite
linguistic constraints imposed by non fluent aphasia. They conclude that routine assessment of
grammar in conversation is as important as testing elicited grammar and that omission of any
one of these formats results in an incomplete picture of the client’s abilities. Also data shown to
be problematic on testing cannot always be assumed to be problematic in real life. The reverse
is also true – individuals who score relatively well on formal tests are not guaranteed problem
free conversation (Hayashi, Ramsberger & Menn, 2000).

2.6.3.8 CA allows for the differentiation between “normal” and aphasic discourse

Perkins (2003) writes that repair in aphasic discourse has complex organisation that appears to
be structurally different in many respects from repair organisation in normal discourse.
Linguistic impairments necessitate greater use of repair work - but also limit the ability to
execute repair within the same turn of the trouble source. Successful repair outcomes must be
collaboratively achieved – not strictly by ‘self’ or ‘other’ as occurs with conversation that is not
linguistically impaired (Schegloff, Jefferson, & Sacks, 1977). Repair organisation embodies a
preference for quick repair to minimise disruption of current interactional business. In contrast
aphasic trouble sources are often not resolved quickly and complex collaborative work is
required for resolution.

Interlocutors operate on a principle of least collaborative effort to complete repair work as
quickly as possible. Repair is also treated as interactionally delicate and becomes a socially
sensitive event - aphasics can often trace trouble sources back to personal insufficiency in
linguistic processing, giving rise to interactional work that minimises threat to face.
Interlocutors have the option not to repair but let misunderstandings or failures in understanding go by – but this can have notable consequences for interaction for the aphasic person who needs collaborative work in order to make a meaningful contribution to conversation.

2.6.3.9 SUMMARY OF CONVERSATION ISSUES

This discussion has looked at the increased awareness of the need to incorporate conversation as well as long term ideas about psychosocial wellbeing and adaptation of both individuals with aphasia and RHD and their partners into communication intervention. Current research by speech language pathologists using CA has been presented and the need for further studies in the interaction between executive skills and aspects of conversation highlighted. The advantages of the CA methodology have been stressed informing its relevance to the current study. By using CA in this research, it is hoped that the recommendation of Oelschlaeger and Damico (2003) is heeded: ‘we need to employ other assumptions about language and conversation and we need to use more authentic research stances if we are to understand the pragmatic life of brain-damaged patients.’ (p. 211).

“…as an injury, aphasia does reside within the skull. However, as a form of life, a way of being and acting in the world, in concert with others; its proper locus is a distributed multiparty system.” (Goodwin, 1996, p. 31)

2.6.4 PHARMACOLOGICAL TREATMENT OF STROKE

The reviews above have focused exclusively on behavioural interventions. Yet, it remains clear that despite our best efforts, some patients remain resistant to intervention techniques. In such cases, failure to make progress is often ascribed to the severity of the brain damage and resulting psycholinguistic deficits. In such cases, compensation and social approaches to intervention are often included in the treatment plan. However, recovery can be induced in different ways. Recovery of function seems to imply the reconnection or perhaps the re-coordination of a network of areas each of which may be specialised in one or more aspect of the lost function but requires the coherent support from others to reach a high level of proficiency (Weiller, 2000). Effective neuroprotection can be provided by antagonising each of the mechanisms that result in cell death. There is long standing evidence, particularly from animal research that brain plasticity and recovery can be influenced by drugs (Wise, 2003). These strategies include (1) replacement of neurotransmitters lost in cell death (2) antagonising
toxic agents released in cell death (3) decreasing excitotoxic substances (4) enhancing calcium blockade (5) introducing antioxidants and (6) restoring normal circulation. In human studies the vast majority of research related to drug treatment has attempted to salvage potentially viable tissue around the edge of the recent infarct either by thrombolytic therapy to encourage reperfusion or by use of neuroprotective agents (Wise, 2003). Antiplatelet or anticoagulant drugs may be appropriate and research evaluating the efficacy of ‘clot busting’ drugs is under way (Fawcett, Rosser & Dunnett, 2001).

Each strategy can prove effective under appropriate precipitating conditions however it must be recognised that there are a diverse range of mechanisms that can apply and feed back on each other. Future strategies must not only characterise particular mechanisms of cell death to a particular disease condition but also develop effective combinations of neuroprotective therapy that block the cycle and cascade of interacting processes that converge on cell death (Fawcett, Rosser & Dunnett, 2001). Several neurotransmitters have been studied with regard to their effect on brain recovery including catecholamines, acetylcholine, gamma-aminobutyric acid (GABBA) and serotonin. Research relating to each of these neurotransmitter systems will be discussed below.

2.6.4.1 Dopamine
Bromocriptine is the most widely studied of the dopaminergic agents. In 1988, Albert, Bachman, Morgan and Helm-Estabrooks found that bromocriptine in conjunction with traditional speech therapy was associated with improvements in verbal fluency, decreased number of pauses, better naming and fewer paraphasias within ten days of treatment in an individual with transcortical motor aphasia. These improvements were maintained for as long as the patient was receiving treatment. One month after bromocriptine withdrawal, scores returned to equal or below those at baseline. A later study replicated findings of increased fluency with bromocriptine in three patients with transcortical motor aphasia (Sabe, Leiguarda & Starkstein, 1992), although when Sabe and colleagues measured the effect of the same drug on seven other individuals with non fluent aphasia, no significant drug or placebo effects were found on any of the measures (Sabe, Salvarezza, Garcia Cuerva, Leiguarda & Starkstein, 1995). Similarly Gupta, Mlocch, Sclaro and Moritz (1995) were unable to document a significant change in aphasia severity, language content or speech fluency with bromocriptine.
However in a more recent study, Gold, VanDam and Silliman (2000) found improved word retrieval scores and increased speed of cognition on bromocriptine. The most dramatic improvement was seen in the participant with the most frontal damage, disrupting the mesocortical dopaminergic pathways (Klein & Albert, 2004). Dopaminergic projections appear to be involved with initiation, fluency, naming and language content and it therefore makes sense that potentiating dopaminergic availability would improve speech and language, although many of the studies have provided less than compelling evidence (Klein & Albert, 2004).

### 2.6.4.2 Norepinephrine

Norepinephrine originates in the locus ceruleus of the pons in the brainstem and projects to the entire forebrain and primary sensory and motor cortex (Klein & Albert, 2004). Afferents to the locus ceruleus arise from the (DLPFC) dorsolateral prefrontal cortex and therefore disruption of norepinephrine is likely to disturb prefrontal functions (McNamara & Albert, 2003). It is interesting that aphasiologists have noted treatment studies using norepinephrine in relation to depression, but have not connected the disturbance of EF to communication within this particular context. Agents that potentiate norepinephrine have been widely available for the treatment of depression and according to some may also improve overall recovery from brain damage (Finklestein, Weintraub, Karmouz, Askinazi, Davar & Baldessarini, 1987). This is contrasted with nortriptyline, a commonly used tricyclic antidepressant that blocks reuptake of norepinephrine, effectively treating depression but leaving neurological function the same (Lipsey, Robinson, Pearlson, Rao & Price, 1984).

### 2.6.4.3 Amphetamines

Amphetamines are the potentiators of the sympathetic nervous system and act to enhance the release of norepinephrine and block its reuptake (Walker-Batson, Curtis, Natarajan Ford, Dronkers, Salmeron, Lai & Unwin, 2001). In animals, depletion of brainstem catecholamines has been demonstrated after experimentally induced infarcts (Kraus, 1995). Pearlson and Robertson (1981) have reported persistent bilateral depletions of levels of norepinephrine and dopamine in distal areas of the cortex and brainstem following right-sided cortical lesions in rats. After induced strokes, animals have been evaluated for effects of dextroamphetamine on motor function, sensory integration and depth perception. Dextroamphetamine was found to be related to enhanced neural sprouting and synaptogenesis, and effects were amplified when paired with practice. It can also enhance the beneficial effects of physical therapy after cortical
Injury (Hesse & Werner, 2003). Furthermore, these improvements have been sustained in animal stroke models (Kertesz & Gold, 2003).

In humans amphetamines have also had positive effects on stroke recovery by reversing the diffuse cortical depression of glucose metabolism (Small, 1994). Walker-Batson et al. (2001) studied 21 patients randomized to administration of either dextroamphetamine or placebo. The group receiving the drug had accelerated rate of recovery although this study was compromised by the fact that the drug therapy was administered during an active period of spontaneous recovery. Several studies have demonstrated the alleviation or complete recovery from post stroke depression with the psychostimulant methylphenidate (Kraus & Burch, 1992; Lazarus, Winemiller, Lingam, Neyman, Hartman, et al., 1992; Lingam, Lazarus, Groves, & Oh, 1988). Grade, Redford, Chrostowski, Toussaint and Blackwell (1998) studied the effects of methylphenidate on stroke rehabilitation in 21 acute stroke survivors. Patients in the treatment group were less depressed and scored better on motor and Activity of Daily Living scales with no adverse effects.

Tiberti, Sabe, Jason, Leiguarda and Starkstein (1998) investigated whether methylphenidate could improve memory in people with organic amnesia resulting from stroke. Since methylphenidate increases the dopaminergic turnover in the DLPFC, as well as increasing the turnover of norepinephrine, dopamine and serotonin from presynaptic nerve terminals it was thought that it may increase attention. However, no neuropsychological benefits were found in this study. On the other hand Johnson, Roberts, Ross and Witten (1992) found that in a sample of 10 patients, 7 showed improved mood and attention span with methylphenidate treatment.

2.6.4.4 Serotonin

Serotonin is also a central neurotransmitter that can be manipulated by various medications whether through selective reuptake inhibitors or through 5HTA serotonin receptor agonists (Klein & Albert, 2004). Reading, Orto, Winter, Fortuna, DiPonte and McDowell (1986) evaluated rehabilitation outcomes in depressed patients treated with trazodone (a serotonin receptor agonist) compared with placebo. The treatment group showed consistent improvements thought to be attributable to activation, motivation and participation. Tanaka and Albert (2001) found improvements in mood and naming in patients with fluent aphasia using a selective
serotonin reuptake inhibitor – fluvoxamine maleate – which was attributable to decreased depression and less perseveration.

2.6.4.5 Acetylcholine

Acetylcholine is found in most places in the nervous system. Primary sources include the two cerebral nuclei at the base of the forebrain. There is some evidence that cholinergic activity is greater in the left hemisphere than in the right, especially in the temporal lobe (Klein & Albert, 2004). There is some evidence that acetylcholine has a neuroprotective effect during ischaemic hypoxia and that 5-citidine diphosphocholine started within 24 hours of onset can increase rate of recovery (Clark, Warach, Pettigrew, Gammans & Sabounjian, 1997).

Piracetam which is actually a gamma aminobutyric acid derivative has direct cholinergic effects via the release of acetylcholine and its effect on recovery from stroke has been widely studied. Learning and memory were improved in animal models of a variety of behavioural procedures in preclinical studies with piracetam. The benefits were most pronounced in animals subjected to hypoxia drug intoxication, electric shock and aging (Genton & Van Vleymen, 2000) and is currently indicated for the treatment of cognitive impairments including memory disturbances in age-related cognitive function or decline, the early stages of dementia (Croisile, Trillet, Fondarai, Laurent, Mauguiere & Billardon, 1993) and post-stroke aphasia (Enderby, Broeckx, Hospers, Schildermans & Deberdt, 1994; Orgogozo, 1998; Poeck, 1998). It was for these reasons that piracetam was strongly considered when this study was designed.

Huber (1999) found that piracetam improved aphasia in acute stroke and as an adjuvant to language therapy in sub-acute and chronic aphasia. Burd, Gekht, Bogolepova and Buklina, (1997) further found that restoration of high mental functions occurred faster during piracetam treatment as compared with basic therapy only. They concluded that piracetam can be prescribed for patients with hemispheric ischemic stroke and in particular those with alterations of cerebral circulation in the internal carotid artery system.

In a preliminary double blind, placebo controlled study on functional communication in two adults with chronic aphasia improvement was noted specifically in conversational skills (as well as reading and writing) over the eight week period of the study (Rudolph, 1998). The two participants demonstrated improvements on two established functional measures, which was
further validated by the observations of significant others and the subjective comments of the participants who reported that they felt “more alert”, “more organised” and could “think more”. These functions were interpreted to be critical to the support of communicative functions, allowing more efficient deployment of communicative resources in the presence of better cognitive control.

Huber, Willmes, Poeck, Van Vleyman and Deberdt (1997) found a significant reduction in overall aphasia severity, especially in written language with piracetam in combination with speech therapy. Kessler, Thiel, Karbe and Weiss (2000) found improvement in cerebral blood flow on PET scans in language areas with concomitant improvements in speech and language. Once again however, this study recruited participants during periods of spontaneous recovery.

Direct cholinergic effects have also been studied with the use of bifemelane in four patients with left temporal lobe lesions. Improvements were noted in auditory comprehension, animal naming and confrontation naming in the two patients who received drug treatment as compared to the two who received speech therapy alone (Tanaka, Miyazaki & Albert, 1997). Aniracetam, another centrally active cholinergic agent also resulted in improvements in naming, word generation and decreased perseveration (Tanaka & Albert, 2001) supporting the potential benefits of cholinergic agents for the treatment of aphasia.

2.6.4.6 Gamma-aminobutyric Acid (GABA)

GABA is an excitatory neurotransmitter acting to inhibit neurotransmitters in all areas of the brain (Klein & Albert, 2004). It interacts closely with glutamate, norepinephrine and acetylcholine. GABA agonists are often used to treat agitation, seizures, spasticity and insomnia (Berthier, Hinojoa, Mdel & Fernandez, 2003). One such agonist, sodium amytal, has been studied for its potential efficacy in treating aphasia. It was administered intravenously to two patients who had aphasia. One showed improved language and the other also improved but returned to baseline when the drug was withdrawn (Walker-Batson, 1998).

2.6.4.7 SUMMARY OF PHARMACOLOGICAL TREATMENT OF STROKE

Although well established for over 20 years, the study of pharmacological agents in the treatment of aphasia and other neurogenic communication disorders is to some extent considered revolutionary. While many research studies have not convincingly shown
amelioration of the target deficit, there is enough evidence to suggest that we should persevere with these lines of enquiry. Firstly, because it makes sense to stabilise the neurochemical environment at the site of lesion, in order to further support behavioural therapy techniques and speed recovery. More importantly, pharmacology offers an optimistic avenue for intervention in the face of intractable behavioural and communication difficulties resistant to other intervention techniques and which may arise from fundamental EF deficits that are not currently addressed in models of aphasia and RHD.

2.7 CONCLUDING COMMENTS

This chapter has introduced the topic of stroke and contextualised the current study within the challenging and under-serviced context of South African. In a country where fighting crime and poverty are among our primary concerns, little is known about stroke, despite the fact that the impact of burden is keenly felt among our older population and in all likelihood will accelerate in the coming years. Contributing to the understanding of the mechanisms that cause stroke and affect recovery in functional and real life settings is therefore of significant importance. Neuroanatomical and neurotransmitter substrates as well as psycholinguistic processing were presented as the first set of relevant contributors towards conversational intelligence.

A number of communication deficits arise from both left and right sided lesions, which also affect EF and therefore potentially impede recovery and undermine the success of traditional therapeutic interventions. Modern approaches which highlight wellbeing and quality of life by focusing on the collaborative nature of communication and encouraging life participation may have more to offer, especially since these approaches focus so heavily on conversation, a cornerstone of our social lives. The importance of conversation and the usefulness of CA in facilitating research about stroke-related communication deficits was therefore highlighted. Finally, the relevance of exploring pharmacological agents in the management of the cascade of brain events following stroke was discussed, with a review of existing literature exploring the effect of pharmacology on communication deficits. The following chapter presents a detailed review of executive functions and their contribution to conversational intelligence.
In the past twenty odd years, executive functions (EF) have formed the focus of over 3000 research studies in a wide range of fields including normal development, addiction (gambling and drug), psychiatric disorders, pervasive developmental disorders, traumatic brain injury, dementia, stroke, Parkinson’s disease, learning disability, attention deficit hyperactivity disorder (ADHD) and bilingualism, among others. Over the past two decades, theories of frontal function incorporating their executive nature have evolved. Considering the earliest theories in relation to current viewpoints, the outlooks are not so much exclusionary as they are more elaborated and integrated. As science and technology have advanced, we have been afforded a view of the brain that continuously expands our horizons and challenges established frontiers. EFs have always been characterized as those processes and abilities that guide and direct goal oriented behaviour. The centrality of the frontal and especially the prefrontal cortex and its reciprocal connections to EF has remained a constant throughout decades of investigation. Now, we are investigating EF as a potentially important feature of conversational intelligence. This chapter will provide an overview of definitions of EF, supporting neuroanatomy, in particular the prefrontal cortex, neurochemical modulation, the impact of ageing on EF as well as provide an overview of early theories of frontal lobe function and their evolution to existing EF models.

3.1 DEFINITIONS

As essential as EF is for human existence, it is as elusive to conceptualise and measure (Filley, 2000). The term executive function defines complex cognitive processing requiring the coordination of several sub-processes to achieve a particular goal (Elliott, 2003). Lezak (1995) states that executive functions are ‘the capacities that enable a person to engage successfully in independent, purposeful, self-serving behaviours’ (p.42). It is widely accepted that executive functions may not be activated during the execution of well-learned routine behaviours but are
enlisted in novel or unfamiliar circumstances, in which no previously established routines for responding exist. Anderson, Levin and Jacobs (2002) operationalize the components of EF as: 1) attentional control – including selective attention, sustained attention and response inhibition 2) goal setting - incorporating initiating, planning, problem solving and strategic behaviour and 3) cognitive flexibility - entailing working memory, attentional shift, self-monitoring, conceptual transfer and self-regulation. Temporal integration and recruiting or integrating multimodal inputs from throughout the brain are also thought to be critical elements of EF (Keil & Kaszniak, 2002).

Executive dysfunction therefore may be reflected by poor planning and organization, difficulties generating and implementing strategies for problem solving, perseveration, inability to correct errors or use feedback and rigid or concrete thought processes (Stuss & Benson, 1986). Qualitative features may include poor initiation, inflexibility, reduced self control, impulsivity and erratic or careless response behaviours and deficient high level communication (Anderson et al., 2002).

### 3.2 EXECUTIVE FUNCTIONS AND CONVERSATIONAL INTELLIGENCE

It is becoming clearer that cognitive functions are important predictors for successful treatment and rehabilitation of stroke patients (Riepe, Riss, Bittner & Huber, 2004). Even people with mild cognitive deficits have been found to be more likely to be institutionalised than the cognitively unimpaired (Riepe et al., 2004). The ultimate goal of any speech-language intervention is to harness learning processes to improve communication in everyday settings, where unpredictable demands and fluctuating conditions require goal oriented behaviour and flexible problem solving (Helm-Estabrooks, 2002). It is therefore critical that we develop a more comprehensive understanding of the realm of EF, its interruption in the presence of neurologic communication deficits and if and how this impacts on everyday interaction as manifested in conversation.

Miyake, Emerson and Friedman (2000) have said in relation to aphasia that everyday communication is not just dependent on language specific processes but also on a host of non-linguistic executive processes. These include paying attention to the communication partner, sustaining attention, appropriately sequencing pieces of information, monitoring ongoing communication processes and employing flexible shifting strategies when necessary. The
integrity of EF may be an important determinant of how well aphasic patients succeed in everyday communication. Differences in degree of communicative success for individuals with aphasia and those with right hemisphere damage as a result of stroke may be related to their abilities to perform certain tasks designed to capture aspects of EF.

There are conspicuously few studies examining the impact of EF on conversational skill in this population. There are notable exceptions. One of the first writers to highlight the impact of executive dysfunction on communication was Hartley (1995). She presented the linguistic, cognitive and executive/behavioural changes that follow brain injury and specified their proposed effects on social communication. Also in relation to TBI, Frankel and Penn (2007) investigated the manifestations of perseveration in conversational discourse and the possible relationships of such manifestations to various executive functions, most significantly behavioural inhibition and interference control.

In 1992, Purdy wrote that people with aphasia lack the ability to initiate, plan, monitor and correct their own communicative performance and thus are unable to use available verbal and nonverbal abilities to achieve their goals. In her study she examined performance of individuals with aphasia on the Porteus Mazes, Tower of London (TOL), Tower of Hanoi (TOH), Wisconsin Card Sorting Test (WCST) and block design from the WAIS-Revised. She also examined the ability of these participants to learn and use a number of communication techniques. She found that participants learned the techniques but only switched them 41% of the time. Those who did switch techniques were successful 67% of the time in their communication efforts. Purdy attributed lack of success on communication tasks to deficits in attention allocation.

Fridriksson, Nettles, Davis, Morrow, and Montgomery, (2006) tested a group of 25 individuals with single event stroke induced aphasia on the WCST and Colour Trails Test (CTT). They measured functional communication outcomes using the ASHA-FACS. Their findings suggested that executive functioning and functional communication were closely related in aphasia. They further suggested that the skills needed for CTT are also needed in functional communication (sequencing, inhibition, planning, cognitive flexibility, working memory, perceptual tracking, grapho-motor skills and sustained and divided attention).
Another important study looking at the impact of attention on conversation was conducted by Hardin and Ramsberger (2004). A non-linguistic computerized training battery was used to determine whether changes in attention translated to more successful conversations with unfamiliar partners. Conversation was measured in terms of the Measured Transactional Success in Conversation model (Ramsberger, Rende & Kurland, 2003 cited in Hardin & Ramsberger, 2004). Statistically significant improvements were made in attentional skills and functional communication, but, notably, no improvements were found on linguistic measures. Their conclusions are highly pertinent to this study. It is possible that conversational success relies more heavily on attentional skills and EF than on linguistic abilities. Also linguistic tasks may not sufficiently stress the attentional system to a degree that they produce changes in function. The implication for future research is twofold. First as was highlighted at the end of the previous chapter, research needs to examine the EF profiles of individuals with stroke in more detail in order to better understand relevant contributions to communication deficit. Second, that complex and taxing communication tasks such as those that occur in everyday conversation need to feature in both our research and clinical assessment and treatment of communication disorders in stroke.

The characteristics described above provide the rationale for investigating EF as a potentially significant component of conversational intelligence, as conversation requires all of the above mentioned skills. Attentional control must be exerted in order to concentrate on the interaction and person/people with whom one is interacting with as well as cope with distractions such as interruptions, or background noise. Goal setting is exercised through the formulation and response to topic initiations, shifts and terminations. Planning and strategic behaviours are often called into play to repair conversational trouble spots. Cognitive flexibility is necessary for integrating previously stated information and using shared background appropriately throughout the exchange. The ability to monitor one’s own contributions by gauging the response of the listener is also an integral skill to successful interaction. As a result of the fact that conversations typically produce novel material, adaptations to flexible and creative communication encounters are necessary. While these skills are critical in single floor interactions (where one person speaks at a time), they are in even greater demand during multiparty interactions which often produce group floors – where meaning is co-created between a number of participants (Edelsky, 1981), who all interrupt, overlap and generally talk at the same time. The ability to integrate multimodal inputs under these circumstances is
essential for successful inclusion in conversation. In order to understand these inputs to conversation, a thorough understanding of EF is needed.

3.3 ANATOMY OF EXECUTIVE FUNCTIONS

3.3.1 The significance of the Prefrontal Cortex (PFC)

The Prefrontal Cortex (PFC), occupying approximately 25% (or up to 1/3) of all cerebral cortex in the brain is disproportionately large in humans (Preuss, 2000), and is generally accepted to be the primary anatomical basis for the EF (Koechlin & Summerfield, 2007). The frontal cortex comprises all of the cortical area in front of the central sulcus. The area just in front of the central sulcus - between it and the precentral sulcus - is primary motor cortex (Brodmann’s area 4). In front of that is the premotor cortex and the supplementary motor area (SMA), both sub-regions of Brodmann’s area 6. All of the cortical area in front of that is PFC (Mesulam, 2002). The dorsolateral prefrontal cortex (DLPFC) extends over the superior and middle frontal gyri. Areas 9, 46, and 9/46 comprise the core of the DLPFC (mid-dorsolateral PFC) (Petrides & Pandya, 1999), while area 8 constitutes the posterior portion of the DLPFC and area 10 the anterior portion. Areas 44, 45 and 47/12, all of which lie on the inferior frontal gyrus, comprise the ventrolateral PFC (Diamond, 2002). In relation to this discussion, it is significant to note that within the carotid system, the anterior and middle cerebral arteries provide the blood supply for all the of the anterior cerebral hemispheres. Furthermore, it is within the distribution of the middle cerebral artery that most strokes occur (Robinson, 2006). It is highly likely therefore that anterior portions of the brain, critical in supporting EF, are susceptible to disruption in the majority of strokes. Figure 3.1 on the following page, depicts the areas under discussion.

3.3.2 Neuroanatomy of the PFC

The prefrontal cortical areas give rise to efferent connections that are directed back to the same areas from which they received input (Petrides & Pandya, 2002). These pathways therefore provide particular prefrontal areas with sensory specific or multimodal information, at the same time providing the means by which the PFC can regulate information processing in the posterior cortical areas. The neuroanatomic subdivisions within the frontal lobes subserve different neurobehavioural functions and can be divided into three different regions (Filley, 2000).
First, sensory information in post-Rolandic areas is provided to the orbitofrontal cortical region. This area is extensively connected to the hypothalamus, amygdala, hippocampus and also the other paralimbic cortices in the temporal pole, insula, parahippocampal gyrus and cingulate gyrus (Mesulam, 2002). The significance of these connections is that they are thought to be critical for the regulation of emotional consequence and motivational value of stimuli. Second, the mid-ventrolateral prefrontal region via its strong bidirectional connections can exercise a top-down modulation of activity for the purpose of strategic judgment, encoding, and retrieval of information (Petrides & Pandya, 2002). Third, the dorsolateral prefrontal region is critical for monitoring of information in working memory, necessary for high level planning and manipulation of information. The interaction between the mid-dorsolateral prefrontal region and the memory system may be exercised via the dorsal limbic pathway that links reciprocally with the hippocampal system. The posterior dorsolateral frontal cortex appears to underlie
attentional processes. All these regions have links to the subcortex so that deep structure damage may also result in EF deficit. If one were to impose the components of conversation onto the concepts described above, the necessity of EF to conversational functioning is easily appreciated. Small wonder then that conversation more than any other communicative act is so vulnerable in the wake of brain insult.

The following sections provide an overview of the neurochemical modulation of the PFC and a summary of relevant research linking these neurochemical substances to various EFs. It is notable that there is no research relating these systems to corresponding EF functions within the stroke population, despite the fact that there is now a well established research base to indicate that EF deficit represents a core deficit of post-stroke disability (Godefroy & Stuss, 2007).

3.3.3 Neurochemistry of the PFC

The cognitive functions of the PFC are among the most fragile in our behavioural repertoire (Arnsten & Robbins, 2002). Abilities carried out by the PFC can also become impaired in so-called “normal” individuals under conditions of stress, fatigue, and with advancing age. Many of these changes clearly result from transient neurochemical changes. Animal research indicates that the PFC is highly sensitive to its neurochemical environment. Ascending monoaminergic and cholinergic systems have marked effects as part of their orchestration of cortical arousal. Dopamine (DA), norepinephrine (NE), serotonin (5HT) and acetylcholine (ACh) may modulate PFC cognitive functions. Research has shown that even small cortical lesions can cause significant disruptions of neurochemical functioning because of disruption of the blood-brain barrier and can affect sites distant from the lesion itself (Klein & Albert, 2004). The complex cascade of events and their repercussions form the basis of a number of rehabilitation approaches – behavioural as well as pharmacological. An understanding of these connections therefore informs the impact of neurochemical disruption in individuals who have experienced stroke, with the resulting disturbance in communication, as well as providing some direction about which systems to target when treating dysexecutive syndromes. Figure 3.2 on the following page, depicts the main anatomical connections between chemically defined ascending systems of the reticular core and the neocortex.
3.3.3.1 Dopamine

Working memory is a critical component of EF and has significant impact on the ability of individuals to participate meaningfully in conversation. The two elements integral to working memory, sensory retrospective memory and sensory prospective memory, allow individuals to remember the past and engage in preparation to act, respectively (Fuster, 1995; Goldman-Rakic, 1995). People are able to construct hypotheses about how to act in the present and future based on previous experience, thus if one is unable to look back, one is unable to look forward, remaining instead in the dislocated present (Barkley, 1998). Therefore, patients with working memory deficits may display a tendency to be more influenced by context and external stimuli and less controlled by internally represented information, giving rise to a temporal myopia (Stuss & Benson, 1986). In conversation, this would result in impaired ability to maintain the thread of discourse, particularly in multi-party interactions, difficulty integrating new
information, poor application of shared information and reduced coherence and organization.

Twenty years of research have established the critical effect dopamine (DA) has on PFC working memory. Too little or too much DA receptor stimulation impairs spatial working memory in monkeys and rodents. Brozoski, Brown, Rosvold and Goldman (1979) demonstrated that monkeys with large catecholamine depletion of the PFC were as impaired as animals with PFC ablations on specifically PFC tasks. This research has allowed investigators to appreciate that both catecholamines (DA and NE) must be substantially depleted to produce marked impairment (Arnsten & Robbins, 2002). The results of the Brozoski et al. (1979) study have been replicated in rats and marmosets.

Deutch and Roth (1990) have also demonstrated that exposure to even relatively mild, uncontrollable stress greatly increases DA release in the PFC, which in turn impairs working memory function through excessive stimulation. These studies have particular relevance as many neuropsychiatric disorders are exacerbated or precipitated by stress, including those related to stroke such as depression and anxiety (Robinson, 2006). Exposure to environmental stress such as loud noise or pharmacological stressors impairs performance of spatial working memory in monkeys and rats with these same stressors improving performance on tasks dependent on the inferior temporal cortex, parietal cortex, striatum and cerebellum. A similar profile has been observed in human studies. In people, noise stress impaired the ability to sustain attention or inhibit the prepotent response in the Stroop Interference Test (Hartley & Adams, 1974).

Interestingly, three tasks shown to be sensitive to lesions of the PFC, spatial delayed response, self ordered sequencing and a model of the Wisconsin Card Sorting Test exhibit differential effects of prefrontal DA loss: deficit, no effect and enhancement respectively (Arnsten & Robbins, 2002). This means that different operations possibly mediated by distinct regions of the PFC may have differing neurochemical needs in terms of the modulatory functions provided by the DA system (Robbins, 2000).

In humans, there are two main sources of evidence for the role of DA in PFC function. These are studies of patients with disorders that implicate the DA system and studies on the effects of dopaminergic drugs in normal subjects. This work is now being augmented with functional neuroimaging, general positron emission tomography (PET) and more recently functional
magnetic resonance imaging (fMRI), to measure effects on regional cerebral blood flow (rCBF).

In 1992, Lange, Robbins, Marsden, James, Owen and Paul showed that l-Dopa withdrawal from a small group of ten Parkinson’s disease (PD) patients selectively impaired their performance on tests of spatial working memory, planning and various tests of visual discrimination learning. Latency as well as accuracy of thinking on the planning task was affected. However, these results are not unequivocal as dopaminergic medication does not always have cognitive benefits. Gotham, Brown and Marsden (1988) provided evidence for differential responses of cognitive functions in response to l-Dopa and explained it in terms of the pattern and course of DA loss within the striatum. Those regions suffering from extensive DA loss would be optimally stimulated by medication. However, other areas that were relatively spared in early stages would potentially be disrupted by medication as the level of DA function would be set supra-optimally by the drug.

The effects of the D2 receptor agonist bromocriptine on working memory and other executive functions in individuals with traumatic brain injury was examined in a double-blind crossover trial with placebo (McDowell, Whyte & D’Esposito, 1998). Bromocriptine improved performance on some but not all tasks thought to be subserved by the PFC. Making inferences about the functions of DA in cognition is less promising in schizophrenia, as antipsychotic medication may produce indirect effects by the remediation of disruptive positive symptoms. Also as with neuroleptic drugs, cognitive functioning can be impaired as a side effect.

When this study was initially conceptualized, the use of methylphenidate (Ritalin) for the drug trial was initially considered. Firstly, because previous research with two individuals who had sustained TBI showed measurable improvements of EF considered critical i.e. behavioural inhibition. Second, because amphetamines and similar compounds have been implicated to produce effects on the dopaminergic system, thereby improving not only attention but also working memory functions as described above. However, the uncertainties about whether the treatment “damps down” unwanted activity or boosts deficient functioning hinders understanding – and this is seen clearly in the treatment of ADHD. Evidence implicates the dopaminergic system and prefrontal nigrostriatal regions in the pathophysiology of childhood ADHD and prefrontal catecholaminergic dysfunction in adult ADHD but to what extent the
beneficial effects of drugs like methylphenidate (Ritalin) depend on modulation of dopaminergic or noradrenergic neurotransmission or both, remains unclear (Ernst, Zametkin, Matohik, Jons & Cohen, 1998). Ultimately, Ritalin was rejected as the drug of choice for this study for far more practical reasons, as discussed later, although this brief discussion bears on the complexity of the choices to be made.

In summary, DA exhibits an inverted $U$ dose-response function in regard to PFC working memory and attentional functions through its actions in animals. Low levels of D1 receptor stimulation are essential for working memory but high levels such as occur during stress impair working memory (Arnsten & Robbins, 2002). In humans, altered DA likely contributes towards many PFC disorders including PD, schizophrenia, ADHD and as this research proposes, dysexecutive syndromes following stroke. Data from normal volunteers suggests that DA mechanisms likely exhibit the same inverted $U$ dose-response curve in humans, just as in animals (Arnsten & Robbins, 2002).

### 3.3.3.2 Norepinephrine

Another neurochemical substance thought to be involved in the regulation of PFC working memory and attention is norepinephrine (NE). Studies in rodents, monkeys and humans have all shown that NE has important beneficial influence on PFC function through its actions at postsynaptic $\alpha$-2 receptors. Young monkeys with working memory impairment induced by local PFC or global catecholamine depletion are improved by treatment of $\alpha$-2 agonists. The same effect has been seen in aged rats and monkeys with naturally occurring catecholamine loss (Arnsten & Goldman-Rakic, 1985). The greater the loss of NE the lower the dose of $\alpha$-2 agonist needed to improve PFC performance (Arnsten & Goldman-Rakic, 1985). Imaging studies in monkeys are also consistent with $\alpha$-2 adrenergic mechanisms enhancing PFC function. Increased rCBF has been observed in the dorsolateral PFC of monkeys treated with guanfacine (an $\alpha$-2 agonist) prior to performing a spatial working memory task.

$\alpha$-2 agonists have also been shown to be particularly effective under distracting conditions (Jackson & Buccafusco, 1991), consistent with research that shows that forebrain NE depletion increases distractibility (Carli, Robbins, Evenden & Everitt, 1983). Higher doses of guanfacine have also been shown to improve performance on object reversal tasks, improve attentional regulation, behavioural inhibition and planning (Steere & Arnsten, 1997).
In humans, α-2 agonists have been shown to improve performance of PFC tasks in patients with PFC dysfunction. For example clonidine improved memory recall and the Stroop Interference Task in patients with Korsakoff’s amnesia (Mair & McEntree, 1986). It has also been effective in improving performance on the Trails B Task in schizophrenia (Fields, Van Kammen, Peters, Rosen, Van Kammen, et al., 1988). Much of the research with α-2 agonists has focused on patients with ADHD, a disorder with prominent PFC dysfunction. Guanfacine has been shown to improve performance on tasks of vigilance, working memory and behavioural inhibition (Scahill, Chappell, Kim, Schultz, Katsovich, et al., 2001). These findings are consistent with the clinical reports that guanfacine reduces impulsivity, a sign of improved PFC function. Imaging studies generally support findings from animal research (Arnsten & Robbins, 2002). These drugs however are typically prescribed in order to lower blood pressure and therefore have direct effects on blood circulation. Given that several of the participants were already taking medication to control blood pressure, and that guanfacine in particular is contra-indicated in individuals with coronary heart disease, these substances could not be considered for use in this trial.

3.3.3.3 Serotonin

Relatively little is known about the precise functions of the indoleamine serotonin (5HT) in cognition especially with regard to PFC function. Current research seems to suggest that the PFC may be more sensitive to catecholamine than serotonergic influences. However, the pharmacology of serotonin is highly complex and according to Arnsten and Robbins (2002), it is likely that serotonergic influences will be observed when the right pharmacological tools are applied. In humans, 5HT appears to modulate functions controlled by the orbitofrontal cortex to a greater extent than those of the dorsolateral PFC, which may explain the importance of 5HT in depression (Arnsten & Robbins, 2002). This is significant to bear in mind and will be returned to in the results and discussion chapter, in the section detailing regulation of affect (6.2.4).

3.3.3.4 Acetylcholine

ACh is commonly associated with the intellectual deterioration of Alzheimer’s disease (AD) from histo-pathological evidence of degeneration of the basal forebrain cholinergic system projecting to the cerebral cortex and a corresponding reduction of neocortical cholinergic markers (Perry, Tomlinson, Blessed, Bergmann, Gibson & Perry, 1978). Similar relationships
have been reported for PD and Lewy Body dementia (Perry, Walker & Perry, 1999). Much of the relevant evidence has been derived from rats and monkeys after they have developed brain lesions of the basal forebrain or received an infusion of cholinergic agents into the prefrontal regions. The implication of the cholinergic system in attentional functions has been substantiated in rats (Sarter & Bruno, 1997).

Other approaches have suggested that ACh contributes to working memory within the rat prefrontal cortex and these effects were more convincing than those obtained following similar infusions of D1 antagonists. There have been relatively few studies of the role of prefrontal ACh in cognition. Marmosets have however demonstrated reductions in distractibility when irrelevant background stimuli were manipulated in response to nonselective excitotoxic lesions of the basal forebrain. These findings are consistent with the existence of strong projections of the orbitofrontal cortex to the basal forebrain with significant cholinergic activity associated with orbitofrontal function (Mesulam, 1995 in Arnsten & Robbins, 2002).

In humans, a study by Furey, Pietrini and Haxby (2000) examining the relationship between cholinergic drug treatments and changes in rCBF showed that ACh can reduce the burden on working memory by increasing visual encoding. To sum up, while animal studies have implicated ACh in attention and working memory, the relevance of current findings to specific cholinergic mechanisms in the human PFC is not well understood.

### 3.4 EXECUTIVE FUNCTIONS AND AGEING

The PFC undergoes one of the longest periods of development of any brain region, taking over two decades to reach full maturity (Sowell, Thompson, Holmes, Jernigan & Toga, 1999a). During the first year of life significant changes occur that allow cognitive advances by one year of age. This brief review focuses on the EF associated with the PFC in ageing. This topic is important to discuss bearing in mind the age of the participants in this sample and the fact that in general, after the age of 55 years, the risk of stroke doubles every ten years (Mlcoch & Metter, 1994). This means that individuals who suffer EF deficits secondary to stroke in later years, may already have experienced a reduction in EF capacity as a result of the ageing process to a greater or lesser extent.
Some older adults show significant cognitive deficits whereas others perform as well as young adults. With age, our brains undergo a series of changes including white and grey matter atrophy, synaptic degeneration, blood flow reductions and neurochemical alterations (Raz, 2000). It is therefore not surprising that older adults perform less well in tasks related to attention, perception and memory (Craik & Salthouse, 2000) as well as have more difficulty developing logical strategies (Allain, Chaudet, Nicoleau, Pinon, Etcharry-Bouyx, et al., 2007). Studies have also demonstrated that ageing can be accompanied by a functional dysregulation of motor cortex excitability during sensorimotor processing, with this deficit becoming progressively evident with greater task complexity (Yordanova, Kolev, Hohnsbein & Falkenstein, 2004).

Some studies have suggested that cardio-respiratory fitness may have a selective protective effect against age-associated cognitive decline as fitness measured by VO2max was a strong predictor of cognition and accounted for more variance in processing resources than in higher-order functions (Newson & Kemps, 2006). Cabeza, Anderson, Locantore and McIntosh (2002) suggest that high performing older adults counteract age-related neural decline by reorganising brain functions. In PET and fMRI studies prefrontal cortex activations are sometimes lateralised, possibly reflecting the nature of the processes and/or stimuli involved. Lateralisation of PFC activations tends to be reduced by aging and has been conceptualised in terms of the Hemispheric Asymmetry Reduction in Old Adults (HAROLD) model. The HAROLD model is supported by functional neuroimaging evidence in a variety of cognitive domains of which the relevant executive functions include working memory (Dixit, Gerton, Dohn, Meyer-Lindenberg & Berman, 2000; Reuter-Lorenz, Jonides, Smith, Hartley, Miller, et al., 2000) and inhibitory control (Nielson, Langenecker & Garavan, 2002). Age related asymmetry reductions have also been observed in electrophysiological and behavioural studies (Bellis, Nicol & Kraus, 2000 and Reuter-Lorenz, Stanczak & Miller, 1999 respectively).

According to a compensation hypothesis (Cabeza, 2002), increased bilaterality in old adults could counteract age related neurocognitive deficits. This hypothesis is also supported by evidence that hemispheric asymmetry reductions may also facilitate faster recovery from brain damage by the recruitment of the unaffected non-dominant hemisphere. An alternative account of age-related asymmetry reduction is the dedifferentiation hypothesis. Here reduced hemispheric asymmetry may reflect age related difficulty in recruiting specialised neural
mechanisms (Li & Lindenberger, 1999). The notion of dedifferentiation is supported by evidence that correlations among different cognitive measures tend to increase with age. According to this hypothesis, age-related asymmetry reductions are just another example of the deleterious effects of aging on the brain (Cabeza et al., 2002).

In their study examining each of these two hypotheses, Cabeza et al. (2002) examined three groups of participants, young adults (20-35 years), Old-High adults (older adults aged between 64 – 78 years who performed as well as the young adults on a battery of cognitive tests) and Old-Low adults (older adults aged 63 – 74 years who performed significantly worse than the younger adults on the same battery). If the HAROLD pattern was present during source memory in Old-High adults, then the findings would lend support to the compensation hypothesis. Conversely if the HAROLD pattern was present in the Old-Low group, this would support the dedifferentiation hypothesis.

Behavioural and PET data showed strong support for the compensation hypothesis. PFC activity during source memory was right lateralised in young and Old-Low participants but bilateral in Old-High adults. These findings would suggest that HAROLD is a beneficial change not a deleterious side effect of aging. The findings imply that the Old-Low group engaged a similar memory network as young participants but used it much less efficiently whereas Old-High participants compensated for age related memory decline by reorganising memory networks.

3.5 THEORIES OF EXECUTIVE FUNCTION

As science has evolved, so too naturally have theories of EF. Starting with Pribram (1960), theories began based on behavioural observations and gradually, increasingly complex neuroanatomical, neuropsychological and neurochemical aspects were incorporated. This review presents the major contributors over the past 50 years. The concluding summary links the theories in terms of their similarities and novel contributions. Each theory contributed towards an understanding of specific components of EF, although it is clear that few incorporated several dimensions of EF or defined the inter-relationships among the components we now consider to be executive in nature.
3.5.1  Pribram – Feedback system

Pribram (1960) conceptualized the frontal lobe as a system involved in the resolution of a state of incongruity which exists between an organism and a stimulus, resulting in a testing of operations until the incongruity is resolved. Thus feedback is provided from results of testing and is used to form future instructions and also assists control. This theory was built on earlier work by Miller, Gallanter and Pribram (1960 cited in Stuss & Benson, 1986) who posited a test-operative-test-exit (TOTE) system viewed as an organizing, coordinating unit. The TOTE system was seen as having two main properties: a planning characteristic which controls both construction of tasks to be performed and the sequence of operations to be carried out. The second property is the operation phase, subserving both actions to be done and the actual operation thus realizing an external representation of neuro-programmes in the brain. In 1973 the notion that behaviour was context-dependent was added to the model. In this way, Pribram (1973) accounted for behaviour in individuals with frontal lobe damage as having absent or inappropriate schedules or routines resulting in impaired behaviour and planning. If external flexible noticing orders are imposed such as markers or cues, markers that are generally internally represented in the undamaged brain, performance may be improved to near normal levels. This theory conceptualizes EF as comprising predominantly monitoring abilities, yet fails to address the most fundamental of skills namely, attention and attentional control. While the ability to monitor and utilise contextual feedback regarding the success or lack thereof in the pursuit of goal directed behaviour is certainly a key element of EF, the exclusion of basic operational systems renders this theory extremely simplistic.

3.5.2  Teuber - Corollary discharge

Teuber’s theory represented a radical change from what had preceded it (Stuss & Benson, 1986). Historically neuroscientists considered that brain function began in posterior sensory regions. However, Teuber proposed that the reverse might actually be a better explanation by demonstrating how anterior anticipatory processing acts on sensory function. Anticipatory discharges travel from motor to sensory areas to prepare sensory structures for anticipated change of voluntary action. Every voluntary movement has two neural correlates – a stream of impulses to effectors and a simultaneous corollary discharge to central receptor structures presetting the latter for predictable changes of input that will be the consequences of the particular motor input. In order to be able to anticipate, the frontal lobes must monitor external stimuli first by feedback from external stimuli, represented by a downward signal to effector
areas, and simultaneously a feed-forward anticipating signal goes from frontal to posterior region to predict the consequences of a selected motor act. Thus corollary discharge depends on intact frontal lobes and basal ganglia.

This theory is supported by the fact that the frontal lobe has reciprocal connections to the somatosensory zones as well as the limbic system. These frontal limbic connections allow anticipation and foresight. Therefore, in planning behaviour, an individual can examine alternate strategies based on verification of possible affective responses associated with each possible strategy. Once again, the behaviour of individuals with frontal lobe lesions who often have flat affect, socially inappropriate behaviour and difficulty in foreseeing action outcomes was seen as being supportive of this model (Stuss & Benson, 1986). This model introduces the notion of temporal integration of stimuli as well as the more complex functions of working memory in providing retrospective and prospective memory. As with Pribram’s contribution, though more sophisticated than other theories that had predated it, Teuber’s model is heavily reliant on one aspect of EF to the exclusion of other relevant inputs.

3.5.3. Luria

Much of Luria’s theory (1980) about executive dysfunction was based on his behavioural observations of the relationship between EF and problem solving. He noted that individuals with frontal lobe damage were unable to analyze problems systematically, and identify important connections and relationships. They typically had no specific plan to solve problems, could not engage in preliminary investigations of the nature and constraints of the problem and evidenced impulsive actions. His model put forward an impairment in self regulation, resulting in EF impairments involving anticipation (unrealistic expectations, failure to appreciate consequences), planning (impulsivity, poor organization), execution (perseveration, difficulty maintaining set) and self monitoring (emotional dyscontrol and poor error recognition).

According to Kagan and Saling (1988), Luria attempted to resolve the conflict between the narrow mechanistic view of cerebral localization and a more holistic view, much as Fuster did later. In this regard, Luria’s work was visionary and predicted the complex and split second timing of the integration of various cortical areas in the process of dealing with novel activity and to which we have only truly had access with the advent of sophisticated neuroimaging technology. Luria (1973) conceptualized higher mental functions as functional systems of
extremely complex composition, requiring the cooperation of many different parts of the brain. Luria conceived of the brain as comprising functional units: a primitive unit, Unit I, which regulates arousal or state of consciousness, Unit II a posterior region housed by the occipital, temporal and parietal lobes and finally Unit III, seated in the frontal lobes. Unit III was viewed as the executive of the brain by being involved with control and evaluation of action. Unit III is concerned with output and information therefore flows from the tertiary cortex through secondary and primary cortices to effectors. This makes the tertiary zone the most complex regulatory mechanism of the brain as it assembles information necessary for action plans, the broad framework within which action is realized, and also verifies its effectiveness.

3.5.4 Damasio – Anatomical-Functional Model

Three basic factors underlie Damasio’s model. The first is that the fundamental guiding principle of behaviour is the preservation of equilibrium. The second is that the general functions of the frontal lobes are to judge and regulate ongoing external perception and based on this, plan appropriate responses. Third, basic neuroanatomical organization underlies these functions (Damasio, 1979).

The frontal lobes decide how to analyze sensory information and also how to respond appropriately to accomplish the most beneficial path of action for both immediate and long term advantages. This is achieved through a series of gating mechanisms. First, a lower system gating, which is almost automatic in nature, is performed by hypothalamic nuclei – relevant for the evaluation of pleasure/pain and motivational loading of a particular stimulus based on past experience. Complex information that requires evaluation of both external and internal rules and postulation of goals demands participation of the frontal lobes and this represents a higher gating system involved in learning more than mere inherited past behaviours. Damasio’s theory appears to incorporate elements of cognitive functioning highly reminiscent of working memory models as well as introducing emotive aspects of EF. The same critique as discussed above applies to this model. While the elements of this model are certainly important to EF, the exclusion of other mechanisms and a lack of integration of the multiple components provide an overly simplified account.
3.5.5  **Fuster - Temporal integration of behaviour**

Fuster (1973) directly addressed the problem of unity and diversity in the frontal lobes. On one level, it appears that lesions in specific areas result in particular deficits. However, these same lesions appear to produce more general problems suggesting they might also be part of a superordinate function. Fuster argues that behaviour represents complex temporal gestalts with each part being important not in and of itself but in relation to each other and to the overall goal. Maintenance of this temporal gestalt and averting interfering stimuli through inhibitory protection are therefore the most characteristic functions of the prefrontal lobe. Fuster (1997) states that this is achieved by two symmetrical functions: a) the prospective function, comprising anticipation, preparation, foresight or set whereby the frontal cortex uses past experience to enable preparation for anticipated events; b) the retrospective function, comprising provisional memory whereby information is held until a goal is attained. The involvement of the frontal lobes is in direct proportion to the establishment of a purpose and need for temporal continuity. Finally the frontal lobes are important in initiation, intention, motivation and vigour by which complex i.e. executive, behaviours are developed. Fuster’s work has been profoundly influential and is incorporated to a large extent in Barkley’s model presented below. However, unlike Barkley, while Fuster presented a more comprehensive view of the characteristics of EF, he did not define their relationship to one another.

3.5.6  **Shallice – Information processing model**

This highly influential and often cited model is best understood as consisting of four components: cognitive units, schemas, contention scheduling and the supervisory attentional system (SAS). Cognitive units are brain functions related to specific anatomical systems such as language or the visual-spatial functions. Schemas are behavioural activities that demand integration of multiple cognitive units for accomplishment even though goal oriented schemas are usually routine, learned, rehearsed and highly specialized programs for controlling such overlearned skills as making breakfast or driving home.

Contention scheduling involves selection of appropriate schemas for combinations of routine behaviours by triggers activated by sensory perception or input of other schemas. Finally the SAS handles non-routine goals and operates under two circumstances. The first is when contention scheduling fails and the second is when there is no known solution as when only weakly activated schemas are evoked (Shallice, 1982).
For routine tasks, contention scheduling may be adequate. As an example, a person can drive home without being truly aware or highly conscious of their surroundings and behaviour. The SAS can in the interim, rest or deal with other information and contention schemas correctly handle the routine behaviour. During this time, capture errors may occur when a strong trigger can activate contention scheduling and lead to an incorrect response as the SAS, which is not monitoring behaviour will not be aware until a later stage. To continue with the example of driving home, a person may find themselves driving to a particular destination but recognizing a familiar turn off may trigger going the wrong way, particularly if they are distracted, fatigued, stressed or preoccupied. The SAS is a frontal function and therefore following frontal damage, contention scheduling often remain effective, resulting in routine overlearned tasks being performed efficiently, but planning, handling novel situations or making changes for new situations are often impaired. In response there are two possible responses to damage. The first is perseveration because strong environmental triggers continue to elicit dominant action schema. The second is distractible behaviour which can occur when triggers are weak or absent and any new input captures contention scheduling procedures at which times, patients may respond randomly (Shallice, 1982). Of all the models described to this point, this one captures areas of EF that are observable in daily functioning. Application of the model to conversational performance can be easily appreciated. If contention scheduling is preserved, often the mechanics of conversation remain intact. Individuals are able to take turns on the basis that turn-taking is a well established, automatic activity when conversing. However, the quality of those turns may often be disrupted when the SAS is impaired when a failure occurs in the ability to assess the unique communicative needs of the current conversational context adequately and respond accordingly.

3.5.7 Stuss and Benson – Behavioural / anatomical approach

Stuss and Benson (1986) put forward a behavioural/anatomical approach in which separate divisions of frontal lobe function are proposed. Each of these frontal systems is intimately linked with other functional systems and damage to distant areas can affect results of any observations or tests used to monitor and identify these systems. This is the first model to propose hierarchical relationships between the varying EF components. The first division is involved in the ability to maintain and organize pieces of information into meaningful sequences and is considered in this model to be largely dependent on the intactness of frontal lobes. Sequencing and the extraction of crucial elements were also considered key to this
division allowing for the production of new, more complex responses from available sequences of data. The second division is concerned with drive, will and motivation. EF were defined as being anticipation, goal selection, preplanning (means-end establishment), monitoring use of feedback. Each represents a separate and testable frontal lobe/executive function. Stuss and Benson’s most critical contribution was their contention that EF and complex attentional systems are integral to one another. In this hierarchical model of cerebral organization they define attention as the foundation of the hierarchy of functional systems. Without the ability to sustain and shift attention and control interference, the other self regulating functions carried by the sequencing and motivation functions can not be effective. In this regard, this model is the most similar to Barkley’s (1997), described below. The organizational division appears to rest on PFC working memory capacity, with a direct impact on planning ability, although this is not specifically the terminology used. Sequencing and extraction have much in common with Barkley’s notion of reconstitution which is described further on. Emotional regulation features strongly and attention is critical to sustaining all. While both the components and organization are similar to Barkley’s, the latter has the advantage of narrowly defined and clearly operationalised concepts, which make hypothesis testing and defense of the theory more manageable.

3.5.8 Barkley – A Hybrid Neuropsychological Model of Executive Functions

Barkley’s model (1997) is unique in that it draws together in a coherent manner six different threads, all highlighted in the models presented above. The first is the central role of attentional systems as the most fundamental governing force in the performance of executive functions as highlighted by Stuss and Benson (1986). In Barkley’s model these systems are subsumed under the term *behavioural inhibition* and comprise the inhibition of a prepotent response, stopping of an ongoing response and interference control. Behavioural inhibition does not cause the executive functions but rather sets the stage as it were for their occurrence, by providing the space or allowing the opportunity for them to occur. In his model, which is presented as Figure 3.4 on the following page, this concept is conveyed by the blunted lines connecting behavioural inhibition to the four executive functions.

The second thread is the incorporation of Fuster’s notion of retrospective and prospective memory and cross temporal organization of behaviour. This concept is termed *working memory* and represents the first of four executive functions. The third thread and second executive
Figure 3.3. Barkley’s model of behavioural inhibition and executive function (1997).
function is *self regulation of affect*. This concept incorporating the regulation of motivation and arousal is important to the work of many of the preceding authors but especially Damasio and Stuss and Benson (1986), who highlighted the importance of motivation as being closely associated with drive, but reflecting a degree of intellectual control i.e. mental control of a basic energizing force (drive). The fourth thread and third executive function in Barkley’s model is *internalization of speech*, which represents planning, problem solving, ability to engage in description and reflection and conform to rule governed behaviour as projected by Luria. The fourth and final executive function and the fifth thread, is reconstitution – as novel a contribution as the EF it represents. Reconstitution is the capacity for the analysis and synthesis of behaviour. It is expressed in fluency and creativity and contains what Barkley calls the syntax of behaviour – or what Stuss and Benson (1986) may have been referring to when they spoke of ‘sequencing.’

Finally, the sixth thread is integrated in the fact that these four executive functions have direct bearing on motor output – its control, syntax and fluency. This is depicted by arrows that lead from behavioural inhibition and each of the four executive functions to *motor control* in the model. This final thread acknowledges the neauroanatomical structures of the PFC, its reciprocal connections and its main role in pursuing goal directed behaviour.

### 3.6 SUMMARY AND CONCLUDING COMMENTS

Original proposals of frontal and ultimately executive function emphasized that posterior portions of the brain specialized in sensory reception and analysis of sensory data, whereas anterior parts of the brain carried out motor executive activities. Current thinking has shifted away from this perspective and instead the frontal lobes are seen as the gathering point for accumulated sensory data where this information is further analyzed and used to initiate a final common pathway for motor responses.

What the preceding theories all have in common is that they recognize the overriding purpose of the frontal lobes as being executive and being involved in the control of novel, non-routine action. Different investigators emphasized various aspects of this control in their theories. Stuss and Benson (1986) acknowledge these contributions in their theory of frontal lobe functioning and perhaps summarize the distinct contributions best in the following way. Frontal lobes perform the supervisory attentional tasks suggested by Shallice, planning and design
formulation proposed by Luria, the establishment of goals postulated by Damasio, and EF (working memory roles) of Fuster. Decades worth of work continues to be verified on neuroanatomical levels in current information theory using fMRI research. Koechlin and Summerfield (2007) for example have shown that the executive system lying along the anterior-posterior axis of the lateral PFC is hierarchically ordered, with control signals relative to events which occurred in the more and more distant past arising from successively more anterior cortical regions.

Barkley’s unique contribution and the reason that his model was chosen as the framework for this research, is that it is the most comprehensive and inclusive model to date. The fact that it is by name and is in nature a hybrid, integrating the best of what others have offered, makes it the most complete and accessible model of executive function. The fact that these perspectives continue to be borne out in current neuroimaging and neuropsychological research is testament to the complexity and sophistication of the prefrontal cortex and its participation in supporting EF.

In conclusion, although not the seat of intelligence the prefrontal lobes are essential for the highest of mental activities; those demanding the control of intelligence. In the role of ‘I’ or consciousness of self, prefrontal structures bridge the gap between complex multi-integrated response mechanisms that make up the brain and a free willed, independently thinking entity - the mind.

This concludes the chapter on executive functions, their prospective contribution to conversational intelligence and their underlying neuroanatomy and neurochemistry. The following chapter presents a discussion on executive dysfunction in neurogenic communication disorders as well as highlighting gaps in the literature related to the assessment of executive dysfunction in the stroke population and proposed pharmacological treatment for these deficits.
Although EF have been researched in a wide range of disciplines, this discussion will be limited to those that have been researched and identified in the presence of more commonly investigated neurogenic communication disorders. Table 4.1 on the following page provides a summary of the neurological disorders associated with executive dysfunction. In relation to Traumatic Brain Injury (TBI) and dementia, there is a long history of EF research. This is probably attributable to the fact that EF and cognitive deficit or decline, are the predominant features of both conditions. The neuropathology associated with TBI and cortical dementia, involving the frontal, especially the prefrontal cortex, and subcortical connections make these clinical populations a likely focus of research endeavour as EF rely primarily on these anatomical areas, as described in the previous chapter. The following discussion summarises research detailing EF characteristics in the following clinical conditions: vascular disease (stroke), TBI, dementia, as well as brief sections on multiple sclerosis, HIV/AIDS, tumour and psychosis. Throughout, the overlap of symptoms in various conditions and similarities to EF deficit in stroke is noticeable. This chapter highlights gaps in the literature with reference to stroke as well as some of the difficulties entailed in embarking on research of this nature. Finally, this chapter concludes with a review of documented pharmacological treatments for EF highlighting the drug used in this particular study.

4.1 EXECUTIVE DYSFUNCTION AND VASCULAR DISEASE

Cerebrovascular disease can take many forms only one of which is stroke. Others are Binswanger’s disease (a form of vascular dementia), with primary damage to the subcortical white matter. Clinical features include slowly progressive impairments of attention, memory, visuo-spatial skills, cognition, emotional competence as well as variable combinations of paresis, spasticity, hyperreflexia, extensor plantar responses, incontinence and gait disorder (Caplan, 1995). Another variant of vascular dementia is lacunar state resulting from small infarcts from occlusion of penetrating arterioles that have been narrowed by atherosclerosis. Clinically, lacunar state presents similarly to Binswanger’s disease with disproportionate executive dysfunction (Caplan, 1995). Changes in white matter known as leukoaraiosis appear
as hypodensities on MRI, in older people. Some evidence suggests that the origin of these findings is vascular in nature and may interfere with executive function (Boone, et al. 1992 in Filley, 2000).

Table 4.1 – Neurological disorders associated with executive dysfunction (Filley, 2000).

<table>
<thead>
<tr>
<th>Vascular disease</th>
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<tbody>
<tr>
<td>Binswanger’s disease</td>
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<tr>
<td>Lacunar state</td>
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<tr>
<td>Leukoaraiosis</td>
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<td>Stroke</td>
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<tr>
<th>Traumatic Brain Injury</th>
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<tr>
<td>Penetrating</td>
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<td>Non-penetrating</td>
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<thead>
<tr>
<th>Degenerative dementia</th>
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<tr>
<td>Frontotemporal dementia (Pick’s disease)</td>
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<td>Alzheimer’s disease</td>
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<td>Subcortical dementia</td>
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<td>Parkinson’s disease</td>
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<td>Huntington’s disease</td>
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<td>Progressive supranuclear palsy</td>
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<th>Multiple sclerosis</th>
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<th>Inflammatory disease</th>
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<td>Infectious</td>
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<td>AIDS</td>
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<td>Neurosyphilis</td>
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<td>Brain abscess</td>
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<td>Fungal infections</td>
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<tr>
<td>Progressive multifocal leukoencephalopathy</td>
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<td>Herpes simplex encephalopathy</td>
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<td>Lyme disease</td>
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<td>Creutzfeldt-Jakob disease</td>
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<th>Noninfectious</th>
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<tr>
<td>Systemic lupus erythematosus</td>
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<td>Polyarteritis nodosa</td>
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<td>Giant cell arteritis</td>
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<td>Behçet’s disease</td>
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<td>Sjögren’s disease</td>
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<td>Isolated angitis of the central nervous system</td>
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<th>Tumours</th>
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<th>Psychiatric diseases</th>
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<td>Schizophrenia</td>
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<td>Depression</td>
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<td>Frontal lobe psychosurgery</td>
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For a long time, the stroke population did not feature in EF research, perhaps because of the fact that it was considered unlikely to find EF deficits in brains with focal damage. Investigators expected to find EF deficits in bihemispheric lesions or disease processes instead (Filley, 2000). Yet as will become clear throughout the review, several cognitive deficits arising from stroke, have more in common with other neurogenic communication disorders in terms of disruption to executive characteristics than was previously anticipated. As a result of the impact of these deficits manifesting so clearly in the communicative domain, speech language pathologists have become motivated to explore them in more depth. In terms of EF disorders in stroke, the discussion will be divided into deficits in individuals with aphasia and those with Right Hemisphere Disorders (RHD).

4.1.1 Executive dysfunction and aphasia

It is hypothesized that language deficits following PFC damage are a consequence of primary cognitive deficits involving, along with action planning, the cognitive processes of memory and attention (Ferstl, Guthke & van Cramon, 1999). However the PFC also plays an important role in semantic processing and selection (Frattali & Grafman, 2005). Specific language and discourse difficulties have been associated with PFC damage in the past. Dynamic aphasia characterized by a reduction in spontaneous speech with a lack of initiation, limitations in the amount and range of narrative expression and the loss of verbal fluency, often arises from lesions of the dorsolateral areas (Frattali & Grafman, 2005). Akinetic mutism which results in failure to communicate by word or gesture, attributable to the absence of the will or drive to communicate can result from damage to the ascending dopaminergic systems originating in the ventral tegmental area and terminating in the supplementary motor area and anterior cingulate (Alexander, 2002). Discourse disturbances in patients with PFC damage can result and include reduced or verbose speech output, poor structure, failure to stay on topic, tangentiality, lack of cohesion and difficulties with temporal sequencing (Ferstl et al., 1999). Furthermore, story comprehension in terms of inferring the main point is adversely affected due to a proposed breakdown of semantic networks that link propositional information into a connected memory structure (Frattali & Grafman, 2005). Processing ambiguity is also hindered in the presence of PFC damage due to a failure to suppress context-inappropriate meanings (Frattali, Wesley & Grafman, 2001).
A substantial and continuously expanding body of research has been established for attention, memory and working memory impairments in individuals with post-stroke aphasia. There is however a scarcity of research related to other EF. Using Barkley’s model to organize the areas of EF, what follows is a summary of existing findings related to attention, working memory, regulation of affect, planning and reconstitution (flexibility) deficits.

4.1.1.1 Attention deficits in aphasia

Deficits of attention or control thereof have been proposed as all or part of the underlying mechanisms responsible for the linguistic impairments that characterize aphasia (McNeil, Matthews, Hula, Doyle, Rubinsky & Fossett, 2005). Laures, Odell and Coe (2003) found that physiologic arousal as indexed by cardiovascular and neuroendocrine measures and vigilance performance, showed that people with aphasia have decreased overall vigilance and non-optimal levels of arousal regardless of whether the stimuli were linguistic or non-linguistic in nature.

Left hemisphere lesioned patients with aphasia have been found to be worse than normals on measures of sustained attention (Glosser & Goodglass, 1990). Accuracy during a semantic judgment task under conditions of focused attention, demonstrated significant decrements in accuracy of performance for individuals with aphasia (Murray, Holland & Beeson, 1997a). The detrimental effects of competing stimuli on other listening (tone discrimination) and speaking (phrase completion) tasks under conditions of divided attention have also been demonstrated (Murray, Holland & Beeson, 1997b; Murray, 2000).

Another area that is becoming popular in research paradigms related to attention and aphasia are dual task conditions during which individuals with aphasia are required to manage or attend to two or more tasks simultaneously. Deficits are often interpreted in terms of deficient resource allocation, or deficient strategies for allocating attention i.e. sharing and distributing attention efficiently between the two tasks as well as deficient availability of attentional capacity to manage the two tasks. Performance decrements have been convincingly demonstrated with individuals with aphasia demonstrating slowed reaction times, accuracy decline or both (Erickson, Goldfinger & LaPointe, 1996; King & Hux, 1996; Murray, Holland & Beeson, 1998; Tseng, McNeil & Milenkovic, 1993). Many of these authors interpret these deficits as being representative of inefficiencies in the allocation of attention (McNeil, Odell & Tseng, 1991;
Murray, 1999). However, others like Korda and Douglas (1997) demonstrated impaired attentional capacity as shown by slower processing speed and greater increases in response time with increased processing load. Whether the deficit lies in diminished availability of attentional resources, impaired ability to allocate resources or a combination of both remains widely debated.

Tabor-Connor, Albert, Helm-Estabrooks and Obler (2000) and Helm-Estabrooks, Tabor-Connor and Albert (2000) have highlighted the significance of attentional processing in auditory comprehension. They maintain that effects such as substantial item by item variability on repeated tests of auditory comprehension and enhanced performance as a result of slowing rate of input, providing emphatic stress or providing an alerting signal, can be attributed to mechanisms related to attention. Helm-Estabrooks et al, (2000) demonstrated that participants enrolled in the Attention Training Program, which targeted improved sustained, selective and shifting attention performed better on tests of auditory comprehension. Wiener, Connor and Obler (2004) specifically identified impairment in inhibition at the lexical-semantic level of language processing in Wernicke's aphasia, reflecting the inability to effectively ignore automatically evoked, distracting stimuli. Their findings suggest that at least part of the attentional difficulties contributing to the striking reductions in auditory comprehension in this population can be attributed to impaired inhibition.

The impact of attention on reading has also been demonstrated. Coelho (2005) presented a case study of an individual with mild aphasia whose ability to read longer, more complex materials was enhanced concurrently with improvements on measures of sustained attention, her ability to cope with distractions and improved concentration.

Other researchers have looked at the link between attention and expressive language. Villiard (1990) and Nespoulous and Dordain (1990) attributed agrammatism to underlying attention and working memory deficits.

A different type of attention deficit can be seen in the presence of neglect, which refers to the difficulty orienting, attending or responding to stimuli presented on one side of the body. It is a symptom most commonly seen with right hemisphere damage, but is not uncommon in left hemisphere damage as well (Murray, 2002).
4.1.1.2 Working memory deficits in aphasia

Recent investigations have suggested that adults with aphasia present with a working memory deficit that may contribute to their language-processing difficulties. Most of the existing working memory research in the aphasic population draws links between working memory deficit and comprehension impairment. Working memory capacity has been conceptualised alternately as a single "resource" pool for attentional, linguistic, and other executive processing or as a system of separate working memory abilities for different types of linguistic information. By using n-back tasks, Wright, Downey, Gravier, Love and Shapiro (2007) measured working memory ability in adults with aphasia for processing specific types of linguistic information, and examined whether a relationship exists between participants' performance on working memory and auditory comprehension measures. They found that on the whole participants’ performance declined as n-back task difficulty increased. Overall they performed better on semantic level than phonological or syntactic level tasks. Notably, participants who performed poorly on the SynBack (syntactic level task) also had more difficulty comprehending syntactically complex sentence structures (i.e. passive & object-relative sentences) suggesting that there are separate working memory abilities for different types of linguistic information and that these deficits can be measured in adults with aphasia and may contribute to comprehension impairment. This research echoes the findings of previous research by Friedmann and Gvion (2003) who proposed that the type of reactivation required by a sentence, as well as the type of memory overload, is crucial in determining the effect of working memory limitation on sentence comprehension in aphasia.

Consistent with the above findings, Francis, Clark and Humphreys (2003) presented a case study where a patient with working memory and comprehension impairments was treated with a therapy programme targeting memory deficit with the anticipation that comprehension would also improve despite not being directly targeted. There was measurable improvement on tasks of repetition suggesting improved working and short term memory but generalisation to comprehension tasks, although noted, was limited.

The collaborative role working memory plays with attention forms the basis for the resource reduction theory. The assumption made by proponents of this theory is that people with aphasia still possess structural and procedural knowledge necessary to perform syntactic analysis but suffer from reductions in working memory capacity for language, which can lead to patterns of
comprehension breakdown (Just, 1995). Reading performance of individuals with aphasia is seen as evidence. Individuals with aphasia slow down their reading precisely on those parts of a sentence that are computationally most demanding. On-line reading processes reflect momentary demands on resources and indicate that these patients’ difficulties occur when the demand on resources is high relative to the supply (Just, 1995; Just & Carpenter, 1992). In a later study, the same results were obtained by Caspari, Parkinson, LaPointe and Katz (1998) who also investigated the relationship between working memory capacity and reading comprehension in aphasia. Their results support the notion that the ability of aphasic individuals to comprehend language is predictable from their working memory capacities.

4.1.1.3 Regulation of affect deficits in aphasia

Depression and anxiety are recognised psychological consequences of stroke, either as a primary symptom related to brain damage or as a secondary symptom in reaction to the effects of living with disability after a stroke and the host of psychosocial adjustments these disabilities entail (Robinson, 2006). In addition, other disorders such as mania (and bipolar disorder), psychosis, anosognosia (denial of illness), catastrophic reaction, apathy, irritability and aggression, pathological laughing or crying (emotional lability) and disturbances of prosody have all been reported post stroke (Robinson, 2006). In terms of the prevalence of co-morbidity with aphasia, the data is complex and difficult to interpret. This is because of differing methods of diagnosis, exclusion criteria in prevalence studies, the setting in which patients were examined and diagnosed and the cutoff scores on rating scales used for diagnosis (Robinson, 2006).

With regards depression, there appears to be significantly greater frequency of major depression occurring in patients with left anterior hemisphere injuries than with any other lesion location and therefore a high rate of comorbidity with aphasia (Robinson, 2006). Post-stroke depression is associated with increased mortality rate as well as poorer rehabilitation outcomes (Linden, Blomstrand & Skoog, 2007). Significantly, in a recent study by Robinson (2006) more than half of the patients examined with generalized anxiety disorder (GAD) had associated major depression. This suggests that GAD is also frequently associated with aphasia which is further borne out as the clinical correlates of anxiety disorder include impairment in activities of daily living and social functioning, which would include deficits in communication (Robinson, 2006).
Santos, Caeiro, Ferro, Albuquerque & Figueira (2006) also found that anger was frequent in acute stroke patients, probably triggered by the brain lesion itself, which interferes with emotional control. However, regulation of affect within the context of EF has not been well researched. With the exception of one important paper, there has been virtually no investigation into disorders of drive and motivation in the stroke population.

In a highly relevant study Piamarta, Iurlaro, Isella, Atzeni, Grimaldi, et al., (2004) assessed the presence and severity of unconventional affective symptoms (apathy, anhedonia and emotional lability) and depression in 33 elderly patients with first ever stroke and evidence of a single supratentorial lesion at neuro-imaging. Patients were submitted to neurological, functional, and affective assessment at a mean interval of 2 weeks after stroke onset. The prevalence of the various affective symptoms was as follows: apathy 15.2%, anhedonia 6.1%, emotional lability 48.5% and depression 57.6%. Apathy and anhedonia showed significant reciprocal correlations and they were also correlated with the executive score and the Barthel index; apathy was also correlated with depression; emotional lability, instead, was correlated only with depression. The study of possible anatomo-functional correlates between unconventional affective symptoms and lesion site did not show significant differences (stroke in the right versus left hemisphere, anterior versus posterior and cortical versus subcortical locations). This study is significant as it links affective disorders specifically apathy and depression to executive deficit, both of which have pervasive impact on recovery and rehabilitation.

4.1.1.4 Problem solving deficits in aphasia

Of all the areas of EF, the presence of problem solving and planning difficulties in aphasia is perhaps the most philosophical and elusive. Kinsbourne, (2000a) states that in adults, most language use is actually internal – mediating the stream of consciousness i.e. we think in words virtually all the time. When trying to address the functions of inner speech the primary question in this context is, “how does disabled inner speech harm cognition?” Contemporary opinion disengages thought (intention) from its verbal realisation. If language coaxes thoughts from the global to the specific then does the absence or disability of language blunt that specificity? Kinsbourne (2000b) asks whether inner speech is abridged in people with aphasia and does that affect how they think? Language, being a symbolic representation system allows us not just to represent concepts but also facilitates our ability to manipulate those concepts.
For many years, speech language pathologists have been at the forefront of advocacy on behalf of our clients with aphasia by insisting that despite compromised language, our clients are essentially intellectually intact and should be treated as such. However, we also need to recognize that the ability to internalize language in the form of inner speech allows us to solve problems covertly – when language is interrupted or disrupted, are we left with less sophisticated, less flexible capacity for analyzing and solving complex problems (Baldo, Dronkers, Wilkins, Ludy, Raskin & Kim, 2005)? In a nutshell, studies that include participants with aphasia must on some level address the questions of the importance of internal verbalization to executive functioning. Clearly these questions represent sophisticated and complex issues that are subtle and to some extent obscure and thus difficult to investigate. On the other hand, the implications are practical and far reaching and speak to ethical issues such as decision making capacity and legal guardianship or independence.

Performance on the Wisconsin Card Sorting Test (WCST) as well as Raven’s Coloured Progressive Matrices has correlated consistently with comprehension and naming. As a result of the fact that the WCST is a nonverbal test of problem solving, this correlation suggests that language impairment can manifest as impaired cognition even when overt verbalization is not required. It has been suggested that covert language processes i.e. “inner speech” may be utilized to support complex problem solving (normals performed worse under conditions of articulatory suppression during their performance of the WCST). More severe language impairments have also been correlated with poor problem solving. Covert verbalization also overlaps with the concept of verbal working memory which has been suggested to support problem solving (Baddeley, 2000, p. 420) in that “people recode materials verbally so as to take advantage of the capacity of the phonological loop for storing serial order”. The repetition subscale on the Western Aphasia Battery (Kertesz, 1982) correlated significantly with perseveration on the WCST so the possibility exists that verbal working memory plays a part in aspects of performance on WCST.

4.1.1.5 Flexibility deficits in aphasia
Cognitive flexibility is often impaired in people with neurogenic communication disorders (Rende, 2000). In individuals with aphasia, reduced flexibility is seen in the presence of perseveration (Glosser & Goodglass, 1990). In addition, researchers are also suggesting that reduced flexibility is the reason that individuals with aphasia experience communication
breakdown when they are unable to restructure the way they think or have difficulty switching cognitive sets in reaction to topic changes or initiating topic shifts (Rende, 2000). Reduced performance on verbal fluency tasks also suggests impaired cognitive flexibility in this population (Varley, 1995).

4.1.1.6 Summary of executive function deficits in aphasia

Attention and working memory deficits in the aphasic population are becoming more frequently documented. The nature of these deficits is also being examined in more specific detail. With reference to Barkley’s model, all areas of EF as outlined have been addressed in this review and although some areas such as planning are not well researched, there is evidence to suggest that individuals with aphasia may suffer from EF deficits in all of the areas characterized as executive. This observation is significant in that such deficits have been linked to poorer rehabilitation outcomes and increased rates of mortality. This points all the more to the need for speech language pathologists to familiarize themselves with EF and begin to address the task of measurement and treatment as part of our communication therapies.

4.1.2 Executive dysfunction and Right Hemisphere Disorder (RHD)

Damage to the right hemisphere is caused by the same mechanisms that cause damage to the left hemisphere: stroke, tumours, head injury or disease processes. However, unlike the left hemisphere, there is no obvious localization of function. One reason is that the right hemisphere has more white and less grey matter than the left side of the brain suggesting more intra-hemispheric connections within the right hemisphere. Thus specific abilities are attributed to processing by large networks instead of relatively restricted areas dedicated to particular functions. As an example, attention processes, which are frequently impaired following right hemisphere damage, are thought to arise from large networks that involve cortical as well as subcortical areas (Lehman-Blake, 2005). RHD deficits are often difficult to diagnose as there are no clear boundaries between normal and abnormal behaviour for many of the pragmatic and cognitive abilities affected by damage to the right hemisphere (Lehman-Blake, 2005). Often these deficits may only emerge after in-depth conversational exchanges and social interaction.

There are myriad deficits associated with RHD including sequencing, problem solving, reasoning, attention and perception, hypo- or hyperresponsiveness and tangentiality (Lehman-Blake, 2005). Deficits in one area impact deficits in another and in some cases may be difficult
to separate. There are also a range of communicative impairments including impaired prosody, metaphoric comprehension, jokes, contextual and emotional adequacy of discourse (Martins, Caeiro & Ferro, 2007). However, how these pragmatic difficulties are specifically linked to executive dysfunction remains poorly researched.

4.1.2.1 Attention deficits in RHD

Just like attention difficulties identified in aphasia, disorders of attention in RHD may be due to reduced attentional capacity or problems with resource allocation. Unilateral spatial inattention or neglect is the most frequent and typical cognitive dysfunction in right hemisphere lesions and may result from different sites of lesion including prefrontal anterior cingulate, parietal and subcortical (Martins et al., 2007). According to Tompkins, Bloise, Timko and Baumgaertner (1994) neglect can affect other language abilities and regardless of whether or not it is resolved, an initial diagnosis of neglect appears to predict greater difficulty with discourse-level language tasks. This relationship is proposed due to the characterisation of neglect as an attention disorder which then impacts on other communicative abilities reliant on attention processes. Significantly, much of the current literature appears to have inferred an attention deficit based on observations of communication and behaviour rather than as a product of actual neuropsychological testing of attention functions per se, a fact which detracts from the strength of the relationships proposed. Attentional problems have also been thought to explain the tangential behaviour often seen in adults with RHD and this is evidenced through an inability to remain focused on a single topic and becoming distracted by internal stimuli. Saldert and Ahlsén (2007) found that poor ability to make inferences was associated with poor sustained attention in RHD. Furthermore, attention difficulties have been proposed as one explanation for why adults with RHD have difficulties with cognitively demanding tasks but not on relatively easier ones (Tompkins, et al., 1994). The presence of perseveration, confabulation and sensitivity to interference are further indications of attention deficit in this population (Ghika-Schmid & Bogousslavsky, 2000).

4.1.2.2 Working memory deficits in RHD

There is a surprisingly wide range of working memory deficits reported in individuals with RHD including poor verbal memory retrieval, deficits of learning and recall of verbal and visuo-spatial material (Davis, 2000). However, relatively few studies have actually researched these deficits systematically and like attention difficulties in this population, deficiencies are
often inferred from behaviour in clinical settings rather than based on specific neuropsychological profiling. However there are exceptions. In a study by Lazar, Festa, Geller, Romano and Marshall (2004), working memory deficit secondary to attention problems in RHD was proposed to account for failure to multitask. Impaired working memory has also been found in patients with spatial neglect due to right hemisphere pathology. These patients may show 'revisiting' behaviour during visual search and cancellation tasks, such that previously encountered targets are treated as if they are new discoveries. Revisiting behaviour is particularly evident when no visible trace is left to inform patients that a particular target has already been detected, implying that spatial working memory may be impaired in neglect (Pisella, Berberovic & Mattingley, 2004).

4.1.2.3 Regulation of affect deficits in RHD

Much of the research related to disturbances of affect, focuses on the interpretation of emotion and ability to infer mood while there is less that centres on emotional regulation. It is well documented that individuals with RHD show reduced appreciation of emotional material (in speech and gesture) and altered expression of emotion (Brownell & Martino, 1998; Gardner, 1994; Martins et al., 2007). Right MCA superficial lesions can induce delirium. Agitation, hallucinations and autonomic hyperactivity have also been reported.

Of the disorders that affect emotional regulation and expression, apathy or disorders of motivation are among the most frequent (Martins et al., 2007). There is also a highly significant association between secondary mania and right hemisphere lesions following stroke (Robinson, 2006). While the co-occurrence of GAD and depression is more frequent after left hemisphere lesion and frequently present with aphasia, GAD is commonly associated with right posterior stroke as well (Robinson, 1998). Finally emotional incontinence, particularly crying, is most predominant after a right-side lesion (Kim 2002).

4.1.2.4 Problem solving deficits in RHD

Specific planning deficits such as dressing apraxia and constructional apraxia have been identified in this population (Hier, Mondlock & Caplan, 1983; Martins et al., 2007). Cognitive deficits associated with RHD like deficient organising, sequencing, reasoning and problem solving are reportedly impaired in as many as half of the adults with RHD (Blake et al., 2002). Yet, they are not well explained, partly as mentioned previously because of the wide range of
cognitive abilities in the general population but also because of the recurring difficulty of such executive functions being imprecisely defined, with closely intertwined concepts that are difficult to isolate. These disruptions are however thought to account for some of the communication deficits manifested as disorganised macrostructure and impaired repair of conversational breakdown (Lehman-Blake, 2005).

4.1.2.5 Flexibility deficits in RHD
As with research pertaining to aphasia, information about flexibility deficits arise primarily from verbal fluency tasks with other difficulties arising secondary to attention problems. Divergent word fluency tasks indicate that individuals with RHD have more difficulty with semantic fluency than with first-letter fluency (Joanette & Goulet, 1986) and generate fewer words than controls (Grossman, 1981). Reduced behavioural flexibility has been seen in continuous perseveration as well as difficulty shifting perspective in this population (Varley, 1995).

4.1.2.6 Summary of executive function deficits in RHD
Literature regarding cognitive and communicative deficits in RHD is often descriptive. Lists of symptoms are presented with a wide range of deficits in behavioural and cognitive abilities implicated in damage to the right hemisphere. However, as mentioned, many of these deficits appear to be inferred and there is limited research which specifically and systematically investigates the presence of EF deficits in this population. Given the range of pragmatic and communication difficulties experienced by this population, thorough profiling of EF would be highly beneficial.

4.2 EXECUTIVE DYSFUNCTION IN TRAUMATIC BRAIN INJURY
Executive dysfunction is perhaps the most frequently recognized and most disabling outcome of traumatic brain injury (TBI) (Adamovich & Henderson, 1992) providing a major barrier to community integration (Gordon, Cantor, Ashman & Brown, 2006). From the reviews above, it appears that there is more in common between stroke related communication difficulties and those disruptions caused by EF deficit in TBI than previously thought and may therefore have similar impact on community integration and communication skill. These impairments have been linked to deficits of the central executive system of working memory (Serino, Ciaramelli, Di Santantonio, Malagu, Servadei & Ladavas, 2006) as well as deficits in self-generative
behaviour and cognitive flexibility/set shifting. Reduced inhibition has also been consistently identified (Busch, McBride, Curtiss & Vanderploeg, 2005). A disruption of problem-solving abilities and difficulties regulating emotion are commonly cited (Gordon et al., 2006).

4.2.1 Attention deficits in TBI

Inhibition deficits in TBI have been documented in relation to a wide number of research tasks and clinical observations including higher incidence of interruptions during conversations (Linscott, Knight & Godfrey, 1996), verbal expansiveness (Prigatano & Fordyce, 1986), poor turn taking skills as evidenced by intrusions (Coelho, Liles & Duffy, 1991; Gillis, 1996; Schapiro & Sacchetti, 1993). Perseveration on the Wisconsin Card Sorting Test (Damasio, 1994) and poor performance on Go/No Go tasks are also seen as signs of poor behavioural inhibition. Participation in Go/No Go tasks have further been reflected in event-related brain potentials and EEG spectral power data (Roche, Dockree, Garavan, Foxe, Robertson & O’Mara, 2004).

Patients with TBI further demonstrate poor interference control by being distractible, where attention is attracted by irrelevant sensory stimuli. Therefore, patients often have poor sustained attention to internal representations and behavioural or mental tasks and many complain of having difficulty concentrating (Stuss & Benson, 1986; Wilkins, Shallice & McCarthy, 1987). Similar to attention difficulties described in individuals with RHD and more lately in some cases in individuals with aphasia, these individuals fatigue easily, have impaired selective attention and scanning, and poor shifting of attention back and forth (Hartley, 1995; Prigatano & Fordyce, 1986). The fact that even individuals with mild head injuries complain of difficulty following a conversation, is not surprising given the multitude of co-occurring mechanisms and processes required for successful interaction (Gillis, 1996; Schapiro & Sacchetti, 1993). These attention deficits manifest in social interactions particularly during group communication (Godfrey & Shum, 2000) and are by and large an unrecognized difficulty for people with stroke related communication difficulties as well. This may also account for difficulty staying on topic and a tendency towards confabulation (Gajar, Schloss, Schloss & Thompson, 1984).

Distractibility also accounts for the fact that individuals with TBI make tangential or irrelevant comments, demonstrating conceptual confusion reflected in loose connections between thoughts
and ideas, poor word selection and a strong tendency to stray from the core message (Linscott et al., 1996; Prigatano & Fordyce, 1986). Speed of information processing is often affected in brain-injured patients (Gillis, 1996). This refers to difficulties registering incoming information and rapid cognitive processing of material as well as rapid output (Schapiro & Sacchetti, 1993). These deficits have all been identified in individuals with both left and right hemisphere lesions resulting from stroke, although there appears to be a perception that such attention difficulties arise mainly from diffuse or widespread damage.

4.2.2 Working memory deficits in TBI

In terms of working memory deficit, researchers have initiated investigations that assess the different sub-components of working memory in order to characterize the essential nature of the disability. Vallat-Azouvi, Weber, Legrand and Azouvi (2007) found marginal group differences with regard to the functioning of the two slave systems (the phonological loop and the visuo-spatial sketchpad) in controls, whereas patients with severe TBI performed significantly poorer on most central executive tasks, particularly on those requiring a high level of controlled processing implicating an impairment of executive aspects of working memory in severe TBI.

In an earlier study Perlstein, Cole, Demery, Seignourel, Dixit et al., (2004) examined working memory function in predominantly chronic patients with mild, moderate and severe TBI and healthy comparison subjects behaviourally and, in a small subset of moderate-to-severe TBI patients, with event-related functional magnetic resonance imaging (fMRI), using a visual n-back task that parametrically varied working memory load. TBI patients showed severity-dependent and load-related working memory deficits in performance accuracy, but not reaction time. fMRI results showed that TBI patients exhibited altered patterns of activation in a number of working memory related brain regions, including the dorsolateral prefrontal cortex and Broca's area. Examination of the pattern of behavioral responding and the temporal course of activations suggests that working memory deficits in moderate-to-severe TBI are due to associative or strategic aspects of working memory and not impairments in active maintenance of stimulus representations. Their results overall suggested that individuals with moderate-to-severe TBI exhibit working memory deficits that are associated with dysfunction within a distributed network of brain regions that support verbally mediated working memory.
A recent and novel study by Piolino, Desgranges, Manning, North, Jokic and Eustache (2007) examined disorders of autobiographical memory. Deficits involved disturbances in sense of remembering, visual imagery, self-perspective and recollection of spatiotemporal details. Stepwise-regression analyses carried out in the TBI patients revealed a significant relationship between an abnormal sense of remembering and executive dysfunction covering both anterograde and retrograde components (critical aspects of working memory). The results suggest that these patients, long after trauma, present autonoetic consciousness and self-perspective disorders, which include sense of identity (the self) as a continuous entity across time, probably related to frontal dysfunction.

4.2.3 Regulation of affect deficits in TBI
Disturbances of affect and regulation of emotion are also well established outcomes of TBI. In their 2004 study Jorge, Robinson, Moser, Tateno, Crespo-Facorro and Arndt, found that individuals with major depression exhibited comorbid anxiety (76.7%) and aggressive behavior (56.7%) and also had significantly greater impairment in executive functions than their non-depressed counterparts. Major depression was also associated with poorer social functioning at the 6-and 12-month follow-up, as well as significantly reduced left prefrontal gray matter volumes, particularly in the ventrolateral and dorsolateral regions. Apathy is also specifically related to executive deficits and this reduction in goal-directed behavior due to lack of motivation constitutes a major neuropsychiatric symptom following TBI (Andersson and Bergedalen, 2002). However based on the reviews above, perhaps it would be more accurate to say that these disorders of motivation and drive are major symptoms following frontal lobe damage – even in the presence of focal lesions.

4.2.4 Problem solving deficits in TBI
The 'dysexecutive syndrome' that occurs following TBI is often associated with difficulties of problem solving (including specific impairments in planning, initiation/plan-implementation and self-monitoring). Research has suggested that one reason people with TBI are poor at everyday planning is that they fail to spontaneously use specific autobiographical memories to support planning in unstructured situations. To this end, Hewitt, Evans and Dritschel (2006) examined whether a self-instructional technique involving self-cueing to recall specific autobiographical experiences would improve performance on a planning task. Two groups of 15 participants who had suffered a closed traumatic brain injury carried out a task in which they
were asked to describe how they would plan eight common unstructured activities, i.e. activities that could be solved in a variety of ways. Group 1 was then asked to describe how to plan a second set of eight unstructured activities. Prior to completing their second set of eight activities, Group 2 underwent training in a procedure aimed at prompting the retrieval of specific memories to support planning. The results suggested that the intervention was effective at increasing the number of specific memories recalled, with a corresponding increase in the effectiveness of the plan and number of relevant steps in the plan.

Patients with TBI also have difficulty developing strategies to organize material to be memorized and therefore display difficulty with rote learning and short-term memory deficits (Gershberg & Shimamura, 1995; Prigatano & Fordyce, 1986). An impairment of abstract attitude is also evident, showing the lack of sophistication related to language use that would allow for the development of meta-thought. Thus, individuals with TBI may interpret verbal messages literally instead of symbolically, thus impairing understanding of the subtle innuendo that often characterizes emotional aspects of conversation (Prigatano & Fordyce, 1986; Schapiro & Sacchetti, 1993). These deficits are characteristically seen in RHD as well.

4.2.5 Flexibility deficits in TBI

Performance on verbal fluency tasks, that demand the accurate and efficient communication of information, is diminished in TBI (Prigatano & Fordyce, 1986). Confrontational story narratives and responses to direct questions or requests for information in both speech and writing are limited in terms of both amount and organization of content. Joint peer communication tasks and other situations or tasks that demand accurate and efficient communication of information, are frequently disrupted (Barkley, 1997).

Breakdown in the processes governing reconstitution is also evident in nonverbal behaviour and in problem solving tasks requiring complex and novel motor sequences or goal-directed creativity. Reduced capacity for interpretive statements such as deriving a moral from a story has also been demonstrated in these patients (Ulatowska, Freedman-Stern, Doyel, Macaluso-Haynes, & North, 1983), indicating a diminished capacity to analyse components and interpret them meaningfully.
Creation of multiple novel complex alternative response sequences, whether in language or motor behaviour such as in gestural or drawing fluency, is often impaired in patients with damage to frontal lobes (Fuster, 1989, 1995; Milner, 1995; Stuss & Benson, 1986). As a result behavioural flexibility is restricted, and perseverative tendencies dominate as behaviours selected and executed come from a smaller pool of possibilities.

Patients with TBI may also manifest greater difficulties with tasks, settings and interpersonal interactions where reconstitution is essential. Social skills in particular require a degree of proficiency in the flexible application of socially governed norms that are context dependent (Argyle & Henderson, 1985).

4.2.6 Summary of executive deficits in TBI

As in the stroke population, deficits across a number of executive skills are well documented in the TBI population, including attention deficits in the areas of both inhibition and interference control, as well as specific sub-types of working memory, regulation of affect, planning and problem solving. Finally reduced flexibility in verbal and nonverbal behaviour is noted. All these behaviours combine to make up what is often called a dysexecutive syndrome in individuals with TBI. Traditionally, RHD secondary to stroke may have been believed to have more in common with the well established sequelae following such diffuse injuries as TBI than aphasia. Yet as the review of EF deficits in aphasia has shown, these initial associations are not as simple or as exclusive as previously perceived. To this end, as we understand more about the similarities between executive dysfunction in stroke and other neurogenic communication disorders, the more we will appreciate the significant challenges faced in community integration and rehabilitation in the stroke population.

4.3 EXECUTIVE DYSFUNCTION IN DEMENTIA

Dementia is diagnosed on the basis of the decline or deterioration in cognitive or thinking capacities in more than one area of cognition but in the presence of unimpaired awareness or alertness (Rabins, Lyketsos & Steele, 1999). Memory is affected by almost every disease that causes dementia. The other impairments – judgment, perception, language, abstraction and calculation – depend on specific diseases and stages of illness. Deficits of EF are well documented particularly in relation to the cortical dementias of which Alzheimer’s disease (AD) is the most familiar example. Executive impairments are common in early AD. Complex
Attentional skills are more frequently affected than other executive functions. There is, however, considerable heterogeneity among AD patients in the pattern of executive dysfunction (Stokholm, Vogel, Gade & Waldemar, 2006). While cortical dementia is often characterized by the inability to perform cognitive functions, subcortical dementias are often characterized by the inability to coordinate such functions. The most striking difference between executive dysfunction in dementia and stroke is that in the former, such difficulties presumably worsen over time while in the stroke population identified deficiencies will remain stable or even improve with recovery and rehabilitation efforts. Certainly it is hoped that in the face of chronic impairment, compensation would be more effectively achieved in individuals with stroke who retain stable neurological status.

Parkinson’s disease (PD) along with Huntington’s disease (HD) and progressive supra-nuclear palsy (PSP) are all types of subcortical dementia that routinely display prominent EF deficits. These are likely to be a manifestation of the strong neuroanatomic connections between frontal lobes and subcortical grey matter structures. Executive dysfunction may be the first sign of cognitive loss. There is significant variation in the manifestation of executive dysfunction in PD as demonstrated in a recent study by Verleden, Vingerhoets and Santens (2007). They performed extensive neuropsychological testing on a sample of 100 consecutive PD patients with motor fluctuations. A component analysis of the major test variables resulted in three components: one concerned with memory/attention, one with visuospatial, and one with executive/motor functions. They found that eighteen percent of the cohort showed no significant impairment on any domain. Fifty-one percent showed impairment in one cognitive domain, most frequently in the executive/motor component (88%). Twenty-four percent performed below normal on two cognitive components, most often executive/motor and memory/attention deficits (96%), and only 7% of the cohort had significant impairment on each derived cognitive component. Deterioration of visuospatial and visuoconstructive skills have also been reported in mild and moderate stages of PD (Stella, Gobbi, Gobbi, Oliani, Tanaka, et al., 2007). In HD executive dysfunction is also an early symptom while in PSP executive dysfunction is prominent in all stages of the illness (Filley, 2000).

In addition to the areas of cognition that are affected by dementia, EF deficits related to attention (especially inhibition), generative processes, planning, deficiencies in the regulation of affect (particularly in the co-occurrence of depression) have also been identified.
4.3.1 Attention deficits in dementia

Typically it is memory impairment that heralds the diagnosis of Alzheimer’s disease (AD) (Cummings & Benson, 1983). However a recent study by Silveri, Reali, Jenner and Puopolo (2007) indicated that it is also the executive (in this study represented by attention) deficits that may not only characterize the onset of dementia but also track AD even at the preclinical stage (Belleville, Chertkow & Gauthier, 2007). Silveri et al., (2007) followed subgroups of patients with different types of mild cognitive impairment who were assessed on various tests of attention and EF. On the basis of scores obtained on the Clinical Dementia Rating at the one year follow-up, patients were redistributed into two groups: those who developed and those who did not develop dementia. Patients who presented evolution to dementia had, at baseline, lower scores than patients who did not evolve on tasks exploring attention and executive functions.

Executive dysfunction is frequently reported with AD as well as the frontal variant of frontotemporal dementia (fv-FTD) (Collette, Amieva, Adam, Hogge, Van der Linder, et al., 2007). Specifically, these authors found similar inhibitory deficits in the two conditions (on the Stroop task) suggesting that inhibitory dysfunction in the two groups of patients depends on a disconnection process between anterior and posterior cerebral areas. Furthermore, there appears to be a large cortical network implicated in executive dysfunction in AD, and this pattern of cognitive-metabolic correlations varies according to disease severity (Bracco, Bessi, Piccini, Mosconi, Pupi & Sorbi, 2007).

4.3.2 Working memory deficits in dementia

Deficits in working memory processes are well documented in AD. In a study by Bayles (2003), the performance data of individuals with mild and moderate Alzheimer's dementia on five tests of language comprehension and four tests of language expression was examined in the context of possible contributions from impaired working memory functions. She argues that diminished scores on tests of language comprehension and production result primarily from attenuated span capacity, difficulty focusing attention, encoding, and activation of long-term knowledge rather than from loss of linguistic knowledge. This perspective is similar to that of Rochon, Waters and Caplan (2000), who also see the working memory impairments in AD as being related to difficulty mapping the meanings of sentences onto depictions of events in the world. On the other hand, some researchers have posited that the working memory deficits seen in AD are secondary to other cognitive deficits among them semantic memory decline.
(Kensinger, Shearer, Locascio, Growdon & Corkin, 2003). This differentiation may also have important repercussions for the language processing and retrieval mechanisms disrupted in aphasia.

4.3.3. Regulation of affect deficits in dementia

In Alzheimer’s disease (AD), depressive symptoms are known to occur even from an early stage of the disease. However, the nature of the impairment of executive functions in depression associated with AD remains unclear, because of the frequent occurrence of the apathy syndrome as a major confounding factor (also central in frontal temporal dementia). In this study Nakaaki, Murata, Sato, Shinagawa, Tatsumi et al., (2007), a comprehensive comparative neuropsychological assessment in AD patients with (n = 21) and without (n = 21) depression was conducted. Their results suggested that depressive symptoms in AD patients further increase the deficits of cognitive flexibility and divided attention.

4.3.4 Planning deficits in dementia

Although not particularly well researched, planning deficits have also been identified in cortical dementias – specifically in AD (Allain et al., 2007). Sixteen participants with AD and thirteen normal elderly controls underwent testing of the "Zoo Map Test" which derived from the Behavioural Assessment of the Dysexecutive Syndrome test battery. The difference between formulation and execution was greater in patients with Alzheimer’s disease than in healthy elderly. Planning impairments mainly correlated with behavioural changes (in particular motivational changes) observed by patients’ relatives. These results suggest that patients with Alzheimer’s disease have some difficulty mentally developing logical strategies and executing complex predetermined plans, which are partially related to behavioural changes.

4.3.5 Flexibility deficits in dementia

Deficits in generative processes which rely on the left orbitofrontal cortex and temporal neocortex to gain access to autobiographical memories, are thought to be responsible for the episodic memory deficiency or autobiographical amnesia experienced by individuals with frontal variant of fronto-temporal dementia (fv-FTD) (Piolino, Chetelat, Matuszewska, Landeau, Mezenge, et al., 2007). In PD, the bradykinesia or slowed voluntary movement are often paralleled by inflexibility in thought manifested in poor performance on the WCST (Filley, 2000).
Notably, the impairment of EF has also been found to be a significant and powerful predictor of future functional decline in individuals with vascular dementia. Boyle, Paul, Moser and Cohen (2004) found that a measure of instrumental activities of daily living (IADLs) had declined significantly over the course of one year and that baseline performance on executive tasks was a significant predictor of IADLs at the one year follow-up, even after accounting for global cognitive dysfunction.

4.4 EXECUTIVE DYSFUNCTION IN MULTIPLE SCLEROSIS
Forty to sixty percent of patients with multiple sclerosis (MS) have cognitive dysfunction. The frequency of cognitive disturbances according to the clinical form is not completely understood and the natural history of these disorders has not been extensively studied. Cognitive deficits can be detected in early stages of the disease. Their frequency increases from clinically isolated syndromes, to relapsing-remitting (RRMS) and secondary progressive MS. Cognitive abnormalities are frequently observed also in primary progressive MS. The most frequently impaired functions are information processing speed, attention and memory. Dementia is uncommon but may disclose the disease. Diffuse cerebral injury, assessed by magnetic resonance imaging, contributes to cognitive dysfunction in MS, probably by interrupting connecting fibers between neuronal networks involved in these cognitive functions. Compensatory mechanisms may occur at early stages but they are limited by extension of brain injury (Brochet, Bonnet, Deloire, Hamel & Salort-Campana, 2007).

4.4.1 Attention and working memory deficits in MS
The attention impairment in subjects with RRMS is thought to be related to slowed central processing, which may be affected in all stages, including impairment of automatic and controlled processing of information and in the motor programme (Balsimelli, Fernanda Mendes, Bertolucci & Tilbery, 2007). This slowing is also reflected in studies assessing working memory capacities by delayed reaction times using n-back tests, which increased with working memory load (Parmenter, Shucard, Benedict & Shucard, 2006).

A study by Nebel, Wiese, Seyfarth, Gizewski, Stude, et al, (2007) investigated cortical activity in focused and divided attention tasks in patients with MS with and without impairments in these specific attention functions compared to controls. They found evidence for the neural
correlate of attentional deficits in MS patients. Their findings also suggested impaired top-down attentional control on sensory functions.

4.4.2 Regulation of affect disorders in MS
In terms of disorders of affect, there is very little research with the exception of depression, which understandably appears to occur as a secondary reaction to compromised quality of life. Beal, Stuifbergen and Brown (2007) published a longitudinal research project in which they examined depression in MS patients over time. Younger age, longer time since diagnosis of MS, progressive forms of MS, and greater extent of functional limitation were predictive of greater depressive symptoms. With the exception of functional limitation, which showed an association with depression at all periods, these variables did not predict changes in depressive symptoms over time. There does not appear to be any research related to disorders of motivation, drive or other executive aspects of emotional regulation in this population although it has been suggested that slowed information processing speed and secondarily, deficient central executive skill, may be core to the cognitive deficits characteristic of depressed MS patients (Arnett, Higginson & Randolph, 2001).

4.4.3 Planning disorders in MS
There is very little research looking at planning ability in individuals with MS. Lazeron, Rombouts, Scheltens, Polman and Barkhof (2004) used fMRI to study planning in moderately impaired individuals on the Tower of London task. They found that performance in MS patients was significantly worse than in controls despite the fact that the pattern and extent of brain activation during planning were maintained. Lazeron et al., (2004) interpreted poor performance as being reflective of exhaustion of adaptive mechanisms.

4.4.4 Flexibility disorders in MS
As far as this researcher is aware, there are no investigations relevant to flexibility and generative abilities in individuals with MS.

4.5 EXECUTIVE DYSFUNCTION IN INFLAMMATORY DISEASES / HIV/AIDS
There are a wide range of inflammatory diseases that have the potential to produce executive dysfunction. Effects are often diffuse and isolated involvement of the DLPFC is rare. Inflammatory diseases can be divided into those that are infectious and those which are not and
these are listed in Table 4.1 above. Although slowed cognition, inattention, personality changes, apathy, disinhibition and intellectual compromise have been described, EF has not received much careful attention amongst these populations. The only exception is the dementia that often complicates AIDS, known as the AIDS dementia complex (ADC) (Filley, 2000), which will be discussed briefly.

ADC is characterized by compromised function in frontal cortical and subcortical brain regions including, impairments in motor control, reaction time, and executive functions (Gray, Wilcox, Zink & Weed, 2006). As was true of MS, existing research suggests that the rate of depressive illness and depressive symptoms are high in people living with HIV/AIDS, but investigations on the causes of depression provide conflicting results. Social, psychological and biological factors have all been suggested as possible causes of depression in people living with HIV/AIDS. The suggestion that depression may be the result of the neurotropic effects of the virus on the central nervous system leading to an 'organic' or secondary depression has major implications in the treatment of HIV/AIDS. In a 2005 study by Judd, Komiti, Chua, Mijch, Hoy et al., 129 people living with HIV/AIDS were recruited from out-patient clinics and primary care settings to complete self-report symptom and personality measures as well as neuropsychological testing and a structured clinical interview. Results suggested that for 'well' people living with HIV/AIDS, there is no distinct subtype of depression and early treatment approaches can be modeled on those used for other non-HIV groups. However the effect and impact of the other multiple factors underlying depression in HIV/AIDS is not understood and requires further longitudinal exploration. Previous research has linked apathy to depression but not to neuropsychological impairment (in terms of attention, concentration, learning, memory, executive function, and psychomotor speed) in 75 men and 58 women with HIV (Rabkin, Ferrando, van Gorp, Rieppi, McElhiney, & Sewell, 2000).

4.6 EXECUTIVE DYSFUNCTION IN TUMOURS
Neoplasms may involve the frontal lobes and cause a variety of neurobehavioural deficits. Depending on the degree of malignancy, tumours may cause a rapid or slow and insidious change in behaviour over a long period of time. Neurological features are due to the effects of an expanding mass either directly invading or compressing the brain in a non-specific manner (Filley, 2000). Executive dysfunction may therefore occur along with headache, seizures, disinhibition or apathy among other classical frontal features. There is some evidence to suggest
that even after a tumour has been excised, patients may still present with deficits of an executive nature (Halbig, von Cramon, Schmid, Gall & Friederici, 2002). The incidence of major depressive disorder has also been found to be triple that found in adult ambulatory cancer patients previously evaluated with DSM criteria. Key predictors included frontal region of tumour location, combined sadness and lack of motivation symptoms and family psychiatric history (Wellisch, Kaleita, Freeman, Cloughesy, & Goldman, 2002).

4.7 EXECUTIVE DYSFUNCTION IN PSYCHOSIS
Perhaps the most well researched psychiatric disorder in relation to EF is schizophrenia. Negative symptoms include inattention, cognitive slowing, stimulus-bound behaviour and perseveration. Poor performance on the Wisconsin Card Sorting Test in combination with reduced cerebral blood flow to DLPFC is a common finding among researchers (Crider, 1997). Recent investigation suggests that sustained and selective attention alterations could be a vulnerability factor to psychotic disorders in general and not specific of schizophrenia (Mulet, Valero, Gutierrez-Zotes, Montserrat, Cortes, et al., 2007). Although, others have argued that executive function deficits appear to be a core component of schizophrenia rather than an effect of acute psychotic disorganization (Reed, Harrow, Herbener & Martin, 2002).

Another frequently investigated disorder is depression which is also associated with DLPFC hypometabolism, impaired attention and motivation which may stem in part from executive dysfunction (Filley, 2000). Finally, frontal lobe psychosurgery which is reserved for highly selected patients with intractable depression, anxiety and Obsessive-Compulsive Disorder is a procedure which involves a precisely limited operation guided by stereotactic techniques and which may result in EF deficits (Filley, 2000).

4.8 GAPS AND CHALLENGES
In 1982 Darley said that aphasic language impairment is ‘disproportionate to impairment of other intellective functions’ (cited in Davis, 2000, p. 4). Yet, clinical experience and research are indicating that executive deficits are common. In fact when considering the anatomy of the EF as described in the previous chapter, it is not surprising that executive dysfunction would result from stroke as it manifests in all neurogenic communication disorders which affect not only relevant anatomical sites but connections to those sites. Furthermore, executive dysfunction may account for the pervasive deficits seen when patients fail to generalise
communication strategies into their every day conversations. Functions related to attention and working memory are relatively well researched. However, although they both represent complex constructs and processes, they only represent two of the areas that are considered critical to EF. Planning, regulation of affect and flexible or generative behaviour are not often studied and certainly there are no comprehensive investigations that look at all these functions together.

Related to this dearth of information, one could say that the most glaring aspect absent from the literature on EF, aside from a lack of comprehensive executive profiles for stroke patients, is an accepted theoretical framework within which to assess, interpret, diagnose and treat executive dysfunction. Despite the fact that theories of prefrontal function and EF specifically exist, much of the literature on EF is descriptive but almost completely atheoretical. Authors purport to study EF but in fact are only concerned with individual or selected few capabilities, usually related to attention or working memory. Results are then interpreted within relevant theories of attention or working memory as the case demands, but not within a comprehensive EF framework.

Still, it is not surprising that there are so few inclusive EF batteries for individuals with stroke given that this area is so challenging to study. One of the reasons is that the components that are supposed to comprise EF are themselves not well defined and the nomenclature is confusing (Tompkins, 1995). Often definitions of EF consist of lists of sub-processes but concepts may overlap on some lists or be at fundamentally different levels e.g. resistance to interference is at a much lower level than planning or problem solving. The same term may be used to refer to conceptually and functionally different things. As an example, EF is sometimes used interchangeably with terms related to attention, despite the fact that there are many skills and underlying abilities that are executive in nature, which are not exclusive to the realm of attention. Then different terms are sometimes used to refer to the same construct and within each dimension, debate rages about whether that particular skill is a unitary general cognitive construct or reflects dissociable processes (Miyake, Emerson & Friedman, 2000).

Another difficulty relates to measurement problems i.e. what task best measures a particular construct? The practice of relying on complex frontal tests is problematic. Although the terms ‘frontal’ and ‘executive’ are often used interchangeably, conceptually they are not identical.
Many people with frontal damage do not exhibit executive deficits while some people who do not have frontal deficits do have EF problems (Murray & Ramage, 2000). To illustrate this, performance on the Wisconsin Card Sorting Test may be compromised in patients with posterior damage who have problems with short term memory for phonological information such as repetition or digit span. However the test is also well known for highlighting perseveration. Task impurity is a real challenge as there are no pure measures that tap EF exclusively yet there are many ways for patients to demonstrate impaired performance.

Another significant problem with measurement is that executive tests are notorious for having poor reliability. There are several reasons. The first is that executive tasks must contain a degree of novelty (Ylvisaker & Feeney, 1998). Once an examiner presents a test again, the patient may use different strategies at different testing times or even within the same session. Performance on measures considered executive that are used to assess these skills may depend crucially on other fundamental abilities. Also individual differences are vast in approaches and aptitudes. Complex skills can be hard to measure on formal tests partly because habitual or automatic routines may be adequate for performing when sufficient structure is provided and in part because so many component processes are necessary for success. Researchers are therefore faced with the dilemma of needing to administer relatively complex tasks in order to highlight deficits that are not apparent when testing simpler cognitive skills (Butler, Rorsman, Hill & Tuma, 1993; Stuss, 1993). However, interpretation is then confounded as the test draws on so many skills for successful performance (Stuss, 1993; Godefroy & Rousseaux, 1997) and speaks for the need to administer control tasks in addition to the executive tasks to rule out confounding variables, such as neglect (Keil & Kaszniak, 2002).

With regard to assessment of EF in the stroke and more specifically the aphasic population, Keil and Kaszniak (2002) highlight a number of further challenges. Most obviously EF tests often have verbal and other language requirements. Even those tests that do not specifically require verbal responses may have language elements to them that pose difficulties in comprehension or reading and therefore presents a high possibility for misunderstanding task instructions (Keil & Kaszniak, 2002). Specifically novel requirements often lead to difficulties in understanding instructions, despite intact comprehension. Frank comprehension deficits can only exacerbate this problem. Also many EF tasks are complex and require multiple sets of rules or steps which make them more sensitive to brain damage but also much more challenging to understand, even
for tasks that are purportedly assessing nonverbal intelligence. Advanced age, a common variable in individuals with stroke, can in and of itself present a tendency towards misunderstanding of complex task instructions (Philips, 1997). When more equitable testing conditions are sought by removing time constraints and simplifying instructions or supplementing them with gesture and drawings, other drawbacks are highlighted. Speeded tasks given with unlimited time compromise the ability to interpret the test to group norms and relaxes standards of test administration to the point where results may be invalidated. The inclusion of more practice examples reduces the novelty so critical to the assessment and researchers may inadvertently provide insight into test performance in an attempt to simplify and disambiguate test instructions.

Furthermore, there are a range of sensory, motoric, perceptual and demographic variables that are not taken into account when using existing EF tests for a neurogenically disordered population (Murray & Ramage, 2000).

Given the variables described above it is perhaps understandable why researchers have balked at the challenge of examining in detail executive dysfunction in individuals with stroke. However, it is in this very arena that we may find the answers to some of our most impenetrable problems. For those patients whose linguistic profiles belie the extent of their difficulties in every day conversations or who appear to manage so well in therapy but cannot generalise their gains to their lives outside their clinicians’ therapy rooms, this study proposes that executive dysfunction holds a critical component of the answer.

So far this chapter has examined EF deficits in a wide range of neurogenic communication disorders. In addition, the challenges of studying executive dysfunction in general and in the stroke/aphasic population more specifically have been addressed. The resulting gaps in the literature have been highlighted with the presentation of this study’s hypothesis that in order to completely understand the communication challenges faced by our stroke patients, we need to start investigating EF as comprehensively as possible, within a theoretically sound framework that defines the relevant aspects of EF and their inter-relationships. The following section presents a discussion related to attempts to treat EF disorders through pharmacotherapy.
4.9 PHARMACOLOGICAL TREATMENT OF EXECUTIVE DYSFUNCTION

In Chapter 2 the mechanisms of recovery from stroke were discussed in detail with the resulting logic that pharmacological treatment of stroke-related symptoms could potentially provide the stabilizing force needed to return neurochemical balance to the damaged area. By using pharmacological agents to salvage the penumbra especially, exposure to behavioural interventions may be more beneficial and be integrated more effectively. In a similar vein, a range of pharmacological agents have been researched in relation to their potential to alleviate effects of executive dysfunction in a wide range of disorders. Interestingly, despite the fact that both areas of research would appear to be informed by similar disciplines (i.e. an understanding of neurochemistry and effects on behaviour), there seems to be surprisingly little overlap between research focused on treating stroke related disorders and EF disorders. This is perhaps a comment on the fact that EF deficit as a result of stroke has not yet filtered into enough relevant research areas.

The neurotransmitter systems implicated in EF were discussed in detail with research connecting their participation to various functions in a number of clinical populations. Therefore this brief review focuses on recent research that specifically looks at the treatment of various EF and outcomes. As in the preceding chapters, the discussion is divided into segments for each neurotransmitter system. The discussion includes investigations into the effects of catecholamines (dopamine, amphetamines and norepinephrine), serotonin, acetylcholine and GABA. Throughout the review, parallels are drawn between this discussion and research related to treatment of stroke. An evaluation of the suitability of available drugs for this research is also included.

4.9.1 Catecholamines

4.9.1.1 Dopamine

Research in experimental animals strongly suggests that stimulation of dopamine D1 receptors in the prefrontal cortex can ameliorate spatial working memory related cognitive deficits, and may even enhance cognitive function in healthy animals. Research in humans has not been able to clearly replicate these findings, partly due to the lack of available agents that can safely be used. In the literature on treatment of stroke, it is not surprising that dopaminergic substances have not been extensively studied, as many of these agents such as bromocriptine and madofinil
have side effects that are potentially harmful to individuals who have had stroke including altering blood pressure and arrhythmia.

Low doses of dopamine D2 receptor agonists such as bromocriptine and pergolide may be able to enhance working memory and executive functions, but these effects may be dependent on the nature of the tasks used and the baseline performance levels of the participants. Thus, the effects of dopaminergic cognitive enhancers may not be simple or uniform across subjects. Systematic studies in humans with carefully controlled task parameters are needed in order to specify the potential cognitive processes open to enhancement with dopaminergics. However, since the DA receptor subtypes in different brain regions appear to differentially influence similar functions, carefully defining the cognitive processes to be tested against potential therapeutics is an equally important goal. Studies in patient groups using selective dopaminergics are rather restricted, but show promise for designing large-scale clinical trials into the cognitive enhancing properties of potential therapeutic agents that act through the dopamine system (Mehta & Riedel, 2006).

In a study by Honey, Suckling, Zelaya, Long and Routledge et al. (2003), 23 healthy elderly participants were scanned in a repeated measures, randomized, placebo-controlled design to measure modulation of physiological connectivity between cortico-striato-thalamic (CST) regions following treatment with drugs which served both to decrease (sulpiride) and increase (methylphenidate) dopaminergic transmission, as well as non-dopaminergic treatments (diazepam and scopolamine) to examine non-specific effects. They demonstrated enhanced functional and effective connectivity of human caudate nucleus following sulpiride treatment, which is compatible both with the anatomy of ascending dopaminergic projections and with electrophysiological studies indicating abnormal coherent oscillations of CST neurons in parkinsonian states.

In patients with schizophrenia, modafinil, a wake-promoting agent believed to operate via the hypocretin/orexin system, has a similar clinical profile to that of conventional, dopaminergic stimulants. Increasing interest in the use of modafinil to improve cognition in schizophrenia as well as attention-deficit/hyperactivity disorder has been addressed because of the association between cognitive performance and functional outcome. Initial findings indicate that modafinil may lead to better executive functioning and attentional performance in patients with
schizophrenia. The results further suggest that patient characteristics such as overall current cognitive functioning levels, genetic polymorphisms, and medication status may be important mediators for the effectiveness of modafinil, allowing for future treatment to be targeted to those most likely to benefit (Morein-Zamir, Turner & Sahakian, 2007).

Recently, research has demonstrated that a functional polymorphism in the catecholamine-O-methyltransferase (COMT) gene impacts prefrontal cognition and raises the possibility of a novel pharmacological approach for the treatment of prefrontal lobe executive dysfunction. To explore this Apud and colleagues (2007) tested the effects of tolcapone, a CNS penetrant specific COMT inhibitor in a randomized, double blind, placebo controlled, and crossover design in normal subjects stratified by COMT (val158met) genotype. COMT enzyme activity was determined in peripheral blood. Forty-seven normal volunteers underwent physiological measurement of prefrontal information processing assessed by blood oxygen level-dependent functional magnetic resonance imaging (fMRI). They found significant drug effects on measures of executive function and verbal episodic memory and a significant drug by genotype interaction on the latter, such that individuals with val/val genotypes improved, whereas individuals with met/met genotypes worsened on tolcapone. fMRI revealed a significant tolcapone-induced improvement in the efficiency of information processing in prefrontal cortex during a working memory test. This study demonstrates enhancement of prefrontal cortical function in normal human subjects with a nonstimulant drug having COMT inhibitory activity (Apud, Mattay, Chen, Kolachana, Callicott et al., 2007).

Rabins, Lyketsos and Steele (1999) promote the use of amantadine, l-dopa, bromocriptine and bupropion which all augment dopamine neurotransmission as agents of first choice for demented patients with productive executive syndromes (i.e. patients who are disinhibited, stimulus bound, intrusive, wandering, distractible or who engage in repetitive behaviour such as hoarding, tapping or vocalizing). Despite its potentially harmful side effects for those at risk for stroke, bromocriptine has been researched in relation to recovery from stroke, although results have been equivocal with some studies evidencing improved verbal fluency and speed of cognition.
4.9.1.2 Amphetamines

Rabins et al. (1999) also cite the usefulness of the amphetamine methylphenidate (Ritalin) in patients with head injury in the treatment of dysexecutive syndromes, with improved focused attention and set switching. Consistent with their findings, Frankel and Penn (2007) also found an amelioration of attention related functions (in particular interference control and behavioural inhibition) with the use of methylphenidate in two participants with TBI. This study was particularly informative in the design of the current research as neuropsychological profiles were augmented with conversational data and improvements in conversational features were also noted related to improved attention in the active phase of the trial. Therefore, the use of methylphenidate was initially the primary choice for this study. There is also a precedent of amphetamines being used in the stroke population with positive results (Small, 1994; Walker-Batson et al., 2001). However, when investigated more thoroughly, it was discovered that among the side effects are possible increase in blood pressure and arrhythmia, possibilities considered too risky for individuals who had already suffered strokes. The possibility of using methylphenidate or in fact any other amphetamine was therefore discarded because of possible side effects relating to heart function and blood pressure.

4.9.1.3 Norepinephrine

In the stroke population, norepinephrine has been found to improve depression and as a result speed up recovery from stroke (Finklestein et al., 1987). Current research also suggests that both norepinephrine and dopamine contribute to beneficial actions of stimulant medications on regulation and attention (Arnsten, 2006; Popper, 2000). There are some reports that atomoxetine, a new and highly selective inhibitor of the norepinephrine transporter improves inhibitory capacity, as measured by the Stroop task. This hypothesis was tested and proved in a study with adults with ADHD who showed improved interference control on the Stroop task, after being treated with atomoxetine for ten weeks (Faraone, Biederman, Spencer, Michelson, Adler, et al., 2005). Furthermore, nonstimulant agents that are effective in the treatment of ADHD tend to affect the norepinephrine system, whereas those affecting only dopamine, or those that affect neither catecholamine, are less potent in reducing ADHD symptoms. Studies of the effects of norepinephrine and dopamine peripheral metabolites by ADHD pharmacotherapies show acute increases in levels of these catecholamines. However their long-term turnover may be reduced (Pliszka, 2005).
Coull (1994) published a review in which the extent to which the manipulations of the noradrenergic system affects cognition was examined. Attentional deficits in attention deficit hyperactivity disorder and visuospatial neglect are often ameliorated by noradrenergic drugs. Noradrenergic beta-blockade suppresses the encoding of emotionally arousing unpleasant stimuli and reduced amygdala activation in healthy volunteers, with potential implications for posttraumatic stress disorder (Chamberlain, Muller, Robbins & Sahakian, 2006). The aged primate brain is prone to degeneration of the locus ceruleus, as well as profound catecholamine depletion in the prefrontal cortex, and so is ideal for psychopharmacological investigation of the role of noradrenaline in frontal lobe function. Elderly monkeys show deficits in performance of the delayed response task, which can be reversed directly by both the mixed alpha(1)/alpha(2)-agonist clonidine, the more specific alpha(2)-agonist guanfacine and also, indirectly, by the alpha(2)-antagonist yohimbine. It is suggested that these results can be explained by an attenuation of the distracting properties of irrelevant stimuli following stimulation of noradrenergic activity. Conversely, distractibility is magnified whenever noradrenergic activity is reduced. This is supported by similar findings in psychopharmacological studies of healthy humans. The exception to this is when the locus ceruleus is likely to be firing, e.g. in times of stress or when novel stimuli are encountered. Clonidine attenuates locus ceruleus firing on such occasions, and so counteracts any beneficial (or deleterious) effects of stress on task performance.

Alpha(2)-Adrenoceptor agents have little therapeutic value in patients with dementia of the Alzheimer's type. However, they may have some clinical use in patients who have a cognitive symptomatology similar to that of patients who have received neurosurgical excisions to the frontal lobes, e.g. deficits in working memory, executive function or focused attention, with relative sparing of episodic short term memory. Patients with Korsakoff's disease, attention deficit disorder or schizophrenia may benefit from treatment with alpha(2)-agents. In particular, idazoxan has putative therapeutic effects in patients with a neurodegenerative disorder, namely dementia of the frontal type.

The complexity of these interactions combined with the fact that many substances that affect norepinephrine transmission also have other drug interactions and may induce arrhythmia, disqualified this class of neurochemicals from consideration.
4.9.2 Serotonin

There is little research regarding serotonin related treatments relative to the other neurotransmitter systems. Serotonin manipulations in healthy volunteers do not appear to affect response inhibition but do affect performance on cognitive tests involving emotionally salient rewards and feedback, suggesting involvement of this neurochemical in affective aspects of impulsivity (Chamberlain, et al., 2006). There is evidence for double dissociations of the effects of manipulations of prefrontal cortical catecholamine and indoleamine (5-HT) systems that have considerable implications in the treatment of disorders such as Parkinson's disease, attention deficit/hyperactivity disorder and depression, as well as in theoretical notions of how 'fronto-executive' functions are subject to state-dependent influences, probably related to stress, arousal and motivation (Robbins, 2007; Valkalopoulos, 2007). In addition, there are indications that a blockade of serotonin receptors (5-HT2A) has proven useful in the treatment of psychiatric disorders (Stein, Hemmings, Moolman-Smook, Audenaert, 2007). In this regard, Tanaka and Albert’s study (2001) which showed decreased depression and reduced perseveration in individuals with fluent aphasia is relevant. However, the drug in that study, fluvoxamine maleate, has significant and health threatening drug interactions, and could therefore not be considered for this study.

4.9.3 Acetylcholine

Several sources of evidence suggest that cholinergic deficits may significantly contribute to dementia in Parkinson's disease (PDD) and acetylcholinesterase inhibitors (ChEIs) have been reported to improve cognitive symptoms in PDD, without worsening parkinsonism (Bedard, Lemay, Gagnon, Masson & Paquet, 1998). Nineteen PDD patients underwent brain perfusion SPECT with Tc-99m-ethyl cysteinate dimer after 6 months ChEIs treatment in order to evaluate the functional correlates of clinical improvement. A clear-cut cognitive improvement was reported in PDD patients with a significant improvement of scores exploring executive functions. This data confirmed the efficacy of ChEIs in the treatment of dementia associated with PD mainly on attention and executive functions, and the functional findings indicate that this cognitive improvement is possibly associated with a pharmacological frontal "re-afferentation" (Ceravolo, Volterrani, Frosini, Bernardini, Rossi, et al., 2006).

ChE-1s also attenuate the cholinergic deficit underlying the cognitive and neuropsychiatric dysfunctions in patients with Alzheimer’s disease (AD). Inhibition of brain acetylcholinesterase
(AChE) has therefore been the major therapeutic target of ChE-1 treatment strategies for AD. AChE-positive neurons project diffusely to the cortex, modulating cortical processing and responses to new and relevant stimuli. Butyrylcholinesterase (BuChE)-positive neurons project specifically to the frontal cortex, and may have roles in attention, executive function, emotional memory and behaviour. Furthermore, BuChE activity progressively increases as the severity of dementia advances, while AChE activity declines. Therefore, inhibition of BuChE may provide additional benefits. The two cholinesterase (ChE) enzymes that metabolize acetylcholine (ACh) differ significantly in substrate specificity, enzyme kinetics, expression and activity in different brain regions, and complexity of gene regulation. In addition, recent evidence suggests that AChE and BuChE may have roles beyond 'classical' co-regulatory esterase functions in terminating ACh-mediated neurotransmission. 'Non-classical' roles in modulating the activity of other proteins, regional cerebral blood flow, tau phosphorylation, and the amyloid cascade may affect rates of AD progression. If these additional mechanisms are demonstrated to underlie clinically meaningful effects, modification of the over-simplistic cholinergic hypothesis in AD that is limited to symptomatic treatment, ignoring the potential of cholinergic therapies to modify the disease process, may be appropriate. The specificity of ChE inhibitory activity, up-regulation of AChE activity and changes in the composition of AChE molecular forms over time, selectivity for AD-relevant ChE molecular forms, brain vs. peripheral selectivity, and pharmacokinetic profile may be important determinants of the acute and long-term efficacy, safety and tolerability profiles of the different ChE-Is (Lane, Potkin & Enz, 2006).

The use of rivastigmine (a cholinesterase inhibitor), has been supported as being beneficial for the treatment of cognitive deficits related to Alzheimer’s disease (AD) (Bullock & Lane, 2007). A new development, the rivastigmine patch is the first transdermal treatment for AD. By providing continuous delivery of drug into the bloodstream over 24 hours, transdermal delivery may offer benefits superior to those of oral administration. This study looked at the efficacy, safety and tolerability of rivastigmine patches. 1195 patients with AD were randomized to placebo or one of three active treatment target dose groups: 10-cm(2) rivastigmine patch (delivering 9.5 mg/24 hours); 20-cm(2) rivastigmine patch (17.4 mg/24 hours); or 6-mg BID rivastigmine capsules. All rivastigmine treatment groups showed significant improvement relative to placebo. The 20-cm(2) patch showed earlier improvement and numerically superior cognitive scores vs. the 10-cm(2) patch. The transdermal patch with rivastigmine may offer
additional therapeutic benefits and may prove to be the best delivery system for this drug to treat AD (Winblad, Grossberg, Frolich, Farlow, Zechner, et al., 2007).

The effects of donepezil, an inhibitor of AchE, were assessed on the cognitive performance of 30 young and healthy subjects. Time-by-group interactions demonstrated significant drug effects that were specific to episodic memory in both the verbal and visual domain. Additionally, donezepil significantly improved long-term visual episodic recall (Gron, Kirstein, Thielser, Riepe & Spitzer, 2005).

As highlighted in Chapter 2, piracetam, a gamma aminobutyric acid derivative with direct cholinergic effects via the release of acetylcholine has an extensive history of use with individuals with stroke. As a result of its innocuous characteristics in relation to drug and disease interactions, and its potential in alleviating cognitive deficits that could have associations to EF, piracetam was the second drug of choice for the current study. In close consultation with UCB Pharmaceuticals, it was strongly advised not to use piracetam owing to the fact that leviteracetam (LEV), a newer more effective derivative was available. LEV is discussed in more detail below, along with research studies that supported its use in the current trial.

4.9.4 GABA

Studies in non-human primates indicate that normal working memory function depends upon appropriate GABA neurotransmission in the DLPFC, and alterations in markers of GABA neurotransmission are well documented in the DLPFC of subjects with schizophrenia (Lewis, Volk & Hashimoto, 2004). Postmortem studies suggest that markers of reduced GABA neurotransmission in schizophrenia may be selective for, or at least particularly prominent in, the subclass of GABA neurons, chandelier cells, that provide inhibitory input to the axon initial segment of populations of pyramidal neurons. Given the critical role that chandelier cells play in synchronizing the activity of pyramidal neurons, the pharmacological amelioration of this deficit may be particularly effective in normalizing the neural network activity required for working memory function. Because GABA(A) receptors containing the a(2) subunit are selectively localized to the axon initial segment of pyramidal cells, and appear to be markedly up-regulated in schizophrenia, treatment with novel benzodiazepine-like agents with selective activity at GABA(A) receptors containing the a(2) subunit may be effective adjuvant agents for
improving working memory function in schizophrenia (Lewis et al, 2004). GABA receptor antagonists are also being explored as future pharmacotherapies for ADHD (Weisler, 2007).

The above review has highlighted some of the recent research related to treatment of EF disorders in a range of clinical populations based on neurotransmitter systems. The catecholamines including dopamine, amphetamines and norepinephrine have been linked with improvements in working memory, disinhibition and attention functions. In this regard, substances affecting these neurochemicals would appear ideal in a drug trial targeting EF in stroke. However, potential side effects on heart function and blood pressure uniformly ruled out any of these substances as possibilities. Serotonin is most often associated with emotional regulation and affective or motivational aspects of attention. Acetylcholine has been established as being significantly involved with memory functions particularly useful in treating the cognitive deficits associated with AD. Finally, GABA has been linked to working memory ability. However, this summary belies the complexity of what must be considered when researching and planning pharmacological treatment of EF deficits. Specific interactions imposed by the neurotransmitter system in question, its interaction with other systems, patient demographic variables, genotype, clinical disorder as well as task parameters make this area both a challenging and intricate area of research.

4.10 LEVITERACETAM (LEV) (KEPPRA ®)

The drug used in the present study is leviteracetam (LEV) a pyrrolidine derivative that has a similar chemical structure to numerous nootropics drugs and is the S-enantiomer of the ethyl analogue of piracetam (Patsalos, 2003). Based on extensive in vitro experiments, the mode of action of LEV does not appear to derive from any interaction with known mechanisms involved in inhibitory or excitatory neurotransmission (Noyer, Gillard, Matagne, Hénichart & Wülffert, 1995) and neither does it appear to influence membrane excitability. It may seem counterproductive to use a substance that specifically does not target any of the neurotransmitter systems described in detail above. However as the review has highlighted, the neurochemical interactions are highly complex and would be difficult to interpret within the scope of the present study. In addition, although many of the substances used in current EF research have also been used in the stroke population, they also have concerning side effects, which would have made them inappropriate choices. In animal models a brain-specific stereoselective binding site has been identified for LEV in synaptic plasma membranes of the
central nervous system (Genton & Van Vleymen, 2000). Figure 4.2 below depicts its chemical structure and its primary pharmacologically inactive metabolite LO57.

Figure 4.1: The chemical structure of LEV and its metabolite LO57.

LEV was initially evaluated in models of cognitive impairment with the primary objective of finding a drug more effective than the better known piracetam (Genton & Van Vleymen, 2000), which has a history of use in patients with aphasia (see discussion above). In preclinical efficacy trials, LEV has been found to improve learning and memory in animals with underlying cerebral ischemia (Gobert, Verloes & Gower, 1988 cited in Genton & Van Vleymen, 2000).

Despite early data that demonstrated that piracetam demonstrated greater and more consistent cognitive benefits than LEV (Genton & Van Vleymen, 2000), more recent studies have started looking at the influence of LEV on cognitive function. Loring and Meador (2004) listed improvements in cognition, concentration as well as increased alertness in children with epilepsy treated with LEV. These improvements occurred in several patients without improved seizure control. Frings, Quiske, Wagner, Carius, Homberg, and Schulze-Bonhage, (2003), demonstrated statistically significant improvements in selective attention, verbal working memory and verbal fluency as well as in a visual planning task in eighteen epileptic patients introduced to LEV. A case study published by Canevini, Chifari and Piazzini (2002), described the disappearance of stuttering behaviours and improvements in verbal fluency in a 34-year-old woman treated with LEV. They postulated that LEV might influence the metabolism of the language area thereby increasing verbal fluency. A particularly interesting case study was reported by Kossoff, Boatman and Freeman (2003). A 5-year-old with Landau-Kleffner
syndrome, a rare epilepsy syndrome notable for language regression, received LEV monotherapy with a discontinuation of carbamazepine and valproic acid. Her seizures were controlled and her language improved substantially in the areas of speech sound perception, word and sentence comprehension, picture naming and auditory working memory.

In contrast however, Neyens, Alpherts and Aldenkamp (1995) did not find any significant improvements in cognitive performance in ten adult patients being treated for epilepsy. Nevertheless they noted a tendency towards better performance on motor tapping with the nondominant hand, faster binary choice reaction times and improved memory performance for simultaneously presented words, while on treatment. They concluded that further investigations with more sensitive designs and prolonged use of treatment need to be carried out.

For the purposes of research, LEV is almost ideal. After oral ingestion its absorption is rapid, linear and almost complete, with peak plasma concentrations occurring approximately one hour later. The extent of absorption is independent of dose and can be ingested without regard to meal times. Steady state plasma concentrations are reached within 24-48 hours. It rapidly crosses the blood-brain barrier to enter both brain extra-cellular and cerebrospinal fluid compartments. LEV is excreted primarily unchanged by the kidneys and is cleared fairly rapidly with approximately 93% of the drug being excreted within 48 hours following oral administration. It also has no drug interactions. These characteristics make it easy to track reactions of participants to the drug because of minimal inter-patient variability in blood concentrations and consequently efficacy, minimal complications with regard to interpretation of therapeutic drug monitoring, minimal interaction potential and relatively rapid changes in response to drug dosages (Patsalos, 2003).

4.11 SUMMARY – THE NEXT STEP
The preceding chapters have laid the foundation for what emerges as the rationale for the present study. Chapter 2 provided an overview of stroke and the resulting communication deficits in both aphasia and RHD. Chapter 2 also reviewed the mechanisms of stroke and its recovery with a review of both traditional and more modern approaches to treatment, including two main areas of focus for the current study – interventions targeting conversation and pharmacotherapy.
Chapter 3 provided a detailed description of EF, their neuroanatomy and neurochemical substrates and the various existing theories that describe them. Barkley’s model was highlighted as the most comprehensive of these as it provides operational definitions and more importantly, a cohesive framework which specifically lays out the relationships and interactions of the EF with one another. This model provides the structure within which this research was designed and interpreted.

This chapter has outlined the various EF deficits arising in a range of neurogenic communication disorders. The critical focus of this chapter was to highlight similarities in executive dysfunction across a wide variety of sites of lesion and disease processes suggesting that EF is widely represented and distributed across the cortex and extremely vulnerable to disruption. Significantly it is under-appreciated in individuals who have suffered strokes. Although there is increasing awareness of the need to assess EF in this population there are virtually no studies to date that have tested EF comprehensively in any group of individuals in chronic stages post onset. The need to do this was highlighted in relation to the fact that it is potentially EF deficit that could account for poor therapy outcomes and lack of generalisation to everyday environments.

Finally, this chapter concluded with a review of pharmacological agents used in the treatment of EF with commentary regarding the characteristics that allowed for consideration for use in this study. As a result, the complexity of the neurotransmitter systems and their interactions was demonstrated. The use of LEV, which does not rely on a neurotransmitter system but which nonetheless affects cognitive and central nervous system functioning, therefore seems an appropriate agent to test in this population. Where researchers need to be mindful of the potentially deleterious side effects of medication, the fact that LEV is a safe drug with no other drug interactions and mild side effects along with its other pharmacokinetic properties makes it an ideal research prospect.

The following chapter presents the aims and general methodology of this study.
CHAPTER FIVE
METHODOLOGY

This chapter presents the aims, design and general methodology of this research as well as relevant details of subsidiary investigations that informed the primary study. Components of the research test battery are discussed and methods of data analysis presented.

5.1 AIMS OF THE STUDY
As argued in the preceding chapters, conversation is a complex cognitive task, which has the potential to open windows onto language as well as EF abilities. These underlying skills provide critical contributions to conversational intelligence and are of particular interest to the speech language pathologist. While EF testing in aphasia and right hemisphere disorders is in its infancy it holds significant promise for more effective assessment and intervention and therefore comprehensive profiling of the executive profile of individuals who have suffered a stroke would be worthwhile.

This study comprised two components – a descriptive element and an experimental one. In the descriptive component, the aim was to document the language, executive and conversational profiles of ten individuals who had experienced a stroke. These profiles were then examined to determine if there were associations among the linguistic, executive and conversational features. In the experimental component, all ten participants took part in a drug trial. Executive and conversational profiles were generated for each of the four stages of the study (baseline, active, placebo and withdrawal) and compared against each other to assess whether LEV effected changes in either the executive or conversational characteristics.

5.2 DESIGN OF THE STUDY
This research took the form of multiple case studies. As a result of the nature and complexity of the variables under investigation as well as the fact that this research does not have a precedent in the literature, each of the ten participants acted as their own controls and were examined individually. These mini ethnographies (Grbich, 1999) are characterized as being one of the best arenas in which quantitative and qualitative research can be combined (Bryman, 1989).
The methodological approaches associated with case studies are eclectic and broad – in fact virtually all methodologies can be drawn upon and case studies rarely depend on one approach (O’Leary, 2004). Case studies therefore allow for in-depth exploration, the examination of subtleties and intricacies. They attempt to be holistic by exploring processes as well as outcomes and investigate the context as well as the setting of a situation (O’Leary, 2004). O’Leary (2004) further maintains that case studies are especially suited for clinical treatment evaluation, as they can integrate clinical services and research and help generalize from research to individual clients, thereby providing a logical generality through detailed descriptions of client characteristics and treatment effects. Case studies can also be more easily replicated than group studies.

Aspects of the study employed a qualitative orientation, specifically the capturing and analysis of conversational data, which utilized a hybrid approach, drawing mainly on Conversation Analysis (CA) to inform principles of video recording, generating transcripts and analysis thereof (Ten Have, 1999; Wooffitt, 2005). In addition to CA, the work of Heath (1986) and Jefferson (2004) was used to capture, transcribe and analyze nonverbal data such as eye gaze and gesture when these variables were considered critical to conveying communicative acts during conversation. Other areas of the study, including the testing of language and executive skills aligned with a quantitative approach in which tests with standardized norms were administered, although some of the executive tests were adapted for use with a stroke population.

In terms of the experimental drug trial, four phases were included: baseline, active and placebo phases as well as a withdrawal phase. The active and placebo phases were randomized by an external source (Head of the Pharmacy Department at Wits University) for each participant and in addition, a double blind precaution was incorporated in order to minimize bias during data collection and interpretation (Drummond, 1996). The list containing the assignment of the drugs and placebos to phase A or B for each participant was sealed and kept by the Head of the Pharmacy Department for the duration of the investigation and revealed only after all data analysis had been completed.
5.3 PHASES OF THE STUDY

5.3.1 Compiling the executive battery

Executive functioning tests are not routinely included among the batteries of speech language pathologists in their assessment of aphasia. There are several reasons for this, discussed in Chapter 4. To review briefly, the notion of executive contributions to language deficits is a relatively new idea in this field. Second, the assessment of EF in this population is highly complex and requires a thorough understanding of the neuropsychological arena from which much of the research on EF comes and from where tests are generally developed. Third, an appreciation of the contextual variables that need adaptation of some tests is critical if the tests used are to provide any meaningful information. Some of these variables include cultural and educational background as these can significantly affect performance (Nell, 1994) and in the case of people with aphasia, the verbal demands required to explain the test as well as verbal requirements for responding, use of the non-dominant hand and slowed rate of information processing to name a few (Keil & Kaszniak, 2002).

Barkley’s model (1997), introduced in Chapter 3 is a comprehensive framework drawing together established research and combining constructs from overlapping theories into a single hybrid model. This provides a more thorough accounting of self-regulating functions than other theories presented as discussed earlier. It was critical to include an experienced neuropsychologist to evaluate Barkley’s constructs, and assist with the development of a battery that would effectively target contributions to EF, provide specific information about various skills and abilities and also be suitable for use with people who had had a stroke or which were adaptable to their individual needs. In addition, he was instrumental in the interpretation of preliminary data and was able to provide input regarding the possible contributions of confounding variables and how to further assess, or make provisions for these in future testing. The completed EF test battery including the constructs assessed, the tests utilized and the rationale for choosing them, the manner in which they were administered and scored and how they were adapted appears in Appendix A in Table A.1. Table 5.4 below also lists the critical areas of EF, their fit with Barkley’s concepts of EF and the tests representative of each construct. In addition, where research has indicated significant practice effects with multiple exposures to the same test, a significant disadvantage of multiple testing (Drummond, 1996), parallel versions were used to counteract this confounding variable. These are marked in Table A.1 with a *.
5.3.2 Piloting the executive battery

The design of the EF battery and its usefulness in merging the information with conversational data needed to be assessed. The battery in its entirety – incorporating language and EF testing as well as conversational sampling was piloted with one of the participants who would later be included in the final ten who took part in the study. The pilot study (Frankel, Penn & Ormond-Brown, 2007) supported the contention that EF deficits are likely to account for numerous manifestations of communication difficulty in aphasia, particularly at a conversational level. In addition, the merging of conversational and neuropsychological paradigms appeared to be successful, with each set of results complementing the other. The pilot study also revealed the need to include a pointing task for tests of digits forward and backward, in addition to the verbal response mode, to rule out the effects of oral or verbal Apraxia.

5.3.3 Ethical considerations

It is standard protocol to submit a request for ethical clearance from the University of the Witwatersrand (Medical) when research is to be conducted with human participants, which was the case with this research. In addition to clearance from the university, clearance was also sought at an international level with the contributing drug company’s head office in Belgium (UCB Pharmaceuticals). Owing to the complexity of the research, the inclusion of multiple and randomized phases and the use of a placebo, there was particular concern regarding the ability to truly gain informed consent (IC) from potential recruits. For this reason, the researcher additionally obtained permission to record the recruitment process, in order to later evaluate its success. Appendices B – F present: the participant and caregiver information sheets, participant consent form, participant consent form for the use of video recordings, caregiver assent form and caregiver assent form for the use of video recordings.

Following data collection for the primary study, the author collaborated with three other researchers to identify potential facilitators and barriers to the process of IC in some of the participants with aphasia, focusing on both verbal and non-verbal components of the interactions. The motivation behind this subsidiary investigation was that people with aphasia are particularly vulnerable when taking part in research studies and there was a degree of concern that recruitment although carefully undertaken may still have been flawed.
The process of informed consent depends on a number of factors which may be compromised in aphasia. Very little research has been conducted on the process and the issue is often neglected in published research. The IC process for three trial participants was examined in detail. Specific portions of the enrolment process dealing with the explanation of the concepts ‘placebo’, ‘randomisation’ and ‘double blind’ were analysed. Analysis methods were qualitative and comprised systematic observation and scrutiny of the video-recorded recruitment as well as feedback sessions with these participants after the study had been completed and their participation in the research was over.

Results of this ethical study demonstrated that the process of IC was widely discrepant. There were marked differences in the way that the participants reacted to the process and in the behaviours of the clinician during each enrolment, also differences in terms of length of enrolment and the degree of confidence with which the researchers believed consent had been authentic. Paradoxically some attempts to facilitate the process seemed to have an inhibitory effect. In addition, a review of published research in three prominent journals in which aphasia research is often produced was undertaken. These articles were analysed to determine how often IC was mentioned and to what extent this process was described. The evidence suggested that IC is often neglected and at best is difficult to obtain. There are multiple influences on the process of IC in aphasia which include the potential for therapeutic misconception i.e. trial participants may believe that every aspect of a research project to which they consent is designed for them specifically and may hence participate out of self interest and interpret and even distort the information received on the trial to maintain this view (Dresser, 2002; Lidz & Appelbaum, 2000; Hochhauser, 2002). The therapeutic misconception is particularly likely to emerge in the context of clinical research because of the close and often sustained relationship between the investigator and the participants.

The process seems particularly jeopardised in qualitative and clinical research. There are many possible reasons why a person might agree to take part in a trial, but there are numerous pitfalls and barriers to the process. In order to combat these pitfalls, certain principles need to be borne in mind. Firstly, that IC is an ongoing process, which should be negotiated throughout every phase of a research project. Second, the process of obtaining IC can not be delegated lightly. Training of research assistants is essential in order that recruiting researchers can adapt flexibly to the individual needs of potential recruits. Third, competence is site specific, meaning that just
because an individual has agreed to take part in one research project does not automatically make them eligible for participation in another. Fourth, language specialists have a particular role to play when recruiting individuals with communication difficulties. Different communication disorders present with different profiles of strength and weakness in terms of communication and these factors need to be considered during the recruitment process. Finally in the paper that stemmed from this research the recommendation was made to consult with a community representative who could serve as a participant advocate for the duration of the study, assisting to mediate between the goals of the researchers and the best interests of the participant (Penn, Frankel, Watermeyer and Muller, accepted). While aphasia is a complex and fruitful testing ground for a number of issues, obtaining genuine IC may be more elusive than we suppose. While language and cognitive problems may be the primary barrier, focus on these aspects should not be at the expense of understanding a range of other contextual and process variables which render the potential participant vulnerable.

5.3.4 Preparation of the drug

The study, which investigated the effect of LEV on executive functions and conversational performance, included randomized active and placebo phases. As the participants and researcher were blind to which phase of the study was being conducted, the drug had to be disguised. At a dosage of 1500mg per day, participants were required to take one 750mg pill in the morning and another of the same dosage in the evening. This dosage was determined by a consulting neurologist who performed preliminary assessments on each of the participants, in collaboration with UCB Pharmaceutical’s research director in Johannesburg. Due to the large size of the tablets, concealing them became a significant challenge. In collaboration with the University of the Witwatersrand Pharmacy Department and UCB Pharmaceuticals, the tablets were prepared in the following way. All LEV tablets were inserted into an opaque capsule. The placebo capsules contained a locally available vitamin called Centrum® (Wyeth Consumer Healthcare) which is similar in size, shape and colour to LEV. Ten packages were then prepared containing a bag of 60 capsules with LEV and a bag of 60 capsules with Centrum®, the packages being labeled with a research participation number from 1 – 10 for each of the ten recruited participants, who were assigned their numbers as they were inducted into the study. The Head of the Pharmacy Department then randomly assigned the bags to an A or B pile, noting which was which for each participant. The list produced is replicated in Table 5.1 below.
This list was then sealed and kept by the Pharmacy Department, until all the research data had been fully analyzed.

Table 5.1: Randomization of Participant Placebo and Active Phases

<table>
<thead>
<tr>
<th>PATIENT NO</th>
<th>BAG A</th>
<th>BAG B</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>PLACEBO</td>
<td>ACTIVE</td>
</tr>
<tr>
<td>2</td>
<td>ACTIVE</td>
<td>PLACEBO</td>
</tr>
<tr>
<td>3</td>
<td>ACTIVE</td>
<td>PLACEBO</td>
</tr>
<tr>
<td>4</td>
<td>PLACEBO</td>
<td>ACTIVE</td>
</tr>
<tr>
<td>5</td>
<td>ACTIVE</td>
<td>PLACEBO</td>
</tr>
<tr>
<td>6</td>
<td>PLACEBO</td>
<td>ACTIVE</td>
</tr>
<tr>
<td>7</td>
<td>ACTIVE</td>
<td>PLACEBO</td>
</tr>
<tr>
<td>8</td>
<td>ACTIVE</td>
<td>PLACEBO</td>
</tr>
<tr>
<td>9</td>
<td>ACTIVE</td>
<td>PLACEBO</td>
</tr>
<tr>
<td>10</td>
<td>PLACEBO</td>
<td>ACTIVE</td>
</tr>
</tbody>
</table>

5.3.5 Initial Neurological Assessment

Following the recruitment process, participants each agreed to meet with a consulting neurologist who examined and confirmed their diagnoses of single incident stroke. In addition he took a case history from the participants and caregivers where this was appropriate, noting co-existing medical conditions. In one instance further investigation was warranted and the participant was referred to a local hospital for a CAT scan. This revealed only the presence of the previous infarct and no new diagnoses emerged. The meeting with the neurologist also provided an opportunity for participants and/or caregivers to present queries or concerns related to the trial drug. Once the participants were cleared for participation, they were formally enrolled in the study and testing was scheduled.

5.3.6 Testing

Each participant underwent initial language testing where the researcher administered the Western Aphasia Battery (WAB) (Kertesz, 1982). This was the only time during the study that formal language testing took place. For the rest of the study, participants completed the entire executive battery at baseline, placebo, active and withdrawal stages. In addition each participant was recorded having a conversation with a family member, friend or the researcher (when no-one else was available), at each of these four stages. Table 5.2 on the following page, lists communicative partners for each of the participants for each of the four phases of the study.
Each participant received Bag A during their first month and instructed to take one capsule in the morning and one in the evening. During the second month, they were given Bag B and again instructed to take one capsule in the morning and one in the evening. A data control sheet was provided and each capsule had to be signed out. The control sheet had to be returned to the researcher as evidence that all 60 capsules had been taken during the 30 day trial periods. An example of the control sheet is provided in Appendix G.

5.3.7 Data Analysis

Data from the language and executive batteries at each stage were scored according to test guidelines, unless the scoring procedures had been modified as indicated in Appendix Table A.1. In such cases, consultation with the neuropsychologist regarding test performance resulted in a decision of whether performance was deemed intact or deficient.
In order to determine whether or not any significant changes occurred in performance over the course of the four stages, a repeated-measures analysis of variance (Howell, 1999) was conducted. This design is useful when there are large individual differences among participants, which lead to considerable variability in the data. When participants are measured repeatedly, their differences can be assessed and separated from random error which would be difficult to distinguish were participants to be measured only once. The main disadvantage of this design is that there is the risk of carry-over effects from one trial to the next. For example, a participant may learn something in Trial 1 that will assist them with performance in Trial 2. This problem can be reduced by counter-balancing the order in which treatments are administered. Thus the placebo and active phases of the study were randomized, in order to allow the carry-over effects to affect both phases equally. In addition, parallel versions were used for tests susceptible to practice effects. Post hoc analysis further revealed where the source of difference was for the one test which indicated changes in performance.

The executive profiles were then drawn up individually for each of the ten participants, in terms of functions that were intact and those that were deficient. In addition, the results were plotted by site of lesion as well as by aphasia type in order to determine whether patterns were evident. The conversational data were submitted to CA.

5.4 PARTICIPANTS

Ten people who had sustained a stroke participated in this study. In addition, there has been concern raised in the literature that writing or drawing for timed neuropsychological assessments may unfairly and significantly penalize the person taking the test when performing with their non dominant hand (Keil & Kaszniaik, 2002). In order to examine this contention a separate control study was conducted. A group of seven controls matched to the stroke participants were assessed on the paper and pen tasks using both their dominant and non dominant hands. The results of this control study are described below (section 5.4.2)

5.4.1 Selection criteria for stroke participants

Participants were not excluded on the basis of gender or prior or current therapy, although these variables were noted.
5.4.1.1 Inclusion Criteria

- Participants were individuals who had suffered single incident cerebrovascular accident (CVA). Multiple events complicate assessment and diagnosis and therefore a single site of lesion resulting from a single event was required. As an example, a history of multiple strokes is commonly associated with vascular disease and has been reported to be the second leading cause of dementia (Hopper & Bayles, 2001).

- Participants’ neurological assessments and examinations were available to the consulting neurologist to confirm diagnosis. Consultation with the participants’ treating general practitioners was imperative to ensure that the prescription of LEV was not contraindicated in combination with current prescriptions or that other medical conditions (e.g. renal impairment) did not preclude participation, although this was unlikely as the drug has convincingly been tested as being safe for use with other medications and medical conditions (Patsalos, 2003; Patsalos, 2000; Radtke, 2001).

- Participants were between the ages of 40 and 65 years. Due to the increasing prevalence of dementia in individuals over 65 years of age (Hopper & Bayles, 2001), recruits were preferred to be 65 years or younger. Dementia is a clinical syndrome defined by the deterioration of memory and cognitive functions, which if present would confound data collected, with its emphasis on cognitive performance and conversational functioning (Potkins, Myint, Bannister, Tadros, et al. 2003).

- Participants had successfully completed secondary education. This criterion ruled out significant premorbid learning disability which may have imposed confounding variables on current language and cognitive functioning (Basso, 1989).

- Participants spoke English as their first language or as a second language with enough proficiency to be able to cope in an English environment and not require an interpreter during testing. Also the researcher was best equipped to analyse and interpret conversational data in the same language as her own.

- Participants were in chronic stages of recovery. Language and cognitive skills often resolve spontaneously over time, particularly in the first few months post onset when physiological recovery processes are taking place (Weiller, 2000). Therefore, potential improvements noted would not be attributed to LEV exclusively but rather to spontaneous recovery. Participants therefore had to be at least three months post-onset.

- Participants presented with corrected sensory visual or hearing impairments.
• Participants demonstrated an ability to comply with the executive functioning test demands. They could therefore repeat or point to digits, read individual letters, numbers and words, count to 25, recognise and name colours as well as have preserved comprehension. The latter was also critical in terms of the participants’ ability to understand the information sheets and be fully participative in the informed consent phase of this study (although see section 5.3.3 regarding Ethical Considerations above).

5.4.1.2 Exclusion Criteria:

• Participants with severe aphasia were excluded from the study. The study relied to a large extent on the ability of the individual to participate in conversation. In addition, participants needed to have a minimum repertoire of preserved skills in order to meet the demands of the executive functioning test battery and supply consent. These skills are not likely to be preserved in individuals with severe aphasia, which includes significant comprehension difficulties (Chapey & Hollowell, 2001).

• Participants with a prior record of alcohol abuse or treatment for toxic substances were excluded, as iatrogenic or surreptitious drug exposure imposes separate and confounding effects on neurological involvement resulting from brain injury (Jovanovski, Erb & Zakzanis, 2005; Manning, Wanigaratne, Best, Strathdee, Schrover & Gossop, 2007).

• Participants with a current history of treatment for psychiatric conditions or mood disorders were excluded. Disorders such as schizophrenia and depression are thought to have implications for neurological involvement with marked behavioural correlates (Clegg, Brurnfitt, Parks & Woodruff, 2007). Depression in particular is a significant variable in this population and may result from a premorbid mood disorder, medication, brain damage or psychological reaction to the stress associated with acquiring communication as well as other possible cognitive and physical impairments (Murray & Chapey, 2001).

• Individuals with swallowing disorders were excluded from the study as participants were required to swallow whole capsules of relatively large size during drug and placebo phases.

5.4.2 Selection criteria for non-stroke participants

The study recruited seven individuals who had not experienced a stroke who served as controls for the pen and paper tasks, for participants who were using their non-dominant hands. These individuals were recruited from the local community in which the researcher lives. Each control
was matched according to sex, age, professional and work experience, handedness and education. As three of the participants with stroke, Cecil, John and Tumi used their dominant hands, matched controls were not recruited for them.

5.4.2.1 Controlling for use of the non-dominant hand

In the control study the seven non-neurologically impaired individuals performed the paper and pen tasks included on the executive battery, namely: Complex figures – copy and recall, Trail Making A and B, Five point test and Design fluency. Results for performance on trials for dominant and non-dominant hand responding were then subjected to a matched t-sample test to reveal the presence of significant differences. An important advantage of this procedure is that it avoids problems associated with variability from participant to participant and allows for control of extraneous variables (Howell, 1999). However, it too carries the disadvantage of carry-over effects where experience with the test material may automatically mean improved performance (Howell, 1999). In order to combat this, controls were randomly assigned to a first trial, whereby the order in which they completed the tasks (dominant or non-dominant hand first) was randomly assigned. Appendices H and I present the information sheet and consent forms for the control participants.

Results of the matched t-samples are presented in Appendix J in tables J.1 – J.6. Without exception there were no significant differences between controls’ results for dominant as opposed to non-dominant hand responding. This suggests that the stroke participants who used their non-dominant hands for responding would not be significantly penalized as has been suggested may happen (Keil & Kaszniak, 2002).

A more interesting finding was that there was a tendency (though not a statistically significant one) for the control participants to score higher on the second presentation of the test, regardless of which hand was used in the second trial. The implication is that in the non-neurogenically impaired sample, participants showed evidence of practice effects to a greater extent than their neurologically impaired matches. It appears that the control participants had a tendency to adopt different strategies and produce more effective responses the second time around. This was contrasted with the stroke participants who generally did not display practice effects to the same extent (with the exception of the Tower of London), suggesting decreased learning and adaptation ability accompanying neurological impairment.
### Table 5.3 – Participant characteristics

<table>
<thead>
<tr>
<th>PARTICIPANT</th>
<th>AGE</th>
<th>TIME SINCE ONSET</th>
<th>SITE OF LESION</th>
<th>HANDEDNESS</th>
<th>ACCOMPANYING MEDICAL CONDITIONS</th>
<th>MEDICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cecil</td>
<td>67</td>
<td>3 years</td>
<td>Left postero-inferior parietal cortex</td>
<td>Right</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Felicity</td>
<td>67</td>
<td>5 years</td>
<td>Right temporo-parietal cortex</td>
<td>Left</td>
<td>Atrial fibrillation Hypercholesterolaemic Hypertensive</td>
<td>Atacand (high blood pressure) Cordarone (antiirrhythmic) Lipitor (high cholesterol) Warfarin (anticoagulant)</td>
</tr>
<tr>
<td>Grace</td>
<td>57</td>
<td>6 years</td>
<td>Left fronto-parietal cortex</td>
<td>Right</td>
<td>Arteriosclerosis</td>
<td>Disprin (anticoagulant) Premarin (hormone replacement) Provera (hormone replacement)</td>
</tr>
<tr>
<td>Jane*</td>
<td>53</td>
<td>6 years</td>
<td>Right fronto-temporal cortex</td>
<td>Left</td>
<td>Epilepsy</td>
<td>Epanutin (anticonvulsant) Premarin (hormone replacement)</td>
</tr>
<tr>
<td>Jeannette</td>
<td>60</td>
<td>5 years</td>
<td>Left fronto-parietal cortex</td>
<td>Right</td>
<td>Epilepsy Hyperintensive</td>
<td>Disprin (anticoagulant) Epinutin (anticonvulsant) Prazosin (high blood pressure) Renitec (high blood pressure) Zocor (lipid altering agent)</td>
</tr>
<tr>
<td>John</td>
<td>40</td>
<td>8 years</td>
<td>Left fronto-parietal cortex</td>
<td>Right</td>
<td>Epilepsy</td>
<td>Phenytoin (anticonvulsant)</td>
</tr>
<tr>
<td>Margaret</td>
<td>64</td>
<td>3 years</td>
<td>Left postero-parietal cortex</td>
<td>Right</td>
<td>Hypercholesterolaemic Hypertensive</td>
<td>Ado Sinvastat (high cholesterol) Disprin (anticoagulant) Prexum (hypertension) Zopiclone (sleeping tablet)</td>
</tr>
<tr>
<td>Mel</td>
<td>59</td>
<td>5 years</td>
<td>Left fronto-parietal cortex</td>
<td>Right</td>
<td>Hypercholesterolaemic</td>
<td>Ecotrin (anticoagulant) Neurobion (multivitamin) Lipitor (high cholesterol) Imovane (sleeping tablet)</td>
</tr>
<tr>
<td>Paul*</td>
<td>63</td>
<td>1 year</td>
<td>Left fronto-parietal lobe</td>
<td>Right</td>
<td>Diabetic Hypertensive</td>
<td>Diamicron (hypoglycemic agent) Disprin (anticoagulant) Metformin (diabetes) Pharmapress (diuretic) Turbовite (multivitamin)</td>
</tr>
<tr>
<td>Tumi</td>
<td>54</td>
<td>17 years</td>
<td>Left temporo-parietal cortex</td>
<td>Right</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
Table 5.3 Participant characteristics cont…

<table>
<thead>
<tr>
<th>PARTICIPANT</th>
<th>HOME LANGUAGE</th>
<th>EDUCATION</th>
<th>QUALIFICATIONS</th>
<th>WORK</th>
<th>LIVING ARRANGEMENTS</th>
<th>COMMUNICATION SUMMARY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cecil</td>
<td>English</td>
<td>15 years</td>
<td></td>
<td>Car salesman</td>
<td>With wife in a house</td>
<td>Anomic aphasia</td>
</tr>
<tr>
<td>Felicity</td>
<td>English</td>
<td>18+ years</td>
<td>Nurse</td>
<td>Previously a nurse</td>
<td>With a full time caregiver in a cottage adjoining son’s property</td>
<td>Right Hemisphere Disorder (RHD)</td>
</tr>
<tr>
<td>Grace</td>
<td>English/Afrikaans</td>
<td>13 years</td>
<td>Secretarial course</td>
<td>Previously administrator for old aged home</td>
<td>With husband in a house</td>
<td>Conduction aphasia</td>
</tr>
<tr>
<td>Jane*</td>
<td>English</td>
<td>13 years</td>
<td>Secretarial course</td>
<td>Previously personal assistant in corporate company</td>
<td>Independent townhouse</td>
<td>Right Hemisphere Disorder (RHD)</td>
</tr>
<tr>
<td>Jeannette</td>
<td>English</td>
<td>15 years</td>
<td>BA English</td>
<td>Previously mechanic</td>
<td>Home for the aged</td>
<td>Conduction aphasia</td>
</tr>
<tr>
<td>John</td>
<td>English</td>
<td>18+ years</td>
<td>BAS (Bachelor of Architectural studies) Master of Architecture</td>
<td>Previously architect</td>
<td>With wife, daughter and father in a house</td>
<td>Broca’s aphasia</td>
</tr>
<tr>
<td>Margaret</td>
<td>English</td>
<td>15+ years</td>
<td>Book-keeping diploma</td>
<td>Previously book keeper</td>
<td>Independent in retirement village</td>
<td>Conduction aphasia</td>
</tr>
<tr>
<td>Mel</td>
<td>English</td>
<td>18+ years</td>
<td>BA English and Art Counseling diploma Marketing Degree</td>
<td>Previously art teacher</td>
<td>Home for the aged</td>
<td>Anomic aphasia</td>
</tr>
<tr>
<td>Paul*</td>
<td>English</td>
<td>18+ years</td>
<td>BCom</td>
<td>Previously managed import/exports for mining company</td>
<td>With wife in a house</td>
<td>Broca’s aphasia</td>
</tr>
<tr>
<td>Tumi</td>
<td>Tswana, Zulu, Shangaan, Xhosa, English, Afrikaans, Sotho, French, Russian</td>
<td>12+ years</td>
<td>Interrupted tertiary education</td>
<td>Studied anthropology and journalism Did not complete degree due to political turmoil and interruptions</td>
<td>Currently political activist Previously poet</td>
<td>With husband in a flat Anomic aphasia</td>
</tr>
</tbody>
</table>
5.4.3 Participant characteristics

The ten participants who had experienced a stroke are represented in Table 5.3 above where their relevant characteristics are listed or described. It should be noted that the study recruited eight individuals with aphasia and two with Right Hemisphere Disorder (RHD). In addition, eight of the participants were monolingual, while two were bi/multilingual preceding their stroke. Following her stroke, Grace only spoke English, while Tumi continued to speak several South African languages, with variable fluency. She reported that she could no longer speaker French or Russian at all – in her words “gone completely”, the languages she acquired most recently.

5.5 PROCEDURES

5.5.1 Data Collection

Data was collected over a period of 18 months; four times for each participant – at baseline, active, placebo and withdrawal phases. There was a minimum period of 1 month between active, placebo and withdrawal phases and therefore each participant’s involvement lasted between 3 and 6 months, depending on the length of time between baseline testing and participation in the first phase (either drug or placebo), as well as the duration of the withdrawal period which was determined for the participants’ convenience. This is depicted diagrammatically below. Some intervals were longer due to participants traveling or unrelated illness. Data was always collected in the participants’ homes.

<table>
<thead>
<tr>
<th>Baseline</th>
<th>Phase 1 (active / placebo) 1 month duration</th>
<th>Phase 2 (placebo / active) 1 month duration</th>
<th>Withdrawal 1 month duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Testing 1</td>
<td>Testing 2</td>
<td>Testing 3</td>
<td>Testing 4</td>
</tr>
</tbody>
</table>

5.5.1.1 Language Data

Language profiles were obtained from performance on the WAB (Kertesz, 1982). Testing was always conducted in the participants’ homes under relatively quiet conditions, in well lit rooms and with breaks provided when deemed beneficial or whenever requested by a participant.
5.5.1.2 Executive Functioning Data

EF testing took place at a time convenient for the participants. As the battery was relatively short, breaks in testing were offered and provided when requested, although they were not usually necessary. The testing environment was the same as for language testing.

5.5.1.3 Conversational Data

Where the participant used a family member or friend for the recording of their conversations, times suitable for them were identified. As far as possible, these interactions were “naturally occurring” i.e. they captured conversations that would have taken place with or without the camera being present as recommended by Ten Have (1999). In some instances however it was impossible to find a partner willing to be video-taped and in such cases, the researcher took on the role of interactant. Grbich (1999) describes this role as the ‘researcher-participant/participant-researcher’. In such cases, the researcher has the flexibility to switch quickly from role to role in the environment and his/her time can be divided roughly equally between the two roles. In this research, participation involved the researcher in interactions with people whose values, actions and behaviours they shared to some extent. In developing an emotional commitment to these concerns the researcher could access a secure and unthreatened (and unthreatening) position (Grbich, 1999).

In both cases, a fixed camera on a tripod was set up in advance of the interaction, positioned to maximally capture all aspects of the interaction from which to derive a complete transcript (including verbal and non-verbal information such as speech, eye gaze, gesture and manipulation of objects). A stable image is preferred as it is more comfortable to look at especially when data needs to be viewed repeatedly (Ten Have, 1999). Recordings were made of approximately thirty minutes of interaction. Ten minutes of these interactions, generally from the middle segments of the recordings were then chosen for analysis (Ten Have, 1999).

5.6 THE RESEARCH BATTERY

5.6.1 Test to Assess Language Functioning

Each of the participants completed the Western Aphasia Battery (Kertesz, 1982). This aphasia test is widely used as it provides a detailed profile of abilities in the four significant areas that are diagnostically sensitive to identifying type and severity of aphasia namely: spontaneous speech, comprehension, repetition and naming. In addition to this, the battery assesses the
ability to perform voluntary movements as well as reading, writing, visuo-constructional ability and calculation. The test is highly structured and proceeds from the simple to the complex. When scored the test provides three summary scores a) an Aphasia Quotient (AQ) derived from performance on the oral language subtests, b) a Language Quotient (LQ) derived from performance on all the language subtests (oral as well as reading and writing subtests) and c) a Cortical Quotient (CQ), derived from performance on all the subtests (Murray & Chapey, 2001).

5.6.2 Tests to Assess Executive Functioning
In the pilot study, a list of constructs considered to tap EF was generated and tests were selected on the basis of whether or not they were felt to assess that construct as well as their suitability for administration to individuals who had experienced a stroke. However for the purposes of this research, a coherent and unifying theory that could operationalize the constructs under discussion and more importantly describe their relationships to one another, was sought. With reference to Barkley’s model therefore, some of the constructs originally presented in Frankel, Penn and Ormond-Brown (2007) have been collapsed into the constructs represented in the Barkley model as indicated by Table 5.4 on the following page.

5.6.3 Data Analysis
5.6.3.1 Analysis of language and executive data
The amount of data generated was considerable and it was clear that not all of it could be presented. Individual profiles were drawn up by scoring test performance on the WAB and executive battery. These profiles reflected individual strengths and deficits in terms of language and EF using baseline data. Baseline data was chosen as it was felt that the truest executive profile would be obtained in the initial phase owing to the fact that executive functions are taxed during novel activity, a variable which may have been compromised in subsequent testing sessions, at least to some extent. Conversational data was analyzed separately. Conversational, language and EF profiles were then compared to find meaningful associations to inform the descriptive phase of the study. EF results from the remaining phases of the study were scored and subjected to a repeated measures analysis of variance to determine significant changes in performance for each participant across the four phases of the study.
Table 5.4 - Constructs and Tests of the Executive Functioning Battery

<table>
<thead>
<tr>
<th>Construct</th>
<th>Tests</th>
<th>Barkley’s constructs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple Attention</td>
<td>Digit Span (forward) (Lezak, Howieson, Loring, Hannay &amp; Fischer, 2004)</td>
<td>Behavourial inhibition</td>
</tr>
<tr>
<td>Complex attention</td>
<td>a) Stroop Colour-Word Interference Test* (Golden, 1978)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>b) Trail Making* (Lezak et al., 2004).</td>
<td></td>
</tr>
<tr>
<td>Nonverbal working memory</td>
<td>a) Self Ordered Pointing Test* (Spren &amp; Strauss, 1998).</td>
<td>Working memory</td>
</tr>
<tr>
<td></td>
<td>b) Wisconsin Card Sorting Test (Ormond Software Enterprises, 1999)</td>
<td></td>
</tr>
<tr>
<td>Memory</td>
<td>Medical College of Georgia complex figures* (Spreen &amp; Strauss, 1998)</td>
<td></td>
</tr>
<tr>
<td>Verbal working memory</td>
<td>Digit Span (backwards) (Lezak et al., 2004)</td>
<td></td>
</tr>
<tr>
<td>Regulation of affect</td>
<td>Neuropsychology Behaviour and Affect Profile</td>
<td>Regulation of affect</td>
</tr>
<tr>
<td></td>
<td>(Nelson, Satz &amp; D’Elia, 1994)</td>
<td></td>
</tr>
<tr>
<td>Planning / scheduling / strategy use / rule adherence/ problem solving</td>
<td>Tower of London* (Shallice 1982)</td>
<td>Internalization of speech</td>
</tr>
<tr>
<td>Concept formation / abstract reasoning</td>
<td>Raven’s Progressive Matrices* (Raven, Raven and Court, 1998)</td>
<td></td>
</tr>
<tr>
<td>Generation / fluency / initiation</td>
<td>a) Five Point Test (Spren &amp; Strauss, 1998)</td>
<td>Reconstitution</td>
</tr>
<tr>
<td></td>
<td>b) Design Fluency (Spren &amp; Strauss, 1998)</td>
<td></td>
</tr>
</tbody>
</table>

* Parallel versions used

5.6.3.2 Conversation Analysis

CA has an established history of use in the field of speech language pathology. Its applicability in describing and highlighting a multitude of communication features and characteristics is highly relevant and described in Chapter 2. The ten minute segments were viewed in detail and transcribed using multiple layers. According to Ten Have (1999), an analyst should make his or her own transcriptions as it is this process that provides access to ‘the “lived reality” of the interaction…’ (Ten Have, 1999, p. 77). As a result of the fact that the researcher is forced to notice details not obviously evident to the listener, transcription works as a “noticing device” (Ten Have, 1999, p. 78). Specific guidelines compiled by Ten Have from the work of Psathas and Anderson (1990) were followed.

1. The time, date and place of the original recording were noted.
2. Participants are identified in the left column by a letter code.
3. The words spoken by each of the participants were transcribed in standard orthography
4. Sounds as uttered are then transcribed including vocal sounds, inhalation, exhalation and laughter. These are included as they are considered to have interactional meaning and contribute to a picture of the rhythm of talk.
5. Inaudible or incomprehensible words are also transcribed to the best guess, in single brackets.
6. Pauses or silences are noted. An informal beat count as a proximate measure of ‘rhythm-sensitive’ pause length was used as described by Jefferson (1989).
7. Overlapped speech and sounds are noted.
8. Psathas and Anderson (1990) have described conventions for elaborating the process rather than the content of talk. These include the noting of latching, cut off word, stretching of words, stress of the word or part thereof, volume markers, and intonation. These are all noted.

Transcription conventions as derived from Jefferson (2004) and Lesser and Milroy (1993) are included in Appendix K.

Once the data had been transcribed, transcripts were analyzed initially in a broad manner in terms of turn taking, topic management and repair. These three features were chosen as they have largely been acknowledged to form the basic tenets of conversational ability in that they are present throughout the entire conversation and are likely to set the context for more micro-level behaviours (Lesser & Perkins, 1999). Furthermore, there is a wide range of research on typical and expected performance in relation to these three components, which made analysis and structuring of an enormous data corpus manageable. Conversational characteristics and summarizing comments were made for each of the ten participants and included in their individual profiles in terms of which conversational structures were generally managed and which were not. The baseline data were then compared to the executive profiles and evaluated for meaningful associations. Transcripts for all the remaining phases of the study were also prepared and examined to determine changes in turn taking, topic management and repair characteristics over the four phases of the study.

5.6.4 **Data confirmability**

There are numerous issues surrounding the truth value of qualitative research. When engaged in positivistic investigative enterprises, specific statistical procedures exist to verify aspects of the
data. However, these methods are not appropriate for use with data of a qualitative nature. Although CA is a methodology that it not usually submitted to any form of confirmability or verification procedure, this is a shortcoming when the data and interpretations generated are used to inform theory and clinical practice.

The omission of any form of reliability checks when using CA as a methodology is an issue that represents the heart of the debate between researchers in positivistic (quantitative) fields and those in qualitative fields, with the former being critical of qualitative research, labeling it unscientific, exploratory, unreliable and impressionistic (Guba & Lincoln, 1994). Conversely qualitative researchers criticize positivistic investigators as attempting to promote one version of truth over another. They maintain that quantitative researchers are seldom able to capture individuals’ perspectives because they rely on remote, inferential materials. Quantitative research is seen as stripping findings of their applicability as a result of exclusionary designs that detract from the relevance of the conclusions reached (Guba & Lincoln, 1994).

Qualitative researchers using CA have defended their wariness of quantification by demonstrating that evidence arising from the sequential context of conversation is compromised if items are removed for counting. The collaborative nature of interaction is at risk of being lost if actions of interlocutors are separated from one another (Schegloff, 1993). Perkins (1995), for example, looked at the judicious use of quantification used in conjunction with CA and concluded that quantification of conversational phenomena can be useful, provided it is interpreted with the findings of the qualitative analysis.

Significantly though, when SLPs use CA with a view to description, assessment and management planning, the clinical and therapeutic implications demand that our observations be verifiable (Hux, Sanger, Ried & Maschka, 1997). Accountability to our clients and to the profession is paramount if we are to continue to uphold speech pathology as a science. Pathology must be accurately described and diagnosed and a common language and purpose between clinicians i.e. concurrent validity among therapists, must be preserved.

How to go about verifying interpretations made on the basis of CA transcripts appears to at least thus far, represent a battle of territory between qualitative and quantitative researchers. Yet, the disagreement about marrying qualitative and quantitative measures when employing CA seems
to be unwarranted. The field of qualitative research provides numerous approaches, which if adopted can be effective in establishing the truth value of a study. Morse (1994) maintains that qualitative and quantitative research do not necessarily represent two contrary research paradigms, as some researchers believe (Henwood, 1996; Neuman, 1994). The inclusion of both approaches speaks to the effort to ensure transferability in a research paradigm that is in fact mindful of issues of authenticity. Denzin and Lincoln (1994) point out that qualitative research refers to an approach that is essentially multi-method in focus and that within the qualitative paradigm, specific criteria that define the soundness or ‘truth value’ of qualitative research already exist. Marshall and Rossman (1995) provide the following guidelines. The terms ‘credibility’, ‘transferability’, ‘dependability’ and ‘confirmability’ replace the positivistic terms of ‘validity’, ‘reliability’ and ‘objectivity’. Table L.1 in Appendix L presents a summary of the qualitative concepts that are significant in establishing the ‘truth value’ of a study, their quantitative correlates and procedures for their implementation.

‘Investigator triangulation’ is a method used to ensure the objectivity or rather the confirmability of the findings. It should be noted that this method has been criticized on the basis that it is unrealistic to expect an external rater to have the same insight into the material from a limited and truncated version of the data as the primary investigator, and therefore it is not recommended by some authors (Morse, 1994). However it is proposed that this form of triangulation has the ability to provide a measure of the degree of rater concordance, which in turn informs the accuracy with which the data has been interpreted.

Furthermore, there is a theoretical and testable basis for utilizing this method of verification. In his writings on neo-phenomenology Langewitz (2007) highlights the importance of the meaning derived from ‘situations’ as opposed to constellations’. Situations are referred to as interactions in which meaning is diffused. Communication is viewed as an immersion of two individuals in a common or shared situation in which the sensations of the lived body provides evidence of the inter-relationship between the participants and informs the way in which they interact with one another as well as their experience of one another. This meaning is derived from non specific ‘atmosphere’ or impressions which are experienced in authentic ways. This notion is differentiated from constellations referring to meaning that is attached to single constituents within an interaction. Langewitz (2007) provides data showing how immediate impressions enable physicians to predict (accurately) how long patients will need in order to convey their
complaints. In a similar logic, the submission of conversational data to an experienced clinician for verification, allows for a third party to observe aspects of the interaction that while they may be difficult to capture in specifically delineated components, still provide an authentic sense of the interaction. These global perceptions have every possibility of being valid judgments on the significance of variables under scrutiny, which provide a form of triangulation and consensus in a methodologically relevant way.

Ten interactions were selected – a sample which contained one interaction for each participant, randomly drawn from the four phases of the study. These interactions were then divided into three groups. The first contained only video recorded material of the interactions. The second contained only the CA transcripts and the third group contained both sets of data. Each data set was then submitted to a different rater. The first set containing only video material was given to an experienced speech language pathologist with a PhD in the field of adult neurogenic communication disorders and extensive clinical experience. The second set (the CA transcripts) was given to a postgraduate linguistics student with a particular interest in CA research. The final set (video and transcripts) was given to a speech language pathologist with experience in treating individuals with neurogenic communication disorders as well as extensive research experience using CA as a methodology. Each of the three raters were asked to analyze their data sets according to broad guidelines relating to specific conversational constructs including turn taking, topic management and conversational repair. Additional comments were invited and a general impression or overriding comment was requested. (The rater information sheet as well as the response form is found in Appendix M). These three perspectives were then compared to the primary researcher’s findings and measured for the degree of concordance.

The comments of the three external raters were compared with the judgments of the researcher to assess the degree of concordance. In the pages that follow, Table 5.5 summarises interpretations for turn-taking, Table 5.6 presents topic management and 5.7 presents repair data.

Interpretations of the primary researcher were most similar to the rater with access to both video material and completed CA transcriptions. The clinician who rated the videos also made interpretations that were for the most part consistent with the researcher’s although they were not as detailed and excluded aspects provided by the rater who viewed both videos and
transcripts. There were occasions when her interpretation differed completely (e.g. for turn taking, she rated Felicity’s turn taking skills as intact, whereas the researcher and third rater stated that her turns were overly long and that she was prone to interrupting). Finally, the least agreement was found between the researcher and the second rater (transcripts only), who often stated that a feature was intact when the other three commented on aspects that were impaired.

This methodological study has some significant implications. The first is that analysis was for the most part confirmed by external raters, adding authenticity to the interpretations made. Second, it was clear that the most reliable rater was the one who had access to both the videos and transcripts. This is important as it suggests that analysis of documented transcripts alone may not be sufficient for accurate and complete assessment. Furthermore, the rater who analysed the transcripts was not a speech language pathologist but a linguist with a particular interest in CA research. Therefore, clinical insight and experience may also play a significant role in the diagnosis and evaluation of which conversational features facilitate or impede successful and meaningful communication. Finally, while the rater who viewed the videos was often in agreement with both the researcher and the third rater (videos and transcripts), there were times when she was not. In addition, her descriptions lacked the same detail that characterized the comments of the researcher and the third rater. This suggests that CA when used in combination with clinical interactions is a more powerful tool and can convey more diagnostically meaningful information than observation alone.

This chapter has presented the aims, general methodology and procedures of the study. Within the scope of the larger study, four smaller studies were conducted. First was a study piloting the use of the EF battery and its integration with conversational data. Second, was the study using controls who were assessed on all the paper and pen tasks of the EF battery using both their dominant and non dominant hands to determine if testing was significantly biased in favour of the dominant hand. Third, an ethical study was initiated assessing the facilitators and barriers to obtaining true informed consent. Finally, a methodological study, triangulating analysis of the conversational data, was conducted. The results of these three studies were described briefly with references provided for material already prepared for publication.

This concludes the chapter on methodology. The following chapter presents the results and discussion of the study.
Table 5.5 – Comparison of judgments regarding turn-taking

<table>
<thead>
<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>PRIMARY RESEARCHER</th>
<th>RATER 1 VIDEOS</th>
<th>RATER 2 TRANSCRIPTS</th>
<th>RATER 3 VIDEO AND TRANSCRIPTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>Extended turns</td>
<td>Protracted turn length</td>
<td>Some interruptions mostly intact</td>
<td>Interruptions Extended turns</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Some interruptions</td>
<td>Unequal turn distribution</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>Intact</td>
<td>Preserved</td>
<td>Intact</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>Intact</td>
<td>Preserved</td>
<td>Intact</td>
<td>Intact</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>Sometimes delayed Length reduced</td>
<td>Preserved</td>
<td>Short, uninformative does not anticipate</td>
<td>Delayed reduced length</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>Intact</td>
<td>Preserved</td>
<td>Anticipates and takes turn but can not convey meaningful information</td>
<td>Takes turns appropriately but can not utilize the turn space</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>Intact</td>
<td>Preserved</td>
<td>Intact</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>Intact</td>
<td>Preserved</td>
<td>Intact but too short</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>Extended turns</td>
<td>Preserved</td>
<td>Intact</td>
<td>Extended turns</td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>Extended turns</td>
<td>Preserved</td>
<td>Intact</td>
<td>Interrupts, does not always take turn promptly, verbose</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>Intact</td>
<td>Preserved</td>
<td>Intact</td>
<td>Intact</td>
</tr>
</tbody>
</table>
Table 5.6 – Comparison of judgments regarding topic management

<table>
<thead>
<tr>
<th>Aphasia Type</th>
<th>Participant</th>
<th>Primary Researcher</th>
<th>Rater 1 Videos</th>
<th>Rater 2 Transcripts</th>
<th>Rater 3 Video and Transcripts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>Initiates but shifts are fragmented</td>
<td>Can initiate, difficulty sustaining</td>
<td>Maintains current topic</td>
<td>Does not stay on topic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Does not maintain topic – distractible</td>
<td></td>
<td></td>
<td>Initiates ideas but strays from discussion</td>
</tr>
<tr>
<td>Mel</td>
<td>Can initiate</td>
<td>Initiates topics to shift – does not always process information – can be repetitive</td>
<td></td>
<td>Does not initiate but contributes to shift</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Does not engage in topic initiation or shift</td>
<td></td>
<td></td>
<td>Some perseveration</td>
</tr>
<tr>
<td>Tumi</td>
<td>Initiates, contributes logically and coherently to shift</td>
<td>Does not engage in topic initiation or shift</td>
<td></td>
<td>Contributes to shift and can initiate</td>
<td>Intact</td>
</tr>
<tr>
<td>Broca’s</td>
<td>Limited initiation</td>
<td>Attempts to sustain topic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>John</td>
<td>Strict limitations to shift contribution</td>
<td>Does not initiate or shift</td>
<td>Does not contribute meaningfully</td>
<td>Does not initiate can contribute but minimally with reduced output</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>Impaired completely</td>
<td>Impaired on all levels</td>
<td>Does not contribute</td>
<td>Impaired</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>can not shift</td>
<td></td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>Good initiation and contribution to shift</td>
<td>Preserved on all levels</td>
<td>Initiates and contributes to shift</td>
<td>Intact</td>
</tr>
<tr>
<td>Jeannette</td>
<td>Initiates topics</td>
<td>Difficulty with initiation</td>
<td>Can initiate but poor contribution to shift</td>
<td>Intact</td>
<td></td>
</tr>
<tr>
<td>Margaret</td>
<td>Initiates topics, does not retain organization. Often incoherent and tangential makes irrelevant statements</td>
<td>Preserved</td>
<td>Intact</td>
<td>Initiates many topics</td>
<td></td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>Does not initiate and has tendency to be tangential and distractible</td>
<td>Maintains current topic</td>
<td>Can initiate, difficulty sustaining</td>
<td>Does not stay on topic</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>Initiates many topics but fragmented, does not maintain topic, disorganized and often incoherent</td>
<td>Initiates topics but lacks coherence</td>
<td>Intact</td>
<td>Fails to initiate topics at appropriate places</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Can initiate but lacks organization , not always coherent, disjointed</td>
</tr>
<tr>
<td>Aphasia Type</td>
<td>Participant</td>
<td>Primary Researcher</td>
<td>Rater 1 Videos</td>
<td>Rater 2 Transcripts</td>
<td>Rater 3 Video and Transcripts</td>
</tr>
<tr>
<td>--------------</td>
<td>-------------</td>
<td>--------------------</td>
<td>---------------</td>
<td>--------------------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>Can initiate self repair or invite other repair Sometimes does not repair when interlocutor initiates repair on his utterance</td>
<td>Utilizes phonological self repair and word repetition Multiple attempts</td>
<td>Initiates other repair</td>
<td>Can self correct Assisted by wife Does not always respond to requests for clarification</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>Struggles with this initiates repair, but perseverative limited strategies gives up.</td>
<td>Multiple self repair revisions perseveres with repair</td>
<td>Mostly smoothly</td>
<td>Attempts repair not always successful then tends not to persevere Does not use alternate strategies</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>Intact – uses a number of strategies</td>
<td>Uses repetition</td>
<td>Intact</td>
<td>Intact</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>Limited, does not often initiate, can not use alternate strategy does not collaborate - passive</td>
<td>Impaired, does not initiate strategies ambiguous and unhelpful</td>
<td>Some repair evident with repetition for emphasis</td>
<td>Does not initiate repair strategy, relies totally on interlocutor</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>Multiple attempts but perseverates Does not make use of alternate techniques</td>
<td>Makes attempts but unsuccessful</td>
<td>Struggles to use new strategies, attempts repair but not at all successful</td>
<td>Can not initiate repair strategies Perseverative</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>Intact makes use of multiple repair strategies. Good use of shared repair with interlocutor</td>
<td>Uses several strategies to self repair</td>
<td>Struggles with repair requires assistance from interlocutor</td>
<td>Initiates self repair, makes use of a number of strategies collaborates well with husband</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>Can not use different strategies reliant on book but this is often unhelpful reliant on interlocutor</td>
<td>Self repair impaired Relies on collaboration</td>
<td>Does not initiate strategies or provide clear feedback</td>
<td>Does not initiate repair strategy Uses diary but largely depends on conversational partner</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>Intact, invites shared repair</td>
<td>Self repair intact</td>
<td>Intact</td>
<td>Intact</td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>Intact</td>
<td>Self repair intact</td>
<td>Intact</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>Intact</td>
<td>Intact</td>
<td>Intact</td>
<td>Intact</td>
</tr>
</tbody>
</table>
CHAPTER SIX

RESULTS AND DISCUSSION

The previous chapter described the methodology for this study. This chapter presents the results of both the descriptive and experimental phases of the research. The preliminary investigation which involved the piloting of the executive battery and its incorporation with conversational data was referred to in the previous chapter.

6.1 SUMMARY OF THE FINDINGS
The most prominent finding of the descriptive phase of this study was that most of the participants presented with significant EF deficits. As predicted, the formal language tests results were not consistently related to the profiles of EF deficits observed across the group. However, EF deficits appeared to be more consistently related to conversational skills as revealed by the CA, confirming the importance of EF skills to conversational intelligence. Specifically, the descriptive phase of the study revealed that impaired inhibition was associated with talkativeness and perseveration. Nonverbal working memory impairments as well as poor problem solving / internalization of speech were associated with organizational and structural impairments in conversation. Difficulties with reconstitution were also associated with difficulty contributing novel and creative responses in conversation although this relationship appeared to be somewhat mediated by behavioural inhibition factors. The two participants with the most intact EF profiles showed the most preserved conversational intelligence and these participants also happened to be the only two participants in the sample who were bilingual. The potential significance of this finding is discussed tentatively below. Poor response inhibition often appeared to have a cascading effect on other EF including working memory, problem-solving and reconstitution. Similar EF profiles manifested variable and unique conversational profiles across different participants, precluding a simple relationship between discrete EF skills and conversation. Findings for each of the participants did not appear to be influenced by the number of interlocutors they had, suggesting that changes observed in conversational skill, could not be attributed to a change in conversational partner. All three participants who showed qualitative changes in their conversations over the course of the study were individuals who had only one interlocutor over all four phases of the research.
In the experimental phase, quantitative findings were disappointing with no statistically significant patterns of improvement being observed in the active phase of the trial. However, qualitative analysis provided some examples of the possible effect of LEV on EF and conversational skills in a subset of the participant sample. These findings are summarized in a model of EF and conversational skills.

6.2 DESCRIPTIVE PHASE OF THE STUDY
Comprehensive profiles including language, executive functions (EF) and conversation characteristics were compiled for each participant. These profiles used data obtained during the baseline phase of testing. Baseline data was chosen as it was likely to produce the purest profile in terms of the EF data. This is because EF tests should present novel tasks and require the participant to perform non-routine activity. In subsequent test phases, although provisions were made to combat carry-over effects (the order of testing was changed and parallel versions were used where tests have known practice effects), it was still plausible that some carry-over effects would be present. As will be shown later, this was subsequently shown to be true for one of the tests in the EF battery (Tower of London).

6.2.1 Language results
The WAB (Kertesz, 1982) was administered to all of the participants with stroke. Of the ten, three participants were classified as having anomic aphasia, four had conduction aphasia, two had Broca’s aphasia and one participant who had a right hemisphere stroke presented as non-aphasic. Table 6.1 below presents the scores for the ten participants on the following sub-tests: spontaneous speech, comprehension, repetition, naming, reading and writing, praxis, drawing, block design and calculation. (The Raven’s Coloured Matrices was not administered as the Raven’s Progressive Matrices was administered as part of the EF battery).
Table 6.1 – Language, praxis, construction and calculation scores for the Western Aphasia Battery

<table>
<thead>
<tr>
<th>PARTICIPANT</th>
<th>SPONTANEOUS SPEECH</th>
<th>COMPREHENSION</th>
<th>REPETITION</th>
<th>NAMING</th>
<th>READING</th>
<th>WRITING</th>
<th>PRAXIS</th>
<th>DRAWING</th>
<th>BLOCK DESIGN</th>
<th>CALCULATION</th>
<th>APHASIA TYPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cecil</td>
<td>18/20</td>
<td>8.3/10</td>
<td>8/10</td>
<td>8.7/10</td>
<td>100/100</td>
<td>85/100</td>
<td>60/60</td>
<td>26/30</td>
<td>8/9</td>
<td>24/24</td>
<td>Anomic</td>
</tr>
<tr>
<td>Felicity</td>
<td>20/20</td>
<td>10/10</td>
<td>10/10</td>
<td>9.2/10</td>
<td>100/100</td>
<td>96/100</td>
<td>60/60</td>
<td>19/30</td>
<td>9/9</td>
<td>24/24</td>
<td>RHD#</td>
</tr>
<tr>
<td>Grace</td>
<td>14/20</td>
<td>8.2/10</td>
<td>6.7/10</td>
<td>8.6/10</td>
<td>76/100</td>
<td>74/100</td>
<td>46/60</td>
<td>26/30</td>
<td>9/9</td>
<td>24/24</td>
<td>Conduction</td>
</tr>
<tr>
<td>Jane*</td>
<td>18/20</td>
<td>9.4/10</td>
<td>6.9/10</td>
<td>9.7/10</td>
<td>100/100</td>
<td>93/100</td>
<td>58/60</td>
<td>22/30</td>
<td>9/9</td>
<td>24/24</td>
<td>RHD#</td>
</tr>
<tr>
<td>Jeannette</td>
<td>13/20</td>
<td>7.45/10</td>
<td>5/10</td>
<td>7.5/10</td>
<td>42/100</td>
<td>48.5/100</td>
<td>40/60</td>
<td>27/30</td>
<td>8/9</td>
<td>18/24</td>
<td>Conduction</td>
</tr>
<tr>
<td>John</td>
<td>13/20</td>
<td>9.95/10</td>
<td>7.6/10</td>
<td>8.5/10</td>
<td>67/100</td>
<td>58/100</td>
<td>56/60</td>
<td>30/30</td>
<td>9/9</td>
<td>24/24</td>
<td>Broca’s</td>
</tr>
<tr>
<td>Margaret</td>
<td>17/20</td>
<td>8/10</td>
<td>6.9/10</td>
<td>8/10</td>
<td>84/100</td>
<td>72.5/100</td>
<td>42/60</td>
<td>24/30</td>
<td>8/9</td>
<td>24/24</td>
<td>Conduction</td>
</tr>
<tr>
<td>Mel</td>
<td>14/20</td>
<td>9.5/10</td>
<td>9.4/10</td>
<td>8.8/10</td>
<td>84/100</td>
<td>78/100</td>
<td>52/60</td>
<td>29.5/30</td>
<td>9/9</td>
<td>24/24</td>
<td>Anomic</td>
</tr>
<tr>
<td>Paul*</td>
<td>3/20</td>
<td>7.65/10</td>
<td>2.2/10</td>
<td>2.8/10</td>
<td>48/100</td>
<td>25.5/100</td>
<td>31/60</td>
<td>11/30</td>
<td>8/9</td>
<td>24/24</td>
<td>Broca’s</td>
</tr>
<tr>
<td>Tumi</td>
<td>13/20</td>
<td>7.9/10</td>
<td>8.4/10</td>
<td>7.1/10</td>
<td>64/100</td>
<td>60.5/100</td>
<td>58/60</td>
<td>22/30</td>
<td>9/9</td>
<td>18/24</td>
<td>Anomic</td>
</tr>
</tbody>
</table>

# RHD: Right Hemisphere Deficit
The three classical aphasia syndromes identified in this sample are well documented in the literature (Kearns, 2005). Their symptoms generally conformed to traditional characteristics as used for diagnostic purposes on the WAB, and which are in bold face below in Table 6.2 (a simplified version of the table presented in chapter 2).

Table 6.2 – Aphasia syndromes

<table>
<thead>
<tr>
<th></th>
<th>FLUENCY</th>
<th>COMPREHENSION</th>
<th>REPETITION</th>
<th>NAMING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonfluent</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Broca’s</strong></td>
<td>-</td>
<td>+</td>
<td>=</td>
<td>=</td>
</tr>
<tr>
<td>Global</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Transcortical motor</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>=</td>
</tr>
<tr>
<td>Fluent</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wernicke’s</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>=</td>
</tr>
<tr>
<td>Transcortical sensory</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>=</td>
</tr>
<tr>
<td><strong>Conduction</strong></td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>=</td>
</tr>
<tr>
<td><strong>Anomic</strong></td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

Key: (+) Relatively unimpaired; (-) Impaired; (=) Variable impairment across patients

Felicity and Jane* were two participants who did not present with aphasia and whose CVAs occurred in the right hemisphere, both manifested symptoms which are consistent with right hemisphere syndromes. Although according to strict scoring criteria, Jane*’s WAB profile characterized her as having conduction aphasia on the basis of relatively poor repetition scores, her performance on other language tasks as well as her discourse characteristics and site of lesion, did not support this diagnosis. Instead after considerable consultation, it was felt that her profile was more consistent with RHD. As most of these features manifested at a conversational level, they will be discussed below.

In the discussion that follows, the data are presented within the framework of Barkley’s theory of EF. In Chapter 4, the discussion presented research related to EF deficits in the presence of stroke and aphasia. In this section, the results are integrated with a discussion of relevant studies that draw associations between various EF deficits and corresponding language and communication difficulties. The sample size in this study was too small to allow for statistical analysis of correlations between EF and communication deficits and therefore associations noted are interpreted with caution in light of whether or not they confirm or contradict previous findings. Because there is very little previous research that has drawn parallels between EF and
conversational features in the stroke or aphasia literature, the conversational data are mostly presented with reference to the TBI and dementia literature where such precedence exists.

6.2.2 **EF and co-occurring communication deficits**

The results below are presented in terms of the five constructs assessed namely: behavioural inhibition, working memory, regulation of affect, internalization of speech and reconstitution. Each section is preceded by a brief definition of the construct under investigation. Results of the executive testing for the current study are then presented. Raw data obtained for each participant, during each of the four phases of the study, are presented in the Appendix N as indicated in each section. These results are discussed first in terms of language results with reference to previous findings in relevant literature, then in terms of the conversational findings and relevant literature where it exists. A summary section is then provided for each of the five constructs.

6.2.2.1 **Behavioural inhibition**

The term behavioural inhibition employed in Barkley’s model essentially describes the processes and functions encompassed by various forms of attention. Attention comprises a complex collection of abilities including: focused attention (the ability to attend to one stimulus in the absence of other distracting stimuli), selective attention (the ability to attend to one stimulus in the presence of distractors), alternating or shifting attention (the ability to alternate between tasks sets), divided attention (the ability to attend to multiple stimuli at one time) and sustained attention or vigilance (the ability to attend to one stimulus infrequently over extended periods of time) (Sohlberg & Mateer, 2001). Slowed information processing has also been shown to be an underlying cause of impaired attention (Ponsford & Kinsella, 1992; Van Zomeren & Brouwer, 1994).

The functional outcomes of disrupted attention processes will consistently result in impulsivity (the inability to inhibit a prepotent response), perseveration (the inability to shift focus or interrupt an ongoing behaviour) and distractibility (inability to resist interference) (Adamovich & Henderson, 1992, Pashler, 1998). Interference control includes focused, selective, divided and sustained attention, with its main role being the protection of the central system from distraction. Inhibition on the other hand is concerned with the ability to shift attention, by being
able to interrupt an ongoing stimulus and adapt to a new more appropriate one. Table 6.3 below presents the tasks utilized for this section of the EF testing.

Table 6.3 – Tests of Behavioral Inhibition

<table>
<thead>
<tr>
<th>CONSTRUCT – BEHAVIOURAL INHIBITION</th>
<th>TESTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interference control</td>
<td>Digit Span (forward) (Lezak et al., 2004)</td>
</tr>
<tr>
<td></td>
<td>Stroop Colour-Word Interference Test (Golden, 1978)</td>
</tr>
<tr>
<td>Response inhibition (shifting)</td>
<td>Trail Making (Lezak et al., 2004).</td>
</tr>
</tbody>
</table>

6.2.2.2 Interference control results

This refers to the control of interferences or distractions in pursuance of goal directed tasks (Barkley, 1997).

The Digit Span test is the most common format in use for measuring immediate attention span. It is closely related to the efficiency of attention or what is also called freedom from distractibility (Lezak et al., 2004). The ability to repeat a string of digits in serial order was preserved in only two of the ten participants – Mel (mild/anomic aphasia) and Felicity (RHD), suggesting that simple attention to verbal material was disrupted in the majority of this sample. This is an interesting finding as although poor repetition is anticipated in individuals with conduction aphasia, two of the participants with anomic aphasia and both of the individuals with Broca’s aphasia as well as Jane* (RHD) had poor scores on this task. Most significantly, results did not differ when the pointing version of the test was administered to the individuals with Apraxia. Appendix N.1 presents the raw scores for this test.

On the Stroop Colour-Word Interference Test six of the ten participants had difficulty as measured by ratios of errors made in trial 1 against errors made in trial 2 as presented in Appendix N.2. The participants who showed deficits in this area included two of the individuals with anomic aphasia (Cecil and Mel), one of the three with conduction aphasia (Margaret), one of the individuals with Broca’s aphasia (John) and both of those with RHD (Felicity and Jane*).
Interference control and language

As outlined in Chapter 4, there has been a recent surge of interest in complex attention factors and their potential impact on language and communication. These studies have for the most part utilized dual task paradigms in order to examine theories of resource allocation. Capacity theories of attention predict that impairments with dual tasks are the result of competition for the same limited capacity attentional resources (Kahneman, 1973; Wickens, 1984, 1989). When the amount of resources or task demands required, to complete both tasks simultaneously, exceeds attentional capacity, decrements or dual task interference will occur in performance.

The WAB measures spontaneous speech in response to questions and picture description, naming, comprehension, repetition, reading and writing. Each area will be addressed briefly in relation to interference control.

Spontaneous speech

The amount of content reflected in speech output as well as the fluency of this output contributes to the spontaneous speech score for the WAB and contributes to the classification of aphasia type. The majority of the participants were therefore classified as having fluent output while only the two individuals with Broca’s aphasia were considered non-fluent. Difficulties with content (which essentially reflects a naming component) as well as decreased length, accuracy and completeness of grammatical structures contributed to lower scores for the non-fluent speakers. Nonetheless, qualitative assessment as based on conversational interactions indicated that even in the presence of relatively intact language scores, discourse was compromised. These difficulties could be attributed to a number of factors, among them response interference deficits, which predominantly manifested as distractibility and impulsivity. These features were clearly evident in the conversational output of those individuals with poor scores on the Stroop Colour-Word Interference Test and discussed more fully under the section detailing conversation findings.

These findings are consistent with previous research which suggests that attentional demands may have a negative impact on spoken language in healthy people as well as individuals with aphasia. Morphosyntactic, lexical and pragmatic components of aphasics’ verbal output were affected by manipulating demands placed on attentional capacity or allocation (LaPointe & Erickson, 1991; Bowles, Erickson & LaPointe, 1992; LaPointe, Kemker, Stierwalt, Heald &
Blanchard, 2004). Message construction, flow of output and sentence construction have all been shown to be disrupted in the presence of attention deficit (Jou & Harris, 1992).

**Naming**

Anomia is ubiquitous in aphasia and it is this deficit that differentiates mild aphasics from normal speakers. Therefore expectedly, all of the participants with aphasia in this sample presented with word finding difficulties on the WAB. The link between anomia and attention difficulties is also not a new one. Linebaugh (1997) demonstrated that on-line word generation in spontaneous activities, such as in conversation, represents the most difficult of all naming activities, as the onus of finding a word relies completely on internal processes of the individual with only the awareness of the context to assist in the location of the appropriate label. These processes are thought to rely heavily on attention (Murray, 2000), although if disrupted they do not in and of themselves cause naming problems as demonstrated by poor interference control in Felicity and Jane* who both had intact naming skills.

In a study comparing attention and intention treatments for naming specifically, Crosson, Fabrizio, Singletary, Cato, Wierenga et al., (2007) showed that patients with moderate, severe and profound word-finding impairment showed gains with both treatments with the first two groups displaying a greater incremental improvement from one treatment phase to the next with the intention than the attention treatment. Furthermore, almost all patients who showed treatment gains on either attention or intention treatment also demonstrated generalization from trained to untrained items.

**Comprehension**

Attention deficits have also previously been associated with comprehension difficulties (Helm-Estabrooks et al, 2000). In right hemisphere damaged (RHD) individuals, sustained attention whilst not being a solitary sufficient factor, might be involved in comprehension problems related to RHD, specifically with reference to the ability to make inferences (Saldert & Ahlsen, 2007). In children with attention deficits, listening comprehension has been found to be impaired, regardless of language ability (McInnes, Humphries, Hogg-Johnson & Tannock, 2003). A group of patients with Parkinson’s disease (PD) were significantly compromised in their ability to attend to certain critical grammatical features of a sentence. A regression analysis identified specific grammatical, semantic, and attentional mechanisms as significant
contributors to PD patients' overall sentence comprehension, accounting for over 97% of the variance in their performance (Grossman, Carvell, Stern, Gollomp & Hurtig, 1992).

Wiener, Connor and Obler (2004) suggested that impairment of inhibition is positively correlated with clinical-behavioural symptoms of severity of auditory comprehension deficits as measured by the Token Test. Their findings support an impairment of inhibition at the lexical-semantic level of language processing, reflecting an inability to effectively ignore automatically evoked distracting stimuli. Wiener et al. (2004) state that in light of their findings, at least part of the attention difficulties contributing to striking reductions in auditory comprehension can be attributed to impaired inhibition.

In this sample, comprehension was relatively intact for all ten participants (although comprehension of complex information and higher order skills was not assessed), despite the fact that six of the ten also presented with inhibition deficits. The ability to process online information within the context of a conversation, especially multiparty interactions, relies on different processes. Despite participants coping well with the demands of the WAB testing, misinterpretations and slowed processing was evident in the context of conversational interaction, as demonstrated below.

**Repetition**

A notably large proportion of the literature on repetition deficits in conduction aphasia, attribute such difficulties to reduced working memory capacity (see discussion in the working memory section below). However, the relationship between working memory and attention factors – such as the ability to resist interference, is complex and highly interdependent (Barkley, 1997). Fuster (1995) states that short term memory (STM), as supported by the prefrontal cortex, is highly dependent on attention, which sub-serves the temporary retention of information. Therefore, the measurement of STM, in individuals with prefrontal deficits amounts to a measurement of the fundamental difficulty these patients have with suppressing attention to irrelevant stimuli and maintaining attention to relevant stimuli.

Testing with this sample did not reveal consistent relationships between interference deficits and repetition difficulties. Some of the participants who consistently scored poorly on all
aspects of attention e.g. Cecil, had intact repetition scores, while others whose interference scores were intact, had poor repetition. Table 6.4 demonstrates this point.

Table 6.4 – Inconsistencies in the relationship between interference control and repetition

<table>
<thead>
<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>INTERFERENCE CONTROL</th>
<th>REPETITION</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>DIGITS</td>
<td>STROOP</td>
</tr>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>x</td>
<td>√</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>x</td>
<td>√</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>x</td>
<td>√</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>x</td>
<td>√</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>x</td>
<td>x</td>
</tr>
</tbody>
</table>

Key: (✓) Intact; (x) Impaired

*Reading* /writing

A common complaint amongst the participants in this research was that reading was difficult because participants reported that they “could not concentrate”. It might be expected that these individuals would present with poor interference scores. These consistencies were true for some participants like Mel and Margaret. However, of the three individuals who achieved 100% on the reading tasks of the WAB, two were Felicity and Jane* who both demonstrated clear interference deficits on the Stroop Word-Colour Interference Test. The other was Cecil (anomic aphasia) who scored poorly on all tasks of attention.

There is existing research examining relationships between reading and attention factors, and to a much lesser extent about writing and attention. Most of the existing literature comes from the field of Attention Deficit Hyperactivity Disorder. Children with attention deficits have demonstrated poorer performance on tasks measuring single word recognition and vocabulary, word attack, contextual comprehension, written spelling, written sentence construction and writing fluency (Elbert, 1993; Resta & Eliot, 1994). Researchers have attributed some features of dyslexia to reduced attentional capacity relative to textual demands (Kershner, Henninger &
Cooke, 1984) and also found co-morbid attention and EF deficits in children with reading difficulties (Bental & Tirosh, 2007).

Attention training has proved beneficial for reading skills in both adults with aphasia (Coelho, 2005) as well as children with reading disability (Solan, Shelley-Tremblay, Silverman & Larson, 2003). In a follow up to his 2005 study, Coelho and colleagues demonstrated improved reading rate and comprehension as well as less variability in comprehension skills for longer passages of text. The authors attributed these changes to more efficient allocation of attentional resources (Sinotte & Coelho, 2007). In addition, specific attention training focusing on self regulation strategies have also been beneficial to children treated for written language difficulties (Chenault, Thomson, Abbott & Berninger, 2006).

Each of the ten participants demonstrated difficulties with some aspect of interference control or freedom from distractibility. Given that the two tests administered for this construct are designed to measure similar skills namely the ability to ward off distraction, and given that of the two the Stroop Colour-Word Interference Test is the more complex, the lack of consistency in findings was initially surprising. While it is understandable that Mel and Felicity who managed the simpler of the tasks did not cope as well with the complex one, it is difficult to explain why the other participants, who managed the Stroop Colour-Word Interference Test, all failed Digit Span.

Perhaps Barkley’s comments (1997) in regard to the types of distraction that may cause interference are pertinent. He states that whether or not distractors disrupt task performance could depend on the prepotency of the response likely to be elicited by the distracting event. In other words a previous experience of responding in a particular way to a particular task may serve to make it more likely that that familiar response will occur despite task demands requiring a new or different response. It could then be hypothesized that the degree of prepotency could be highly individual, depending on the participants’ previous experience. In addition the extent to which executive functions taking place during that task performance require protection from such interference will also determine whether or not distractions actually disrupt performance. In the ADHD literature, distinctions are made between distractions that are inherent within the task as opposed to distractions outside of the immediate task. Task-irrelevant thoughts during performance may be a source of distraction from a more
internal source (Barkley, 1997). Therefore the source and type of distraction coupled with individual experience and functioning could account for discrepancies in the test findings.

Interference control and conversation

The two distinct behavioural manifestations of poor interference control are distractibility and impulsiveness. In conversation these features would be noted by the following characteristics: difficulty retaining the purpose of an exchange or following a conversation (Gillis, 1996, Schapiro & Sacchetti, 1993), especially during group communication (Godfrey & Shum, 2000; Prigatano, 1986) or while carrying on a second activity like walking and talking in individuals with dementia (Cocchini, Della Sala, Logie, Pagani, Sacco & Spinnler, 2004); difficulty staying on topic and a tendency towards confabulation (Gajar, Schloss, Schloss & Thompson, 1984); a tendency towards making tangential or irrelevant comments (Linscott, Knight & Godfrey, 1996; Prigatano & Fordyce, 1986); misinterpretations of speech with inappropriate reactions to conversational partners and interruptions (Linscott et al., 1996).

Most of the existing research relating interference control to conversational features comes from the TBI literature with contributions from dementia (Muller & Guendouzi, 2005; Perkins et al., 1998) and right hemisphere research (Tompkins, 1990). However there is almost no existing literature which draws parallels between attention function and conversational success in individuals with aphasia. Notable exceptions are work by Hardin and Ramsberger (2004) and Ramsberger (2005) demonstrating that participants who underwent attention training showed significant changes in attentional tasks as well as in functional communication skills as measured by story retelling. What is particularly significant about these studies was that these improvements were not reflected in linguistic function as tested by the PICA. This result led the authors to conclude that either conversation may depend more on attention and executive abilities than linguistic skills, or that the linguistic tasks did not sufficiently stress the attentional system to a degree that they produced changes in function, a conclusion that was supported by the current investigation.

Conversation Analysis (CA) results for the ten participants showed a distinctly clear and consistent pattern in this regard. Participants who showed deficits on both the digits forward as well as the Stroop Colour-Word Interference Test displayed evidence of the distractible behaviour described above. Individuals who showed impaired performance on only one of the
two tests consistently displayed distractible features in conversation when they were impaired on the Stroop Colour-Word Interference Test, but not the digits forward test. Table 6.5 on the following page demonstrates this significant finding. The conversational excerpts that follow demonstrate each of the features described above. Every participant who demonstrated distractibility is represented in the excerpts below.

Table 6.5 – Interference control with presence of distractibility in conversation

<table>
<thead>
<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>POOR INTERFERENCE CONTROL</th>
<th>PRESENCE OF DISTRACTIBILITY IN CONVERSATION</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>DIGITS</td>
<td>STROOP</td>
</tr>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>✓</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>x</td>
<td>✓</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>x</td>
<td>✓</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>x</td>
<td>✓</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>x</td>
<td>✓</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>✓</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>x</td>
<td>x</td>
</tr>
</tbody>
</table>

Key: (✓) Intact; (x) Impaired

**Difficulty following the exchange**

Several of the participants provided self reports that they found it difficult to follow group interactions including Mel and Margaret. In *Example i* below, John (JH) fails to follow information online. M and John are discussing the route he takes to the University Speech and Hearing Clinic which he attends once a week. M has asked John which highway off-ramp he takes (line 161). When John cannot recall the name, M begins to offer some alternatives. The first suggestion was in fact the correct one however John does not recognize the correct name when it is presented. Instead, M suggests two more road names then repeats the selection again in slower, short sentences and phrases before John finally recognizes the name of the turn-off in line 171.
Example i

161 M >Which turn off do you take?< (.) >do you remember<?
162 JH No
163 M >Empire Rd<?
164 JH No. (shaking head)
165 M Jan Smuts?
166 JH No. (shaking head)
167 M Even before?

(1)

168 M There’s St. Andrews↑
169 JH Andrews? No. (shaking head)
170 M Jan Smuts turn off then there’s Empire
171 JH Empire Rd (hand to head shakes head) Empire Rd

Difficulty staying on topic

Three examples are provided. In the first, Cecil is in conversation with his wife (RS) who asks him a direct question in line 045. Cecil starts to answer the question but then becomes distracted by a related line of thought (lines 047 and 048). Instead of returning to the question his wife asked, the conversation then shifts to the car he bought as demonstrated by his wife’s follow up question in line 054.

Example ii

045 RS where did you have breakfast?
046 (5)
047 CS um we went to this (.) we went to eat no I went I went to get the car you know I got
048 that uh ok there’s another thing.(2) I got a it’s called a (6) a a Scenic a Scenic
049 RS A Scenic yes
050 CS until I got (. ) that (3) name (1) “people would talk” I said Snee Shnik Schnee Snich (3)
051 I’m trying to say that’s the whole thing says when it says Sce-Scenic well I found it now↑
052 RS mm
053 CS I found what it is ↓(2) it’s a nice car↓ it’s um↑ white
054 RS What’s the mileage?

The next example, presents Jane* (JR) also responding to a question asked of her by the researcher (R) but then finding it difficult to remain on topic. In line 056 the researcher asks Jane* a question related to her writing. Jane* answers the question in line 057 but then elaborates. What follows is a disorganised and loosely connected flow of ideas which covers a range of six different topics, none of which are coherently linked to the original topic which related to Jane*’s writing.
**Example iii**

056  R  are you writing more just because you (1) practising more? or↑
057  JR  no.
058  R  because you’ve got-
059  JR  no actually, what I need to do is (. ) I need to do really is to get my self confidence back.
060  R  right.
061  JR  I need to be on my own, in my own little place. like you know (1) wherever you’ve met me, you’ve met me in different places↑
062  R  Ja.
063  JR  And I- I’m used to being (1) I I bought you know I had my own home. I had a good job. everything was great until I got sick. didn’t matter whether I was married or not because I brought them up on my own. I didn’t I didn’t have a have a a hu- husband to help me. S***’s got his own business and C***’s got her own business. >had nothing to with with the father<
064  R  Ja..
065  JR  in fact C*** and W*** never came from the same father and he is in university in England >has got nothing to do with that<

In this third example, Margaret’s distractibility is clearly evident. She (MS) and the researcher (R) were discussing a serious concern about the fact that she had been experiencing numbness in her one hand. The researcher had been encouraging her to consult her doctor, when she had a mild coughing attack (line 096). She makes an irrelevant comment about the video camera being on and then gets up to open the door to allow some fresh air into the room. This then prompts her to explain why the door was locked and the conversation turns to a discussion about her granddaughter. At no point subsequently in the conversation does she return to her concerns about her health despite the fact that when she raised the topic she exhibited considerable anxiety regarding her symptoms.

**Example iv**

088  MS  You see. I think- why does it feel like that?
089  R  That numbness?
090  MS  Mm.
091  R  I don’t know
092  MS  That worried me
093  R  Ja
094  MS  Especially when at at night
095  R  Ja
096  MS  And then in the afternoons I feel (coughs) sorry (coughs) oh I keep forgetting you have this thing on that um the-
097  R  Ja It is on
098  MS  Let me get a bit of air here (gets up and starts unlocking the balcony door) I had to do this because of my my grandchild. I’m telling you she um she got hold of the balcony and she also got hold of my key for the bathroom↑
And she gave me uh quite a hard time in two days. Oh shame. She’s feeling very diff- difficult you know that’s the whole thing (sits down again). She’s only six. Mm. She’s just started school.

Tendency towards making tangential or irrelevant comments

Example v below presents Jane* (JR) in discussion with the researcher (R) where she makes several tangential comments. The conversation reads like a series of offshoots with each idea then sparking their own derivative, although her thoughts do not always appear to be associated. Initially the researcher and Jane* are talking about Jane*’s health as she had recently found out that she may have uterine cancer. In the middle of this discussion (line 033), Jane* says something completely unrelated about a therapy activity she participated in with her student therapist at the university clinic she attends. There is another tangential comment when while talking about her writing, she mentions her son going away (line 039). This offshoot becomes clearer later in the conversation when Jane* mentions that her son has a lap top which she wants to use to practice her writing on, while he is away, however this is not explicitly stated the first time and the fact of his departure appears random and irrelevant in the context of the conversation. The excerpt ends with an evaluation about her memory which is also unrelated to the previous statement about her son (line 041) and which stems from her conviction that her writing difficulties have nothing to do with poor memory skills.

Example v

021 JR no (1) he’s saying that I have cancer of the of the parts of the (gestures to her abdomen)  
022 R mm↑  
023 JR but he says that it could be the loop? but if well if it is, then they ju- they use a la ser  
024 R °right°  
025 JR mm↓  
026 R to remove the°=  
027 JR =the parts. Ja=  
028 R °the affected parts°.  
029 JR mm.  
030 R Oh shame Jane* I’m so sorry  
031 JR Hm?  
032 R Are you ok? I mean (. ) how are you f-  
033 JR I’m not well (. ) I don’t actually >I don’t want to< upset my children because they think
>you know< she doesn’t talk about it and and M***1 it was great\ and I thought you
know what (.) I haven’t been writing for a long time and >I (1) actually lost the feeling
of writing again<

034  R   ja?
035  JR  and she gave me her (1) lap top↑
036  R   right↑
037  JR  and she said let’s try it
038  R   ja.
039  JR  look it’s not it’s because I’m left handed right? and it actually I said to Sh***2 if
he’s going away ‘cos he’s going away for a while
040  R   mm?
041  JR  so I can whatever I do wrong↓ cos my memory is ºit was goodº

Misinterpretations of speech

Mel, who by her own self report sometimes finds it difficult to follow conversations when there
are a number of people or when the information conveyed is complex, demonstrates a
misunderstanding of her boyfriend’s (A) question. The topic centers around what Mel (MZ) will
wear to an upcoming wedding the two of them are attending together. In line 012, A asks Mel if
she has another outfit if the one she was planning to wear does not fit her. Mel misinterprets the
question as a request for details about the dress and begins to demonstrate that there is detail on
the shoulders and sleeves. A’s confusion is evident with his question in line 014 and then he
rephrases the question in line 016. Mel continues to describe the dress by elaborating that it is
the one she wore to her own wedding. A asks the question a third time, at which point Mel
realizes that she has misunderstood his previous questions and answers him (line 019).

Example vi

008  A  Will it fit you?
009  MZ  No we- I don’t know.
010  A  But I told you, you must put it on (.) see.
011  MZ  (smiles) if-
012  A  and if that doe↑sn’t↓ what else have you got?
013  MZ  No it ºthere are some things thereº (gesturing to her shoulder)
014  A  What?
015  MZ  and I have some of the things there (gesturing to the cuff of her sleeve)
016  A  Have you another nice dress?
017  MZ  Yes I want to wear that one it’s mm- wh-what I want to wear that one was I own
wedding (1) um that I wore to my own wedding.
018  A  Yes I understand. But if that doesn’t fit, have you got anything else?
019  MZ  OH. another (2) I don’t know.

1 The name of her student therapist
2 Her son
Interruptions and intrusions were rare among this group of participants. Although some like Cecil, Felicity and Jane* were prone to interrupt or fail to judge the end of the transition space accurately. In the example below, Felicity interrupts three times, misjudging the end of the researcher’s (R) turn. The first two times, the researcher is mid-sentence, when Felicity begins her own turn (lines 013 and 014 and lines 028 and 029). The third time, the researcher has asked a question (line 032) but intonation patterns indicate that she has not quite finished asking the question. Felicity seems to have some difficulty interpreting intonation, paying attention only to the linguistic content and beginning her answer as soon as the linguistic text indicates that a question has been asked (line 033). This difficulty with prosodic interpretation has been demonstrated previously in individuals with right hemisphere damage (Hird & Kirsner, 2003).

Example vii

011 R I’ve joined a book club
012 FG Oh↑ good.
013 R (hhh) Since our last- chat
[ ]
014 FG Mind you
015 FG my last book club my book club we don’t ge- this is one of their books
016 R Yeah↓
017 FG but we don’t get so↑ (1) technical. we just say if we liked the book or not. we don’t (.) go into characters, and whether the characters (1) grow↑ and
[... portion omitted from the transcript]
026 R Ja↓ I must say I’m quite excited. >I’ve only been to one mee↑ting↓<, they meet (.) once a month↑--
027 FG Ja
028 R and↑
[ ]
029 FG that’s a bit the right time
030 R “ja”
031 FG cos then you’ve got (. ) four maybe five weeks to finish the books you choose.
032 R Ja. How many do you normally choose↓, at a time?
[ ]
033 FG I normally take
034 FG for I normally take well- (2) if the next book club’s four weeks away, I must have at least four↑ books. Five weeks, at least fi↓ve.

“…a mild stroke indeed, but I cannot do any two things simultaneously…Noise is brutality, every aphasic will tell. And truly there should be a law against it” (Moore, 1994, p, 102).
Interference control – summary

Associations between various language functions as tested by the WAB and interference control were inconsistent. There are a few potential explanations for this. The first is that deficits in EF may not be readily apparent in the tightly structured and hierarchically designed test format of this formal language battery. There appears to be better consistency between EF findings and conversational characteristics. Six of the ten participants performed poorly on the Stroop colour word test indicating difficulty with the suppression of distracting stimuli i.e. a susceptibility to distraction. All of these participants demonstrated conversational features consistent with behaviours previously identified in the literature as being representative of poor interference control. Many of these features such as the tendency to be tangential, reduced cohesion with disorganised or off-target output have also been described with attention difficulties as being characteristic of right hemisphere damage (Tompkins, 1990). This data suggests that such difficulties are not exclusive to right hemisphere or diffuse lesions, such as have been identified in both cortical and subcortical dementias and multiple sclerosis, and can be present in individuals with focal left hemisphere infarcts. This finding is highly significant as it speaks to the need for the inclusion of more authentic assessment formats to fully understand the communication of brain injured adults.

The second possible reason for lack of association between language and EF results speaks to the great level of heterogeneity in stroke populations. The combination of functions and skills within the presence of noted linguistic and/or pragmatic deficits imposed by stroke is unique and depends on a variety of psychosocial and individual variables, which may further account for why some participants showed deficits on the supposedly less complex of the tasks while managing the more complex one.

A final reason for this lack of conclusiveness is the intricacy of the constructs under discussion. Each construct individually represents a multifaceted notion which interacts in complex ways with one another. Results may be dependent on test construction, administration, or scoring. Relationships can only be determined relative to the specific conditions and parameters of a given situation.
6.2.2.4 Response inhibition results

This is the capacity to inhibit a prepotent response and stop an ongoing response, by shifting to a different one (Barkley, 1997).

Table N.3 in the appendix presents the results for the Trail Making test. Performance throughout was variable both across and within participants. At initial testing, four individuals presented with poor set switching on this test: Cecil, Mel, Jane* and Margaret.

Response inhibition and language

As the previous section detailed current findings linking language functions to attention, this section will only address the primary behavioural manifestation of poor inhibition – perseveration - as it relates to the language functions assessed by the WAB.

Spontaneous speech

Many existing theories of perseveration are derived from aphasic performance on structured tasks, such as naming. Although perseveration is noted in spontaneous speech in clinical observations, there is little literature on its manifestation in this context. There is a strong body of evidence suggesting that perseverations have their origin in language processing deficits at both semantic and phonological levels (Hirsh, 1998; Moses, Nickels & Sheard, 2007). According to Moen (1993) perseveration is often found in the speech of anomic patients; and normally perseveration of a specific word will take one of two forms: either (1) the perseverated word occurs interspersed throughout a dialogue or (2) it occurs only once or twice following its first occurrence.

An assessment of the spontaneous speech component of the WAB revealed that seven of the ten participants displayed evidence of perseveration during administration of this sub-test, not all of whom performed poorly on Trail Making. Perseveration is generally defined as the inappropriate repetition of a word, groups of words or ideas in the face of changing contextual demands. In addition, Jane* who scored poorly on Trails, did not show evidence of perseverance in the spontaneous speech task. Perseverations took several forms, which are detailed below in Table 6.6 together with an assessment of their performance on Trail Making.
Table 6.6 – Perseveration in spontaneous speech and performance on Trail Making

<table>
<thead>
<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>TYPE OF PERSEVERATION</th>
<th>EXAMPLE</th>
<th>PERFORMANCE ON TRAILS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>Continuous (word level) (Cecil is also a premorbid stutterer, repetitions are attempts at self correction, not apraxic)</td>
<td>‘There’s a flas, a fla a flag’ ‘She’s got a flat a flaks, flask’</td>
<td>Impaired</td>
</tr>
<tr>
<td>Mel</td>
<td>Recurrent (phrase level)</td>
<td>‘I don’t know why they’ve got the needle to me but they do have the needle to me ok I don’t know what they’ve got a needle to me but they do have a needle to me but I want to know’</td>
<td>Impaired</td>
<td></td>
</tr>
<tr>
<td>Tumi</td>
<td>Recurrent (phrase level)</td>
<td>‘What is this? Oh my God oh my God and I know oh my God boyfriend man woman oh my God oh my God’</td>
<td>Intact</td>
<td></td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>Recurrent (word / phrase)</td>
<td>‘woman and man walking I don’t know sitting the river rug woman is walking coffee or tea man walking no reading a book’</td>
<td>Intact</td>
</tr>
<tr>
<td>Paul*</td>
<td>Continuous (word level) Recurrent (phrase level)</td>
<td>‘to uh um work to work to to uh no to re- to um tree work is to um to working work yes no work to’</td>
<td>Intact</td>
<td></td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>-</td>
<td>-</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>Recurrent (word)</td>
<td>‘radio shoes woody wood shoes take it off a garage woody wood a kite a tree’</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>Recurrent (phrase level)</td>
<td>‘Its trees… and with lots of trees and a house and lots of trees as well.’</td>
<td>Impaired</td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>-</td>
<td>-</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>-</td>
<td>-</td>
<td>Impaired</td>
</tr>
</tbody>
</table>

Bold type indicates perseverated utterances

**Comprehension and repetition**

There is no existing literature on associations between shifting abilities and the ability to comprehend or repeat verbal material other than that perseverative responding can interfere with test performance. As with the spontaneous speech findings above there were no clear associations between these skills and set shifting measured by Trail Making.

**Naming**

In the area of aphasia, shifting deficits or the manifestation of perseveration have been discussed predominantly in relation to naming tasks, as well as in response to assessment
demands (Bryant, Emery, & Helm-Estabrooks, 1994). Theories about perseveration have also been generated to a large extent based on naming performance (Albert & Sandson, 1986; Buckingham, Whitaker & Whitaker, 1979; Helm-Estabrooks, Bayles & Bryant, 1994; Hirsh, 1998; Lundgren, Helm-Estabrooks, Magnusdottin, & Emery, 1994; Santo-Pietro & Rigrodsky, 1982; Vitkovitch & Humphreys, 1991; Wepman, 1972; Yamadori, 1981). Naming on the WAB is assessed through object naming, word fluency, sentence completion and responsive speech. Table 6.7 below presents the presence of perseveration during these tasks with performance on Trail Making. As is evident, results are variable with some individuals manifesting perseveration during verbal tasks but having intact performance on Trail Making (Paul*) and others showing no perseveration at all during verbal naming tasks, but impaired performance on Trail Making (Jane*).

Table 6.7 - Presence of perseveration during naming and performance on Trail Making

<table>
<thead>
<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>NAMING</th>
<th>WORD FLUENCY</th>
<th>SENTENCE COMPLETION</th>
<th>RESPONSIVE SPEECH</th>
<th>PERFORMANCE ON TRAIL MAKING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>-</td>
<td>√</td>
<td>-</td>
<td>-</td>
<td>Impaired</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>-</td>
<td>√</td>
<td>-</td>
<td>-</td>
<td>Impaired</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Intact</td>
</tr>
<tr>
<td>Broca's</td>
<td>John</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>√</td>
<td>-</td>
<td>√</td>
<td>-</td>
<td>Intact</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>-</td>
<td>√</td>
<td>-</td>
<td>-</td>
<td>Impaired</td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Impaired</td>
</tr>
</tbody>
</table>

Key: (✓) perseveration present; (-) perseveration absent

**Reading / Writing**

Perseveration has been noted in writing in aphasic individuals where individual letters, groups of letters, words or phrases are repeated in inappropriate contexts (Albert & Sandson, 1986; Christman, Boutsen, & Buckingham, 2004; Sandson & Albert, 1987). No perseveration was noted during reading tasks, but three individuals John, Margaret and Paul*, displayed evidence in written tasks expressed as letter and part word repetitions. Again, results were inconsistent with test results on Trail Making.
Shifting / Response inhibition and conversation

There is scarce existing literature linking deficits in shifting or inhibition to conversation skills. Researchers have found that perseveration or impaired inhibition may manifest in individuals with TBI as a tendency to stray from the conversational topic at hand to a preferred point of reference, which is then repeated continually (Body & Parker, 2005; Frankel & Penn, 2007; Godfrey & Shum, 2000). Also in relation to individuals with TBI, poor response inhibition has been demonstrated in an inability to gauge when it is appropriate to end their turn and give another participant a chance to speak, resulting in verbose and lengthy turn characteristics (Gillis, 1996; Schapiro & Sacchetti, 1993). Coelho, Liles and Duffy (1991) in their study of five TBI patients and five control subjects found that adults with TBI took a greater number of turns, which they attributed to poor inhibition. Children with ADHD have also been found to present with talkativeness (Barkley, Cunningham & Karlsson, 1983) and intrusive behaviour during conversation, suggestive of impaired inhibitory control (Malone & Swanson, 1993). Aside from an inability to interrupt ongoing responses, poor inhibition is manifested in repetitive verbal disorders such as echolalia in individuals with cognitive decline including Alzheimer’s disease and frontotemporal dementia (Blair, Marczinski, Davis-Faroque & Kertesz, 2007).

In this sample, conversational data suggested that a number of participants presented with the features of talkativeness with extended turns, and perseveration during conversational speech. However as with the language results these were not consistent with the results from Trail Making. Instead, the Wisconsin Card Sorting Test (WCST) which was assessed under the working memory construct has been considered a definitive test of response inhibition and perseveration. Participants with fewer perseverative errors achieved a higher number of correct sorts. Table 6.8 below shows participants who displayed inhibition difficulties in conversation with those who showed impaired shifting in Trail Making and the WCST. With one exception, the participants who managed the WCST did not display inhibitory deficits in their conversational discourse, while the other seven participants had features of poor response inhibition in their conversations. Jeannette, who scored well on the WCST, presents with one idiosyncratic perseverative utterance ‘woody wood’, which recurs during her spontaneous speech. Aside from that there are no other perseverative characteristics in her conversational output.
Table 6.8 – Evidence of poor inhibition and perseveration on Trails and the WCST.

<table>
<thead>
<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>EVIDENCE OF POOR RESPONSE INHIBITION</th>
<th>PERSEVERATION ON TRAIL MAKING</th>
<th>PERSEVERATION ON WCST</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>Talkativeness, extended turns, word level perseveration</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>Perseveration at word and phrase level</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>Topic repetition, perseveration at word and phrase level</td>
<td>-</td>
<td>√</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>Perseveration at word and phrase level</td>
<td>-</td>
<td>√</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>Perseveration at word level</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>Talkativeness, extended turns, topic repetitiveness</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>Talkativeness, intrusions</td>
<td>-</td>
<td>√</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>Talkativeness, extended turns, topic repetitiveness</td>
<td>√</td>
<td>√</td>
</tr>
</tbody>
</table>

Key: (√) perseveration present; (-) perseveration absent

The remainder of this section presents examples from conversational interactions demonstrating features of poor inhibition identified above.

*Talkativeness / extended turns*

The four individuals who presented with extended turns Cecil, Felicity, Jane* and Margaret violated turn taking conventions by speaking for extraordinarily long periods of time about themselves or topics related to them. Once the topics were exhausted, Felicity in particular had difficulty introducing new topics and did not share in the responsibility of introducing new elements into the discussion, a finding consistent with previous research on individuals with right hemisphere damage (Hird & Kirsner, 2003). In the two examples below the researcher (R) talks to Felicity (FG) and Cecil (CS) speaks to his wife (RS). The lengthy turns are evident for Felicity in turns 041 and 043 in which she utilizes a considerably longer turn space than the researcher. The same is true of Cecil in turns 001, 006 and 014.
Example i

038 R You must miss your work
039 FG <Oh I do::> and I used to love it so much
040 R Where was it? you were↑ (.at the Gen?
041 FG I was at the Gen because I worked for the heart foundation as a- (1) everything
you know. <Unfortunately> a lot of it was fundraising but I enjoyed that too and then a lo-
some was (.) sort of↑ not patient education but, talking to people about preventing
heart disease and stroke. There’s an irony for you (laughs) and um and how I got to
work at the Gen↑ I was going to, I knew that they had a a rehab unit and it was based in
the intensive at the cardiac intensive care and I went to see the sister who ran it and she
was telling me about her job, and she and (1) she was saying that she was going to have
to work more full time and she was trying to find somebody to do her job >did I know
anybody< I said I would like to, it sounds marvellous. so I spoke to the head who was
the cardiologist E*** K*** and they both said cos I mean, you wouldn’t- couldn’t have
put me in an intensive care unit then cos I didn’t even know how the machines worked,
whi↑ch button to press, cos I was out of practical nursing for twenty years already but
(2) it’s talking to patients (2) is still the same.
042 R Ja ja absolutely
043 FG And so that’s where I started. that↓ I did that in the mornings, and I went to
the heart foundation in the afternoons, and did all my haranguing. My <phone runs> and
things then. So, I really had TWO jobs no wonder. I remember thinking once I was
driving home from somewhere and my mind was going around with all these things and
I thought to myselfº I feel like a snowball travelling downhill. What’s going to happen
when I get to the bottom? This might have been what happened when I got to the bottom
(laughs)
044 R Sounds like you had a very busy-
045 FG And I had children

Example ii

001 CS um ok um (4) uh and I went to (2) I thought it was first it says (1) I thought it says um
(2) I thought it was says ‘Rust Berg’ you know ‘Rust Berg’ and I phoned I said what is?
and I phoned um (1) I said what is Ru- Rust? and then (.). when I came there is Ru-
RusTENberg (.) you know what I’m saying?
002 RS yes you you weren’t sure what the name of the place is that what you
[-] when I said
[>] >no no no< I know (3) uh I
003 CS had to phone (2) um I***
004 RS “mm”
005 CS and I said uh I’m going to get (.). uh you know the car (.). and I the- and I the- I said you
know I phoned him and I said um (1) um I said Rust Rust berg he says what is
“Rustberg” then I said I said you know that that place so I said you know (.). then I
came here and it said Rustenberg so that’s the whole thing my (1) my (2) uh mind (1)
mind the mind
007 RS mm
008 CS um (3) when I (4) I can f- I could feel the (2) uh in their mind (1) in their mind where is
009 CS says Rust but (.). doesn’t (.). doesn’t come in↑ you know what I’m trying to say?
010 RS it doesn’t come out “>you trying to say<“

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so it says it says Rustberg I said what? "anyway"

so now (.).what it is is Rustenberg (.). anyway uh (3) we went with (2)uh G*** you know (1) um (4) ts! with (5) uh (3) uh you know the rain (2) hard pour (.). pour pour RS pouring

(1) can’t believe it we going slow slow slow (.). until (.). there’s a place called Dies Diek (2) Diekstill Diek Diekstiel something when they (2) with all the people (4) then we could go much much better (3) um† (picks up coffee and takes a sip)

Interpretations of talkativeness are however complex. Talkativeness is an identified although debated feature of communication in elderly individuals (Gould & Dixon, 1993; Juncos-Rabadán, Pereiro & Rodríguez, 2005). Findings have also shown that elderly persons will have a greater tendency to talk “off-target” than younger adults (Arbuckle, Nohara-LeClaire & Pushkar, 2000). One theory is the “style difference” theory postulated by Giles, Fox-Harwood and Williams (1994) which states that ageing leads to a pragmatic evolution whereby a greater emphasis and value is placed on conversation. Arbuckle et al., (2000) however propose that the increase in irrelevant content can be attributed to age-related inhibitory mechanism deficit. Stine, Cheung and Henderson (1995) see the decrease in content density, increments in irrelevant content and reduction of overall cohesiveness to be representative of working memory deficits which can no longer keep information in mind while actively processing and adjusting said information efficiently.

Miyake, Friedman, Emerson, Witzki, Howerter, and Wager (2000) propose that such inhibitory and working memory deficits, should actually be interpreted as an executive functioning deficit affecting the control and coordination of complex information into working memory. This is coupled with changes in the social situations of some elderly persons and may create an increased need for communication which drives the talkativeness in the presence of reduced content and cohesiveness.

Juncos-Rabadán et al. (2005) state that research into the influence of ageing in discourse, needs to relate characteristics of the discourse to both cognitive and psychosocial factors. If this is true for normal aging, then it is even more necessary for elderly individuals with neurological damage. The confluence of numerous variables here complicates clear interpretation. Within the sample, three of the four participants who appear to be “talkative” are the three oldest and qualify as “older adults”. In addition, two of those individuals live alone. Margaret especially
spoke frequently of her loneliness and difficulty securing conversations. Normal ageing processes exert influences on cognitive flexibility and these must be taken into account when looking at the attention, inhibition and working memory deficits manifested on testing. The variable that weights in favour of interpreting this observed talkativeness in these participants, to deficient inhibitory processes is the fact that for both Cecil and Margaret changes on this dimension were seen during the active drug phase (this will be addressed in the discussion relating to the results of the experimental phase of the study.

**Perseveration**

Perseveration as manifested in these conversational samples was notably linked to the disruption of repair abilities. Trouble spots in conversations are traditionally and in the majority of cases self corrected within the same turn transition space in which the trouble occurs (Schegloff, Jefferson & Sacks, 1977). There is emerging evidence from the aphasia literature however that in fact successful repair is achieved more collaboratively as a joint activity between speaker and listener (Oelschlaeger & Damico, 2003; Simmons-Mackie & Damico, 1996). Still, the person with aphasia needs to demonstrate some ability to contribute to the repair, even in as much as they negate or accept their conversational partners’ guesses at what they were trying to say. Difficulties contributing to repair as a result of perseveration are demonstrated in the examples below. In *example iii*, Paul* (PF) argues with his wife (RF) about the hand brace the Occupational Therapist has given him to wear. He has a strong objection to wearing it but cannot express why he does not want to. His wife volunteers a number of possible reasons, (line 019, 029, 031, 039 and 043) ranging from the brace being uncomfortable to causing pain. In a different part of the transcript not presented here she also asks if it makes him uncomfortable emotionally, if he feels a stigma when wearing it, but none of these are accepted by Paul*. Paul* has limited expressive output during conversations but perseverates in this sequence on the words ‘no’, ‘its’ and ‘to’ and repeats ‘I don’t know’. Ultimately, despite a protracted sequence, there is no resolution.

*Example iii*

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>013</td>
<td>RF</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>014</td>
<td>PF</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>015</td>
<td>RF</td>
</tr>
</tbody>
</table>
See al ready (1) it’s not as (. ) cramped as it was. NO it’s it’s s s s u:m no not (3) ss ss, (2) to to went uh no (3) no
You feel it’s too much? No. it’s (2) it’s (3) ja.
Wh-What is actually? what is? What’s worrying you about this?
you tell M***3 what’s worrying you no no no noº I don’t know (2) whe (3)
no she did say you must (.) wear it (1) in the day as well now.
and if it’s hur ting, I’m just looking hereº no.
not catching again? no no It’s ss ss (2)
If the fingers are hurting? No. No. No.
Then why don’t you want to wear it?
During the day?
Ja. we’ll the::: its it’s (2) too too too it it
Uncomfortable.
NO no s s sº I (1) d-don’t (1) (audible sigh out) I::: (1) don’t (1) know (2) how (1) I (1) ss ssº turn to to toº no NOº >it’s it’s it’s s s sº<
Ja but I’m just worried why you don’t (1) want to wear it during the day↑
Yes↓ (2)
<is it somewhere hur ting g or unco mfortable?>
No it’s s s sº
So then why don’t you want to wear it?

no it’s (1) IT’S (1) WHEN YOU (1) GET (2)
BO SSY.
No no↓ so. I↑ I: do- don’t (audible sigh) IT WILL BE I I I I I don’t know it’s (3)
ja-Well it just worries me that (1) I you know w-want to understand why what’s what’s wrong with wearing it during the day?
No it’s it’s the way, (1) (audible sigh) No↓ its its eh
Cos M*** said to wear it

3 The name of the OT
In the second example Mel (MZ), talking to her boyfriend (A) becomes stuck on a question she is trying to ask about what time he is to be fetched from a nearby clinic where he is being admitted for a minor routine procedure. In line 107, Mel initiates the question but finds herself repeating the same phrase ‘what happens if he sees you’ when she meant ‘fetches’. In the same turn, Mel gives up the communication attempt by making a loud noise (MMM) and then burying her head in her hands (a common outcome of her tendency to perseverate and frequently noted in her interactions throughout the study). A allows her some time to regroup her thoughts and encourages her to try the question again (108) which she does with success in line 109.

Example iv

107 MZ wh- what if uh uh (.) no↓ yes↑ uh <what if thing there uh sees you at uh> five ‘o clock
what happens if Wiver sees you at agh! What if he sees you mm↓(2) MMM (buries head in hand)

108 A relax relax slowly (3) take a deep breath and (.) talk again

109 MZ um <what happens if I*** fetches you at five ‘o clock> or six ‘o clock? what
happens then?
6.2.2.5

Response inhibition - summary

Response inhibition essentially manifests as perseveration – an inability to stop an ongoing response or prevent a previous response from reoccurring in the face of changing contexts. The literature related to language abilities is heavily weighted towards naming. In this study, perseveration was noted for some individuals during spontaneous speech, naming and writing tasks, but these were not consistent with the findings from the performance on Trail Making. Instead the WCST was shown to be more sensitive to the measurement of this dimension. As with interference control above, there was also greater association between perseveration and conversation than other language measures. Impaired inhibition in conversation was reflected in both talkativeness and perseveration. The only inconsistent result was Jeannette’s, who performed well on the WCST, but presented with an idiosyncratic phrase which recurred in her spontaneous output. However, conceptually, she did not present as perseverative in the same way that John, Mel and Paul* did nor was she unable to inhibit ongoing responses like Cecil, Felicity, Jane* and Margaret. In this sample it appears that impaired response inhibition has conversational associations that are clearly identifiable. A discussion regarding talkativeness in elderly individuals cautioned against over-interpretation of this feature and highlighted the complexity of variables that may contribute to garrulousness.

6.2.2.6   Working memory

Working memory is the ability to hold information and events in mind while manipulating that information. Furthermore it is the ability to reach into the past (retrospective memory) and project into the future (prospective memory) in order to inform and organize behaviour. Table 6.9 provides a reminder of the tests used to assess this construct.

Table 6.9 - Tests of Working Memory

<table>
<thead>
<tr>
<th>CONSTRUCT</th>
<th>TESTS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ii) Medical College of Georgia complex figures (Spreen &amp; Strauss, 1998)</td>
</tr>
<tr>
<td></td>
<td>iii) Digit Span (backwards) (Lezak et al., 2004)</td>
</tr>
<tr>
<td></td>
<td>iv) Wisconsin Card Sorting Test (Ormond Software Enterprises, 1999)</td>
</tr>
</tbody>
</table>
Working memory – results

The four tests produced variable results. Grace and Tumi were the only participants who performed within normal limits on the Self Ordered Pointing Test (SOPT) during the baseline phase (See Appendix table N.4). While for the complex figures task Cecil, John, Jane* and Margaret were unable to recall sufficient details to score adequately (Appendix tables N.5 and N.6). Grace and Tumi along with Jeannette managed an average number of sorts on the Wisconsin Card Sorting Test (WCST) (Appendix tables N.7 – N.9). Finally, Felicity was the only participant to manage the digits backwards task (Appendix table N.10). Table 6.10 below summarises performance for each participant on the three tasks.

Table 6.10 – Performance on working memory tests

<table>
<thead>
<tr>
<th>Aphasia Type</th>
<th>Participant</th>
<th>Self Ordered Pointing Test</th>
<th>Complex Figures</th>
<th>WCST</th>
<th>Digits Backwards</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>x</td>
<td>√</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>√</td>
<td>√</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>x</td>
<td>√</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>√</td>
<td>√</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>x</td>
<td>√</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>x</td>
<td>√</td>
<td>x</td>
<td>√</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
</tbody>
</table>

Key: (√) Intact; (x) impaired

All participants (with the exception of Felicity) found digits backwards the most taxing. Participants who failed the complex figures also failed the SOPT and the WCST. As with digits forward, the pointing version of this test did not produce different results for individuals with Apraxia. Although it is not considered a verbal test by many clinicians and researchers, the verbal component of the digits backwards task appeared to make it significantly taxing (although according to Lezak et al, (2004), this test is also a good indicator of visual tracking skills, as successful performance requires the visual manipulation of numbers in working memory).
Working memory and language

Working memory has alternately been conceptualized as a single resource pool for attentional, linguistic and other executive processing and as a conglomeration of separate working memory abilities for different types of linguistic information. Research results suggest that working memory abilities for different types of linguistic information can be measured in adults with aphasia and therefore favours theories postulating separate working memory abilities for different types of linguistic information (Wright, Downey, Gravier, Love & Shapiro, 2007). This position is strongly supported by Friedmann and Gvion (2003) who showed that there is a highly complex relationship between working memory limitations and sentence comprehension. Their research suggests that the type of reactivation required by a sentence as well as the type of memory overload experienced, are critical determinants in whether or not sentences will be accurately processed. Such theories form the basis for the reasoning that working memory deficits may contribute to language processing difficulties. Strong correlations between reading comprehension, working memory capacity and language functions support the notion that the ability of individuals with conduction aphasia to comprehend language is predictable from working memory capacities (Caspari, Parkinson, LaPointe & Katz, 1998).

Spontaneous speech

In this sample, classifications of aphasia provide a general framework that predicts spontaneous language functioning. Previous research has associated working memory with the following aspects of spontaneous speech. In a group of individuals with mild aphasia, phonemic and semantic paraphasias with self correction were attributed to impairments of the phonological loop and central executive components of working memory (Ronnberg, Larsson, Fogelsjoo, Nilsson, Lindberg & Angquist, 1996). In TBI working memory has been shown to be associated with naming skills in verbal fluency tasks (Bittner & Crowe, 2007). In addition, deficits in verbal working memory have been associated with Apraxia in individuals with Parkinson’s disease (Howard, Binks, Moore & Playfer, 2000). Adams and Gathercole (1995) demonstrated that children with good phonological working memory produced speech that was significantly more grammatically complex, contained a richer array of words and included longer utterances than children with limited phonological working memory capacity. Likewise Radanovic, Azambuja, Mansur, Porto and Scaff (2003) predict that working memory deficits may impair sentence planning, leading to verbal production and vocabulary acquisition disturbances. Table 6.11 on the following page provides a summary of spontaneous speech features for each of the
Table 6.11 – Features of spontaneous speech associated with working memory and performance on working memory tests.

<table>
<thead>
<tr>
<th>Aphasia Type</th>
<th>Participant</th>
<th>Paraphasias</th>
<th>Apraxia</th>
<th>Grammatical Complexity</th>
<th>Anomia</th>
<th>Length of Utterance</th>
<th>Digits Backwards</th>
<th>Nonverbal Working Memory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>√</td>
<td>-</td>
<td>Reduced</td>
<td>√</td>
<td>Reduced</td>
<td>Impaired</td>
<td>Impaired</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>√</td>
<td>√</td>
<td>Reduced</td>
<td>√</td>
<td>Reduced</td>
<td>Impaired</td>
<td>Inconsistent</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>√</td>
<td>-</td>
<td>Reduced</td>
<td>√</td>
<td>Reduced</td>
<td>Impaired</td>
<td>Intact</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>√</td>
<td>-</td>
<td>Reduced</td>
<td>√</td>
<td>Reduced</td>
<td>Impaired</td>
<td>Impaired</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>√</td>
<td>√</td>
<td>Reduced</td>
<td>√</td>
<td>Reduced</td>
<td>Impaired</td>
<td>Inconsistent</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>√</td>
<td>√</td>
<td>Reduced</td>
<td>√</td>
<td>Reduced</td>
<td>Impaired</td>
<td>Intact</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>√</td>
<td>√</td>
<td>Reduced</td>
<td>√</td>
<td>Reduced</td>
<td>Impaired</td>
<td>Inconsistent</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>√</td>
<td>-</td>
<td>Reduced</td>
<td>√</td>
<td>Intact</td>
<td>Impaired</td>
<td>Impaired</td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>-</td>
<td>-</td>
<td>Intact</td>
<td>-</td>
<td>Intact</td>
<td>Intact</td>
<td>Inconsistent</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>√</td>
<td>-</td>
<td>Reduced</td>
<td>-</td>
<td>Intact</td>
<td>Impaired</td>
<td>Impaired</td>
</tr>
</tbody>
</table>

Key: (√) Feature present; (-) feature absent
participants that have in the past been associated with working memory abilities. The table also presents their working memory skills divided into performance on the verbal working memory task (digits backwards) and performance on the nonverbal tasks.

The most striking feature of the table is the uniform presence of expressive language difficulties in the presence of difficulties with digits backwards. Without exception, all those participants who presented with reduced grammatical complexity, reduced length of utterance, anomia and paraphasias were also impaired on digits backwards. Even Jane* who was not aphasic presented with reduction of grammatical complexity. Felicity, who was the only individual who did not present with any of these difficulties was also the only participant to manage the digits backwards task. At the risk of oversimplifying the data, it is clear that classic aphasia symptoms observable in the majority of the sample including paraphasias, anomia and reduced complexity and length of utterance co-exist with deficits of verbal working memory. While the link between receptive aspects of speech has begun to enjoy widespread acceptance, it appears that aspects of working memory (particularly the phonological loop) may influence expressive speech as well. Differential performance on the three nonverbal tasks may reflect a hierarchical relationship with the complex figures being a less difficult task than the SOPT and WCST. It probably also represents the fact that the Complex Figures Test does not only rely on strategic memory but on a range of other skills that may in fact have facilitated performance due to the intactness of other cognitive domains in this group of participants.

**Comprehension**

The relationships between working memory and comprehension and language processing are far better developed than its expressive counterparts and reference was made to such deficits in Chapter 4. A growing body of evidence attributes deficits in working memory to sentence comprehension difficulties (Friedmann & Gvion, 2003; Moser, Fridriksson, & Healy, 2007; Radanovic et al., 2003; Rochon, Waters, & Caplan, 2000; Waters & Caplan, 2005; Wright et al., 2007). The availability of long term memory has also been found to mediate relationships of both working memory and background knowledge with listening comprehension (Was & Woltz, 2007). Working memory capacity has further been shown to impact on the ability to process higher order and figurative language such as metaphor comprehension (Chiappe & Chiappe, 2006), proverb comprehension (Moran, Nippold, & Gillon, 2006) and the ability to make inferences (Moran & Gillon, 2005).
In this sample although the participants all presented with essentially preserved comprehension skills on the WAB, one might expect that poorer scores for working memory would also coincide with relatively poorer scores on the comprehension sub-tests. However this was not the case. Table 6.12 ranks the participants in terms of their comprehension scores from highest to lowest, with their performance on the working memory battery. The lack of a pattern of associations is evident.

Table 6.12 – Ranked comprehension scores and performance on working memory tests

<table>
<thead>
<tr>
<th>PARTICIPANT</th>
<th>COMPREHENSION SCORE</th>
<th>PERFORMANCE ON WORKING MEMORY TASKS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>SOPT</td>
</tr>
<tr>
<td>Felicity</td>
<td>10</td>
<td>x</td>
</tr>
<tr>
<td>John</td>
<td>9.95</td>
<td>x</td>
</tr>
<tr>
<td>Mel</td>
<td>9.5</td>
<td>x</td>
</tr>
<tr>
<td>Jane*</td>
<td>9.4</td>
<td>x</td>
</tr>
<tr>
<td>Cecil</td>
<td>8.8</td>
<td>x</td>
</tr>
<tr>
<td>Grace</td>
<td>8.2</td>
<td>√</td>
</tr>
<tr>
<td>Margaret</td>
<td>8</td>
<td>x</td>
</tr>
<tr>
<td>Tumi</td>
<td>7.9</td>
<td>√</td>
</tr>
<tr>
<td>Paul*</td>
<td>7.65</td>
<td>x</td>
</tr>
<tr>
<td>Jeannette</td>
<td>7.45</td>
<td>x</td>
</tr>
</tbody>
</table>

Key: (√) performance intact; (x) performance impaired

Naming

The role of working memory in facilitating naming or word retrieval is also attracting interest. In a study by Fillingham, Sage and Ralph (2006), two groups of people with aphasia were compared in terms of their response to naming therapy using either an errorless or errorful treatment design. They found comparative results for the two groups regardless of language skill. However, significantly, participants who made more progress during errorful learning were those with better working memory skills. The critical role that working memory and executive factors play in the recovery of word retrieval in aphasia has been highlighted.

Improvements in word finding ability, in response to training, has been attributed to working memory functions by other investigators (Crosson, 1999). Cornelissen, Laine, Tarkiainen, Jarvensivu, Martin and Salmelin (2003) state that training effects reflect more effective phonological encoding and storage of trained items through the engagement of a left
hemispheric word-learning system. This reflects the link between left inferior parietal lobe activity to the phonological storage component of verbal working memory. However, as with the comprehension scores, there was no pattern of higher naming scores being associated with an overall better working memory profile. In fact some of the participants like Jane* who presented with the highest naming score on the WAB also presented with the worst working memory profile.

**Repetition**
Reduced working memory capacity has been put forward as one of the primary explanations of poor repetition ability in individuals with conduction aphasia and is in fact defined by some as a phonological short term memory deficit, resulting from an increased activation decay rate (Koenig-Bruhin & Studer-Eichenberger, 2007). However, once again, the language results did not bear out this prediction consistently. While some individuals with conduction aphasia like Margaret performed consistently poorly on all tests of working memory, others like Grace performed relatively well and had difficulty only with digits backwards. In addition, working memory deficits were not confined to those individuals with conduction aphasia but were present in all the participants to some extent, suggesting that perhaps working memory, like attention, is particularly vulnerable to brain damage.

**Reading**
Working memory has a demonstrated impact on reading abilities in children, young and older adults as well as in individuals with aphasia. In children both the phonological and the executive systems are important predictors of age-related changes in reading (Swanson & Howell, 2001). In addition working memory, specifically efficiency of the central executive, has been found to be predictive of word recognition and reading comprehension (Cain, Oakhill & Bryant, 2004; Swanson & Ashbaker, 2000). A study examining the effects of aging on reading found that it was working memory capacity and meta-comprehension components rather than age that accounted for different patterns in comprehension of texts (De Beni, Borella & Carretti, 2007).

In a previous study strong correlations were found between working memory capacity, language functioning and reading comprehension in individuals with aphasia (Caspari et al., 1998). However in this sample this was not the case. As with the attention scores individuals
with good reading performance did not necessarily have intact working memory capabilities, while good working memory performance were not associated with higher reading scores.

**Writing**

Writing is a complex multi-component process during which a number of sub-processes compete for working memory resources (Kellogg, 2001). As with the reading scores, no consistent relationships were evident between reading scores and performance on the working memory battery in this sample. Previous research has highlighted the importance of working memory in the writing process to facilitate planning, writing and revisions and construction of a macrostructure (Olive, 2004; Vanderberg & Swanson, 2007).

**Working memory and conversation**

Working memory deficit typically involves the inability to recall new information or maintain and manipulate current information. Based on this, several conversational characteristics have been predicted or observed in individuals with TBI: inability to maintain the thread of social interactions (Dickinson & Givón, 1997; Hartley, 1995; Gillis, 1996; Schapiro & Sacchetti, 1993); difficulty using cohesive ties (Mentis & Prutting, 1987); repetition of ideas or statements (Hartley, 1995); impaired recall of shared information (Hartley, 1995); disorganized discourse (Hartley, 1995); difficulty integrating new information (Hartley, 1995) and providing only limited information when asked (Hartley, 1995).

In individuals with dementia, working memory deficits are frequently held accountable for decreased coherence in conversations (Dijkstra, Bourgeois, Allen & Burgio, 2004; Mcpherson, Furniss, Sdogati, Cesaroni, Tartaglini & Lindesay, 2001) and decreased ability to keep track of multi-party interactions (Alberoni, Baddeley, Dellasala, Logie & Spinnler, 1992). The conversational extracts below demonstrate some of these features considered characteristic of working memory deficit.

**Inability to maintain the thread of social interactions**

In the first, Felicity (FG) shows a classic example of being unable to maintain the thread of discourse in conversation with the researcher (R). She begins the thread of conversation by saying that she finds it hard to concentrate and that she has a tendency to get sidetracked (turn 039). She provides an insightful analogy of putting something “on the back burner” but not
being able to bring it forward again. She then starts to give an example of what happens to her when she gets sidetracked with the words ‘cos what I often do…’ (turn 041) but then instead becomes distracted and veers off into a discussion on positive thinking.

Example i

039 FG I know that sometimes (.) I don’t concentrate and I have the experience that- that I allow myself to be sidetracked.
040 R mm
041 FG I do- (1) instead of keeping >whatever it was< on the backburner and bringing it back forward again, I can’t seem to do that anymore. ‘cos what I often do when people think I’m sleeping all the time° (1) or just having a power sleep ° or whatever° is I practise my lateral thinking.
042 R oh ok
043 FG from this there’s an arrow to that and sometimes THAT only seem connected to me but sometimes I think that if I used those as an illus- illustration in a talk↑ they might be quite good for some(.)body else. For instance for (2) I’ve been thinking of positive, of positive thinking
044 R mm↑ hm↓
045 FG cos that’s what I think makes su↑ch a difference to one’s attitude

Repetition of ideas or statements

In the example below, Mel (MZ) and her boyfriend (A) are talking about an event at a local synagogue to which A is going on Saturday. Mel enquires about which synagogue it is but A does not remember. A begins talking around the word he cannot recall by providing shared information to Mel. However she can not make use of it and instead repeats her questions twice more (059 and 075) before understanding that A does not remember the name of the suburb.

Example ii

054 A Saturday morning I***’s picking me early and we going to shul° to
055 MZ Um What shul?
056 A To to uh ts! (sighs) (2) um (3) to >what’s his name< the one they going to perform the chupah°
057 MZ yes what-
058 A he did he did uh T*** chupah as well
059 MZ what shul?
060 A Uh sh- it’s it’s Chabad° shul in ts I can’t think of the place chabad shul in uh
061 MZ Oh
062 A Oy °what’s wrong with me° I can’t think of the place uh A*** S***
063 MZ Yes?

---

4 Synagogue
5 A Jewish wedding ceremony
6 A Hassidic sect
Impaired recall of shared information

In an attempt to make conversation with John (JH), his friend (M) asks about their shared experience of having lunch together with a group of familiar friends. Initially, John struggles to recall the event at all (047), then cannot remember who else was with them. M becomes noticeably upset and tries to prompt John into remembering (058, 060, 064 and 066) by asking the names of the people who were with them. As the conversation progresses M identifies their common friends and names them, at which point John recognizes them (069) and later becomes angry at the implication that he did not know who they were (095).

Example iii

046 M What did you get up to on Saturday?
047 JH uh (4) wait uh (looks up, points to M, rubs head) (3) no (holds up hand in wait gesture, one finger up pointing) (4) "eating" eating (3) (points directly at M) M*** (4) you (.) you eating.
048 M "mm.." (2) who was all there that you (.) remember? (4) that you know of there? at the lunch (5)
049 JH I don’t know (shakes head)

A tea
(5)
050 M You came to my house on Saturday for lunch↓
051 JH >Yes yes yes< (nods head)
052 M and (. ) who was there that you (1) know?
(6)
053 JH four people (holds up four fingers)
054 M mm hm.
055 JH no THREE people (holds up three fingers) (2) no no. (looks up hand in wait gesture)
056 M ok and and their names?
057 JH I don’t know (shakes head)
(2)
058 M But you know↑ I mean
059 JH Mm↓ (shakes head)
060 M But you know >you reco- you know them you know< five people at the (1) lunch
061 JH Mm.
062 M who’d you know?
063 JH Two girls (holds up two fingers) and (1) boy (.) nothing um
(4)
064 M But all those that you know↓ that were at the lunch,
065 JH Yes yes
066 M What are their names?
067 JH >I don’t know< (shakes head, shrugs shoulder) (. ) nothing M***
068 M And H***? surely↑ you know H***,
069 JH JA:: ja
070 M ok↑
071 JH oH***o
072 M ja.
[portion of transcript omitted]
092 M Oh >well when I was asking< who you knew there >you said you didn’t know
anybody’s name< you knew who they were↑
093 JH ah↑ (head to side shrugging and shaking head)
094 M You didn’t know H↑***↑ you didn’t know↑ Ch***.
095 JH NO (looks up and pulls head back, face shows denial or rejection of what M is saying)
096 M Oh and who else was there that you knew? th- that you know?

Disorganized discourse

Working memory skills allow conversational partners to relate stories in a coherent and organized manner by allowing them a perspective of past events. In the example below, Margaret demonstrates how difficult it is for her to relate a series of events in a sequenced manner. She (MS) is telling the researcher (R) about the fact that her granddaughter took a balcony key and went onto the balcony by herself, refusing to allow Margaret access, by holding the door closed. Subsequently, Margaret managed to prise her fingers off the door and gained access to the balcony. She then gave her granddaughter a light smack and phoned her
daughter to have a talk with the granddaughter, after which things settled down. In the telling, the sequence of events is jumbled starting with the fact that after she had taken the key, Margaret smacked her. She proceeds to say that her granddaughter had started crying at which point she threatened to call her daughter. In the middle of the narrative she inserts some irrelevant details about who carries spare keys in the complex. She returns to the fact that she had called her daughter. Only at this point towards the end of the narrative does she present the initiating event, which began with the child closing herself behind the balcony door. In the telling the story was difficult to follow because of the lack of organization and coherence.

Example iv

110 MS We::ll you know what happened? um I was in the bathroom and she she unlocked the key like it, and then she was crying because I gave her I gave her- the leg a little (slaps leg)
111 R Mm.
112 MS Smack and she went waah! And you know↑ here from one o clock till till three
113 R oh its quiet
114 MS it’s supposed to be ja, and she started Waah and then I said to her, I’m going to get hold of mommy now and you going to listen to her because I thought what ELSE can I do?
115 R ja
116 MS I couldn’t get hold of the key and I was quite, (1) I was really worried about it and I thought now what if I don’t get hold of the key?
117 R Ja
118 MS But of course the sister there must be >something that we can use< that you know I↑ I’ve got the normal key that you have,
119 R uh
120 MS for for the door and whatever but um(.) they must have another >anyway<
121 R Did she give it back to you?
122 MS She did. She listened to me
123 R Oh ok
124 MS With my daughter (1) and then she spoke to her. she spoke to to her
125 R Ja
126 MS and she said to her now you go and have a little bit of a rest and you leave Granny alone for a while because-
127 R right
128 MS especially with the balcony
129 R ja
130 MS she started doing this↓ you know. I mean she’s got all strapped out and whatever
131 R oh
132 MS and she got(.) she wouldn’t let open the- she wouldn’t let me. the balcony wouldn’t let me you know she just
133 R oh she was just holding onto the door and keeping it closed
134 MS that that’s↑ it↓and I said you must come here because I’m must- I must close that. She
wouldn’t let me so in the end I had to prise her her? (gestures with one hand pulling the fingers of the other hand)

135 R finger
136 MS fingers, and in the end I got hold of her and I smacked her
137 R mm
138 MS and that’s what started it ooaa! I had a terrible time.

**Difficulty integrating new information**

Cecil (CS) and his wife (RS) are talking about their day. RS speaks about a new shop that has opened in their area. She introduces the topic by setting the scene in familiar territory orienting Cecil to the place she is referring to (line 125). Cecil responds with ‘right’ (Line 126) suggesting that he has followed this information. But seconds later he asks his wife where the shop is located (130) suggesting that in fact he had not integrated the information the first time it was presented.

**Example v**

125 RS They’ve opened up here in (. ) Sandringham do you know where all the kosher shops are?
126 CS Right.
127 RS It’s a kosher shop, selling just milk products
128 CS Yes?
129 RS And they’ve got a few tables and chairs
130 CS Where abouts?
131 RS Next door to the fish place, by you↑ know↓ Tiberius? you know where I get the fish from? right next door.
132 CS Right

**Providing only limited information when asked**

In this final example, John (JH) is trying to get his friend (M) to recall the name of a place they visited together on holiday. M responds by putting forward several guesses and asking questions that may clue him as to what John is talking about. Despite this shared experience, John finds it difficult to provide useful information, volunteering nothing and leaving the burden of the conversation to M. This extract is interesting specifically because John’s expressive skills are actually better than reflected in this conversation. Yet he appears unable to integrate past experience to inform the current interaction, almost giving the impression of being distinctly unhelpful and uncommunicative.
Example vi

192 M What was happening there?
193 JH Um I don’t know
194 M You talking about something else around there?
195 JH >Yes yes yes yes< um:::
196 M Where we went to?
197 JH >Yes yes<
198 M When we were there? and we drove through?
199 JH No
200 M Free state?
201 JH Yes yes (2) no?
202 M Kruger Park?
203 JH Uh:: who was there? I don’t know. um:::
204 M In that area?
205 JH >YES yes yes yes<
206 M In the Kruger Park lodge?
207 JH No
208 M Winkelspruit?
209 JH No
210 M Um one of those towns?
211 JH >Yes yes yes<
212 M I don’t know. it’s another town in that area?
213 JH >Yes yes yes<
214 M There (. ) White River?
215 JH No
215 M Graskop?
216 JH No
217 M Sabie?
218 JH No
219 M There’s like that you stayed before?
220 JH No no
221 M Visited?
222 JH Yes ONCE
223 M Oh Pilgrim’s Rest
224 JH PIL(.)GRIM’S REST
225 M You talking about that grave?
226 JH >Yes yes yes<
227 M With the soldier?
228 JH Yes
229 M Who’s feet had to face east west and the rest facing north south? Ja.
230 JH Robbers
231 M Robbers Grave ja
232 JH Long ago long ago ah

6.2.2.7

Working memory – summary
Years of research have consistently produced findings that working memory impacts on language functions, including those tested on the WAB. Yet in this sample with these participants, expected associations were not always found. The most consistent result to emerge was the presence of phonological loop deficits in the presence of expressive language difficulties in spontaneous speech. This was well contrasted with Felicity’s results who, in the presence of intact verbal working memory, was the only participant to present with intact expressive language across all areas assessed. The other results were variable with no clear pattern relating to language profiles. These findings suggest several things. First, that working memory is highly susceptible to brain damage and is present throughout the sample in different ways, irrespective of site of lesion or type of aphasia. This is further reinforced by the presence of working memory deficits identified in other diffuse and connection pathologies such as dementia and multiple sclerosis. Second, that the complex relationship between attention functions and working memory may affect these results as the latter is to a large extent dependent on the former. Third, different language tasks may yield different results and the WAB may not be sufficiently taxing to uncover working memory deficits. Finally, it is clear from the contrasting results on the EF battery that different elements of working memory and other cognitive functions were tapped by the tests chosen. It would therefore be inappropriate to discuss findings in terms of a uni-dimensional construct of working memory. Rather there seems to be at least one clear distinction between working memory capacity tapped by digits backwards reflecting capacity of the phonological loop of working memory and the nonverbal tasks reflected by the SOPT, representative of the central executive and visual sketchpad. Consistent with this proposal, expressive difficulties including anomia, the presence of paraphasias, reductions in length and complexity of utterance all co-existed with poor performance on digits backwards. The nonverbal assessments, specifically performance on the SOPT and WCST, appeared to have more consistent manifestations of organizational deficits and structural impairments in conversation, coinciding with a more executive component of working memory in terms of the ability to integrate, sequence and organize conversational contributions. In this regard all of the participants who demonstrated poor scores on the SOPT (with the exception of Jeannette, who scored well on the WCST) demonstrated working memory deficits in conversation. (Paul’s data is not included in this analysis as his expressive language was so limited. His lack of ability to use alternative or compensatory means of communication made interpretation of his conversation in this regard entirely speculative).
6.2.2.8 Regulation of affect

Regulation of affect refers to emotional self control and the ability to self-regulate drive, motivation and arousal in pursuance of goal-directed action (Barkley, 1997). Table 6.13 below presents the test used to assess this construct.

Table 6.13 - Test of regulation of affect

<table>
<thead>
<tr>
<th>CONSTRUCT</th>
<th>TEST</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regulation of affect</td>
<td>Neuropsychology Behavior and Affect Profile (Nelson et al., 1994)</td>
</tr>
</tbody>
</table>

Regulation of affect – results

There is virtually no existing precedent for examining communication behaviours with reference to disorders of affect. The Neuropsychology Behavior and Affect Profile (NBAP) (Nelson et al., 1994) measures disorders of the following emotional constructs: Indifference (Apathy), Inappropriateness (unusual or bizarre behaviour), Pragmatic deficits, Depression and Mania. Table N.11 in the appendix presents results for the ten participants. For the most part, the ten participants did not present with significantly disordered affect. The construct which is referred to in the NBAP as ‘Pragnosia’ refers to pragmatic difficulties. Some examples of questions weighted for this construct are: ‘The patient has a problem recognizing voices of friends and family; The patient tends to take conversations literally; The patient seems to miss the point of a discussion; In a conversation the patient does not seem to know when to stop and let others talk.’ Of the five constructs measured, inclusion of pragmatic deficit as an emotional construct is the least convincing although pragmatic aspects of communication are certainly affected by affective disorders. Still, identification of the other affective states make this tool a useful one.

In this sample, four individuals were characterized by their significant others as having a disorder of affect. Interestingly both John and Paul*, the two individuals with Broca’s aphasia were identified as being depressed, apathetic and having pragmatic communication difficulties. Jane* was characterized as being inappropriate and manic and Margaret was characterized as having pragmatic difficulties.
Regulation of affect - language and conversation

According to Barkley the ability to regulate affect allows for the creation of positive emotional and motivational states when angered, frustrated, disappointed, saddened, anxious or bored. These self directed actions may involve self-comforting, self-directed speech, visual imagery, and self-reinforcement. The absence of these regulating features would therefore result in difficulty modulating those same emotions and lead to excessive expressions of anger, frustration, lack of motivation when disappointed, sad, anxious or bored. This description is consistent with previous literature related to disorders of drive, mood and emotional expression.

With respect to disorders of drive and motivation, Luria (1980) described a loss of or weakness in drive, interest and motivation. This apathy and indifference to surroundings, described as a blunting of affect and emotional responses by Blumer and Benson (1975), represents disorders of self-activation (Laplane, 1990 in Stuss, Gow & Hetherington, 1992). As a result these patients have a tendency to demonstrate increased helplessness and therefore a greater dependence on others. During conversational interactions, these individuals may only produce restricted or minimal output, placing a greater burden on their conversational partner to carry the responsibility for the interaction (Miller, 1989 in Crockford & Lesser, 1994; Ylvisaker & Szekeres, 1994).

It is notable therefore that the two participants with the least ability to generate spontaneous output (John and Paul* Broca’s aphasia) were also characterised as being indifferent. There is a suggestion therefore that significantly reduced output in these two participants may have affective contributions, reflecting difficulty with initiation of activity and an inability to moderate drive and motivation in the pursuance of communicative acts. This is particularly evident in conversational characteristics, where reduced participation may in fact reflect reduced ability to engage in self-activation. This was clearly depicted in example vi above for John in conversation with his friend M (see working memory – providing only limited information when asked). The extract clearly demonstrates John’s inability to mobilize any resources to contribute to the conversation. He is instead a passive participant, waiting for M to generate the solution to his problem. The same is true of Paul* during his interaction with his wife (see response inhibition – perseveration). Despite the fact that he clearly has strong objections to wearing his brace, he is strikingly unable to mobilize any form of communication to indicate why this is the case.
Disorders of mood are also common in relation to brain injury, the most common of which is depression which is also associated with indifference and social withdrawal (Prigatano, 1986), consistent with findings from this sample. Acoustic and perceptual properties of depressed people’s speech have been identified as slower, quieter and more monotonous with less variation in prosody than that of ‘happy’ people (Alpert, Pouget & Silva, 2001; Barrett & Paus, 2002). This was not found in the current sample.

A less common result of focal brain injury, although more prevalent in right hemisphere lesions is mania, a characteristic identified in Jane*. She displayed irritability, compulsive and restless behaviour (she admitted to being caught shoplifting during the course of the study) as well as a shallow and immature sense of humour. There were times when she engaged in excessive self-disclosures, also consistent with mania as well as her characterization as being inappropriate (Gajar et al, 1984). Hartley (1995) in her work with individuals with TBI anticipates the following communication effects arising from disordered emotional regulation: inappropriate laughter, excessive talking, inappropriate sexual comments, excessive complaining and expression of anger, unpredictable social behaviour, non-purposeful behaviour that can be distracting to self and others and initiation of unnecessary questions. In example i below Jane* demonstrates several of these features. She (JR) and her cousin (B) are talking about a mutual relative who is not well and requires nursing care. In turn 089 Jane* laughs inappropriately, after a making a childish joke about the relative’s weight. In line 102, she makes a sexually loaded joke in response to the previous comment by her cousin after which she again laughs inappropriately. Finally in line 114 Jane* makes an inappropriate and unpredictable comment about how she could be involved in her relative’s care.

Example i

088  B     And I mean basically <G*** just needs to know: that somebody is sleeping> with- (1) in the house. you know? <if she needs any sort of nursing I’m sure they’ll, they’ll get a qualified personlush> because even you I mean you’ve said you’ll go and you’ll help and do или and J*** you could <never lift her> an:д: put her on the toi:let and things like that [ "No nobody could she’s quite a big girl" ]

089  JR     she’s a (laughs) BIG girl (laughs)

090  JR     [portion of transcript omitted]

100  JR     I don’t know↓

101  B     But they can always hire a care↑giver↓ (2)

102  JR     I can- I hire ‘em too you know.
Finally the pragmatic difficulties representative of the pragnosia construct on the NBAP, and which were identified for John, Margaret and Paul* have been discussed in relation to difficulties with inhibition and working memory above.

6.2.2.9

**Regulation of affect - summary**

It is difficult to draw conclusions about associations between language functions and regulation of affect. At most, we could suggest that in this sample, the expressive language deficits, which are more severely affected in the individuals with Broca’s aphasia (certainly in terms of fluency, length and complexity of utterance), may have contributions from deficits in drive and mood. In addition, some of the communicative features expressed in verbose, uninhibited and disorganized conversational discourse for Jane* may have contributions from disordered drive and expression of emotion. This is an area that certainly merits greater research interest and which could potentially have a profound impact on the way in which we view communication symptoms of aphasia as well as other neogenic communication disorders with known co-existing disorders of affect – such as apathy and depression in dementia or depression in multiple sclerosis.
6.2.2.10  **Internalization of speech**

This construct refers to the ability to engage in rule governed behaviour. It encompasses problem solving, planning, reasoning rule adherence and rule generation (Barkley, 1997). Table 6.14 below lists the tests used to assess this construct.

Table 6.14 - Tests of internalization of speech

<table>
<thead>
<tr>
<th>CONSTRUCT</th>
<th>TESTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internalization of speech</td>
<td>i) Tower of London (Shallice, 1982)</td>
</tr>
<tr>
<td></td>
<td>ii) Raven’s progressive matrices (Raven et al., 1998)</td>
</tr>
</tbody>
</table>

**Internalization of speech – results**

The tests in this section were chosen with extreme care owing to the fact that the contribution of internal speech is thought to play its most critical role in this domain and has been shown to be susceptible to disruption in individuals with aphasia (Keil & Kaszniak, 2002). Tests were chosen for their load on non-verbal reasoning skills and lack of reliance on verbal output or mediation. Despite this, most participants displayed impaired functioning on both tests. For the Tower of London, only two individuals, Grace and Tumi, performed within normal limits (Tumi particularly well, scoring well above average for this test). Results are presented in Appendix table N.12. However, on Raven’s progressive matrices nobody exhibited intact performance although Grace’s score was borderline (Appendix table N.13).

**Internalization of speech and language**

As with the previous section, there is virtually no research that has looked at the impact of planning (as in problem solving or reasoning) capacities on language functioning. ‘Planning’ as a notion is highly complex and exists on a continuum from the conceptual to the motoric. In the context of executive functions, the emphasis clearly falls on the conceptual nature of planning. The challenge to apply poor planning to varying levels of conversational output is considerable and would have to include planning in terms of more global features such as topic management to discrete aspects such as syntactic construction of sentences.

The most obvious finding on which to comment is the fact that Felicity whose speech retains fluency, complexity and length of utterance consistently scored the lowest of all the participants...
on Raven’s progressive matrices and was among the least successful on the Tower of London. Whereas several other participants displayed significant and consistent learning effects for the latter test, Felicity’s performance was impaired on three of the four occasions that the test was administered. There were no other associations to be drawn from performance on the WAB to performance on the EF tests of planning. This was predominantly because scores (except for Grace and Tumi) fell within the same range, in the presence of varying language profiles. Conversational features were however interesting to note as these appeared to be more sensitive to planning skill on a broader level.

Internalization of speech and conversation
The ability to use planning skill to manage various aspects of conversation had more consistent associations with the EF battery. According to Hartley (1995), poor planning would be reflected in disorganized, fractured discourse, lack of coherence, excessive details and numerous revisions and false starts. These features were evident in a number of the participants’ conversations. When impaired, these difficulties had a marked impact on topic management in terms of the ability to manage coherent and organized discourse related to a topic and make planned, logical topic shifts. Many had features of fractured conversation, where topics were initiated, but were not coherently related to the preceding topic and then would be abandoned before being elaborated (This was particularly true of Jane* although Cecil and Jeanette also demonstrated these features). Repair was also significantly affected in those with planning difficulties as they appeared unable to engage in the self-reflection needed to initiate a different strategy for communication (John in particular). These findings are notably contrasted with Grace and Tumi who despite not necessarily having better language skills, presented with better organizational and executive features in their conversations.

Table 6.15 on the following page summarises the findings of the planning tests for each of the participants as well as conversational features that reflect impaired planning ability.

Intact planning
Whereas the previous sections have presented examples of impaired performance, this section also presents conversational samples of the two individuals with intact planning skills. Example i below presents a repair sequence between Grace (GB) and her husband (RB), which reflects intact planning skills. They have been discussing the fact that Grace has some money that a
fellow patient had given her as part of a collection for the student therapists at the university where they both attend a clinic. Her husband has misunderstood where the money has come from and whom it is for as reflected in lines 067 and 068. Grace immediately plans a repair strategy in which she will use an external aid to convey the information she needs. She begins by instructing her son to retrieve a file from a different room for her (069). She then restructures the discussion and makes a meta-comment ‘let’s try again’ (line 070). She then repeats salient information to convey the essential core of the message (line 070). This immediately serves to clarify the main source of confusion (071). Furthermore, once she has the file, Grace locates the names of the people she is talking about to further clarify the protagonists in her story. The breakdown is successfully repaired and commented on (line 087).

Table 6.15 – Results of planning tests and associations with conversational features.

<table>
<thead>
<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>TOWER OF LONDON</th>
<th>RAVEN'S</th>
<th>CONVERSATION ASSOCIATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>x</td>
<td>x</td>
<td>Lacks organisation, can be fractured, excessive revisions</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>x</td>
<td>x</td>
<td>Lacks coherence and organization, excessive false starts and revisions</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>√</td>
<td>x</td>
<td>Uses external aids to assist coherence and organization</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>x</td>
<td>x</td>
<td>Decreased ability to engage in self reflection during conversation</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>x</td>
<td>x</td>
<td>Difficult to interpret, poor expressive output</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>√</td>
<td>Borderline</td>
<td>Uses aids and a number of strategies to create coherence and organization</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>x</td>
<td>x</td>
<td>Lacks coherence and organization, fractured</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>x</td>
<td>x</td>
<td>Lacks coherence and organization, excessive details, false starts and revisions</td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>x</td>
<td>x</td>
<td>Lacks coherence and organization</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>x</td>
<td>x</td>
<td>Lacks coherence and organization, fractured discourse</td>
</tr>
</tbody>
</table>

Key: (√) Intact; (x) impaired

*Example i*

067 RB But then why have you got her money if she’s going to go to Wits? (2)
068 RB or have I got the story all wrong?
069 GB (to her son off camera) please won’t you uh uh look with my um (1) file right next to my
desk

(talking to her husband again) uh let’s try again. yesterday J*** wife is
give me

Oh J***’s wife gave you the money

GB Yes

Oh not the Black lady

No is the let’s see oh thanks (taking the file from her son) I hope it’s in here

You started speaking about J***’s wife and then you started speaking
about the Black lady

Ja oh alright ok here

J*** H*** that’s the the old man that’s this one here

Ja ja ja (1) there’s

T*** Now Is that the- that the one you’re talking about? She’s the one writes the poetry

Ja like this this is a whole this new one

Ok so J***’s wife gave you some money cos they not going. J***’s not going to Wits

Ja they not going anymore

So this money you must give to T*** is that

Ja to give it to her cos she’s

Collecting for the students oh No I got the story wrong

ja I’m sure I got it all wrong (laughs)

no well we worked it out now

Tumi also uses external aids to assist her with the management and structuring of information in
discourse. In example ii below she (TD) is talking to the researcher (R) about a machine
available at a local clinic which is supposed to generate sensation and induce muscle relaxation.

When the researcher asks her what the machine is supposed to do (line 039), she responds by
handing over a pamphlet and instructing the researcher to read the information (040). The
ability to use external aids was significantly lacking in a number of participants who would
have benefited from them and who have used them successfully in therapy (Mel, John and
Paul*).

Example ii

AND THIS ONE good but now, I’m feeling this (indicates hand)

Oh really

Um But four o’ clock, run

what’s it supposed to do?

uh (1) and read (gives R pamphlet to read) but too much line and come back twelve o’
clock or one o’ clock eh taxi come back.

It takes too long and there too many people, waiting for them
Poor planning

The best example of poor planning during conversation came from Margaret. She (MS) and the researcher had been talking about Margaret’s son and his girlfriend. Margaret told the story of how she met the girlfriend under circumstances that was extremely embarrassing for her. However, the story is disorganised and includes excessive detail. The story begins incoherently in line 012. There are also excessive revisions during this turn and Margaret specifically makes a comment about needing to include extra information in order to make sense of the story, ‘I’ve got to explain something as well’, which in actual fact is irrelevant to the story she is telling about her son. In line 016 she makes another revision and starts the story again ‘Let me just rather...’ The extract is full of unnecessary and tangential detail related to her dog. This rambling account is deficient of planning and monitoring skills and is highly reflective of the fact that Margaret scored particularly poorly on the EF tests for planning.

Example iii

012 MS November the following um (2) uh uh um in March in March uh I uh all of a uh I had this a uh G***8 says to me, ‘Mom won’t you come and have a bit of uh um dinner.’ So I said, ‘oh ok wha- what is it for?’ and it was about I’ve got to explain something as well to make sense about what I’m gonna say my dog the big do we we had two
013 R Mm
014 MS And still the same ones you know now but he was a pup at that at that time now he’s um the eldest eh I mean the eh the eh the big dog he had a he went lame
015 R Right
016 MS And all of a sudden this is how it started let me just rather I I’ll tell you this and he said to me, ‘Mom do me a favour just go uh and see what’s wrong with him and he can’t he seems lame.’
017 R Ja
108 MS so I said, ‘Oh all right’ and after- when I finished what I had to do and then I’ll go there you know went there I noticed he’s absolutely lame and with the long paw like this and with nails scratching like this and and with blood.
019 R mm. oh ⁹no°.
020 MS And what on because you see because he was lame↑
021 R Right
022 MS He had dragged this (gestures with her hand to indicate dragging movement)
023 R Right
024 MS This is what happened. so in the end I managed to get hold of a vet and I said um, ‘I don’t know what’s wrong with the dog.’ but I said um which is couple of a few blocks away from here actually and I said if I mentioned it um I said, ‘he’s a big dog and I I can’t manage I can’t manage him.’
025 R Right
026 MS But I so they said to me ‘oh they have an ambulance but it’s gonna cost you nogal⁹

⁸ Margaret’s son
listen to this (laughs) um it’s gonna cost uh it’s gonna cost two hundred and whatever just for that couple of
027 R A few blocks
028 MS And I said, ‘No ways! I’ll wait for my daughter and she will she’ll come with me and because he’ll eventually he’ll get in the car which he’s done before and he’ll just lie there and then we’ll go to the vet. Why must I lose that? lose that?
029 R Spend all that money
030 MS Spend all that money and then I still don’t know what’s wrong with him
031 R Mm
032 MS And etc. anyway the whole story eventually after about it was after about two weeks the vet said um that uh he’s either got tic uh fever or um or somebody’s with the roof or something has been uh uh what do you call it um huh um ts! You know uh uh fix I mean stones or something have been thrown into the thing and hurt him or what was the other thing? oh and the other thing is that something to do with the way he was and I said, ‘No it sounded very strange.’ But anyway and then eventually my um after the then the vet said to me um ‘let’s just give it two days and we’ll see what’s wrong and we’ll give him m- pills and all this sort of thing and when he’s used when he’s when he’s a bit better we’ll see what he’s like and they wanted to put a uh uh ha like a harness
033 R Uh huh
034 MS So that you know so anyway so and then that the other vet was on was off
035 R right
036 MS so this man said to him the the the man vet said oh no um he says i- it’s gonna cost you at least ten thousand whatever and I s- and then I said to G*** you know that’s what this vet said. ‘Mom I can’t afford that.’
037 R ja
038 MS I can’t pay ten thousand or twelve thousand or whatever so sai- so I said well I don’t know what you- I don’t know what to do then but I’ll wait then the other vet comes because she said to me uh I’m sure that sh- she’ll go he will get better as he goes along but with this uh and then when he was off uh in again he was in again when he was what uh what do you call it when he’s not there not working
039 R Ja
040 MS then he’ll then she’ll be back
041 R right
042 MS the next day and when she came she said no let’s give it a bit of time and after a whi- after a week he was he was looking after- so much better
043 R mm
044 MS and he’s a beautiful dog he’s only three years
045 R ja
046 MS not even three years
047 R ja
048 MS so when this happened uh I said to G*** look I’ll um I’ll uh I’ll see what he’s like at the vet and but he seems to be getting better
049 R right
050 MS you know and and which he did
051 R ja
052 MS with pills and all this sort of this thing and the vet said to me instead of wasting leaving the poor dog there for a whole week I’m sure he’ll be alright with the pills and stuff

9 An Afrikaans word meaning ‘on top of that’
because you know everything the rest of him he was fine

anyway what happened on the Friday I said to to G*** look I’ll what I’ll do um I want you to come and actually have a look at the the- and he said to me, ‘Oh I haven’t got time to do that.’ so I said, ‘Why not?’ so he said, ‘because I can’t come tomorrow or whatever it will probably have to be a Tuesday’ and see the uh the difference uh um he’ll have to see the

the dog by then so I said, ‘oh but why can’t you come some time during the Saturday?’

no and then he didn’t explain anything you see this is why it made made it all funny he said, ‘Mom I want you to to go there on Saturday.’ but I I’ve um uh but I I’m going to I’m have we gonna do a bit of dinner ‘we gonna have dinner’ that’s all he said

so now I didn’t know what had happened but what had happened is apparently the the the girlfriend I hadn’t met her I didn’t even know that she had

yes so what I did I I I went in and the next thing I saw all these cars here and all these strange people here and balloons and what have you and I thought, ‘What the hell is going here G***?’ I said, ‘You said dinner.’

and I said now you you telling me that I must ‘Oh it’s E***’s it’s her thirtieth birthday.’

and you said, ‘Who’s E***?’

so I and then I didn’t know her either so I felt this big (shows thumb and finger in a “small” gesture) and I thought, ‘Now hang now I’ve got to walk into these people people.’ I didn’t know her mom and dad and all this sort and I didn’t know any of them nobody I just too-

why did he make it so awkward for you?

Yes and I I I felt terrible because I didn’t even have a present

I didn’t even know what it is about I didn’t know anything why couldn’t he just say to me um mom it’s gonna be E***’s it’s gonna be a surprise which they knew obviously they knew by the Saturday and of course I come in right in the Saturday and and I couldn’t do and I didn’t know what I I just (sighs)

10 The girlfriend
6.2.2.11 Internalization of speech - summary

The language results obtained from the WAB did not appear to have associations with the EF tests for planning and problems solving. However, all the participants whose performance on the EF battery consistently indicated poor planning skills also reflected a lack of organization and coherence in their conversational discourse. Once again, it is interesting to note the parallels between this finding and poor planning and disorganized discourse in dementia. The Tower of London appeared particularly sensitive to good problem skills which were reflected in the conversations of Grace and Tumi.

6.2.2.12 Reconstitution

Reconstitution refers to the analysis and synthesis of behaviour resulting in the ability to produce verbal and behavioural fluency as well as goal-directed creativity (Barkley, 1997). Table 6.16 lists the tests used to assess this construct.

Table 6.16 - Tests of reconstitution

<table>
<thead>
<tr>
<th>CONSTRUCT</th>
<th>TESTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reconstitution</td>
<td>i) Five Point Test (Spreen &amp; Strauss, 1998)</td>
</tr>
<tr>
<td></td>
<td>ii) Design Fluency (Spreen &amp; Strauss, 1998)</td>
</tr>
</tbody>
</table>

Reconstitution – results

Results of the five point test are presented in Appendix table N.14 while those for design fluency are presented in N.15. On the whole, participants were impaired on these tasks, displaying high percentages of perseveration on the five point test and producing very few unique designs on design fluency. The exceptions were again Tumi and Grace whose skills on both tests were intact.

Reconstitution and language

The only area of language use that has received attention relative to this executive function is verbal fluency. Research has suggested that the temporal cortex subserves word retrieval constrained by semantics, whereas frontal regions are more critical for strategic word retrieval constrained by phonology (Baldo, Schwartz, Wilkins & Dronkers, 2006). Verbal fluency as
measured by FAS tasks appears to be more dependent on the left frontal cortex whereas nonverbal tasks such as design fluency recruit both left and right hemisphere processes (Baldo, Shimamura, Delis, Kramer & Kaplan, 2001). Working memory capacity has also been implicated in verbal fluency tasks and is thought to account for perseveration when working memory capacity is overloaded (Azuma, 2004). Also, decreased scores on design fluency tasks have been associated with depression in a group of individuals with Parkinson’s disease following subthalamic stimulation (De Gaspari, Siri, Di Gioia, Antonini, Isella, et al., 2006).

On the WAB category fluency is assessed as part of the naming component of the test. Yet, given that letter fluency and not category naming is thought to recruit more frontal and executive resources, it is not surprising that there was no association between performance on the WAB and performance on the design fluency and five point tests. Spontaneous speech as measured on the WAB did not provide insight into nonverbal fluency performance either. One may anticipate that decreased levels of generative ability may also be associated with generally reduced language scores, especially in terms of expressive components. However, in the face of varying language profiles rates of perseveration were equivalent and the number of unique designs produced uniformly low, as again Felicity and Jane* who had the highest spontaneous speech scores also scored poorly on tests on both reconstitution tasks. Paul* who displays the most severely affected expressive language, presented with the highest number of unique designs, (after Tumi and Grace) and had amongst the lowest levels of perseveration on the EF battery tests for this construct.

One of the only other studies to be published associating elements of flexibility and creativity with language functions in general comes from Flaherty (2005), who suggested that frontal lobe deficits are associated with decreased idea generation in part because of inflexible judgments about an idea’s worth. This phenomenon finds its clearest expression in verbal creativity or lack thereof. Flaherty (2005) proposes that temporal lobe changes are associated with mania and increased idea generation often at the expense of quality while frontal lobe lesions produce sparse speech, cognitive inflexibility and depression. These trends are also thought to shape non-linguistic creativity. This theory was not consistent with the data obtained during this research. In this sample, the majority of participants presented with lesions which affected more than one hemisphere, sometimes involving both frontal and temporal hemispheres. While it is true that some participants displayed some characteristics predicted by Flaherty (2005) e.g. John
did in fact present with sparse speech, depression and decreased creativity, and Jane’s* behaviour was characterized as manic, the results of this research do not fit neatly into the framework proposed.

_Reconstitution and conversation_
Frankel and Penn (2007) found that in two individuals with TBI, poor scores for reconstitution were associated with perseveration during conversation affecting topic management. The inability to generate or initiate new topics of discussion or contribute meaningfully to topic shift was related to decreased capacity for generative and flexible behaviour on verbal fluency tasks. Importantly, this construct was found to be mediated by behavioural inhibition. Furthermore, Penn, Frankel, Watermeyer and Russell (under review) have suggested that reconstitution is reflected in the ability to generate and have available a number of communication strategies for adaptive and flexible use as well as the creative use of communication strategies. On the other hand, reduced ability would lead to decreased ability to generate alternate communication strategies as well as decreased verbal output in terms of amount of words and/or number of ideas. Thus deficits of reconstitution are hypothesised to have a significantly negative impact on topic management in terms of initiating topics and contributing to shift as well as repair.

Grace and Tumi, the two participants who displayed intact reconstitution skills, also demonstrated good ability to initiate new topics, contributed meaningfully to shift and used a variety of communication techniques to facilitate repair of breakdown. The examples below demonstrate the variety of strategies used by Grace (GB) to resolve communication breakdown with her husband (RB).

_Example i_
**Using external source to facilitate communication** – see example from Internalization of speech above.

_Example ii_
**Using writing**

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>019</td>
<td>GB</td>
<td>Steps um</td>
</tr>
<tr>
<td>020</td>
<td>RB</td>
<td>Steps?</td>
</tr>
<tr>
<td>021</td>
<td>GB</td>
<td>(writes)</td>
</tr>
<tr>
<td>022</td>
<td>RB</td>
<td>sketches?</td>
</tr>
<tr>
<td>023</td>
<td>GB</td>
<td>ja that sort of thing</td>
</tr>
</tbody>
</table>
**Example iii**

**Using circumlocution**

041 GB did have the other one was uh (4) u:m (1) um (3) “jam” no um (3) uh that that little in the (1) fridge um

042 RB Fishpaste?

043 GB JA that I used that as well I did so it was well good

**Example iv**

**Using gesture or enactment**

071 GB sorry but anyhow it’s I didn’t hear that much but he swore that from (. ) h-he got all this out again a-at the um (1)

072 RB all the putty out

073 GB ja uh:: (3) u:m (8)

074 RB leg?

075 GB NO sitting on your (acts out sitting and flushing) (. ) TOILET (laughs)

076 RB (laughs) oh on the toilet ok ja so all the putty he took out

**Example v**

**Breaking words up into component syllables to facilitate articulation**

043 GB …all that everybody knitting except me I took my::: um (3) tap uh (1) tap (. ) es (. ) try [ ^ostry^ ]

044 RB

045 RB That’s^ it.

In example vi Tumi demonstrates a number of topic shifts. The researcher (R) had asked about Tumi’s daughter. In response Tumi (TD) brought out a picture of her with her family and spent some time talking about her. The researcher then asked about the kind of work Tumi’s daughter does (line 056). In line 063 there is a good example of repair, where Tumi initiates the use of circumlocution to describe the type of her work her daughter does. She shifts the topic again in line 069 talking about the fact that her son-in-law also changed jobs to be able to earn more money. In 071 she introduces the fact that her daughter is divorced also adding where the ex-husband is from. In 079, Tumi sensitive to the slight lapse introduces the next shift, related to the fact that her daughter will be coming back to South Africa. The conversation progresses with Tumi elaborating, providing pertinent, relevant information.

**Example vi**

056 R And what does your daughter do there does she work is she

057 TD Ja eh university finish and then work eh um oh my God I I don’t know my husband knows eh em teacher but change now work somewhere in Toronto somewhere

058 R oh ok (. ) so does she teach? is that what she does?

059 TD no:: eh
no she did a teacher’s degree but she’s doing something else
>ja ja ja ja<
ok
eh em:: eh (1) eh same thing ehm (1) eh water food whatever
oh like to do with environmental issues
ja whatever
ok that’s interesting though
ja ja and then money too so this one (pointing to her son in law in the picture) change I don’t know what happened. but anyway
and she’s happy there?
ja but now divorced children shame eh YOUNGsters I don’t what happened. Why? Suddenly. I don’t know this one. eh husband Jamaica
oh
ja
she married a Jamaican man
ja
ja
but they Canada so now divorced so I don’t know
shame that’s sad hey
and now next year eh home
oh she’s coming to visit.
no eh
she’s coming back to live?
ja
oh really?
but eh embassy but works works ts! South Africa or something
so has she been? has she got a job offer or is she looking?
no no no ja ja
she’s gonna look for a job first
ja ja
and then if she can find work she’ll come back
June July August October November yes work but embassy eh work eh eh eh he told me eh eh work embassy in South Africa embassy check always
I know they they quite strict I think everywhere the emigration laws are very strict
Ja
Is she a Canadian citizen?
Ja Canadian. Me dual and her Canadian so
Oh so she’s got to- Is she applying for South African citizenship?
Ja
Or is she†-
Mm last year

In contrast, John and Paul* who both performed poorly on these tests on the EF battery also manifested reduced verbal output, inability to generate ideas either by initiating topics for discussion or contributing meaningfully to them or contributing to repair during breakdown (see
examples above). Repair was also significantly compromised in Jeannette and Mel, who both found it difficult to generate and use alternate strategies when a breakdown occurred. Mel has a tendency throughout the conversations recorded to become highly frustrated and to give up on repair attempts by saying, ‘I can’t talk.’ She needs to be supported and encouraged to make another attempt or engage in collaboration with her interlocutor. Example vii below presents an interaction between Jeannette (JD) and the researcher (R) where Jeannette is talking about her work with her ex-husband as a ‘spanner-girl’. The researcher does not understand what Jeannette has said and what results is a protracted repair sequence. Jeannette perseveres for the most part, (there is an initial reaction on her part to ‘leave it’ – line 170). She also collaborates with the researcher to some extent, attempting to repair by repeating what she has already said (line 176, 178, 189 and 209). However she is not able to initiate the use of a new strategy to facilitate the resolution until well into the interaction. She finally uses circumlocution but only after the researcher has finally put forward the correct interpretation (line 202).

Example vii

165 JD (2) and uh <uh:m uh skanner guh a skanner girl (2) a skanner girl
{shereez}> a boy-a boyfriend and a skanner girl u::m (3)
166 R ‘I’m not getting this’
167 JD BA
168 R ja?
169 JD and a skanner girl uh woody wood huh a car and a skanner girl uh a cars
(1) a cars
167 R he was- he sold cars?
168 JD no mm mm.
169 R he↑
170 JD um alright (waves hand in a ‘leave it’ gesture)
171 R oh he was a ca::r like he was a mechanic
172 JD mm
173 R he had his own body-shop and he (. ) looked after cars
174 JD mm
175 R and fixed cars
176 JD mm so woody wood huh spanner girl (1) a skanner girl uh woody wood a
skanner girl fixing the cars↑
177 R ja↓
178 JD BA and a skanner girl (hhh)
179 (3)
178 R so he had a degree he had a BA
179 JD no that one
180 R P*** did.
181 JD No THAT one (pointing to herself)
182 R YOU had a BA
183 JD mm hm
Another interesting finding from the conversational data was the fact that Felicity found it significantly difficult to initiate new topics of conversations. When the researcher introduced a topic, Felicity would often use this as a spring board to discuss a number of ideas related to the topic (often tangentially) until that particular thread was exhausted. Subsequently she was unable to initiate another topic of conversation and appeared insensitive to lapses in the conversation, instead waiting for the researcher to secure a new topic. Example viii demonstrates this. Felicity (FG) is talking to the researcher (R). Line 141 is the end of a previous topic where Felicity was saying how lucky she felt to be able to still read even after her stroke. After the researcher responds there is a three second gap, during which Felicity makes no attempt to say anything else. The researcher introduces a new topic about a video shoot that Felicity had participated in, which was supposed to be screened on a local television.

---

Example viii

Felicity (FG) is talking to the researcher (R). Line 141 is the end of a previous topic where Felicity was saying how lucky she felt to be able to still read even after her stroke. After the researcher responds there is a three second gap, during which Felicity makes no attempt to say anything else. The researcher introduces a new topic about a video shoot that Felicity had participated in, which was supposed to be screened on a local television.

---

11 Jeannette’s daughter
programme. The topic is exhausted by the end of turn 157 and there is again a long silence, which Felicity makes no attempt to break. The researcher circles back to the previous topic to add more information. This continues until line 169, at which point the topic is closed. After that there is a long 12 second lapse, during which Felicity again makes no attempt to break the silence. The video recording shows her to be sitting in her chair, looking passively at the researcher and then around the room. When it is clear that Felicity is not going to contribute a new topic, the researcher does so.

Example viii

141 FG Makes a big difference.
142 R Ja. (1) “it does.”
(3)
143 R Do you know? remember you told me that they did some filming, for (.) that pro↑gramme↓ that was going to be on on a Saturday morning?”
144 FG I know, it’s never come r- up yet.
145 R it hasn’t been ON?
146 FG I don’t think so.
146 R Because I actually <recorded> the programme that weekend, and then I never (.) got around to watching it and now

   [ ]

   Well don’t worry.

148 R And now↓ I don’t even know where it is. it’s on one of our tapes but I (.) I just never

   [ ]

   (laughs)

149 FG actually got around to↑ seeing itº
150 R Well don’t even look at it, it never came on
152 R It never↑ came on.=
153 FG =Lots of people listened to it from the beginning, to the end and then it- I never came on
154 R Oh↑ dear↓ so I wonder what they did with it?
155 FG I don’t know↑ maybe it just wasn’t suitable, or whatever.
156 R Oh. ok well now↑ at least I don’t feel like I missed out.
157 FG No.
(4)
158 R “Oh well”º
(7)
159 R I hadn’t (.) thought about it for a while, and then it occurred to me that I actually hadn’t, (1) “followed it up.”º
160 FG I think they said it was coming on sometime in Ju↑ly↓ and they were go↑nna↓ phone (1) S*** A**** from Stroke Aid, but I spoke to her last week, and she said she hadn’t heard a word.
161 R Mm.
162 FG She’s also the hell in↓ she’s been looking e↑very↓ Saturday.
163 R Oh shameº (laughs)
164 FG (laughs)
The other three individuals in the sample: Cecil, Jane* and Margaret do not demonstrate the same difficulties despite the fact that their scores for the reconstitution tests were also quite significantly impaired. These individuals to a large extent introduce many topics of discussion, which like Felicity, are usually not particularly well managed especially by Jane* and Margaret as discussed above. Nonetheless when breakdowns occurred, all three of these participants were able to either engage in self repair, or invite collaboration quite effectively in a proactive manner. It is possible that the substantial evidence of distractibility during conversational exchanges in these individuals overrides the limitations that may be imposed due to reconstitution difficulties. This supposition is consistent with previous research by Frankel and Penn (2007) which noted that the ability to generate new ideas and manage them appropriately once generated, is linked to the function of behavioural inhibition. Attention factors mediate the manifestation of reconstitution in complex activities like conversation.

6.2.2.13

<table>
<thead>
<tr>
<th>Reconstitution - summary</th>
</tr>
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<tbody>
<tr>
<td>As with some of the previous sections, there is virtually no previous literature that has examined the cognitive capacity for flexibility and generative skill in relation to specific language functions, with the exception of verbal fluency. Also, language scores from the WAB were notably inconsistent with the results from the EF battery. Conversational data provided greater consistency and there was evidence to suggest that some of the difficulty contributing novel and creative responses manifested at a conversational level, although this construct appears to be mediated, at least to some extent, by behavioural inhibition factors.</td>
</tr>
</tbody>
</table>
6.2.3 Results with reference to aphasia type

As demonstrated, results of the EF battery are difficult to integrate with the language profiles. Significantly, the majority of participants demonstrated notable impairments in most areas tested, regardless of language skill. There are inconsistencies in terms of executive features and expected language outcomes, with regard to the profiles of the participants. The fact that the WAB may not tax executive skills sufficiently to allow for comparable results is possibly the best account for these discrepancies, particularly when the conversational data appears to provide a more consistent fit with the EF findings.

An analysis of preserved function and impairment was undertaken in terms of types of aphasia to see whether any useful associations emerged. Table 6.17 on the following page presents a summary of the participants’ performance on the EF tests. In addition, there is a space under each participant’s name indicating whether these deficits were manifest in their conversations as discussed in detail above. As is evident, performance is quite variable within aphasia types. Some associations are more robust than others but are not exclusive to only one type of aphasia. In particular both verbal spans (forward and backward), reflective of immediate span of attention and verbal working memory skills respectively, were impaired in almost all aphasic individuals (with the exception of Mel who managed digits forward).

For the individuals with anomic aphasia, Cecil and Mel’s profiles are the most alike, although Cecil is slightly more impaired. While Mel is able to manage digits forward, she displays deficits in all areas tested with the exception of regulation of affect, where she and Cecil remain intact. Their conversational profiles are highly consistent with one another’s in terms of implicating similar EF deficits, although these actually manifest in very different ways. The only area of inconsistency is the lack of reconstitution difficulties in Cecil’s speech, which as mentioned before may be influenced or mediated by inhibition factors. Tumi on the other hand has a much stronger profile than either of them despite presenting with similar language characteristics – to Mel especially. A proposed hypothesis for this discrepancy is presented below in section 6.2.8.
Table 6.17 – Summary of EF results and conversational skills for all ten participants, grouped according to type of aphasia

<table>
<thead>
<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>INTERFERENCE CONTROL</th>
<th>RESPONSE INHIBITION</th>
<th>WORKING MEMORY</th>
<th>REGULATION OF AFFECT</th>
<th>INTERNALIZATION OF SPEECH</th>
<th>RECONSTITUTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x x</td>
</tr>
<tr>
<td></td>
<td>Conversation</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>√</td>
<td>x</td>
<td>x x</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>√</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>=</td>
<td>x x</td>
</tr>
<tr>
<td></td>
<td>Conversation</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>√</td>
<td>x x</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>x</td>
<td>√</td>
<td>√</td>
<td>x</td>
<td>=</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Conversation</td>
<td>√</td>
<td>√</td>
<td>√</td>
<td>√</td>
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<td></td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>x</td>
<td>x</td>
<td>√</td>
<td>x</td>
<td>x</td>
<td>x x</td>
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<td></td>
<td>Conversation</td>
<td>x</td>
<td>x</td>
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<td>x</td>
<td>x</td>
<td>x x</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>x</td>
<td>√</td>
<td>√</td>
<td>x</td>
<td>=</td>
<td>x x x</td>
</tr>
<tr>
<td></td>
<td>Conversation</td>
<td>?</td>
<td>x</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>x</td>
<td>√</td>
<td>√</td>
<td>x</td>
<td>√</td>
<td>√ x</td>
</tr>
<tr>
<td></td>
<td>Conversation</td>
<td>√</td>
<td>√</td>
<td>√</td>
<td>√</td>
<td>√</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>x</td>
<td>x</td>
<td>√</td>
<td>x</td>
<td>√</td>
<td>x x</td>
</tr>
<tr>
<td></td>
<td>Conversation</td>
<td>√</td>
<td>x</td>
<td>√</td>
<td>√</td>
<td>√</td>
<td>x x</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x x</td>
</tr>
<tr>
<td></td>
<td>Conversation</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>RHD</td>
<td>Felicity</td>
<td>√</td>
<td>x</td>
<td>√</td>
<td>x</td>
<td>=</td>
<td>x x</td>
</tr>
<tr>
<td></td>
<td>Conversation</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>√</td>
<td>x</td>
<td>x x</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x x</td>
</tr>
<tr>
<td></td>
<td>Conversation</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td></td>
</tr>
</tbody>
</table>

Key: (√) intact; (x) impaired; (=) inconsistent / variable responses across tasks; (?) insufficient basis for judgment
John and Paul*, the two individuals with Broca’s aphasia, present with similar EF profiles although John is more severely impaired. Both presented with impaired interference control (Paul* to a lesser extent), and although they managed set switching on the Trail Making test, both presented with perseveration on both the WCST and design fluency, which explains the presence of perseveration in conversation. In addition, they both experienced difficulty on working memory, planning and reconstitution tasks. Significantly, they share identical profiles for disturbances of affect. Despite having a marginally stronger EF profile, Paul’s expressive language is more compromised than John’s. Based on his output it is impossible to draw certain conclusions about more complex EF skills and their respective manifestations in conversation and therefore these can not be compared.

In the group of individuals with conduction aphasia, none of the participants have similar profiles. Margaret was significantly impaired in all areas of EF tested. In terms of affective disturbance, Margaret is perceived as being unable to cope with pragmatic demands of interactions. Conversational profiles are again consistent with EF results and nonverbal tests like the Stroop Colour-Word Interference Test and the nonverbal working memory tasks appear more predictive of conversational characteristics than the digit span tasks. Like Tumi, Grace stands out from this group as being intact in all areas with the exception of tasks requiring repetition of digits (forwards and backwards) and this is also discussed below. Jeannette’s profile is also not consistent with anyone else’s. Her executive skills are not as well preserved as Grace’s nor are they as markedly impaired as Margaret’s. Although she has been marked as presenting with perseveration in conversation, this is based on the recurrence of ‘woody wood’ in her spontaneous speech. This may in fact be more characteristic of the integral relationship between working memory and inhibition skills, which results in the uninhibited expression of a phrase, kept inappropriately active and primed in working memory.

Finally Felicity and Jane* who presented not with aphasia but with RHD and who had similar language profiles, demonstrated variation with regards both EF profiles and conversational characteristics. There were some similarities though in that both demonstrated poor performance on the Stroop Colour Word Interference Test, reflecting poor interference control. Both showed features of poor topic maintenance and a tendency towards tangentiality. Despite performing adequately on Trail Making, Felicity demonstrated perseverative tendencies on the WCST and both showed impaired response inhibition or shifting evidenced in excessively long
turns. While Felicity demonstrated interruptions and intrusions, Jane*’s conversation was characterized by topic repetitiveness. Their EF profiles were also differentiated in terms of the working memory tests with Felicity performing better on some measures. However, she too evidenced some working memory deficits which were evident in her conversation (such as losing the thread of conversation) while Jane*’s discourse was more fractured and disorganized, also reflecting poor planning. The similarities appear to end here, although both demonstrated poor reconstitution skills in the EF tests, Jane* did not actually manifest this deficit in conversation, whereas Felicity did (failure to introduce new topics). Also Jane* was perceived as evidencing significant disturbance of affect, whereas Felicity did not.

6.2.3.1

**Aphasia type - summary**

Initially it was hoped that profiling based on type of communication difficulty and the stratification of language performance into different types of aphasia and RHD would provide some understanding of the neurolinguistic contributions to conversational intelligence. However, although some relationships emerged, the data was too divergent on the whole to provide clear patterns. In some cases where there was both similar language and EF profiling, conversational features manifested in unique ways. In other cases, like in the group with conduction aphasia, three completely different profiles emerged. For Paul* language appeared to have a far more significant mediating factor on conversation than his EF profile. Two individuals stood out by having similar language profiles to other participants with similar types of aphasia to them, but relatively intact EF and high levels of conversational intelligence. On the whole language facility did not generally predict conversational skill, and similar EF deficits, manifested variably, arguing for specific and unique manifestations of alterations in brain functioning in response to brain injury, imposed on an already individualised system. The measurement of conversational intelligence therefore becomes an exercise in the description of a wide range of personalised factors and variables.
6.2.4 Results with reference to site of lesion

A growing body of neuro-physiological research using functional imaging techniques such as fMRI and PET scans has also contributed to the neurological sites implicated in performance of a variety of neuropsychological and executive tasks (Damasio & Anderson, 2003). Several strands of research have convincingly argued that cognitive control is not attributable to a single unitary system but rather emerges from the interaction of separable systems, which are responsible for complementary control functions (Gruber & Goschke, 2004). These systems include the prefrontal cortex, inferior parietal cortex and anterior cingulate cortex (Abutalebi & Green, 2007). The chief neural component is the prefrontal cortex which supports several classes of executive disorders, which have been differentiated roughly into behavioural and cognitive domains (Godefroy & Stuss, 2007). This distinction is compatible with two major functional/anatomical dissociations within the frontal lobes (Stuss & Levine, 2002). Cognitive aspects of EF are mainly supported by the circuit from the dorsolateral frontal cortex, involved primarily with spatial and conceptual reasoning and the behavioural component by the lateral orbital and medial frontal/anterior cingulated circuits involved in emotional processing. Exactly how these discrete regions and their differential connections contribute to the executive role of the prefrontal cortex remains to be delineated (Elliot, 2003). Figure 6.1 from Abutalebi and Green (2007) presents a schematic model of the areas involved in executive control.

Figure 6.1 – Schematic representation of areas involved in cognitive control
Furthermore, different disease processes affect specific sites of lesion differentially, which accounts for variations in clinical profiles related to cognitive and behavioural dysexecutive syndromes. Although, it is interesting to note, as discussed in chapter 4 regarding executive deficits in other neurogenic communication disorders, that the two features most consistently identified across all the disorders reviewed, were attention-related constructs (critical to cognitive executive functioning) and disorders of affect (representative to behavioural executive disorders). Table 6.18 below briefly summarises the existing literature in terms of whether or not deficits in each relevant area of EF have been identified. The table also highlights where no research exists yet. Also, neuropsychological studies have suggested that there is no unique system controlling all aspects of behaviour and cognition rather multiple executive processes that can be selectively impaired (Shallice & Burgess, 1996). Cognitive dysexecutive syndromes are characterised by deficits in response initiation and suppression, sustained attention, set shifting, divided attention and task coordination (i.e. behavioral inhibition), working memory and strategic memory processes, problem solving, rule deduction and planning (i.e. internalization of language) and deficits in information generation (i.e. reconstitution) (Godefroy & GREFEX, 2004 cited in Godefroy & Stuss, 2007). Behavioural dysexecutive syndromes on the other hand are characterised by global hypoactivity, global hyperactivity, perseveration, disturbance of emotion and social behaviour, anosognosia, disorders of sexual behaviour and confabulation (i.e. regulation of affect) (Godefroy & GREFEX, 2004 cited in Godefroy & Stuss, 2007).

Table 6.18 – Profiles of disorders of EF in neurogenic communication disorders

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Cognitive Dysexecutive Symptoms</th>
<th>Behavioural Dysexecutive Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Behavioural Inhibition</td>
<td>Working Memory</td>
</tr>
<tr>
<td>APHASIA</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>RHD</td>
<td>√</td>
<td>-</td>
</tr>
<tr>
<td>TBI</td>
<td>√</td>
<td>-</td>
</tr>
<tr>
<td>DEMENTIA</td>
<td>√</td>
<td>-</td>
</tr>
<tr>
<td>MULTIPLE SCLEROSIS</td>
<td>√</td>
<td>-</td>
</tr>
<tr>
<td>HIV/AIDS</td>
<td>√</td>
<td>-</td>
</tr>
<tr>
<td>TUMOUR</td>
<td>√</td>
<td>-</td>
</tr>
<tr>
<td>PSYCHOSIS</td>
<td>√</td>
<td>-</td>
</tr>
</tbody>
</table>

Key: (√) disorder identified; (-) no research yet conducted
Table 6.19 on the following page summarises recent research findings of neuroanatomical substrates for cognitive executive functions assessed in this research (Dagher, Owen, Boecker, & Brooks, 2001; Damasio & Anderson, 2003; Duncan & Owen, 2000; Graybiel, 2000; Leung, Skudlarski, Gatenby, Peterson, & Gore, 2000; Liu, Banich, Jacobson, & Tanabe, 2006; Mevorach, Humphreys & Shalev, 2006; Sumitani, Tanaka, Tayoshi, Ota, Kameoka, et al., 2006).

In terms of the ten participants for this study, site of lesion was initially inferred from types of aphasia or communication disorder, and was also confirmed through hospital records of brain scans and radiology reports. There is well established research indicating that Broca’s aphasia coincides with lesions of the left frontal lobe specifically the posterior aspects of the third frontal convolution (Caplan, 2003). Conduction aphasia on the other hand, represents a disconnection between the sound patterns of words and the speech production mechanism. Lesions are usually located in the arcuate fasciculus and/or cortical connections between Wernicke’s and Broca’s areas (Caplan, 2003). Finally, anomic aphasia has been associated with lesions of the inferior parietal lobe or connections between the parietal lobe and temporal lobe, although this type of aphasia may follow lesions in many different areas (Caplan, 2003). These broad delineations are reflected in this sample. As discussed in chapter 4 right hemisphere deficits do not claim the same degree of localisation and disturbances are attributed to disruptions of larger networks with greater cortical representation.

With reference to stroke and the development of executive deficit, site of lesion is usually considered to be the main determinant. Infarcts involving the frontal lobe occur with high frequency estimated between 20% and 25% (Bogousslavsky, 1994) although this fact alone does not account for the high frequency of executive deficit in stroke. Studies have indicated that anterior and posterior strokes result in impaired performance on tests of EF as do subcortical strokes involving the fronto-striatal circuits. In relation to this, the mechanism of stroke is also highly significant. Most strokes arise from territorial infarct, specifically within the distribution of the middle cerebral artery (MCA) which spreads over the cerebral hemispheres and gives rise to as many as 20 penetrating branches, which in turn supply the lateral portion of the basal ganglia and thalamus and the posterior limb of the internal capsule (Robinson, 2006). The executive deficits associated with MCA infarcts are usually prominent on the cognitive domain while behavioural dysexecutive syndromes are often moderate or even
absent. When present, global hypoactivity with apathy is the most frequent disorder (Godefroy & Stuss, 2007). Significantly, 8 of the 10 participants in this study experienced MCA infarcts. Their executive profiles were in fact largely consistent with the cognitive dysexecutive syndromes described in the literature with Paul’s disturbance of affect conforming to general features of global hypoactivity accompanied by apathy and aspontaneity (Robert, Clairet, Benoit, et al., 2002).

Table 6.19 – Neurological sites involved with aspects of EF

<table>
<thead>
<tr>
<th>CONSTRUCT</th>
<th>NEUROLOGICAL SITE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Behavioural inhibition</td>
<td>Anterior cingulate cortex</td>
</tr>
<tr>
<td>Interference control</td>
<td>Left prefrontal cortex</td>
</tr>
<tr>
<td>Response inhibition</td>
<td>Frontothalamic circuit</td>
</tr>
<tr>
<td></td>
<td>Prefrontal cortex</td>
</tr>
<tr>
<td></td>
<td>Parietal cortex</td>
</tr>
<tr>
<td>Working memory</td>
<td>Dorsolateral and posterior ventromedial frontal cortex</td>
</tr>
<tr>
<td></td>
<td>Inferior parietal lobule</td>
</tr>
<tr>
<td>Internalization of speech</td>
<td>Anterior cingulate gyrus</td>
</tr>
<tr>
<td></td>
<td>Anterior dorsolateral prefrontal cortex</td>
</tr>
<tr>
<td></td>
<td>Basal ganglia</td>
</tr>
<tr>
<td></td>
<td>Inferior frontal</td>
</tr>
<tr>
<td></td>
<td>Right caudate nucleus</td>
</tr>
<tr>
<td>Reconstitution</td>
<td>Anterior cingulate cortex</td>
</tr>
<tr>
<td></td>
<td>Basal ganglia</td>
</tr>
</tbody>
</table>

John and Jane* both presented with subarachnoid hemorrhage, the fourth most frequent cause of stroke (Robinson, 2006) while John presented with complicated rupture of an anterior communicating artery aneurysm. According to Godefroy and Stuss (2007), the disorder mainly concerns behaviour, attention and strategic working memory. The behavioural executive disorders can include hypoactivity (John), hyperactivity with distractibility and disinhibition (Jane*) as well as perseveration (John), and disturbances of emotion and social behaviour (Jane*). Also according to these authors the cognitive dysexecutive syndrome is usually mild except for attention and response suppression deficits, although that contention was not necessarily borne out in this research. When looking at Barkley’s model, the constructs of EF are hierarchically arranged. Behavioural inhibition is placed above the other EF depicting the reliance of the other functions on behavioural inhibition, essentially attention-related processes,
for effective recruitment and deployment. In addition the link between inhibition and working memory functions is essentially inextricable. Therefore, the possibility that the EF deficits observed for John and Jane* on testing could be interpreted as being secondary to attention and behavioural executive deficits is worth considering. One way to test this hypothesis would be to find a pharmacological agent more effective in treating underlying attention difficulties, and observe whether other EFs improved as a result. Table 6.20 below presents details of the ten participants in terms of mechanism of stroke, site of lesion, and presence or absence of cognitive / behavioural dysexecutive syndromes.

Table 6.20 – Sites of lesion for the ten participants

<table>
<thead>
<tr>
<th>PARTICIPANT</th>
<th>MECHANISM OF STROKE</th>
<th>SITE OF LESION</th>
<th>COGNITIVE DYSEXECUTIVE SYNDROME</th>
<th>BEHAVIOURAL DYSEXECUTIVE SYNDROME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cecil</td>
<td>MCA infarct</td>
<td>Parietal</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td>Grace</td>
<td>MCA infarct</td>
<td>Fronto-parietal</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td>Jeannette</td>
<td>MCA infarct</td>
<td>Fronto-parietal</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td>John</td>
<td>Subarachnoid hemorrhage</td>
<td>Fronto-parietal</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Margaret</td>
<td>MCA infarct</td>
<td>Parietal</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td>Mel</td>
<td>MCA infarct</td>
<td>Fronto-parietal</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td>Paul*</td>
<td>MCA infarct</td>
<td>Fronto-parietal</td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Tumi</td>
<td>MCA infarct</td>
<td>Tempero-parietal</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td>Felicity</td>
<td>MCA infarct</td>
<td>Tempero-parietal</td>
<td>√</td>
<td>x</td>
</tr>
<tr>
<td>Jane*</td>
<td>Subarachnoid hemorrhage</td>
<td>Fronto-temporal</td>
<td>√</td>
<td>√</td>
</tr>
</tbody>
</table>

Key: (√) present; (x) absent
6.2.4.1

**Summary - Site of lesion**

Cognitive control is proposed to be located in the prefrontal cortex, inferior parietal cortex and anterior cingulate cortex together with subcortical connections and this is evidenced by neuroimaging research. Furthermore, these areas are precisely those that are commonly affected by stroke and other disease processes or injuries that result in a disruption of communication and other skills reliant on cognitive control. Whereas sequelae of damage following stroke are by no means exclusive to cognitive dysexecutive syndromes, these appear to predominate owing to the frequency of occurrence of MCA infarctions which can affect the frontal, parietal and temporal lobes as well as subcortical territory. When behavioural dysexecutive syndromes occur, the most frequent is global hypoactivity with apathy. These associations beg the question then of whether communication deficits arise independent of executive dysfunction, or whether they are secondary to powerful disruptions of the processes of cognitive control that allow for the access, selection, assembling and execution of neurolinguistic processes.

6.2.5 **The impact of bilingualism on executive functions**

Two individuals in this sample, Grace and Tumi, stand out for having essentially intact EF and conversational skills across all areas assessed. These abilities can not be explained relative to type of aphasia or site of lesion. Instead, it is suggested that these preliminary findings may support some emerging literature related to the effects of bilingualism on the brain. It has been argued that individuals who are bilingual retain certain advantages in terms of cognitive control and flexibility (Bialystok, 2007), which would be evident in greater adaptability in using language in context (Penn, 2007). In a recent study, Penn, Frankel, Watermeyer and Russell (submitted) examined this contention by comparing EF and conversational profiles of bilinguals (represented by Grace and Tumi) to monolinguals, using the data from this study.

The participants’ EF performance was ranked using non parametric statistics (Mann-Whitney -Test) (Siegel, 1956) and features of conversation were analysed. Results indicated significant differences for Grace and Tumi when compared against the monolingual participants on all measures (except Raven’s progressive matrices, where performance was uniformly impaired) and manifested in observable ways in conversation. These findings are particularly interesting
as, if they can be further corroborated in more carefully constructed research designed to investigate this phenomenon, they may have significant implications related to recovery from brain injury, choice of treatment options and response to therapy as well as subject selection for research.

6.3 EXPERIMENTAL PHASE OF THE STUDY

6.3.1 Statistical results

In order to determine whether pharmacological therapy using LEV, induced any changes, a repeated measures analysis of variance was conducted. ANOVA compared performance on the EF battery during the active phase as compared to performances at any of the other three phases of the study (baseline, placebo and withdrawal). There was some hope that at least some of the measures would show significant improvements on LEV. However, this was not the case and no significant changes were observed during the active phase for any of the constructs under investigation. Appendix O presents the statistical analysis for each of the tests (Tables O.1 – O.13).

One test showed significance at p<0.05 for the Tower of London (Appendix table O.7a). However, post-hoc analysis (Appendix table O.7b) revealed that the difference arose between the first administration of the test at baseline and the other three phases of the study, reflecting a significant practice effect on this task, despite the fact that parallel versions had been used.

6.3.2 Qualitative assessment of executive functioning results

Despite the fact that there were no statistically significant changes while on the drug, some observations are worth making. Margaret and Cecil both demonstrated normalization of the interference score on the Stroop Colour-Word Interference Test. This improvement was unique to the active phase of the study for these two participants, with no carryover effects into the other phases of the study, and should reflect an improvement in the ability to resist distraction and show improved sustained attention or concentration. In addition Cecil also showed improvement on the WCST (scoring within normal limits), Ravens (where his score was borderline - a notable improvement) and the five point test, where his percentages of perseverative responding reduced to normal limits. The common factor in all these tasks is the reduction of perseveration during performance. Frankel and Penn (2007) proposed that impaired interference control was the basis for recurrent perseveration in their participant AA. In that
study, improved performance while taking Ritalin also resulted in improved interference control with less perseveration during working memory and reconstitution tasks and a moderate improvement in the ability to contribute to topic shift. It is therefore proposed that Cecil’s improved performance during the active phase of this study could be attributed to improved resistance to interference, resulting in more efficient interactions between executive control and performance on other cognitive tasks. The following section discusses the manifestation of these improvements in conversation.

6.3.3 Conversational results

The conversational transcripts were examined for evidence that reflected the improvements noted above for Cecil and Margaret. In addition, the transcripts for the other eight participants were analyzed in detail to determine changes in functioning. One other participant’s conversations (Jeannette) showed evidence of improved functioning although this was not reflected in the EF test scores. Once again, these findings seemed to be confined to the active phase of the study, without any lasting effects into the following phases of the study.

During the baseline phases, Cecil’s conversation was characterized as showing evidence of poor interference control (distractibility, difficulty staying on topic); poor response inhibition (talkativeness, word level perseveration), working memory difficulties (problems integrating new information) and poor planning (disorganized output). Margaret also demonstrated poor interference control (distractibility, tangential, irrelevant comments), poor inhibition (talkativeness), poor working memory (incoherent output) and impaired planning (disorganized output). Jeannette produced word level perseverations as well as poor planning in terms of output that was lacking in organization and coherence and impaired reconstitution (difficulty with initiating or adapting communication strategies to facilitate repair). An analysis of Cecil’s, Jeannette’s and Margaret’s transcripts comparing baseline, placebo and withdrawal phases to the ones generated during active phase revealed interesting differences. Each transcript was examined for evidence of the EF deficits described above. Each time an EF deficit was noted per turn, it was counted. The occurrences for each feature (under each construct) in each phase of the study was then noted and compared against the number obtained during the active phase. Table 6.21 on the following page reflects the findings.
Table 6.21 – Number of observations of impaired EF in conversation

<table>
<thead>
<tr>
<th>Feature</th>
<th>CECIL</th>
<th>JEANETTE</th>
<th>MARGARET</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BASELINE</td>
<td>PLACEBO</td>
<td>WITHDRAWAL</td>
</tr>
<tr>
<td>Interference control</td>
<td>5</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Response inhibition</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Working memory</td>
<td>8</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>Planning</td>
<td>3</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Reconstitution</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Key: (-) Deficits in this feature not noted
The results are worth noting. For each of the three participants, there were some qualitative changes when comparing impaired features during off-drug phases and the active phase. Cecil did not display any of the features of distractibility, or excessive talkativeness during the active phase. In addition, although there were two instances where trouble integrating new information were evident, Cecil appeared to compensate better by repeating salient information instead of forgetting it, as in the other phases.

Example i below is an extract from Cecil’s (CS) conversation during the active phase, when he talks to his wife (RS) about their plans for when their daughter visits from Australia. His repetition of salient information is evident in lines 101 and 103, where he has requested information from his wife, and then repeats it. In other samples, Cecil has demonstrated a tendency to ask for or be given information, which he acknowledges but then fails to recall and subsequently asks for it again. There is no evidence of extended turn taking in this sample, and Cecil does not stray from the topic at hand, but rather proceeds in a series of logical shifts, talking about the fact that a friend of his has offered his house on the Vaal dam to them for when their daughter arrives. When Cecil’s wife mistakenly thinks that he has been talking about family that are currently in the country and makes a comment in that regard (line 122), he immediately recognizes her error and repairs the miscommunication (123).

Example i

<table>
<thead>
<tr>
<th>Line</th>
<th>CS</th>
<th>RS</th>
<th>CS</th>
<th>RS</th>
</tr>
</thead>
<tbody>
<tr>
<td>099</td>
<td>So you say uh K*** will be in in where March?</td>
<td>About the 18th of March</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100</td>
<td>Yes eight 18th of March</td>
<td>Eighteenth of March</td>
<td></td>
<td></td>
</tr>
<tr>
<td>101</td>
<td>March</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>102</td>
<td>We must do a few nice special things with her for a change we must plan something else she’s on holiday</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>103</td>
<td>She’s got all the friends</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>104</td>
<td>Ja she’s got friends but we must go somewhere nice like go to one of the game parks just for a night or two drive around in a four wheel drive you don’t have to drive I’ll drive or she will</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>105</td>
<td>I spoke I spoke to N*** N***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>106</td>
<td>N***</td>
<td>N*** N*** F***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>107</td>
<td>Mm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>108</td>
<td>So um</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>109</td>
<td>What did he say</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

12 Their daughter
13 Their friend
113 CS He said, ‘if it’s a like a lovely day with K*** will be, you must just phone. Go to the Vaal.’ He’s got all that. How many I told him we got >how many people we’ve got a few people< (1) He’s got twenty rooms at their house.

114 RS Twenty
115 CS Twenty
116 RS They not short of space
117 CS Well we’ve been there before
118 RS Yes
119 CS But I didn’t I didn’t know didn’t
120 RS You didn’t realise that there were so many
121 CS Ja that was it was like all this
122 RS I don’t think they’ll be here long enough to share a weekend cos they going to Cape Town this weekend but whatever
123 CS I’m not talking about THEM
124 RS Oh you mean when K*** comes
125 CS K***
126 RS Did we take them her there last time did she come with to the river
127 CS No no

Jeannette showed a marked reduction in the occurrence of her perseverated utterance (less than half). She was further able to express herself in a more organized and coherent manner, showing better planning skills in her output. Finally, she was better able to initiate strategies to resolve communicative breakdown, whereas during off-drug phases, she was almost completely reliant on her interlocutor to do this for her. In the active phase she used circumlocution and writing to convey her message with success.

In example ii Jeannette (JD) and the researcher (R) are talking about Jeannette’s food preferences. In line 150, Jeanette can’t say the word ‘muffins’. She initiates the use of writing as a strategy which then triggers her recall of the word. She stops writing and instead says the word she is looking for (152). She comments on the fact that she cannot recall how to write the second part of the word, using gesture to supplement her message.

*Example ii*

145 R What do you enjoy?
146 JD Salad
147 R And?
148 JD salad again (laughs)
149 R and salad and salad (laughs)
150 JD and uh (1) and uh (1) um ok write it
151 (writes ‘muff’) 
152 JD moffins
153 R oh muffins
JD: I love muffins. What. You see I finished but I can remember the name ‘muffins’ but I woody wood a name I (.) that one I know (pointing to the part of the word she wrote) but that one I don’t (pointing to the space where the second part of the word should be)

R: So you could remember how to write the first part but not the second part

JD: Mm. mm.

This excerpt is contrasted with the following examples from the other phases of the study. In example iii from the baseline phase of the study, Jeannette (JD) is talking to the researcher (R) about her stroke. The frequent use of the idiosyncratic perseveration ‘woody wood’ is noticeable throughout the exchange. In addition, her reliance on her diary to provide details with which to inform the conversation is evident from lines 118 to 125, when she finally locates the information she needs. However in the interim she relies on the researcher to guess and provide alternatives for her consideration.

Example iii

JD: ‘cos I (1) walk (. ) and um (1) woody wood a chair and I fall so another fall woody wood hospital (1) and um another (2) so I said woody wood too much

R: mm↑

JD: so I’m getting betterº

(4)

R: so (.) where was the first place you went to? to the Gen? first to Helen Joseph?

JD: I↑ don’t remember (. ) that one ((pointing to the name of the hospital in her diary))

R: to the Joburg Gen?

(4) ((JD shrugs))

JD: and a cage↑ (3) a cage

R: a cage?

JD: ‘cos um woody wood, huh fall.

R: oh so they put up -

JD: and woody wood huh um woody wood teeth I haven’t food and um uh /gasses/

R: your glasses broke

JD: no↑ u::m woody wood huh breaking so I can’t see I can’t see (2) lo::ng time ago (1) shit (2) long time ago

R: Where were you when you fell? (1) Were you at home?

JD: no uum

(5)

((starts paging through her diary))

JD: no um

(4)

R: were you with your kids?

JD: ((shakes head))

R: were you alone?
Finally, Margaret’s data showed quite striking improvements. Like Cecil her active phase conversation revealed less excessive talkativeness, the ability to remain focused on a topic without becoming tangential and making irrelevant comments. She appeared more organized and coherent in her discourse. Three examples are presented reflecting improved conversational output for each of these participants.

Margaret (MS) and the researcher (R) are talking about Margaret’s recent bout of flu. This extract is clearly different from her other interactions. There is a marked absence of irrelevant and tangential comment. She is able to follow a well sequenced, organised set of ideas and is essentially coherent.

*Example iii*

034 MS You know what I think happened with me? I took you know when I went to to L***'s house and I I didn’t have enough of the (.) the visitors (2)
035 R oh your vitamins
036 MS vitamins. There now. I always have that and for two years I never had a problem. >I didn’t have to worry about you know< I didn’t have flu and I was FINE. I was always healthy and all that sort of thing and then of course I didn’t have enough here
037 R ja
038 MS so I couldn’t get hold of my pill*=lls and what have you. So that’s why it had to happen I’m sure
039 R mm
040 MS because I didn’t have enough
041 R ja your immune system=
042 MS =pills
043 R =was a bit depressed
044 MS =yes ja ja that’s what I think happened. But ↓ one of those things that happen.
045 R well hopefully now that you’ve got antibiotics
046 MS mm
047 R I hope you get over it. You must be careful though hey. not too much exercise and stress
048 MS oh I’m not gonna do that until I’m rea::llly finished
049 R well ja

14 Her daughter
I mean ts! you know what I’m saying.
I do. until you over it completely. I know what you mean
Thanks. I didn’t do anything here. I didn’t let me (puts coffee cup down; coughs) (sighs) I didn’t have any games, or anything like that. Nothing.
Ja
because I’ve been, all I want is to sleep. That’s all I wanted. Terrible. hey?
Well sometimes your body just needs to rest >to kind of work whatever it is out of your system<
Then that was like that.

6.3.4 Summary of drug effects
On the whole this study is disappointing given the fact that piracetam studies have demonstrated modest gains in terms of cognition and communication. LEV was initially evaluated in models of cognitive impairment with the primary objective of finding a more effective drug than piracetam (Genton & Van Vleymen, 2000). Although LEV appeared to have some impact on the interference score for both Cecil and Margaret with apparent corresponding changes in conversation and some qualitative improvement for Jeannette, the findings are not particularly convincing and do not appear to provide compelling evidence for further use of LEV in this population.

Although studies exist evaluating the effect of certain drugs on specific cognitive functions, class I randomized controlled trials addressed the efficacy of pharmacological and rehabilitation treatment agents remain limited in the field of executive and attentional dysfunctions, especially in stroke (Forsyth & Jayamoni, 2003). Pharmacological intervention has been used in rare clinical trials for some behavioural disorders similar to those seen in dysexecutive syndrome, although most have been conducted in nonstroke patients, mainly in patients suffering from closed head injury and dementia. In apathy and motivational deficits, common agents are those related to the ascending monoamine pathways, i.e. dopaminergics and amphetamines. In some studies significant behavioural improvement has been reported although benefit to everyday life activities remains to be evaluated (Forsyth & Jayamoni, 2003; McDowell, Whyte & D’Esposito, 1998; Powell, al-Adawi, Morgan & Greenwood, 1996). For impulsivity and disinhibition open trials have reported the efficacy of beta-blockers (Chatham Showalter & Kimmel, 2000; Shankle, Nielson & Cotman, 1995) although the Cochrane review (Fleminger, Greenwood & Oliver, 2003) has highlighted the need for better evaluation of drugs in these disorders. Certainly, it appears that agents that have direct impact on stabilizing attention
functions would be potentially more effective, although participants would have to be carefully
selected and monitored owing to some of the deleterious (albeit rare) side effects on heart
rhythm and blood pressure, which are clearly counter indicated in individuals who have had
strokes.

This concludes the results and discussion, the following chapter presents the general discussion
and concludes the study.
7.1 SUMMARY OF THE INVESTIGATION

Conversational intelligence is a concept, coined with reference to Howard Gardner’s notion of multiple intelligences (2004). In much the same way, our ability to converse depends on many independent, interconnected and sometimes interdependent processes. These variables are as wide ranging as personality, cultural background, self esteem and communication context. This study has focused on some of the neuroanatomical, neurochemical, psycholinguistic and neuropsychological mechanisms that contribute towards conversational intelligence and the way in which these factors and hence our ability to interact is disrupted in the face of damage to the brain through stroke.

7.1.1 The primary investigation

This study comprised two parts. In the first descriptive stage, language, executive functioning and conversational characteristics of a group of ten individuals with strokes were described. Language features were derived from performance on the WAB (Kertesz, 1982), while executive functioning (EF) abilities were interpreted from a specially designed battery measuring the constructs of behavioural inhibition, working memory, regulation of affect, internalization of speech and reconstitution (Barkley, 1997). Conversational data was produced by video-recording the participants with familiar interlocutors and subjecting the data to Conversation Analysis. This data was then analysed to determine associations amongst the different components investigated.

In the second experimental stage, the ten participants (8 of whom were aphasic and 2 of whom had right hemisphere syndrome) took part in a drug trial. They were each administered a dosage of 1500mg of Leviteracetam on a daily basis for one month. The administration of the drug was assigned to phase 1 or phase 2, which was randomly alternated with a placebo condition, to which both the researcher and the participants were blind. These phases followed baseline data collection which provided the details for the descriptive stage of this research and preceded a
withdrawal phase. Executive functioning and conversational data were therefore collected four times, while the language data was only collected at initial baseline testing.

7.1.2 The secondary investigations
In addition to the main aims, a total of five subsidiary studies were derived from this research. The first involved the piloting of the EF battery to determine its usefulness in relating its findings to conversational data. Results of this study are published and summarized in chapter 5. The second study involved a control investigation to determine whether significant differences existed between performances on selected EF tests when using the dominant as opposed to non-dominant hand. The third study was a methodological investigation, which served two purposes - namely to provide a form of authentication of the interpretations reached by the researcher with respect to the conversational data, and also to evaluate the usefulness of CA as opposed to other forms of analysis. The fourth study investigated the effectiveness of the informed consent process with this group of participants. Results of this study were also summarized and presented chapter 5. Finally the last study compared bilingual to monolingual performance on EF tests and conversational skill, with these participants and these results were briefly presented in the previous chapter.

7.2 GENERAL FINDINGS
7.2.1 Descriptive phase
Three participants presented with anomic aphasia, two with Broca’s aphasia, three with conduction aphasia and two with right hemisphere deficits. Most of the participants presented with significant EF deficits across all areas assessed, with few remaining pockets of intact skill. Two participants (Grace and Tumi) were exceptional in that they demonstrated essentially intact profiles excluding performance on serial digit recall (forward and backward) and Raven’s progressive matrices.

In general there was little consistency between EF and language features, although some relationships were more robust. Without exception all the individuals with aphasia and Jane* who presented with reduced grammatical complexity failed the working memory task represented by the digits backwards test, while Felicity, was the only person who showed intact performance as well as normal grammatical complexity in her verbal output. This implies a strong involvement of the phonological aspects of working memory in facets of expressive
language. Also, there appeared to be a relationship between affective disorders and expressive language. The two individuals with the most reduced expressive language were also characterised as being depressed and apathetic, while Jane* who was characterised as being manic and inappropriate presented with corresponding elements in her language output. However, manifestation of language and EF deficits were individual and although some features and characteristics were similar across language profiles, for the most part, there was not enough consistency to be able to draw convincing prototypes.

On the other hand, the conversational data seemed to provide a good reflection of the EF features. To a large extent, these also corresponded broadly to cognitive and behavioural dysexecutive syndromes described by site of lesion in the literature. Also, characteristics of deficient behavioural inhibition tended to have pervasive effects on many other executive functions dependent on attention factors for adequate control. When participants demonstrated significant response inhibition deficits, accompanied by perseveration, they also tended to show performance decrements as a result of shifting difficulties and perseveration on tasks measuring working memory, problem solving / planning and reconstitution.

However, despite similarities in EF profiles conversational symptoms manifested in different ways. While some individuals with poor interference control were unable to stay on topic, others had a tendency to interrupt, or showed various combinations of disruptive features in their conversations. Significantly, the EF deficits observed in the individuals with aphasia and RHD are not dissimilar to those found in other neurogenic communication disorders. While aphasia is no doubt characterised by specific and well established disturbances of language that impact on comprehension and expression, the notion of how to have a conversation and how to be an effective conversational partner, appears to depend more on executive functions than on language. It would be interesting to submit some of the conversation samples, particularly those of the fluent speakers, together with samples from individuals with TBI, or dementia, and observe the accuracy with which conversational styles are categorised into their respective etiologies. Based on the results from this study, it would not be surprising to see some stroke patients classified as more diffuse etiologies on the basis of their respective EF deficits. Instead, as we refine this type of research it may be possible to identify EF deficits on the basis of various conversational features, as demonstrated in Figure 7.1 using an adaptation of Barkley’s model on the following page.
The performance of Grace and Tumi, who presented with intact EF and accompanying competent conversational skills, could not be explained by site of lesion or type of aphasia. Instead, a hypothesis about the protective role of bilingualism on the brain was proposed. The data suggests that the experience of speaking more than one language provides the bilingual brain with greater flexibility, adaptability and ability to shift sets. These characteristics manifest in greater control over communicative acts and have implications for future research and intervention.

7.2.2 Experimental phase

The statistical data showed only one change, reflecting a notable practice effect on the Tower of London between the initial baseline performance and subsequent administrations. However, there were three qualitative changes worth noting. First Cecil and Margaret both demonstrated a normalization of the interference score on the Stroop Colour-Word Interference Test. In addition, for Cecil this was accompanied by improvements across a number of tests including the WCST, the Five Point Test and although his score was not quite in the average range for Raven’s it improved significantly to being borderline. These results were not replicated during any other phase of the study. These findings, though not statistically significant were reflected in the conversational data. Finally, Jeannette, also demonstrated observable improvements in her conversational skills while in the drug phase, but these were not reflected in her EF results.

As discussed in the previous chapter, it is difficult to specify an exact reason for such poor response to the trial drug. Previous trial indications suggested that it had significant potential to improve some of the executive deficits present in the individuals in the sample. Nonetheless, the literature in this area is characterised by ambivalent results. As suggested in chapter 3, this could reflect the complexity of the symptoms under investigation and the individual manner in which factors combine and respond to chemical manipulations. Of all the pharmacological agents under review, the catecholamines, and especially the amphetamines seem to hold promise in terms of stabilising inhibitory and attention related abilities, which theoretically could have a follow on effect on other aspects of EF. More studies, recruiting medically stable individuals, with poor EF, carefully monitored at low to moderate dosages of catecholamine substances, are needed.
Figure 7.1 – Disordered features of conversation relative to EF deficiency

**Regulation of affect**
- Hyporesponsiveness
- Hyperresponsiveness

**Internalization of speech**
- Discourse lacks organisation and coherence
- Fractured
- Excessive revisions and false starts
- Reduced self reflection and monitoring
- Excessive detail

**Working memory**
- Difficulty maintaining thread of conversation
- Repetition of ideas and statements
- Impaired recall of shared information
- Disorganized output
- Difficulty integrating new information
- Providing limited information on request

**Behavioural Inhibition**
- Response inhibition:
  - Talkativeness
  - Perseveration
- Interference control:
  - Difficulty following an exchange
  - Difficulty staying on topic
  - Tangentiality, irrelevant comments
  - Misinterpretation of speech
  - Interruptions

**Conversational control**
- Poor ability to repair conversational breakdown
- Limited ability to mobilise, generate alternate communication strategies
7.2.3 Control study
Results of the control study revealed non-significant differences between performances using dominant vs. non-dominant hand. A tendency towards improved performance on the second administration of the tests proved more noteworthy, regardless of which hand was used. This suggests greater learning effects for the non-impaired sample than for the neurologically involved group.

7.2.4 Confirmability of the data
Of the three raters who examined the data, the rater with access to both video material and transcripts displayed the highest degree of concordance with the researcher both in terms of similarity of findings as well as in terms of the extent of the detail noted. The clinician with access to the video material showed the next highest agreement, although her interpretations were often less detailed and included less insight than the researcher and rater who had both videos and transcripts. These findings suggested the significant role of clinical training and experience in the recognition of clinical pathology, but also the usefulness of Conversation Analysis in bringing depth and further insight to the assessment of these features.

7.3 METHODOLOGICAL ISSUES
Several issues related to the constructs under investigation, the notion of ecological validity and the choice of tests as well as aspects of the confirmability study present topics for closer inspection.

7.3.1 Language testing
It was evident from the results that the WAB was not a good predictor of either executive dysfunction or conversational competence. This finding is not uniquely relevant to the WAB and has been echoed by other researchers with reference to other formal language tests (e.g. Hardin & Ramsberger, 2004). On the WAB, the spontaneous speech score is derived from responses to quite structured questions as well as a picture description. As Oelschlaeger and Damico (2003) have pointed out, the fact that formal tests do not actually assess language in context makes them incomplete tools. Conversation has been shown to be highly sensitive to contextual, neurological and neuropsychological influences. Its inclusion in communication assessment batteries would therefore offer deeper insights into the abilities of our clients to integrate a variety of skills and adapt to dynamic, multi-faceted interactions.
7.3.2 Ecological validity and EF testing

The second issue relates to the tests chosen for the EF battery. In relation to the ecological validity of EF tests Mesulam (cited in Cripe, 1996) makes the following statement. ‘The behavioural changes associated with frontal cortex damage… tend to be exceedingly complex, variable, difficult to define in technical terms and almost impossible to quantitate by available tests.’ (p. 184). Goldberg and Podell (2000) present the differentiation of veridical problem solving (as tapped by tests) as opposed to adaptive problem solving (which more closely resembles real world activity). The definition of ecological validity is the functional and predictive relationship between a patient’s performance on a set of neuropsychological tests and their behaviour in a variety of real world settings. Also inherent to the concept of ecological validity is the assumption that demand characteristics within these various settings are idiosyncratic and fluctuate as a result of their specific nature, purpose and goals (Sbordone, 1996).

Sbordone (1996) further offers some thoughts on the critical issues that need to be considered when attempting to draw conclusions about real world performance from EF testing. The first is that physical testing conditions may significantly weaken any generalization about patients’ behaviour in real world settings. In this study, the researcher was careful to administer tests in quiet, virtually optimal environments, with good lighting, freedom of noise and distractions and the opportunities to take breaks. Sbordone (1996) makes the point that our lives are not carried out in quiet rooms but rather often in situations that are less than conducive to optimal performance. The type and scope of tests under such conditions may be inappropriate, inadequate or generate inaccurate or unrealistic expectations of behaviour in the real world. In this regard, it is possible that some of the test results are in fact inflated and that the testing situation allowed for better performance than would be achieved in more demanding circumstances. This is worth bearing in mind particularly when investigating constructs like ‘freedom from distractibility’. An example to illustrate this is the fact that Mel, in her recorded conversations with her boyfriend, typically had a range of intact behaviours. She was able to initiate topics, and contribute to shift. However, she maintains that when she is in a group, she is unable to follow the thread of the conversation and the input from multiple parties becomes overwhelming and difficult to follow.
Sbordone (1996) makes another point related to the interactions between examiners and patients immediately prior to and during testing. He says that these interactions can mask the extent of impairments, particularly when therapists take on the role of tester, owing to the fact that our role is traditionally facilitative and mediating. Perhaps the involvement of an objective third party, who will not have continued interactions with the participants, is worth considering when such testing is required.

In addition, just like the structure of current language batteries appears to mask functional communication deficits, so too the structure of many EF tests also serves to hide behavioural deficits. In this regard he stresses the need to talk to other people in the patient’s life and gather real world data in order to further inform a clinical profile regarding areas of strength and difficulty.

7.3.2 Task sensitivity and specificity

It is difficult to critique the tests chosen without acknowledging the challenges and complexities involved in choosing relevant tests for individuals who have suffered a stroke and more particularly who present with aphasia. While there are no doubt dozens of tests available that are better suited to measuring certain constructs than the ones chosen for this battery, their usefulness with individuals with aphasia is undermined because of their reliance on verbal responses or the need for complex verbal instructions. One example is the Multiple Errands Task which has been shown to have good ecological validity when measuring planning and strategy use (Shallice & Burgess, 1991). However, the test requires reading, motor mobility (being able to move in and out of shops on a street), the ability to use money and communicate effectively with shopkeepers. It therefore remains a limitation of this research, that some of the tests chosen were selected on the basis that they were the best available that could meet the needs of the people to whom they were being administered. This highlights a potentially fruitful area for future research, involving the design and validation of relevant and meaningful EF test batteries for individuals with aphasia.

Another significant challenge lies in the difficulty of disassociating between several cognitive functions, particularly with respect to behavioural inhibition, attention and working memory. One of the central principles of the Barkley model is that the appearance of poor sustained attention, in individuals with ADHD, actually represents a reduction in the control of behaviour
by internally represented information, contributed to by the four executive functions. While Barkley (1997) makes a strong case for the differentiation between sustained attention and inhibition, the broader role of attention and its implications in his model and for this study, need to be addressed more directly.

Attentional processes incorporate a wide range of functions, including arousal, vigilance, selective attention and shifting attention (Adamovich & Henderson, 1992; Hartley, 1990). In addition, slowed information processing has been shown to be an underlying cause of impaired attention (Ponsford & Kinsella, 1992; Van Zomeren & Brouwer, 1994). These processes and the functions they serve, have much in common with those implied by behavioural inhibition. Specifically, the ability to shift the focus of attention appears commensurate with Barkley’s implication of inhibition in terms of the ability to interrupt ongoing responses.

In addition, the ability to inhibit inappropriate shifts of attention closely resembles Barkley’s notion of interference control. It is especially significant that in the last few years, the field of cognitive psychology has produced three major theories of attention, all of which cite as their fundamental precept the fact that the primary role of attention is to protect central mechanisms from interference caused by irrelevant input (Milliken & Tipper, 1998). This perspective is expressed in Broadbent’s Filter Theory (1958), Wally and Weiden’s work on lateral inhibition (1973) and the work related to single cell recordings by Moran and Desimone (1985).

Furthermore, the functional outcomes associated with disruption of these processes are essentially the same. Whether viewed from the perspective of Barkley’s model or from cognitive and neuropsychological perspectives, disruptions to the processes outlined, will result in impulsivity (the inability to inhibit a prepotent response), perseveration (the inability to shift focus or interrupt an ongoing behaviour) and distractibility (inability to resist interference) (Adamovich & Henderson, 1992, Pashler, 1998). Therefore, it appears that behavioural inhibition as described by Barkley, could be interpreted as a construct related to attention.

Another intricate relationship presented in Barkley’s model, is that between behavioural inhibition (attention) and working memory. As is evident from the material presented above, Barkley (1997) separates these functions. Evidence in support of this dissociation comes from neuropsychological studies by D’esposito, Detre, Alsop, Shin, Atlas, and Grossman (1995),
Knight, Grabowecky and Scabini, (1995), Vendrell, Junque, Pujol, et al. (1995) and Williams and Goldman-Rakic (1995) as well as neuroimaging research, which has found separate anatomical supports for these two constructs. Iverson and Dunnett (1990) established that inhibitory functions are ascribed to the orbital-frontal regions of the prefrontal cortex and its reciprocal connections with the ventromedial region of the striatum. Working memory on the other hand, appears to be subserved by the dorsolateral region of the prefrontal cortex and its reciprocal connections to the more central region of the striatum.

However, within the literature about working memory, much controversy exists. Watt (1996) highlighted the inseparability of the theoretical constructs of attention and working memory by drawing attention to the fact that in several clinical batteries the same test is used to assess both constructs (Kinsella, 1998). As has been demonstrated in this study, interpretation of test performance is complicated. Some researchers view memory span tasks (such as Digits forward) as reflections of verbal or phonological working memory (Baddeley & Wilson, 1993; Gathercole & Baddeley, 1993). Others consider immediate memory span to be reflective of attention processes (Nell; 1994, Van Zomeren & Brouwer, 1994).

Fuster (1995) partially attributes the existing contradictions to the fact that Short Term Memory (STM) as supported by the prefrontal cortex, is highly dependent on attention, which sub-serves the temporary retention of information. Therefore, the measurement of STM, in individuals with prefrontal deficits amounts to a measurement of the fundamental difficulty these patients have with suppressing attention to irrelevant stimuli and maintaining attention to relevant stimuli. This deficit is epitomized by failures of STM at delayed matching and responding because of the inability to suppress interference from competing and untimely tendencies (Fuster, 1989, 1995).

Other difficulties that may arise on immediate span tasks in individuals with prefrontal memory difficulties are the inability to use context spontaneously to facilitate recall, impaired use of context for storage and retrieval, inability to organize material to be learned and impulsivity, all of which are related in some way to attention (Lezak, 1995).

Furthermore, Barkley (1997) notes that according to Fuster (1989), failure to adjust motor performance given feedback concerning its ineffectiveness (ascribed to behavioural inhibition)
may actually reflect an interaction between behavioural inhibition (attention) and the retrospective-prospective functions of working memory. The individual fails to hold in mind, information on the success of his or her responding on the immediately preceding trials (retrospection), which then feeds forward to influence or even stop immediately future responses (prospection). This suggests a cessation, shifting and re-engagement of inhibition that relies on an inextricable link between attention and working memory.

Two significant issues arise from the discussion above. Firstly, the discussion highlighted the fact that behavioural inhibition reflects essentially the same processes as ascribed to attention and therefore could be construed as a component of attention. The second implies that attention and working memory are inextricably linked and that deficits in the former, will anticipate deficits in prefrontal memory function. Interpretations regarding the dissociations of functions in the presence of a deficit in behavioural inhibition must therefore be made with extreme caution. Despite controversy in the related literature, there seems little doubt that the relationship between the constructs exists.

Bearing in mind the above discussion, the tests used in this research are discussed below, in terms of sensitivity and specificity and their ultimate usefulness in research of this nature.

Two tests were used to assess different aspects of interference control, Digits forward was used to assess simple span of attention while the Stroop Colour Word Test was used to assess the ability to resist distraction in a more sustained and effortful task. This test is the format most commonly used for measuring span of attention (Lezak, 2004) and is generally not considered to be a test of verbal skill. Despite this, the point digit span was specifically used for individuals with significantly compromised verbal abilities in order not to confound the data. The Stroop Colour Word Test is a highly sensitive test of interference control, showing the ability to dissociate between findings that were specifically related to poor ability to withstand interference as opposed to other factors. Its biggest detraction is that it requires speeded verbal responding which even a ratio score may not be able to control for in this population. A similar test with fewer verbal demands would certainly be more ideal. Still, there was a high degree of correspondence between findings on these tests and conversational features, suggesting that in fact these tests if interpreted carefully, can highlight some potential attention difficulties underlying conversational features.
Response inhibition or the ability to shift attention was measured with the Trail Making task. This test was not a particularly sensitive test when interpreted in terms of scores produced. Several participants were able to perform well on the task, but showed distinct difficulty with the ability to shift attention on other more complex tasks, such as the Wisconsin Card Sorting Test, Tower of London and fluency tasks. Difficulties with response inhibition were also often found in conversation (e.g. John, Paul*, Felicity) despite good performance on Trail Making, suggesting that this task may not be taxing enough to highlight deficits in response inhibition. Still it may be a useful tool if interpreted qualitatively, in terms of number of errors, instances of perseveration and adaptation to Task B from Task A.

Working memory was assessed in multiple ways on this battery, using the Self Ordered Pointing Test (SOPT), Digits Backwards (probably the purest of the tests chosen), as well as the Complex Figures Test and the Wisconsin Card Sorting Test. Of all the tests chosen for this construct, the participants’ performance on the Complex Figures Test was disproportionately much stronger than on the other tests of working memory. Although it has been described as a test of working memory because it relies on the ability to hold in mind, organize and reproduce complex information, it is also a test of planning, visuo-spatial skill and learning. It is possible that the element of the test that taps long term learning capacity accounted for the discrepantly high performance across all participants for this test relative to other tests for working memory. This differentiation is articulated by Damasio and Anderson (2004) who state that defects in working memory, attention and executive control may result in major dissociations between well-preserved memory capacity and severely impaired utilisation of these abilities in real-life situations. As Melinder, Barch, Heydebrand and Csernansky (2005) point out the assumption that more difficult tasks have greater discriminating power is not always accurate. They caution that the critical factor for task selection and matching is discriminating power, not task difficulty.

The SOPT as well as Digits backwards, were perhaps the purest or most specific of all the tests chosen in the entire battery. It was also interesting that poor performance on the SOPT a non-verbal test of working memory was consistently associated with organizational aspects of conversation mostly related to topic management. On the other hand, Digits backwards, was more consistently associated with reduced sentence length and complexity and word finding
difficulties. Further research linking these constructs under stricter and more specific testing conditions is certainly warranted to tease out these relationships.

It is very difficult to interpret results of the WCST in terms of working memory constructs only. The information gleaned from this task is particularly useful when interpreted in light of its link with the inhibitory functions discussed earlier. It is the ability to use retrospective memory to inform current decision making or lack thereof that comes to the fore during administration of this test. Therefore as a test of the breakdown between inhibition and working memory functions, manifested in perseveration, this task is extremely useful. However, comprehension of the test instructions can be extremely challenging for people with aphasia. In addition, conceptualization difficulties may impair performance regardless of executive dysfunction (Keil & Kaszniak, 2002). As an aside, it was noted that this test is extremely unpleasant to take. Most of the participants experienced extreme frustration and expressed overtly or covertly, that the aim of the test was to deceive and otherwise undermine them. If for no other reason, inclusion of this test in other batteries needs to be carefully considered.

The Neuropsychology Affect and Behaviour Profile was used to assess affect and is a useful tool in that it can discriminate among several types of affective disorders. It was specifically for this reason that it was chosen above others such as the Neurobehavioral Rating Scale by Levin, High, Goethe, Sisson, Overall, Rhoades, Eisenberg, Kalisky and Gary (1987), as it assesses constructs related to personality changes following brain injury and has a record of research investigating its validity and reliability. The placement of the category Pragnosia within the battery is however questionable. As a construct, Pragnosia is designed to measure deficits in the appropriateness of social behaviour, attributing such behaviour to emotional and personality changes. The behaviours listed as comprising this construct are familiar to speech language pathologists in relation to individuals with problems of a pragmatic nature (Grundy, 1995). It is however difficult to associate emotional changes secondary to brain injury to all manifestations of pragmatic deficits. In some cases, pragmatic difficulties are directly related to and therefore secondary to communication difficulties, rather than due to any emotional disturbance necessarily. In addition, slowed information processing, may also affect communicative skills such as word finding and topic initiation and therefore the tool’s ability to differentiate between cognitive-communicative deficits and apathy or indifference is poor.
Planning or the ability to use internalized language was assessed with the Tower of London and Raven’s Progressive Matrices. The Tower of London is a complex task whose purpose has been widely debated in the literature owing to the fact that it loads on separate factors from other EF tests (Keil & Kazsniak, 2002). This suggests that it has unique demands or cognitive processes in comparison to typical clinical executive tests including both sequential and spatial elements. However, because patients with left anterior damage have been shown to perform normally on Block Design but were impaired on the Tower of London Shallice (1982) has urged that this test should in fact load on planning factors specifically. The difficulty with this task, and the ones that target reconstitution as well, is that the effects of impaired inhibition and working memory appear to be pervasive. It is therefore difficult to isolate the effects of poor planning or indeed identify such deficits with complete confidence, from the cascade caused by poor inhibition and interference control. However, more so than with performance on the Raven’s Progressive Matrices, individuals with significantly poor performance on Tower of London, also appeared to show poor planning of macrostructures in their conversations and this is a potentially valuable finding.

Unlike the findings with Tower of London, performance on the Raven’s Progressive Matrices was so uniformly poor across the sample that it yielded little discriminatory power. This test appears to tap too many skills including visuoperceptual, primary visual, abstract reasoning and concept formation as well as relying heavily on intact aspects of attention. It appeared to be particularly vulnerable to deficits in inhibition and perseverative responding was often the source of incorrect responding. The most interesting finding in relation to this test was the fact that Cecil achieved an almost borderline score (an important, although not statistically significant improvement relative to other phases of the study) which also coincided with reduced distractibility and perseverance as manifested on several other tests during his active phase.

Finally, the tests of reconstitution, were the Five Point Test and Design Fluency. The Design Fluency task has been shown to be highly sensitive to frontal damage but has low specificity owing to the fact that many types of brain damage lead to impairments on this measure (Ellis & Young, 1996). Both of these fluency tests may be confounded by the roles of visuoperceptual and visuoconstructive processes in successful completion. However, this research appears to imply that poor performance on these tasks, like with planning, is secondary to behavioural
inhibition. Perhaps it is because of all the EF, attention is one of the most vulnerable to disruption and the ability to initiate creative, fluent and novel responses is highly dependent on the efficient distribution of attention resources. Table 7.1 below presents a list of the tests that were felt to be particularly useful relative to the conversational features with which they were associated.

Table 7.1 – Useful EF tests and their associations with conversational features

<table>
<thead>
<tr>
<th>TEST</th>
<th>CORRESPONDING CONVERSATIONAL FEATURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroop Colour-Word Interference Test</td>
<td>Difficulty staying on topic&lt;br&gt;Difficulty following an exchange&lt;br&gt;Tendency to make tangential or irrelevant comments&lt;br&gt;Misinterpretations&lt;br&gt;Interruptions</td>
</tr>
<tr>
<td>(Trail Making) when interpreted qualitatively</td>
<td>Talkativeness / Verbosity&lt;br&gt;Perseveration</td>
</tr>
<tr>
<td>Self Ordered Pointing Test</td>
<td>Inability to maintain the thread of a topic&lt;br&gt;Repetition of ideas or statements&lt;br&gt;Impaired recall of shared information&lt;br&gt;Disorganized discourse&lt;br&gt;Difficulty integrating new information&lt;br&gt;Providing limited or restricted information when asked</td>
</tr>
<tr>
<td>Wisconsin Card Sorting Test</td>
<td>Difficulty monitoring flow of inappropriate communication&lt;br&gt;Difficulty shifting from ineffective communicative strategies to more adaptive ones</td>
</tr>
<tr>
<td>Digits backwards</td>
<td>Reduced sentence length and complexity, anomia</td>
</tr>
<tr>
<td>Tower of London</td>
<td>Planning the macrostructure and sequence of discourse</td>
</tr>
<tr>
<td>Design Fluency</td>
<td>Reduced number of topic initiations&lt;br&gt;Reduced ability to contribute to shift</td>
</tr>
<tr>
<td>Five Point Test</td>
<td>Reduced ability to generate alternate communication strategies to facilitate repair</td>
</tr>
</tbody>
</table>

7.3.3 Confirming authenticity of the conversational findings

Several issues need to be considered here. First, is the fact that while most of the participants had one conversational partner for all four phases of the study, usually a spouse, some like Jane* had two. It is usually common practice when using CA as a methodology in a study with multiple phases that factors such as time, setting and nature of interaction as well as interlocutor
remain constant in order to allow the researcher to effectively measure change across time (Ten Have, 1999). However when conducting this research, this was not possible in all cases. For the most part, the time, nature and setting of the interaction were stable across all four phases relevant to each participants’ specific circumstances, even when there was a change in conversational partner. The scripts were carefully analysed for changes in the conversational competence of these participants, to determine whether changes in interlocutor had notably deleterious or enhancing effects on the presence or absence of executive features as well as management of conversational nuances. Such changes were not noted. It was felt that the informal nature of the interactions, and the familiarity with both the researcher and the other conversational partners, mitigated any discrepancies that may have arisen due to varying conversational partners.

However, this raises a different issue about some of the observations and interpretations of the conversational data, where the researcher was the interlocutor. The fact that the researcher participated as a conversational partner, may characterise these interactions as a form of ‘institutional interaction’ as described by Drew and Heritage (1992), from the participants’ point of view. Such interactions are often characterised by the the ‘lay’ speaker producing significantly longer turns than the ‘professional/institutional representative’. Another feature of institutional interactions is that it is typically the ‘professional’ who changes topic. Therefore, in the case of Felicity for example, long turn length and lack of topic initiation, may not strictly be an outcome of EF impairment, but rather a reflection of certain social conventions.

The results of the study assessing confirmability of the conversational findings reflected the least concordance with the linguist who analysed the transcripts only. This result was interpreted to indicate the important role of clinical experience and knowledge in recognising clinical symptoms. However, more than that there is a theoretical basis for needing data that provides access to all aspects of an interaction as discussed with reference to Langewitz’s work on neo-phenomenology. The ability to analyse situations, in this respect, have a sense of conversational competence or intelligence, allows researchers and clinicians to compare and contrast impressions to arrive at a more complete and accurate description of a clinical presentation. By submitting even qualitative material for verification, we provide greater authenticity to our assessments and clinical judgements.
‘Pure logical thinking cannot yield us any knowledge of the empirical world; all knowledge of reality starts from experience and ends in it …’ (Albert Einstein in Cripe, 1996, p. 200).

### 7.4 IMPLICATIONS OF THE STUDY

#### 7.4.1 Clinical implications

Although there is a growing awareness of the possibility of EF deficits in our stroke patients, thorough evaluation and consideration of such deficits in treatment planning have not yet infiltrated routine practice. Results of this research suggest that we need to be particularly mindful of possible executive dysfunction not only because of the impact on functional everyday communication acts but because their potential to pervade and influence every aspect of the intervention process. More specifically, EF may ultimately prove to be a powerful predictor of whether or not therapy benefits will be generalised into real world settings.

In this respect there is a theoretical debate about how to plan treatment. Sohlberg and Mateer (1989) argue that abstract and complex skills like reasoning, problem solving and other EF rely so heavily on core processes such as attention, visual processing, memory and language that these impairments should be treated before even assessing higher level skills areas. Others, like Ylvisaker and Szekeres (1994) say that this hierarchical conception is inexact and misguided. They maintain that improvements in apparently higher level operations like organization are essential for effecting changes in lower level processes like attention. They also reason that some problem solving tasks are quite straightforward whereas some attention tasks are cognitively more demanding (Tompkins, 1995). One rehabilitation technique that has met with some success is goal management therapy, specifically designed to address goal directedness. The major objective of this therapy is to train individuals to stop and think before acting. Success may be assisted when the individual can use other abilities such as self talk to monitor and direct behaviour (Godefroy & Stuss, 2007).

Results of the current study imply that in fact deficits in behavioural inhibition and working memory could have profound effects on other skills like planning and reconstitution. The problem with treating strategic planning and organization first is that in the face of inhibition and working memory deficits, patients may not remember to use their techniques when required to do so. As Barkley points out “The problem then… is not one of knowing what to do but one
of doing what you know when it would be most adaptive to do so.” (Barkley, 1997, p. 78). These difficulties may provide the explanation for why many of our clients are successful in using alternate strategies and mastering various communication techniques - within the confines and structure of the therapy room. Even when the therapy programme includes other individuals and is widened to embrace a number of different communicative situations with a broad group of people, as long as there is some form of structure or mediation, it is likely that our clients will experience success. Their difficulty lies in self-activation, in the ability to independently integrate those skills in adaptive ways in changing and unique environments, in response to fluctuating communication demands. This may not be so easily done, without treating the fundamental inhibition and working memory skills on which self-regulation relies. Perhaps in such cases where EF deficit is intractable, the social approaches which rely heavily on environmental support and collaboration from significant others to help with cueing and mediation are ideal.

It is also relevant to examine some of the existing literature which reports results of treatments aimed at remediating these deficits. While some researchers have reported successful transfer of the treatment of attention to functional language skills, on the whole the existing treatment studies are disappointing.

In attempting to treat aspects of attention, Marshall and Barnes (2007) investigated the use of a treatment strategy which used directed vision in order to counteract effects of rapid auditory extinction without success. Murray, Keeton and Karcher (2006) used the Attention Process Training-II program to improve the attention abilities of RW, a patient with mild conduction aphasia and concomitant attention and working memory deficits. Their results showed that RW improved on trained attention tasks and made modest gains on standardized tests and probes that evaluated cognitive skills related to treatment activities. Nominal change in auditory comprehension and untrained attention and memory functions was observed, but neither RW nor his spouse reported noticeable improvements in his daily attention or communication abilities. As mentioned in previous chapters other authors have reported benefits of attention training on comprehension (Helm-Estabrooks et al., 2000), reading (Coelho, 2005), naming (Crosson et al., 2007) and conversational speech (Hardin & Ramsberger, 2004).
Some treatment studies related to the improvement of working memory have also been published. Majerus and van der Linden (2001) reported a single case of a patient with fluent conduction aphasia, where short term memory was successfully treated. They further interpreted their findings to suggest that the intervention had assisted to normalize activation decay thus allowing for the repetition of increasingly longer strings of words and sentences. However, they did not demonstrate a corresponding improvement in functional communication. Francis, Clark and Humphreys, (2003) reported on a single case treated for working memory and short term memory deficits. Certain aspects of short-term and working memory improved post-therapy, notably an increase in digit span and an ability to repeat more words in sentences. There was a limited generalisation of improvement to comprehension tasks, meaning that the client could understand longer sentences and required fewer repetitions.

The research thus far has reported modest improvements in discrete skills that are directly treated with very few studies indicating gains in functional settings. This trend indicates that structured retraining may enhance specific skills, but that positive changes in broader attention and untrained functions are less likely. The fact that so few show transferability to functional communication makes their relevance questionable. In this regard, Murray (2004) has suggested that pharmacological treatment of nonverbal deficits in aphasia needs to be considered, owing to the limited success of behavioural approaches. This will be further discussed below in relation to implications for future research.

In the absence of clearly defined drug protocols for language and EF disorders in stroke, treatment plans need to be, as always, carefully and individually crafted with specific regard to the relative strengths and deficits in our patients’ EF profiles. Significantly poor inhibitory ability and depleted working memory capacity skill make it appear unlikely that strategies that rely on self-initiation are primary treatment choices. Perhaps these patients would be better served by an emphasis on environmental support and compensatory strategies that are generated by other advocates in the patient’s life. This is not to say that we should not explore executive treatments for these patients. The challenge is to find creative ways in which the environment and communication opportunities can be primed to exert the level of structure needed to activate useful conversational approaches. On the other hand, those patients like Grace and Tumi who present with essentially intact self-monitoring systems could make good use of those
techniques that require self-monitoring, self-activation and proactive choice and utilization of various strategies.

7.4.2 Research implications

The area of EF deficit in adults with aphasia and stroke is an exciting field promising creative and novel research endeavours. Spordone (1996) in relation to EF testing and ecological validity states that we have reached a point in our investigations where ‘we know enough to know how much we don’t know.’ (p. 41). This is true of EF deficits in this population as well.

There are numerous challenges ahead, starting with operationalized definitions of the processes and skills that comprise EF. Future projects will continue to delineate the relationships of various EF to one another, their neurological substrates and neurochemical interactions. To this end, the advance and increasing frequency with which neuroimaging is available, will benefit this area, adding to an already imposing body of knowledge. This research has suggested that the relationship between language and EF is opaque, and yet there is a sense that perhaps in aphasia, some of the language deficits observed may in fact have executive contributions which impose pervasive impediments on the ability to access, mobilize and employ psycholinguistic factors. Ultimately, this harks back to the question about the relationship between thought and language and how the different constructs influence one another. Further study with individuals with aphasia would hopefully open doors to this intricate enigma.

Of particular interest is the relationship between disorders of affect and functional communication outcomes. This is an area ripe for investigation and has significant impact on how we interpret communication symptoms of aphasia.

In addition, despite a history of dedicated research over the last couple of decades, the concept of drug therapy for the treatment of aphasia is considered to some extent ‘novel’ and ‘revolutionary’ (Klein & Albert, 2004, p. 193). Research studies have not convincingly demonstrated overwhelming evidence to support the routine use of drugs as either a complementary or alternative treatment for the language and cognitive deficits associated with stroke. Rigorous research is still needed to determine which deficits, in which patients and in which situations will benefit from which types of drug therapy. Well-designed, randomised,
well controlled clinical research studies with numbers of participants, great enough to support treatment protocols are greatly needed (Greener, Enderby & Whurr, 2004).

In order to generalize findings and to ensure cross-setting transferability, the temptation to recruit large numbers of participants to studies is understandable. However, as this project has demonstrated the heterogeneity and inherent uniqueness of each case, arising from individual conglomerations of variables must be taken into account. More studies that concentrate on well documented and carefully delineated single cases have intrinsic value as they can be used to debunk existing theories, bring new variables to light, provide supportive evidence for a theory and be used collectively to form the basis of a theory (O’Leary, 2004).

7.5 CONCLUDING COMMENTS
Stroke is the forerunner of vascular disease in the epidemiologic or health transition and all evidence suggests that South Africa is well into that transition. It is a devastating condition with high levels of disability not least of which is the loss or impairment of communicative functions.

Executive function deficits exist in individuals who have experienced stroke. These deficits may be present irrespective of age, gender, handedness, language localization, type of aphasia or site of lesion. Even in the most preserved patients, there may be some small executive deficit that provides a deeper explanation into communication strengths and difficulties. EF is extremely sensitive to its neurological environment and as a result is highly susceptible to disruption. Individuals with executive dysfunction will adapt and respond uniquely depending on a range of variables and these adaptations and reactions will be observable in the act of having a conversation.

In much the same way that Howard Gardner (2004) talks about intelligence being a multifaceted gestalt of many aptitudes, the ability to make one’s way around the social necessity of conversation, represents an accumulation of multiple ‘intelligences’ and dispositions. The identification of relevant constructs is a start to understanding these factors, but they are uniquely and distinctively affected by individual circumstances.
In completing this research, one has the sense that the variables we observe, describe, manipulate and interpret are merely external features of a system that is almost inexplicable in its complexity and awesome in its depth and sophistication. As we learn more and attempt to test different ways of integrating various factors to the benefit of our clients, new relationships and patterns will emerge. Our skill in classifying and refining these blueprints will also progress. For now, we have just scratched the surface.


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**APPENDIX A**

Table A.1: Executive Functioning Battery

<table>
<thead>
<tr>
<th>BARKLEY’S CONSTRUCTS</th>
<th>TEST</th>
<th>RATIONALE</th>
<th>ADMINISTRATION AND SCORING</th>
<th>ADAPTATIONS</th>
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</thead>
<tbody>
<tr>
<td><strong>BEHAVIOURAL INHIBITION</strong> (Interference control)</td>
<td>Digit Span (forward) (Lezak, Howieson, Loring, Hannay &amp; Fischer, 2004).</td>
<td>This test measures efficiency of attention i.e. freedom from distractibility.</td>
<td>Tester presents strings of digits of increasing length. Participant repeats verbatim. Scored in terms of number of digits recalled. 5-7 = normal, 4 = borderline.</td>
<td>For individuals with verbal or oral Apraxia, the point digit span was also used (this parallels the digit span test in all respects except that the response modality does not require speech). This was used to determine whether a nonverbal response format affected performance.</td>
</tr>
<tr>
<td></td>
<td>Stroop Colour-Word Interference Test* (Golden, 1978)</td>
<td>This well established task of attentional inhibition is regarded as being sensitive to executive functions (Golden. 1978; Lezak, 1995). In particular, breakdown on this task signifies difficulty warding off distractions (Holst &amp; Vilkki, 1988). Although it requires speeded verbal responding, the ratio scores control for general reading speed.</td>
<td>In trial 1, participants read a list of words printed in various colours. In trial 2, they name the colour the word is printed in, which does not correspond to the name of the colour that is written. Scored in terms of the ratio of time taken to complete trial 2 compared to trial 1. Norm = 2.28</td>
<td>Materials were enlarged and placed directly in line of vision. Scored as ratio between trials rather than in terms of absolute time taken to completion.</td>
</tr>
<tr>
<td>Response inhibition</td>
<td>Trail Making* (Lezak et al., 2004)</td>
<td>This is a well established, sensitive test of attention shifting (as well as motor speed and visual search), backed by a solid body of research (Spreen &amp; Strauss, 1998). Although the test uses letters and numbers, it is considered by many to be essentially a nonverbal test (Burgess &amp; Strauss, 1998).</td>
<td>In trial 1, participants connect consecutive numbers from 1 to 25. In trial 2, they join alternating letters and numbers in consecutive order i.e. 1,A,2,B,3,C. Scored in terms of the ratio of time taken to complete trial 2 compared to trial 1. Norm = 2.2</td>
<td>Pathways for Trails A and B were identical (i.e. the placement of consecutive numbers and letters followed the same spatial pattern for both trials) to avoid excess weighting of motor component. These were then changed for each phase of the study. For the one participant with hemi-neglect, materials were enlarged and placed in line of unaffected vision. Scored as ratio between trials rather than in terms of absolute time taken to completion.</td>
</tr>
<tr>
<td>BARKLEY’S CONSTRUCTS</td>
<td>TEST</td>
<td>RATIONALE</td>
<td>ADMINISTRATION AND SCORING</td>
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<tr>
<td>WORKING MEMORY</td>
<td>Self Ordered Pointing Test* (Spreen &amp; Strauss, 1998).</td>
<td>This test is a nonverbal assessment of working and strategic memory. Evidence suggests that this task captures important aspects of executive functioning (Spreen &amp; Strauss, 1998).</td>
<td>The same set of stimulus items are arranged in varying layouts on different pages. Examinees are required to point to a different item on each page, with the sole restriction being that they are not allowed to point to a stimulus already chosen. The test is divided into four sections, consisting of six, eight, ten and twelve stimulus sheets. Three consecutive trials of each section are administered. Scored in terms of number of errors (items chosen more than once). Mean number of errors = 4.68 (2.53)</td>
<td>Materials were enlarged and placed directly in line of vision.</td>
</tr>
<tr>
<td>Medical College of Georgia complex figures* (Spreen &amp; Strauss, 1998)</td>
<td>Storage and recall of complex information over a lengthy delay period is considered a working memory function, particularly when that information needs to be organised through the use of strategies as in Complex Figures tests (Barkley, 1997). This nonverbal test specifically addresses all these aspects of working memory function and does not rely on verbal or linguistic response.</td>
<td>Participants were given a complex figure to copy. After a 30 minute delay period they were requested to replicate the drawing from memory. This test is scored in terms of elements retained and graded according to accuracy, distortion and location of the reproduction. Mean score for copy = 32.31 Mean score for recall = 16.56</td>
<td>Materials were enlarged and placed directly in line of vision. The copy task acted as a control to ensure that participants could in fact see and draw the target figure before being required to hold it in mind and replicate it from memory.</td>
<td></td>
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</table>
Table A.1 cont… Executive functioning battery

<table>
<thead>
<tr>
<th>BARKLEY’S CONSTRUCTS</th>
<th>TEST</th>
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</tr>
</thead>
<tbody>
<tr>
<td>WORKING MEMORY cont…</td>
<td>Wisconsin Card Sorting Test (Ormond Software Enterprises, 1999)</td>
<td>This test measures a number of different skills. Traditionally it is the classic test of set shifting, however it is also characterised as a test of working memory owing to its assessment of effective processing of feedback information to monitor and adjust performance. It has also been characterised as a test of concept formation and abstract reasoning skills (Lezak et al., 2004). Its utility with individuals with aphasia has been rated as being high (Keil &amp; Kaszniak, 2002).</td>
<td>Examinees are required to sort cards according to three principles: colour, form and number., using feedback of ‘correct’ or ‘incorrect’ from the examiner. After ten correct sorts the sorting principle is changed without warning. Scores include number of categories achieved, proportion of errors and perseveration.</td>
<td>Computer mouse manipulated by tester</td>
</tr>
<tr>
<td></td>
<td>Digit Span (backwards) (Lezak et al., 2004)</td>
<td>This test is used in many neuropsychological batteries to determine ability working memory or mental tracking ability (Barkley, 1997)</td>
<td>The tester presents a series of numbers which the participant has to repeat in the reverse order. 4-5 = normal; 3 = borderline/defective</td>
<td>As with digits forward, a pointing format was used in addition to verbal formats where verbal apraxia was suspected of confounding results.</td>
</tr>
<tr>
<td>REGULATION OF AFFECT</td>
<td>Neuropsychology Behaviour and Affect Profile (Nelson, Satz &amp; D’Elia, 1994)</td>
<td>This is one of the very few affect inventories, developed specifically for use with neurologically involved individuals available with norms for the stroke population (Spreen &amp; Strauss, 1998). It includes assessment of constructs frequently implicated in disorders of mood, motivation and expression.</td>
<td>A familiar caregiver or significant other reads the questionnaire and marks agree or disagree for each item. Agree items are then tallied for each construct assessed and compared to T-scores to examine presence or absence of that feature.</td>
<td>None needed</td>
</tr>
</tbody>
</table>
Table A.1 cont… Executive functioning battery

<table>
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<tr>
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<tbody>
<tr>
<td>INTERNALIZATION OF SPEECH</td>
<td>Tower of London* (Shallice, 1982)</td>
<td>This test is a well accepted measure of planning skill (Keil &amp; Kaszniak, 2002). For successful completion examinees must plan ahead in a series of sub-goals, follow specified moves and monitor and change plans as necessary. Usefulness with individuals with aphasia is considered high (Keil &amp; Kaszniak, 2002)</td>
<td>Participants need to move pegs from a starting position to a given goal position in a limited number of transfers. This test is usually scored in terms of accuracy, latency to initial move and total time completion. In this research, although these measures were all taken, interpretation relied heavily on qualitative observations.</td>
<td>Timing constraints were interpreted with caution. Detailed notes were used in terms of number of attempts to correct completion, number of attempts before failing or giving up, number of different strategies used vs. perseverance on same strategy.</td>
</tr>
<tr>
<td></td>
<td>Raven’s Progressive Matrices* (Raven, Raven &amp; Court, 1998)</td>
<td>This test has been shown to correspond strongly with verbal measures of intelligence. It is considered a test of logical reasoning and problem solving. Administration in the language impaired population seems entirely viable although deficits with constructional praxis (rules out during initial testing) may confound interpretation (Keil &amp; Kaszniak, 2002).</td>
<td>A series of patterns requiring matching and analogy problems are presented. Examinees are required to conceptualise the relationships within the design in order to choose the correct option to complete the pattern. Accurate responses are summed and ranked.</td>
<td>Material presented directly within visual field for participant with neglect.</td>
</tr>
</tbody>
</table>
Table A.1 cont… Executive functioning battery

<table>
<thead>
<tr>
<th>BARKLEY’S CONSTRUCTS</th>
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<tbody>
<tr>
<td>RECONSTITUTION</td>
<td>Five Point Test (Spreen &amp; Strauss, 1998)</td>
<td>Both the five point task and the design fluency task fall under the general heading of ‘Design Generation’, an effective means of exploring executive functioning. It taps into the ability self-monitor, remember and follow the given rules, use of strategies but primarily productivity and ability to vary responses rapidly and produce novel and creative output. The major difference between the two tests is the degree to which structure is provided.</td>
<td>The examinee is given a page on which 40 squares in a 5 x 8 array are printed, each containing five symmetrically and identically arranged dots. The examinee must make as many different figures a possible within five minutes by connecting the dots with straight lines, without repeating any figure.</td>
<td>None needed</td>
</tr>
<tr>
<td>RECONSTITUTION</td>
<td>Design Fluency (Spreen &amp; Strauss, 1998)</td>
<td>See above</td>
<td>The free condition of this test was administered where participants were asked to invent drawings that represent neither actual objects nor nameable forms (e.g. geometric shapes) that are not merely scribbles. Both tests are scored in terms of the number of unique designs created. For the Five Point Test, a percentage of perseverative responses is calculated. 15% perseveration is considered significant (Spreen &amp; Strauss, 1998). For Design fluency, the norm = 16.2 designs.</td>
<td>None needed</td>
</tr>
</tbody>
</table>
APPENDIX B

PARTICIPANT AND CAREGIVER INFORMATION SHEET

This information sheet is to be read with the researcher, in conjunction with the specially adapted participant consent form with pictorial aids.

INTRODUCTION

Hello, my name is Tali Frankel and I am a PhD student in the Speech Therapy department at Wits University. I am inviting you to consider participating voluntarily in my study. The document you are reading is an information sheet. In it I will describe the reasons for why the study is being done, how it will be conducted and what would be required of participants who are willing to volunteer. I will read this document with you in conjunction with a specially adapted form with pictures that will help to make the discussion clear to you. You can ask questions at any time about any aspect of this study. When we have finished reading the information sheet and talking about the study, I will make sure that you have understood everything that we have spoken about by asking you some key questions and giving you more time to ask questions or clarify any information. I will give you some time to think about our discussion and phone you the within the next few weeks to ask if you have made a decision about participating. If you agree, I will ask you to sign a form that shows that you consent to participating. You will then be given a copy of the form you sign to keep.

WHY IS THIS STUDY BEING DONE?

As we learn more about strokes, we understand that not only do people who have had strokes suffer from difficulties communicating and talking, but also with thinking. By this I mean that people may have difficulty paying attention to things, remembering things or organising and planning their daily activities. I am interested in these communication and thinking difficulties that result from strokes. I particularly want to see if these thinking difficulties exist and if they do what kind of difficulties they are. I also want to see if these difficulties influence the way you are able to have conversations with your friends and family members. We are also learning that some medicines may be helpful in assisting recovery from strokes. I want to see if in fact medicine can help improve thinking and conversational abilities. As you have had a stroke, which took place at least six months ago and live at home within your community, you are being invited to volunteer. If you agree to take part in the study you will be one of about ten people who will be asked to participate in the same manner.
NAME OF MEDICINE BEING USED IN THE STUDY
If you participate in this study you will take medicine called Keppra. This has to be prescribed by a
doctor. In this study the doctor will be a neurologist called Dr. Saffer. He will examine you and talk to
your present doctor. Together they will decide if it is safe for you to participate in the study. If they are
cconcerned that other medications or medical conditions make it unsafe for you to participate, you will
not take part in the study.

WHAT DO I HAVE TO DO?
The study will take place over a period of approximately thirteen weeks.
1) You will be tested by a neurologist who can give me information about your stroke. If you choose to
participate, your medical records must be available to this neurologist (Dr. Saffer) who can make sure
that there will be no danger to you if you take the medicine used in this study. Dr. Saffer will visit you
at your home in order to do the assessment. If this is not possible, I will arrange transport for you to a
different venue. Dr. Saffer and I will also talk to your current doctor to make sure that it is safe for you
to participate in the study.

2) I will visit you at home in order to test your thinking skills. I will ask you to do several different tests
with me that will take approximately three hours. The testing may take longer especially if we take
breaks when you want them. During the testing I will ask you to do the following:
   a) Repeat numbers after me in the same order that I say them to you
   b) Repeat numbers after me in the reverse order to the one in which I say them to you
   c) Read single words
   d) Name the colours that several words are printed in
   e) Join together numbers and letters in a specific order
   f) Look at detailed pictures and copy them from memory as well as with the picture in front of you
   g) Look at beads on a peg and then move a different set of beads to match the first set in as few
      moves as possible
   h) Make many different kinds of line patterns by connecting a series of dots
This testing will take place four times during the following weeks of the study.
Week 1
Week 5
Week 9
Week 13 (Final week)
I will do all this testing with you. Some of the tests will be timed.

3) You will have to video-tape yourself having a conversation with someone that you talk to often. You and I will discuss times when you usually have a conversation with a friend or family member. I will set up the video camera in advance in your home or at your friend or family member’s homes. I will leave when you start having the conversation. If there is no-one with whom you regularly converse or it is difficult to find a convenient time, I will act as your conversation partner. You will be videotaped for about half an hour. Only ten minutes of the recording will be used, probably the middle ten minutes. These recordings will take place in the same weeks as the thinking tests:

   Week 1
   Week 5
   Week 9
   Week 13 (Final week)

After you give me the video tapes I will watch them very carefully. I will choose ten minutes from the conversation and write down everything about it. I will analyse the information to see how you manage the conversation. I will give some of the pages of the conversation to another researcher and ask them to tell me what they think about how you manage the conversation.

4) You will nominate someone close to you, who spends a lot of time with you to fill out a form. This person, if they agree will fill out a form that tells me about the way you cope with your daily communication and other activities. This form gives information about your emotional wellbeing and behaviour. They will fill out the form four times, in the same weeks when we do the thinking tests and the video recordings.

   Week 1
   Week 5
   Week 9
   Week 13 (Final week)

5) You will be part of a drug trial. This means that for one month you will take medicine called Keppra. The Keppra tablet will be inside a capsule, which you may not open. You must swallow the capsule whole.
You will take 2 capsules a day, every day, for 1 month. This is a dosage of 1500 mg of Keppra a day – 750 mg per capsule, one to be taken in the morning and one in the afternoon. You will also have to take a capsule that has no medicine in it. Therefore for another month in the study, you will have to take 2 capsules every day for one month which have no medicine in them. Again you will have to swallow the capsules whole without opening them. You will take one in the morning and one in the afternoon.

Both the capsules with the medicine and the capsules without will be given to you by a person you nominate – this person must live with you. They will sign out every capsule to make sure that you take every single one at the right times, every day.

You will not be able to tell which month you are taking the medicine and which month you are taking the capsules without medicine. The capsules will look identical. I will also not know when you are taking the medicine and neither will the person who gives you the capsules. We will only find out after the study is finished and we have analysed all the results from all the testing.

You will therefore either take the medicine during Weeks 2, 3, 4 and 5 and then swap and take the capsules without the medicine in Weeks 6, 7, 8 and 9. Or it could be the other way round – take the capsules without the medicine in Weeks 2, 3, 4 and 5 and then swap and take the medicine in Weeks 6, 7, 8 and 9.

The reason for making you take capsules with and without medicine in them is that it is one of the best ways we can see if Keppra really helps or not.

The study therefore has several stages. The time line below will help you to visualise the time frame for the study and what will be done at each stage:

<table>
<thead>
<tr>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 5</th>
<th>Week 6</th>
<th>Week 9</th>
<th>Week 13</th>
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</thead>
<tbody>
<tr>
<td>Baseline testing</td>
<td>Start taking</td>
<td>Start taking</td>
<td>Testing 3</td>
<td>Testing 4</td>
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<tr>
<td>1) Neurologist</td>
<td>1st set of</td>
<td>2nd set of</td>
<td>1) Thinking</td>
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<tr>
<td>2) Thinking tests</td>
<td>capsules</td>
<td>capsules</td>
<td>tests</td>
<td></td>
<td></td>
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<tr>
<td>3) Friend/family</td>
<td>1) Thinking tests</td>
<td>2) Forms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>to fill in form</td>
<td>2) Forms</td>
<td>3) Videotape</td>
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<td></td>
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<tr>
<td>4) Videotape conversation</td>
<td>3) Videotape</td>
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<td>2) Forms</td>
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<td>3) Videotape</td>
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</tbody>
</table>

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WHAT HAPPENS TO ALL THE TEST RESULTS?
At the end of the study, I will write up all the results in a thesis and submit the thesis for a PhD in speech therapy to Wits University. I will also try to publish the results in a professional journal. You will be given the results of the study if you would like to know them. If your doctor or therapists you are currently seeing would like to see the results and you give permission, they will also be given a copy.

WHAT ARE THE BENEFITS OF TAKING KEPRA?
I am hoping that Keppra will allow you to concentrate better, learn more effectively, remember things more easily and be available to mediation from your environment i.e. input from your therapists, family members and friends. I hope that you will therefore be able to communicate better in conversations and feel more able to plan, organise and monitor your daily activities. These benefits are not guaranteed and we will have to wait for the outcome of the testing to determine whether or not you have benefited from the treatment.

WHAT ARE THE RISKS OF TAKING KEPRA?
The side effects that can occur are seen very rarely. Keppra has been seen to be safe for use in stroke patients. However, the following side effects can occur: nervousness and insomnia, hypersensitivity to the skin, loss of appetite, nausea, dizziness, headaches, palpitations, drowsiness, abdominal pain and weight loss during prolonged therapy. If you experience any discomfort or become concerned about taking the medication at any time during the study, you have the right to withdraw immediately.

No negative consequences to you or the project will result.

WHY WOULD I BE WITHDRAWN FROM THE STUDY EARLY?
1) If you begin to experience negative side effects from the medication that are harmful to your health, while you are taking the capsules.
2) If new medications or medical conditions that are contraindicated when taking Keppra are identified or prescribed during the course of the study.
WHAT HAPPENS IF I BENEFIT FROM THE DRUG?
At the end of the study, you will not be given any more medication even if you have benefited from it. Keppra must be prescribed. If you would like to continue taking Keppra, you will need to consult with a neurologist and your doctor about this possibility. If they feel that this would be beneficial to you, you will have to purchase Keppra at your own expense.

WHAT WILL THIS STUDY COST ME?
The study will not cost you anything. I have been given funds in order to provide you with the Keppra free of charge. You will be compensated for travel and doctor’s consultations will either be free of charge or paid for you by my research grants.

CONFIDENTIALITY
Your identity and the identity of your family members or friends who participate will be protected throughout the study. Your names will not appear on any documents, unless you specifically want it to. The video material will be destroyed after it has been analysed, unless you give separate permission to allow me to keep the material for future research purposes.

WHAT ARE MY RIGHTS?
1) You have the right to withdraw from the study at any time, with immediate effect with no negative consequences to you or the project.
2) You have the right to withhold any of the video material from me
3) You have the right to see the results of the study and have these results distributed to your doctor and therapists should you so desire.
4) You have the right to contact me at any time with any queries or concerns
5) You have the right to contact Dr Saffer the neurologist about any of your medical concerns

If you have any questions you need to ask me or Dr Saffer, our contact details are below.

Tali Dr. Saffer
Cel - 072  492  0664
APPENDIX C

PARTICIPANT CONSENT FORM

I _______________________ (name of participant), the undersigned, agree to volunteer

in this study in writing. In giving consent I understand the following:

1. This research is for a degree – PhD at Wits

2. I will need to give all my medical records – and be

examined by a doctor Dr Saffer to see if I can participate.

Dr Saffer will also speak to my treating doctor – they will consult and decide if
it is safe for me to participate in this study.

The study will take 13 weeks all together
3. I will be tested at home to see how good my memory, attention and thinking skills are by the researcher – Tali. I will be asked to do several things including: repeating numbers, pencil and paper tasks, reading, naming colours, copying pictures, moving beads on sticks and drawing patterns.

This testing will take place 4 times, during weeks, 1, 5, 9 and 13.

4. 30 minutes of conversations with people I talk to at home will be video recorded, written down and analysed.

This will also take place 4 times, during weeks, 1, 5, 9 and 13.

10 minutes of each recording will be used.
5. My friend / spouse / son / daughter / boyfriend / girlfriend / relative will fill out a form describing how I communicate and behave in different situations. This will also take place 4 times, during weeks, 1, 5, 9 and 13.

6. I will be on a drug trial. During the study I will take Keppra. 2 capsules every day for 1 month.

I will also be given 2 capsules with no medicine every day for 1 month. This will take place during Weeks 2 - 9.

No-one will know which capsules have medicine and which ones do not.

6. I may experience some benefits – I may have improved concentration, memory, problem solving, planning and organisation. But there are no guarantees.
7. I may have some side effects
   a) nervousness
   b) insomnia,
   c) extra sensitivity to the skin,
   d) loss of appetite and nausea,
   e) dizziness and headaches,
   g) pain in the stomach and weight loss
   h) tremor
   i) feeling sleepy

8. If I have these side effects I have the right to leave the study immediately!
9. The results will be written in a thesis for a degree. I can have the results of the study, so can my doctor or therapist if I want.

10. After the study is finished, I will receive no more medication, even if the Keppra helped me during the study. If I want to continue taking the Keppra, I must check with Dr Saffer and my doctor. If they think it is a good idea, I will have to pay for the medicine myself.

11. My name will not be used at all during the study or afterwards.

12. I can hold back any video material from the researcher. The videos may be used in the future for research.

13. I can leave the study at any stage, for any reason and without negative consequences to myself or the project.
14. I can contact Tali at any time to ask about the study. I can also contact Dr. Saffer if I need to ask him a medical question.

15. I have read or spoken about and understood this document and the information sheet. I recognise Tali Frankel as the primary researcher for this project and agree to participate in her study.

__________________________   __________________________
Participant name (Print)    Participant signature

__________________________   __________________________
Date and place

__________________________   __________________________
Researcher’s name (Print)    Researcher signature

__________________________   __________________________
Date and place

__________________________   __________________________
Witness name (Print)     Witness signature

__________________________
Date and place
APPENDIX D

PARTICIPANT CONSENT FORM FOR THE USE OF VIDEO RECORDINGS

I ______________________ (participant’s name in print) the undersigned give written consent to confirm my participation in this study. In giving consent I understand the following:

1. **30 minutes** of conversations with people I talk to at home will be
   - video recorded,
   - written down
   - and analysed

   10 minutes of each recording will be used

   This will take place **4 times, during weeks, 1, 5, 9 and 13** of the study.

2. Portions of these transcripts will be given to an external rater who will analyse the conversations.

3. I have the right to withhold any of the video material at any time from the researcher.

   At the end of the study, Tali will keep the tapes for future research.
4. My name will not be used at all during the study or afterwards, unless I want it to be.

5. I have the right to withdraw from this study at any time, with immediate effect, without any negative consequences to myself, the participant or the project.

6. I have read and fully understood the accompanying information sheet and agree to be videotaped for this study.

7. I can direct any of my enquiries to Tali Frankel, the primary researcher for this project at any time at the details listed on the signature page.

Participant name (Print)   Participant signature

Date and place

Researcher’s name (Print)   Researcher signature

Date and place

Witness name (Print)   Witness signature

Date and place

Tali
Home - (011) 640 1989  Cel - 072 492 0664
APPENDIX E

CAREGIVER ASSENT FORM

I ______________________ (caregiver’s name in print) the undersigned give written consent to confirm my participation in this study. In giving consent I understand the following:

1. I am participating in research that is being conducted for the purposes of a PhD in speech therapy at Wits University.

2. As part of this research I will fill out a form that measures the participant’s communication skills and behaviour across a variety of situations. I will fill out this form during weeks 1, 5, 9 and 13 of the study.

3. I will also be in charge of giving the participant capsules during weeks 2 – 9 of the study. I will be given 1 set of capsules for weeks 2 – 5 and another set of capsules for weeks 6 – 9. The sets look identical but one set contains Keppra while the other does not. I will give the participant two capsules, one in the morning (8:00 a.m.) and one in the afternoon (4:00 a.m.) every day for a month from first set 1 and then from set 2. I will fill in the control sheet by ticking each capsule as it is taken and signing at the end of each day. This way we can ensure that every capsule is taken on time, every day for 2 months. I will not know when the participant is taking the medicine and when they are taking the capsules without.

4. Results of this study will be published in a thesis that will be submitted for a degree. The results may also be published in a professional journal. The results will be made available to me upon my request and subject to permission from the participant.

5. My identity will be protected throughout this study and my anonymity is assured.

6. I have the right to withdraw from this study at any time, with immediate effect, without any negative consequences to myself, the participant or the project.

7. I have read and fully understood the accompanying information sheet and agree to voluntarily participate in this study.

8. I can direct any of my enquiries to Tali Frankel, the primary researcher for this project at any time at the details listed on the signature page.
SIGNATURE PAGE

__________________________   __________________________
Caregiver name (Print)    Caregiver signature

__________________________
Date and place

__________________________   __________________________
Researcher’s name (Print)    Researcher signature

__________________________
Date and place

__________________________   __________________________
Witness name (Print)     Witness signature

__________________________
Date and place

Tali
Home   -  (011) 640 1989
Cel     -  072 492 0664
APPENDIX F

CAREGIVER ASSENT FORM FOR THE USE OF VIDEO RECORDINGS

I ______________________ (caregiver’s name in print) the undersigned give written consent to confirm my participation in this study. In giving consent I understand the following:

1. I will be videotaped having a conversation with the participant a total of four times during the course of this study. These recordings will take place at the participant’s home during weeks 1, 5, 9 and 13 of the study. The video machine will be set up by the researcher who will then leave me and the participant to have as natural a conversation as possible. The conversation will be recorded for approximately half an hour.

2. The researcher will view the tapes and transcribe and analyse approximately ten minutes of each of the four recordings.

3. Portions of these transcripts will be submitted to an external rater who will analyse the conversations.

4. I have the right to withhold any of the video material at any time from the researcher.

5. My identity will be protected throughout this study and my anonymity is assured.

6. I have the right to withdraw from this study at any time, with immediate effect, without any negative consequences to myself, the participant or the project.

7. At the end of this study, I and the participant agree to let the researcher keep them for future research purposes.

8. I have read and fully understood the accompanying information sheet and agree to be videotaped for this study.

9. I can direct any of my enquiries to Tali Frankel, the primary researcher for this project at any time at the details listed on the signature page.
## APPENDIX G

### PARTICIPANT DATA CONTROL SHEET

#### PHASE 1 – BAG A

<table>
<thead>
<tr>
<th>Day</th>
<th>Capsule 1 – 8:00 a.m.</th>
<th>Capsule 2 – 6:00 p.m.</th>
<th>Signed</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Saturday 4 March</td>
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<td></td>
<td></td>
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<tr>
<td>2 Sunday 5 March</td>
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<td>3 Monday 6 March</td>
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<td>4 Tuesday 7 March</td>
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<td>5 Wednesday 8 March</td>
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<tr>
<td>6 Thursday 9 March</td>
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<td>7 Friday 10 March</td>
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<td>8 Saturday 11 March</td>
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<td>9 Sunday 12 March</td>
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<td>10 Monday 13 March</td>
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<td>11 Tuesday 14 March</td>
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<td>12 Wednesday 15 March</td>
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<td>13 Thursday 16 March</td>
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<td>14 Friday 17 March</td>
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<td>15 Saturday 18 March</td>
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<td>16 Sunday 19 March</td>
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<td>19 Wednesday 22 March</td>
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<td>20 Thursday 23 March</td>
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<td>21 Friday 24 March</td>
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<td>22 Saturday 25 March</td>
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<td>23 Sunday 26 March</td>
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<td>24 Monday 27 March</td>
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<td>25 Tuesday 28 March</td>
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<td>26 Wednesday 29 March</td>
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<td>27 Thursday 30 March</td>
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<td>28 Friday 31 March</td>
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<td>29 Saturday 1 April</td>
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<td></td>
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<tr>
<td>30 Sunday 2 April</td>
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<td></td>
</tr>
</tbody>
</table>
INTRODUCTION
Hello, my name is Tali Frankel and I am a PhD student in the Speech Therapy department at Wits University. I am inviting you to participate in my study. The document you are reading is an information sheet. In it I will describe the reasons for why the study is being done, how it will be conducted and what would be required of participants who are willing to volunteer. I will read this document with you. You can ask questions at any time about any aspect of this study. When we have finished reading the information sheet and talking about the study, I will make sure that you have understood everything that we have spoken about by asking you some key questions and giving you more time to ask questions or clarify any information. I will give you some time to think about our discussion and phone you the next day to ask if you have made a decision about participating. If you agree, I will ask you to sign a form that shows that you consent to participate. You will then be given a copy of the form you sign to keep.

WHY IS THIS STUDY BEING DONE?
I am conducting another study where I will be looking at the issues of conversation and thinking skills in people who have had strokes. As part of that study, I will be asking several people who have had strokes to complete some pencil and paper tasks that involve drawing lines and pictures. As the people who are participating in that study may not be able to use their preferred hand (either because of weakness or paralysis of the hand and / or arm following the stroke) I need to be careful about the way I interpret their test results. Using their less-preferred hand may make their test results different to what they would be if they were using their preferred hand. In order to see whether or not there would in fact be a difference between using a preferred as opposed to a non-preferred hand, I am conducting this present study.

You are being invited to participate because you fall within the same age range, have the same preferred hand and have the same level of education as the people who will be participating in the stroke study. If you agree to participate, you will be one of approximately 30 people (men and women)
who will be asked to complete a range of tests using either your non-dominant hand first and then your dominant hand (right hand) or alternatively your dominant hand first and then your non-dominant hand.

WHAT DO I HAVE TO DO?

The study will take place over a period of approximately one hour.

1) You will be tested by me at your home. You will be asked to
   i) Look at a piece of paper with letters and cross some of them out
   j) Join together numbers and letters in a specific order using a pencil
   k) Look at detailed pictures and copy them from memory as well as with the picture in front of you
   l) Find your way through a paper maze by drawing with a pencil
   m) Make many different kinds of line patterns by connecting a series of dots

2) After you have completed the five tasks above, you will complete the same tasks with a slightly different form of each test that is the same level of difficulty, this time using the other hand. The order in which you complete the tasks above (i.e. whether you use your dominant or non-dominant hand first will be randomly decided).

WHAT HAPPENS TO ALL THE TEST RESULTS?

I will look at all the test results of people doing the tests with their left and right hands and see if there is a significant difference in performance depending on what hand is used. This will be done by using statistical formulae. I will include my findings of this study when I write up the results of the stroke study, in a thesis and submit the thesis for a PhD in speech therapy to Wits University. If there is a significant difference between the left and right hand tests, I will use the information for the way you completed the tests with your left hand for comparison against the performance of the stroke patients. I will also try to publish the results in a professional journal. You will be given the results of the study if you would like to know them.

WHAT ARE THE BENEFITS OF PARTICIPATING?

There are no direct benefits to you if you participate in this study. If you volunteer you will help me to determine the best way of interpreting the tests from the stroke study and increase what we know about the difference between dominant and non-dominant hand performance on these tests.
WHAT ARE THE RISKS?
There are no risks to you if you participate in this study.

WHAT WILL THIS STUDY COST ME?
The study will not cost you anything. I will visit you at your home at your convenience. I will provide all stationary to be used.

CONFIDENTIALITY
Your identity will be protected throughout the study. Your name will not appear on any documents.

WHAT ARE MY RIGHTS?
1) You have the right to withdraw from the study at any time with immediate effect with no negative consequences to you or the project.
2) You have the right to see the results of the study
3) You have the right to contact me at any time with any queries

If you have any questions you need to ask me my contact details are below.
Tali

Home  – (011) 640 1989
Cel  - 072 492 0664
APPENDIX I

CONTROL GROUP CONSENT FORM

I _________________________ (name in print) the undersigned, give written consent to confirm my participation in this study. In giving consent I understand the following:

1. I am participating in research that is being used for the purposes of a PhD in speech therapy at Wits University.
2. As part of the research I will be tested on a number of paper and pencil tasks where I will be asked to do the following:
   a) Look at a piece of paper with letters and cross some of them out
   b) Join together numbers and letters in a specific order using a pencil
   c) Look at detailed pictures and copy them from memory as well as with the picture in front of you
   d) Find your way through a paper maze by drawing with a pencil
   e) Make many different kinds of line patterns by connecting a series of dots
3. I will complete the above tasks using my left hand. Then I will do the same tasks with slightly different versions of the tests, of similar difficulty with my right hand.
4. I will complete these tasks in my home at no cost to me, with no risks or benefits.
5. The results will be submitted in a thesis as part of a bigger study on stroke patients and the information may be published in a journal. The information gathered may be used to compare the performance of the stroke patients to my performance on these tests.
6. My identity will be protected and my anonymity is assured.
7. I have the right to see the results of this study and they will be provided upon my request.
8. I have the right to withdraw from the study at any time and to decide not to complete the tests without penalty or negative consequence to me or the project.
9. I have read and fully understand the accompanying information sheet and agree to participate voluntarily in the study.
SIGNATURE PAGE

___________________________   __________________________
Participant name (Print)    Participant signature

__________________________
Date and place

__________________________
Researcher’s name (Print)    Researcher signature

__________________________
Date and place

__________________________   __________________________
Witness name (Print)     Witness signature

__________________________
Date and place

Tali contact details:

Home (011) 640 1989
Cel      072 492 0664
## APPENDIX J

### CONTROL MATCHED t-TESTS

#### Table J.1 – t-tests for Trails A

<table>
<thead>
<tr>
<th>Participant</th>
<th>Dominant</th>
<th>Non Dominant</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>45</td>
<td>39</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>86</td>
<td>36</td>
<td>50</td>
</tr>
<tr>
<td>3</td>
<td>46</td>
<td>37</td>
<td>9</td>
</tr>
<tr>
<td>4</td>
<td>30</td>
<td>30</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>31</td>
<td>43</td>
<td>-12</td>
</tr>
<tr>
<td>6</td>
<td>51</td>
<td>39</td>
<td>12</td>
</tr>
<tr>
<td>7</td>
<td>97</td>
<td>50</td>
<td>47</td>
</tr>
<tr>
<td>Mean</td>
<td>55.14</td>
<td>39.14</td>
<td>16.00</td>
</tr>
<tr>
<td>SD</td>
<td>26.21</td>
<td>6.20</td>
<td>23.53</td>
</tr>
<tr>
<td>N</td>
<td>7</td>
<td>7</td>
<td></td>
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</table>

\[ t = 1.80 \]

\[ p = 0.1221 \]

#### Table J.2 – t-tests for Trails B

<table>
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<tr>
<th>Participant</th>
<th>Dominant</th>
<th>Non Dominant</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
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<td>38</td>
<td>19</td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>3</td>
<td>48</td>
<td>42</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>44</td>
<td>44</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>44</td>
<td>42</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>68</td>
<td>49</td>
<td>19</td>
</tr>
<tr>
<td>7</td>
<td>133</td>
<td>76</td>
<td>57</td>
</tr>
<tr>
<td>Mean</td>
<td>64.86</td>
<td>47.29</td>
<td>17.57</td>
</tr>
<tr>
<td>SD</td>
<td>31.34</td>
<td>13.12</td>
<td>19.36</td>
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<tr>
<td>N</td>
<td>7</td>
<td>7</td>
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\[ t = 2.40 \]

\[ p = 0.0532 \]

#### Table J.3 – t-tests for complex figures (Copy)

<table>
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<th>Participant</th>
<th>Dominant</th>
<th>Non Dominant</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>36</td>
<td>36</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>36</td>
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<td>36</td>
<td>36</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>36</td>
<td>36</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>36</td>
<td>34.5</td>
<td>1.5</td>
</tr>
<tr>
<td>6</td>
<td>36</td>
<td>36</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>36</td>
<td>36</td>
<td>0</td>
</tr>
<tr>
<td>Mean</td>
<td>36.00</td>
<td>35.79</td>
<td>0.21</td>
</tr>
<tr>
<td>SD</td>
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<td>0.57</td>
</tr>
<tr>
<td>N</td>
<td>7</td>
<td>7</td>
<td>7</td>
</tr>
</tbody>
</table>

\[ t = 1.00 \]

\[ p = 0.3559 \]

#### Table J.4 – t-tests for complex figures (Recall)

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<tr>
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<th>Non Dominant</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>7</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>19</td>
<td>18</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>9.5</td>
<td>12</td>
<td>-2.5</td>
</tr>
<tr>
<td>4</td>
<td>18</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>5</td>
<td>17</td>
<td>16.5</td>
<td>0.5</td>
</tr>
<tr>
<td>6</td>
<td>12</td>
<td>10</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>0</td>
<td>9.5</td>
<td>-9.5</td>
</tr>
<tr>
<td>Mean</td>
<td>11.93</td>
<td>11.71</td>
<td>0.21</td>
</tr>
<tr>
<td>SD</td>
<td>6.78</td>
<td>4.08</td>
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<td>7</td>
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\[ t = 0.10 \]

\[ p = 0.9217 \]

#### Table J.5 – T-tests for five point test

<table>
<thead>
<tr>
<th>Participant</th>
<th>Dominant</th>
<th>Non Dominant</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12.5</td>
<td>7.5</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>2.5</td>
<td>2.5</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
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<td>0</td>
</tr>
<tr>
<td>4</td>
<td>32.5</td>
<td>7.5</td>
<td>25</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>3</td>
<td>5</td>
<td>-2</td>
</tr>
<tr>
<td>7</td>
<td>24</td>
<td>13</td>
<td>11</td>
</tr>
<tr>
<td>Mean</td>
<td>11.71</td>
<td>6.14</td>
<td>5.57</td>
</tr>
<tr>
<td>SD</td>
<td>12.24</td>
<td>4.18</td>
<td>9.64</td>
</tr>
<tr>
<td>N</td>
<td>7</td>
<td>7</td>
<td>7</td>
</tr>
</tbody>
</table>

\[ t = 1.53 \]

\[ p = 0.1772 \]

#### Table J.6 – T-tests for design fluency

<table>
<thead>
<tr>
<th>Participant</th>
<th>Dominant</th>
<th>Non Dominant</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>2</td>
</tr>
<tr>
<td>3</td>
<td>27</td>
<td>16</td>
<td>11</td>
</tr>
<tr>
<td>4</td>
<td>37</td>
<td>25</td>
<td>12</td>
</tr>
<tr>
<td>5</td>
<td>23</td>
<td>24</td>
<td>-1</td>
</tr>
<tr>
<td>6</td>
<td>22</td>
<td>27</td>
<td>-5</td>
</tr>
<tr>
<td>7</td>
<td>12</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>Mean</td>
<td>23.57</td>
<td>19.86</td>
<td>3.71</td>
</tr>
<tr>
<td>SD</td>
<td>7.55</td>
<td>5.70</td>
<td>6.26</td>
</tr>
<tr>
<td>N</td>
<td>7</td>
<td>7</td>
<td>7</td>
</tr>
</tbody>
</table>

\[ t = 1.57 \]

\[ p = 0.1677 \]
APPENDIX K

Conversation Analysis Transcription Conventions (Jefferson, 2004; Lesser & Milroy, 1993)

D: speaker identification
Word some form of stress, via pitch or amplitude
(laugh) transcriber’s descriptions, e.g. laughter or head nod
? rising intonation
. falling or terminal intonation
, continuing intonation
( ) short pause
(0.0) gaps in tenths of seconds
"word" softly spoken, quieter than the surrounding talk
CAPITALS Relatively loud speech
>< Relatively fast speech
[ ] onset and offset of overlapping talk
- cut-off
↑ marked shift into higher pitch in the utterance-part immediately following the arrow
*** omitted text, to protect participant’s confidentiality
== Latched utterance (no pause between turns)
:: Prolonged sound or syllable
APPENDIX L

Table L.1 – Quantitative and qualitative constructs of reliability

<table>
<thead>
<tr>
<th>QUANTITATIVE CONCEPT</th>
<th>QUALITATIVE CONCEPT</th>
<th>METHODS OF IMPLEMENTATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal validity – the degree to which findings correctly map phenomena in question</td>
<td>Credibility – the ability to show that the participants were accurately identified and described</td>
<td>• Provide an in-depth description showing the complexities and interactions of the variables, providing boundaries on the research (Marshall &amp; Rossman, 1995).</td>
</tr>
<tr>
<td>External validity – the degree to which findings can be generalized to other settings</td>
<td>Transferability – the ability to demonstrate the applicability of one set of findings to another context</td>
<td>• Engage in triangulation, a process of bringing together more than one source of data to add rigour and depth (Denzin &amp; Lincoln, 1994)</td>
</tr>
<tr>
<td>Reliability – the degree to which findings can be replicated by another inquirer</td>
<td>Dependability – the ability to account for changes in design or conditions in the phenomena under investigation</td>
<td>• Data triangulation – collect data over sufficient situations to account for variation to ensure adequacy (Janesick, 1994; Morse, 1994)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Data triangulation – use measures that are theoretically cohesive and sample purposefully to ensure appropriateness of data (Janesick, 1994)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Theory triangulation – use multiple perspectives to interpret the data (Janesick, 1994)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Methodological triangulation – use multiple methods to study the problem, including quantitative measures such as standardized results (Morse, 1994)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Interdisciplinary triangulation</td>
</tr>
<tr>
<td>Objectivity – the degree to which the findings are bias-free</td>
<td>Confirmability – the ability of the findings to be confirmed by another</td>
<td>• Collect and store an audit trail, to ensure that interpretations are open to scrutiny and falsification and that an inspection of procedures, protocols and decisions adopted can be made (Morse, 1994).</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Investigator triangulation - Submit work to an external researcher to confirm the findings and implications suggested (Marshall &amp; Rossman, 1995).</td>
</tr>
</tbody>
</table>
Appendix M

Dear Rater

Thank you so much for participating in this research. This instruction sheet will inform you about your role.

Ten participants took place in this research. All had strokes, eight presented with aphasia while two individuals presented with a right hemisphere lesion and were not aphasic. You are being requested to describe various aspects of their conversational competence by either: a) reviewing conversational transcripts, b) watching a DVD or c) reviewing the transcripts as well as watching the DVD. There is one interaction provided for each of the ten participants. Participants are numbered 1 – 10 corresponding to the order in which they appear on the DVD, or the number of the transcript provided.

You will then fill in the accompanying forms – one for each of the ten participants. We are interested in three conversational characteristics – turn taking, topic management and conversational repair. These three have been chosen because of the fact that they are generally present throughout most interactions, as well as the fact that there is a large corpus of data related to normal practice against which to compare this data. Please write down your impressions regarding how the ten participants manage each of these aspects, with respect to particular elements noted in the description sheet provided.

In addition, there is a section for your comments on nonverbal behaviours should you feel that there is something specific that should be noted.

Please feel free to contact me anytime with queries.

Best regards
Tali Frankel
011 640 1989 OR 072 492 0664 OR tdfranky@absamail.co.za
PARTICIPANT 1 – CS with his wife

<table>
<thead>
<tr>
<th>CONVERSATIONAL CHARACTERISTICS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Turn taking (promptness, interruptions, anticipates turns, length)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Topic management (ability to initiate, contribute to topic shift, sustain a topic, terminates or responds to topic termination)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Repair (spontaneous self-correction, self corrects when interlocutor indicates has not understood, initiates requests for clarification from interlocutor, repairs interlocutor’s utterance)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Nonverbal aspects (eye contact, use of gestures, facial expression, head nodding / shaking, fluency, rate of speech, intonation)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Final comment</td>
</tr>
</tbody>
</table>
### APPENDIX N

**EXECUTIVE FUNCTION RESULTS**

**RESULTS OF BEHAVIOURAL INHIBITION – INTERFERENCE CONTROL**

Table N.1 - Results of digits forward (Norm = 5 -7)

<table>
<thead>
<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>BASELINE</th>
<th>ACTIVE</th>
<th>PLACEBO</th>
<th>WITHDRAWAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>6</td>
<td>5</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>3</td>
<td>3</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
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<tr>
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<td>Felicity</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>8</td>
</tr>
</tbody>
</table>

Bold type indicates intact performance

Table N.2 - Results of the Stroop Colour-Word Interference Test (Norm = 2.28 – 2.55)

<table>
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<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>BASELINE</th>
<th>ACTIVE</th>
<th>PLACEBO</th>
<th>WITHDRAWAL</th>
</tr>
</thead>
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<tr>
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<tr>
<td></td>
<td>Mel</td>
<td>4.13</td>
<td>2.31</td>
<td>1.55</td>
<td>1.65</td>
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<td></td>
<td>Tumi</td>
<td>1.64</td>
<td>1.33</td>
<td>1.73</td>
<td>1.26</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>5.63</td>
<td>3.4</td>
<td>3.3</td>
<td>3.4</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>1.01</td>
<td>0.98</td>
<td>0.99</td>
<td>1.02</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>1.14</td>
<td>1.03</td>
<td>1.36</td>
<td>1.30</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>3.5</td>
<td>4</td>
<td>1.87</td>
<td>1.37</td>
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<tr>
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<td>Jeannette</td>
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<td>1.16</td>
<td>1.09</td>
<td>1.09</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>32</td>
<td>2.634</td>
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<tr>
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</table>

Bold type indicates intact performance
### RESULTS OF BEHAVIOURAL INHIBITION – RESPONSE INHIBITION

Table N.3 - Results of Trail Making (Norm = 2.2 - 2.3)

<table>
<thead>
<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>BASELINE</th>
<th>ACTIVE</th>
<th>PLACEBO</th>
<th>WITHDRAWAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
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<td>3.47</td>
<td>2.73</td>
<td>4.55</td>
<td>2.74</td>
</tr>
<tr>
<td></td>
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<td>3.66</td>
<td>4.69</td>
<td>2.84</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>1.26</td>
<td>3.86</td>
<td>5.48</td>
<td>2.35</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>2.74</td>
<td>2.18</td>
<td>1.36</td>
<td>2.22</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>1.47</td>
<td>1.57</td>
<td>0.69</td>
<td>1.28</td>
</tr>
<tr>
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<td>Grace</td>
<td>2.15</td>
<td>1.74</td>
<td>2.07</td>
<td>2.14</td>
</tr>
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Bold type indicates intact performance

### RESULTS OF WORKING MEMORY

Table N.4 - Results of Self Ordered Pointing Test (Norm = 4.68 (2.53))

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Bold type indicates intact performance
APPENDIX N cont…

Table N.5 - Results of complex figures copy task (Norm = 30.79 – 32.31)

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<td>John</td>
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Bold type indicates intact performance

Table N.6 - Results of complex figures recall task (Norm = 14.21 (6))

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Bold type indicates intact performance
APPENDIX N cont…

Table N.7 - Results of Wisconsin Card Sorting Test in terms of categories sorted (Norm = 4 - 6)

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<td>Tumi</td>
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<td>3</td>
<td>0</td>
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<td>John</td>
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<td>0</td>
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<td>0</td>
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<td>1</td>
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Bold type indicates intact performance

Table N.8 - Results of Wisconsin Card Sorting Test in terms of number of errors

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<td>&lt;2</td>
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<tr>
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<td>2</td>
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<td>John</td>
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<td>&lt;1</td>
<td>&lt;1</td>
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<td>&lt;1</td>
<td>&lt;1</td>
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Bold type indicates intact performance
### APPENDIX N

Table N.9 - Results of Wisconsin Card Sorting Test in terms of perseverative responses (Norm = Percentile rank ≥ 45)

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<tr>
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<td>Tumi</td>
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<td>2</td>
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<td>&lt;1</td>
<td>&lt;1</td>
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<td>&lt;1</td>
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Bold type indicates intact performance

Table N.10 - Results of digits backwards (Norm = 4-5)

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Bold type indicates intact performance
### RESULTS OF REGULATION OF AFFECT

Table N.11 - Results of regulation of affect

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<tr>
<td></td>
<td>Tumi</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>Jane*</td>
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Bold type indicates intact performance

### RESULTS OF INTERNALIZATION OF SPEECH

Table N.12 - Results of Tower of London (Norm = SS 85 – 115)

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<td>SS = 125</td>
<td>SS = 95</td>
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Bold type indicates intact performance
APPENDIX N cont…

Table N.13 - Results of Raven’s progressive matrices (Norm = Percentile rank ≥ 45)

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<tr>
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<td>Jane*</td>
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<td>Felicity</td>
<td>Below basal</td>
<td>5</td>
<td>6</td>
<td>6</td>
</tr>
</tbody>
</table>

Bold type indicates intact performance

RESULTS OF RECONSTITUTION

Table N.14 - Results of five point test (Norm = <15% perseveration)

<table>
<thead>
<tr>
<th>APHASIA TYPE</th>
<th>PARTICIPANT</th>
<th>BASELINE</th>
<th>ACTIVE</th>
<th>PLACEBO</th>
<th>WITHDRAWAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>20%</td>
<td>7.5%</td>
<td>19%</td>
<td>15%</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>47%</td>
<td>4%</td>
<td>13.5%</td>
<td>23.3%</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>5%</td>
<td>8.8%</td>
<td>7.5%</td>
<td>10%</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>22.5%</td>
<td>12.5%</td>
<td>18%</td>
<td>17.5%</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>17.5%</td>
<td>4%</td>
<td>13.8%</td>
<td>12.9%</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>0%</td>
<td>3.1%</td>
<td>3.3%</td>
<td>13.8%</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>25%</td>
<td>21%</td>
<td>22.5%</td>
<td>19.4%</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>15%</td>
<td>10.3%</td>
<td>7.8%</td>
<td>2.5%</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>30%</td>
<td>20%</td>
<td>17.5%</td>
<td>17.5%</td>
</tr>
<tr>
<td>None</td>
<td>Felicity</td>
<td>50%</td>
<td>17.5%</td>
<td>34.4%</td>
<td>42.5%</td>
</tr>
</tbody>
</table>

Bold type indicates intact performance
## APPENDIX N cont…

Table N.15 - Results of design fluency (Norm = 15.5)

<table>
<thead>
<tr>
<th>Aphasia Type</th>
<th>Participant</th>
<th>Baseline</th>
<th>Active</th>
<th>Placebo</th>
<th>Withdrawal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomic</td>
<td>Cecil</td>
<td>3</td>
<td>8</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Mel</td>
<td>4</td>
<td>10</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Tumi</td>
<td>15</td>
<td>4</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Broca’s</td>
<td>John</td>
<td>5</td>
<td>8</td>
<td>16</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>Paul*</td>
<td>8</td>
<td>10</td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td>Conduction</td>
<td>Grace</td>
<td>16</td>
<td>12</td>
<td>8</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Jane*</td>
<td>4</td>
<td>6</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Jeannette</td>
<td>3</td>
<td>12</td>
<td>12</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Margaret</td>
<td>4</td>
<td>6</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>None</td>
<td>Felicity</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>4</td>
</tr>
</tbody>
</table>

Bold type indicates intact performance
## APPENDIX O

### RESULTS OF ANOVA - REPEATED MEASURES ANALYSIS OF VARIANCE

#### ANOVA FOR BEHAVIORAL INHIBITION – INTERFERENCE CONTROL

**Table O.1 – ANOVA for digits forward**

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treat</td>
<td>3</td>
<td>1.47500000</td>
<td>0.49166667</td>
<td>2.12</td>
<td>0.1216</td>
<td>0.1479</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>6.27500000</td>
<td>0.23240741</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.5663  
Huynh-Feldt Epsilon 0.6844

**Table O.2 – ANOVA for Stroop word colour interference test**

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>treat</td>
<td>3</td>
<td>52.000000</td>
<td>17.333333</td>
<td>0.35</td>
<td>0.7888</td>
<td>0.7108</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>1333.500000</td>
<td>49.388889</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.6735  
Huynh-Feldt Epsilon 0.8695

#### ANOVA FOR BEHAVIORAL INHIBITION – RESPONSE INHIBITION

**Table O.3 – ANOVA for trail making**

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treat</td>
<td>3</td>
<td>9008.07500</td>
<td>3002.69167</td>
<td>0.84</td>
<td>0.4861</td>
<td>0.4861</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>97026.67500</td>
<td>3593.58056</td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.7841  
Huynh-Feldt Epsilon 1.0791
### APPENDIX O cont…

## ANOVA FOR WORKING MEMORY

### Table O.4 – ANOVA for the self ordered pointing test

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>treat</td>
<td>3</td>
<td>124.075000</td>
<td>41.358333</td>
<td>1.80</td>
<td>0.1714</td>
<td>0.1903</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>621.175000</td>
<td>23.006481</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.7235  
Huynh-Feldt Epsilon 0.9617

### Table O.5 – ANOVA for recall of complex figures

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>treat</td>
<td>3</td>
<td>59.818750</td>
<td>19.939583</td>
<td>1.17</td>
<td>0.3397</td>
<td>0.3335</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>460.368750</td>
<td>17.050694</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.6782  
Huynh-Feldt Epsilon 0.8779

### Table O.6 – ANOVA for WCST in terms of categories sorted

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>treat</td>
<td>3</td>
<td>4.100000</td>
<td>1.366667</td>
<td>1.04</td>
<td>0.3897</td>
<td>0.3746</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>35.400000</td>
<td>1.311111</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.6905  
Huynh-Feldt Epsilon 0.9004

### Table O.7 – ANOVA for WCST in terms of errors

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>treat</td>
<td>3</td>
<td>328.275000</td>
<td>109.425000</td>
<td>1.41</td>
<td>0.2610</td>
<td>0.2706</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>2092.97500</td>
<td>77.517593</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.5376  
Huynh-Feldt Epsilon 0.6375
APPENDIX O cont…

Table O.8 – ANOVA for WCST in terms of perseveration

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>treat</td>
<td>3</td>
<td>525.875000</td>
<td>175.291667</td>
<td>1.29</td>
<td>0.2972</td>
<td>0.2990</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>3662.375000</td>
<td>135.643519</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.5601
Huynh-Feldt Epsilon 0.6741

Table O.9 – ANOVA for Digits backwards

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>treat</td>
<td>3</td>
<td>0.8750000</td>
<td>0.29166667</td>
<td>1.24</td>
<td>0.3162</td>
<td>0.3142</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>6.3750000</td>
<td>0.23611111</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.6627
Huynh-Feldt Epsilon 0.8501

ANOVA FOR INTERNALIZATION OF SPEECH

Table O.10a – ANOVA for Tower of London

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>treat</td>
<td>3</td>
<td>1247.40000</td>
<td>415.800000</td>
<td>3.71</td>
<td>0.0236</td>
<td>0.0399</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>3029.10000</td>
<td>112.188889</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.7265
Huynh-Feldt Epsilon 0.9674

Table O.10b – Post Hoc analysis for source of variance Baseline vs. Phases, 1,2 and withdrawal

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1</td>
<td>980.100000</td>
<td>980.100000</td>
<td>6.10</td>
<td>0.0356</td>
</tr>
<tr>
<td>Error</td>
<td>9</td>
<td>1446.900000</td>
<td>160.766667</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## APPENDIX O cont…

Table O.11 – ANOVA for Raven’s progressive matrices

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>treat</td>
<td>3</td>
<td>58.7000000</td>
<td>19.5666667</td>
<td>0.78</td>
<td>0.5129</td>
<td>0.4749</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>673.3000000</td>
<td>24.9370370</td>
<td></td>
<td></td>
<td>0.5022</td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.6908
Huynh-Feldt Epsilon 0.9010

### ANOVA FOR RECONSTITUTION

Table O.12 – ANOVA for five point test

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>treat</td>
<td>3</td>
<td>110.462750</td>
<td>36.820917</td>
<td>0.48</td>
<td>0.6978</td>
<td>0.5850</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>2064.354750</td>
<td>76.457583</td>
<td></td>
<td></td>
<td>0.6140</td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.5296
Huynh-Feldt Epsilon 0.6247

Table O.13 – ANOVA for design fluency

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>Anova SS</th>
<th>Mean Square</th>
<th>F Value</th>
<th>Pr &gt; F</th>
<th>Adj Pr &gt; F</th>
</tr>
</thead>
<tbody>
<tr>
<td>treat</td>
<td>3</td>
<td>18.2000000</td>
<td>6.0666667</td>
<td>0.82</td>
<td>0.4943</td>
<td>0.4476</td>
</tr>
<tr>
<td>Error(treat)</td>
<td>27</td>
<td>199.8000000</td>
<td>7.4000000</td>
<td></td>
<td></td>
<td>0.4680</td>
</tr>
</tbody>
</table>

Greenhouse-Geisser Epsilon 0.6083
Huynh-Feldt Epsilon 0.7549