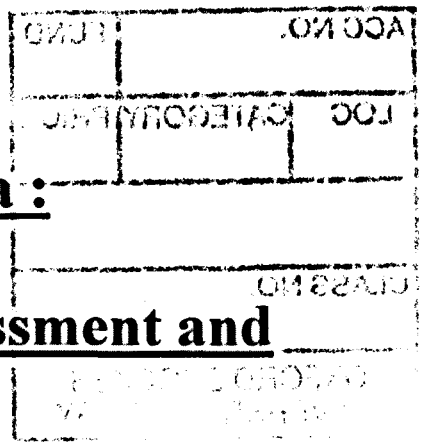

Apraxia :

Analysis of assessment and rehabilitation.

Jennifer Annette Butler

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i. Abstract

This project explored two main areas: the assessment of apraxia and the intervention effectiveness in rehabilitation of the apraxic condition. This was achieved through a group study and a series of single case designs. Three experimental groups were used to investigate clinical tests and the kinematics of movement; apraxic (n=17) and non-apraxic (n=13) left hemisphere damaged patients, and normal control subjects (n=11). Using computergraphic techniques, the data provided evidence of disruption to the temporal-spatial aspects of movement in apraxic people, which was not related to modality of testing, though some normal kinematic profiles were found within the apraxic group. Clinical assessments used to identify apraxia showed no relationship one with another which suggested each was identifying different aspects, or sub-types of a heterogeneous condition. Some tests were found to have low internal consistency, though inter-rater reliability through the observer-judgment process was high. A test devised for identifying agnosia was shown to relate to possible cognitive-perceptual processes or intact vision-to-action routes in the apraxic movement output. Dissociations found between clinical assessments for apraxia and kinematics of movement were explained in relation to different compensatory movement strategies employed by the apraxic patients, and/or as evidence for possible 'sub-types' of the apraxic condition. Analysis also suggested that different task demands might determine compensatory movement strategies and produce altered movement kinematics.

This group study was followed by a series of single cases, two of which charted the 'natural history' process in recovery of apraxia using task performance and kinematic analysis as outcome measures. Evidence for spontaneous recovery over a six week period was shown in one case. Four single case ABA design investigations were then carried out on individuals with ideomotor and ideational apraxia to determine the effectiveness of intervention strategies. Specific sensory stimulation protocols were evaluated with no convincing evidence for effectiveness of the intervention, though both natural recovery improvements and learning effects were seen in the outcome measures. Variability of performance was a feature of all cases studied and could be considered a feature of the apraxic condition. Task break-down strategies were also evaluated in functional activities and demonstrated some effectiveness in a case of ideomotor apraxia,

though a case with an ideational component indicated a more intractable condition. The strategy was not seen to generalise to other unpracticed tasks.

In conclusion, the associations and dissociations found between movement kinematics and the clinical assessment tests for apraxia suggested the presence of 'sub-types' within the blanket diagnosis of the condition. Identification of such sub-types might be facilitated by the development of the agnosia test newly devised for this project. Finally, research into intervention effectiveness in apraxia calls for further investigation to determine what procedures might be used with different sub-types of the condition.

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Chapter 1. INTRODUCTION

Apraxia is a condition usually resulting from left hemisphere brain damage which leaves the individual unable to perform normal functional activities. The impairment is generally considered to lie in the motor planning/motor control elements of the system rather than in any primary motor or sensory deficit. The main controversial areas in the field of applied apraxia research lie in debate about how to *define* the condition, how to identify it and measure its severity in any individual, how to chart progress in recovery and how to measure outcome of rehabilitation.

The definition of the term 'apraxia' is problematic, with the majority of literature defining the condition by exclusion. Historically apraxia has been defined by what it is *not*, rather than identifying positive characteristics of the signs or functional deficits The most frequent definition used in apraxia research is taken from Geschwind (1975, p. 188):

> "disorders of the execution of learned movement which cannot be accounted for either by weakness, incoordination, or sensory loss, or by incomprehension or inattention to command".

The difficulty in the use of exclusion criteria for the condition is that many patients have co-existing deficits. The clinician, therefore, has to make a judgement about what might, or might not, be attributable to those other deficits. This judgement relies upon individual experience and expertise. The nature of these 'expert' opinions has been called into question, and researchers have argued for more precise, standardised clinical assessments which might overcome some of these difficulties (Miller 1986, Tate & McDonald 1995). No *widely-used* standardised assessment has been developed for identifying the presence of apraxia. Some attempts have been made to produce such a tool (Fraser & Turton

1986), but the majority of assessments used and quoted widely in neuropsychological literature are non-standardised (DeRenzi et al 1980). The assessments fall into two main categories; gesture imitation and use-of-objects tests. They attempt to identify two main divisions of apraxia, ideomotor and ideational apraxia, and all rely upon observer judgement of movement performance using a variety of scales.

The use of computergraphic technology has been an avenue of research that attempted to identify elements of movement that might be characteristic or unique to apraxia. Research in motor control explored the kinematics of apraxic movement to try to elicit an understanding of normal motor processing, motor planning, action control and in order, then, to make judgments about the disruption to these aspects of movement that occur in apraxia (Poizner et al 1990, Hermsdorfer et al 1996). Ultimately such investigations could provide reliable, objective, quantitative measures that might assist in the identification of the condition. The technology might become a method of assessment in itself. The study recorded in this thesis aimed to verify the limited evidence, to date, of the temperal-spatial disruption of movement in apraxic people. It also aimed to compare the movements of normal healthy individuals with those of brain-damaged individuals with and without apraxia. If unique elements could be identified, then the use of this technology in the assessment and monitoring of recovery in apraxic patients would clearly be a valuable asset in rehabilitation, and an advance on the observer judgments currently used.

The importance of assessment and diagnosis of the apraxic condition is clear. For each individual, the identification of the elements of the apraxic condition leads to a statement of the aims of rehabilitation which, in turn, leads to the use of appropriate intervention

strategies. Objective and reliable assessment tools allow for accurate assessment of progress in rehabilitation and an evaluation of the efficacy of the interventions being used. Tools which can monitor and measure elements of the apraxic condition and which can be used as evaluative outcome measures are a critical part of rehabilitation research.

The course of recovery in apraxia is poorly documented (Basso et al 1987). Monitoring recovery, using a range of accurate, reproducible and sensitive outcome measures would provide useful baseline evidence from which to make judgements concerning intervention research. A resource of information concerning the changes in movement performance in apraxia over time could then be used to compare recovery profiles using experimental interventions. This study, therefore, aimed to record longitudinal data using single cases.

Rehabilitation literature concerning research into the efficacy of interventions in apraxia amounts to less than five papers throughout the world (Wilson 1988, Pilgrim and Humphreys 1994, Riddock et al 1995, Maher & Ochipa 1997). Many texts are available which *describe* approaches to rehabilitation or interventions in this area, but none cite evidence for *efficacy* of such treatments (Siev et al 1986, Miller 1986, Rosenthal et al 1990). In the current climate of evidence-based health care it is clearly critical to evaluate practice and determine which interventions have an effect and which do not. Also if it can be established which ***type*** of apraxia most benefits from which interventions this, too, could influence practice. This study aimed, therefore, to evaluate a sensory stimulation protocol which had been suggested by clinicians in paediatrics as being worthy of investigation for the adult apraxic patient (Ayres 1985, Croce 1993). A series of single case investigations were considered to be the most appropriate methodology for this aspect of the research using a variety of outcome measures including computergraphic

analysis of movement. In addition, a limited research base had been established (Wilson 1988) concerning the use of task break-down as a strategy for rehabilitation in apraxia. This strategy was also evaluated during the course of the research project using single case methodology.

The research participants for these studies were recruited at a rehabilitation centre which caters mainly for people with profound and multiple disabilities. They did not, therefore, represent those with mild or transient brain damage and all were at least three months post-incident. Patients attending the rehabilitation centre had generally completed their acute medical treatment and had often received some initial rehabilitation prior to arrival.

In summary, this study set out to review assessment tools for identifying apraxia and to evaluate the use of computergraphic technology as an assessment and monitoring tool. The project also intended to collect data that monitored recovery of apraxia and to evaluate efficacy of intervention in the rehabilitation process. In particular, sensory stimulation protocols and task break-down strategies were investigated.

Chapter 2. REVIEW OF THE LITERATURE

2.1 Introduction

In order to explore the key themes of assessment and intervention in apraxia, this literature review first highlights the definitions of the terms commonly used in apraxia research and considers the controversies surrounding classification of *types* of apraxia. The identification of errors made by people with apraxia is examined with evidence for differences elicited according to modality stimuli. The conflicting evidence relating apraxia to lesion site is also presented. Research literature concerning clinical assessment batteries is explored, as is the potential use of computergraphic technology in the identification of movement profiles in apraxic patients. It is important to clarify the characteristics of apraxia and to identify the best methods of assessment for the condition. These actions have the prime purpose of enabling swift and accurate conclusions to be drawn regarding the presence and degree of apraxia in the individual and targeting appropriate interventions to improve functional performance. The review also examines the limited literature concerning the rehabilitation needs of people with apraxia together with the sparse research evidence concerning outcomes of intervention.

2.2 Epidemiology

The World Health Organisation (WHO) 1997 report stated that over 4.6 million deaths per year are accounted for by cerebrovascular disease and stroke, mainly in the over-65 age group. This equates, in England and Wales, to an incidence of 100,000 people per

year experiencing a first stroke (or cerebrovascular accident, CVA) of which 10,000 are under retirement age (Stroke Association 1996). At any one time there are reported to be 350,000 people in England and Wales with a stroke (Stroke Association 1996). The 'Health of the Nation' document (Department of Health 1992) reported stroke as being responsible for 12% of all deaths in 1991, and the target set for the health service aims at reducing the deaths from stroke in people aged 65-74 from 265 per 100,000 population to no more than 159 per 100,000. In America, 600,000 people per year are reported experiencing a stroke (American Stroke Association 1998) although this figure is suggested to be conservative.

Apraxia is generally accepted as being associated with left hemisphere brain damage (Geschwind 1975, De Renzi et al 1980, Kertesz & Ferro 1984, Faglioni & Basso 1985, Basso et al 1987, Alexander et al 1992, Heilman & Rothi 1993, Tate & McDonald 1995) with research mainly carried out with those having a unilateral cerebrovascular accident. Whilst the *incidence* of apraxia in left-hemisphere brain damaged patients is considered to be high (Maher & Ochipa 1997) few published statistics are available. Rothi, Raymer and Heilman (1997) reported the incidence of apraxia in left hemisphere damaged subjects of Leipmann's (1905) studies as high (20 of the 41 patients), whilst the Kertesz and Ferro (1984) exploration of lesion size and location reported 80 apraxic and 68 non-apraxic patients in the 'acute phase' group (less than one month after a left hemisphere cebrebrovascular accident), and 57 apraxic and 86 non-apraxic patients in the chronic (three months post-stroke) group. Indeed, Poeck (1986) suggested that apraxia was a *frequent* syndrome occurring in 80% of patients with a cerebrovascular accident (CVA)

sustained in the Middle Cerebral artery of the hemisphere dominant for language, though it is unclear whether such incidence refers to the acute phase following stroke, or longer term. Basso et al (1987) considered that patients recovered spontaneously from apraxia over time with only 13 of 26 patients still showing evidence of apraxia five months post-stroke, and only five of those patients still apraxic on a third examination at an unspecified later date.

What might be concluded from the limited published data is that, potentially, about half the patients with left hemisphere damage following a CVA may show apraxic signs in the acute phase following stroke. The incidence data would also suggest that all patients with left hemisphere damage who require rehabilitation should be tested and screened for the presence of apraxia to determine whether this is a factor contributing to their functional deficits. In addition, if praxis ability does recover spontaneously then any intervention study must look carefully at what might be considered such a spontaneous recovery from apraxia and what might be considered improvement attributable to the intervention itself.

2.3 Definitions, taxonomy and classification of apraxia.

The term 'apraxia' is used throughout this thesis in keeping with the neuropsychological literature relating to the topic and to distinguish the condition from 'dyspraxia' which is more appropriately used in the paediatric field in relation to developmental dyspraxia in children. 'Apraxia' is used extensively in the neuropsychological research literature relating to the adult condition which follows brain damage. This is despite *'a'* meaning

the *absence of* or *lack*, and *'dys'* meaning *difficult, abnormal, or faulty* and generally implying a less severe impairment. Unfortunately most rehabilitation texts refer to the adult condition in question as *dyspraxia*, though this is changing, and consequently some confusion exists in the clinical rehabilitation field over this nomenclature.

Definitions

The condition of apraxia is generally described by exclusion; by saying what it is *not*. Originally this was not the case, as reported by Faglioni and Basso (1985) in an historical perspective of the original work by Leipmann (1900) who first described and named the condition. This early definition stated that apraxia was a deficit affecting the purposeful organisation of voluntary (learned) actions, elicited by assessing gesture copying ability. Pramstaller and Marsden (1996) interpreted Liepmann's definition of apraxia as being an interruption of the continuous interaction between kinetic memory and the transcoding of those schemas into adequate innervation patterns. This original position by Leipmann postulated that apraxia was only apparent on focused testing of gestures and not disruptive to everyday life. This view is not quite so widely accepted today (De Renzi & Lucchelli, 1988, Cicerone & Tupper, 1991, Foundas et al., 1995).

Both Poeck (1985) and Freund and Hummelsheim (1985), within their definitions of apraxia, had the notion of *higher order* motor disturbance or interruption. More simply De Renzi (1985) suggested a definition of apraxia as an inability to select the correct motor programme, or as a condition where the patient is unable to deliberately make a choice amongst the repertoire of his motor patterns.

The most frequent definition used in apraxia research is, however, taken from Geschwind (1975, p. 188) as :

> "disorders of the execution of learned movement which cannot be accounted for either by weakness, incoordination, or sensory loss, or by incomprehension or inattention to command".

As Tate and McDonald (1995) pointed out, this definition does not help in identification of the disorder when so many patients are likely to have coexisting motor, sensory and language deficits. Extrapolating the degree to which such impairments contribute to the observed motor performance during testing is a difficult clinical task, heavily reliant upon the expert judgment and personal experience of the clinician (Miller 1986, Poeck 1986, Kirshner 1991). Indeed the role of the 'expert' lies exactly in being able to judge what can be attributable to primary or other deficits, and what elements of performance cannot be otherwise ascribed and might therefore be considered apraxic in origin.

Other definitions offered by researchers have reiterated the same exclusion description; that adult-onset apraxia is an impairment of the ability to perform skilled, learned or purposeful movement because of acquired brain disease or injury but ***not*** due to any primary motor or sensory deficit, nor attributable to lack of comprehension, attention or willingness to perform the movement (Kirshner 1991, Heilman & Rothi 1993, Croce 1993, Tate & McDonald 1995). So the evidence shows what it is not, and researchers currently are working to say what it is by attempting to define the condition by performance deficits and/or lesion correlations.

The three major approaches to the consideration of the apraxia phenomenon might each be considered inadequate in some way. To consider apraxia just in relation to observed

behaviour, for instance, at the disability level does not sufficiently discriminate what is observed due to apraxia and what is observed due to a variety of motor or sensory impairments, nor does it attempt to explore the rationale and underlying mechanisms accounting for the disrupted performance. Yet the accepted definitions of apraxia focus on that observable behaviour, as this is what is accessible to researchers and clinicians alike. This particular element of the apraxia debate has been refined by researchers attempting to describe, in precise terms, the error attributes which are particular to apraxia and not any other movement disorder (see section 2.4). Such descriptions have focused both on behaviour observed during formal testing (Roy 1983, Haaland & Flaherty 1984, Raade et al 1991, Mozaz 1992) and during naturalistic functional performance (Mayer et al 1990, Schwartz et al 1991).

A consideration of the underlying impairment is considered by many workers in the field to be the way forward. Attempts to explore the theoretical construct which might explain and illuminate the apraxic condition have focused on the cognitive components required of the apraxia testing conditions which might explain the variety of manifestations of behaviour from patients given the blanket diagnosis of 'apraxia'. These different cognitive components of movement performance have been explored through consideration of the different modalities of testing (see section 2.5). De Renzi et al (1982) concluded from their studies of use-of-objects tests that apraxia could be modality specific and thus indicative of a heterogeneous condition. Roy and Hall (1992) supported this approach and argued that different modalities of testing (verbal, visual and real-object) elicited different movement performances and could be considered indicative of different apraxia *types*. Other researchers, though, have provided evidence that types of

errors made by people with apraxia remained the same *regardless* of cues (McDonald et al 1994). Other research groups have attempted to explore the underlying construct of apraxia by looking at the kinematics of visually guided movements (Fisk & Goodale 1988, Goodale et al 1990, Poizner et al 1990, Clark et al 1994, Hermsdorfer et al 1996) - see section 2.8. The quantitative data on the temporal-spatial aspects of apraxic movement has been used to explain the elements of movement performance which are disrupted in the condition though very limited research, to date, has considered the different kinematics according to modality conditions. Thus the merging of ideas concerning the different cognitive impairments within the apraxic condition which result in heterogeneity of apraxic performance, with the rich kinematic data of movement performance in those apraxic 'types' has not yet been established. A suggestion (Hermsdorfer et al 1996) that the strategies adopted by patents to compensate for their movement deficit, and the success or otherwise of those strategies, could go some way to linking and explaining the interaction between the kinematic profiles, the clinical errors and the modality-specific manifestations of apraxia. A third approach to understanding apraxia has linked the condition to damage in anatomical regions (see section 2.6). Whilst the left (dominant) hemisphere is confirmed as being associated with apraxia, the relationship to lesion location within that hemisphere is highly controversial with many cited cases offering differing views (Table 2.1).

This difficulty of definition and approach to the study of apraxia is not an unusual one in neuropsychology. For example, in the studies of hemineglect a special issue of the journal *Neuropsychological Rehabilitation* (1994) published 25 papers from eminent researchers in the field, all offering different views, different opinions, and different

perspectives on the various aspects of spatial neglect. Just as in the area of apraxia, Berti and Rizzolatti (1994) considered whether symptoms that are usually classified as neglect phenomena could be explained by a *single* theoretical model. They put forward a view that could provide such a model, but others offer different models and views. Milner and Harvey (1994) discussed the "bewildering heterogeneity" (pp. 177) manifest in neglect and how a taxonomy of the condition might be forthcoming through 'symptoms' (*sic*) identification. Marshall and Halligan (1994) stated that the manifestations of neglect were so well known and so well-established that they had the name 'neglect'. But the authors argued that this naming was not an explanation of the disorder. In *apraxia* however, even the manifestations are not well-established and are still subject to differing views so it is unsurprising that no single explanation, nor any single theoretical construct, is yet forthcoming. In addition the attempt to define apraxia in relation to the differing 'types' and errors of performance has followed the approach taken in neglect studies, yet this clearly is not necessarily the most productive way forward. Apraxia research is in the stage of development where neglect research was about ten years ago, so when the phenomena of neglect remains full of controversy regarding the nature of the condition it is unsurprising that apraxia researchers are also struggling to find their way and that little consensus is apparent.

Classifications of apraxia

The naming of apraxia would appear to be particularly problematic within the research and clinical literature but there seems to be a broad acceptance of two major types of the condition: **ideational** apraxia and **ideomotor** apraxia. However many other names are

used, particularly in clinical tests, when describing different presentations of the condition. Indeed Kirshner (1991) and Tate and McDonald (1995) both highlighted the anomaly of some conditions being labeled as a type of apraxia, yet not arising out of dysfunctions pertinent to apraxia as originally described and defined. This is particularly problematic in rehabilitation texts where *constructional apraxia* and *dressing apraxia* are described with the apraxias and yet would probably be more correctly considered to be dysfunctions of visuo-spatial origin which manifest in particular performance deficits. They are mostly associated with right hemisphere lesions (Neistadt 1989, Morera-Fumero & Rodreguez1990), though Sunderland et al(1994) highlighted evidence relating 'constructional apraxia' also to left hemisphere lesions as a function of time since onset of the brain damage. These two 'apraxias' would appear to be descriptive terms of disordered behaviours seen during drawing tasks or dressing activities, and are operationally defined in terms of those observed behaviours. Rothi and Heilman (1997) suggested that the term apraxia had been used non-discriminately (in the cases of constructional and dressing apraxias) with disorders of action where sensory-perceptual dysfunction has not been ruled out. Thus in 'constructional apraxia' Rothi and Heilman suggested that the observed behaviour cannot be entirely explained by the movement aspect of the deficit. Supporting this argument further, Kirshner (1991) suggested that the underlying deficit with these two misnamed conditions involved disordered body image and visuospatial and topographical reasoning. Benton and Tranel (1993) suggested a more appropriate term might be 'visuoconstructive disability'. Poeck (1986) also argued that 'dressing apraxia' should not be considered within the framework of

apraxia as it was considered to be just one of the many consequences of spatial disorgansation and "in most cases also of left-sided neglect" (pp 129).

Taking into account the differing opinions and evidence from published research, it is the authors opinion that ideomotor apraxia is a heterogeneous condition which involves, in underlying impairment, a dysfunction primarily in the motor action elements of performance interacting with different cognitive requirements for particular motor activities. The consequences of these dysfunctions are manifest in temporal-spatial elements of movement. The constructional and dressing 'apraxias' are considered not to have the motor action element as the *primary* impairment component of their disorder, but rather a visuo-spatial and cognitive deficit which shows itself in certain operational performances. It would be less confusing if they did not have the label of 'apraxia' attached to them. The association of constructional and dressing 'apraxias' with right hemisphere lesions would also support the case for suggesting that these conditions are separate from the ideomotor apraxia condition. A recent addition to the misnomer list is that of 'diagonistic apraxia' (Tanaka et al. 1996), used to describe alien hand syndrome.

Whilst the majority of authors have agreed to a 'motor' and an ideational apraxia dichotomy, within the 'motor' division there may be two types identified; limb-kinetic apraxia and ideomotor apraxia. The former is said to involve clumsiness, awkwardness, loss of speed and loss of smoothness of movement. This, again, might not be considered to fulfill the accepted definition of apraxia as similarities between the general description of performance deficits in 'limb-kinetic apraxia' and those seen in recovering paresis, Parkinson's disease and cerebellar damage are obvious. In recent years this earlier confusion has been largely resolved with nomenclature refined to an

ideational/ideomotor dichotomy with these names being embedded within the literature as forming the two main branches or types of the condition. This dual classification of the apraxic condition is the one accepted for this thesis, with acceptance of potential 'sub-types' of the condition which might emerge through exploration of motor performance under different conditions and task requirements.

Ideational apraxia

Ideational apraxia is generally defined in two ways. Faglioni and Basso (1985) and Pramstaller and Marsden (1996) both interpreted Liepmann's original definition of this type of apraxia as involving an inability to carry out sequential motor activity. The person with ideational apraxia cannot perform a *sequence* of movements, the elements of which when taken individually and in isolation can be performed. Other researchers, however, have preferred an alternative definition, referring to an inability to use real objects in both a simple and sequential manner (De Renzi 1985, Barbieri & DeRenzi 1988, Ochipa et al 1989, Kirshner 1991) although De Renzi and Lucchelli (1988) suggested that a more accurate description might be an 'amnesia of usage' of objects. Poeck (1983) defined ideational apraxia as a disturbance, or loss of, the conceptual organisation of movements; a loss of 'idea' of movement sequences. In practice, despite these different definitions of ideational apraxia, the clinician must try and differentiate and diagnose the condition and Tate and McDonald (1995) suggested that there is common agreement that an inability to use multiple objects in a sequence is characteristic of this disorder. Clinical assessments reflect this general concept for identification of ideational apraxia.

Ideomotor apraxia

In contrast to ideational apraxia, Liepmann's original view (as cited by Faglioni & Basso, 1985) suggested that the person *does* know what he has to do; does have the *idea* of the action requested but is unable to call upon the innervation needed to execute the action whether on verbal command or imitation, and when errors occur the patient is aware of the failures and tries to correct them. In addition, Leipmann's view (as postulated by Faglionni & Basso 1985) was that ideomotor apraxia was not disruptive of everyday life and only apparent on special testing. Tate and McDonald (1995) stated the classical view; that ideomotor apraxia is elicited through requests for movements with conscious processing being disrupted (i.e. it affects the intentionality of movement), but with a preservation of actions when automaticity is involved within a contextual framework. This view is being challenged now by increasing evidence from rehabilitation studies (Mayer et al 1990, Cicerone & Tupper 1991, Schwartz et al 1993, Foundas et al. 1995) where movement and actions in daily activities were seen as clumsy, awkward and disruptive to successful completion of tasks.

More precise definitions of ideomotor apraxia have arisen from descriptions of errors seen in the condition. Heilman and Rothi (1993) suggested that people with ideomotor apraxia have difficulty with the selection, sequencing and spatial orientation of movement. Such classification of error types seen in ideomotor apraxia has become very popular in recent years in research groups attempting to establish the unique attributes of apraxia (Roy 1983, Haaland & Flaherty 1984, Fraser & Turton 1986, Raade et al 1991, Schwartz et al 1991, Harrington & Haaland 1992, Mozaz 1992). Whilst these descriptors of errors have become useful in aiding diagnosis of the condition, it remains

unclear whether such errors are unique to, and diagnostic of, ideomotor apraxia or equally applicable to ideational apraxia and other movement disorders.

Other classifications of apraxia seen in the literature include those relating to the body part that the condition affects; i.e. buccofacial (or oral) apraxia, limb apraxia and axial apraxia. It might be considered that this is a less useful taxonomy than the classification by *type* of apraxia (ideational and ideomotor) as it merely identifies the body part affected and not the difficulties experienced by the patient.

Some researchers have argued a case for classification according to area of lesions (e.g. callosal apraxia, supramarginal apraxia). Many reports of apraxia with deep subcortical lesions and/or basal ganglia lesions have appeared in the literature (Agostoni et al 1983, DeRenzi et al 1986, Watson et al 1986, Mozaz et al 1990, Leiguarda et al 1994, Nadeau et al 1994, Classen et al 1995, Pramstaller & Marsden 1996). Whilst reports of such localised subcortical as well as cortical lesions are now well documented and accepted as being associated with apraxia, Tate and McDonald (1995) pointed out that damage to such areas of the brain did not automatically or necessarily lead to apraxia. It might be reasonable to conclude, therefore, that a 'lesion-area' classification system is not particularly useful for the clinician.

For the purpose of this particular study, a working definition of apraxia was adopted such that it was considered to be an observable phenomenon of movement disruption following left hemisphere damage. The movement disorder was considered to be observable through testing procedures using previously published guidelines, and the heterogeneous nature of the condition being evident with errors displayed according to different modality conditions (verbal, visual, real-object). Such errors were judged

according to the Raade et al (1991) classification in relation to the content, the temporal and the spatial components of the requested movement performance and discounting all the other elements that might influence disruption of movement (e.g. paralysis, sensory deficit, not understanding instructions, inattention). The attribution of the diagnosis 'apraxia' to any individual was made using *combined* scores from three tests of ideomotor apraxia (De Renzi et al 1980, Haaland & Flaherty 1984, Alexander et al 1992 - see appendices D1-D3). The apraxic group might be considered, therefore, rather inclusive when compared to other published studies in that some patients may have scored nearly fully on one of the tests (and would, therefore, have been judged as non-apraxic in those studies which only used one apraxia assessment) and yet scored poorly on another test and were thus included in the apraxic group. The apraxic group in this study therefore included some patients with a mild apraxia, as well as those with discernible differences and dissociations between the three ideomotor test scores, suggesting that these were measuring different components of the apraxic condition. The major element for attribution into the apraxic or non-apraxic groups was performance on the De Renzi (1980) gesture imitation test with a cut-off score of less than 68, but with the other tests scores taken in consideration.

2.4 Observed behaviour in apraxia

In the attempt to establish the unique attributes of apraxia, part of the definition of apraxia has involved the kinds of errors made by the person with the dysfunction during testing. Assessment and diagnosis of apraxia involves scoring or describing those movement errors which correspond to the accepted range of apraxic errors recognised in

the literature. The adjudged errors may be observed both during formal assessment and during functional activities in a meaningful (naturalistic) context. Both situations, Arnadottir (1990) suggested, were a critical part of obtaining a diagnosis and of giving the clinician a full appreciation of functional consequences for the patient.

The descriptions of errors have become more focused in recent years. Roy (1983) described apraxic errors occurring in both sequences of action or in single gestures, and included omissions, difficulty terminating movements, repetitions, disturbances to the order of movements in a sequence, coordinating limbs in time and space and perseveration of movement. Roy (1983) continued this list with details of spatial misalignment of movements in apraxia where performance might be in the wrong plane, or the patient might use a body part *as* the object in question (BPO), or might exhibit a verbalisation of the performance without actually completing the action itself. Haaland and Flaherty (1984) noted that types of errors in apraxic movement included hand position inaccuracies, wrong orientation of hand or arm, gestures not being made on the correct body part, clumsiness, delayed responses, and using the body part as the object. An attempt to categorise errors was made by Raade et al (1991) to help with scoring the quality of performance in what was called buccofacial and limb apraxia (Table 2.1). This categorisation of errors has been adopted as a useful tool in the clinical examination of apraxia as it described specific elements of performance in different areas of the movement component. The need for 'expert judgment' to interpret such errors, however, was apparent.

Table 2.1 Error type categories in ideomotor apraxia (Raade et al, 1991).

Error type	Description of errors
a] Content	
Perseveration	A response that includes all or part of a previously produced movement.
Nonrelated	Movement accurately produced but not associated in content to the target.
b] Temporal	
Sequencing	Alteration in characteristic sequence of movement including addition, deletion, and transposition of movement elements.
Timing	Alteration in timing or speed of movement including increased, decreased or irregular rate of production.
Occurrence	Any multiplication of single cycles of movement, or the reduction of a repetitive cycle of movement to a single event.
Delay	Delay in initiating movement.
c] Spatial	
Internal configuration	Abnormality of required finger/hand posture to reflect recognition of imagined tool.
Amplitude	Any amplification, reduction or irregularity of the normal amplitude of the requested movement.
Orientation	Difficulty orienting the fingers/hand/arm to the imagined 'object'.
Movement	Disturbance of the normal characteristic movement required.
Extraneous	Additional or extra movement(s) produced by non-targeted joints or body parts.
Body part as object	Using a body part as the object itself (e.g. using the fist as a hammer, using fingers as a comb, using finger as a toothbrush).
d] Other	
No response	Verbal comment such as "I can't" or "No".
Unrecognizable response	Movement response that is not recognizable and shares no temporal or spatial features of the required movement.
Verbalization	Production of a verbal response or written output instead of the movement response requested.

Duffy and Duffy (1989) had identified that a use of a body part as an object (BPO errors) could ***not*** be considered indicative of left-hemisphere pathology and apraxia, as its presence was elicited in normal adults. This finding was supported by Mozaz et al (1993) and McDonald et al (1994) who found no difference in incidence of BPO errors in normal

controls and left brain damaged individuals. Mozaz (1992) reviewed the types of responses and response errors reported in the literature in order to determine whether some errors were indicative of, and could be used to distinguish between, different types of apraxia. No quantitaive data was presented but rather a qualitative analysis of descriptions of errors which led the author to conclude that whilst certain errors showing an executive deficit might be associated with ideomotor apraxia, and other errors showing an ideational component which could be indicative of ideational apraxia, such errors were as much a function of the testing procedures as they were indicative of the condition being assessed. Mozaz (1992) itemised ideomotor errors to include amplitude of movement, clumsiness, misuse of objects, deviations from the normal position of hand or arm (spatial orientation), mislocation of actions, movement involving the wrong joint in performance, or delay in initiation of movement.

Ideational apraxic errors were identified as unrecognisable performance, verbalisation instead of performance, body part as the object, and no recognition of errors made. Mozaz (1992) suggested that all responses and errors must be related to modalities used during testing procedures, types of gesture used (meaningless or meaningful) and whether errors occurred during simple or complex movements. Tate and McDonald (1995) concluded that the bewildering array of qualitative features of apraxic errors given in the literature confused more than clarified the position for the clinician, and suggested that as the clinical reliability of such classifications remained untested they must be used with caution.

Information about reliability of diagnosis using error categorisation during testing for apraxia is sparse. De Renzi et al (1980) in describing the gesture-copying assessment

tool for apraxia suggested a cut-off point of 62 (of a maximum score of 72) to apply a diagnosis of apraxia but made no reference to inter-rater reliability of judging patient performance on the test nor, indeed, whether more than one judge was used. Poeck (1986) stated that diagnosis of apraxia was generally made on the basis of personal experience and intuition, and expressed concern at the lack of standardised battery of tasks for apraxia assessment. An additional cause for concern according to Poeck (1986) was the need for acceptance of a range of performance scores rather than a cut-off point in order to account for test-retest variability. McDonald et al (1994) reported a high inter-rater reliability (89%) in judging error types in apraxic patients using four raters in consensus pairs looking at videotaped gesture production. Rothi et al (1997) also described the process of videotaping all gestural productions made by patients during testing and a then minimum of two judges scoring those videotapes. No reliability data was presented, but rather the process being one of discussion and consensus between the judges.

In the clinical situation, rather than a research project, a diagnosis is usually made by a sole clinician in real-time observation of errors during the assessment procedure. Rarely is a videotape used for later analysis and scoring. This does leave the clinician open to criticism in terms of reliability of diagnosis, and reliability of scoring within the individual tests. Corroborating evidence from observed functional performance by the patient, as well as descriptions of performance deficits by the patient himself, from other staff and from family carers can be used to support the clinician's diagnosis but the concern remains about the process of assessing for apraxia using non-standardised tests based upon subjective judgment of performance.

Rehabilitation research looking at *functional* deficits and errors in performance are sparse. Mayer et al (1990) argued for assessment to move beyond the constraints of formal examination for apraxia, and on to observations in the context of naturally occurring actions in order to have "ecological validity" (pp 265). Their paper described the development of a complex action coding scheme which enabled reliable recording and analysis of quantitative measures of performance during everyday activities. The authors pointed out that day-to-day variability in performance was the rule rather than the exception. This coding scheme has been further developed and described by Schwartz et al (1991). Whilst this has enabled case study data concerning functional errors to be recorded accurately and systematically over time, it is considered unlikely to be of practical use to clinicians due to the sheer complexity of the coding and scoring system.

The attempts to provide descriptions of apraxic errors from pertinent literature sources has made it clear that people with apraxia are not a homogeneous group with respect to either error types or to the testing procedures which elicit those errors. What is needed in this research area is a definitive tool for the assessment of apraxia which is simple but comprehensive and which will quantify movement errors and aid identification of possible intervention strategies.

2.5 Impairment in apraxia

As well as considering the observable behaviour and motor performance in apraxia, in order to understand the underlying deficit and impairment of apraxia an examination of modality differences during testing procedures has been carried out by many research teams. This exploration has been a means of extrapolating the cognitive differences of

movement performance requirements which could illuminate the different manifestations of the apraxic condition. There is considerable debate in the research literature concerning such modality effects of testing for apraxia. Roy and Hall (1992) suggested that people with ideomotor apraxia were often impaired in both verbal command and imitation during testing procedures, but frequently demonstrated a better performance using the real object. Whilst there is general agreement that any tests for apraxia should involve these three (verbal, visual and real-object) conditions there is conflicting evidence, as Tate and McDonald (1995) stated, concerning the type of movements and gestures that should comprise the assessment tasks. In addition, different research groups have used different names to describe such movements (e.g. meaningful vs. non-meaningful gestures, transitive vs. non-transitive movements, representational vs. non-representational movements, or symbolic vs. non-symbolic movements).

De Renzi and Lucchelli (1988) found a relationship between the inability to pantomime gestures in response to verbal command and an incapacity to imitate gestures (visual representation). Many authors have reported that imitation of *meaningful* gestures is easier for the apraxic patient than pantomime following verbal command (Poeck et al 1980, De Renzi et al 1980, Alexander et al 1992). Alexander at al (1992) suggested that imitation provided a model of movement for the patient and therefore facilitated performance. This notion is reflected in testing procedures in many research reports, with the order of modality being kept constant so that imitation is used *after* the person has failed to pantomime correctly to verbal command. Alexander et al (1992) also reported that imitation performance was better than verbal command pantomime *whether or not* the patients had poor auditory comprehension. This led the authors to support

those researchers who argued the case for imitation-only tests for eliciting apraxic errors. In addition they asserted that modality-specific effects would be seen in test results and therefore did not support those studies using modalities interchangeably or cumulatively in testing for apraxia. This was, to some extent, supported by McDonald et al (1994) who claimed that whilst performance improved in relative terms with imitation and real object cues, the *types* of errors made by people with apraxia remained the same regardless of the cues (modalities) provided.

De Renzi et al (1980) had argued the case for imitation of meaningless (non-symbolic) gestures being more challenging to the motor system as they have not been practiced and over-learned, and therefore would elicit apraxic errors more reliably than well-known, meaningful gestures. Such reasoning, it should be pionted out, flies in the face of the definition for apraxia which expresses notions of disruption to 'learned' and 'purposeful' movements, neither of which resonate with the meaningless gestures which the patient is requested to perform. Nevertheless the development of an imitation test for apraxia (De Renzi et al 1980) involving both meaningful and meaningless gestures was achieved, and is now widely accepted as sensitive and reliable in the diagnosis of apraxia (Tate & McDonald 1995) though no reliability or validity data is available for the test.

De Renzi et al (1982) also reported a use-of-objects test using verbal, visual and tactile modalities showing evidence of gesture impairment related to the modality involved. They reported some patients performing remarkably less well in one modality compared to the other. They concluded that apraxia could be modality-specific (i.e. contingent upon a specific lesion interrupting pathways connecting sensory association areas with movement planning areas). Having reviewed apraxia examination methods extensively,

De Renzi (1985) concluded that whether the movement requested was symbolic or meaningless, transitive or intransitive, simple or complex, the crux of the deficit lay in the patient being unable to select appropriately from his movement repertoire.

In rehabilitation research it is clearly important to incorporate all of these elements into the assessment procedure to elicit the conditions under which individuals perform better or worse. This would help to target intervention strategies using the 'best performance' modality. In addition, carrying out a variety of assessment tests allows a judgement concerning the dissociation of one test with another for each individual, and would also help in differentiating between the (potential) different *types* of apraxia.

2.6 Anatomical basis of apraxia

Another avenue of investigation has been to explore the anatomical basis of apraxia. Whilst lesion site as a classification system might not be considered useful (e.g. supramarginal apraxia, callosal apraxia), one of the questions that has challenged researchers of apraxia was the extent to which the *dysfunction* could be related to specific lesion sites within the brain. Heilman and Rothi (1993) stated that the majority of cases of apraxia (in right handed patients) were associated with left hemisphere lesions. The importance of the left hemisphere to praxis and the identification of specific areas within that hemisphere devoted to voluntary action and guiding of movement is well documented (Halsband & Passingham 1985, Kertesz 1985, Fisk & Goodale 1985; 1988, Passingham 1987, Halsband & Freund 1990, Ikeda et al 1992, Goodale et al 1994, Haaland & Harrington 1994, Jennerod et al 1994, Jackson & Husain 1996). Certainly the relationship between left (dominant) hemisphere lesions and apraxia has been confirmed

and accepted by researchers from the early Leipmann work onwards (Geschwind 1975, De Renzi et al 1980, Kertesz & Ferro 1984, Faglioni & Basso 1985, Basso et al 1987, Alexander et al 1992, Heilman & Rothi 1993, Tate & McDonald 1995). The evidence for specific lesion localisation within that hemisphere which ***causes*** apraxia is, however, equivocal.

In their review of the historical perspectives of apraxia, Faglioni and Basso (1985) stated that Liepmann's early paper in 1900 proposed a disconnection theory to explain the disruption to praxic function. This related to parietal and callosal destruction which caused an isolation of part of the sensory-motor system such that apraxia was evident when tested through one sensory modality alone. It would appear that Liepmann, while acknowledging that different expositions of apraxia would follow from different lesion locations, did not suggest a *centre* for praxis where gestures were processed and movement was programmed (Faglionni & Basso 1985). Liepmann had proposed that parieto-occipital lesions would have more ideational qualities, whilst supramarginal gyrus (intermediate parietal) lesions would have more ideomotor qualities.

Geshwind (1975), in developing the disconnection proposition for apraxia, considered that the motor association pathways were the critical ones and that destruction of these explained most apraxic conditions. These included lesions involving Wernicke's language area, the supramarginal gyrus, the premotor region, subcortical connections between the two (the arcuate fasciculus) and callosal lesions which could all lead to a disruption in praxic function particularly in relation to carrying out verbal commands. When the patient was unable to imitate gestures, Geschwind (1975) attributed this to disruption of the pathways from the visual association cortex contained in the arcuate fasciculus. A

synopsis of studies relating apraxia to lesion site is given in Table 2.2 and indicates how evidence has concentrated on left hemisphere involvement with some considerable controversy concerning the influence of callosal and basal ganglia damage in the apraxic condition. Right hemisphere lesions have also been reported as leading to apraxia, but these are much less common than left hemisphere involvement and the literature has presented such cases in relation to handedness and/or dissociation with language deficits (Poeck & Lehmkuhl 1982, Haaland & Flaherty 1984, Archibald 1987, Mozaz et al 1990).

What is evident from all the above studies is that apraxia has consistently been demonstrated as arising from a *number* of localised areas in the cortex, as well as from lesions in subcortical structures and connections. However, as Tate and McDonald (1995) remarked, it must be remembered that despite the evidence of such lesion studies, many other studies demonstrated that dysfunction in those areas did not *necessarily* and automatically lead to apraxia. So it remains to be determined whether apraxia is actually related to specific sites within the left hemisphere and whether specific dysfunctions are associated with focal areas of damage. No attempt has been made, in apraxia research to date, to relate rehabilitation outcomes to lesion site nor to establish which rehabilitation strategies might be more effective according to lesion location.

Table 2.2 Synopsis of apraxia and lesion site studies.

Authors	Date	Findings
Basso et al	1980	123 cases. Left hemisphere stroke. No differences between apraxic and non-apraxic relating to lesion size and location except non apraxic higher incidence of deeper lesions.
Agostoni et al	1983	7 cases. Ideomotor apraxia associated with basal ganglia and thalamic lesions in both hemispheres.
Watson & Heilman	1983	Single case. Corpus callosum lesion associated with apraxia
Kertesz & Ferro	1984	177 cases. Deep lesions in parietofrontal & occipitofrontal connections plus lesions in anterior corpus callosum all associated with apraxia. Lesion size and location positively correlated with severity of apraxia.
Rothi & Heilman	1985	Review. Supramarginal gyrus & angular gyrus led to different dysfunctions in apraxia compared to disconnection lesions involving premotor and motor areas.
Freund & Hummelsheim	1985	11 cases. Premotor cortex lesions led to limb-kinetic apraxia.
Faglioni & Basso	1985	Supplementary motor area (SMA) damage led to apraxia when anterior corpus callosum involved.
Watson et al	1986	2 cases. SMA damage led to disturbance in spatial and temporal ordering of movement in object use.
Graff-Radford et al	1987	Single case. Corpus callosum lesion associated with apraxia.
Passingham	1993	Animal studies. SMA ablations showed role in selection of appropriate movements.
Halsband et al	1993	Premotor cortex involved in correct retrieval of movement following a visual cue. SMA involved in internally remembered movement sequences, retrieval of self-initiated movements & in temporal aspects of motor planning.
Heilman & Rothi	1993	Review. Apraxia and lesion site dependent on pattern of language and motor dominance in the individual.
Leiguarda et al	1994	10 cases. Corticobasal degeneration associated with ideomotor and ideational apraxia.
Nadeau	1994	Single case. Left thalamic infarction associated with severe ideomotor apraxia.
Pramstaller & Marsden	1996	Meta-analysis. 82 cases. Lesions *solely* confined to basal ganglia rarely caused apraxia. Internal capsule, superior longitudinal fasciculus & fronto-striatal connections are areas most commonly associated with apraxia.

2.7 Assessment and examination of apraxia

The tests for apraxia described in the literature are many. Until a reliable and standardised battery is available, Tate and McDonald (1995) suggested that clinicians and researchers alike should use the type of batteries developed by De Renzi et al (1980) for in-depth examination of apraxia, but that there should be an additional inclusion of qualitative descriptions of the type of errors made during the assessment procedures.

The tests for ***ideomotor*** apraxia described in the research literature fall into two main categories; 1. gestural tests using verbal command and imitation, or imitation alone,

2. use-of-objects tests using the three modalities discussed above: verbal, visual and tactile (using the real object).

The content of both these types of tests differs from research group to research group but there is a gradually developing consensus. Some researchers have used both types of tests, others have used only the imitation tests. Scoring has also differed across research groups, but all tests do appear to take into account the theoretical notion that a] there is a need to test across modalities, b] meaningful/meaningless gestures may elicit differences in different groups of patient and therefore should be differentiated in the tests, c] single gestures and sequences of movement may discriminate between different types of apraxia, and d] use-of-object tests may provide some useful information for the clinician especially in terms of error types observed. Summaries of assessment battery components and scoring systems for movement imitation tests are given in Table 2.3 and for use-of-objects tests in Table 2.4.

Fraser and Turton (1986) developed the Cambridge Apraxia Battery using 27 stroke patients and showed high inter-rater reliability, but as this particular attempt at a

standardised test was not accepted or adopted by any of the researchers in neuropsychology the result was to make it redundant. What is interesting about the assessment tools currently accepted and used in the diagnosis of apraxia is that no reliability and validity data is available for them. These are tests compiled and devised through expert opinion about what might elicit apraxic errors in patients, and as more and more researchers use and value these tools they acquire a 'classic' aura. In addition each subsequent assessment tool that is suggested by researchers uses the 'classic' tests as criterion reference without questioning whether or not these early tests are actually measuring what they say they are measuring. It should also be pointed out that in the published literature, many researchers do not adhere to a principle of using a battery of different examination procedures in order to capture the heterogeneous nature of apraxia, but rather continue to diagnose apraxia relying upon a single test.

Neiman et al (1994 & 1996) took a different approach from other research groups and looked towards using the Kaufman Hand Movement Test as a method of assessing apraxia. This test, originally devised to assess attention and concentration, involved copying hand gestures and the authors have reported its high concurrent validity as a measure of limb apraxia.

The examination for ***ideational*** apraxia has concentrated on using a variety of objects in a sequence and there does appear in the literature to be a growing general consensus between research groups on what those tasks could involve. A summary of test components and scoring systems for identification of ideational apraxia is given in Table 2.5.

Table 2.3 Summary of movement imitation tests.

Authors	Date	Test Items	Scoring system
De Renzi et al (appendix D1)	1980	Movement imitation only :- 12 meaningful movements 12 meaningless movements (including hand and finger gestures both static and in sequences of movement)	3 = flawless on 1st trial 2 = flawless on 2nd trial 1 = flawless on 3rd trial 0 = impaired performance
Haaland & Flaherty (appendix D2)	1984	Verbal command and imitation :- 5 meaningful movements 5 meaningless movements	Number of errors.
Poeck et al	1982	Verbal command and imitation :- 10 meaningful movements 10 meaningless movements (including equal numbers of unimanual and bimanual movements in each section).	Correct/incorrect (pass/fail) scoring. Cumulative end score of passed items.
Raade et al	1991	Verbal command only 20 movements	Pass/fail scoring. *Types* of errors recorded.
Alexander et al	1992	Verbal command and imitation. Meaningful and meaningless movements plus object use pantomime.	5 = correct performance. 4 = correct performance after an error. 3 = BPO error 2 = repeated, undifferentiated movement. 1 = perseverative movement. 0 = no movement.
Leiguarda et al	1994	Verbal command and imitation	3 = correct performance. 2 = partially correct performance. 1 = weak resemblance. 0 = incorrect/unrecognisable performance.

Table 2.4 Summary of use of objects tests.

Authors	Date	Test items	Scoring
De Renzi et al	1980	Visual presentation of common objects. Verbal request for gesture of object use.	2 = flawless performance. 1 = correct at 2nd trial. 0 = incorrect performance.
Haaland & Flaherty	1984	5 common objects in three conditions :- verbal command, imitation of gesture, using the real object.	Number of errors counted.
Riddock et al	1989	38 common household objects in two conditions: verbal command & visual presentation of object. Some objects requiring bimanual pantomimed performance.	Score 1 for each correct gesture.
Buxbaum et al	1995	Common objects in three conditions :- verbal command, imitation of gesture, using the real object.	3 = correct performance. 2 = partly correct performance 1 = partial resemblance. 0 = unrecognisable performance.

Table 2.4 Summary of use-of-objects tests.

Table 2.5 Summary of ideational apraxia tests

Authors	Date	Test items	Scoring
De Renzi & Luccelli (appendix D4)	1988	6 task items using multiple objects (paper into file, polishing shoes, mailing a letter, lighting candle, making cup of tea, locking a box).	Number of errors counted.
Riddock et al	1989	Same items as De Renzi et al (1988) above.	Number of errors counted.
Leiguarda et al	1994	3 items (Torch and batteries, mailing letter, padlock & key)	Number of errors counted.
Motomura & Yamadori	1994	Combination of De Renzi & Luccelli (1988) and Leiguarda et al (1994) items.	2 = correct performance 1 = incorrect but recognisable. 0 = unrecognisable performance.
Buxbaum et al	1995	4 items (making toast, preparing lunchbox, wrapping a gift, packing a school bag).	Number of errors counted.

Table 2.5 Summary of ideational apraxia tests.

As is clear from this wide variety of assessment procedures and scoring methods, much continues to rely upon the examiner's judgment and expertise in observing errors. The examiner/clinician must decide to what extent such errors can be attributable to other neurological deficits, and what can not and therefore might be considered to be evidence of apraxia. Tate and McDonald (1995) were probably right to conclude the need for development of a detailed clinical assessment tool with reliable and explicit scoring criteria that is more objective than observer expertise. Such objective measures might emerge from clinical tools or might arise from use of the new computergraphic technologies which can analyse limb movement and quantify errors in a reliable, standardised and objective manner. For the clinician, in the absence of a 'gold standard', the tests currently available are better than no test at all. Nevertheless care must be taken to look at the accumulation of evidence from both formal testing and functional performance observations before arriving at a conclusion concerning the presence and type of apraxia which might affect any individual patient. It is suggested that the presence of one or more movement errors is not, in itself, diagnostic of apraxia, but must be taken and weighed in balance as a part of the whole clinical picture.

2.8 Kinematic analysis of apraxic movement

The difficulties in clinical testing for apraxia and reliance on observer judgment of apraxic errors led Goodale et al (1990) to argue the need for more objective, quantitative measures to be used in the analysis of motor performance in people with different types of hemisphere lesions. Such movement analysis, exploring the kinematics or components of movement has built upon the work of Jeannerod (1984, 1988 and 1990) and Soechting

and Terzuolo (1990) and attempted to uncover the nature of the loss of motor control in apraxia and look for evidence which would support or refute the clinical evidence for the construct of apraxia at the level of impairment. Fisk and Goodale (1988) noted that people with left hemisphere lesions took longer to complete tasks, had a lower peak velocity of movement, had a characteristic prolonged period of low velocity at the end of a 'reach' towards a target and were less accurate in their location of the target when compared with right hemisphere lesion patients and control subjects. Right hemisphere lesion patients were shown to have a longer time in *initiation* of movement compared to the control group in this study. Fisk and Goodale (1988) suggested that the left hemisphere group were deficient in their ability to update and modify the trajectory of their reach as the movement unfolded and also perhaps being unable to utilise proprioceptive information to determine the position of the limb. They conjectured that this would result in greater dependence upon *visual* information to achieve the required movement, in turn leading to both increased time and decreased accuracy in performing the movement. This study was followed up by looking at right hemisphere lesion patients to determine the role of the right hemisphere in visually guided movement.(Goodale et al 1990). The deficits in trajectories in reaching to a target shown by those *without* clinically-apparent neglect signs led the authors to suggest that the interaction between motor behaviour and cognitive and perceptual processes should not be ignored. This has implications in apraxia research where the influence of vision and perception, particularly in the testing procedures could be important in understanding the nature of the condition (Riddock et al 1989) but also in illuminating the different performance outputs according to modality of testing (verbal, visual, real-object use - see section 2.5).

Unfortunately kinematic studies with apraxic patients have mainly been with small numbers due to the complexity of the computergraphic recording, the vast array of data that is produced, the time-consuming and difficult nature of analysis of such recordings as well as the difficulties in patient recruitment. Nevertheless, individual cases and small n studies do provide useful and important data, and build up to provide a body of knowledge to which researchers can refer. Poizner et al (1990) investigated the kinematics of two apraxic patients using computergraphic technology. Gestures such as winding down a car window and carving a turkey were used to establish the timing and spatial elements of movements in the ipsilateral limb. In contrast to the Fisk and Goodale (1988) study, Poizner and colleagues' project showed differences in the initiation of movement in apraxic patients when compared with normal controls. In the two cases studied, there was shown to be delay in starting the movement and also an initial hesitation before the movement was fully begun, revealed by a slow build up of velocity. Difficulties and hesitations were also observed in the transition phases of movement components and a general loss of fluidity of movement in the apraxic subjects. There was no indication, in this Poizner et al study, of the low velocity pattern at the end of movement seen in the Fisk and Goodale research. This may have been due to the different nature of the movements being analysed; one being a single movement aiming for a target, the other being a repeated movement, representative and meaningful. Other kinematic analyses (Poizner et al 1990) found errors and over-use of proximal joints in movements in the apraxic patients. It was suggested that such compensatory movements were an attempt by the apraxic people to gain control of their distal musculature. Spatial

orientation of movement was also judged to be impaired in the two cases presented by Poizner et al (1990).

Later kinematic research by Poizner et al (1995) on three people with ideomotor apraxia used a 'slicing bread' gesture as the basis for analysis. They found an interruption of normal coordination in these cases which was consistent in both gesture performance and actual tool use. These same apraxic subjects were reported in Clark et al (1994) who looked at spatial planning deficits. Again for both gesture (slicing bread) and for actual tool and object use, the apraxic participants were shown to have deficits in plane of motion and in the shape of their trajectory of movement. They also showed a dissociation of the normal relationship between speed and trajectory of movement. Poizner et al (1997) concluded that people with apraxia show deficits in the spatio-temporal aspects of movement and in the coordination of joint movements both in verbal command conditions and in actual tool use. They suggested that such observed deficits could be attributed to the disruption of 'visuokinaesthetic motor representations of learned movement' (pp 108) stored in the posterior parietal area, or from a separation of those representations from the motor or premotor areas, rather than the disconnection hypothesis (Geshwind 1975) between the language and motor areas where actual tool/object use should not be disrupted . These authors have suggested that the use of kinematic studies to determine the underlying impairment of apraxia could allow for discovery of new subclassifications of the disorder as well as detecting subtle forms of the condition.

One 'large number' study in the kinematics of apraxia has been published. Hermsdorfer et al (1996) chose to use meaningless gestures in the kinematic analysis of apraxic

movement in both left and right hemisphere damaged subjects. This study reported right brain damaged subjects performing as well as the control subjects in spatial accuracy and kinematics of movement trajectory. The left hemisphere damaged subjects (n=20, both apraxic and non-apraxic) were reported to have a higher frequency of kinematic errors compared to the other groups. These errors did not, though, correlate with apraxic errors. Indeed, four apraxic subjects in this study were reported as having demonstrated a completely normal kinematic profile. This dissociation between clinical assessment of apraxic errors in action and the kinematic components of movement is an intriguing one. One the one hand, in clinical assessment the observed errors fall mainly into the spatial components of the movement (though judgments of the temporal components are also made) as well as in judging the position and orientation of the hand and limb. In the kinematic analysis great emphasis is placed upon the temporal component (particularly on initiation time and completion time of the task) and on the velocity and acceleration aspects of movement that are not within the clinician's observable domain. At this very basic level, it could be argued that the two forms of examination would inevitably be dissociated and they are measuring different things. Hermsdorfer et al (1996) argued that the inadequate preprogramming of movement and the errors in the *determination* of the target movement could be considered two *independent* sequels of left hemisphere lesions and would thus lead to the dissociations shown in the study. Alternatively, Hermsdorfer and colleagues have suggested, the dissociation could be ascribed to the different strategies which apraxic patients might adopt to compensate for a loss of mental representation of the target position. Patients might adopt a strategy of slow, controlled movement to achieve the required end position resulting in abnormal kinematics, or they

might move smoothly and normally to a *rough approximation* of the target position ignoring the movement deficiency. Hermsdorfer et al suggested that depending on the success or otherwise of these strategies, the associations and dissociations between kinematics and clinically observed apraxic errors might occur. The 'strategy for coping' explanation is compelling, especially as different strategies for compensation for the deficit could account for both the cognitive and perceptual interactions which have been suggested by researchers and the heterogeneity of the manifestations of the condition. Such explanations could also account for the trajectory and kinematic profiles found in the Poizner et al and the Fisk and Goodale (1988) studies cited previously.

The advances of technology have allowed researchers to examine the subtle deficits, seen with hemispheric lesions, which are not apparent in normal observation techniques. At present there is conflicting evidence in the research literature over the kinematic elements which might characterise apraxic movement although there is compelling evidence of a disruption of the temporal-spatial elements of movement. Clearly a replication of previous work needs to be undertaken with a greater number of subjects, in order to clarify the potentially unique kinematic characteristics of apraxia and to explore the different interactions between modality of eliciting movement performance and kinematic output. Further studies of this nature might also help to identify the suggested 'sub-types' of apraxia within the heterogeneous condition. A confirmation of the dissociation between kinematic profiles and apraxic errors would also be a useful contribution to the understanding of the underlying impairment of apraxia. It could be argued that if this were achieved then kinematic analysis might be a useful objective tool for the identification of apraxic conditions. It could also provide objective evidence of change

and recovery in these patients and could, therefore, be used as one of the outcome measures in investigations of the effectiveness of intervention in rehabilitation.

2.9 Rehabilitation issues

Research relating to rehabilitation of apraxia and effectiveness of intervention is sparse. The limited published work that is available has generally relied upon expert opinion concerning what might be effective strategies for rehabilitation. For example, Fraser and Turton (1986) have made some suggestions for therapeutic approaches based upon how the individual patient responded and scored in the Cambridge Apraxia Battery (Fraser and Turton 1986). Miller (1986) similarly reviewed and described approaches which might be attempted with the apraxic patient, but did not investigate the effectiveness of such interventions. The first major attempt to evaluate a rehabilitation strategy used with an apraxic person was reported by Wilson (1988). In this case study, a break-down of tasks and a chaining procedure were used together with verbal mediation. This showed dramatic effects on functional performance in two tasks: drinking from a cup, and sitting on a chair and positioning it correctly at a table. Pilgrim and Humphreys (1994) also reported the effectiveness of verbal mediation strategies in the regulation of motor acts in a case of ideomotor apraxia.

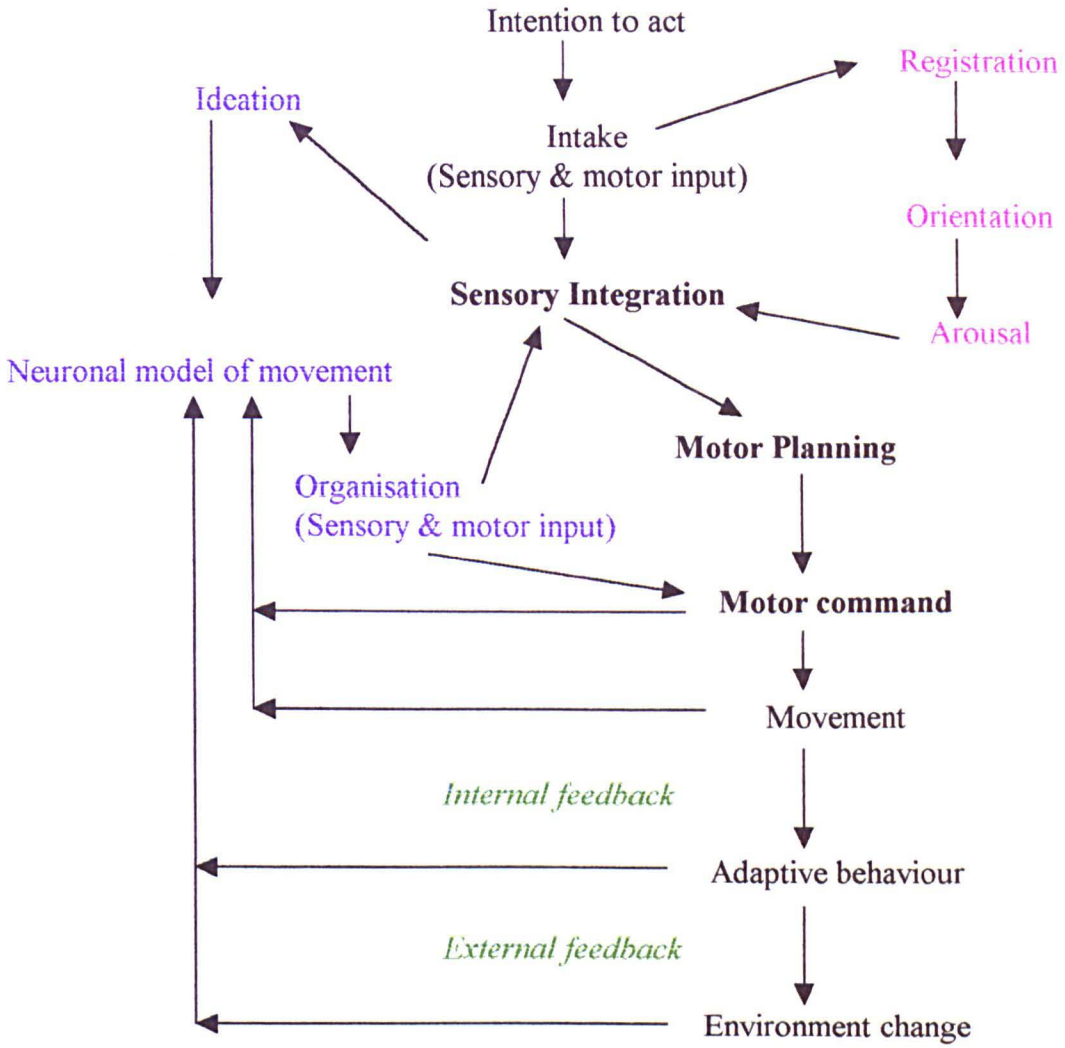
Mayer et al (1990), whilst not describing rehabilitation *per se*, argued the case for examining apraxia performance in a naturalistic setting using functional tasks both for initial diagnosis and for monitoring change. They reported errors in action affecting daily performance in those with both ideomotor and ideational apraxia. Such observational approaches for apraxia assessment and monitoring recovery are supported by several

workers (Carr & Shepherd 1980, Miller 1986, Arnadottir 1990, Schwartz et al 1993, McDonald et al 1994, Foundas et al 1995) though all have agreed that objective measures are critical and that reliability of observed behaviour is problematic. A combination of formal clinical assessment, kinematic analysis of movement and behavioural observations of functional activities clearly needs to be established. Such a combination would provide the most rigorous approach for initial diagnosis as well as for monitoring change and recovery processes both in the clinical field and for research purposes.

The rehabilitation strategies and therapeutic interventions described, but not evaluated, in specialist manuals and texts (Siev et al 1986, Rosenthal et al 1990) have focused on both isolated movement components and practice in functional tasks. The involvement of cognitive processes to enhance motor performance was suggested by Croce (1993) who argued that people with apraxia needed verbal cueing to encourage them to look at what they were doing and where they were going. That level of visual cueing, Croce suggested, should be accompanied by the provision of a visual model of the movement performance. This should be coupled with verbal mediation from the patient (or therapist) saying what they were doing or going to do. Riddock et al (1995) reported evidence suggesting improved movement action performance with such verbal cueing strategies and visual feedback in a case of ideomotor apraxia.

Croce (1993) also stated that a multifaceted intervention approach should incorporate sensory stimulation procedures. This philosophy arose from the work of Ayres (1985) who had reported sensory stimulation intervention effectiveness in developmental dyspraxia in children. Ayres (1985) had suggested that the main difficulty in apraxia lay in the neural activity which takes place before the motor execution is begun (Figure 2.1).

Figure 2.1 Model of motor control and praxis (taken from Ayres 1985)



Ayres argued that although developmental dyspraxia and adult-onset apraxia could be viewed as an output disorder because the motor component was more observable, the crux of the dysfunction lay in the sensory information integration and planning part of the motor system. Ayres (1985) reported a consistent relationship between developmental dyspraxia and a disrupted tactile system. This research led to the development of a treatment programme based on stimulating the tactile system in all sensory modalities to

improve motor output in developmentally dyspraxic children. In attempting to integrate the underlying construct of dyspraxia and adult-onset apraxia Goodgold-Edwards and Cermak (1989) reported two major processes of movement control: sensory organisation, which integrates orientation information from visual and somatosensory input, and muscle coordination which determines the temporal sequencing and distribution of muscle contractile activity. The authors suggested that problems arose in both the clinical conditions (developmental dyspraxia and adult-onset apraxia) when there was an inability to correctly integrate body senses or an inability to select the most functionally appropriate sensory information. It was suggested, therefore, (Ayres 1985, Goodgold-Edwards & Cermak 1990) that sensory input and sensory enhancement would facilitate the development of that necessary integration of the sensori-motor system in order that skilled purposeful actions might be performed. Morris (1997) criticised the lack of strong empirical research to support theories linking sensory-perceptual dysfunction with dyspraxia but, nevertheless, recognised the sensory integration intervention programme as being the most frequent approach carried out with developmentally dyspraxic children, and stressed that the essential component in assessment was a careful evaluation of sensory-perceptual function. Croce (1993) used Ayres' philosophy and theories, and the sensory integration therapy protocols arising from those assertions, to recommend a research priority to examine the effectiveness of such intervention procedures in adult-onset apraxia.

The lack of empirical evidence for effectiveness of rehabilitation in apraxia (as well as most other neurological disorders) highlights the most pressing need for rehabilitation research in neuropsychology. Such measurement of the effectiveness of intervention

would need to incorporate a reliable quantitative analysis of task performance and evaluation of kinematic changes in movement performance, as well as functional outcomes.

Chapter 3.

STATEMENT OF THE PROBLEM AND AIMS OF THE RESEARCH

The literature review has highlighted a number of issues and problems in the assessment and rehabilitation of those with apraxia. There is conflicting evidence of both the nature and the type of errors in movement made by people with apraxia. The relationship of apraxia to lesion location is unclear. The assessment of apraxia by identification of (arguable) error types is heavily criticized in the research literature. Such identification relies upon 'expert' judgment to extrapolate, from observation of an abnormal motor performance, what may or may not be attributable to coexisting primary deficits in the patient and what could, therefore, be attributable to apraxia. This largely subjective decision-making in the diagnosis of apraxia is reported to be heavily dependent upon the individual clinician's expertise and experience. To add to the confusion, the assessment tests widely used and accepted in apraxia research and in the clinical field are non-standardised with little published evidence of inter-rater reliability in the diagnosis of apraxia nor of validity of the tools. More objective measures of assessment are required and the use of computergraphic technology might provide such a source of information. The limited kinematic studies in apraxic movement as have been reported to date demonstrate conflicting findings. The lack of consensus could be partly, or wholly, attributable to both the conceptually different movement performances which were being measured and the nature of a heterogeneous condition grouped under a blanket term of apraxia. Most studies have used single case analyses (Poizner et al 1990 and 1995, Clark et al 1994) with only one study published to date using larger numbers of subjects (Hermsdorfer 1996).

The most critical gap in the knowledge base is in the area of applied rehabilitation research. The dearth of studies investigating effectiveness of intervention is indicative of the early stage of research in this area. There are various interventions suggested in therapy texts as being appropriate to use with people with apraxia, but little evidence that any of these cause change either in isolated elements of motor performance or in function. Neither are the different techniques evaluated one against another. There is no evidence or indication that these techniques work better with one *type* of apraxia than with another, or according to levels of severity of the deficit. Whether the effectiveness of intervention might have a relationship to the underlying pathology is also unknown.

The research questions arising from the literature review could be viewed under two main themes:

1. Assessment of apraxia

i] Does kinematic analysis of apraxic movement performance aid identification of the condition? Can it provide an objective, reliable measure to confirm or refute the clinical tests relying on observer judgment and support the notion of an underlying construct of apraxia? Does it provide evidence for sub-classifications of apraxia?

ii] Do kinematic data, using group comparisons, support published findings of single case analysis of movement deficits?

iii] Can the kinematic differences in apraxic and non-apraxic movement performance be used as an indication of change and/or recovery of praxic ability over time in those with apraxia?

2. Intervention in apraxia

i] Do the normal intervention procedures, chosen and used by experienced therapists, demonstrate an effect on motor performance immediately after intervention and/or over time?

ii] Does the addition of a specific sensory intervention protocol have an effect on motor performance immediately after intervention and/or over time?

iii] Does functional change accompany improvement in isolated motor performances?

iv] Can task break-down, combined with verbal and visual mediation strategies, lead to improvements in the practiced tasks?

v] Does the patient profile (lesion site, underlying pathology, severity of apraxia) influence the effectiveness of intervention?

vi] What is the 'natural history' of apraxic movement as determined by kinematic analysis? What are the changes that occur over time? Is there evidence for spontaneous recovery from apraxia?

Two main aims for this project arose from these research questions. The first aim was to analyse particular movement deficits which occurred in apraxia for purposes of assessment and outcome measures. The second aim was to explore strategies of intervention and rehabilitation.

These aims were translated into specific objectives :

Firstly -

1i] - to determine the presence of deficits in movement performance in apraxic patients using computergraphic techniques and to identify components of that movement which might be unique to apraxia using group comparisons.

1ii] - to analyse the relationship of movement performance to input modality in testing and also to site of brain lesion.

1iii] - to determine the relationship between kinematic analysis of movement performance and the clinical apraxia tests.

1iv] - to determine whether kinematic analysis of movement provided evidence to identify 'sub-types' within the apraxic condition.

1v] - to determine the usefulness of kinematic analyses as outcome measures of the effectiveness of intervention.

Secondly -

2i] - to evaluate specific intervention strategies for apraxia using single case design.

2ii] - to determine whether different patient profiles (underlying pathology, severity of apraxia) influence effectiveness of intervention.

Specifically the research hypotheses were that :

1. there is a difference in movement performance, as shown by kinematic analysis, between three groups: those with clinically diagnosed apraxia, those who are non-apraxic but brain damaged, and normal controls.

2. there is a difference in apraxic movement, shown in kinematic analysis, according to the modality of testing.

3. the addition of a sensory stimulation programme leads to an improvement in motor performance in apraxic patients compared to previous (baseline) interventions.

4. task breakdown combined with verbal and visual mediation strategies leads to improvements in practiced functional tasks.

Chapter 4. METHODOLOGY

In order to carry out the aims of the research project to evaluate assessment protocols in apraxia, including determining the movement deficits which might be unique to apraxia, and assess the effectiveness of intervention strategies with apraxic people it was decided to have two components to the study. The **first part** of the project was a *group study* using both clinical assessment tools and computergraphic recording equipment to determine whether apraxic movements had unique kinematic components. The **second part** of the project involved *single case* analyses from both 'natural history' and experimental design data.

Elements of methodology were common to both parts of the research project and are described in this chapter, with individual details of methodological importance highlighted in the main data chapters.

4.1 The setting - Rivermead Rehabilitation Centre

The setting for all patient assessment and intervention procedures was a rehabilitation centre in the outskirts of Oxford but which serves a wide geographic area of Oxfordshire, Buckinghamshire, Northamptonshire and beyond. The rehabilitation center caters for both in-patients and out-patients with severe, usually complex, neurologically-based disability mostly related to stroke or head injury. Clients' ages range from 20-70 years. There is a physiotherapy department with a hydrotherapy pool, occupational therapy department, speech and language therapy services, social work department, psychology department, specialist disability information and advice services, an independent living unit, facilities for re-establishing leisure activities and skills, as well as a ward for in-

patients. People are referred to the center by GP's, hospitals, and sometimes by social workers, families or clients themselves. There is a recognition of the need for specialist, prolonged and intensive rehabilitation services after the initial medical or emergency care is completed, and/or after initial rehabilitation programmes have been completed. The center is part of the NHS.

The center has, as a guiding philosophy, a client-centered approach to rehabilitation with a focus on the needs and desires of the client and his family (McGrath et al 1995). The main objectives are to concentrate on the aspirations of the client and family and to ensure that all interventions relate to those wishes. The ultimate aim is to enable each client to lead the best life he/she can within the limits of any permanent damage he may have; the client himself chooses those areas of life and functioning he/she would like to concentrate on. Most clients are discharged within nine months. Some clients remain involved with the rehabilitation center on an out-patient basis for several years while some attend for a limited assessment period of a few weeks long after their initial injury or illness to receive advice on final placement or on work or occupation.

The patients involved in this research project were those with a severe brain damage or injury requiring tertiary rehabilitation following in-patient treatment. All patients were assessed in the same room, at the Rivermead rehabilitation centre, throughout the study.

4.2 Subjects

Three experimental groups were devised for the first (kinematic) study; two groups of clinical subjects (apraxic and non-apraxic) and one group of normal healthy controls. Recruitment of clinical subjects followed normal informed consent procedures as

approved by the Regional Ethics Committee (appendix 1- COREC approval letter, appendix 2 - patient consent letter) and was carried out as soon as was practically possible after admission. Patients with unilateral left hemisphere lesions, as defined by clinical diagnosis from medical notes and CT scan, were approached for participation in the project. If consent was obtained the patients were screened for the presence of apraxia. Normal control subjects were recruited through verbal request for volunteers and information leaflets distributed throughout the university and the local population (appendix 3 - normal control consent letter). These subjects were tested at the School of Occupational Therapy, Oxford Brookes University.

Exclusion criteria from both parts of the research project (group study and single case) were :

a] the presence of other neurological disease or injury (e.g. Parkinson's disease, Multiple Sclerosis, learning disability)

b] severe aphasia so that the patient was unable to understand the testing procedure. The cut-off score for comprehension on the Frenchay Aphasia Screening Test 'FAST' (Enderby et al 1986) was set at 3 or less, on the advice of the Speech and Language therapist. The FAST score was available for all patients as part of their admission procedure to Rivermead Rehabilitation Centre.

c] emotional or behavioural problems which suggested that research participation would be counter-productive to the patient's well-being or which would interfere with testing procedures. Advice and approval for participant recruitment was sought from the medical consultant in charge and also the treating clinical psychologist at the Centre.

d] dementia or cognitive impairment of a severity that rendered the patient unable to participate in testing procedures. Advice was sought from the consultant and the clinical psychologist concerning the cognitive abilities of each patient.

The recruitment of participants for the single case intervention studies followed identification and suggestion by the consultant of the rehabilitation centre as to potential suitability. The inclusion criterion was the presence of ideomotor or ideational apraxia as elicited by clinical assessment. No specific underlying pathology was excluded. The exclusion criteria were the same as for the group study (identified above) and normal consent procedures were followed.

4.3 Equipment - Kinematic Recording

An identical protocol for kinematic recording was used for all subjects in both the group study and single case investigations. Using the Qualisys MacReflex analysis system (Qualisys AB, Partille, Sweden) recordings were made of subjects' upper limb movement during a drinking task. Following calibration procedures an infra-red camera was placed two metres from the subject at a constant height and with fixed lens settings. Recordings were then made, in real time at 50 frames per second, of the two dimensional positions of infra-red markers placed upon the subject's arm and hand. Markers were positioned at four specified locations on the subject's left arm and held in place using elastic and Velcro straps plus sticky pads to ensure maximum possible accuracy of recording. The location sites were as follows :

1] A shoulder marker was placed over the axis of the joint on the lateral aspect.

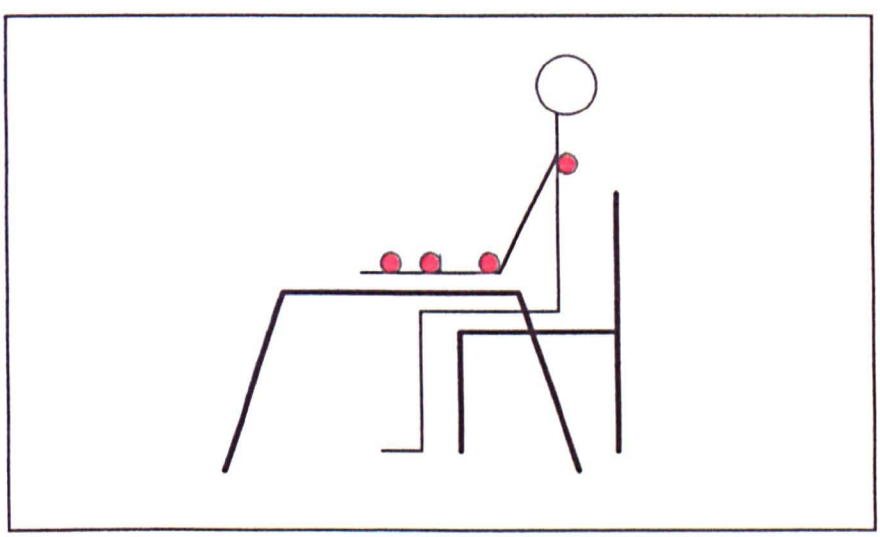
2] An elbow marker was placed over the axis of the joint on the lateral aspect.

3] A wrist marker was placed on the posterior aspect of the wrist on the line joining the radial and ulnar condyles.

4] A hand marker was positioned on the posterior aspect of the hand over the metacarpals.

The starting position for the task was with the subjects seated at a table, left hand resting on the nearest edge of the table palm turned inwards, and the elbow at 90° such that all markers were clearly visible and facing the camera (figure 4.1). A red spot was placed on the table fifteen inches from the edge nearest to the subject to act as a 'target' identifier.

Figure 4.1: Subject position and infra-red marker placement.



The choice of experimental task took into account the need for it to be meaningful and also capable of being used in similar modality conditions to the normal clinical tests; i.e. to verbal command, to visual modeling (copying tester gesture) and using a real object. The task needed to be simple enough to be capable of completion by most patients. A

difficult task that led to failure of completion in a number of patients would not provide the kinematic recordings useful for analysis. A drinking task was selected and was repeated three times in different modality conditions:

1. *Verbal modality*; instructions given. "Pretend there is a mug on the red spot on the table. Using your left hand, reach for this mug, pick it up, take a (pretend) drink from it, return it to the red spot and put your hand back to its starting position".

2. *Visual modality*; instructions given. "Copy this movement." The assessor, sitting opposite the subject, made the gesture of reaching for an imagined mug on the red spot, taking a drink from it, returning it to the red spot and placing the hand back in the starting position.

3. *Real object use / tactile modality;* a mug was placed onto the red spot with the handle placed to the left, and subjects were asked to repeat the movement as before but this time using the real object.

Recordings were taken of each movement from the time the tester gave the starting instructions of "Three, two one, **go**". Each subject had one trial in each of the modality conditions, not only to capture the 'naive' and first motor performance to each modality cue but also to exclude practice effects. It was also considered to be the most sparing of patient time and effort given the clinical assessments to be carried out at the same time.

4.4 Apraxia assessment

Five clinical tests of apraxia were selected in order to capture all the elements deemed (by the research literature) to be important in the examination and diagnosis of the condition. Test selection, therefore, included items of object use with differing modality of

instruction including gesture tests to verbal command and imitation with meaningful vs. non-meaningful elements, single gestures vs. sequences of movement, and personal vs. peri-personal space. A test for ideational apraxia was included using multiple objects in a sequential manner. These tests were based upon, or taken from, the work of De Renzi et al (1980), DeRenzi and Lucchelli (1988), Haaland and Flaherty (1984), and Poeck (1986). Details of these are given in full in Appendices D1-D4.

For the group study, the clinical participants (all with left hemisphere lesions) were asked to perform the tasks with the left 'unaffected' hand (i.e. the limb ipsilateral to the lesion) in order to exclude any effect due to hemiparesis or sensory impairment. Normal, healthy controls were also asked to use the left (non dominant) hand. In the single case research, where bilateral brain damage was present, both hands were tested. For all apraxia testing, the participants were seated at a table in a quiet room with the assessor seated opposite. The following standardised instructions were given to all subjects:

> "I am going to ask you to make some movements with your arm and hand, and also to pretend to do things using your hand. First of all I will tell you what kind of movement to make. Then I will ask you to copy some movements which I will make. Let us try a couple of movements out so that you can see what I mean."

Two demonstration and practice items were then completed, first in verbal modality :

"Put the palm of your hand on the table top", "Make a closed fist with your hand."

This was followed by two items in the copying/visual modality :

"Copy this" (tester places palm on table top),

"Copy this" (tester makes a closed fist).

The first test used in the examination procedure was that from Haaland and Flaherty (1988) as it was considered simple and straightforward for the patient to start with. The test used gestural performance of meaningful and non-meaningful movements in both personal and peripersonal space to both verbal command and visual (copying) instructions (see appendix D2). Scoring was made on a three-point scale according to observer judgment of the quality and content of movement performance following Raade et al (1991) error-type categories (Table 2.1). The scores were given as 2 for correct performance, 1 for partially correct or identifiable performance, and 0 for incorrect performance (De Renzi et al 1980, scoring guidelines).

The second test carried out was adapted from De Renzi et al (1980) and Haaland and Flaherty (1984), and involved pantomimed and real use of six single common objects (see appendix D3). The test was repeated three times with a different modality used for each one :

1. *Verbal modality*. No object was visible. The instruction given was:

   "Pretend to hold a ..... and show me how you would use it", for each of the test objects.

2. *Visual modality*. Each of the objects was placed in turn on the table at a standard distance from the test participant, and the instruction given was :

   " Show me how you would use this".

If the person made to reach out and touch the object, additional instructions were given to merely look at the object and, again, just *pretend* to hold it and use it.

3. *Tactile modality* or real use of the object. In this condition the participant was given the object and asked to demonstrate how he/she would use it.

Scoring was on a 2, 1, 0 scale as for the first test used.

The third test carried out during the examination procedure involved screening for ideational apraxia and was taken from De Renzi and Lucchelli (1988). Six complex sequencing tasks using multiple objects comprised this test (see appendix D4). During the test, if the clinical subject did not have the physical ability to manipulate the objects bimanually, the examiner assisted by steadying the object or helping complete the started action (e.g. opening the envelope to allow the letter to be placed inside once the patient indicated this was what he/she was attempting to do). The patient was also prompted and guided towards objects if he/she appeared to be struggling in the task, but was not prompted or instructed how to actually perform the task. These guidelines arose from those suggested by de Renzi and Lucchelli (1988) to be used in carrying out the assessment test.

The fourth test used in the research protocol was a screening test to ensure that patients tested were able to recognise objects. This was done to ensure that any errors made during the pantomimed use of objects test (appendix D3) was not due to a visual agnosia; of not knowing what the objects were for. It was placed at this point in the examination procedure to give patients a rest from movement tests and provide a different kind of stimulation. This test was adapted from an original devised by Rushworth (University of Oxford, Department of Experimental Psychology) and comprised twenty pages, on each of which were three black and white photographs of common objects. In each set of three pictures the objects were similarly shaped and had similar orientations (see appendix E for examples). Two of the three objects shown on

each page were similar in their use, with the third being the 'odd one out'. The pages were presented to the patient in turn and instructions given :

> "Look at the pictures in front of you. There are photographs of three objects. Point to the one that you think is the odd one out, or is different in some way from the other two."

(e.g. knife, fork, *toothbrush.* - cabbage, *football,* cauliflower. - *frying pan,* table tennis bat, tennis racquet). Scoring was on a right/wrong basis with 1 point for each correct answer. It was considered that pointing to the correct picture might indicate that agnosic problems were not influencing the results of the apraxia tests, though it must be noted that this was not a validated or standardised screening tool.

The fifth and last test used in the examination for apraxia was the movement imitation test as described by De Renzi et al (1980) and consisted of 24 items of symbolic and non-symbolic movements. Half of the movements involved independent finger activity and half used gross hand and arm movements. The items also involved either holding static positions or executing active movements (appendix D1). This test only used the visual/copy condition with scoring, as suggested by De Renzi et al (1980), on a four point scale of 3, 2, 1, 0, depending on whether a correct performance was elicited on first, second, or third presentation or was totally incorrect. The maximum score was, therefore, 72.

Inter-rater Reliability

To test the accuracy and reliability of apraxia identification through these observational tests, six patients were assessed with the researcher and Rushworth (DPhil. student at the

University of Oxford) scoring independently using the same test battery (Table 4.1). Scoring of patient performance by the two observers was shown to have a high correlation particularly in the movement imitation test (Haaland & Flaherty 1988 - appendix D2) and the single object test (De Renzi et al 1980, Haaland & Flaherty 1988 - appendix D3) where r= 0.99 for both tests. Such concordance of judgment was undoubtedly due to the simple nature of the three category decision required (essentially - normal, impaired, or unrecognisable). The more complex movement imitation test (De Renzi et al 1980 - appendix D1) also provided a high level of agreement although the scoring was not quite as close as the previous two tests (r= .96). The two researchers

Table 4.1 Inter-rater reliability data

Movement imitation test Max. Score = 40 (appendix D2)		**Single object test** Max. Score = 36 (appendix D3)		**Movement imitation test** Max. Score = 72 (appendix D1)	
Rater 1	**Rater 2**	**Rater 1**	**Rater 2**	**Rater 1**	**Rater 2**
36	38	35	35	70	72
28	28	22	21	60	62
30	31	20	20	57	55
33	33	17	17	57	58
27	27	19	20	52	50
36	36	27	27	49	52

observed patients in 'real time' and not via videotape and were thus not able to discuss the reasons for scoring, nor the nature of the errors made, actually during the performance. Retrospective discussions relying on memory for events and written comments/observations made during the assessment process were inadequate for a thorough and rigorous comparison of rater judgments. This was unlike the reliability studies in McDonald et al (1994) and the consensus diagnosis in Rothi et al (1997). The

two raters were, though, in total agreement in the identification of the presence or absence of apraxia in each of the patients assessed.

4.5 Timed tasks

During the single case investigations a variety of timed tasks were used as outcome measures. An officially calibrated stopwatch (Road Time Trials Council certificate) was used for accurate timing. A countdown protocol was devised to remove, as far as possible, any researcher reaction-time variable from the data. The patient was informed of the procedure to be used in starting off the task. The watch was started, the patient given warning of the time and then the instruction "Five, four, three, two, one, go" was given as the seconds counted down. The patient was requested to begin the task on the "Go" instruction.

4.6 Data management.

Conventional statistical analysis (t-tests, ANOVA, correlation statistics) was completed for the group study. The Bonferonni and Student-Newman-Keuls post hoc tests were used in the analyses of variance throughout the research data in this thesis with $p < .05$ as the level of acceptance. These tests were considered to offer an acceptable level of stringency, though not quite as conservative as the Sheffe correction, and are commonly used in the neuropsychological literature.

Analysis of data in the single case design aspects of this thesis needed special consideration. Visual inspection is the most commonly used method of examining single case data, looking at graphical displays across the different phases of the experiment (Sunderland 1990, Bush &

Marascuilo 1992) and this method of analysis is reported throughout the neuropsychological rehabilitation literature (de-Weerdt et al 1989, Nickels et al 1991, Edmans & Lincoln 1991, Byng et al 1994) although Edmans and Lincoln (1991) recognised the differing opinions concerning single case analysis by explaining that due to the serial dependency in their data the use of t and F tests were inapplicable, and that

"results reported are based on visual inspection of the graphs and are purely subjective" (pp 144).

Other authors (Parsonson & Baer 1992), too, are of the opinion that there is value and reliability in plotted data and visual analysis in single case research.

Opinion differs, however, as to whether this is the best and only method of analysis. For example Barlow and Hersen (1984) suggested that reliance on such visual screening methods leads to greater likelihood of making a Type II error (i.e. concluding that the intervention did *not* produce a true effect) than if reliance were placed upon statistical analysis. Similarly Morley and Adams (1991) argued that some authors "extol the virtues of visual analysis of the data (and) argue that if there is an effect it will be very obvious" (pp. 97) yet the subjectivity of such analysis (the position and orientation of the person doing the analysis) determined which features of the graphs were highlighted. The complexity of the issues in analysis of single case design was highlighted by Brodie et al (1994) who concluded that, by and large, researchers in small n designs have ignored statistical analysis of the data. Morley and Adams (1989 and 1991) have shown, however, how both graphical displays and statistical analysis on single case data can be achieved. Time series analysis is a common method used for single case analysis, though such techniques are not always applicable as criteria for such analysis include having large numbers of data points in each phase of the design as well as equal intervals between

those data points (Barlow and Hersen 1984). Within this Doctoral study the particular data sets fulfilled neither of these criteria (having small numbers of data points and unequal intervals between those data points) so time series analysis techniques could not be performed.

An addition made to the straightforward graphical displays and visual inspection in single case design can aid interpretation of data. When there is variability in the data, and a non-obvious visual difference between phases of an ABA design as displayed by the graphs, then examination of the regression lines within each phase can highlight phase differences (Morley & Adams 1991, Prada & Tallis 1995). It is argued, however, that such analysis is only appropriate if the regression lines are meaningful as a predictor of one variable, given the value of another variable (Munro & Page, 1993, Bryman & Cramer 1994), though low predicting regression lines could still be considered valuable in the judgement of change in the data over time. When regression lines in single cases are not considered meaningful nor predictive (low correlation *r* figures) then the data can be assessed for serial dependency using autocorrelation statistics. Such a procedure is not without its problems. It is considered by most authors that there is a high likelihood that data collected from any individual over time will show a degree of serial dependency (Brodie et al 1994) although Barlow and Hersen (1984) caution that "serial dependency is not a *necessary* characteristic of single-case data or observations over time" (pp 290).

These opposing views highlight the differences of opinion in statistical approaches to single case analysis and the difficulties in satisfying peer evaluation and critique in single case research. Although Barlow and Hersen (1984) stated that data revealing no serial dependency

> "can be treated as independent observations and can be subjected to conventional statistical analyses" (p. 288).

most authors would be reluctant to accept an assumption of such data points being totally independent as failure to *show* serial dependency in the data does not mean that serial dependency is not there, merely that it has not been found.

Given the debate and differing opinions in the literature regarding analysis of single case data this doctoral research has presented a combination of analyses. Graphical displays of the data sets have been visually analysed with support (or otherwise) for apparent trends in the data through linear regression analysis. Supportive descriptive statistics using means, standard deviations, broadened medians, range of scores and inter-quartile ranges have also been used to explore the differences and similarities in phases of the ABA designs in each single case presentation to consider whether differences between the experimental phases could be confidently stated or not, and to determine intervention effects.

Chapter 5.

KINEMATIC ANALYSIS OF APRAXIC MOVEMENT PERFORMANCE .

5.1 Methodology

5.1 [i] Subjects

Clinical participants with unilateral left hemisphere lesions were recruited for the research as described in chapter 4.2 and assessed for the presence of apraxia at the rehabilitation centre following normal consent procedures. 30 clinical cases were assessed, 17 of which were identified as apraxic and 13 as non-apraxic. This selection into the apraxic/non-apraxic categories was based mainly on the cut-off score of less than 68 on the De Renzi et al (1980) movement imitation test, but also with a criterion of a reduced score on at least one other test. Normal control subjects were tested and recorded at the School of Occupational Therapy, Oxford Brookes University. Clinical and demographic data of participants are given in Table 5.1.

A total of 11 healthy controls were, as a group, matched as near as possible in age with the apraxic group as this was considered to be potentially more influential in movement than gender. There was, however, a significant difference in age between the non-apraxic group and the matched-for-age apraxic and control groups ($F=6.19$ $2,38$ $p< .01$). Covariant analysis indicated, though, that age was not an influencing factor in performance in the clinical tests. There was an unequal distribution of male and female subjects in both the non-apraxic and control groups largely due, in the case of the clinical group, to the chance factors involved in admission to the rehabilitation centre. As a consequence of difficulties in recruitment and the pragmatics of testing, it was not possible to balance groups by gender.

Handedness was also seen to be unevenly distributed between groups. The apraxic group were all right handed, while five of the 13 non-apraxic group were left handed. This might be an artifact of the experiment or it might bear consideration when linking location of praxis with hemispheric functioning. Virtually all subjects in the clinical groups had some degree of language impairment. Only two of the non-apraxic group were considered to have an intact language system. Both these patients were also left handed.

Table 5.1 : Group study - Summary of clinical and demographic data.

	Apraxic group	Non-apraxic group	Controls
Number in group	17	13	11
Age (Years) : Mean	55.82	46.31	57.36
SD	7.35	9.39	9.55
Range	45 - 69	27 - 60	45 - 72
Gender	8 male. 9 female.	10 male. 3 female.	3 male. 8 female.
Handedness	17 right. 0 left.	8 right. 5 left.	10 right. 1 left.
Language function	17- affected. 0 - OK.	11 - affected. 2 - OK	0 - affected. 11- OK.
Lesion location	5 - MCA	2-MCA	11 normal
	1 - ACA	2 -ACA	
	3 - SAH	1 - SAH	
	1 - parietal lobe	2- parietal lobe	
	1 - internal capsule	2 - internal capsule	
	0 - basal ganglia	2 - basal ganglia	
	6 - 'other'	2 - 'other'	
Time since onset (in weeks)			N/A
Mean time	19.53	17.73	
Range	8 - 52	10 - 30	
SD	11.16	6.1	
Barthel score			full
Mean	10.4	14.23	
Range	1 - 20	5 - 20	
SD	4.75	4.95	

Key : MCA - middle cerebral artery, ACA - anterior cerebral artery, SAH - subarachnoid haemorrhage

The lesion location in both clinical groups was taken from CT scan reports. The level of detail in these reports was usually minimal and consequently of little use, in research terms, in trying to relate such lesions to the characteristics of the apraxia. The category 'other' given in table 5.1 includes several instances of unknown location, or when 'left hemisphere lesion' was the only evidence in medical notes of the lesion. So initial aims to relate apraxic movement errors to lesion site were, therefore, modified.

Although there was a wider range of time since onset in the apraxic group this was due to the inclusion of a single patient assessed 52 weeks after the cerebrovascular accident. Despite this, the time since onset of the cerebral insult was not shown to be statistically different between the apraxic and non-apraxic groups [$t(30) = -.52$ *NS*]. *Without* the distorting single case, however, the apraxic group's range of time since onset was 8-29 weeks (mean = 17.21, SD = 6.87) and matches well with the 10-30 weeks of the non-apraxic group (mean = 17.73, SD = 6.1).

The Barthel ADL (activities of daily living) Index is a measure of the degree of independence in an individual (Wade & Collin 1988, Wade 1992) with a maximum possible score of 20 indicating full independence in all major areas (bowels, bladder, grooming, toilet use, feeding, transfer, mobility, dressing, stairs and bathing). Wade (1992) reported the widespread use of the Barthel index with its well-established validity and reliability. These scores are routinely taken at Rivermead Rehabilitation Centre and so were available to the researcher from the medical notes. There was shown to be a significant difference between the apraxic and non-apraxic group in their levels of independence as measured by the Barthel scores [$t(30) = 2.09$ $p < .05$].

5.1[ii] Procedure

All clinical participants were assessed in the same room at the Rehabilitation Centre though at different times of the day as this was dependent upon their individual rehabilitation timetable and availability. Issues associated with fatigue were considered, but it was concluded that any effects were likely to partial out between groups. Also all patients were asked, at time of testing, whether they felt able to participate and were not too tired. Each person was seated at a table (height 28 inches) either in a standard chair (floor to seat height : 16 inches) or a wheelchair (according to his level of physical independence in being able to transfer safely from wheelchair to chair). During assessment procedures the researcher was seated directly opposite the person. The procedure was explained again to the participant before starting the assessment to remind him/her of the information given previously and also to reassure him/her of the nature of the activities, the time involved and his/her contribution.

5.1[iii] Apraxia assessment

The selected five apraxia tests (previously described in chapter 4.4 and given in appendices D1-D4) were administered to each participant of the research. Clinical participants and normal controls were requested to perform the tasks with their left hand. For the clinical group this was to exclude any effect due to hemiparesis or sensory impairment. Following the clinical assessment tests, the procedure (described in chapter 4.3) for kinematic recording of movement during a drinking task was carried out. There was one trial in each of the three modality conditions; verbal command, visual imitation and real object use.

5.2 Results

5.2[i] Clinical assessments.

The first analysis completed was an evaluation of the apraxia assessments used in the research, their relationship to other research variables and the differences between experimental groups. A visual inspection of the raw data scores in the apraxic group indicated an inconsistent pattern across the tests for ideomotor apraxia (Table 5.2).

Table 5.2 Apraxic group: scores on apraxia tests

Case	Movement imitation test (appendix D2) Max. Score 40	Single object test (appendix D3) Max. Score 36	Movement imitation test (appendix D1) Max. Score 72	Ideational apraxia test (D4) Max. Score 12
1	28	22	60	9
2	30	21	57	7
3	33	17	57	7
4	27	19	52	12
5	36	27	49	11
6	21	11	39	10
7	39	34	42	10
8	19	23	48	11
9	23	19	53	10
10	27	16	67	9
11	26	25	42	10
12	31	24	52	9
13	20	9	59	6
14	34	13	62	12
15	20	27	52	10
16	32	16	49	9
17	22	12	37	9

These assessments (De Renzi et al. 1980, Haaland & Flaherty 1984. Appendices D1, D2, and D3) chosen for identification of apraxia were, therefore, analysed to determine whether or not each clinical test score could be shown to statistically correlate one with another (Table 5.3). None reached significance level and would, therefore, seem to be

tapping into different aspects of the condition or to quite different underlying features of performance. This lack of association between the different ideomotor assessment scores is an important point in consideration of sub-types of apraxia and will be raised in the discussion section 5.3 of this chapter. Furthermore, in the apraxic group, none of the ideomotor apraxia tests correlated with the tests for ideational apraxia, agnosia or with the score on the Barthel Index for independence (table 5.3).

Table 5.3 : Group study - Clinical assessment scores; correlation data.

Clinical Tests	Apraxic Subjects only
a] Ideomotor apraxia tests	
Test 1 (appendix D2) **with Test 2** (appendix D3)	r = .46 (*NS.* n=17)
Test 1 (appendix D2) **with Test 5** (appendix D1)	r = .005 (*NS.* n=17)
Test 2 (appendix D3) **with Test 5** (appendix D1)	r = - .23 (*NS.* n=17)
b] Ideomotor apraxia tests with other clinical tests.	
Test 1 (appendix D2) **with Test 3** - ideational apraxia (appendix D4)	r = .09 (*NS* n=17)
Test 1 (appendix D2) **with Test 4** - agnosia (appendix E)	r = - .35 (*NS.*) n=17)
Test 1 (appendix D2) **with Test 6 : Barthel Index**	r = .35 (*NS.* n=17)
Test 2 (appendix D3) **with Test 3** - ideational apraxia. (appendix D4)	r = .29 (*NS* n=17)
Test 2 (appendix D3) **with Test 4** - agnosia (appendix E)	r = - .09 (*NS.* n=16)
Test 2 (appendix D3) **with Test 6 : Barthel Index**	r = -.25 (*NS* n=17)
Test 5 (appendix D1) **with Test 3** - ideational apraxia. (appendix D4)	r = -.23 (*NS.* n=17)
Test 5 (appendix D1) **with Test 4** - agnosia (appendix E)	r = .37 (*NS.* n=16)
Test 5 (appendix D1) **with Test 6 : Barthel Index**	r = .42 (*NS.* n=17)

The clinical assessment scores for the three experimental groups were then analysed to determine the extent to which the apraxic and non-apraxic groups differed and whether the non-apraxic and control groups were essentially the same. There were significant differences between the groups in scores on the clinical apraxia tests (Table 5.4) with the post hoc tests indicating (at $p < .05$) that the major difference lay between the apraxic group and the other two groups (non-apraxic and controls). This is not surprising as the clinical groups themselves were chosen on the basis of the clinical test scores.

There were no significant differences shown in any of the apraxia test scores between the normal control group and the non-apraxic group (Table 5.4). The range of scores within each of the three experimental groups shows that, for most tests, some overlap occurred between groups. This is explained by patients performing reasonably well on one kind of test for apraxia, yet doing particularly badly on another adding support to the rather heterogeneous nature of the apraxic condition. In order to quantify the accumulated evidence of ideomotor apraxia provided by the three tests (appendices D2, D3 & D1), the scores were converted into percentages. For each subject the mean of these scores was then taken to give a percentage score for ideomotor apraxia. The differences between the brain-damaged groups when this formulation was completed, demonstrated a clear demarcation with no overlapping within the score ranges (see table 5.4). Analysis of variance on these scores indicated highly significant differences between groups (unless otherwise indicated, the significance level accepted for all group differences was $p < .001$) with post hoc tests indicating that the difference lay between the apraxic group and the other two (non-apraxic and control) groups. Unless otherwise indicated, the significance level accepted for post hoc tests throughout this doctoral study was $p < .05$.

Table 5.4 : Group study - Clinical assessment scores; differences between groups.

	Apraxic	Non-apraxic	Controls
Test 1 (max. = 40) Ideomotor apraxia			
(See appendix D2)	**F (2,38) = 35.32**	**p< .001**	
Mean	27.53	39.15	39.91
Standard Deviation	6.8	1.52	0.3
Range	19 - 39	36 - 40	39 - 40
Test 2 (max. = 36) Ideomotor apraxia			
(See appendix D3)	**F(2,38) = 60.43**	**p< .001**	
Mean	19.53	34.46	36
Standard Deviation	6.59	2.5	0
Range	9 - 34	29 - 36	36
Test 5 (max. = 72) Ideomotor apraxia			
(See appendix D1)	**F(2,38) = 61.08**	**p< .001**	
Mean	51.82	70.23	70.82
Standard Deviation	8.03	1.64	1.25
Range	38 - 67	68 - 72	69 - 72
Mean % scores for Tests 1, 2 & 5.			
	F(2,38)= 110.69	**p< .001**	
Mean	65.01	97.05	99.38
Standard Deviation	10.23	4.06	0.62
Range	46.2 - 83.42	88.33 - 100	98.61 - 100
Test 3 - ideational apraxia (max. = 12) (See appendix D4)			
	F(2,38) =18.15	**p< .001**	
Mean	9.47	11.53	12
Standard Deviation	1.66	0.97	0
Range	6 - 12	10 - 12	12
Test 4 - agnosia (max. = 20) (See appendix E)			
	F(2,38) =10.52	**p< .001**	
Mean	14.25	18.62	19.73
Standard Deviation	4.67	2.66	0.47
Range	4 - 20	12 - 20	19 - 20
Test 6 : Barthel Index for independence (max. = 20)			
	F(2,38) =17.27	**p< .001**	
Mean	10.4	14.23	20
Standard Deviation	4.75	4.95	0
Range	1 - 20	5 - 20	20

The test for *ideational* apraxia also showed significant differences across groups (see table 5.4). The post-hoc tests again indicated that this difference lay between the apraxic group and the other two (non-apraxic and control) groups at $p < .05$. It was clear from the clinical test scores that the apraxic group contained a proportion of people with *both* ideomotor *and* ideational apraxia. A clinical judgment was made regarding a diagnosis of ideational apraxia based upon a score of 9 or less (from a maximum of 12) on the multiple-objects sequencing test (De Renzi & Lucchelli 1988 - appendix D4). Referring to Table 5.2 therefore, cases 1, 2, 3, 10, 12, 13, 16 and 17 would be considered to show evidence for ideational apraxia. When this sub-group were compared with the rest in the apraxic group (scoring 10 and over in the test for ideational apraxia) no significant differences were found in their scoring on the *ideomotor* apraxia tests [movement imitation test D2 $t(15) = 0.19$ *NS.*, single object test D3 $t(15) = -1.56$ *NS.*, movement imitation test D1 $t(15) = 1.47$ *NS*] nor in the agnosia test [$t(15) = -1.44$ *NS*] nor in the Barthel index scores [$t(15) = 1.00$ *NS*]. The two subgroups were, therefore, considered similar enough in all critical elements to make meaningful analyses of their performance on the research tasks as a single group.

The test for agnosia was also seen to be significantly different between groups (Table 5.4); again the post hoc tests indicated that the difference lay between the apraxic group and the other two groups ($p < .05$). Further examination of the agnosia score data revealed that both clinical groups (apraxic and non-apraxic) had people within them who displayed some difficulties with the screening test, though the range and standard deviation of scores within the apraxic group were by far the larger (Table 5.4). In order to establish whether the elements which determined agnosia test performance had also

influenced performance in the other clinical tests, covariant analysis techniques were employed. Covariant analysis (ANCOVA) allows the data to be examined in a way that considers how much influence an additional variable (the covariant) has when considering differences between groups. The extraction of that influence provides a more accurate estimate of the real difference amongst groups (Munroe & Page 1993).

Covariant analysis of these group data indicated that the scoring on the agnosia test did not significantly influence scoring on *most* of the other clinical tests, the group category (apraxic, non-apraxic or control) consistently being demonstrated as the major influencing variable in predicting differences between the clinical scores. In the Haaland and Flaherty (1984) ideomotor apraxia test using both verbal and visual modality conditions (appendix D2) the agnosia test scores were not a significant influence [$F(2,36) = 3.37$ *NS*] whereas the group category was significantly predictive of differences in scores [$F(2,36) = 30.47$ $p < .001$]. Similarly the scores in the test for pantomimed use of objects (appendix D3) were not being influenced by the elements in the agnosia test [$F(2, 36) = 0.11$ *NS*], the main effect being the group category [$F(2,36) = 38.6$ $p < .001$]. The test for ideational apraxia (D4) was also shown not to be influenced by the agnosia components [$F(2,36) = 1.57$ *NS*] but again the diagnostic category of group was a significant influence [$F(2,36) = 8.1$ $p < .01$] though not as strongly predictive as in the other clinical tests. The one clinical test which ***did*** demonstrate having been influenced by the elements in the agnosia screening test was the De Renzi et al (1980) 24 item test (appendix D1). In this test, agnosia test scores were shown to predict scoring on the ideomotor apraxia test [$F(2,36) = 4.7$ $p < .05$] though the group category (apraxic, non-apraxic, or control) remained considerably the better predictor and major influencing variable [$F(2,36) = 32.38$

$p < .001$]. Consideration of the cognitive and perceptual elements within the agnosia test which were shown to be influencing the De Renzi (1980) test for apraxia, which not only was used in this research as the major criterion for diagnosis of ideomotor apraxia but is also the test deemed most robust by researchers (Tate & McDonald 1995), will be made in the discussion section 5.3 of this chapter.

The Barthel score also revealed a difference between groups (see table 5.4); the post hoc tests indicating the difference lay between the control group and the two clinical groups ($p < .05$) as would be expected. This analysis of variance technique might be considered the more robust in looking for differences between groups than the t-test previously employed looking for differences between the apraxic and non-apraxic groups alone [$t(28) = 2.09$ $p < .05$]. Before accepting that the significant difference in independence levels lay between the control group and the two clinical groups it was important to employ covariant analysis techniques to determine how far, if at all, the level of independence of an individual was influencing, and therefore predictive of, scores on the clinical tests for apraxia. As with the agnosia test, covariant analysis revealed that the Barthel score did not significantly influence scoring on *most* of the other clinical tests with the group category consistently being demonstrated as the major influencing factor. The Barthel scores were not significant influences in the Haaland and Flaherty (1984) ideomotor apraxia test [$F(2,35) = 3.65$ *NS*] nor in the single object test (appendix D3) [$F(2, 35) = 0.33$ *NS*], whereas the group category was significantly predictive of differences in scores in both tests [$F(2,36) = 15.49$ $p < .001$ and $F(2,35) = 39.72$ $p < .001$]. The test for ideational apraxia (D4) was also shown not to be influenced by the components of independence [$F(2,35) = 0.94$ *NS*] but again the diagnostic category of

group was a significant influence [$F(2,35) = 7.56$ $p < .01$]. Once more, the clinical test which ***did*** emerge as having been influenced by independence was the De Renzi et al (1980) test. Barthel scores were shown to predict scoring on the ideomotor apraxia test [$F(2,35) = 5.83$ $p < .05$] although, just as before, the group category (apraxic, non-apraxic, or control) remained considerably the better predictor and major influencing variable [$F(2,35) = 37.57$ $p < .001$]. It could be considered, therefore, that the two clinical groups did ***not*** contain the same severity of disability, although they were significantly different from the control group. It could also be argued that although any differences in apraxia scores between the groups could be ***largely*** attributable to the presence of the apraxic condition, the influence of independence level and severity of disability cannot be discounted.

In matching the control subjects to the apraxic group, age had been considered a potential influencing variable in motor performance. To establish whether age did have a significant effect or influence on the clinical test scores, covariant analysis was completed. No significant influences were discovered, attributable to age, in the any of the clinical test scores; F values lower than 0.9 were demonstrated for all ANCOVA analyses. As with the other covariant analyses carried out, the group category was the highly statistically-significant feature predicting differences in *all* clinical test scores ($p < .001$).

As the De Renzi et al (1980) test was considered to be highlighting influencing variables more than the other clinical tests, an investigation into the test content itself was considered worthwhile. There were not enough subjects in the study to undertake a factor analysis, but Cronbach's alpha reliability test would indicate the level of internal

consistency in the clinical tests being used (Bryman & Cramer 1994) . Munro & Page (1993) have pointed out that this method is highly appropriate for instrument development, particularly in the early stages, as it highlights the extent to which items of a test go together and also identifies weak items which might be omitted. An alpha reliability factor of 0.8 or above is considered to be (as a rule of thumb) an acceptable level of internal reliability of additive scales such as were being used in this research project (Bryman & Cramer 1994), although other authors suggest alpha values above 0.9 demonstrate a test being internally consistent but non-discriminatory (Tabachnick & Fidell 1996). The movement imitation test (Haaland & Flaherty 1984, appendix D2) had an α of .72, with the items in verbal command 'hand on nose', 'hand under chin', 'finger on ear' and 'thumb on forehead' contributing the most. Thus, *meaningless* gestures (without a model for the patient to copy) were shown to be the most demanding on the motor system for praxis. The test items 'wave good-bye' in the verbal command and 'hand behind your head' in the copy condition were shown *not* to be differentiating between groups in this study and therefore not contributing much to the scale. In other words they did not provide information that had not been learnt from other more discriminating items in the test. Removal of those weak items from the test raised the alpha value to 0.798 thus enhancing the internal consistency of the scale.

The object/tool use test (appendix D3, from De Renzi et al 1980, Haaland & Flaherty 1984, Alexander et al 1992) had a high α of .85, with all items contributing well to the test except for real use of the pen. In contrast, the test for ideational apraxia (appendix D4. De Renzi & Lucchelli 1988) used with these subjects had a very low α of .185 . The test had *no* internal consistency, but was addressing different aspects of performance with

each individual item rather then revealing a single underlying difficulty. It might be argued, however, that if the number of *errors* made during each test item had been recorded and used as a scoring system (as had been suggested by some research groups) then a more robust finding might have occurred. The 2, 1, 0 scoring used for these test purposes might not have been sufficiently discriminating.

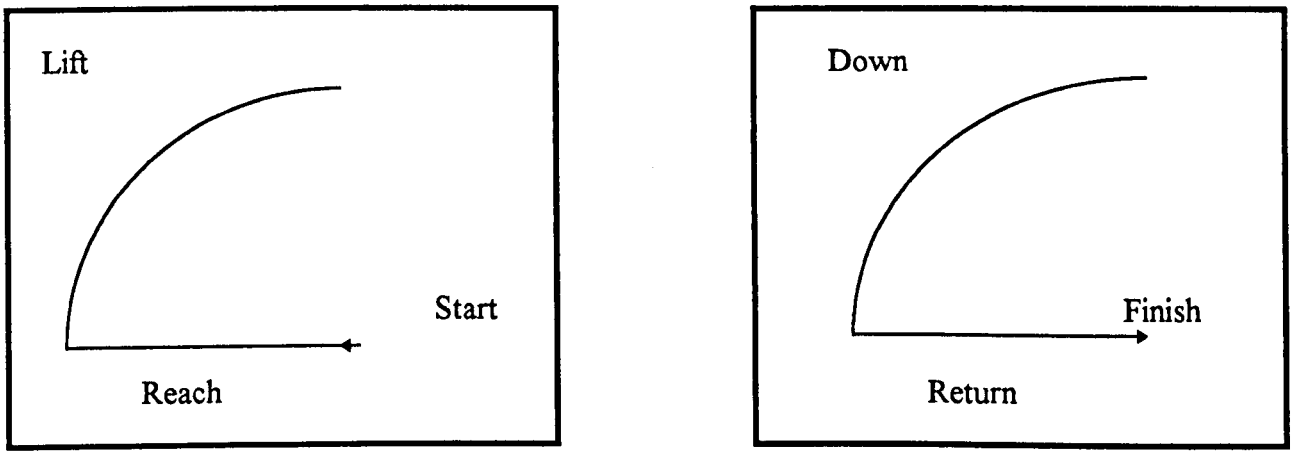
The test devised by the author and a colleague for the screening of agnosia had an α of .93 which is considered, by some workers in psychometrics, to be 'too high' and an indication of uni-dimensionality (Munro & Page 1993). It is considered that such a high α score indicates that the individual test items were so highly correlated one with another that the test would discriminate in exactly the same way with just a few items. This is not to say that in the particular case of the agnosia test it was not measuring something valuable, if uni-dimensional, in the research project but that it could have done so just as well with fewer items. Just what this uni-dimensional factor was, will be addressed in the discussion section 5.3. A development of the test with more discriminating items could be a worthwhile future project.

The best test, in terms of internal consistency, was the gesture copying test (De Renzi et al 1980. Appendix D1) with α of .85. Every item in this test was shown to be contributing to eliciting the underlying dysfunction. It might be argued, therefore, that this was the most sensitive test in the battery and might be the reason that it picked up the influence of other variables like independence and agnosia.

5.2 [ii] Kinematic analysis

Movement analysis of the experimental drinking task was compared between the three groups and also in relation to clinical test scores. Movement during the drinking task was analysed in four phases : 1. reaching out from the starting position to the target 'red spot' on the table, 2. lifting the hand up to the mouth, 3. hand going back down to the table from the mouth, 4. returning the hand from the target 'red spot' on the table to the starting position (see figure 5.1).

Figure 5.1 Illustration of the experimental drinking task .



It should be noted that not all subjects could be included in all aspects of the kinematic analysis due to vagaries and difficulties with the computer software used for analysis of movement recordings, with no recourse to re-recording those subjects at a later date. These practical difficulties of the research are discussed later in the thesis.

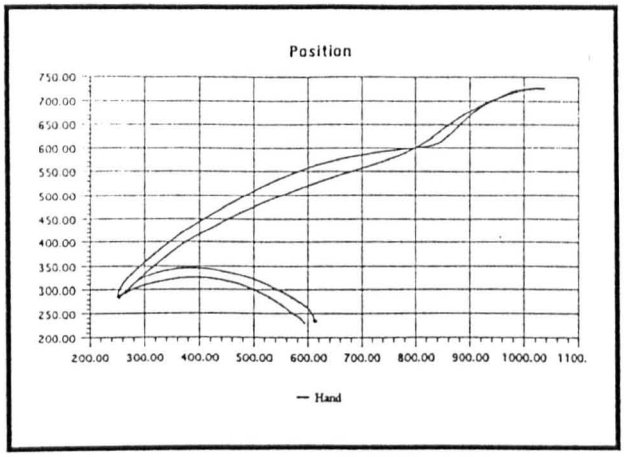
Trajectories :

The trajectories of the control group were seen to be smooth, even and consistent in the spatial pathways executed for both outward and returning phases of the task. This typical

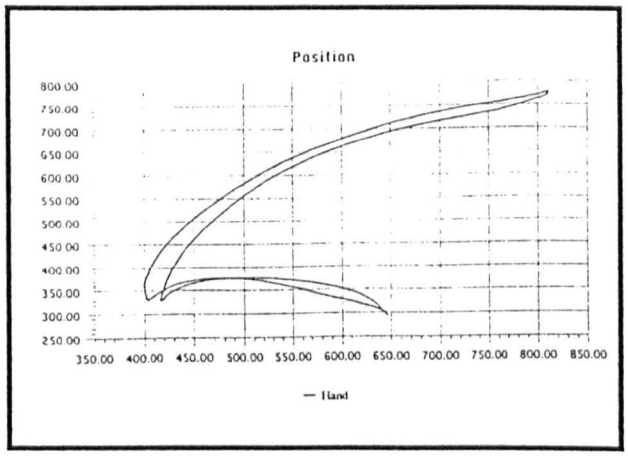
trajectory is illustrated in Figure 5.2 (a & b) in contrast with an apraxic hand trajectory Figure 5.2 (c & d). Non-apraxic trajectories are also illustrated.

Figure 5.2 : Examples of *whole* movement trajectory of the hand during the drinking task.

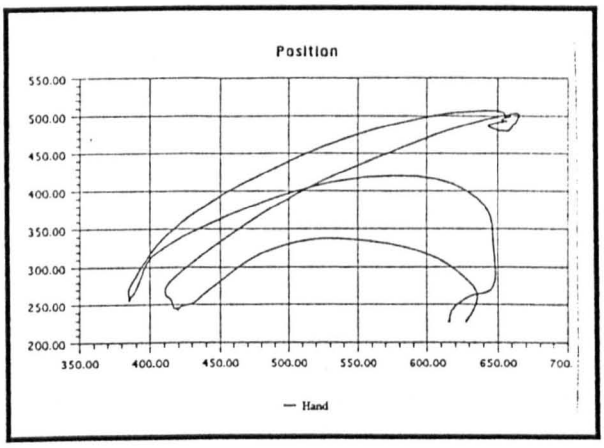
a] Control subject.



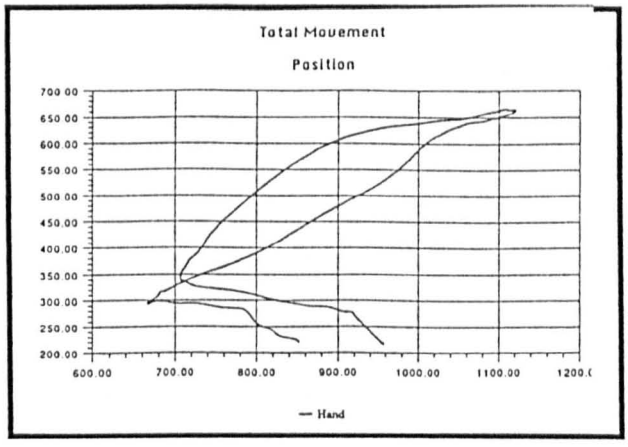
b] Control subject.



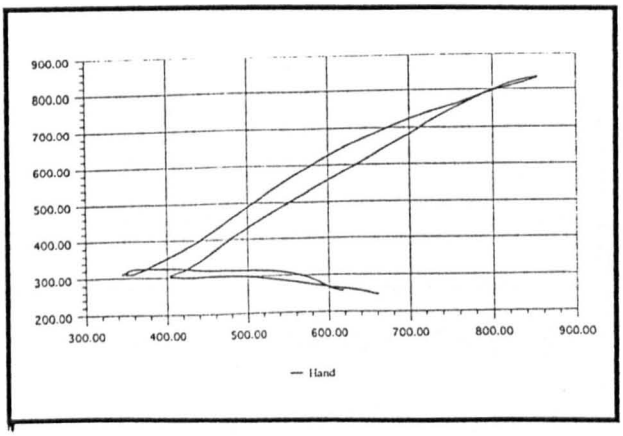
c] Apraxic subject.



d] Apraxic subject.



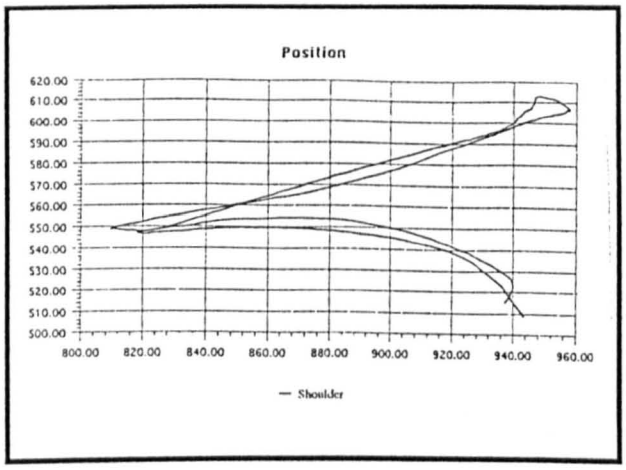
e] Non- apraxic subject.



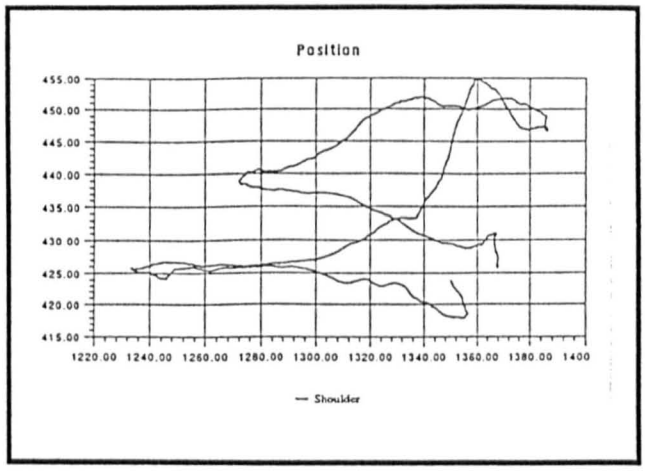
This efficient and smooth movement in the control group was also illustrated in the use of the shoulder during the drinking task, where a distinct pattern was traced by the infra-red marker as shown in Figure 5.3 (a & c), but again contrasts with the shoulder movement elicited in the apraxic subjects - Figure 5.3 (b & d). Non-apraxic shoulder trajectories are illustrated in Figure 5.4.

Figure 5.3 : Examples of the *whole* movement trajectory of the shoulder during the drinking task.

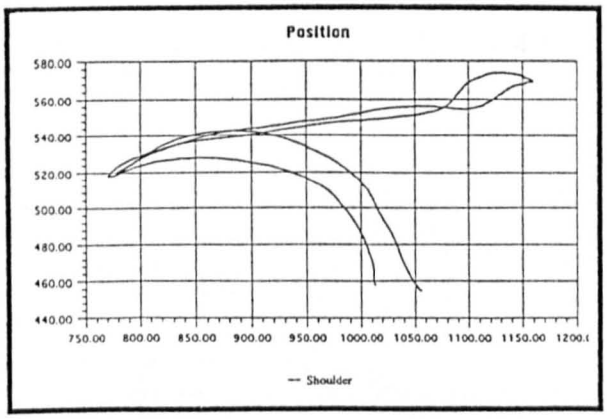
a] Control subject.



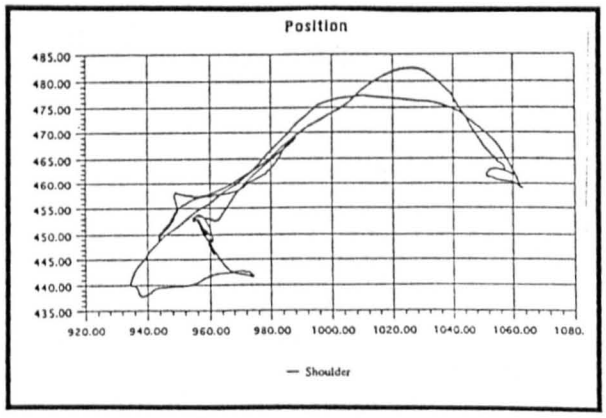
b] Apraxic subject.



c] Control subject.



d] Apraxic subject.

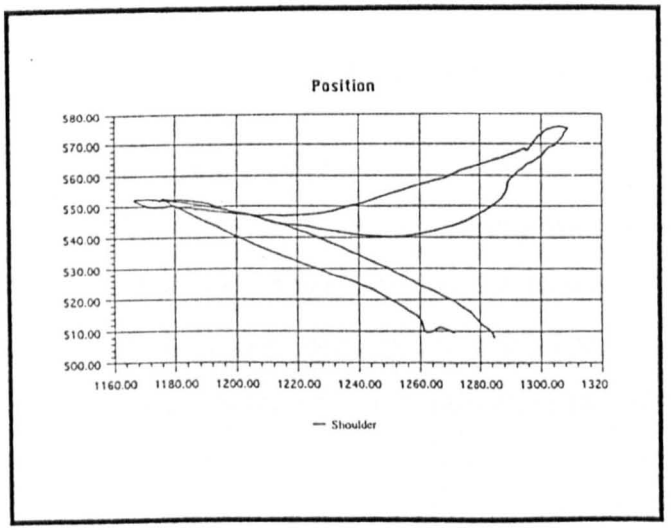


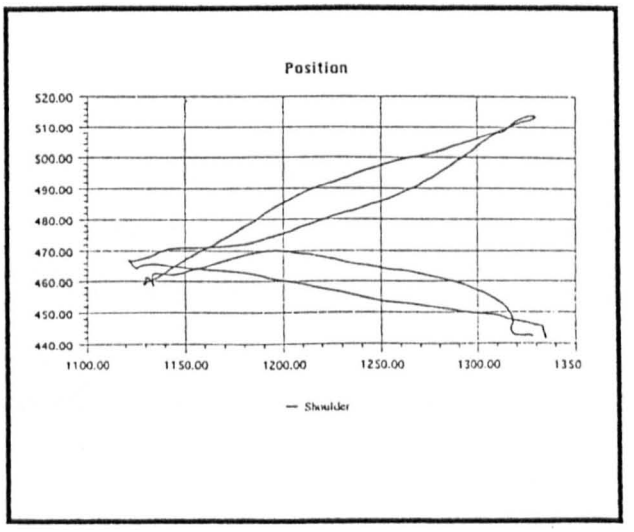
As illustrated, the visual representation of the trajectory recordings for the apraxic group were markedly different from those of the control group. All subjects were able to

complete the task in all modality conditions, but their inability to create a smooth, coordinated and well-modulated movement output was evidenced by their markedly different trajectory recordings.

The people in the apraxic group demonstrated an irregular and somewhat hesitant pattern of movement in the whole of the task, with an inconsistency of pathway of movement in space during the outward and return elements of the drinking task (Fig. 5.2). They did not show the efficiency of movement seen in the control group. One of the most marked features of the apraxic group kinematic recordings was seen in the shoulder movement. As illustrated in Figure 5.3 the apraxic group demonstrated grossly irregular patterns of shoulder movement in comparison with that shown by the control group. The non-apraxic group on the other hand showed very similar trajectory recordings to those of the control group with a general smoothness and efficiency of movement pathway. Some people in the non-apraxic group did, however, show some of the inconsistent elements of shoulder movement demonstrated by the apraxic subjects though not to the same degree. Examples of these shoulder trajectories are given in Fig. 5.4

Figure 5.4 : Examples of the non-apraxic group shoulder movement trajectory

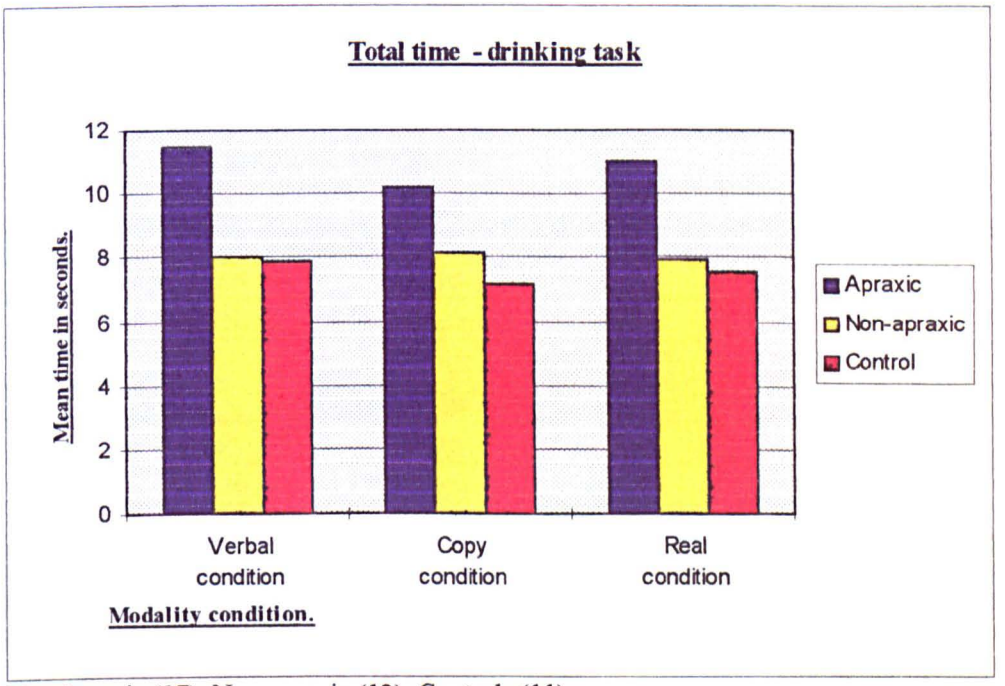




Time taken :

The total time taken to complete the drinking task was shown to be significantly different ($p < .05$) between the three experimental groups in all modality conditions as illustrated in Figure 5.5 with the details given in Table 5.5. This completion time was taken from the *actual* movement, reaction/initiation time not being included in the measurement. The non-apraxic group was seen to be very similar in task completion time to the control group of subjects. In all conditions the control group had the quickest mean time with

Figure 5.5: Group study - Time taken for completion of drinking task.



N = Apraxic (17). Non-apraxia (13). Controls (11)

the smallest standard deviation, showing a marked consistency of performance across subjects. The non-apraxic group showed similar mean scores to the control group but with a much wider range of scores and a correspondingly larger standard deviation. The apraxic group had the slowest mean scores, the largest standard deviation and the greatest range of times, indicating the widest variability between subjects (Table 5.5).

conditions lay between the apraxic group and the other two (non-apraxic and control) groups ($p < .05$).

Reaction times :

Reaction time as measured by initiation of movement from the 'Go' command was analysed across groups and across modality conditions (Table 5.5). The apraxic group, once again showed the slowest mean reaction times, the largest standard deviations

Table 5.5 : Synopsis of timing data for the experimental drinking task.

Total time (in secs.).	**Apraxic**	**Non-apraxic**	**Controls**
Verbal condition.			
[$F(2,37) = 4.96$ $p < .05$] Mean	11.5	8.02	7.92
Standard Deviation	4.29	3.43	1.78
Range	3.4 - 18.5	2.52 - 14.36	5.44 - 10.8
Copy condition			
[$F(2,37) = 3.72$ $p < .05$] Mean	10.19	8.15	7.20
Standard Deviation	3.64	2.95	1.43
Range	4.74 - 19.1	3.2 - 13.86	5.04 - 9.32
Real condition.			
[$F(2,37) = 4.4$ $p < .05$] Mean	11.02	7.95	7.59
Standard Deviation	4.61	2.67	1.23
Range	4.38 - 23.18	4.56 - 12.44	5.72 - 9.82
Reaction time(in secs.).			
Verbal condition.			
[$F(2,37) = 1.59$ *NS*] Mean	0.63	0.28	0.27
Standard Deviation	0.87	0.38	0.24
Range	0.02 - 3.18	0.02 - 1.2	0.04 - 0.84
Copy condition.			
[$F(2,37) = 1.08$ *NS*] Mean	0.4	0.26	0.26
Standard Deviation	0.37	0.24	0.24
Range	0.02 - 1.06	0.02 - 0.7	0.02 - 0.8
Real condition.			
[$F(2,37) = 3.27$ $p < .05$] Mean	0.35	0.10	0.26
Standard Deviation	0.34	0.10	0.2
Range	0.02 - 1.22	0.02 - 0.32	0.02 - 0.62

across all conditions and the broadest range of responses when compared with the non-apraxic and control groups. No statistically significant difference was found in either the verbal or the visual modality conditions (Table 5.5). A significant difference was found, however, in the reaction time for real object use between the apraxic and the non-apraxic groups [$F(2,37) = 3.27$ $p < .05$]. Interestingly this was the only instance where the non-apraxic group performed better than the control group in terms of speed and minimal range of scores across subjects.

Task phases :

The different *phases* of the drinking task were also examined for group differences across modality conditions. Figure 5.6 illustrates clearly how for each phase of the drinking task in the verbal condition, the apraxic group took longer than the other two groups. There was little demonstrable difference between the non-apraxic and control groups. This same pattern was repeated across the other two modality conditions (see Figures 5.7 and 5.8) with a larger standard deviation shown by the apraxic group (Table 5.6).

Figure 5.6 : Phases of the drinking task. Group differences in verbal condition.

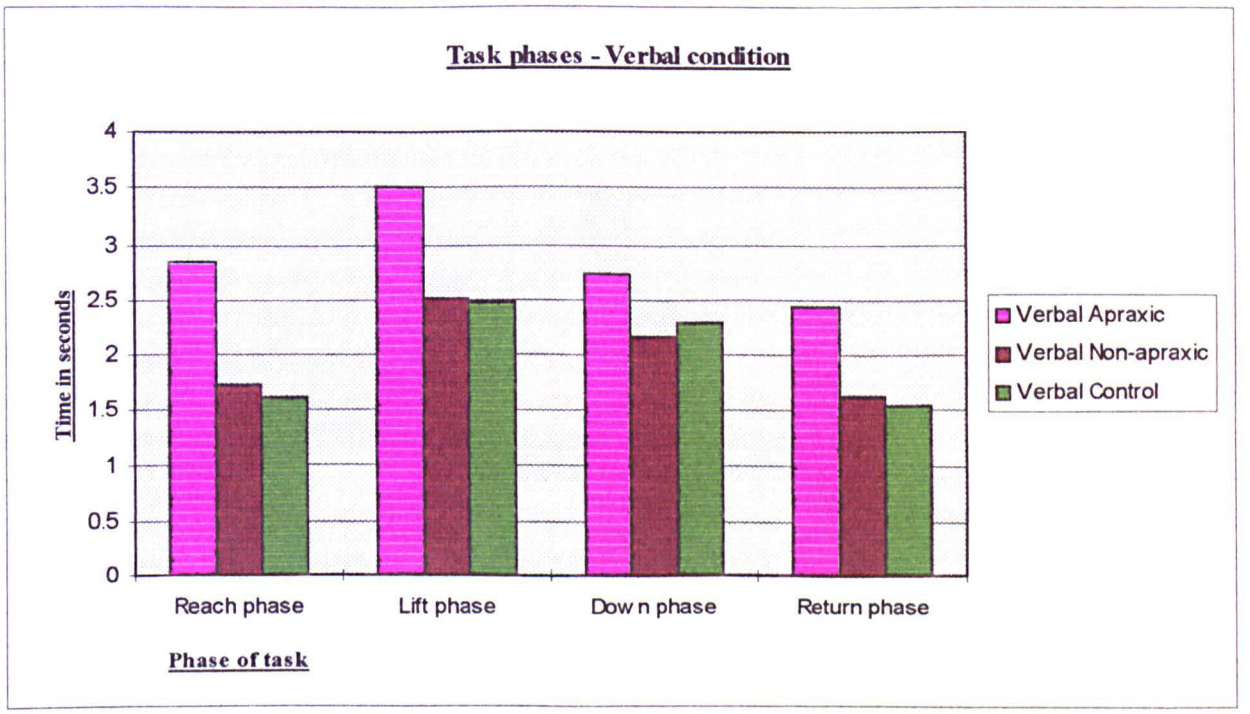


Figure 5.7 : Phases of the drinking task. Group differences in visual/copy condition.

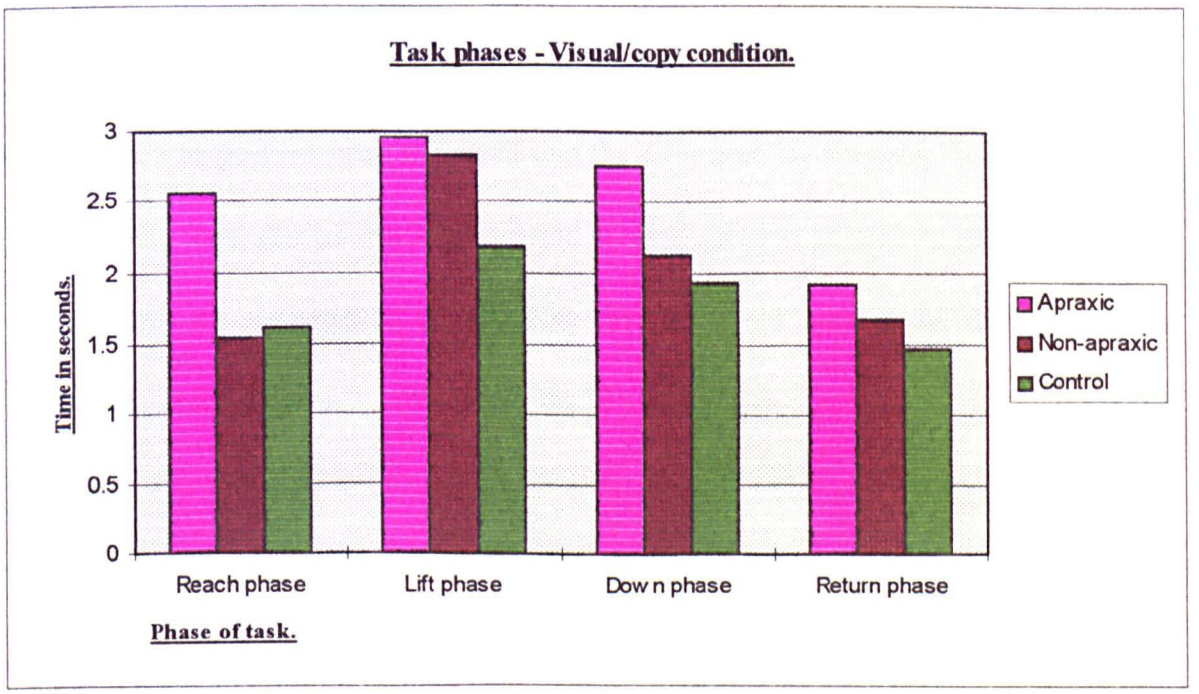
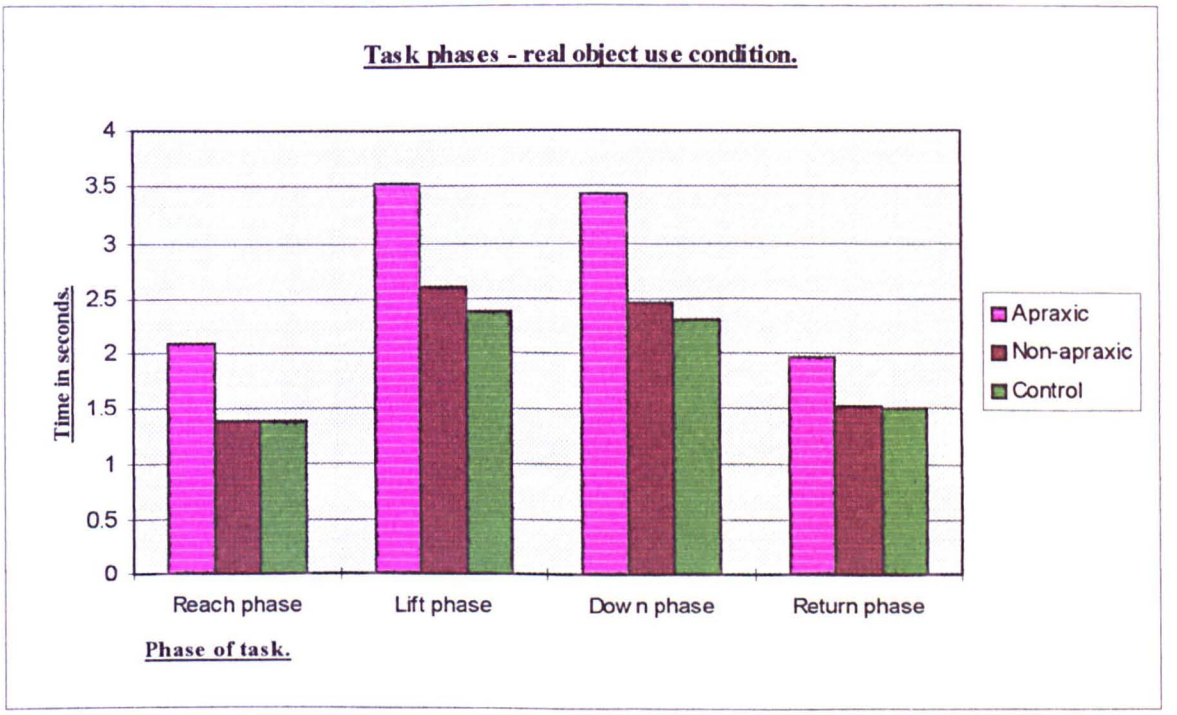


Figure 5.8 : Phases of the drinking task. Group differences - real object use.



As detailed in Table 5.6, the reach phase of the drinking task evoked significant differences between groups in the time taken to complete that phase in all modality conditions. The post hoc tests indicated that the difference lay between the apraxic group and the other two groups (non-apraxic and control groups) in each condition ($p < .05$). The lift and down phases of the drinking task were not seen to be different across conditions or across groups, apart from the real object use condition. Here a significant difference was found between the apraxic group and the other two groups in the 'down' phase timing (Table 5.6). The return phase of the task elicited differences between groups in both the verbal and real object conditions of the experiment (Table 5.6) with the post hoc tests indicating that the apraxic group showed the performance deficit.

Table 5.6 : Group differences in time taken in phases of drinking task.

	Verbal condition.			Copy condition			Real condition		
	Apraxic	Non-apraxic	Controls	Apraxic	Non-apraxic	Controls	Apraxic	Non-apraxic	Controls
Reach phase (in secs.)	**F= 3.97 2,37**	**p< .05**		**F= 7.88 2,37**	**p< .01**		**F= 4.15 2,37**	**p< .05**	
Mean	2.85	1.72	1.62	2.57	1.54	1.62	2.09	1.37	1.38
Standard Deviation	1.65	1.3	0.38	0.92	0.75	0.54	1.04	0.61	0.27
Range	1.00 - 6.18	0.4 - 5.12	1.01 - 2.14	0.7 - 4.12	0.66 - 3.46	0.64 - 2.72	0.86 - 5.14	0.8 - 3.08	1.02 - 1.94
Lift phase (in secs.)	**F= 2.64 2,37**	***NS***		**F= 1.3 2,37**	***NS***		**F= 2.3 2,37**	***NS***	
Mean	3.5	2.51	2.48	2.95	2.82	2.19	3.53	2.61	2.39
Standard Deviation	1.72	1.3	0.57	1.55	1.23	0.48	2.07	1.1	0.42
Range	0.8 - 7.26	0.56 - 4.72	1.81 - 3.5	0.92 - 6.54	0.92 - 4.96	1.67 - 3.06	1.3 - 8.82	1.32 - 4.06	1.9 - 3.16
Down phase (in secs,)	**F= 1.15 2,37**	***NS***		**F= 1.3 2,37**	***NS***		**F= 4.07 2,37**	**p< .05**	
Mean	2.73	2.17	2.29	2.75	2.11	1.92	3.44	2.45	2.32
Standard Deviation	1.34	0.86	0.68	1.3	0.82	0.51	1.5	1.04	0.42
Range	0.72 - 5.48	0.6 - 3.46	1.38 - 3.5	1.04 - 5.16	0.54 - 3.22	1.3 - 2.84	1.26 - 7.3	1.16 - 4.56	1.56 - 2.84
Return phase (in secs.)	**F= 3.29 2,37**	**p< .05**		**F= 1.29 2,37**	***NS***		**F= 3.26 2,37**	**p< .05**	
Mean	2.44	1.64	1.55	1.92	1.68	1.46	1.97	1.52	1.5
Standard Deviation	1.37	0.76	0.58	0.95	0.61	0.44	0.68	0.45	0.41
Range	0.9 - 5.9	0.94 - 3.54	0.76 - 2.38	0.18 - 3.4	0.94 - 2.94	0.84 - 2.26	0.56 - 2.94	1.1 - 2.68	1.06 - 2.32

In order to explore the relationship between the task phases, correlation statistics across phases for all subjects in all three conditions were employed. Results revealed that each phase time was highly correlated with every other phase (ranging from $r = .44$ $p < .01$ but with the majority of correlation scores $r = .65$ and above $p < .001$). In all subjects, therefore, if a person were fast in one phase it was highly predictive that he would also be fast in all other phases, and conversely if slow on one phase he would be slow on all others. Covariant analysis indicated that age and gender were not influencing variables in the time taken over the experimental drinking task in any of the three experimental conditions [age: verbal condition $F(2,36) = 0.003$ *NS*, copy condition $F(2.36) = 0.03$ *NS*, real object condition $F(2,36) = 0.63$ *NS*. Gender : verbal condition $F(2,36) = 1.74$ *NS*, copy condition $F(2.36) = 0.23$ *NS*, real object condition $F(2,36) = 0.87$ *NS*]. The 'group' factor remained the one highly predictive of the total time taken in the task [verbal condition $F(2,36) = 5.44$ $p < .01$, copy condition $F(2.36) = 3.49$ $p < .05$, real object condition $F(2,36) = 4.13$ $p < .05$].

Analysis of time differences indicated that the reaching out into space and returning the hand back towards the body were areas of particular difficulty for the apraxic group. Other kinematic analyses (Fisk & Goodale 1988) have demonstrated similar results for apraxic subjects in these two phases of reach and return and this phenomenon will be addressed in the discussion section 5.3(ii).

A comparison of the peak velocity of movement in each of the phases of the task was undertaken in order to determine whether this gave an indication of the dynamics of movement and relative impairment of those with apraxia. In general, the apraxic subjects

showed reduced velocity peaks during most phases of the task in each of the modality conditions compared with the non-apraxic and control groups (Table 5.7). As with the timing components of the phases of the drinking task previously described, an overall trend emerged with the apraxic group showing the lowest velocity peaks, the control subjects showing the greatest velocity of movement, and the non-apraxic group being near to the control scores or lying in between the other two groups. In nine of the twelve phases, however, the non-apraxic group demonstrated the greatest variability between subjects (as shown by the standard deviation scores) indicating on this measure at least, a heterogeneity of performance.

In the ***reach*** phase of the experimental task in the verbal condition significant differences were demonstrated between the control group and the apraxic group in the level of peak velocity achieved (Table 5.7) with the mean velocity peak of the control group being almost double that of the apraxic group though the range within each group was very broad. No other phases of the task in the verbal condition showed significant group differences (see Table 5.7).

The copy condition of the task showed significant velocity differences between groups in the ***return*** phase only with the post hoc tests indicating the difference lay between the apraxic group and the normal control group. Again, large variability between subjects in each group was observed (Table 5.7). Finally, the real-object-use condition only showed a significant difference in peak velocity between the apraxic group and the other two groups during the 'down' phase of the drinking task (Table 5.7). These group differences found in the various phases according to modality conditions were not found to be

Table 5.7 : Group differences in peak velocity during phases of the drinking task.

	Verbal condition.			Copy condition			Real condition		
	Apraxic	Non-apraxic	Controls	Apraxic	Non-apraxic	Controls	Apraxic	Non-apraxic	Controls
Reach phase (in mm/sec/sec)	**F(2,35)= 4.18**	**p< .05**		**F(2,34)= 3.03**	*NS*		**F(2,35)= 2.12**	*NS*	
Mean	226.76	316.8	400.53	262.25	316.54	414.31	336.12	302.68	514.82
Standard Deviation	126.77	190.13	152.41	155.89	160.40	158.81	354.85	73.59	186.36
Range	76.02 - 505.42	102.39 - 663.58	143.12 - 672.52	54.83 - 584.16	115.47 - 605.16	228.19 - 726.85	74.6-1560.04	192.12-402.1	199.2-868.88
Lift phase (in mm/sec/sec)	**F(2,35)= 0.18**	*NS*		**F(2,34)= 1.37**	*NS*		**F(2,35)= 1.43**	*NS*	
Mean	630.95	670.61	584.18	616.44	777.12	824.51	463.71	587.37	595.66
Standard Deviation	255.15	534.97	138.05	260.56	506.1	264.58	219.76	274.90	207.61
Range	304.66 - 1324.61	245.74 - 2141.0	416.97 - 850.84	279.16 - 1274.3	425.13 - 1274.3	443.54 - 1135.36	209.89	262.1-1216.2	264.7-960.96
Down phase (in mm/sec/sec)	**F(2,35)= 0.14**	*NS*		**F(2,34)= 1.83**	*NS*		**F(2,35)= 4.92**	**p< .05**	
Mean	773.52	793.48	794.25	720.13	921.64	901.46	505.14	729.97	743.72
Standard Deviation	357.91	510.33	209.91	306.07	361.17	224.79	235.47	242.63	196.29
Range	293.45 - 1568.82	312.5 - 2161.85	490.85 - 1295.9	224.12-1410.66	525.96-1785.3	636.38-1305.16	178.6-1016.8	335.9-1126.0	509.1-1153.8
Return phase (in mm/sec/sec)	**F(2,35)= 1.5**	*NS*		**F(2,34)= 3.95**	**p< .05**		**F(2,37)= 0.99**	*NS*	
Mean	354.29	305.33	467.36	287.07	358.18	484.74	404.05	362.60	509.05
Standard Deviation	290.84	127.01	191.28	189.99	131.81	199.99	297.17	137.12	244.73
Range	112.18 - 1062.55	145.71 - 491.4	256.5 - 907.5	23.72-689.99	131.89-629.66	290.15-1013.46	28.1-1299.39	155.2-668.89	145.8-1092.6

influenced by either age or gender from the covariant analysis [age: verbal condition reach phase F(2,33) =0.16 *NS*, copy condition return phase F(2.32)=0.82 *NS*, real object condition down phase F(2,33) =3.64 *NS*. Gender : verbal condition reach phase F(2,33) =0.00 *NS*, copy condition return phase F(2.32)=0.49 *NS*, real object condition down phase F(2,33) =0.1 *NS*]. The group variable remained the major predictor of differences in peak velocity in the reach phase of the verbal condition [F(2,33) =3.91 $p < .05$], in the return phase of the copy condition [F(2,32)=3.67 $p < .05$] and in the down phase of the real condition [F(2,33) =4.57 $p < .05$].

In summary the apraxic group, in certain phases of the drinking task, were shown to be deficient in movement dynamics. Whether or not this was linked to a *mis-timing* of those dynamics could be determined by comparing the time through the movement phase within which the velocity peak was reached. In addition, looking at the movement recording to determine the existence of a long, low-level readjustment phase at the end of movement performance (as demonstrated by previously published research; Fisk & Goodale 1988) would provide evidence for a temporal mis-ordering of movement in apraxia. No relationship was found between the peak velocity data and the velocity timing data across groups. Some significant differences were found between groups in the percentage time through the phase when the velocity peak was achieved (Table 5.8) but only in the lift and down phases of the task in the verbal condition and in the lift phase of the real-object-use condition. Post hoc tests indicated that the differences lay between the apraxic group and the normal controls. No significant differences were seen in any phases of the *copy* condition (Table 5.8). Age and gender were, again, shown to be non-influencing factors.

Table 5.8 : Group differences in percentage time through phases of velocity peak in

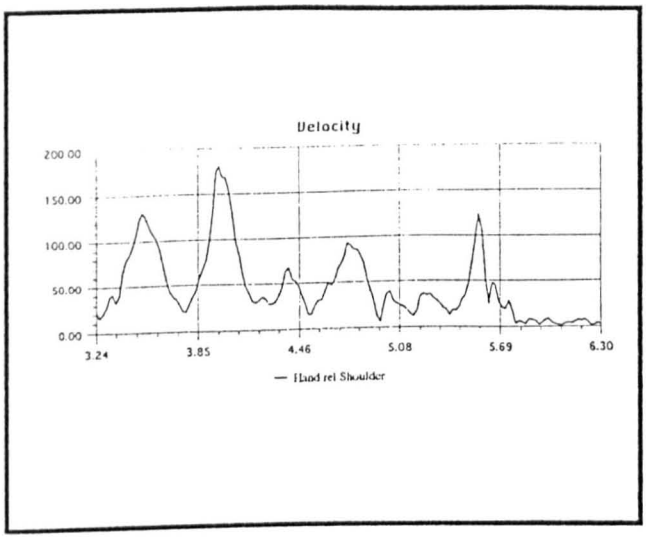
	Verbal condition.			Copy condition			Real condition		
	Apraxic	Non-apraxic	Controls	Apraxic	Non-apraxic	Controls	Apraxic	Non-apraxic	Controls
Reach phase (% time)	**F=0.88 2,34**	*NS*		**F= 0.1 2,35**	*NS*		**F= 0.51 2,34**	*NS*	
Mean	23.32	32.45	28.61	23.61	26.41	25.37	29.52	22.44	128.17
Standard Deviation	19.3	20.3	10.8	18.77	17.65	11.7	25.17	10.09	6.74
Range	2.9 - 62.62	8.06 - 68.42	10.68 - 50	3.41 - 74.44	5.88 - 74.14	8.05 - 43.3	5.71 - 88.71	2.35 - 40	17.65 - 41.67
Lift phase (% time)	**F= 4.94 2,35**	**p< .05**		**F= 0.46 2,35**	*NS*		**F= 4.29 2,34**	**p< .05**	
Mean	45.03	32.35	28.24	32.56	38.17	34.14	34.12	27.59	22.15
Standard Deviation	20.56	6.83	8.04	13.06	17.55	15.14	13.82	8.79	4.72
Range	20.28 - 95.84	18.33 - 42.21	14.71 - 41.32	10.81 - 54.35	15.71 - 69.12	18.35 - 70.2	7.88 - 62.05	13.76 - 41.94	15 - 31.01
Down phase (% time)	**F= 6.15 2,35**	**p< .01**		**F= 1.24 2,35**	*NS*		**F= 1.38 2,34**	*NS*	
Mean	33.89	43.91	55.87	45.54	37.68	47.06	38.83	35.87	45.76
Standard Deviation	17.59	17.61	11.12	16.85	9.66	17.13	15.25	7.09	17.10
Range	6.98 - 68.75	16.24 - 68.67	38.64 - 78.61	15.58 - 67.28	20.69 - 55.56	15.85 - 71.13	8.12 - 75.38	27.18 - 48.54	13.45 - 70.45
Return phase (% time)	**F= 1.23 2,35**	*NS*		**F= 1.69 2,35**	*NS*		**F= 0.5 2,34**	*NS*	
Mean	56.74	60.29	46.85	48.66	58.91	52.71	47.4	51.41	43.86
Standard Deviation	21.47	23.75	17.18	15.40	11.28	14.56	17.2	19.84	14.84
Range	11.36 - 80.85	26.21 - 88.7	20.51 - 84.87	22.73 - 77.51	44.44 - 79.35	34.78 - 85.23	19.51 - 89.29	25 - 85.45	21.67 - 73.91

Whilst a great degree of variability was shown between subjects in each of the groups, visual analysis of the velocity recordings in the different phases showed that certain patterns could be elicited. As illustrated in Figure 5.9, examples of the haphazard and inconsistent performances from the apraxic group in the reach phase of the verbal condition contrasted markedly with those of the controls. The apraxic subjects did not show a sustainable movement with smooth and gradual build up of velocity followed by a smooth deceleration until the target was achieved, as in control subjects' recordings. Rather, their output had several peaks and troughs as if there was a hesitancy and a continual readjustment of control over the reach movement time. The long, low-velocity period reported in other published research (Fisk & Goodale 1988) was also observed in these recordings, the extent varying with each individual apraxic subject. Similar hesitancies and multiple peak recordings were noted in the return phase of the copy condition in the apraxic subjects, which again were contrasted with the smoother performance recordings of the control subjects (Figure 5.10).

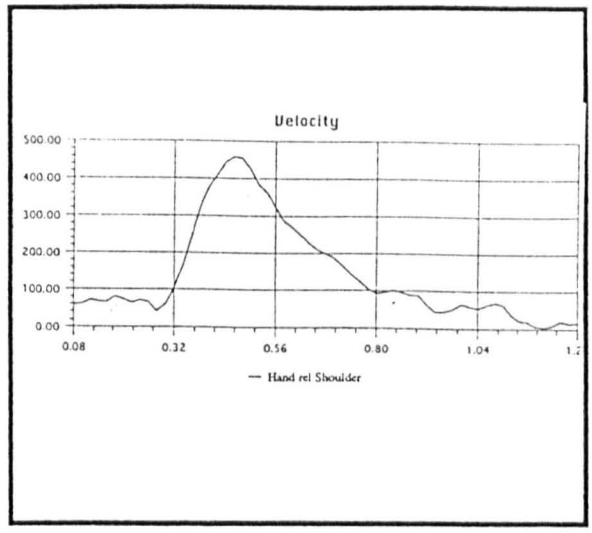
Figure 5.9 : Examples of velocity recordings during drinking task.

Reach phase, verbal condition.

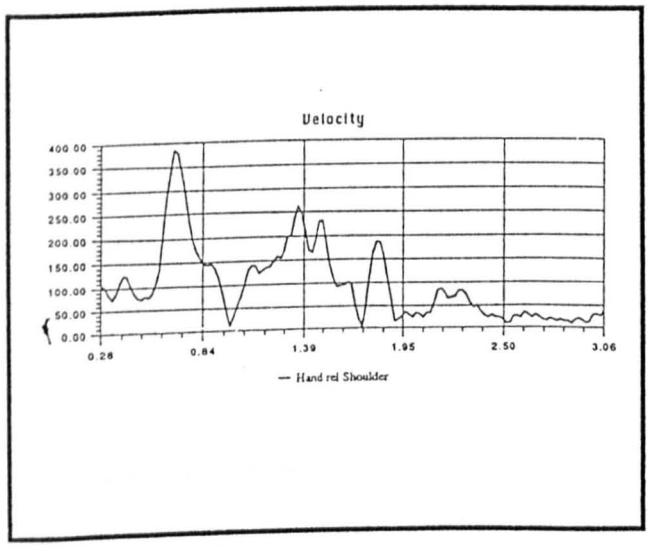
a] Apraxic subject



b] Normal control



c] Apraxic subject



d] Normal control

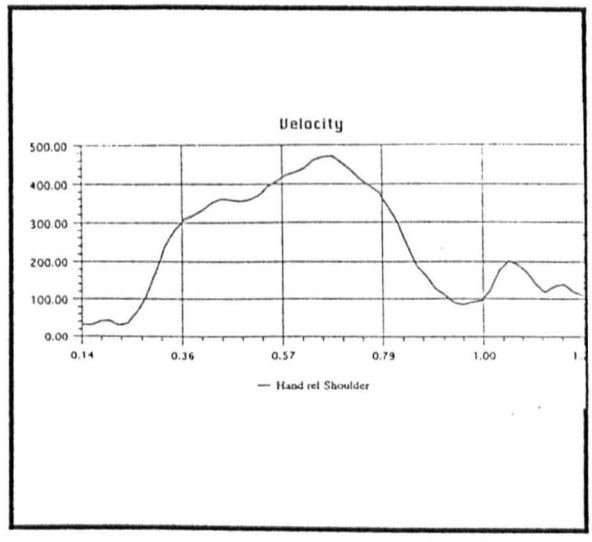
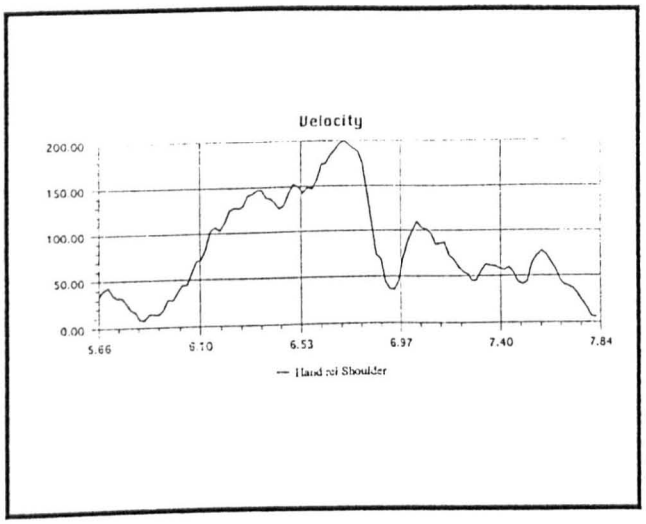


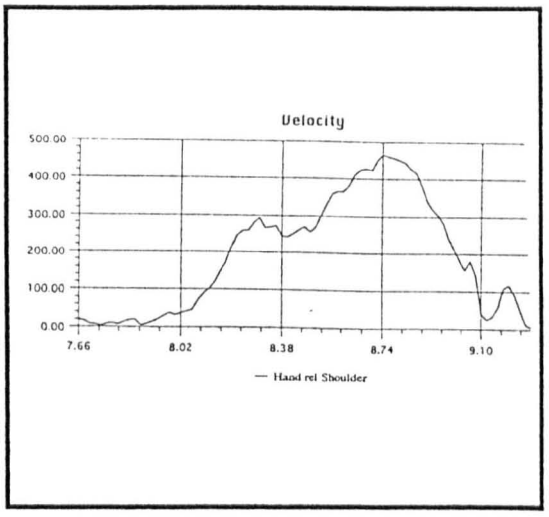
Figure 5.10 : Examples of velocity recordings during drinking task.

Return phase, copy condition.

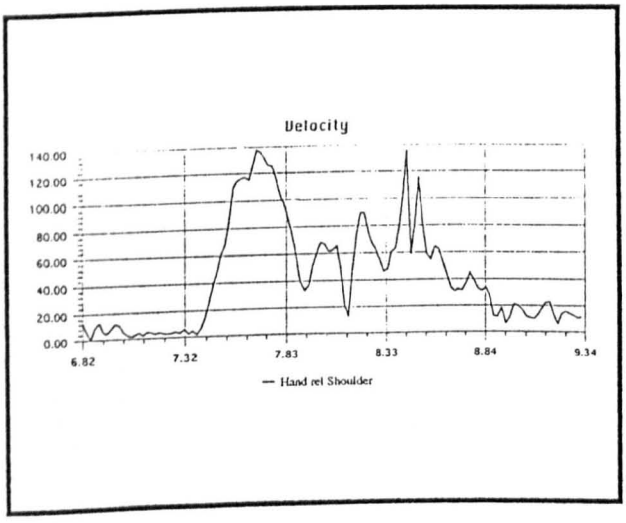
a] Apraxic subject.



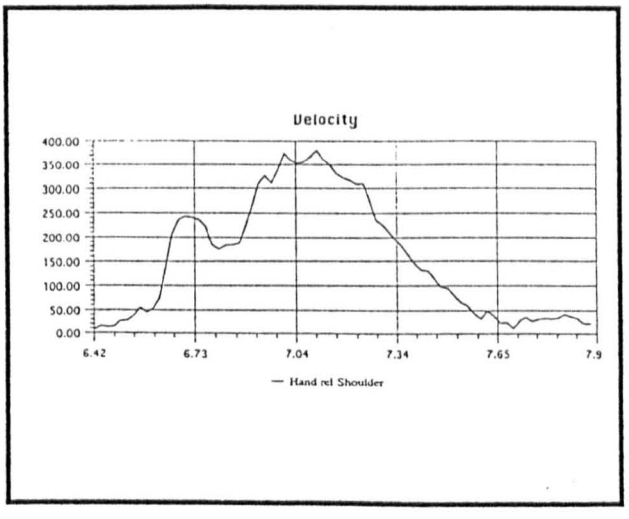
b] Control subject.



c] Apraxic subject.



d] Control subject.



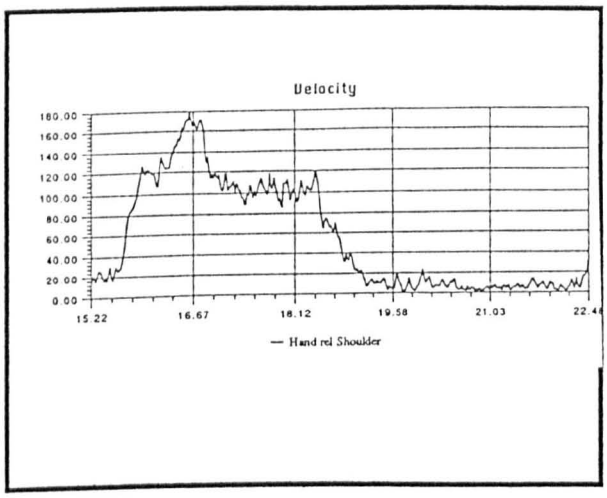
When the real object (a mug) was used to carry out the drinking task, the recordings clearly illustrated the long, low velocity patterns at the end of movement phases reported in other research studies (Fisk & Goodale 1988). The apraxic group were shown to need

longer periods of time with deliberate, slow adjustment to manage the placement of the object on the table when compared to the control group (see Figure 5.11).

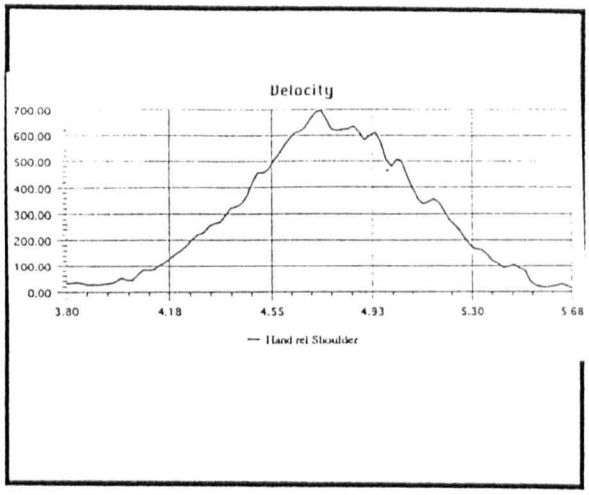
Figure 5.11: Examples of velocity recordings during drinking task.

Down phase, real object condition.

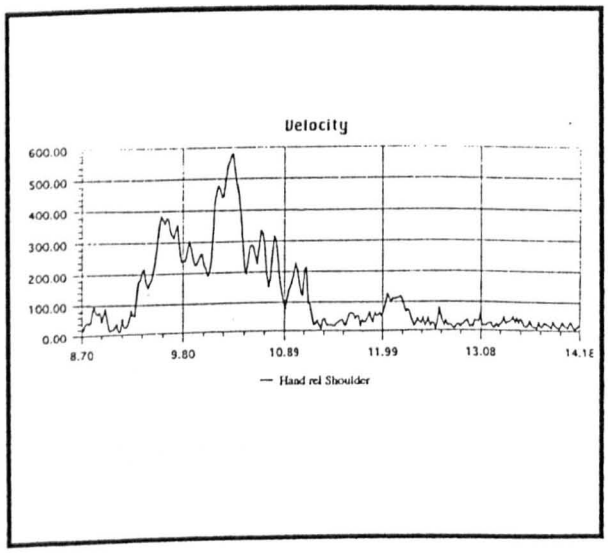
a] Apraxic subject.



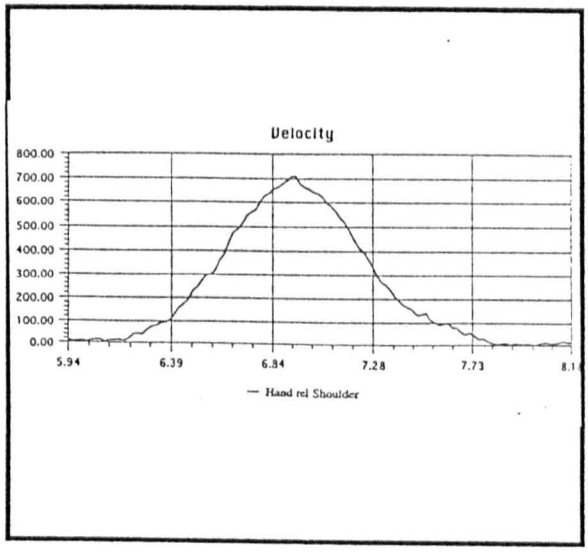
b] Control subject.



c] Apraxic subject.



d] Control subject.



It should be emphasised, however, that not all apraxic subjects showed dysfunctional kinematic recordings, either in trajectory or in velocity patterns, thus suggesting a possible dissociation of the kinematics of the experimental drinking task from the clinical test scores. So whilst the kinematics of movement were shown to be different between groups in some aspects of the drinking task in all modality conditions, those differences (mainly with a level of significance of $p < .05$) were not straight-forward nor did they necessarily equate with the clinical test performances. This association/disassociation between kinematics of movement and clinical test performances and scores will be addressed in the next section of this chapter.

5.2(iii) Relationship with clinical test scores

a]Clinical groups:

In order to determine whether level of independence was influencing speed and performance on the drinking task, correlational analysis was completed between time taken on the experimental task and Barthel Index score. No relationship was found in any of the experimental conditions across the clinical groups (non-apraxic and apraxic) : Verbal condition $r=.37$, $n = 30$; *NS*, Copy condition $r= .30$, $n = 30$; *NS*, Real object use condition $r= .33$, $n =30$; *NS*. This suggested that level of independence was not influencing the performance on the task. As the numbers in the clinical groups were relatively small it was decided to categorize the Barthel scores to check that no relationship existed between independence and experimental task performance. The percentage Barthel scores were divided into low (0-45%), medium (45.1 - 70%) and high

categories (70.1 - 100%) after examining frequency charts to ensure an even distribution. The ranges of scores within each category for the two brain damaged groups were 5-45% (low), 50-70% (medium) and 80-100% (high). No significant differences were shown in total task times in any condition relating to Barthel score (verbal condition $F(2,25) = 1.78$ *NS*, copy condition $F(2,25) = 1.15$ *NS*, real object condition $F(2,25) = 1.29$ *NS*). It could be concluded, therefore, that level of independence was not a significant influence on task performance in the clinical groups.

Scores on the apraxia tests were analysed to determine possible relationships with experimental task performance using the clinical groups alone, as it was considered that the (full scoring) ceiling effects from the control group would render analysis inappropriate. No relationship was found between any apraxia test and the total time taken for the drinking task in any condition (see Table 5.9).

Table 5.9 : Correlation data- apraxia test scores and total time on drinking task (apraxic group only).

	Total time - verbal	**Total time - copy**	**Total time - real**
Clinical test (D2) Haaland & Flaherty (1984)	r= 0.057 n=17; *NS*	r= 0.088 n=17; *NS*	r= -0.177 n=17; *NS*
Clinical test (D3) De Renzi et al (1980), Haaland & Flaherty (1984)	r=0.09 n=17; *NS*	r= 0.124 n=17; *NS*	r= 0.205 n=17; *NS*
Clinical test (D1) De Renzi et al (1980)	r= -0.152 n=17; *NS*	r= -0.201 n=17; *NS*	r= -0.201 n=17; *NS*

So although significant differences had been established between groups both in clinical test scores and in the completion (total) times in the experimental task, no correlation between the two components was demonstrated.

Multiple regression analysis was carried out, using the clinical groups only, to determine the extent to which the clinical test scores were ***predictive*** of movement performance in the drinking task. There was confirmation of the rigour of the De Renzi et al (1980) test with its emergence as the best predictor of total time taken in the ***verbal condition*** of the experimental task [$F(1,26) = 7.75$, $p < .01$]. When the two other ideomotor apraxia tests (gestural and object-use; appendices D2 & D3) were added into the regression analysis they were shown to have little unique variance outside the De Renzi test. So whilst all three tests still predicted total time taken for the drinking task in the verbal condition they were no more effective in doing so than the De Renzi test on its own. However, the De Renzi test was shown to be only accounting for 22.9% of the total variance in the total time taken for the drinking task. Other unknown factors were, therefore, involved but the inclusion of the agnosia score added only 5% to the variance and reduced the predictive effect [$F(1,26) = 6.83$, $p < .01$].

A different pattern emerged from analysis of the clinical tests in predicting performance in the ***visual/copy condition*** of the drinking task. The De Renzi et al (1980) test accounted for only 19% of the total variance but was the best ideomotor apraxia test predictor for the total time taken on the task [$F(1,26) = 6.1$, $p < .05$]. The addition of the other two ideomotor apraxia tests (gestural and object-use; appendices D2 & D3) into the regression analysis only increased the variance accounted for by 1.6%. When, however,

the score for the agnosia test was added into the equation immediately after the De Renzi et al test, it added 15% to the variance. Thus the two tests together accounted for 34% of the variance and were better predictors of the total time taken in the copy condition [$F(2,25) = 6.45$, $p < .01$] than the three apraxia tests as described above. Given that the agnosia screening test was influencing and addressing some aspect relevant to the experimental drinking task performance in this copy condition, possibly a visual/perceptual element or some cognitive component, the agnosia score was entered into the multiple regression equation as the first item for consideration. It accounted for 32% of the total variance and was highly predictive of the total time taken in the copy condition of the drinking task [$F(1,26) = 12.3$ $p < .01$]. Adding the De Renzi et al (1980) test next accounted for little more than an additional 1.9% of the variance, and whilst the two test together were still highly predictive of the copy condition performance [$F(2,25) = 6.45$ $p < .01$] they were less predictive than the agnosia test alone. The addition of the other two ideomotor apraxia tests (gestural and object-use; appendices D2 & D3) into the regression analysis only increased the variance accounted for by 1.6%. The strong influence of the agnosia test was an unexpected result.

In the ***real-object-use condition*** of the drinking task, the De Renzi et al test accounted for 20.73% of the variance in predicting total time taken to complete the task [$F(1,26) = 6.8$, $p < .05$]. In this condition, the addition of the agnosia test next into the multiple regression analysis increased the variance accounted for by 20.1% and increased the predictive ability of the tests on task performance [$F(2,25) = 8.62$, $p < .01$]. The agnosia scores were again clearly influential. Re-analysis of the data using the agnosia test as the

first entered item in the multiple regression indicated that for the condition of real-object-use, the scores on the agnosia test alone accounted for 39.4% of the variance and were highly predictive of total time taken to complete the task [$F(1,26) = 16.89$ $p < .001$]. The addition of the De Renzi test into the equation only added 1.4% to the variance accounted for and although the two tests together still were predictive of performance time, they was less than the agnosia test alone [$F(2,25) = 8.62$, $p < .01$]. So, once more when a visual/perceptual aspect in drinking task performance was inherent in the condition, the agnosia test scores were shown to be highly predictive.

b] Apraxic Subjects:

The apraxic subjects, as a separate group, were analysed for the degree to which the clinical test scores could predict performance on the experimental task. In other words, was severity of apraxia predictive of task performance? These apraxic group scores on the clinical tests were first analysed for correlations with performance on the drinking task. They were all shown to be very poorly related to the total time taken to complete the task in all conditions. Each correlation coefficient was below the level of statistical significance with r at less than 0.3 for each calculation [$r=.3$, $n=17$; *NS*]. Scores on clinical tests for apraxia, which might be considered to give a measure of *severity* of the condition, were not shown to have a relationship with time taken on the experimental task in any modality condition.

In order to determine whether this was a robust dissociation or an anomaly due to small numbers in the apraxic group, two categories of 'not severe' and 'severe' patients were

created ('low' scores ranging from 38-51, and 'high' scores ranging from 52-67 in the De Renzi et al 1980 test) to see if this cruder division could predict differences in the experimental task performance. The categories were determined by frequency analysis of scores in each of the ideomotor apraxia tests and by dividing the group according to where the 50th percentile lay. This provided a fairly arbitrary, but equal, distribution between the groups. There were no significant differences between 'low' and 'high' score categories in the apraxic group in any components of the drinking task. The scores were not predictive of total time taken in any modality condition, nor in time taken in any *phase* of the task. This finding was replicated in all of the ideomotor apraxia tests taken individually as well as when using the cumulative percentage score across all three tests and dividing the apraxic group in to 'low' (range 46.2%-61.29%), 'medium' (range 61.6% - 65.74%), and 'high' (range 69.17% - 84.42%) scorers : [Verbal condition $F(2,14) = 1.62$ *NS*. Copy condition $F(2,14) = 1.19$ *NS*. Real-object condition $F(2,14) = 2.04$ *NS*]. As well as this robust dissociation between the clinical test scores and the temporal components of the experimental task, the other kinematic components available for quantitative analysis, such as velocity and acceleration profiles, showed no statistically significant differences between the 'low' and 'high' score categories of patients in the apraxic group in any phase or in any condition of the experimental task.

This important negative finding supports the published work of Hermsdorfer et al (1996). Several plausible explanations for this dissociation could be considered. The ***severity*** of apraxia, as measured by each of the clinical tests, was not shown to be predictive of performance in the drinking task in this analysis and one explanation might be that it is the

presence or absence of apraxia as an underlying dysfunction that is the critical determining difference between group performances. Alternatively it could be argued that the clinical tests were unrelated to the temporal and kinematic components of the chosen experimental task, or as Hermsdorfer and colleagues suggested more a matter of the individual adaptive strategies adopted by the apraxic patients.

If the clinical test scores were unrelated to the kinematic components of the experimental task then those test scores would not be expected to contribute to the variance of performance in the apraxic group. Multiple regression analysis demonstrated this to be the case. Even the De Renzi et al (1980) test, which might be considered the most sensitive of the assessments, accounted for only 6.9% of the variability in the verbal condition of the drinking task [$F(1,14) = 1.05$ *NS*], 11.9% in the visual/copy condition [$F(1,14) = 1.89$ *NS*], and 7.2% in the real object use condition [$F(1,14) = 1.08$ *NS*]. The addition of the other two (gestural and object-use) tests for ideomotor apraxia added little to the predictive value of the De Renzi et al test alone and the tests remained non significant in being able to predict time taken over the task. The impact of the agnosia test scores once more became apparent, however. In the verbal condition of the drinking task, after the clinical tests for ideomotor apraxia had been put into the multiple regression equation little variance was accounted for by the addition of the agnosia test. When placed *first* in the equation, however, 23% of variance was accounted for by the agnosia test scores. This was still not significantly predictive of performance in the experimental task in the verbal condition [$F(1,14) = 4.28$ *NS*], but nevertheless better than the predictive effect of the De Renzi et al test first applied. In the ***copy condition*** of

the experimental task a different pattern emerged. The addition of the agnosia screening test score was shown to add 31% to the identified variance even *after* the three ideomotor apraxia tests (appendices D1, D2, D5) had been put into the multiple regression analysis, and the addition of this test to the analysis raised the regression analysis from *NS* to a level of significance [$F(4,11) = 3.55$, $p < .05$]. Clearly the agnosia test was showing an association with the copy condition performance. A regression analysis was, therefore, carried out using the visual agnosia test score as the *first* variable entered into the equation. It accounted for 54.8% of the variance and had a high predictive ability of the total time taken on the task by the apraxic subjects [$F(1,14) = 16.96$, $p < .001$]. In this condition, the addition of the De Renzi test into the equation added only 0.5% to the identified variance and the predictive ability of these two together was shown to be less than the agnosia test alone [$F(2,13) = 8.05$, $p < .01$]. The other clinical test scores similarly had little predictive effect and added only 0.6% and 0.4% to the variance.

This same phenomenon occurred in the ***real object use condition*** analysis, where the De Renzi test accounted for only 7% of the variance of timed performance with 0.4% and 0.9% being added by the other clinical test for ideomotor apraxia, yet the addition of the agnosia screening test score added 46% to the identified variance and gave the four tests together a predictive ability of just less than significance level [$F(4,11) = 3.3$, $p = .052$ *NS*]. When the visual agnosia test score was the first variable entered into the equation 47.96% of the variance was accounted for and it was shown to have a statistically significant predictive ability of the task time [$F(1,14) = 12.9$, $p < .01$]. The addition of the

three ideomotor apraxia tests to the regression analysis accounted for little or none of the variance (0.01% for test D1, 2.6% for test D2, 3.9% for test D3) and reduced the predictive ability of the tests, when taken together, to non-significance. This confirmed the earlier analysis using both clinical groups that the clinical tests for apraxia were not associated with performance on the experimental task, and that the agnosia test was highlighting some component of performance particularly in relation to the copy and real-object conditions.

A t-test analysis was carried out, using the apraxic group only, to determine whether any differences existed in task performance between those with ideomotor apraxia alone compared to those with both ideational and ideomotor apraxia. No significant differences were found between the two groups using total time to complete the task as the dependent variable [Verbal condition $t(15) = 1.1$ *NS.* Copy condition $t(15) = 0.9$ *NS.* Real object condition $t(15) = 0.55$ *NS.*]

5.3 Discussion

5.3(i) Clinical tests for apraxia

Analysis of the clinical tests used in the diagnosis of apraxia in this project supported the claim by Tate and McDonald (1995) that the test compiled by De Renzi et al (1980) is the most reliable and sensitive of those used in research. The present study has highlighted some of the difficulties with the other tests for apraxia (Haaland & Flaherty 1984, appendix D2 and D3) in pantomimed gesture and in pantomimed use of objects with some internal consistency problems being evident in those tests. None of the tests

for ideomotor apraxia were shown to correlate with any other, suggesting they were measuring different aspects of a condition and yet each test alone showed highly significant differences ($p < .001$) between the three experimental groups. This might suggest that although the tests were highlighting different aspects or sub-groups of a condition, the underlying condition was one and the same. This research has added further support for the need for a standardised and reliable diagnostic tool for use in both clinical work and research. Given that these tests do not have the rigour that could be achieved from a standardised battery, then the basis for the original division of the clinical subjects into apraxic and non-apraxic groups might be called into question. It could be argued, however, that the use of *multiple* tests in the diagnostic procedure used in this study might counter such a claim. The researcher/clinician had the opportunity to observe the subjects, looking for apraxic errors, over a longer period than would have been achieved if just one test had been used. Furthermore, the lack of any correlation between the different test scores in each apraxic subject could be considered to support the contention that the identification of the wide range of performance deficits within this heterogeneous group relies upon using a ***range*** of tests in the diagnostic procedure. In fact, it could be considered critical. This, if accepted, suggests flaws in the work of those research groups which only use one clinical test to determine the presence, or otherwise, of apraxia in any individual. Alternatively it might be suggested that in using the variety of tests, the 'apraxic' group of this study were more heterogeneous than might have been desirable for research purposes as it included, perhaps, different sub-categories of apraxia that might not have been included if only one apraxia test had been used for

diagnosis. However, any results which *were* established from such a heterogeneous group would attest to the robustness of such findings in relation to the general aspect of apraxia rather than specifically to one 'sub-type'.

The apraxic group in this study was shown to have a wide *range* of scores on each of the clinical tests. So not only was this group heterogeneous in possible sub-category types, it also contained a range of severity levels within the blanket term 'apraxia'. About half of this group showed evidence for both ideomotor *and* ideational apraxia (8 of 17) while 9 of 17 demonstrated errors associated with the ideomotor condition alone. A larger number of subjects capable of being categorized into apraxia **sub-types** might have added rigour to the study, but the availability and suitability of patient subjects is a constant difficulty in this field of inquiry. The general pragmatics of clinical research always make large group studies problematic in short time schedules. Clearly this group cannot be adjudged to be a homogenous group (apart from the ***presence*** of apraxia in all its forms) but rather a disparate heterogeneous one. Despite these limitations relating to the apraxic 'pool', a number of observations regarding the kinematics of apraxic movement performance seen in the group can be made.

5.3(ii) Kinematic analysis of movement.

This study has supported the previous work of Poizner et al (1990) and Hermsdorfer et al (1996) who presented evidence for the disruption of both the fluidity of movement and movement timing in apraxic subjects. In particular Poizner et al (1990) noted a hesitancy of movement and a 'searching or groping behaviour' (pp. 99) for the appropriate

movement in the apraxic subjects. Such hesitancy and spatial misalignment was observed in this current study and seen in the trajectory patterns of movement in the apraxic group when compared with the controls. It might be suggested that such movement hesitancy and the searching for correct movement patterns could be attributed to the breakdown of the normal temporal-spatial elements of learned motor patterns and an inability to readjust movements continually in a systematic and efficient manner as they unfurl towards the target goal. There appeared to be rather a stop-start or surging manner of adjustment with over and under-compensation of movement parameters. Such factors might indicate the possibility of an inefficient or interrupted feedback system. Such inefficiency would naturally lead to a longer task time. This was borne out by the current results where there was evidence of longer time taken to complete the experimental task by the apraxic subject group. This also supports the research of Fisk and Goodale (1988) where apraxic subjects were shown to take longer in task completion.

The task completion time difference with apraxic subjects would appear to be a robust finding across different research groups (Fisk & Goodale 1988, Haaland & Harrington 1994, Hermsdorfer et al 1996) and replicated in this current study. This despite the fact that each study has adopted a different kind of movement; pointing to target, repetitive gestures, meaningless gestures, meaningful gestures across different modality conditions. Clearly timing of movement is used in all research projects using kinematics as it is a straightforward and accessible measure, yet Hermsdorfer et al (1996) has suggested that strategies for managing apraxia might be different within each individual. If so, then Hermsdorfer's argument of some apraxic subjects adopting a slow, on-line controlled

movement, others adopting the fast 'rough approximation' of movement strategy, and others (being unaware of their deficit) executing a temporally normal movement to the wrong target position should result in a bipolar distribution of timing within the apraxic group. This was not shown in the current study where a range of timing was apparent. In addition it might be expected that those with ideational apraxia (being unaware of their deficit) would perform differently from those with ideomotor apraxia in their temporal control, yet this was not found in this study. Timing of movement, at a simple level, can say something about the efficiency of action performance. Judgments about apraxic movement carried out in the clinical assessment include a temporal element, but the majority of error judgments are made upon spatial aspects. Thus a dissociation between timing of movement alone and apraxia assessment scores might be considered to be inevitable. The nature of this dissociation with clinical assessment will be raised in section 5.3(iii).

Clark et al (1994) pointed out the shape of the trajectory pathways was different in the apraxic subjects. Similar evidence to support this assertion has been presented in this study. As well as the shape and spatial misalignment of the trajectory pathways in the apraxic group, an overuse of proximal joints was also shown in this experiment. This is in keeping with, and adds support to, the case study work of Poizner et al (1990). It might be suggested that such an overuse of the shoulder could be the primary difficulty in the spatial misalignment shown in the hand trajectory and not, as Poizner et al (1990) suggested, as a compensation for distal joint malcontrol. Unless the shoulder is stabilized in space, the hand cannot achieve good and efficient movements. Thus an erratic

shoulder joint and upper body movement might act to increase the difficulties in motor execution, with the motor system needing to adjust more parts of the upper limb during the movement than would normally be required. This could explain the hesitancy and the 'searching' type of movement behaviour seen in the apraxic subjects.

The abnormal velocity patterns might also have some basis in this overuse of the proximal joint in the apraxic group. Hermsdorfer et al (1996) pointed out that bell-shaped velocity profiles were characteristic of skilled pre-programmed movements. This was clearly seen to be the case with the control group in this current study. Hermsdorfer et al (1996) also demonstrated that multi-peaked velocity patterns with prolonged adjustments were rare in normal control subjects, but frequently seen in left hemisphere damaged subjects. This was only partially supported by the current study where the typical multi-peaked pattern was seen in the apraxic groups' velocity recordings in all phases of the experimental drinking task, though it was *not* a feature of the non-apraxic clinical group. The current study would suggest that the multi-peaked velocity patterns were more indicative of the presence of apraxia than just left hemisphere lesion *per se*, and would indeed support the suggestion made by Hermsdorfer et al (1996) that these particular apraxic subjects had adopted an hesitant, on-line control of movement which would not be a strategy required of non-apraxic subjects whose praxis system was intact.

Looking for explanations why this study should only find multipeaked patterns in the apraxic group and Hermsdorfer et al find such patterns in left hemisphere damaged patients regardless of presence of apraxia, two possible aspects are evident. Firstly in the absence of detailed MRI scans, which might provide information on the specific location

and size of lesions within each group, no speculation can be made about whether the finding is related to differences in lesion location between the apraxic and non-apraxic group in comparison with the subjects in the Hermsdorfer et al (1996) study. Secondly the different nature of the experimental tasks used in the Hermsdorfer study and in this project might also help to explain these discrepant findings.

This study has also provided evidence that some of the apraxic subjects have, occasionally, demonstrated a normal kinematic profile with trajectories similar to the non-apraxic subjects and with bell-shaped velocity patterns through the task phases, thus lending support to the Hermsdorfer et al (1996) published research. Again the 'strategy' explanation offered by the Hermsdorfer researchers could account for these normal profiles especially as the target position in this current study was not measured, so information relating to accuracy of reaching the 'red spot' during the verbal and copy conditions of the drinking task is unavailable.

So it remains to be established how far the multi-peak velocity patterns are *indicative* of the presence of apraxia, but it might be suggested that such a presence in a kinematic profile would only exist if the individual has needed to consciously think about and process their movement in a deliberate way. Thus such multi-peak patterns might only be seen in those with apraxia completing a target-based task, but not necessarily in *every* apraxic person where different strategies might be employed. Hermsdorfer's explanation of different strategies producing different kinematic profiles is an attractive one that could be explored further, perhaps in qualitative exploration of patients' experience of and approach to their difficulties.

The evidence presented by this study has suggested that the apraxic subjects have particular difficulties in the reaching out and return phases of the drinking task. This difficulty in reaching out into peripersonal space and returning to interpersonal space could be interpreted in several ways. It might be a linked to the proximal joint mis-use, because reaching movements produce more shoulder and body movement than the lifting movement in attempting to achieve the target position. Thus more adjustment and monitoring would be needed during these horizontal plane phases of the experimental task, leading to the group differences seen in this project. This would be in harmony with the work of Poizner et al (1995) where apraxic subjects showed disruption of the well-organised relationships between upper limb joints seen in normal control subjects, with evidence of multiple irregularities in the use of shoulder and upper body movements. Alternatively, or in addition, it might imply a particular disruption to the spatial aspects of movement and a difficulty in carrying out the spatial plan of a movement, especially when that spatial plan has to involve a representation away from the personal body space to a given target. Raade et al (1991) have also reported particular difficulty in the spatial aspects of movement relating to 'away-from-body' movements in apraxic subjects. The current kinematic analyses would seem to confirm those findings by demonstrating a disruption of spatio-temporal representation of movement during the drinking task. It is also supportive of the work of Fisk and Goodale (1988), Poizner et al (1990) and Clark et al (1994) all of whom highlighted the impaired spatial orientation of movement shown in apraxic patients. The return of the hand from a spot away from the body to a place at the edge of the table is less easily explained by such spatial disruption, although the

movement might also be conceived as moving in peripersonal space with adjustments needing to be made to achieve the target spot. Such movement also requires sustained control and modification of the proximal joints (shoulder joint and shoulder girdle) and these have been suggested as potentially being implicated in the spatial misalignment seen in apraxic subjects. The particular difficulty in the apraxic subjects in this part of the movement might, alternatively, be related to their inability to organise completion of the movement required. The motor planning memory of the task requirement, by this end phase, may well have deteriorated. This, it could be suggested, would result in the apraxic group having more difficulty in the motor performance than those participants with intact motor planning systems.

An alternative explanation to the particular difficulties with 'reach' and 'return' aspects of the experimental task could be sought in relation to the strong and unexpected influence of the agnosia test in predicting task performance. The reach and return elements of the drinking task are directly under visual control and the movement is 'in sight' for the whole of its duration. The other phases of the task (lift and down) lose the visual contact for a part of their duration which would lead to a more automatic (i.e. not visually guided) movement performance. Thus a possible explanation of the different phase performances could be that these were related to a similar factor or phenomena being picked up by the agnosia test, perhaps in relation to vision-to-action routes.

Modality differences were not a strong feature of this experimental task though the greatest time differences between groups was shown in the verbal condition. This lends some support to the body of evidence that a verbal command is more difficult for apraxic

people in that they have to formulate their own internally remembered motor representations. Such additional difficulty, over and above the visual modality/copy condition where a model of the movement is provided, would lead to an increased time to complete the task. This was indeed found in the current study and adds support to the notion of the intact vision-to-action routes in the apraxic subjects with disruption to the semantic-action routes in the condition (Riddock et al 1989, Pilgrim &Humphreys 1991). The real-object-use condition elicited movement deficiencies in the apraxic group which added support to the clinical evidence suggesting that the condition does affect real-world performances in everyday life. This was particularly notable in the 'down' phase of the task where the apraxic subjects demonstrated difficulty in making the rapid and efficient adjustment needed to the movement performance. They had to take into account the weight of the object in placing the mug to the target spot on the table and appeared to find this particularly difficult. This was not a feature of brain damage *per se* but only of the apraxic condition as the non-apraxic group did not display such difficulties. It would appear that those *without* apraxia were able to make the rapid adjustments and integration of sensory input, when using a real object, and produce a smooth motor output to the extent that the kinematic measurements showed little or no difference between the gesturing conditions and the real-object-use condition.

This study found no strong evidence to support the work of Poizner et al (1990) in relation to reaction time differences in apraxic subjects. Reasons for this discrepancy could relate to the nature of the tasks within each study (repetitive gesture vs. meaningful single gesture) or that the number of subjects (two) in the Poizner study could be mis-

representative of the apraxic population. Fisk and Goodale (1988) also reported no difference in reaction/initiation times between apraxic subjects and normal controls.

5.3(iii) Kinematic analysis and relationship to clinical tests.

All the clinical tests for apraxia showed significant differences ($p < .001$) between groups (taken individually and as a mean % score from the ideomotor apraxia tests) with the apraxic group showing the lowest scores. The time taken to complete the drinking task in all modality conditions was shown to be significantly different between groups ($p < .001$) with the apraxic group being the slowest. The post hoc tests revealed that the difference lay between the apraxic group and the other two (non-apraxic and control) groups for both the drinking task and the clinical assessments. However, it emerged that these two elements of action performance in apraxic subjects were not related.

The De Renzi et al (1980) test for diagnosis of apraxia was shown to be the most sensitive of the clinical tests used in predicting performance on the drinking task. Yet it only accounted for a maximum of 30% of the variance in completion time for the drinking task in different conditions. Analysis of the data in relating apraxia test scores (i.e. *severity* of the condition) to completion time of the task showed no relationship. This absence of relationship between kinematic disruption and apraxic errors in the diagnostic tests is an interesting phenomenon and requires explanation.

At a very straightforward level it might be asked why a general 'overall time' measure might relate to clinical apraxia assessment at all. Temporal aspects of movement are included in the judgment of errors performed, but the majority of the decision-making

process relates to the *spatial* aspects of the movement or gesture performed. Thus the timed task could be said to capture, in the timing element, only a very small part of the action output from the apraxic person. Other kinematic analysis relating to the spatial elements of the task (trajectory, velocity patterns) could provide associations with the clinical tests, though this was not borne out by Hermsdorfer et al (1996) who also found dissociations between the kinematic data and clinical aspects of apraxia. Unfortunately, in this study, the software and computer-programming facilities were not available to quantify these spatial elements of the kinematics in order to study their relationship with the elements of the clinical test scores in any completely thorough and rigorous way. A visual inspection in relation to experimental group was, largely, all that was achievable.

Alternatively, the lack of relationship found by Hermsdorfer and colleagues in the spatial elements of the kinematics might suggest that timing of movement is as good a way as any to tap into the qualities of action performance. In the absence of sophisticated equipment and/or computer support, then timing of movement performance might be a simple way to quantify action. Hermsdorfer et al (1996) suggested that the dissociation between the kinematics of movement and the clinical evidence for apraxia could be attributed to differences in strategies adopted by the patients. This is a compelling explanation for the results seen in this research as well as that of Hermsdorfer and colleagues. Such an argument provides explanation not only for the multi-peak kinematics seen in some patients, also the long, low-velocity periods at the end of movement phases, and also for the normal kinematic patterns seen in some of the apraxic

group. Evidence for each of these kinematic profiles were described in this current study. Unfortunately the Hermsdorfer study did not provide data on the timing of movements, so a comparison cannot be made with such data from this project. If the Hermsdorfer explanation is a correct one, however, then the timing of movement should reflect the different strategies that were employed by the patients and a wide range, including normal timing should be observed. This was seen to be the case with the apraxic group (Table 5.5) where the broadest range and widest standard deviation was noted (including 'normal' times) compared to the other experimental groups.

The dissociation between clinical errors and kinematic profiles found in this study might, though, be evidence to support the 'sub-classifications' of apraxia argument. It might be considered that the subjects in the apraxic group, being a heterogeneous collection, were each performing according to a separate set of underlying impairments. Thus the sub-types would perform in different ways both in clinical tests and in the experimental task and account for the individual 'anomalies' seen within the group data. This notion has potential for further exploration using large number groups to tease out the possible 'sub-types' according to both clinical and kinematic performance. Such investigation of the relationship between the kinematic and the clinical aspects of apraxia is clearly called for.

An unexpected finding in the research was the influence of the agnosia screening test devised by the author and colleague. It has been demonstrated how this test was a major influence in predicting time of performance for both non-apraxic and apraxic groups (although the influence was the stronger in the latter) in both the copy and real-object

conditions of the drinking task, yet not for the verbal condition. Several explanations might be forthcoming. Very simply, it was possible that the agnosia test (shown to be uni-dimensional from alpha reliability analysis) was eliciting something as straightforward as impairments or limitations in visual acuity in the subjects. Although spectacles, if prescribed, were worn during assessment procedures they may have become less than adequate for the individual following his/her stroke. If so, seeing the target spot on the table during the experimental task would also have been problematic leading to hesitation and 'searching' for the required target position. Thus the relationship between the agnosia test and task performance would be established. This explanation is probably unlikely as patients made it very clear, either through language or facial expression, when they were unable to clearly see the photographs in the agnosia test. The red spot on the table was large and clearly contrasted with the pale blue of the table top.

An alternative explanation could be that the agnosia screening test was also tapping into a cognitive construct relating to the ability of the patient to understand instructions, or to speed of information processing or even related to intelligence. If any of these were to be the case then the level of understanding would be consistent for both the agnosia test and the experimental task. This explanation might be discounted, however, as the clinical impression during examination procedures did not indicate problems with cognition, nor was their cognitive ability highlighted as problematic by other rehabilitation staff. In addition, the tasks were both were very simple and straightforward. During testing all patients produced behaviour and responses that indicated an understanding of the requirements of both the agnosia test and the drinking task.

A third, and possibly the most likely, explanation would take into account the nature of the visual and perceptual elements of the agnosia test. The agnosia test required patients to make judgments of 'odd one out' relating to their *knowledge of objects* as represented by black and white photographs of visually similar items. This interpretation of visual representation to 'knowledge of object' and action might bear some relationship to the underlying impairment in apraxia which results in people not being able to produce actions to verbal commands but can more easily respond to the visual input of a gesture to copy or to the real object. In other words that the agnosia test reflected the intact vision-to-action route (Riddoch et al 1989) in apraxic patients. The agnosia test was highly predictive of performance in the copy condition of the drinking task where the movement was perceived and interpreted from a model provided by the examiner, and also to the real-object condition where the object was present. The agnosia test was not predictive of performance (time to complete) the verbal condition of the drinking task. Riddock et al (1989), and Pilgrim and Humphreys (1991) both suggested that action to visual presentation, either of gesture or object, might be preserved because of intact links between input and action which by-pass impaired semantic-action routes. The agnosia test, the copy and real-object conditions in the experimental task might all be reflecting the intact vision-to-action route in the apraxic group, and thus performance on the test would be highly predictive of performance in the tasks. If this explanation holds true, then the test would be one worth developing and refining as a valuable tool both in the diagnosis of apraxia, and in determining the particular qualities of individual apraxic sub-types and their underlying impairments.

Further research would seem to be called for in which the use of detailed single case methodology might contribute some useful information particularly in relating kinematics of movement performance to clinical test scores. Such approaches would also allow an investigation into whether the kinematics of movement performance **change** during the recovery process in apraxia. Similarly, other studies are needed to ascertain whether such changes could be associated with intervention strategies and thus be used in evidence-based practice. It would also be useful to establish whether the apraxic performance elements shown in this current study could be replicated across different underlying pathologies such as head injury.

5.4 Conclusion

Kinematic analysis of movement in a drinking task across different modality conditions has indicated differences between people with apraxia and groups of clinical non-apraxic people and normal controls. Evidence of slowed movement performance was shown in the apraxic group and was demonstrated to be an element of the apraxic condition but *not* of brain damage alone. Disruption of the temporal-spatial elements of movement was demonstrated by abnormal trajectories of the hand and arm movements and also in the abnormal velocity patterns seen throughout the task. Whether such trajectory and velocity patterns were indicative of the apraxic condition alone has yet to be conclusively established.

An absence of relationship between the kinematic disruptions of movement performance and the clinical tests for apraxia was clearly demonstrated in this study, adding supportive

evidence to previously published research. It was hypothesised that such a dissociation could be attributable either to the different strategies which might be employed by individual patients to compensate for their movement unpredictability, or to the heterogeneity of the apraxic condition with different 'sub-types' performing in highly disparate ways. The influence of the agnosia screening test in predicting movement performance in the experimental task (copy and real-object conditions) was suggested to relate to the intact vision-to-action routes in apraxic patients.

1

[1] Public output from chapter data :

Bateman A., & Butler J.A. (1995). Review : Macreflex Motion Analysis System. Psychology Software News. September. pp 16-18

Butler J.A.(1995) Kinetic Criteria for the Diagnosis of Ideomotor Apraxia? Abstracts: 4th IBRO World Congress of Neuroscience. Kyoto, Japan. pp 514.

Butler J.A. (1995). Kinematic Analysis of Movement Performance in Ideomotor Apraxia: Providing a Focus for Targetting Rehabilitation Strategies? British Neuropsychological Society. London, annual meeting. (April 1995)

Butler J.A. & Howells K.F.(1995). Kinetic Errors and Modality Differences in Ideomotor Apraxia. Proc. British Psychological Society. 3(1).pp 78

Butler J.A., & Howells K.F.(1994). Quantitative Analysis of Apraxia. Proc. 11th Int.Cong. World Federation Occupational Therapy. Vol 3. 1156-1158.

Butler J.A., Howells K.F., Jordan T.C. (1994). Kinetic Errors in Apraxia::Are They Diagnostic? Proc. British Psychological Society. 2(1). pp 47.

Butler J.A., & Howells K.F. (1993). Quantitative Analysis of Apraxia. In : Conference Proceedings. British Journal of Occupational Therapy 56(9). 339-346.

Chapter 6. CASE 1 :

MT - Sub-arachnoid haemorrhage. 'Natural history' of apraxia..

6.1 Case background

MT, a 48 year old woman, was assessed shortly after arriving at the Rivermead Rehabilitation Centre and three months following a sub-arachnoid haemorrhage affecting the left cerebral hemisphere. At the time of initial screening assessment MT had no functional movement in her right arm or leg and had a Barthel ADL score (Wade 1992) of 9 being partially dependent in most areas except for bathing (dependent) but she was fully continent. Some help was needed with her personal grooming, though she had been scored as independent in the medical notes. Thus her 'true' score might more accurately be given as 8 out of a possible 20. MT was severely aphasic with a FAST score of 4 for comprehension and 0 for expressive speech (Frenchay Aphasia Screening Test, Enderby et al 1986). She was left handed.

Informed consent for participation in the study was obtained from MT's husband, in the presence of MT, as it was considered that her aphasia was of a severity that might preclude full understanding of the explanations given. In doing this it was acknowledged that MT herself was able to make her desires and wishes known, and would make it very clear to the researcher if she were unhappy with any of the procedures or did not want to continue at any time.

The movement errors made during initial assessment procedures indicated that MT exhibited features of both ideomotor and ideational apraxia. (Table 6.1 shows initial assessment scores). Her aphasia was considered to be influencing some elements of the

apraxia tests, particularly in the verbal command for meaningless gestures in Test 1 (appendix D2). At the initial assessment, MT had particular difficulties with gesturing object use but more in the visual modality condition than in the verbal modality condition. This might indicate that aphasia alone was not the sole contributor to MT's difficulties during the tests. *Despite* her aphasia, the verbal modality condition produced a better movement performance than the copy condition. MT had a tendency to perseverate in her movement actions and when gesturing object use. She made some body-part-as-object (BPO) errors, but was able to use two of the six objects (comb and pen) correctly in the real/tactile condition. The other objects in the test were used incorrectly but in a recognisable manner. The gestural movement errors made during testing were noted to be more frequent when the movement related to her own body and face, whereas her fine distal movements away from the body were mainly well acted. She was unable to copy any *sequences* of movement.

During the *ideational* apraxia test (appendix D4) many sequencing errors were made. For example, MT was unable to carry out any meaningful activity with the objects for lighting the candle. During the tea-making part of the test, MT made such errors as pouring water into the sugar bowl, placing the teabag into the saucer, making slicing gestures with the teaspoon, and 'pouring' gestures with the sugar bowl. In attempting to lock the box MT placed the padlock and key inside the box and repetitively opened and closed the lid of the box. All these errors indicated the presence of both ideomotor and ideational apraxia.

6.2 Aims of the study.

Using this single case, it was considered that an exploration of the natural history of apraxia would add to the understanding of the condition. This study would :

- monitor the natural progression of recovery over a period from 3 - 14 months post-Sub Arachnoid Haemorrhage (SAH) of a person with apraxia.
- examine the kinematic profile of one individual over time.
- determine whether the kinematic features noted in apraxic subjects from the group study were seen in this individual and whether those features changed over time.
- explore the relationship between clinical assessment scores and kinematic features of movement performance over time.

6.3 Procedure

MT was assessed at the Rivermead Rehabilitation Centre and on each occasion was seen in the same room using the same methods and equipment previously described for both apraxia examination and kinematic recording. This study recorded MT's performance in apraxia tests and in the drinking task. These were carried out at intervals during her rehabilitation process and on one occasion after discharge from the rehabilitation centre. Five apraxia assessment scores were obtained over an eleven month period and, after the initial clinical screening, four kinematic recordings were made over the following nine months. During the investigation MT participated in daily therapy sessions which included physiotherapy, occupational therapy and speech and language therapy.

6.4 Results

6.4(i) Apraxia assessments

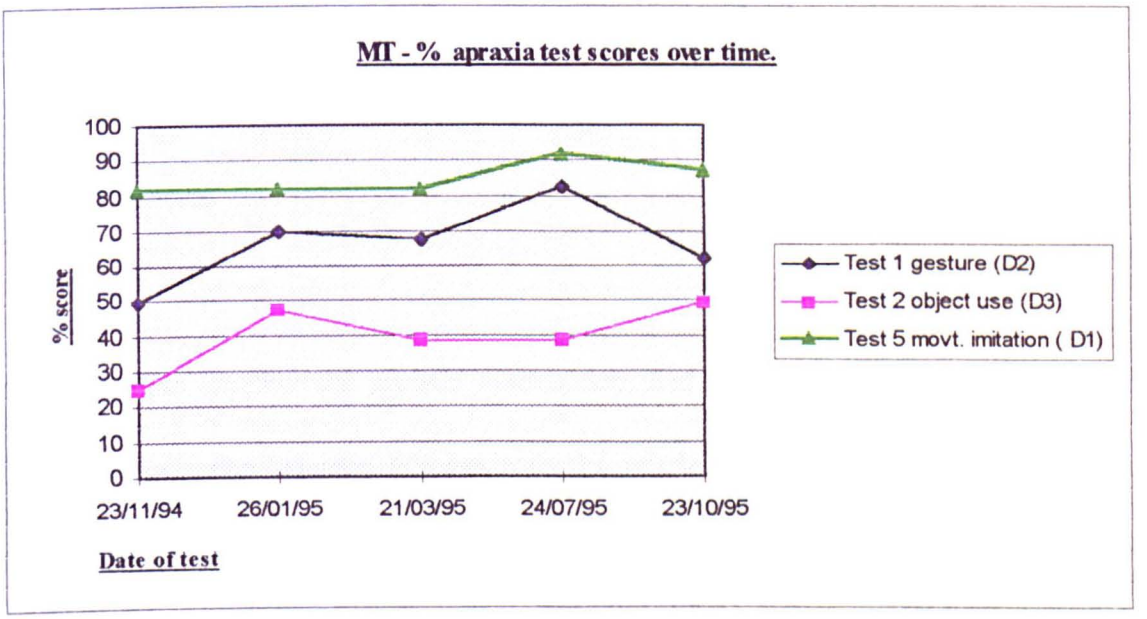
Despite all scores on the clinical assessments showing an improvement over an 11 month period when Time 1 (T1) and Time 5 (T5) are compared (Table 6.1), none of the patterns observed gave a 'straight-line' progression from T1 to T5. Test 4 was particularly erratic but over a small range (75% - 90%) while Test 1 was similarly erratic but over a greater range (52% - 82.5%). It was also noted that Test 5 scores remained unchanged from T1-T3 before showing improvement, while Tests 1 and 5 peaked at T4 (11 months after the cerebral insult). Throughout the eleven month period under review, following MT's admission to the rehabilitation centre, MT displayed many errors in attempting to copy sequences of movement and this did not appear to alter appreciably over time. At T4, her ability to gesture to verbal command was noticeably improved, but this was not sustained

Table 6.1. MT apraxia assessment scores (raw and %) over time.

Clinical Apraxia Tests *(Time post-SAH)*	**23/11/94** *(3 mths.)* **T1**	**26/1/95** *(5 mths.)* **T2**	**21/3/95** *(7 mths.)* **T3**	**24/7/95** *(11 mths.)* **T4**	**23/10/95** *(14 mths.)* **T5**
Test 1 - gesture copying (max. = 40) (appendix D2)	20 (50%)	28 (70%)	27 (67.5%)	33 (82.5%)	25 (62.5%)
Test 2 - object use (max. = 36) (appendix D3)	9 (25%)	17 (47.22%)	14 (38.89%)	14 (38.89%)	18 (50%)
Test 5 - DeRenzi et al. (max. = 72) (appendix D1)	59 (81.94%)	59 (81.94%)	59 (81.94%)	66 (91.67%)	63 (87.5%)
Mean % scores for Tests 1, 2 & 5.	52.31%	66.39%	62.78%	71.02%	66.67%
Test 3 - ideational apraxia (max. = 12) (appendix D4)	6 (50%)	7 (58.33%)	8 (66.67%)	6 (50%)	12 (100%)
Test 4 - agnosia (max. = 20) (appendix E)	15 (75%)	18 (90%)	17 (85%)	16 (80%)	17 (85%)

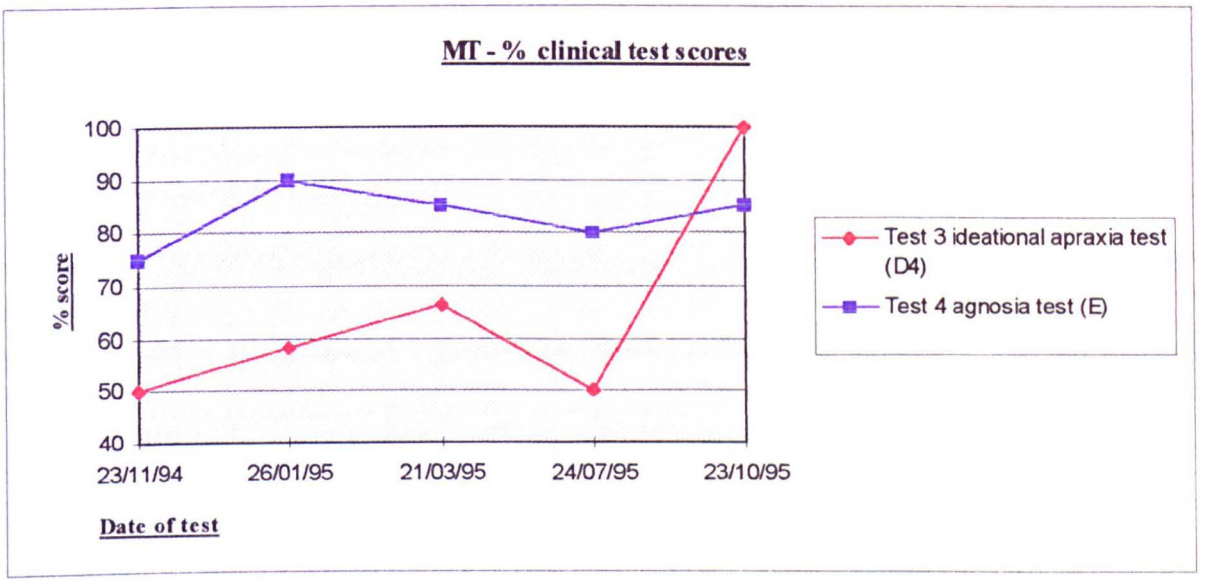
through to the assessment at T5. MT's ability to *copy* gestures gradually improved over time as did her abilities to use several objects in a sequential and meaningful way. The trend of change between the period covering 3-14 months post-stroke is illustrated in Figures 6.1 and 6.2. Some test scores decreased from T4 to T5. This latter period

Figure 6.1: MT % ideomotor apraxia assessment scores over time



covered the time from discharge from the rehabilitation centre when MT spent the majority of her time in her home rather than participating in a rehabilitation programme. This may, then, be evidence of deterioration in motor performance in the absence of therapy. It does, however, contrast with the most notable improvement in test performance (that of the ideational apraxia test score) which occurred during that latter time spent away from the rehabilitation centre. This score rose from 50% to a full 100% at the last assessment date (Figure 6.2).

Figure 6.2 - MT % ideational apraxia and agnosia test scores over time.



To determine whether the apraxia assessments had indicated any significant change in MT's performance between admission to the rehabilitation centre and her discharge, a paired t-test was used on the individual scores in the data. This analysis confirmed a significant difference between **all** the clinical assessment scores shown in Table 6.1 from T1 to T5 [$t(4) = -4.49$ $p < .05$]. When, however, the ideomotor apraxia test scores **only** were analysed (tests 1, 2 and 5), no significant differences were found over time [$t(2) = -3.93$ *NS*]. The lowest percentage change in score (5.57%) of all the clinical assessments between the first and last assessment of MT was that from the DeRenzi et al (1980) test (test 5 in Table 6.1). This test was shown to be the most sensitive and reliable of the ideomotor apraxia tests in the group study (chapter 5). It may be, therefore, a more reliable indicator of change in MT's praxic ability or, rather, the lack of it than the 25%

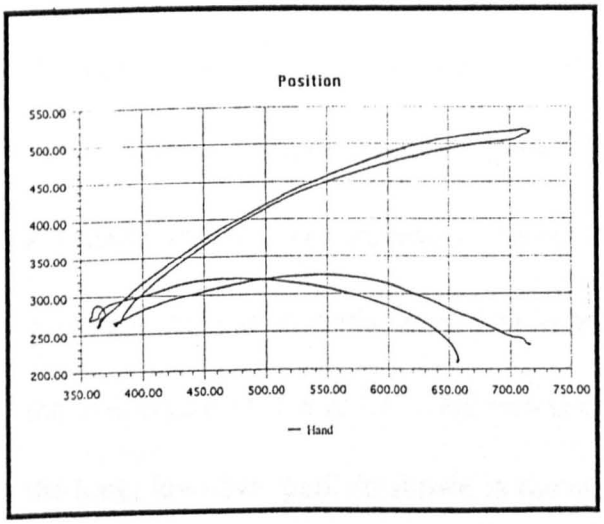
increase in score shown in the object use test (appendix D3) which might have reflected a change in MT's comprehension ability more than praxic ability.

6.4(ii) Kinematic analysis

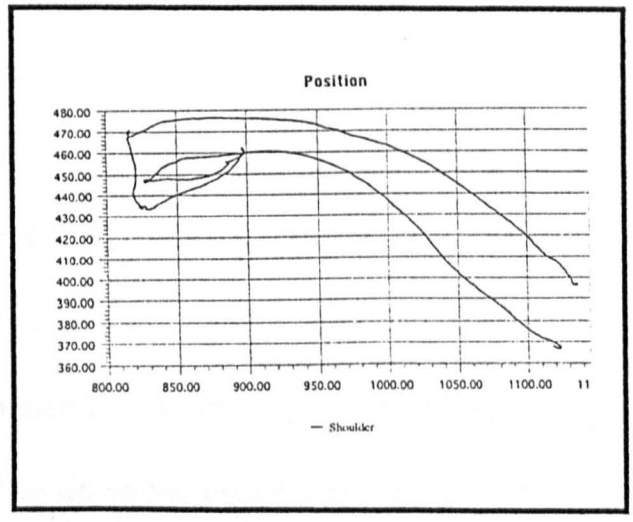
At the first trial in the verbal condition, the kinematic analysis of the drinking task showed that MT had a good spatial trajectory as seen in the hand recording in Figure 6.3. However, the shoulder movement revealed a pattern more typical of the apraxic condition as witnessed in the group study evidence previously described (see Figure 6.3), with extraneous movement and a loss of spatial consistency in the movement pattern.

Figure 6.3 : MT Drinking task hand and shoulder trajectories, verbal condition.

a] Hand trajectory



b] Shoulder trajectory

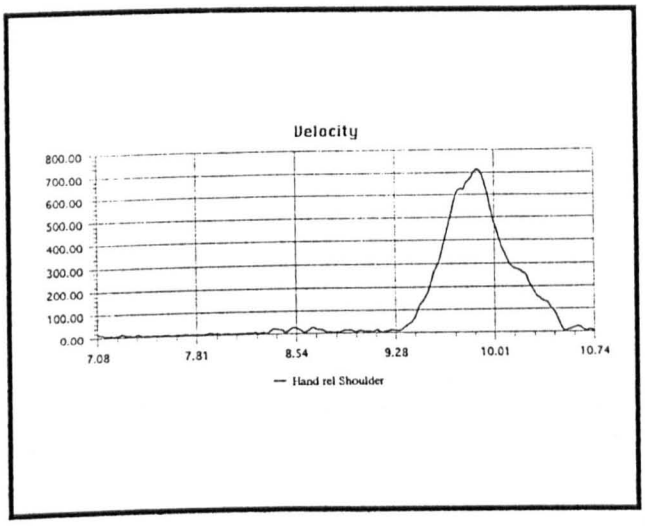


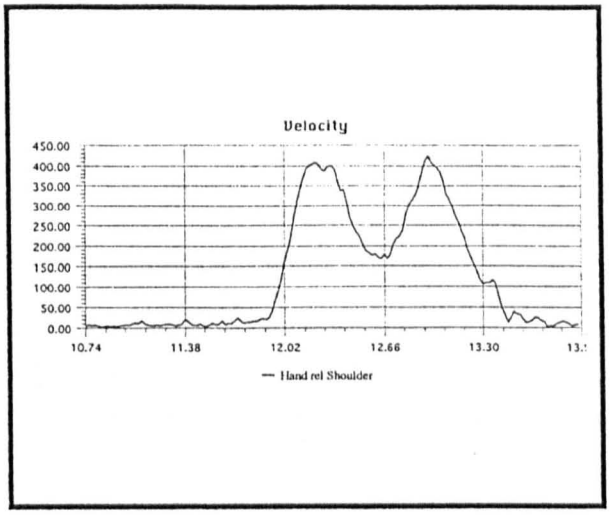
The velocity patterns shown by MT throughout this verbal condition performance showed clear low-level periods between the different phases of the task (Figure 6.4).

Such patterns were observed in the group study (chapter 5) and in previous published research (Fisk & Goodale 1988) as being a feature of apraxic movement.

Figure 6.4 : MT - velocity patterns in phases of the drinking task, verbal condition.

a] Lift Phase **b] Down Phase**

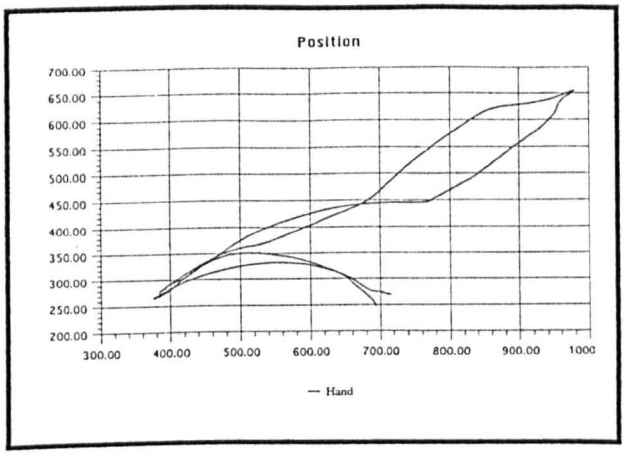




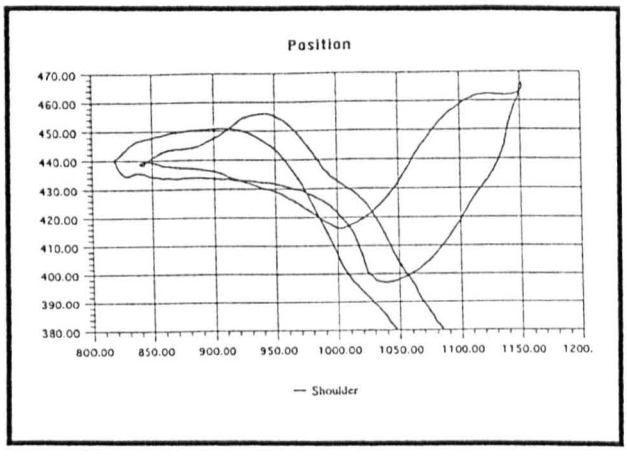
The *copy* condition of the experimental drinking task performed by MT at the first trial (time 2) showed very different patterns from those of the verbal condition in the kinematic analysis. The trajectory recording for the copy condition demonstrated a less consistent pattern of spatial alignment in the hand and an overuse of the shoulder during the task (Figure 6.5 a & b). This was accompanied by velocity patterns which had lost the long, low-level periods shown in the verbal condition but gained more of the multi-peak shape associated with apraxic movement (see Figure 6.5 c & d).

Figure 6.5 : MT - kinematic analysis of the copy condition of the drinking task.

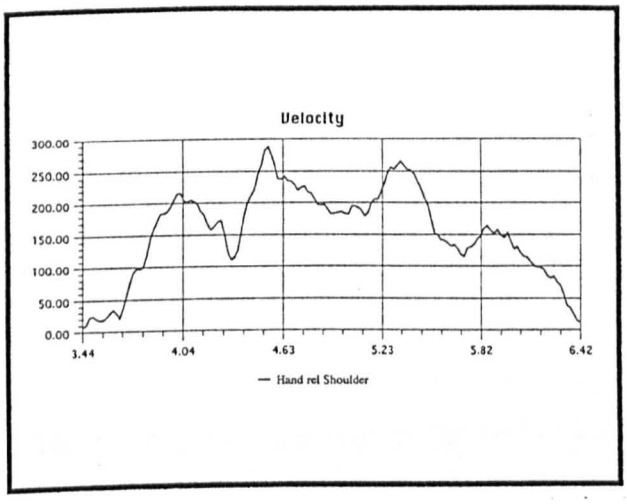
a] Hand trajectory



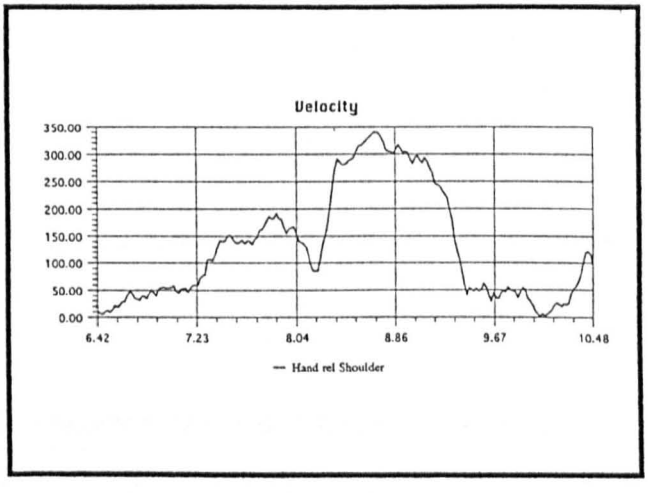
b] Shoulder trajectory



c] Velocity patterns - Lift Phase



d] Velocity patterns - Down Phase

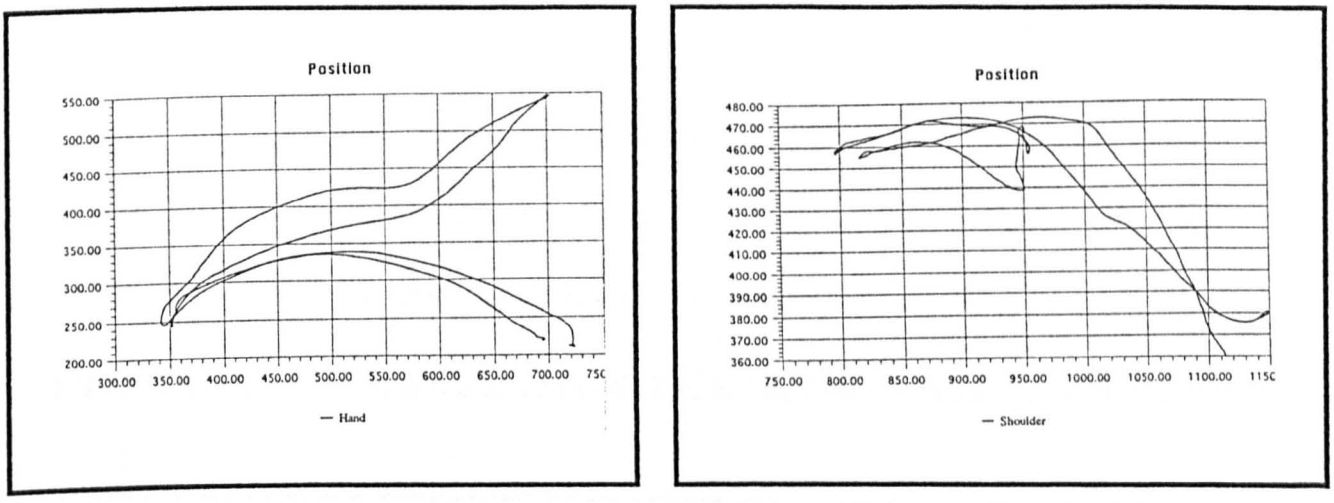


The real-object-use condition of the drinking task at the first trial showed different trajectory patterns yet again. MT demonstrated a different spatial pathway in using her hand to carry the mug up to her mouth and then replace it onto the table top. She again over-used her shoulder to complete this task but in a different manner than in the previous two task conditions (Figure 6.6) but consistent with trajectory patterns demonstrated in the group study as being associated with the apraxic condition. The

different sensory input from the weight of the mug, the tactile sensations from gripping the handle and the need to orientate the object correctly all impact on the required motor output. The ability to integrate the information and produce a smooth motor performance was shown in the group study to be impaired in apraxic subjects.

Figure 6.6 : MT - Trajectory patterns. Drinking task, real-object-use condition.

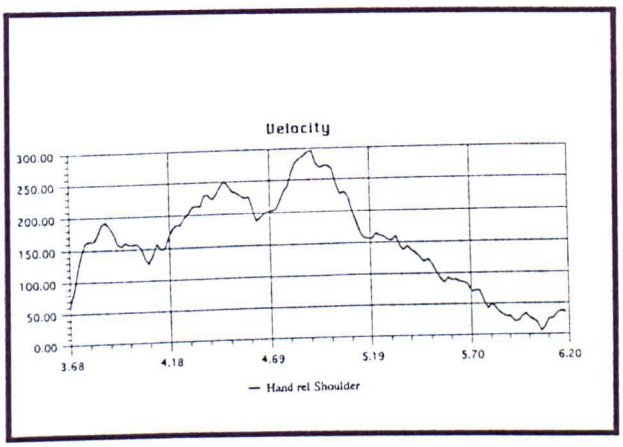
a] Hand trajectory **b] Shoulder trajectory**



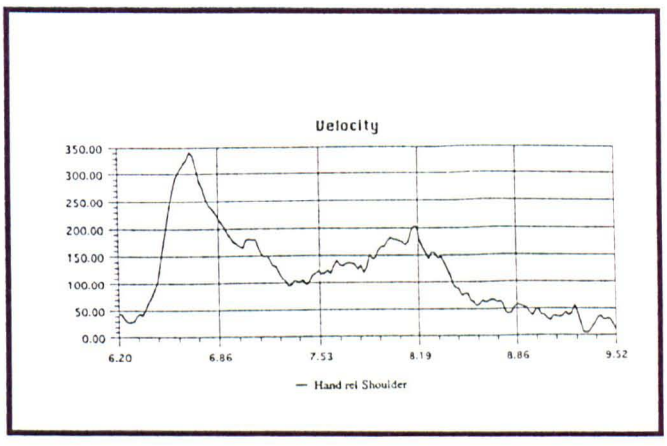
The velocity patterns seen in the real-object-use condition retained a similarity to those seen in the copy condition except in the 'down' phase where an early peak in the phase was followed by a long slowing down period in preparation for placing the mug onto the table top (Figure 6.7).

Figure 6.7 : MT -Velocity patterns; drinking task, real-object-use condition.

a] Lift Phase

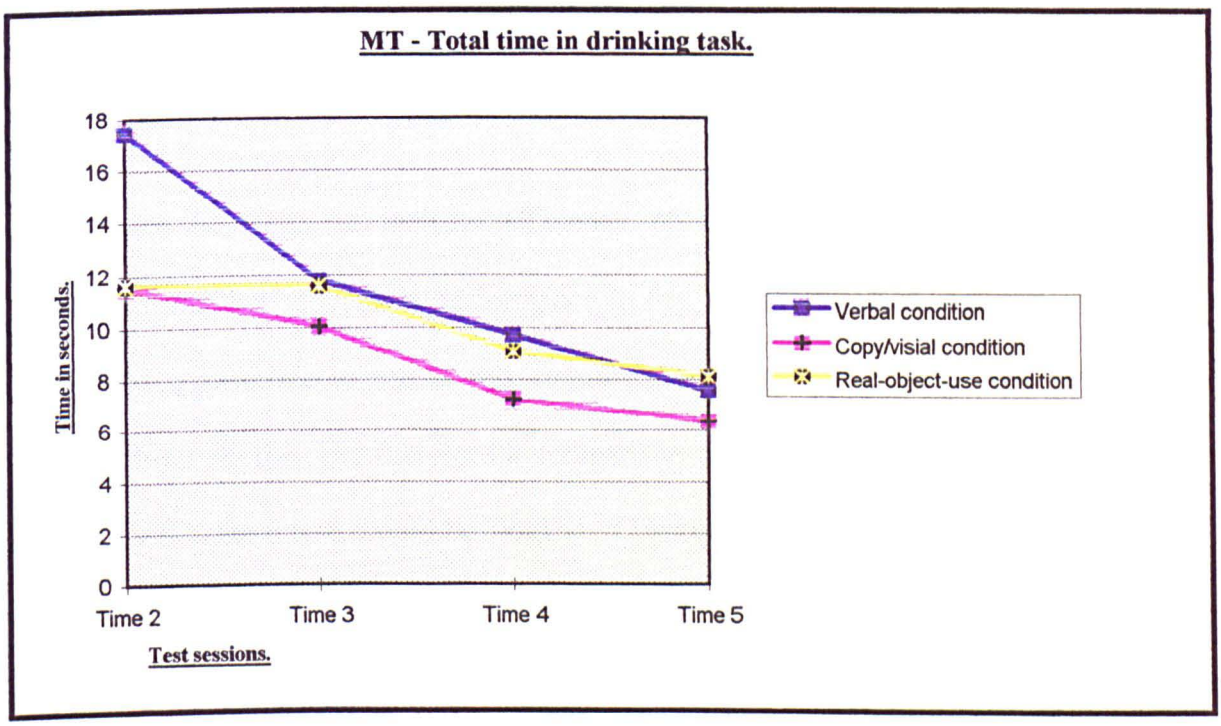


b] Down Phase



Over the nine month period covered by the kinematic testing period, MT demonstrated a gradual increase in speed and efficiency of movement. A significantly shorter total time was taken to complete the task in all conditions (Figure 6.8) and shorter times were

Figure 6.8 : MT - Total time taken to complete the drinking task. All conditions.



shown in each *phase* of the task (Table 6.2).

Table 6.2 : MT- Kinematic timing data over time.

Timing data (in seconds)	23/11/94 T1	26/1/95 T2	21/3/95 T3	24/7/95 T4	23/10/95 T5
Verbal condition					
Total time	*Clinical*	17.46	11.82	9.74	7.54
Reach phase	*tests*	5.36	2.88	2.04	1.70
Lift phase	*only*	3.66	3.2	1.04	1.52
Down phase	*on this*	3.2	2.94	5.48 *	1.62
Return phase	*date.*	3.36	1.48	1.18	1.84
Copy/visual condition					
Total time		11.54	10.04	7.22	6.36
Reach phase		3.20	1.2	1.54	2.02
Lift phase		2.98	2.84	2.10	1.60
Down phase		4.06	2.78	2.24	1.60
Return phase		1.06	1.78	1.30	1.14
Real-object-use condition					
Total time		11.62	11.58	9.08	8.10
Reach phase		2.78	3.14	2.14	1.50
Lift phase		2.52	2.54	2.38	1.92
Down phase		3.32	3.56	2.92	2.84
Return phase		2.10	2.24	1.58	1.84

** Perserveration error during this phase with repetition of action.*

If *time* can be taken to indicate efficiency of action, then MT demonstrated an improvement in her movement performance in this aspect. ANOVA statistics were not considered appropriate to use in this circumstance as each task phase was inherently different from another and therefore a comparison of differences between those phases, inherent within the F test, would be illogical. Consequently a paired t-test was completed on the timing data for all phases and all conditions of the drinking task to determine whether significant changes occurred between the start and the finish of the investigation (T2 and T5). Significant differences in time were shown between T2 and T5 using data

from all conditions [t(11) = 4.58 p< .001]. In analysing each experimental condition independently, however, only the verbal condition showed significant differences in phase timing between T2 and T5 [t(3) = 4.47 p< .05]. No significant differences in phase timing were shown in the visual/copy condition [t(3) = 2.37 *NS*] or in the real-object-use condition [t(3) = 2.98 *NS*].

Analysis of the influence of the agnosia test on drinking task performance (total time for completion) was also carried out. High correlations were found between the test and the verbal condition performance (r= .74 *NS*), the copy condition (r= 98 *NS*) and the real-object condition (r=.87 *NS*) though with such a small data set none reached a level of statistical significance. Multiple regression analysis showed that whilst the agnosia test accounted for more of the variance (54.9%) than the De Renzi apraxia test score (46.7%) when each was put first into the regression equation, neither tests were predictive of performance in the total time to complete the drinking task in the *verbal* condition [F(1,2)=2.43 *NS*. and F(1,2)=1.75 *NS*]. A similar effect was found for the copy condition with the agnosia test accounting for 96.9% of the variance, whilst the De Renzi test accounted for 88.3% of the variance when each was put first into the regression equation but, again, neither test was predictive of performance in the total time to complete the drinking task in the *verbal* condition [F(1,2)=32.13 *NS*. and F(1,2)=7.55 *NS*]. The real-object condition *did* show a predictive effect from the De Renzi test which accounted for 99.8% of the variance [F(1,1) = 5292 p< .01] whilst the agnosia test accounted for 76.1% of the variance when put first into the regression equation but was not predictive of timed performance [F(1,1) = 3.2 *NS*]. These statistical results should be viewed with some caution due to the small data available, but nevertheless the agnosia test is clearly

accounting for more of the variance in the copy and real conditions of the drinking task than in the verbal condition. This supports the evidence from the group study.

The kinematic analysis of the drinking task also revealed that MT showed a tendency towards increased velocity of movement over time. However, this increase in velocity was not consistent through all phases nor in all conditions. If an increase in velocity peak is taken as an indicator of improved motor performance, then the overall pattern shown by MT (Table 6.3) suggested such an improvement over time. The group study data, on

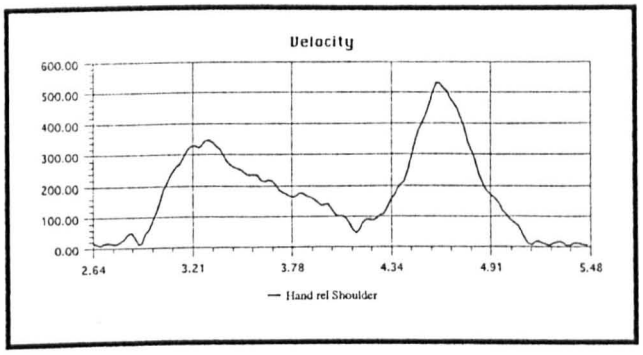
Table 6.3 : MT- Velocity peak data over time.

Velocity peak (in mm/sec.)	23/11/94 T1	26/1/95 T2	21/3/95 T3	24/7/95 T4	23/10/95 T5
Verbal condition					
Reach phase	*Clinical*	150.72	192.53	206.14	341.0
Lift phase	*tests*	713.57	262.11	431.70	518.23
Down phase	*only*	425.00	459.07	602.61	638.14
Return phase	*on this*	292.96	700.98	439.52	532.25
Copy/visual condition	*date.*				
Reach phase		175.32	267.93	297.33	325.26
Lift phase		287.79	530.76	519.27	530.12
Down phase		341.00	579.81	462.86	595.18
Return phase		404.11	415.07	634.12	641.67
Real-object-use condition					
Reach phase		239.62	467.75	254.83	395.78
Lift phase		296.27	271.62	344.99	454.19
Down phase		342.11	370.46	290.26	384.71
Return phase		399.01	390.83	602.23	625.76

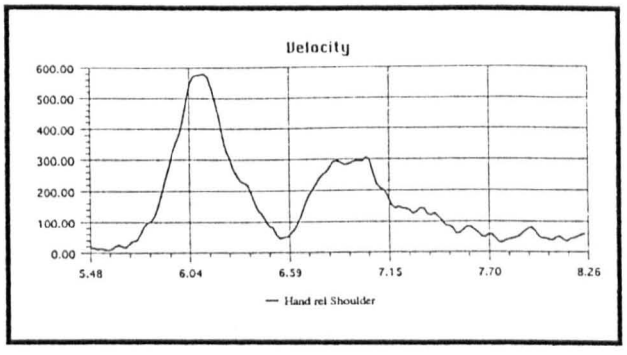
the other hand, demonstrated conflicting evidence concerning the usefulness and relevance of such peak velocity data. Consequently, using this element of the data as an indicator of 'improvement' in MT must be with caution.

Figure 6.9 : MT - examples of velocity peak patterns over time.

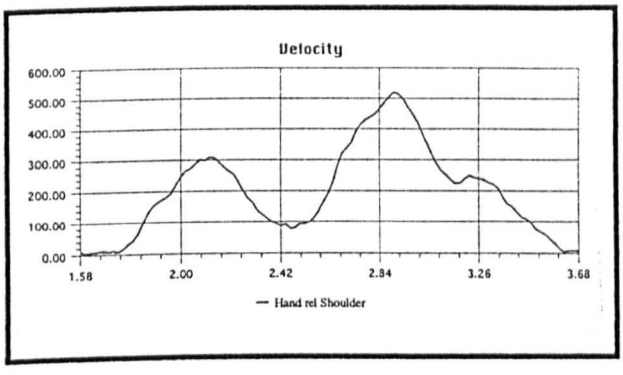
a] Time 3 - Lift Phase (copy condition)



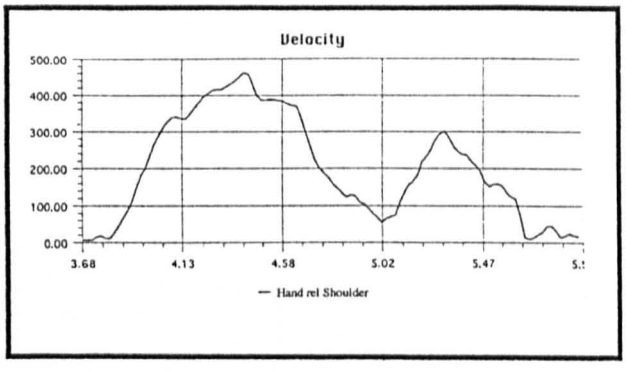
b] Time 3 - Down Phase (copy condition)



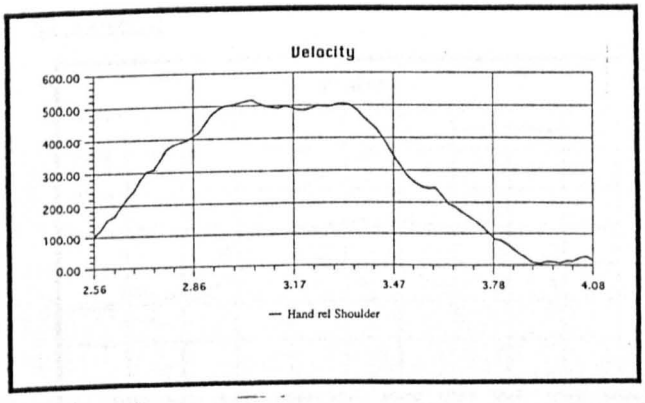
c] Time 4 - Lift Phase (copy condition)



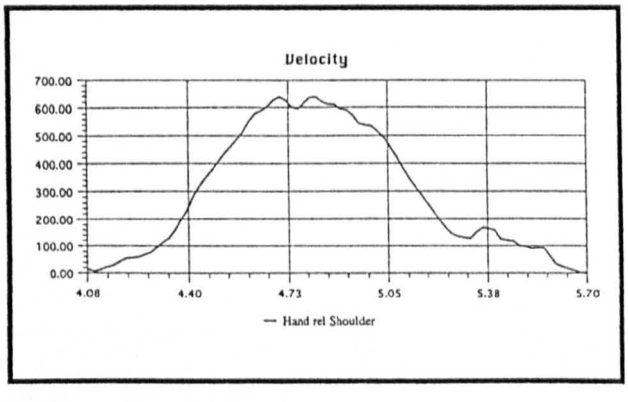
d] Time 4 - Down Phase (copy condition)



e] Time 5 - Lift Phase (copy condition)



f] Time 5 - Down Phase (copy condition)

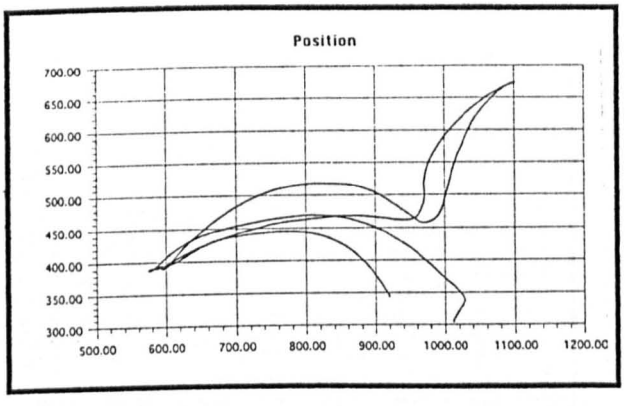


Other improvements were demonstrated by the trajectory patterns and the shape of the velocity curve elicited during each phase of the experimental task (Figure 6.9). As time

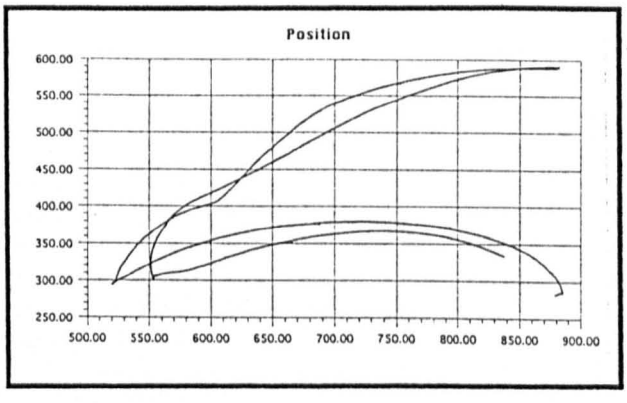
progressed, the velocity shape began to lose some of the multi-peak phenomena seen in the first test session, along with the long, low-level velocity periods. A larger, wider, bell-shaped curve began to emerge in some of the recordings of movement velocity, with overall smoother recording patterns (Figure 6.9). As can be seen, the shapes moved from the multi-peak type with features identified as being associated with apraxic subjects in the group study, to bell-shaped patterns more akin to those seen in the non-apraxic and control subjects. The trajectory patterns also show similar, though not as marked,

Figure 6.10 : MT - Examples of hand trajectory over time. Visual/copy condition.

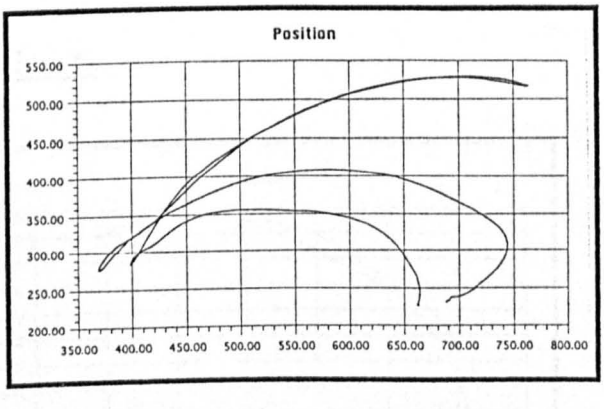
a] Time 3.



b] Time 4.



c] Time 5

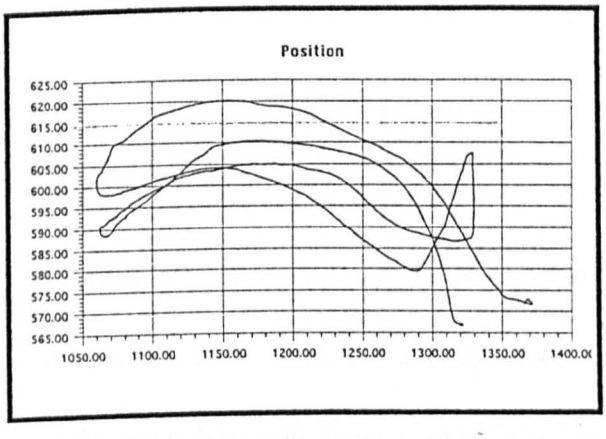


changes over time. This was illustrated by the hand trajectories in the visual/copy condition with smoother, more aligned spatial pathways being used during the task (Figure 6.10)

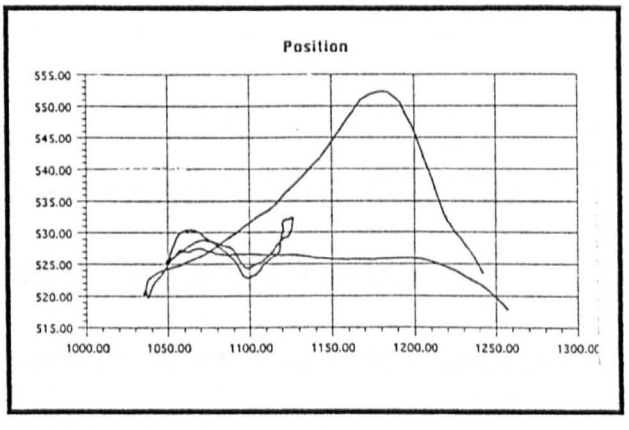
The recording of the shoulder movement over time remained abnormal (despite some changes) when compared with the patterns associated with the non-apraxic and normal control groups shown in the group study (Figure 6.11).

Figure 6.11 : MT - Examples of shoulder trajectory over time. Visual/copy condition.

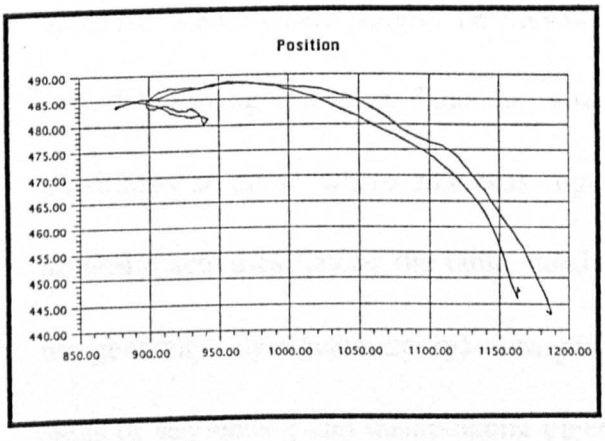
a] Time 3.



b] Time 4.



c] Time 5.



Changes were, therefore, demonstrated over an eleven month period (3-14 months post SAH) in both clinical assessment performance and in kinematic elements of movement performance in a naturalistic drinking task. MT also improved slightly in her levels of independence, with an end score of 12 of a possible 20 on the Barthel Index, though she remained partically dependent for feeding, transfers, mobility, climbing stairs, dressing and bathing..

6.5 Discussion

The general trend of improvement seen over time in the clinical assessment scores could be taken to indicate some improvement in praxic function. This improvement, though small, enabled MT to assist and participate in her functional self-care activities though this change was barely picked up by the Barthel score change due to its broad category scoring system. MT remained severely disabled and required assistance for most activities. The ideomotor apraxia assessment scores themselves did not show significant differences between Time 1 and Time 5 in MT's history of recovery, but a large change was seen in the ideational apraxia test score between Time 4 and Time 5, though this apparent improvement should be viewed with caution. This change occurred in the period following discharge from the rehabilitation centre. It could be argued that the conditions at home where MT was regularly and constantly helping and assisting in domestic activities (laying the table, dusting the house, preparing meals, washing/drying up, generally tidying/clearing up) were giving her many daily opportunities to practice the skills of sequencing and manipulating objects. The quantity and quality of such practice

opportunities in this domain could not be achieved in the rehabilitation centre when other therapy sessions, daily programme constraints and therapist availability would all work to limit these practice opportunities. At home, with her husband constantly enabling MT to practice, she worked to achieve as much as she was able. Indeed, she was able to devote her day to such practice activities. Alternatively, and a more likely explanation, concerns MT and her husband having prior knowledge of the test components. MT may have practiced and rehearsed similar specific tasks at home in an attempt to 'aid recovery' and thus this score may be merely the result of a practice effect not true change in abilities.

Kinematic improvements *were* observed over time, and again this could be considered supportive evidence for the dissociation between clinical tests and kinematic components of movement performance. It has been shown that MT made gradual improvements in timing in the drinking task over time (Table 6.2) and significant differences were shown between Time 2 and Time 5 (7 and 14 months post trauma) in speed of task completion. Extrapolating from the data analysed in the group study, where the non-apraxic subjects were quicker than the apraxic subjects and where the control subjects were the quickest of all, it might be expected that such reduction in time to complete each phase of the task shown by MT could be taken as objective and robust evidence of improvement in praxic performance.

Similarly the patterns of spatial alignment shown in the trajectory of the hand during the drinking task changed in the direction consistent with those seen in the non-apraxic and control subjects of the group study. This was particularly noticeable in the visual/copy

condition. The over-use of the shoulder by MT during the task was also shown by the kinematic recordings to be reduced over time, mirroring the pattern seen in the group study data by the non-apraxic and control subjects. This supports previous research suggesting that spatial accuracy in movement performance is associated with normal praxis (Hermsdorfer et al 1996).

The velocity patterns shown by MT in the early stages showed major difficulties in changing from one phase of the task to the next. This was consistent with other research evidence which indicated difficulties and hesitancy in transition stages of movement in apraxic subjects (Poizner et al 1990). These long low-velocity periods between the task phases diminished over time, as did the multi-peaked patterns also suggestive of the apraxic condition. At the last recording assessment MT demonstrated the bell-shaped velocity patterns associated with normal controls in the group study, and with skilled pre-programmed movements (Hermsdorfer et al 1996). She showed a general trend towards higher velocity peaks over time. This may be taken as evidence to indicate improvement or recovery of praxic abilities and would support the work of Fisk and Goodale (1988) where apraxic subjects were shown to have lower velocity peaks in movement performance than control subjects.

It could be argued that all such kinematic changes demonstrated in the natural history of MT demonstrated an improvement in praxic abilities, both in relation to temporal and spatial components. This appears to be the first kinematic evidence of recovery in apraxia and represents the start of objective and quantifiable data in monitoring change in the apraxic condition.

The dissociation between the kinematic components of movement and the clinical test profiles is demonstrated in this single case *over time*, and supports the evidence presented in the single occasion 'snapshot' measurements taken in the group study (Chapter 5). MT clearly had some difficulty in comprehending the verbal command sections of some of the clinical tests although she was able to understand and complete the verbal condition of the drinking task with accuracy (Figure 6.3). The hesitancy shown between the phases of the drinking task in the verbal condition was in keeping with the body of research which has suggested that apraxic people have the most difficulty when formulating their own internally generated motor representations (De Renzi et al 1980, Poeck et al 1982, Alexander et al 1992). Despite her aphasia MT performed better on some of the verbal command sections of the clinical tests than in the visual/copy sections. The scores achieved by MT in the visual/copy sections of the clinical tests gradually changed over time but showed only small improvements. This was partly due to the category system of scoring where a performance might be qualitatively 'better than last time' yet could still only be scored in the 'impaired performance' category. Thus subtle changes could not be recorded except as qualitative, observational comments and were not reflected in the test score. In contrast, the kinematic analysis of the visual/copy condition of the drinking task demonstrated change over time in a clear, objective, quantitative form.

In this single 'natural history' study, the influence of the agnosia test in relation to action performance on the drinking task in the copy and real-object conditions has been shown. Whilst not reaching a level of statistical significance, possibly due to the small data set,

such results add weight to the evidence of the group study in suggesting that this test might be reflecting the intact vision-to-action routes available to apraxic patients.

6.6 Conclusion

This 'natural history' investigation of apraxia has shown changes over time particularly in kinematic components of movement performance. The kinematic features changed from patterns associated with apraxia to those more in keeping with control subject data previously described. This study has demonstrated kinematic analysis of change and recovery in an apraxic subject and is the first to report such changes in this field. It is suggested that the kinematic analysis has provided objective data and evidence of change more than that shown by observational data, apraxia test scores or independence levels as measured by the Barthel index.

The negligible change in clinical assessment scores supported the evidence for a dissociation between the clinical measures and kinematic analysis of movement. In addition, a relationship between the agnosia test and the movement performance in the experimental drinking task was demonstrated and added support to similar findings from the group study.

Further longitudinal data are needed to verify this first finding. Particularly valuable would be to look at whether the changing patterns in the kinematic analyses were replicated across subjects. It would also be interesting to see whether there were consistencies between the different types of apraxia in terms of the scope for improvement.

Chapter 7. CASE 2 : *LS. Head injury. Spontaneous recovery from apraxia.*

7.1 Case Background

LS, a 17 year old left handed man, sustained a closed head injury resulting in a right hemisphere haemorrhage particularly affecting the frontal lobe. During the road traffic accident he also sustained abdominal injuries and a pelvic fracture. On presentation for assessment four months after his accident, LS demonstrated left sided weakness and a mild ataxia of gait. His speech and language function remained unimpaired. This presentation suggested a left hemisphere dominance for language with right hemisphere dominance for motor control (handedness). His pre-morbid cognitive abilities were judged to be low average and his schooling had been impaired by dyslexia. He was currently attending his local Technical college, training to work as a chef. There was a suggestion from his foster parent that he might have had developmental dyspraxia as a child, but had attained full motor abilities and skills and, for example, was able to use knives with competence in his training as a chef.

Psychological assessment completed half-way through this study indicated that LS had impaired memory function, poor visuo-spatial attention span and impaired performance on the Wisconsin Card Sorting Test consistent with impaired frontal lobe function. Initial clinical assessment of LS (Table 7.1) provided evidence for the presence of a mild ideomotor apraxia expressed when the motor task demands were high, either in complexity of movements or in spatial aspects. During the assessment procedure and recording sessions there was evidence of poor concentration and attention, with prompts needed to alert LS to the task in hand. Informed consent to participate in the study was

given by LS following the normal procedures. This included informing LS's foster parent of the project and obtaining parental agreement for him to participate.

7.2 Aims of the study

Using this single case, it was considered that assessing and monitoring LS's progress would provide evidence to -

- replicate the previous 'natural history' study but with a different underlying pathology.
- monitor kinematic changes in apraxic performance weekly to look for variability and short-term changes.
- relate kinematic performance to cognitive and visuo-spatial abilities.
- explore the kinematic components of gestural and object-use performance.

7.3 Procedure

LS was assessed at the Rivermead Rehabilitation Centre. On each occasion he was seen in the same room using the same methods and equipment previously described for both apraxia testing and kinematic recording. Due to LS's left hemiparesis and his left-handedness, it was decided to assess and record all movements using the non-dominant right hand. This would replicate the previous studies in non-dominant upper limb movement recordings and address the need to exclude hemiparetic effects on movement. During a six-week period, LS was recorded on eight occasions performing the experimental drinking task in each of the three modality conditions. On each occasion, to replicate in part the studies of Poizner et al (1990), additional recordings were made of movement gestures. Instructions given were :

i] 'Show me how you would hold a hammer and use it' (HAMMER)

ii] 'Show me how you would hold a saw and use it' (CARVE)

iii] 'Show me how you would wind down a car window' (WIND)

These were each repeated in a visual/copy modality condition (i.e. "Copy this" The researcher then demonstrated the movement).

The clinical apraxia assessment battery was completed at the beginning and end of the six week period. A further recording of the drinking task and the three additional gestural movements was made a month later. During the period under investigation LS participated in daily therapy sessions which included physiotherapy (concentrating on activities for balance, coordination and stamina) and occupational therapy (concentrating on self care skills, social and community skills, activities and projects to enhance mobility, stamina, arm function and higher-level cognitive function).

At the end of the 10 week period of investigation, and in the light of the results from kinematic analysis, visuo-spatial abilities were assessed paying particular attention to body schema. The Rivermead Perceptual Assessment battery indicated impairment ***only*** in 3D complex shape copying. A further assessment was made in LS's ability to judge distance of objects, both from his own body and from other objects. The requested judgments were of a variety of short and longer distances within the normal, familiar assessment environment.

7.4 Results

During the assessment procedures and kinematic recordings, LS had some difficulty sustaining his attention to the task and needed occasional prompts from the researcher.

He was often impulsive in his actions, with a tendency in the apraxia tests to attempt gestures or movements before the full instructions or demonstrations had been given. On such occasions he was asked to stop and wait until the full instructions or demonstration had been given. He was able to comply with these instructions but it resulted in a second demonstration of the test item being given which was outside the given research protocol. On all occasions in testing, LS gave the impression of trying his best to achieve the movement or gesture requested, though this was frequently accompanied by inappropriate remarks or lengthy comments about his performance. Whilst this made testing a lengthy procedure it was not judged to interfere significantly with the research protocol.

7.4(i) Apraxia assessments

Initial clinical assessment of LS (Table 7.1) provided evidence of mild ideomotor apraxia.

Table 7.1 : LS - Apraxia assessment scores (raw and %) over time.

Clinical Tests *(Time post-HI)*	23/4/96 ***(4 mths.)*** **T1**	10/6/96 ***(5.5 mths.)*** **T2**
Test 1- gesture copying (max. = 40) (appendix D2)	38 (95%)	40 (100%)
Test 2 - object use test (max. = 36) (appendix D3)	29 (80.5%)	36 (100%)
Test 5 - DeRenzi test, gesture copying (max. = 72) (See appendix D1)	56 (77.7%)	70 (97.2%)
Mean % scores for Tests 1, 2 & 5 (ideomotor apraxia).	84.4%	100%
Test 3 - ideational apraxia (max. = 12) (See appendix D4)	11 (91.6%)	12 (100%)
Test 4 - agnosia (max. = 20) (See appendix E)	19 (95%)	20 (100%)

Apraxic errors were shown when the motor demands were high, either in movement complexity or spatial complexity. The De Renzi test (appendix D1) elicited the most errors from LS, especially in the complex finger and hand gesture copying, and in sequencing of movement. The most common errors made were movements or hand positions being in the wrong plane. Timing errors were also exhibited. LS made body part as object (BPO) errors in the verbal command section of the object-use test (appendix D3). At times he used his left, rather than right, hand to perform the gestures during testing and required reminding of the procedure. This might be regarded as evidence of an attentional difficulty.

LS's performance in the ideational apraxia test showed evidence of problems associated with frontal lobe dysfunction rather than apraxic errors. Most of the tasks were eventually completed successfully, but he displayed difficulties in planning, organisation and anticipation of consequences of his actions. He required prompting during this test to complete it, but did recognise when he had made errors (e.g. realising he had padlocked the box but had left the lid open, he corrected his error by closing the lid and then re-padlocking the box successfully).

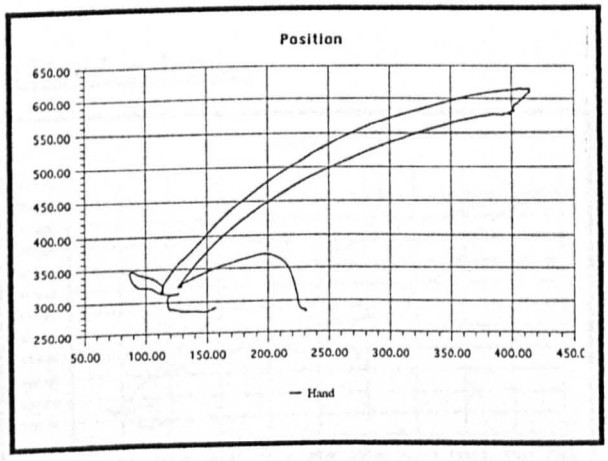
Reassessment six weeks later, using clinical tests, showed no convincing evidence of apraxia (Table 7.1) with only minimal difficulty remaining in successfully copying two of the complex finger movements in the De Renzi test.

7.4(ii) Kinematic analysis

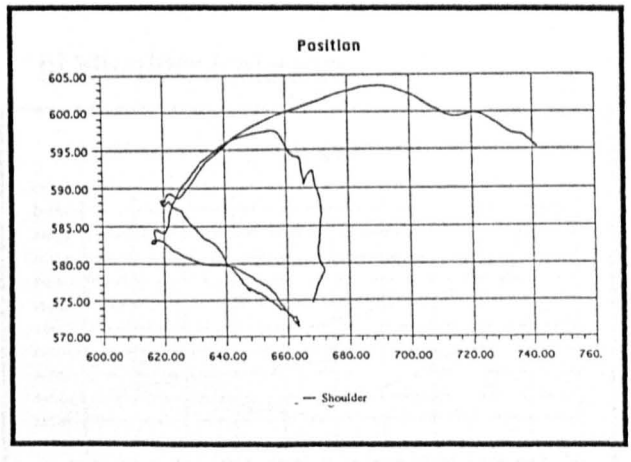
Kinematic analysis at the initial recording showed LS displaying a trajectory pattern consistent with those associated with apraxia in the group study. He was inconsistent in his spatial use of movement in bringing his hand to his mouth and returning it down again (Figure 7.1a). LS did not return his hand to the correct finishing position during the task, but stopped the task by resting his hand on the table top a little way from the target 'red spot' and verbally reporting that he had finished. The pattern of his shoulder movement during the task was also typical of those seen in the apraxic subjects in the group study (Figure 7.1b). Velocity patterns during the verbal condition of the drinking task showed evidence for long, low-level periods at transition stages of movement particularly between the lift and down phases (Figure 7.1c & d).

Figure 7.1: LS - Kinematic data from the drinking task. Time 1, *Verbal* condition

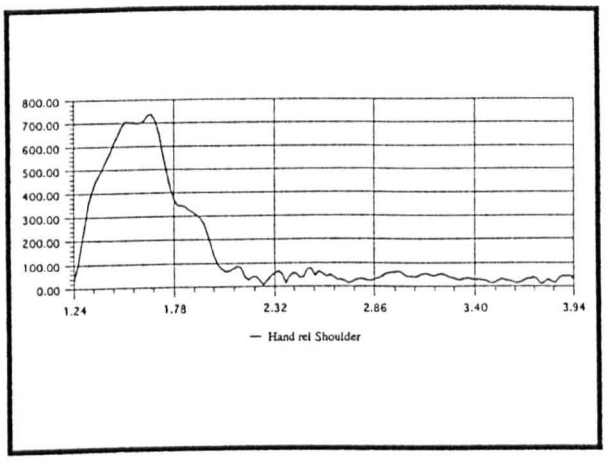
a] Hand Trajectory



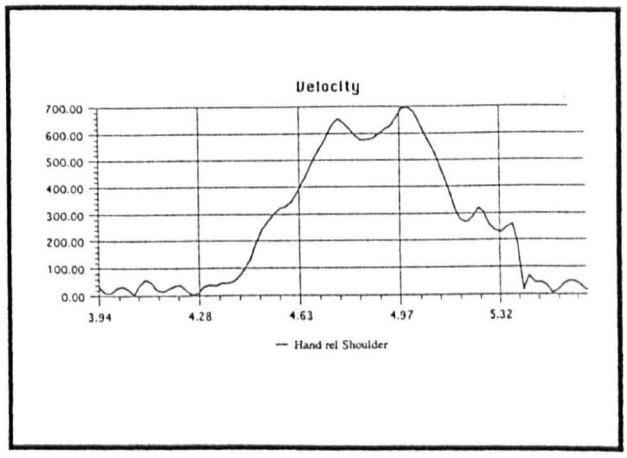
b] Shoulder trajectory



c] Velocity - Lift phase



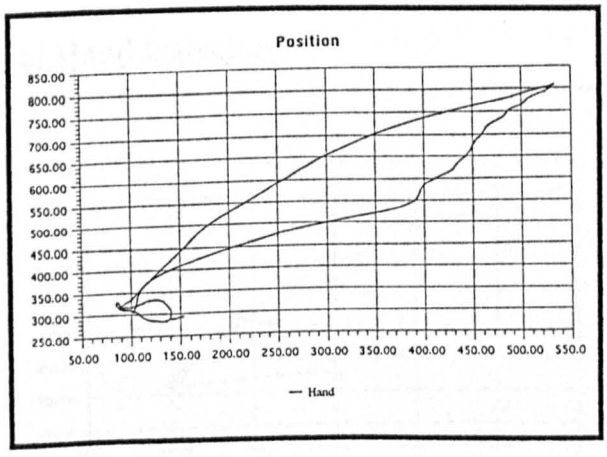
d] Velocity - Down phase



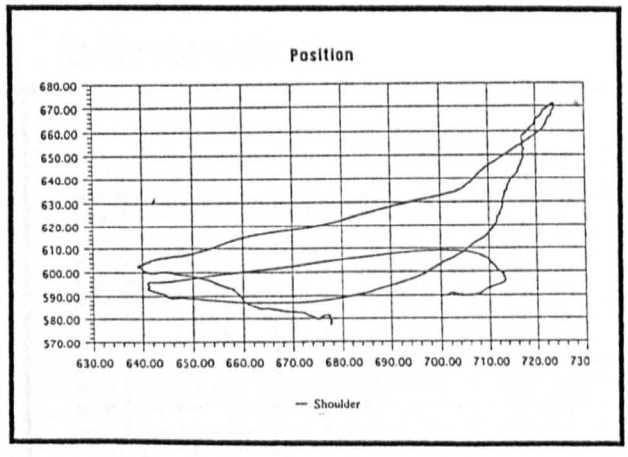
In the copy condition of the drinking task LS was again unable to comply with the start and finish-point rigours of the protocol (Figure 7.2a). The hand trajectory exhibited during the lift and down phases of the task demonstrated an inconsistent use of space during the movement execution. Over-use of the shoulder was also seen in the recordings (Figure 7.2b) as was some evidence of low velocity periods at the end of phases though this was less extreme than in the verbal condition (Figures 7.2 c and d).

Figure 7.2 : LS - Kinematic data from the drinking task . Time 1, *Copy* condition.

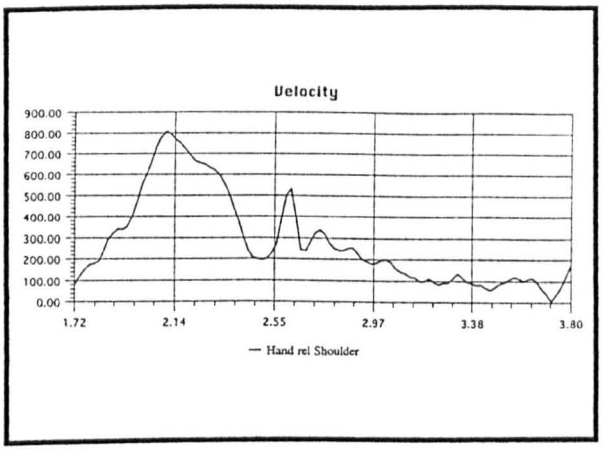
a] Hand trajectory



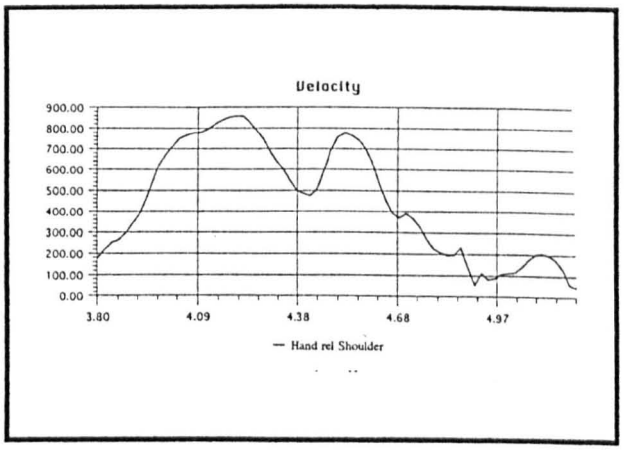
b] Shoulder trajectory



c] Velocity - Lift phase



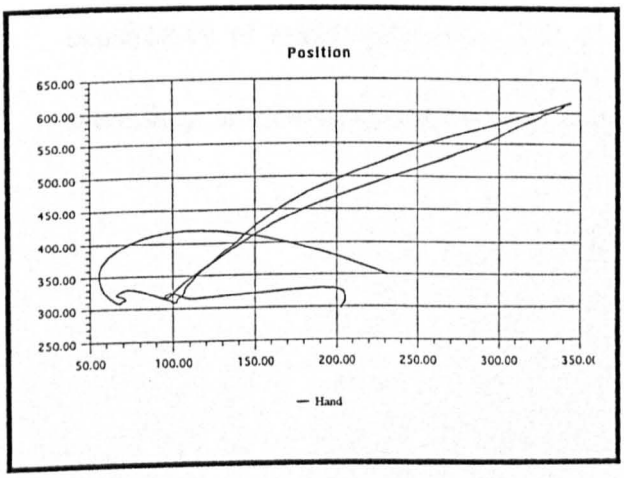
d] Velocity - Down phase.



The real-object-use condition of the drinking task at Time 1 produced recordings from LS which demonstrated a better spatial consistency in the lift and down phases than had been seen in the other conditions. However there were extraneous movements and inconsistent patterns in the reach and return phases of the task (Figure 7.3a). Over-use of the shoulder was also evident (Figure 7.3 b) with extraneous little movements of that joint being picked up in the recordings.

Figure 7.3 : LS Kinemaic data; drinking task. Time 1, *real-object-use* condition.

a] Hand trajectory



b] Shoulder trajectory

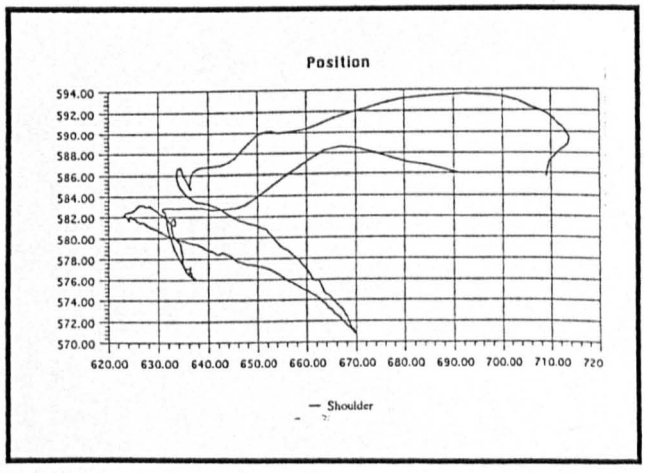
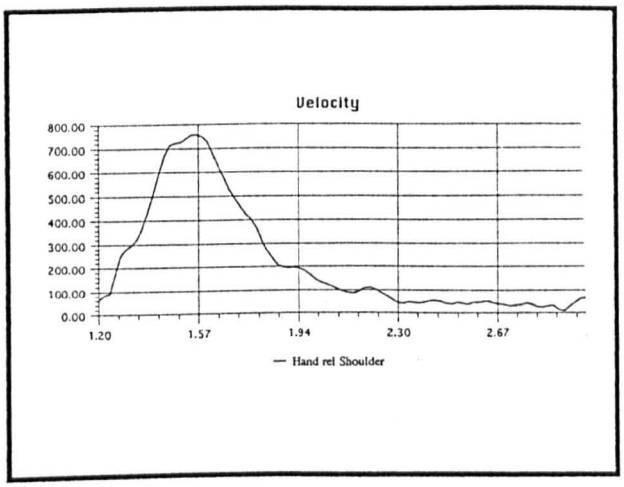
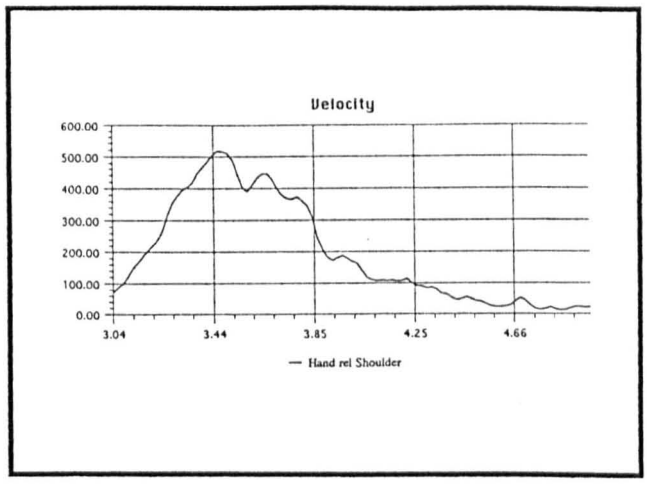


Figure 7.3 (cont.)

c] Velocity - Lift phase



d] Velocity - Down phase

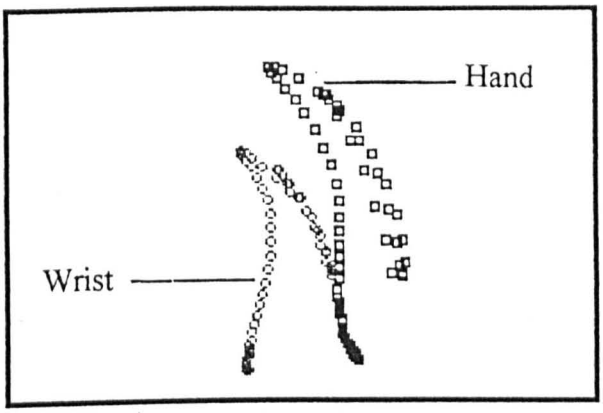


Again, in the real-object-use condition, the low-level periods in velocity patterns were seen between task phases (Figure 7.3 c & d) indicative of hesitation during transition stages of movement. These patterns have been associated with apraxic movement in the group study.

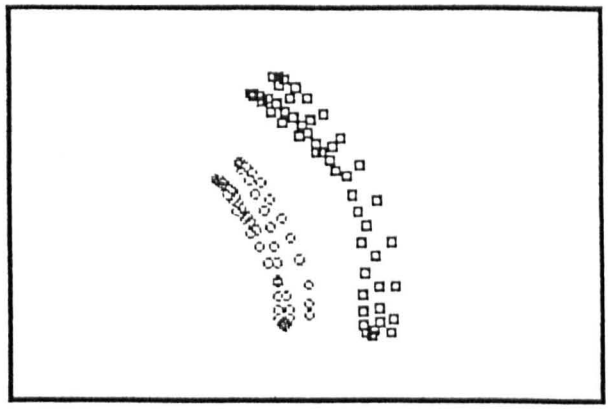
The three object-use pantomimes (as described by Poizner et al 1990) were examined in both verbal and copy conditions. In each of the gestures requested, LS demonstrated a better performance in the copy condition than the verbal condition as shown in the consistency of spatial pathways used by the hand (Figure 7.4 - wrist and hand marker recordings are shown during one 'cycle' of movement).

Figure 7.4 : LS - Object -use pantomime. Time 1.

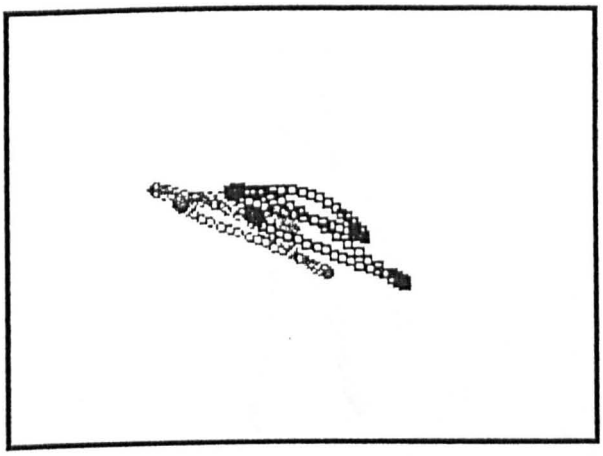
a] 'HAMMER' - verbal condition



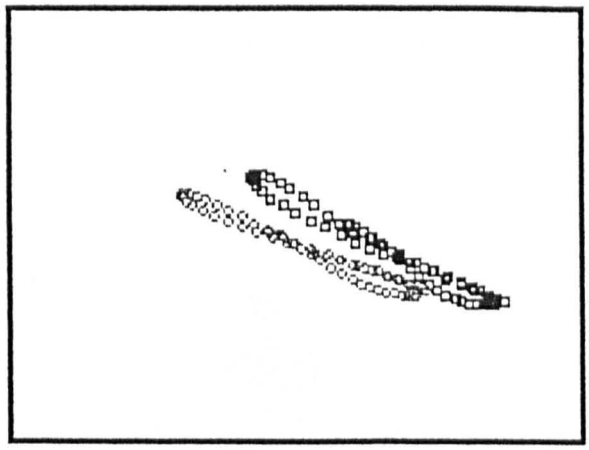
b] 'HAMMER' - copy condition



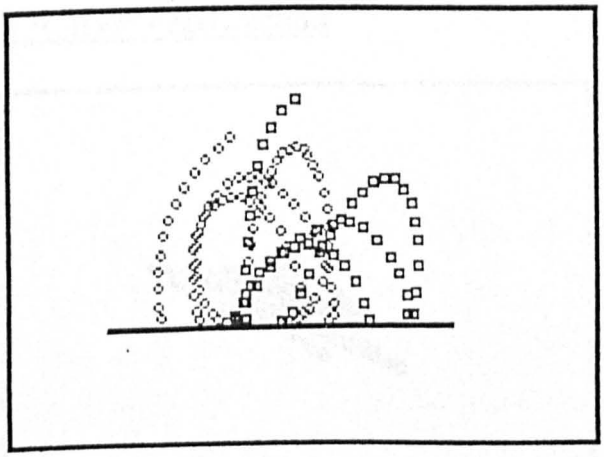
c] 'CARVE' - verbal condition



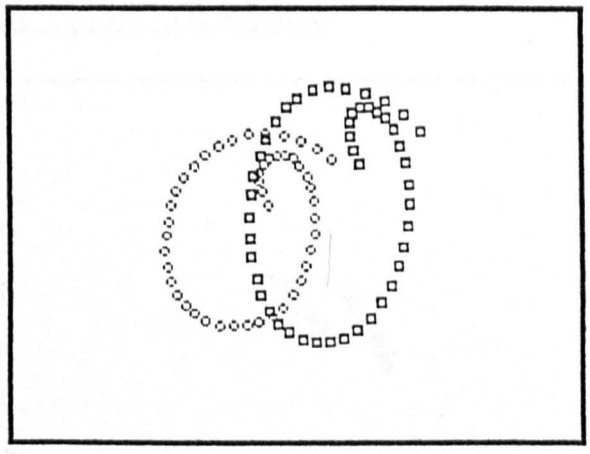
d] 'CARVE' - copy condition



e] 'WIND' (car window) - verbal condition



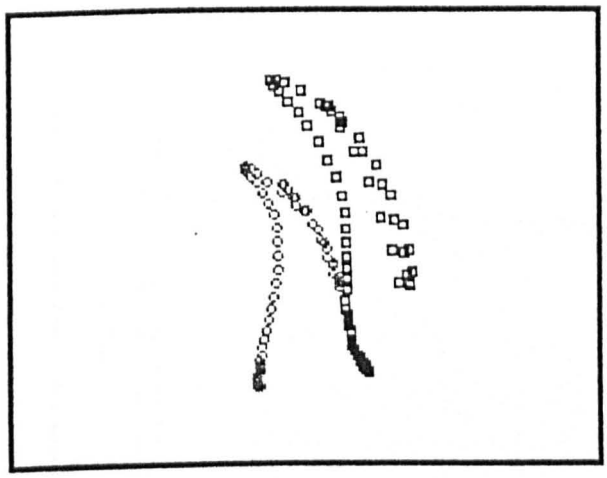
f] 'WIND' (car window) - copy condition



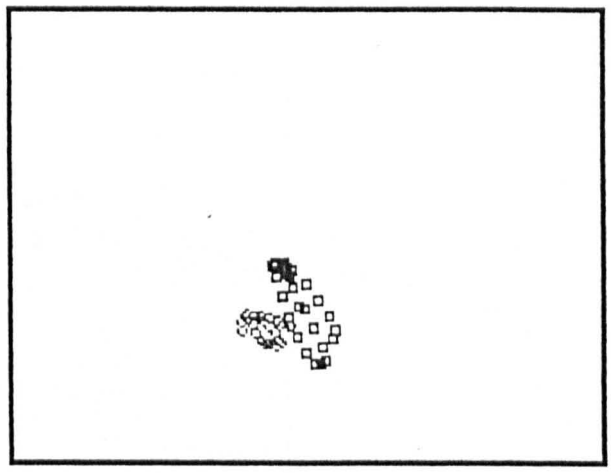
Unfortunately in the 'winding a car window' action, LS moved in such as way as to be outside the range of the recording equipment. Despite this, the expected circular movements made by the hand in gesturing the action could be seen to be less spatially consistent in the verbal condition than the copy condition (Figure 7.4 e & f). An additional feature noted in these pantomimed actions was the increasing poverty of movement displayed as the action continued (Figure 7.5).

Figure 7.5 : LS - Pantomimed action in time sequence.

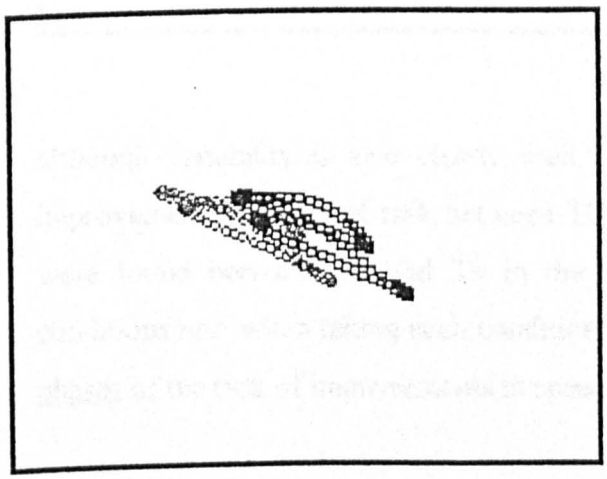
a] 'Hammer' - early action



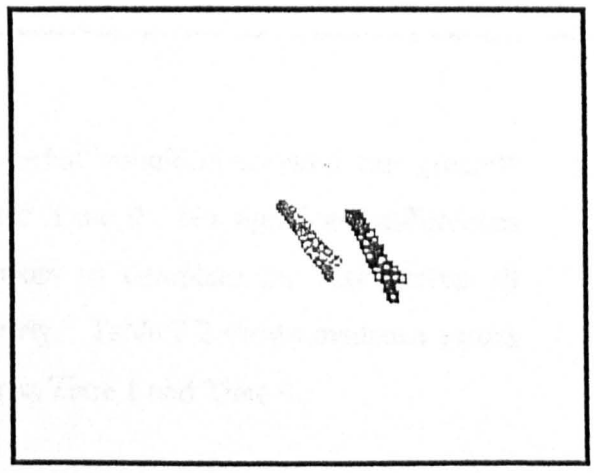
b] 'Hammer' - later action



c] 'Carve' - early action



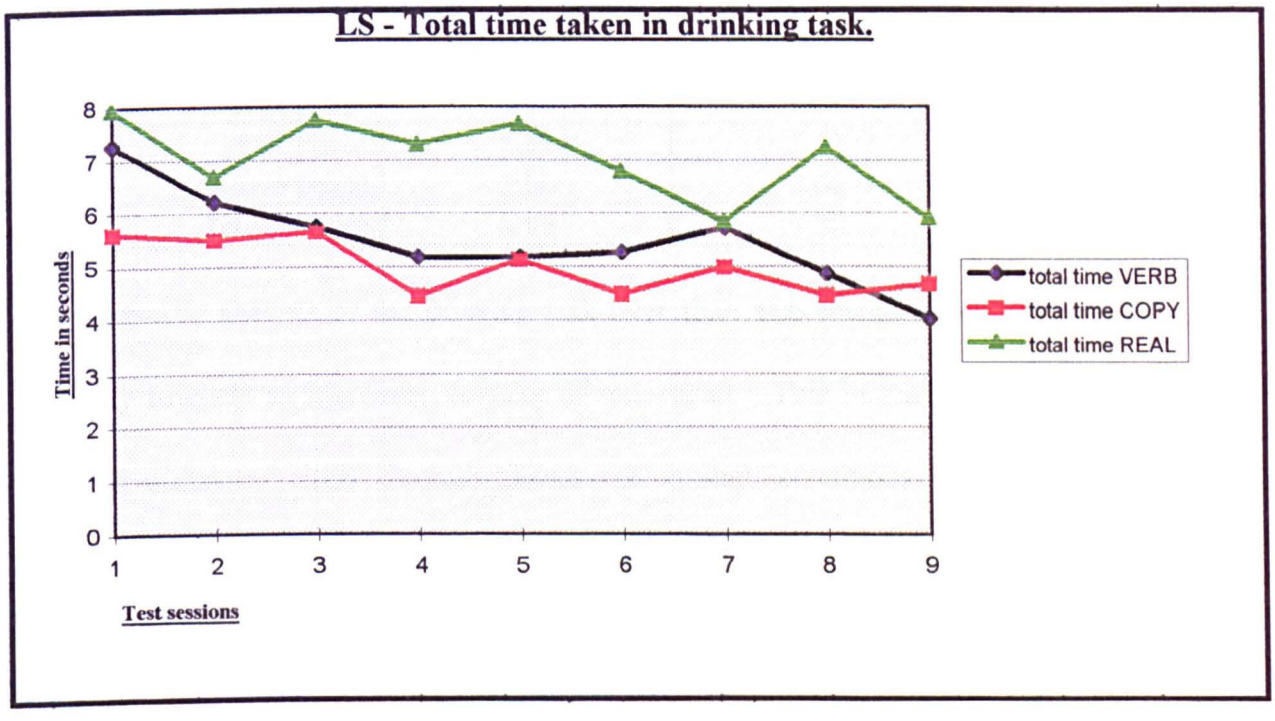
d] 'Carve' - later action



This appeared to be a feature only of the verbal condition where, as the seconds ticked by, LS was unable to sustain the complete movement action and gradually diminished his motor output.

Over the six week period, and on the last recording a month further on, LS displayed changes in his movement performance consistent with the improvements noted in the apraxia test scores. LS demonstrated a trend of quicker performances in the drinking task (Figure 7.6)

Figure 7.6 : LS - Total Time taken in drinking task - all conditions.



although variability is also clearly seen. The verbal condition showed the greatest improvement in speed of task between Time 1 and Time 9. No significant differences were found between T1 and T9 in the time taken to complete the task across all conditions nor when taking each condition separately. Table 7.2 shows evidence across phases of the task of improvements in speed between Time 1 and Time 9.

Table 7.2 : LS - Time taken in phases of the drinking task at T1 and T9

	29/4/96 **T1**	1/7/96 **T9**
Verbal condition		
Reach phase	1.04	0.84
Lift phase	2.70	1.16
Down phase	1.72	1.14
Return phase	1.80	0.88
Copy/visual condition		
Reach phase	1.06	0.92
Lift phase	2.10	1.50
Down phase	1.48	1.36
Return phase	0.98	1.88
Real-object-use condition		
Reach phase	1.16	0.90
Lift phase	1.84	1.62
Down phase	2.00	2.08
Return phase	2.96	1.30

The kinematic analysis of the drinking task over time also revealed that LS showed a tendency towards an increased velocity of movement. This increased velocity pattern was not consistent either through phases nor through conditions. Table 7.3 clearly demonstrates, however, the velocity changes between Time 1 and Time 9. These differences whilst not statistically significant [$t(11)= -1.78$ *NS*] might, nevertheless, be taken as indicators of improved motor performance since they are at least consistent with evidence from the group data study and the MT case study previously described.

Table 7.3 : LS - Velocity peak data at T1 and T9.

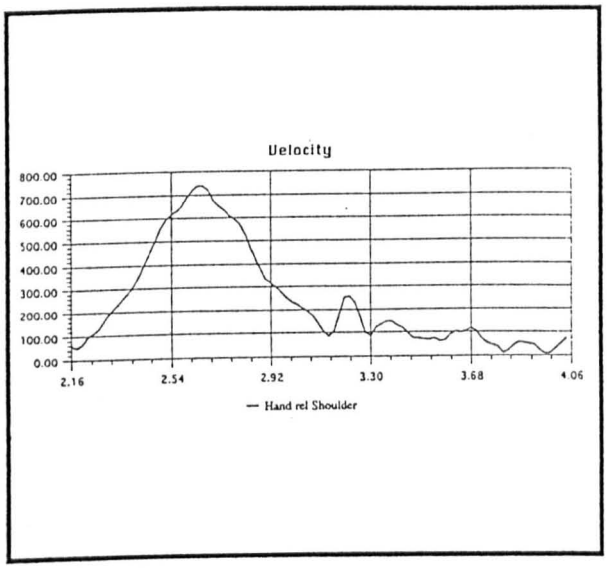
	29/4/96 **T1**	1/7/96 **T9**
Verbal condition		
Reach phase	325.69	357.03
Lift phase	736.96	950.45
Down phase	696.86	1079.30
Return phase	231.41	651.36
Copy/visual condition		
Reach phase	210.00	378.21
Lift phase	809.09	732.60
Down phase	857.90	1162.18
Return phase	181.67	652.84
Real-object-use condition		
Reach phase	160.61	222.04
Lift phase	757.63	666.04
Down phase	516.45	626.79
Return phase	564.59	378.57

Other changes, consistent with evidence for improvement in movement performance, were demonstrated by the velocity curves for each of the phases of the task. As with the previous case study (MT), the velocity patterns demonstrated by LS gradually lost the long, low-level periods between task phases which had been evident at the start of the study (Time 1). *Examples* of these changes in velocity patterns (Figure 7.7) illustrate the gradual appearance of the normal bell-shaped curve associated with the control group data described in the group study and reported elsewhere (Hermsdorfer et al 1996).

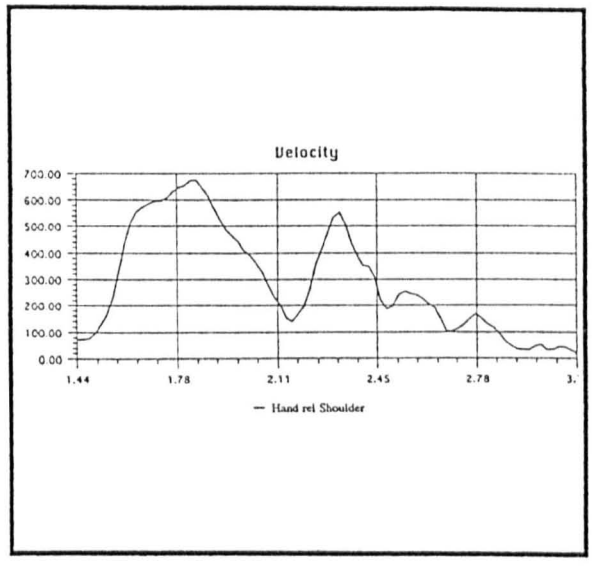
Figure 7.7 : LS - examples of velocity pattern changes over time.

a] Lift phase, *verbal* condition

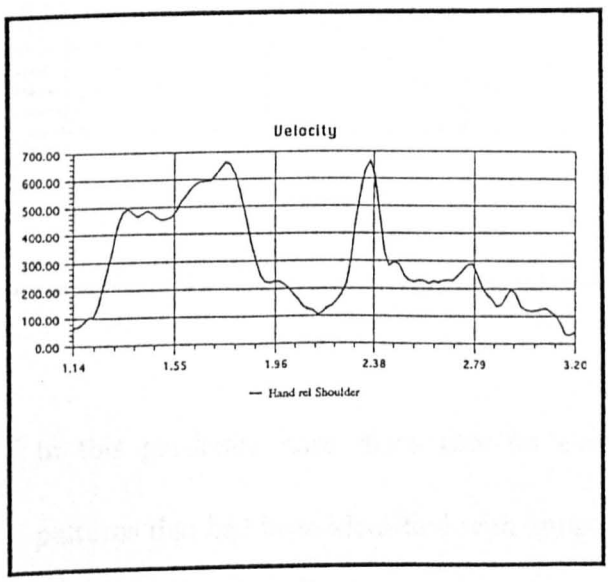
Time 2



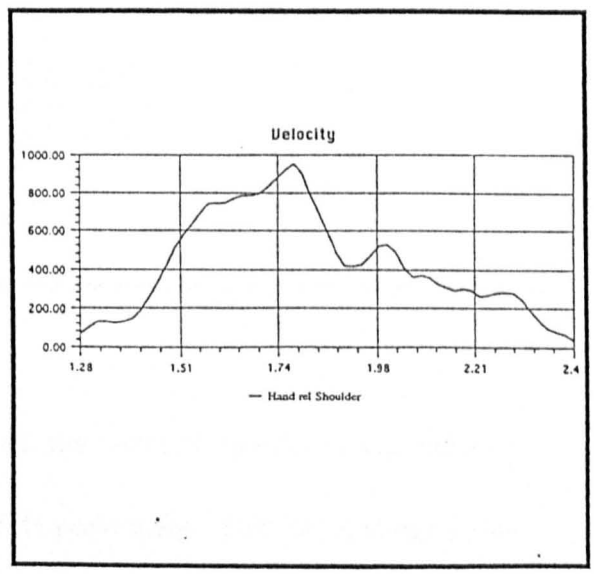
Time 4



Time 6

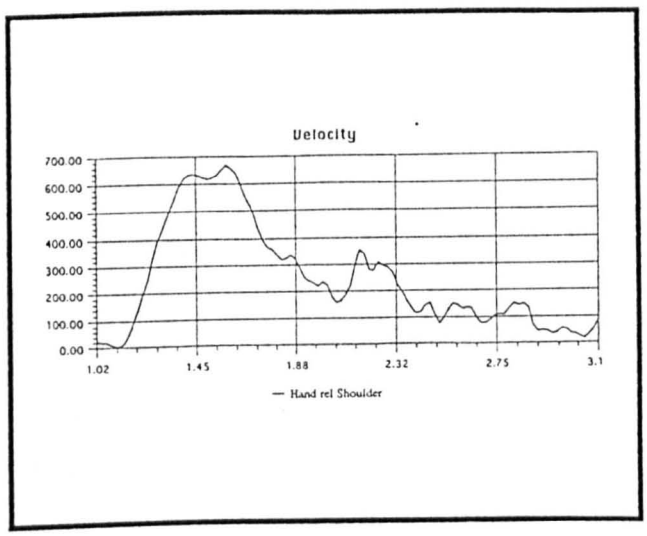


Time 9

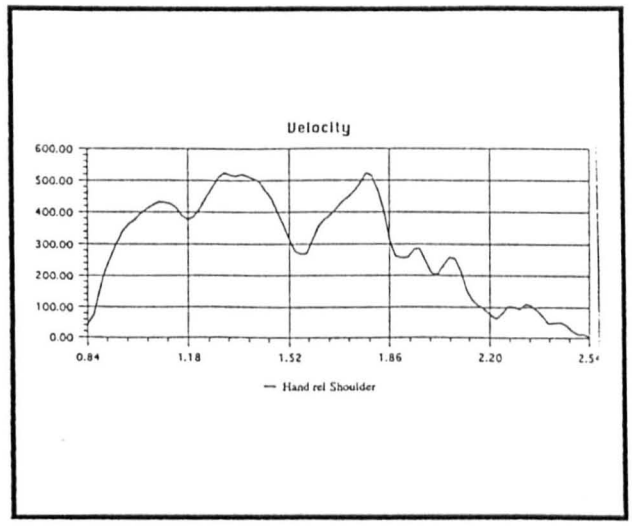


b] Lift phase, *visual/copy* condition

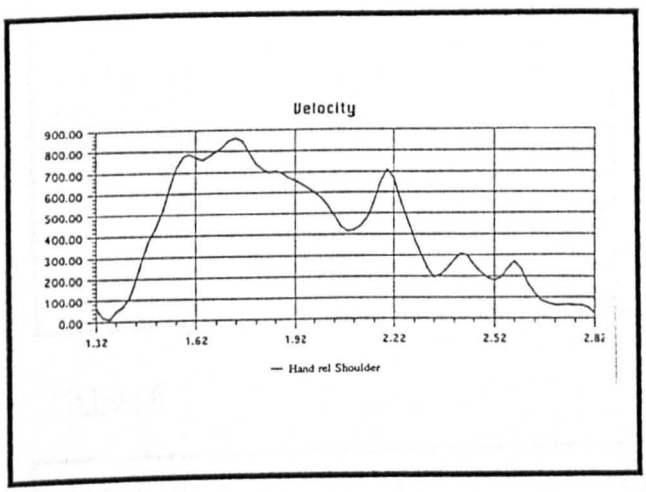
Time 2



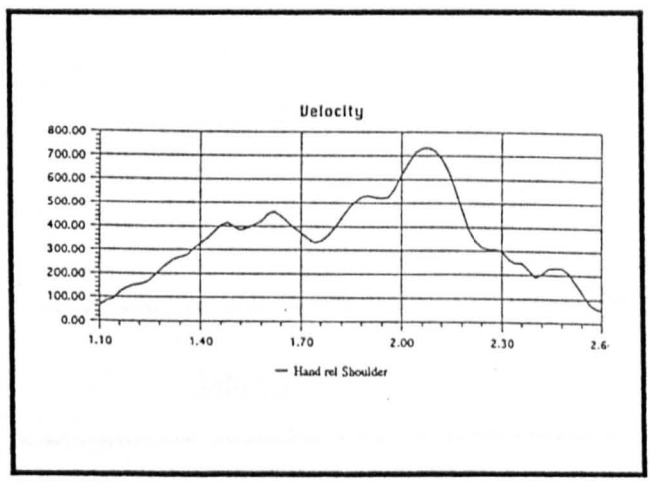
Time 4



Time 6



Time 9



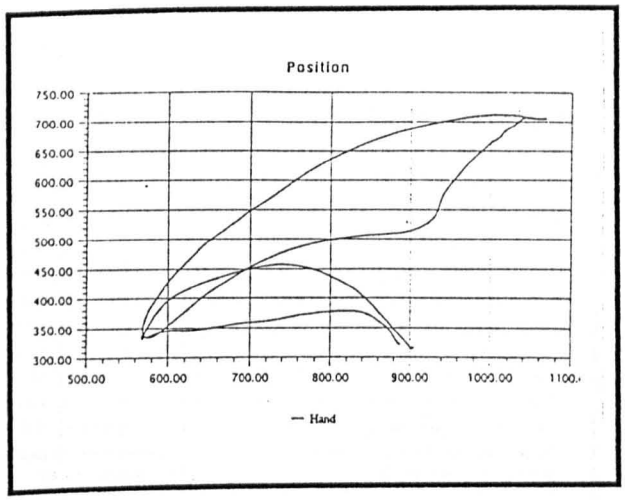
In this particular case there was no evidence of the marked multiple-peak velocity patterns that had been identified with apraxic subjects previously. This might suggest that LS was *not* adopting a slow, process-approach to movement but rather a strategy of rough approximation, as suggested by Hermsdorfer et al (1996). This, as a strategy,

would be in keeping with LS's tendency to be impulsive, consistent with frontal lobe injury behaviours.

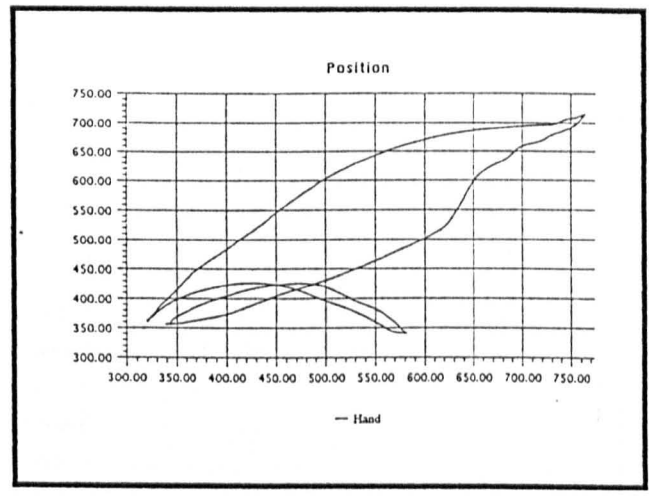
Despite these velocity pattern changes illustrated above, LS remained impaired in his use of spatial pathways during the drinking task throughout the ten-week study period (Figure 7.8). This was particularly evident in the hand trajectory during the visual/copy

Figure 7.8 : LS - Examples of hand trajectory recordings over time. (*visual/copy* condition)

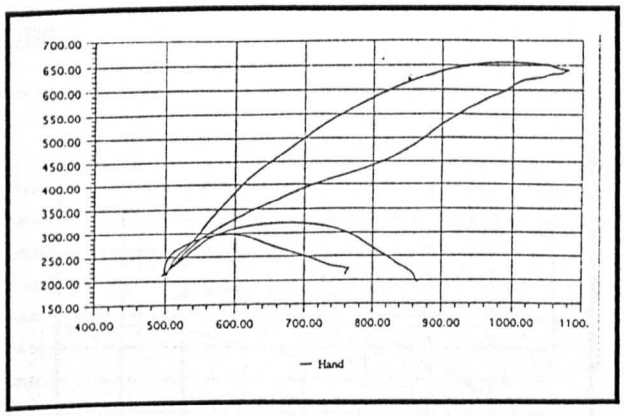
Time 2



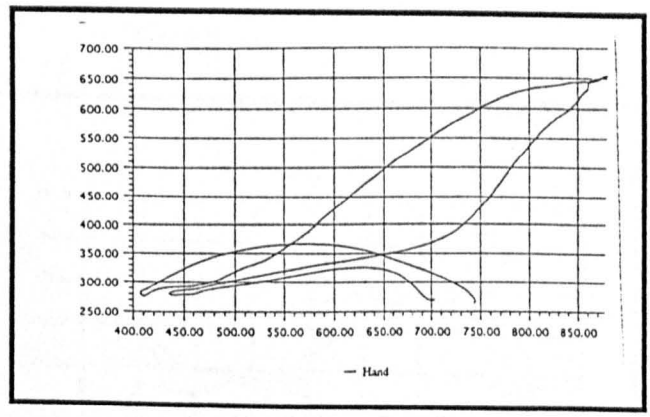
Time 4



Time 6



Time 9

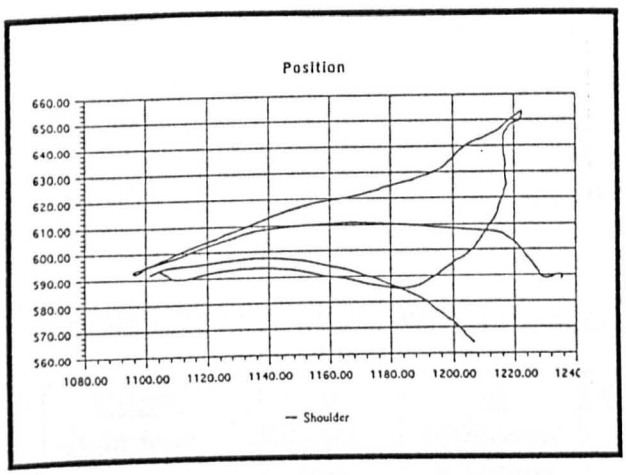


condition of the task, but it also applied to the verbal condition. The inconsistent use of a spatial pathway to complete the lift and down elements of the task are evident from the

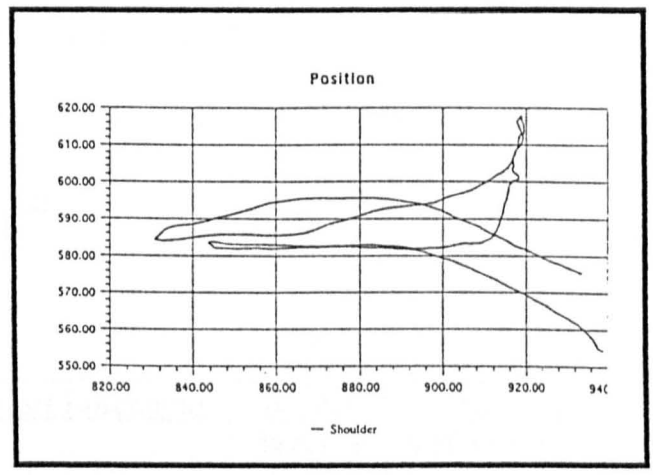
recordings. There is clearly no communality or mirroring of a spatial pathway during those task phases. The shoulder recordings also documented little change over the study period (Figure 7.9). All repetitions of the task demonstrated an over-use, and an abnormal use, of the joint to complete the drinking task when compared to normal recordings demonstrated in the group study. The shoulder trajectories demonstrated by LS were consistent with those associated with the apraxic subjects in the group study data (chapter 5).

Figure 7.9 : LS - Examples of shoulder trajectory recordings over time (Visual/Copy condition).

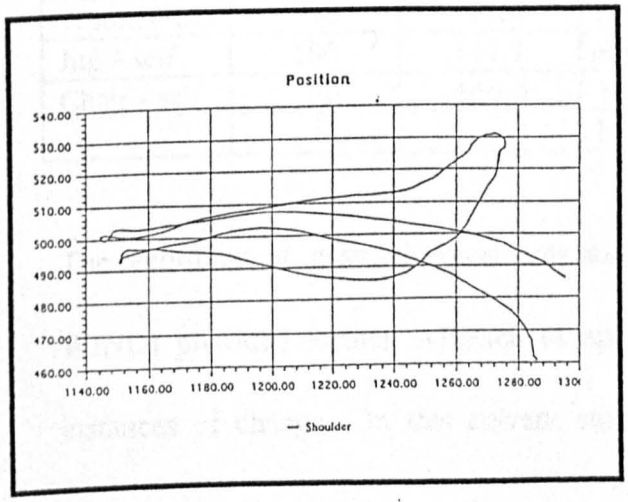
Time 2



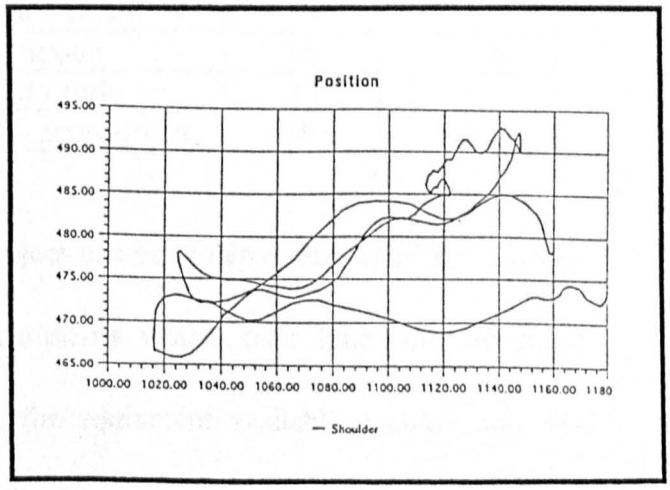
Time 4



Time 6



Time 9



In the light of these spatial orientation deficits demonstrated by the trajectory patterns which had shown no change over time, LS's visuo-spatial abilities were assessed with particular attention being paid to body schema. The Rivermead Perceptual Assessment Battery (Whiting et al 1985) indicated LS scoring well within the normal range, with no difficulties exhibited in the body schema elements of the assessment. The only areas of difficulty highlighted by the assessment procedure was in 3D complex shape copying. A further assessment of ability to judge distance of objects positioned around the normal testing environment indicated a wide range of both over and under-estimation of distance. The errors made by LS in the estimates did not follow any coherent pattern relating to object-self judgments or object-object judgments (Table 7.4). His abilities to estimate the spatial position of objects was judged to be unremarkable and did not provide evidence for any particular spatial deficit.

Table 7.4 : LS - Estimation of distance.

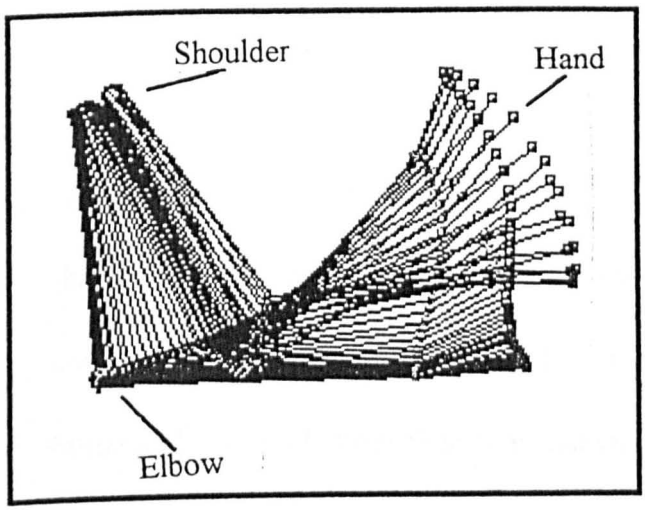
Object judgments	Actual distance (in cm)	% Difference in answer	Object judgments	Actual distance (in cm)	% Difference in answer
Pen - watch	60	+66.6	Chair - watch	60	+66.6
Pen - self	60	-16.6	Brush - watch	30	0
Watch - self	30	-33.3	Spoon - brush	10	-5
Jug - self	180	+11.1	Pen - spoon	18	-16.6
Chair - self	90	+66.6	Spoon - fork	40	0
			Fork - screwdriver	60	+66.6

The recordings of gestural movements and object-use pantomime (HAMMER, CARVE, WIND) provided further evidence of apraxic errors which, over time, showed some instances of change. In this current study, the equipment available enabled only 2D

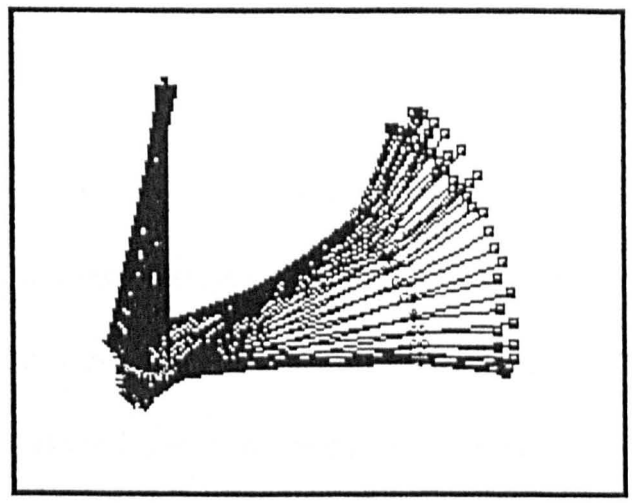
recordings of movement to be made (not 3D) so lateral motion could not be determined. Thus only horizontal and vertical elements of the movement performance could be examined. The stroboscopic reconstructions of movement in each of the three gestures illustrated deficits in both spatial orientation and distal joint control. During the gesture of HAMMER to *verbal* command at Time 1 (Figure 7.10a) a forward motion of the arm

Figure 7.10: LS - HAMMER gesture over time.

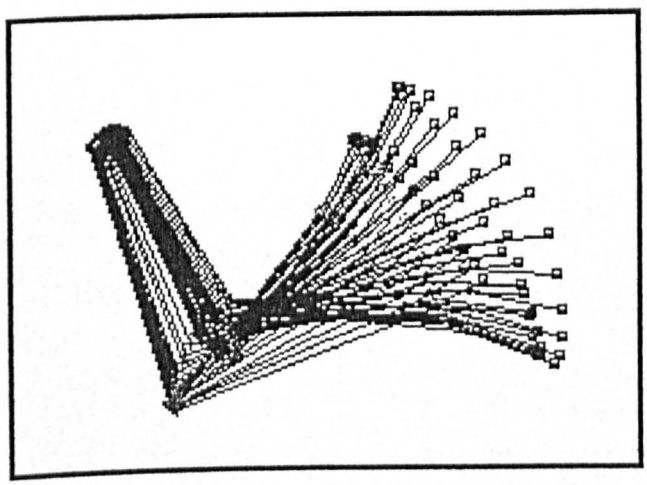
a] Time 1. *Verbal* command



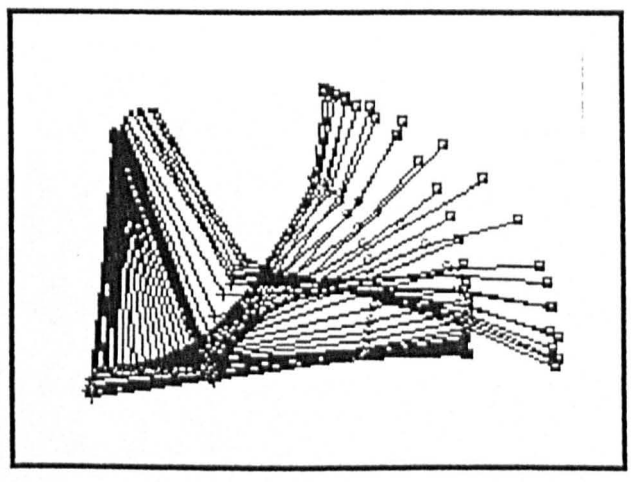
b] Time 1 . *Copy* command



c] Time 9. *Verbal* command



d] Time 9 . *Copy* command

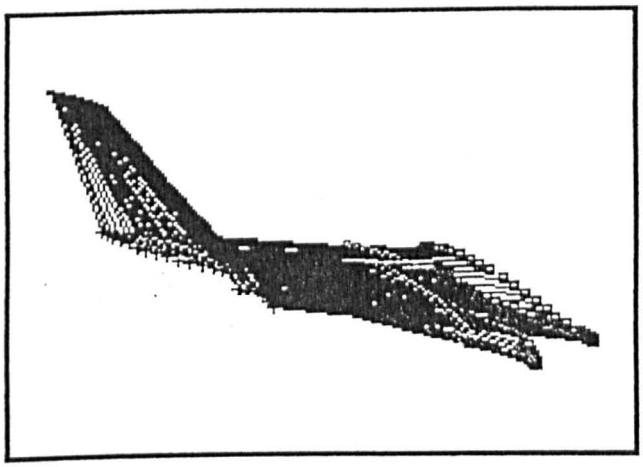


was observed. The over-use of the shoulder and elbow in carrying out this movement resulted in the loss of spatial constancy such that the up and down hammer motion was displaced in front of, or behind, the central line of target at each repetition. This contrasted with the movement achieved to the *copy* command in Time 1 when a spatial constancy was achieved with minimal use of the shoulder and elbow (Figure 7.10b). The recordings made of the HAMMER gesture at Time 9 demonstrated no appreciable improvement in distal joint control and spatial constancy to verbal command (Figure 7.10c) and in addition, at that last time of testing, the copy command also demonstrated the same spatial and joint errors (Figure 7.10d).

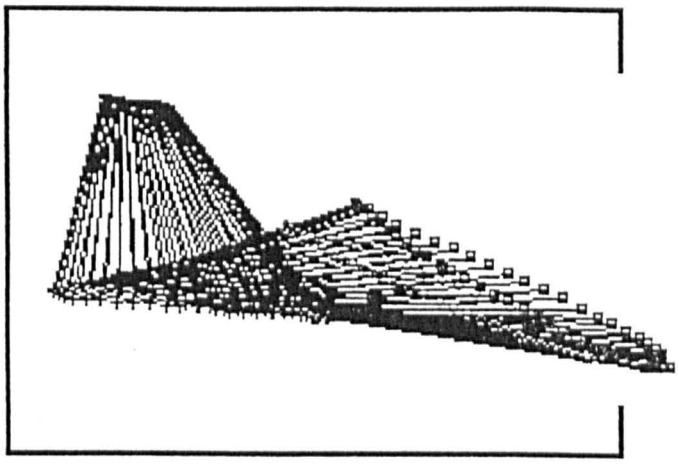
In contrast, the CARVE gesture, at Time 1, showed a deficit in spatial orientation such that LS was completing the movement with a downward trajectory in both verbal and copy conditions (Figure 7.11a & b). In Time 9 recordings, however, the expected horizontal plane of movement was carefully observed for both conditions with little variation in trajectory (Figure 7.11c & d).

Figure 7.11 : LS - CARVE gesture over time.

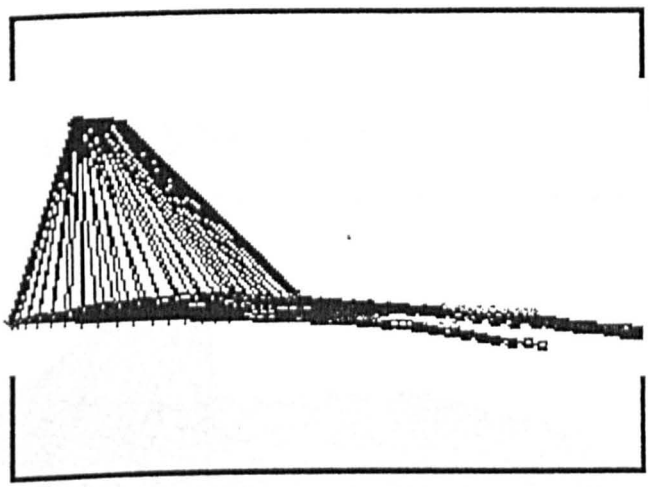
a] Time 1. *Verbal* command



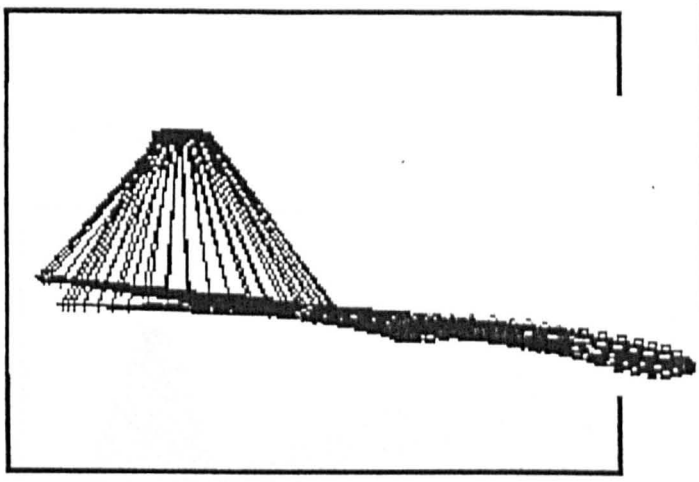
b] Time 1 . *Copy* command



c] Time 9. *Verbal* command



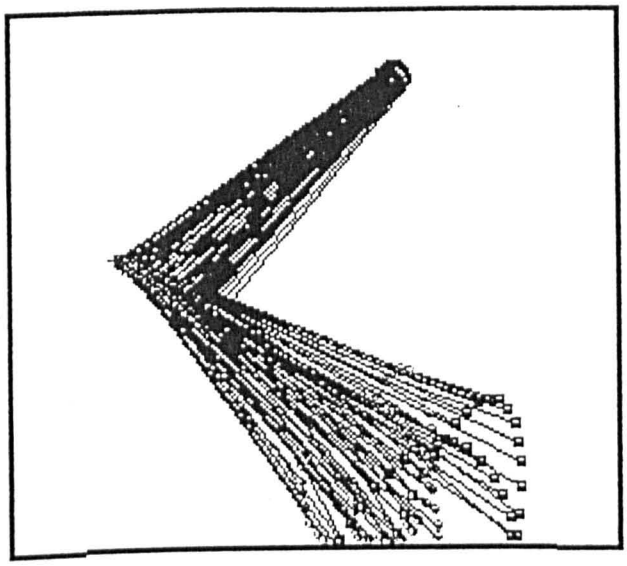
d] Time 9 . *Copy* command



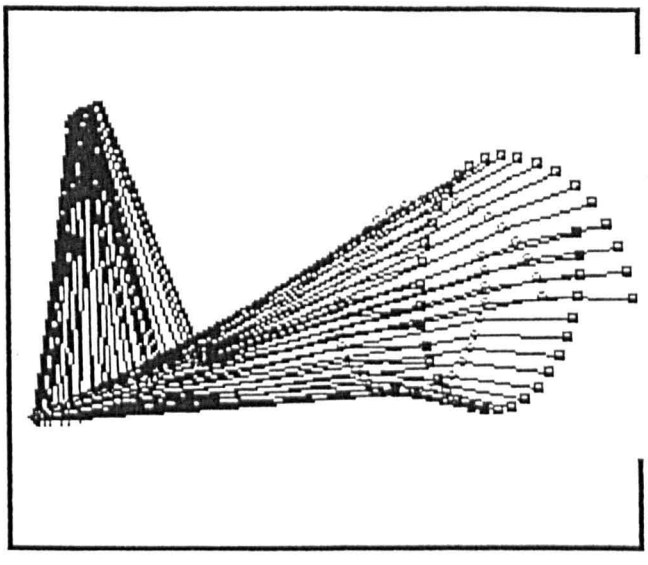
Some changes over time were also noted in the WIND gesture requested of LS in both verbal and copy conditions. As previously described, the movements made by LS to the WIND verbal command went out of the camera range as the movements were displaced linearly downwards throughout the course of the movement in a downward spiral manner. The copy command condition demonstrated a smoother circular movement

Figure 7.12 : LS - WIND gesture over time.

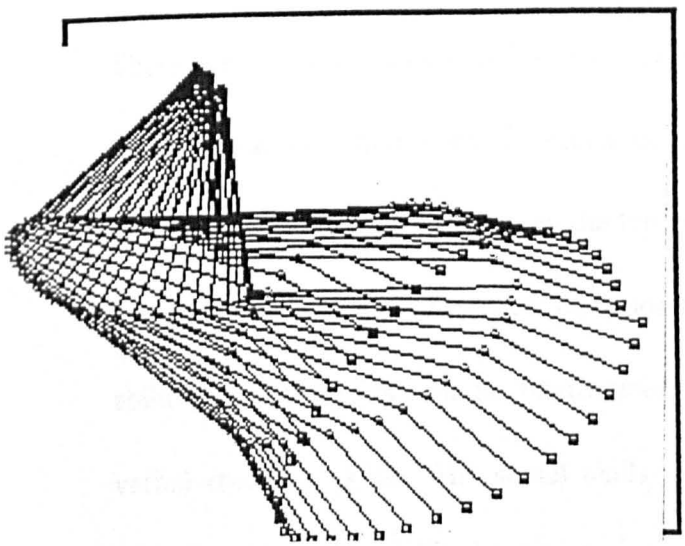
a] Time 1. *Verbal* command



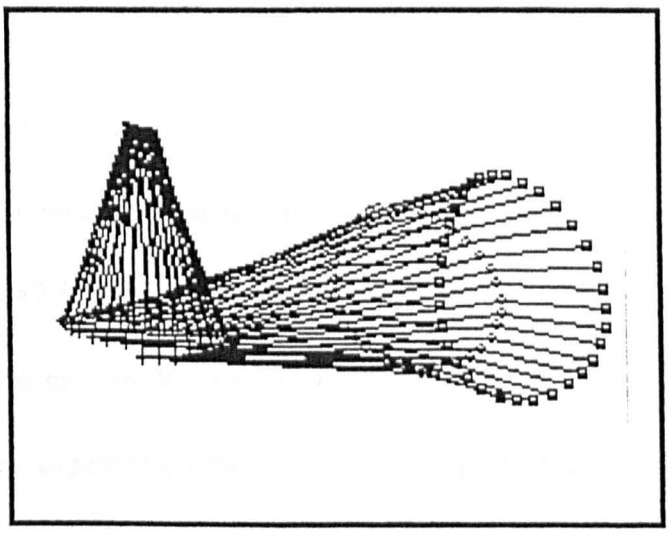
b] Time 1 . *Copy* command



c] Time 9. *Verbal* command



d] Time 9 . *Copy* command



repeated around a well-defined centre point, but this centre point was displaced in a forward and backward direction rather than downwards as in the verbal condition. So a spatial constancy could not be achieved by LS in the progress of the movement repetitions (Figure 7.12a & b). In the recordings made at Time 9 (Figure 7.12c & d) the

WIND gesture was more controlled in both verbal and copy conditions, although the verbal command elicited a rather exaggerated movement. The element of spatial constancy throughout the movement repetitions was demonstrated most precisely in the copy condition (Figure 7.12 d).

In all three gestures at Time 1 large variability across movement replications was noted, but this was greatly reduced by Time 9. This provided some evidence of an increased ability by LS to control and spatially orientate the movement requested of him. Software of a sophistication required to analyse the kinematic elements of the three gestures more precisely was not available for this study.

The kinematic analysis carried out on the drinking task and the gestural performance by LS over a ten week period showed evidence of some improvement in praxic abilities. There were some inconsistencies in the responses, particularly concerning the extent of spatial awareness and control, which contrasted with the improvement noted in the apraxia assessment scores. During the ten week period of the investigation LS was noted to have improved, though to still have some deficits in : concentration span, attentional abilities, ability to organise his environment and plan an activity like making a meal, and verbal memory skills. His social skills were improving although some inappropriate behaviour was still exhibited at times.

7.5 Discussion

This single case study charted the recovery of a young man with an identified mild ideomotor apraxia four months after a closed head injury, which was no longer apparent

six weeks later according to the apraxia assessment tools. The recovery was confirmed by a repeated apraxia assessment ten weeks after the initial examination (i.e. four weeks after the monitoring period). It could be argued that the errors observed during the initial assessment for apraxia were purely an indication and manifestation of concentration and attentional problems; that it was not ideomotor apraxia at all. This was not borne out by the clinical impression obtained during the assessment procedures where clear spatial and temporal errors in gesture and movement, consistent with evidence for apraxia, were exhibited. LS could recognise, during the De Renzi et al test, when a gesture he had made was inaccurate. Despite his best efforts LS was unable to replicate the hand position to his satisfaction. In addition, the researcher/clinician took into account the attentional deficits apparent in LS's performance when determining whether the errors made could be attributable to those deficits or to apraxia. Although LS needed prompting and alerting to the tasks on several occasions he gave the appearance, at each element of the test, of trying his utmost to complete it in the requested manner. It was considered that *despite* the attentional and concentration deficits exhibited by LS during assessment, the evidence pointed to the likelihood of an underlying apraxic difficulty.

The research field concerned with investigating the attentional system indicates clearly that the relationship between this system and the control of general mental processing, including motor processing, is well established (Posner & Petersen 1990). Posner and Petersen (1990) suggested that the attentional system was instrumental in orienting to sensory events, in particular to spatial locations. In the case of LS, the deficit of attention could have been a possible explanation for the loss of spatial orienting skill with LS not

attending to the visual sensory events during his actions. The integration of the sensory and motor systems in accurate hand action is well established (Jeannerod 1988, Riddock et al 1989, Jeannerod 1990, Jackson & Husain 1996). If the attention system deficit had interrupted that integration in LS, then deficits in motor performance would be evident. Posner and Petersen (1990) suggested that not only did the attentional system function to prepare the cognitive systems to respond to high priority signals but also to *sustain* alertness. In the case of LS, the deterioration and poverty of movement during the gesture tasks (HAMMER, CARVE, WIND) over 5 seconds duration, could be considered examples of the attentional system failing to sustain LS's alertness in carrying out the movement required. It might also be suggested that the kinematic recordings of the drinking task in failing to show change in the spatial trajectories of movement, were picking up a particular element of movement in spatial orienting that was related to the impaired attentional system. This might then explain how some elements of the kinematic recording demonstrated improvements in movement performance relating to motor output (timing, velocity peaks and patterns) whilst the spatial trajectories remained largely unchanged. Additional research which explored the role of the attentional system in apraxia would be a useful addition to understanding the apraxic condition. In addition, such studies could highlight potential areas for intervention in apraxia.

Improvement in completion time for the drinking task was demonstrated over the ten week period of the investigation. This improvement was not statistically significant, possibly due to ceiling effects. Other notable changes were seen in the velocity peak patterns in the task phases. This provided support for the previous 'natural history' study

(MT) with evidence of improvement in apraxia being demonstrated by the emergence of the bell-shaped velocity curve rather than the early peak with long low-level periods at the start and end of phases of movement. This suggested that LS had improved in his ability to make changes in the plane and angle of movements, and that the transitional movement phases were no longer so effortful or difficult to execute.

Using gestural performance in kinematic analysis was attempted for the first time in this single case investigation. The gestural performances of LS were noted to be better in the copy condition than in the verbal condition in all three items (HAMMER, CARVE, WIND). This was consistent with previously reported research in apraxia (De Renzi et al 1982) where internally generated gestural performances were deemed more difficult for the apraxic person than when a model of the movement was provided for them. Although this study attempted, in part, to replicate the work of Poizner et al (1990) the restrictions of the available 2D system meant that only certain elements could be explored. All three gestures (HAMMER, CARVE and WIND) were actions carried out in a unidirectional mode. The equipment was not capable of picking up any lateral movements made by LS during his performance of the gestures. Nevertheless, similarities in movement performance found in this case study were in agreement with those of Poizner et al (1990). In particular, the stroboscopic reconstructions illustrated the variability of movements with constantly changing amplitudes and spatial orientations. LS showed the *downward* movement displacement in the WIND gesture as did Poizner's case study. Differences were found, though, in the copy condition of this WIND gesture where a *forward and backward* displacement of movement was noted around a central

point. This was not reported in the Poizner study and may have merely been an anomaly in this particular case. Alternatively it might have been another manifestation of spatial inconsistency seen in apraxic movement. The notion of spatial constancy during the progress of movement repetitions might be an element to explore in future research.

No further kinematic analysis of these gestural performances was possible in this study. Evaluation of the velocity rhythms through the cycle of repetitions of the various movements might have been fruitful. In particular, to determine the constancy or otherwise of velocity patterns and where any dissociations lay could have said something of the nature of repetitive movement in apraxia. In addition, relating such velocity patterns to those found in the drinking task, especially in relation to changes in direction or movement phases, might have provided further evidence for apraxic improvement over time. A 3D analysis to look for shifts away from the unidirectional plane of 'correct' movement would have been a useful addition to these single case results.

7.6 Conclusion

This single case has shown evidence of an improvement in apraxic performance over a six week period in a young head-injured man. During this time his physiotherapy and occupational therapy interventions were not specifically targeted towards his apraxic condition and thus his improvement might be considered to be consistent with the 'spontaneous' recovery reported by Basso et al (1987). The role of the attentional system in LS's performance was considered in this investigation and some elements of change could be attributable to improved sustained attention. This single case has

provided some evidence for the existence of attentional and cognitive components in ideomotor apraxia that could disrupt performance output. Further studies to explore the role of the attentional system in apraxia, and its contribution to the spatial elements of apraxic movement, could enhance the understanding of this condition. An investigation concerning the value of attention training in apraxia might also prove a catalyst for further intervention research. This might include understanding how far the verbal and visual mediation strategies, suggested by rehabilitationists for apraxia intervention, affect motor performance by acting as an attention alerting strategy.

This case has added support to the previous 'natural history' case study in demonstrating kinematic changes in movement performance. The kinematic patterns moved from those associated with apraxic performances towards those patterns more associated with the normal control group data. This could be taken as evidence of improvement or recovery in apraxia. The kinematic analysis has provided quantitative, reliable evidence of change in elements of movement performance, unseen by the observational nature of the apraxia tests. Such objective measures have also highlighted the particular spatial elements of movement output that remained impaired in this case. Further studies, using single case methodology, are needed to verify these data concerning kinematic patterns of recovery. A useful addition to the research in this area would also be an ABA design single case investigation to look for evidence of intervention efficacy in the apraxic condition, using both clinical assessments and kinematic analysis.

Chapter 8. CASE 3: *GP. Head injury. Ideomotor apraxia.*

8.1 Case Background

GP, a twenty one year old right-handed woman, sustained a severe head injury as the result of a road traffic accident. The Post Traumatic Amnesia (PTA) estimation from medical notes was given as six weeks. A CT scan taken shortly after the accident indicated a right parietal haemorrhage; an MRI scan taken ten months later reported widespread and extensive bi-hemispheric cortical damage especially in the right posterior parietal lobe, left inferior parietal lobule and left frontal lobe (motor and pre-motor areas). There was also extensive sub-cortical white matter destruction, especially in the right hemisphere with a small area of damage to the right caudate nucleus.

At the time of the first assessment for this research project when GP first arrived at the rehabilitation centre it was seven months post-accident. She had recovered from the severe internal injuries sustained at the time of the accident. She was ambulant, mute (due to a buccofacial apraxia as diagnosed by the speech and language therapist) but with good comprehension. GP communicated via a spelling board. She was dependent in all her self care activities. A neuropsychological assessment completed by the psychologist over a period of several months concluded that GP's level of intellectual functioning would place her in the 'low average' range with particular difficulty in speed of information processing.

GP had an unusual and complex physical presentation of impairments. Assessment of physical function provided evidence of a disturbance of both motor and sensory functions in both hands. Specifically, there was shown to be full passive range of movements in all upper limb joints bilaterally, but reduced active movement in the wrist and fingers. There was loss of active opposition in the thumbs but without loss of thenar bulk. At rest, both

hands were held with the thumbs flat to the side of the palm, and with the metacarpo-phalangeal joints of the fingers held in hyper-extension. There was evidence of sensory mislocation in all modalities in the distal portion of both upper limbs (i.e. stimulation of the fingers was perceived as coming from the hands, stimulation of the hands was perceived as arising from the lower part of the forearms). Sensation in the upper arm and proximal part of the forearm was intact. Proprioception was intact for shoulder and elbow joints, but diminished in the wrist and fingers of both upper limbs.

Apraxia assessment tests were completed (appendices D1-D4) and results provided evidence for the presence of a severe ideomotor apraxia. The test scores at the first assessment (Table 8.1) and the performance errors made by GP were in keeping with evidence for an underlying dysfunction in motor planning. In the De Renzi et al test (appendix D1), although GP was unable to carry out the complex finger and hand gestures she was able to attempt the large upper limb gestures. Errors made by GP

Table 8.1 : GP - Clinical assessment scores (raw and %) at first examination.

Clinical Tests	Right Hand	Left Hand
Test 1- gesture copying (max. = 40) (appendix D2)	17 (42%)	16 (40%)
Test 2 - object use test (max. = 36) (appendix D3)	14 (38.8%)	18 (50%)
Test 5 - De Renzi test, gesture copying (max. = 72) (appendix D1)	6 (8.3%)	6 (8.3%)
Mean % scores for Tests 1, 2 & 5.	29.7%	32.8%
Test 3 - ideational apraxia (max. = 12) (appendix D4)	0 (unable)	
Test 4 - agnosia (max. = 20) (appendix E)	19 (95%)	

included inaccurate timing of movements, sequencing errors and inaccurate plane of movement or hand posture. There was gross over-use of the elbow and shoulder in all gesture and movement copying during this test. GP showed a notable difficulty in placing her hands in the correct plane and orientation to her face. In both gesture copying and object-use pantomime tests (appendix D2 & D3) all movements to both verbal command and copying were recognizable but incorrect, showing errors of timing, speed, wrong plane of movement and incorrect orientation of the hands. Elbow and shoulder movements were used extensively and inappropriately by GP in her attempt to perform the movements and gestures requested. Again, there was evidence of particular difficulty in positioning the hands near to the face. With all these errors fulfilling the accepted notions of performance deficit in ideomotor apraxia (in content, timing and spatial aspects - Raade et al 1991) an informed judgment was made that these observed behaviours were attributable to ideomotor apraxia deficits.

In the assessment used to determine the presence of *ideational* apraxia (appendix D4) no score was possible as GP was unable to manipulate the objects sufficiently. She was, however, able to indicate where, how and in what sequence each should be used. It was considered unlikely, therefore, that she had an ideational apraxia as such an awareness of objects and their use would not be apparent in a person with that dysfunction.

Performance on all tests for apraxia was confounded by the motor and sensory impairments experienced by GP. Taking this into account, the errors made by GP were of the type and quality recognized and identified as apraxic in origin, and the accumulation of evidence from the tests suggested a severe ideomotor apraxia in both upper limbs. There was no evidence of an ideational apraxia, nor of visual agnosia.

Informed consent to participate in a pilot project concerning evaluation of a sensory stimulation intervention was obtained following normal procedures.

8.2 Aims of the study

Using this single case the aims of the study were to :

- evaluate the effectiveness of a sensory stimulation intervention protocol on movement performance in a person with severe ideomotor apraxia after head injury.
- evaluate the effectiveness of task breakdown on self care independence in a person with severe ideomotor apraxia.
- explore the variability of motor performance in the apraxic condition.

8.3 Pilot study

A four-week pilot project was devised using an ABA design, to establish the practicalities of carrying out an evaluation of intervention with GP. This included assessing GP's ability to cope with the rigours of a research protocol, and to look for any preliminary indications that sensory stimulation procedures could produce change in motor performance. GP was fully informed of the proposed protocol and the potential for a longer intervention study later on. She was enthusiastic about participation for two reasons. Firstly it gave an overt structure and rigour to her rehabilitation programme. Secondly it had an understandable rationale for her following her anecdotal report of hurting her right hand during a fall and, for the half-hour duration of consequent pain, being able to move and use her hand "like normal" (*sic*).

8.3(i) Procedure

An ABA single case design was chosen as an appropriate methodology for this pilot study since it enabled an intervention effect to be determined (Barlow and Hersen 1984). A two week baseline period was planned with apraxia-sensitive tasks and control tasks being used, together with kinematic recordings. This was followed by one week's intervention period using the same tasks and recordings immediately before, and immediately after, the sensory intervention procedure. This enabled analysis of the immediate effects of intervention as well as looking for performance change over the five day period. A further period of one week mirrored the first baseline phase of the experiment. Each daily session lasted 45 minutes in total. During the baseline and post-intervention periods the time in the 45 minute session not taken up with testing and movement recording was spent in "conversation".

Apraxia-sensitive tasks and control tasks proved extremely difficult to find for GP, given her motor and sensory deficits and her mutism. The provision of control tests was never fully resolved as upper limb motor tasks were largely impossible for her to perform and would be sensitive to the intervention protocol rather than acting as a control. In contrast foot-tapping, perceptual or memory tasks resulted in her performing at ceiling level. This is an acknowledged flaw in this protocol. Apraxia-sensitive tasks were compiled to mirror, but not practice, the clinical assessments. Thus a variety of object-use gestures was compiled using examples from published work (Alexander et al 1992, Riddock et al 1989) and carried out in three modality conditions as before; verbal command, copy/visual model, and real-object use (Table 8.2). Four items, randomly chosen, were used on each test occasion. In addition, appropriate movements and gestures were

compiled to provide an imitation test in verbal and visual modalities (Table 8.3). A selection of ten items was randomly given at each test occasion.

Table 8.2 : GP - Items in pilot protocol object use test.

OBJECT USE TEST	
Baseline phase items *Show me how you would hold and use.....*	**Intervention phase items** *Show me how you would hold and use.....*
Saw	Fan
Clothes peg	Screwdriver
Paintbrush	Eraser
Tin opener	Fork
Corkscrew	Shaving brush
Spanner	Spoon to stir
Iron	Jug to pour
Potato masher	Scissors

Table 8.3 GP - Items in pilot protocol movement imitation test.

MOVEMENT IMITATION TEST	*"Show me how you....." / "Copy this....."*
Hitch-hike	Wipe sweat from your brow / forehead
Beckon someone over	Hold a cigarette as if to smoke it
Brush your hair	Hold a straw as if to drink
Polish / dust a table	Rub your nose to warm it.
Flip a coin	Cup your ear as if to hear better
Wipe a window	Roll plasticine
Strike a match	Take off a hat
Throw a ball	Play the piano
'Thumbs up' sign	

A simple timed task (which involved pointing at given targets on a piece of paper in sequence from a designated start position) was carried out with each hand. During the intervention period this timed task was completed immediately before, and immediately after the sensory stimulation protocol.

The sensory stimulation protocol was devised to be manageable during a 45 minute therapy session and mirrored the sensory intervention protocol used with children with developmental dyspraxia. This involved tactile and proprioceptive stimulation using different sensory pathways. The rationale for such intervention (see section 2.9 pages 41 - 42 for details) revolves around the notion that full sensory-motor integration is needed for controlled and accurate motor output and that such integration of the sensory-motor system is aided by a programme of stimulation using all sensory modalities (Ayres 1985, Cermak 1985, Croce 1993). As the *tactile* system has a role in organisation of performance, this is the major sensory modality used in stimulation programmes (Ayres 1985). By such methods it is argued that the stimulation and alerting of the sensori-motor system would enhance motor output. Stimulation of the sensory system as a means to improve neuropsychological deficits is not confined to apraxia alone. Prada and Tallis (1995) reported the use of electrical stimuli to the skin to treat hemineglect.

The specific sensory stimulation protocol for this study involved spending 20-25 minutes applying sensory input to the patient's forearms, hands and fingers using all tactile modalities:

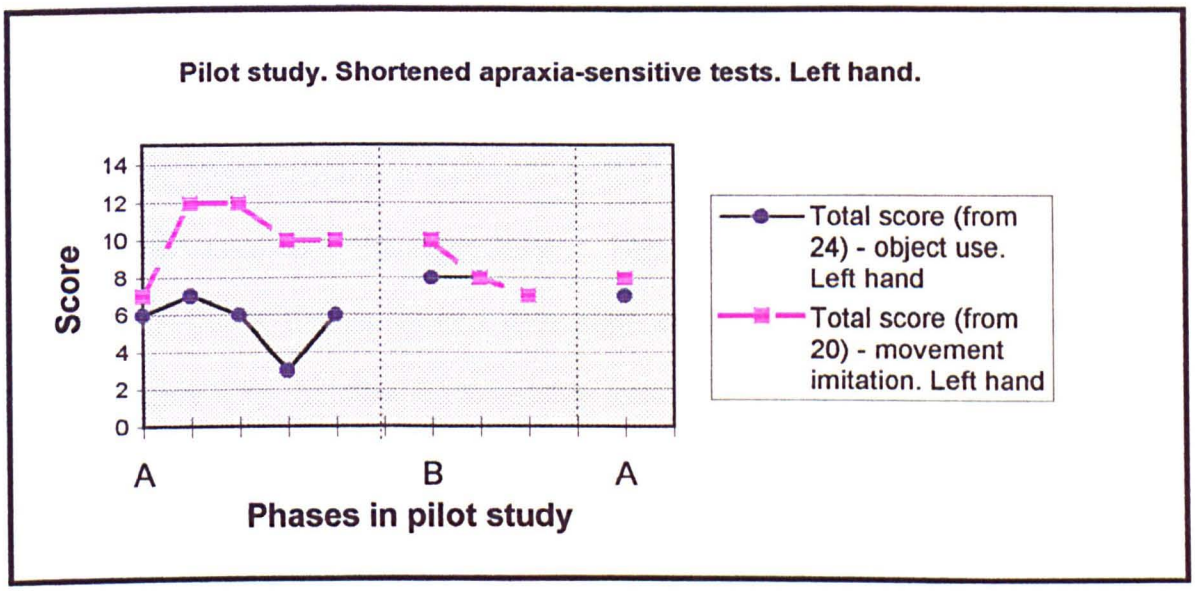
- deep pressure: massage using oil or cream.
- sharp touch: using a nail brush in rotating movement on skin.
- proprioception: leaning on and putting weight through arms and wrists as well as pushing/pulling activities.
- soft touch: using a soft cloth and applying long strokes.
- self-touch: patient stroking and smoothing her hands over forearms and hands.

During this four week pilot study, descriptive observations of activities and performance were made during physiotherapy, occupational therapy, and speech and language therapy sessions. Additionally observations were made at everyday activities as GP performed them (lunchtime eating and drinking, getting washed and dressed, walking in the grounds). These data were obtained to look for notable events, actions or comments which might have influence on the experimental protocol and to obtain information on the general pattern of functioning of GP.

8.3(ii) Results

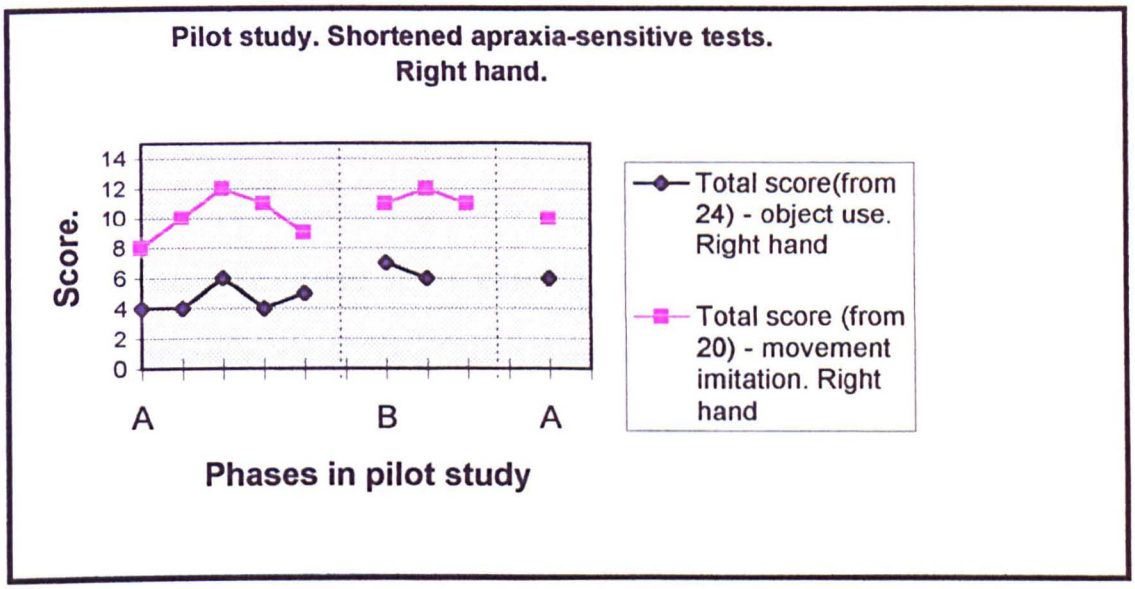
Visual analysis of the data collected in the pilot study indicated a mixed picture, especially between the 'clincal' tests and the timed task. The short apraxia-sensitive tests devised for this study showed a variable pattern of performance in the left hand in the baseline phase, a reduction in scoring in the movement imitation test in the intervention phase, and the one data point in the post-intervention phase demonstrated no real change from baseline performance or even a deterioration. (Figure 8.1)

Figure 8.1 Pilot study : Shortened apraxia-senstive test scores - left hand



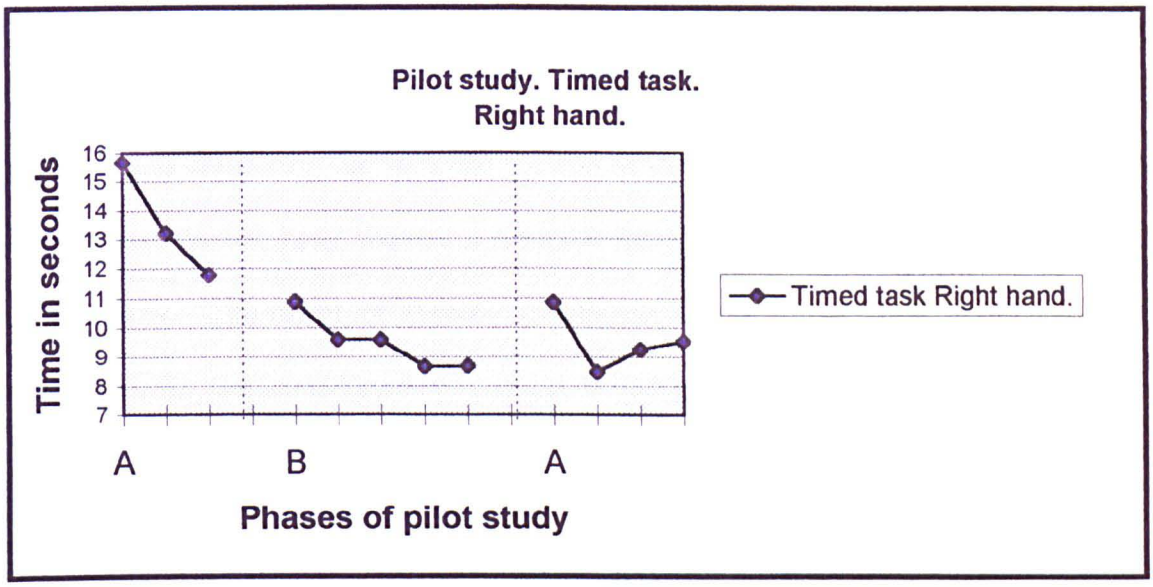
The right hand performance in these test might, though be considered to be marginally improved in the intervention phase, though some evidence of trend in improved performance abilities might be interpreted from the limited data points. The one post-intervention data point showed no convincing change (Figure 8.2).

Figure 8.2 Pilot study : Shortened apraxia-senstive test scores in ABA phases Right hand.



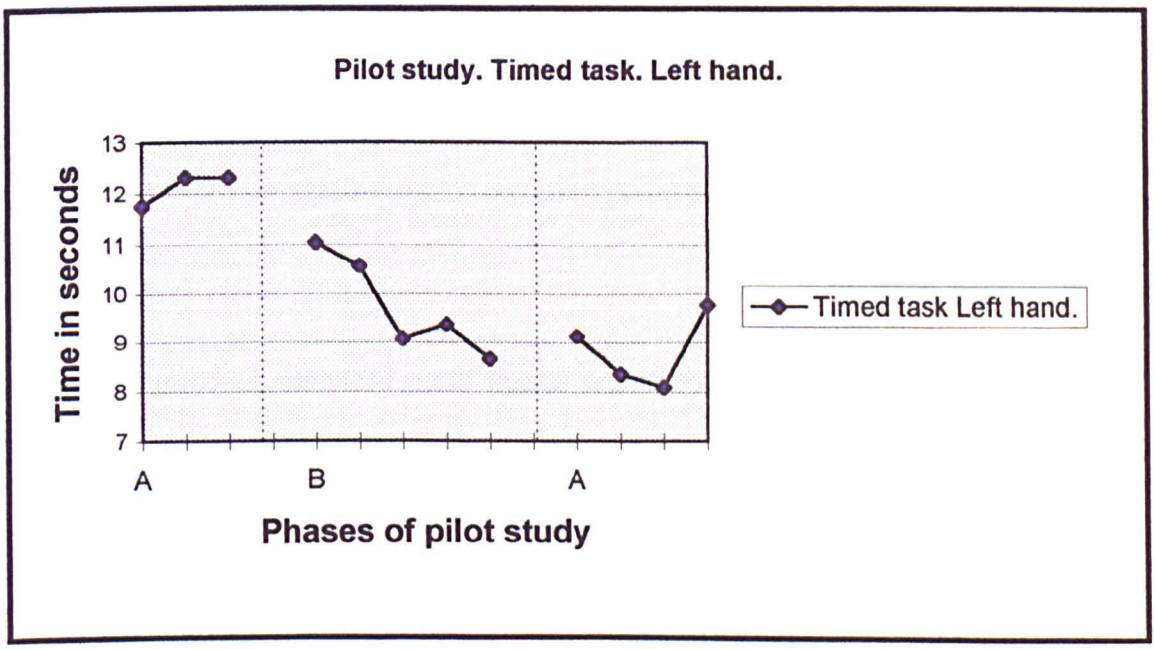
The timed task, when plotted on a graph indicated that motor learning was occuring in the baseline phase such that a marked reduction took place in the time taken to perform the 'circles' task with the right hand. This improvement was maintained in the intervention phase, though showed some signs of deterioration in the post-intervention phase (Figure 8.3). So clear is the baseline trend, that no 'B' phase improvement could be attributable to the intervention itself.

Figure 8.3 Pilot study : timed task, right hand.



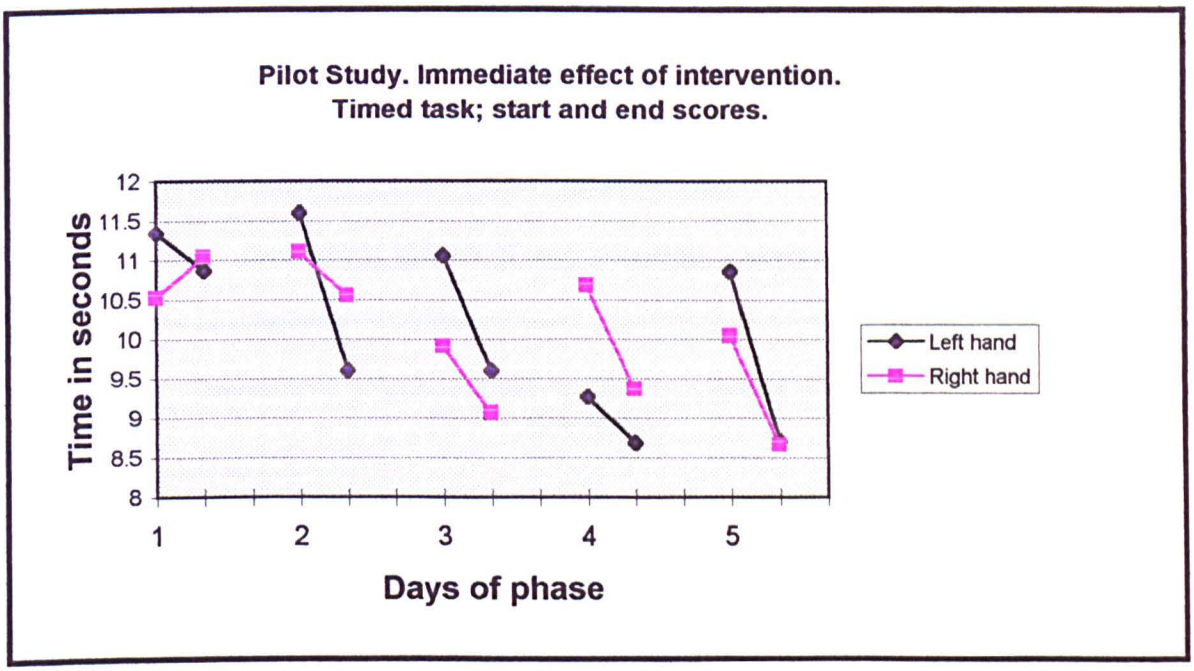
The left hand performance was somewhat different in that the baseline deteriorated, if anything, though a marked improvement in speed to complete the task was demonstrable in the intervention phase. This improvement was partly maintained and enhanced in the post-intervention phase, but the last data point showed loss of speed (Figure 8.4).

Figure 8.4 Pilot study : timed task, left hand.



This graphical plotting of the time taken to complete the task using the data taken at the end of each therapy session hides, though, the *immediate* effects of the sensory stimulation protocol in the intervention stage. The right hand showed a marked improvement comparing the start and the end of the treatment session in each of the five days, whilst the left hand showed a simmilar trend in the latter four days of the phase (Figure 8.5). The effect of sensory stimulation to the upper limbs could have been responsible for this immediate improvement in task performance.

Figure 8.5 Pilot study : Immediate effects of intervention. Timed task.



A similar trend of enhanced performance was noted in the apraxia-sensitive tests when the scores obtained at the beginning of the 45 minute sensory stiimulation intervention session were compared with those obtained at the end (Figures 8.6 and 8.7). The improvement in movement imitation test both to verbal command and copying gestures

Figure 8.6 Pilot Study : Movement imitation test.

Immediate effects of intervention - start and end scores. Right hand.

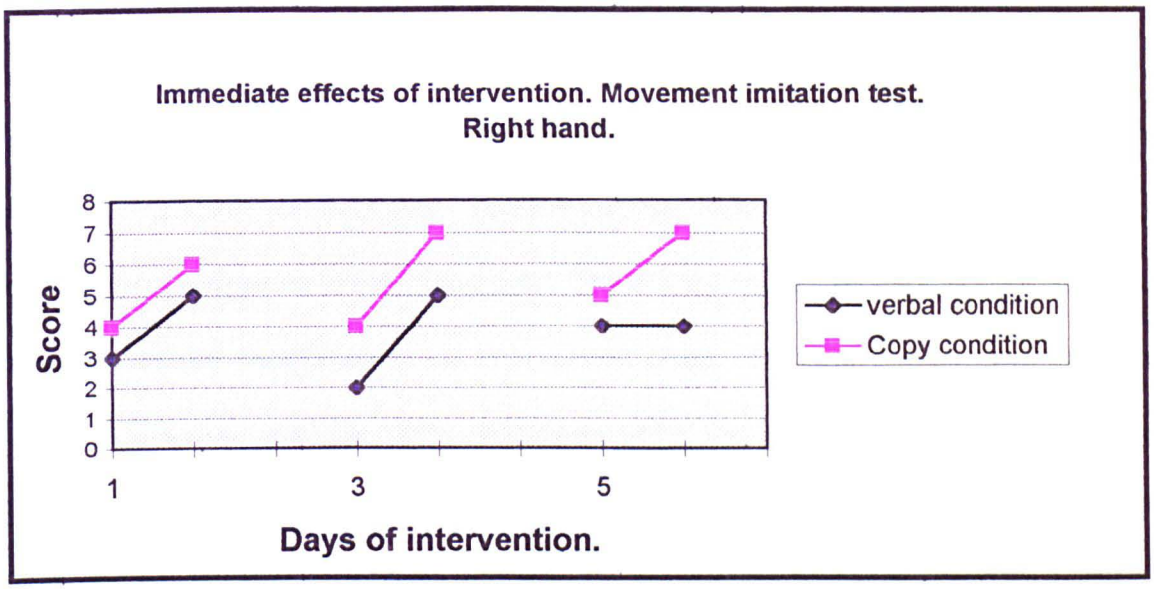
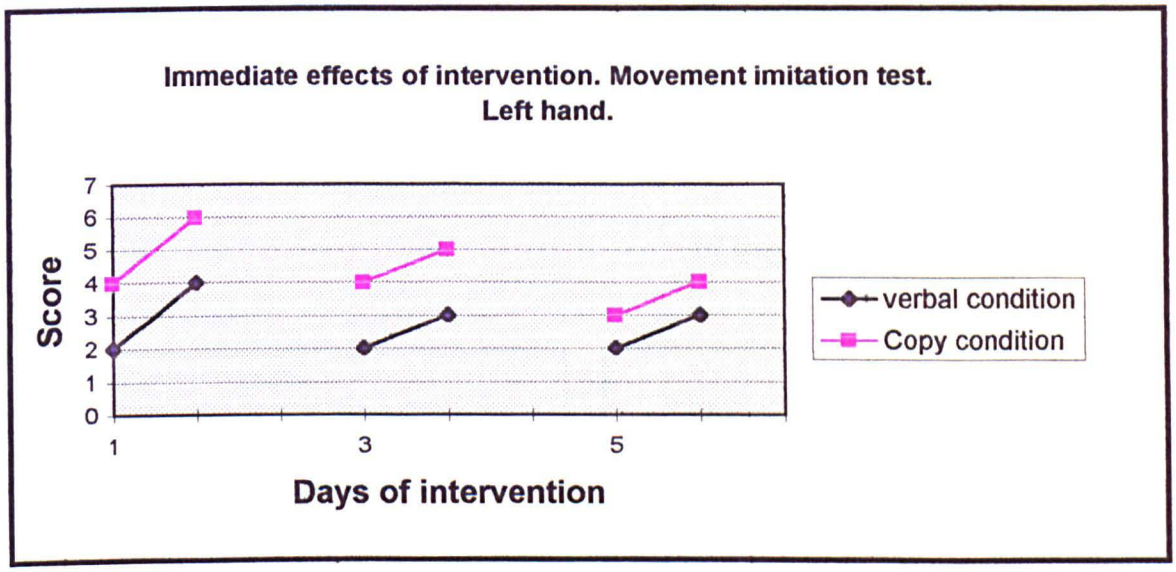


Figure 8.7 Pilot Study : Movement imitation test.

Immediate effects of intervention - start and end scores. Left hand.



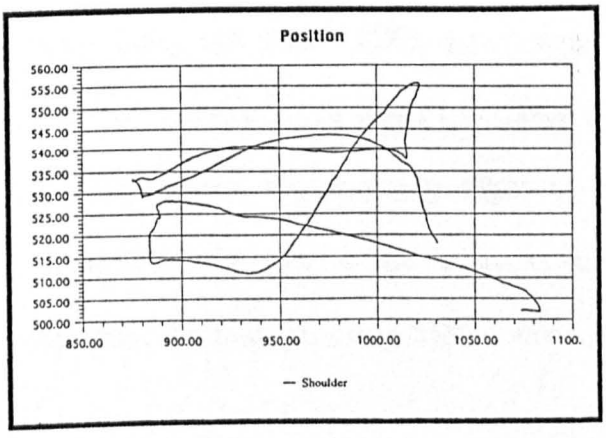
was seen in both right and left hands, though the subjectivity of the judgements in the observed gesture performance must require that these results be viewed with some

caution. However, taken with the objective evidence of timing data for task performance it could be considered to add weight to the contention that the sensory stimulation enhanced GP's motor performance.

Kinematic recording and analysis in this pilot project were limited due to time constraints within the rehabilitation timetable. Three recordings were possible following the protocol previously described for the drinking task. These were completed immediately before and after the sensory stimulation in the intervention phase. Only the verbal condition of the drinking task was used for these recordings using the left (non-dominant) hand. Few differences were observed in the kinematic profile of the drinking task *immediately* following the sensory stimulation protocol, but some **were** noted between day 1 and day 5 of this intervention period. There was less use of the shoulder on day 5 compared to day 1 (figure 8.8) and some reduction in the long low-velocity periods between phases (figure 8.9).

Figure 8.8 - GP Shoulder movement during drinking task. Intervention phase, pilot study.

a] Day 1



b] Day 5

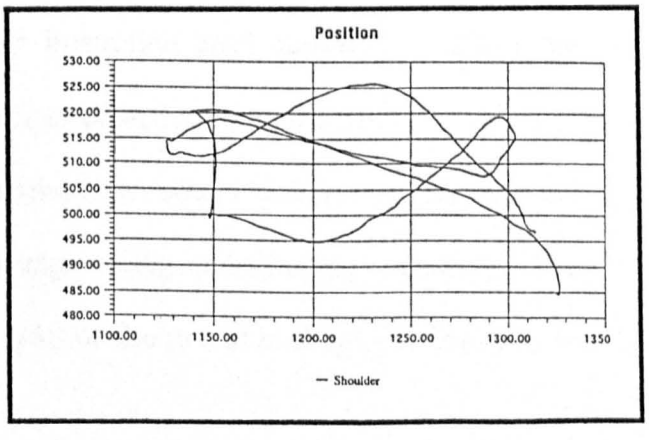
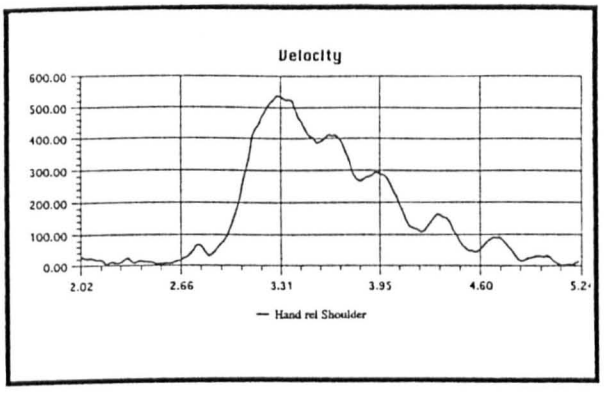
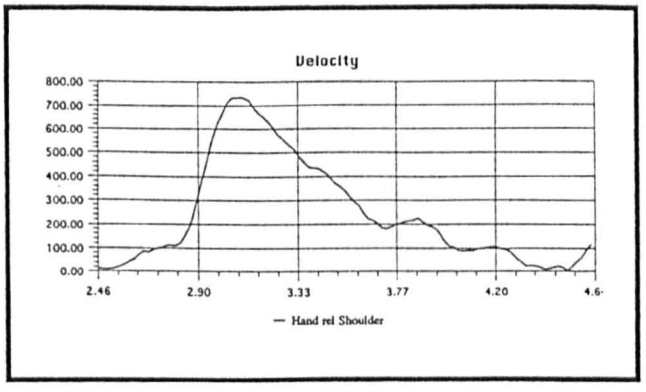


Figure 8.9 - GP Velocity peak during drinking task. Intervention phase, pilot study.

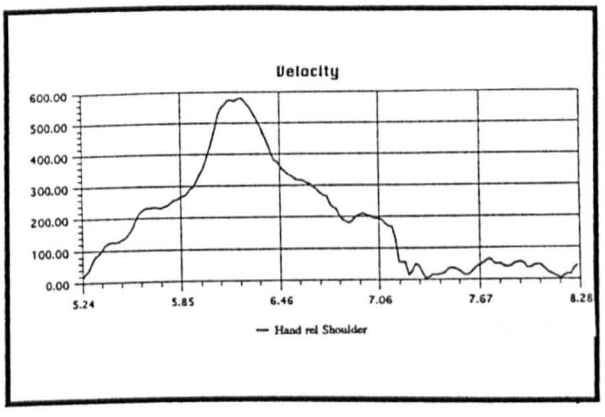
a] Lift phase - Day 1



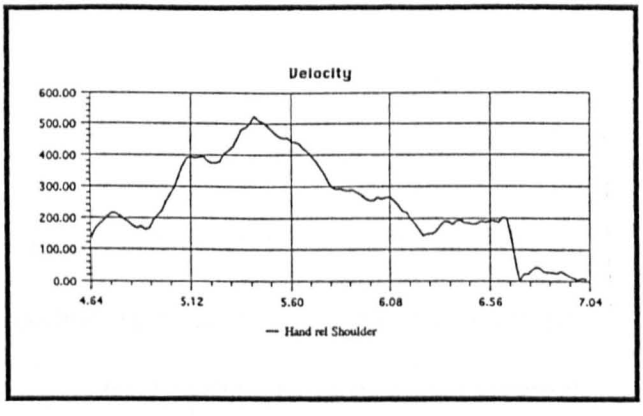
b] Lift phase - Day 5



c] Down phase - Day 1



d] Down phase - Day 5



Observation data obtained over the four week pilot study (gathered in three-hour periods daily), together with information taken from discussions with GP, indicated several elements which seemed to affect her performance during therapy sessions and during daily living activities. GP's mood state and frustration level appeared to affect the number of attempts she made to achieve a task or objective. She seemed very aware of her appearance and how she might be perceived by others and was frustrated and embarrassed by some of her attempts to manage eating and drinking 'normally'. She indicated her anger at being 'talked down to' (*sic*) or shouted at in shops, for instance. In

such circumstances she preferred not to attempt a task rather than make herself appear 'foolish' in public.

Following weekend leave GP was noted to be less willing to try and talk, and less fluid in her movement. This might have been due to not practicing at home, or an indication of mood state at needing to return to the rehabilitation centre after a weekend. Alternatively, it might have been an indication of her feelings of dependency upon her mother as the latter reported occasional tempestuous arguments at home. When GP was feeling cold, and on the occasion of having a head cold, her performance in physiotherapy was reduced. Generally though, GP gave the appearance of trying very hard in all her therapy sessions and seemed motivated to achieve independence.

8.3(iii) Discussion

In attempting to devise short apraxia-sensitive tests which would make the monitoring of change manageable within the 45 minute timetabled session, it was clear that although these might indicate altered motor performance to the trained observer, the traditional category scoring system where the 'impaired but recognisable performance' was the only category available. Any *subtle* changes in ability could not be scored. An attempt was made to improve the scoring system using the error categorisation taxonomy described by Raade et al (1991) (Table 2.2) but this was shown to be impractical, time consuming and potentially unreliable when judging real-life performance of gestures. Analysis of a taped performance would have resolved this difficulty, but GP was unwilling to be videoed. These tests did demonstrate their capability in showing change immediately after sensory stimulation but did not indicate any clear effect between the ABA phases.

The timed task, being quick and simple to administer, demonstrated its use in the project certainly in highlighting the immediate improvement in motor performance following a session of sensory intervention. It is interesting to note that both the timed task and the clinical tests mirrored the effect of improvement directly after sensory stimulation. A general *phase* effect, though , from one day to the next, could not convincingly be assumed from visual inspection of the plotted data. A longer pilot study might have been more helpful, certainly a longer baseline period would have helped eliminate the learning effects noted in the timed task. Balanced against this needed to be an awareness of GP's cooperation and tolerance to the research protocol and her eagerness to start on a longer sensory stimulation programme.

The changes that *were* seen in both task performance and clinical tests, lent weight to the need for a more realistic and prolonged intervention study. The kinematic analysis of the drinking task showed mixed results concerning the quality of GP's motor performance related to the experimental intervention. The limited data available therefore suggested the need for a longer study. Observation of activities of daily living indicated that physical elements, mood state and health state (feeling cold, menstruation, having a cold, personal relationships) could all affect GP's motor performance and ability to participate in her rehabilitation programme.

8.3(iv) Conclusions

The pilot study indicated that GP was able to cope with the rigours of a research protocol. She agreed to participate in a longer project following normal consent procedures. The pilot study also demonstrated the time constraints under which such a project would have to be managed, particularly in the performance measures used before

and after each daily therapy session which could not reduce the therapy time by too much. Simpler measures for monitoring change were needed. Such measures needed to be quick, easy and reliable to administer, quantitative in nature and a meaningful representation of the changes being worked towards as therapeutic goals. It was deemed critical, therefore, that the treating therapists involved in GP's management be invited to become involved in the project. It was decided that the kinematic recording of movement would run separately from, but parallel to, to the intervention project and be used as an outcome measure in evaluating change. Periodic clinical apraxia assessment with the full tests, rather than the shortened versions devised, would also be used to assess change.

8.4 Intervention Study

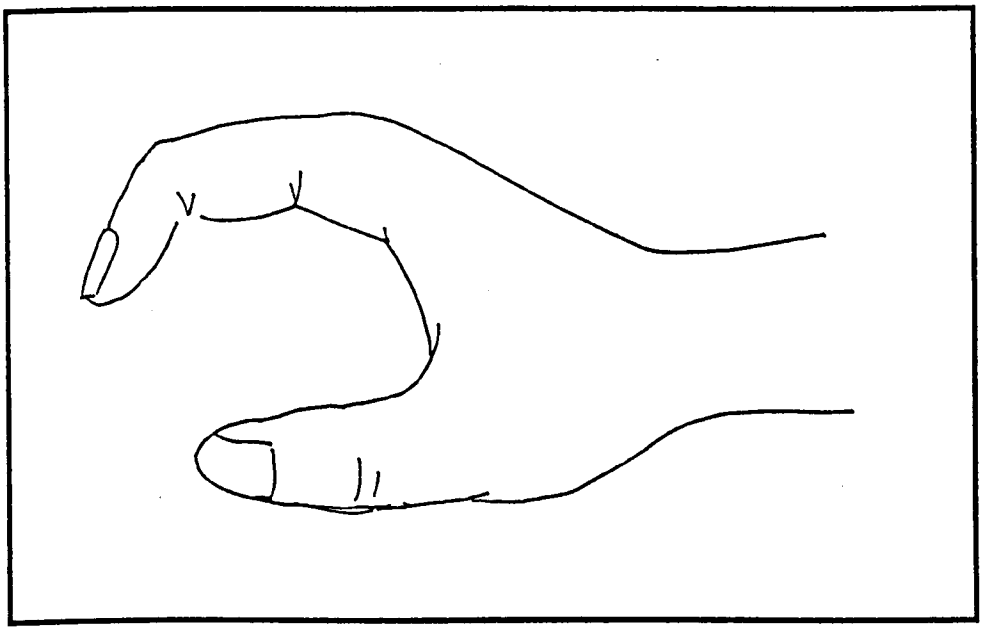
8.4(i) Procedure

Following the pilot study, a longer ABA case study was designed. In consultation with the rehabilitation team, new performance measures were devised which were considered appropriate to monitor the changes being worked towards in GP's rehabilitation programme. These measures were designed to be manageable, reliable and quick to administer before and after the therapy sessions with GP. In addition the measures would be recognised by other therapists and therefore generalisable to other rehabilitation settings.

The physiotherapists chose to measure opposition in both hands. Calipers were used to measure the distance between tips of thumb and little finger. In addition a measure of the ability of GP to form a rounded cylinder shape with her hands was achieved by measuring

the distance of the maximum web space of each hand using caliper points placed at specified hand architecture markings. These measurements were taken by the same person throughout the case study trial, **not** the treating therapist. The occupational therapist chose, as an outcome measure, a simple timed task (picking up, and moving, nine 1-inch cubes from the near edge of a table to a specified area in the centre of the table). Other measures used were wrist extension, ulnar deviation and radial deviation

Figure 8.10 Drawing of hand forming a cylinder shape.



using goniometry. The timed task, most readily prone to motor learning effects, was practiced twice daily for five days prior to the start of the baseline phase in order to mitigate against contaminating the data by practice effects.

During the baseline phase these measures were taken, as appropriate, at the start of each physiotherapy and occupational therapy session. They were repeated when each session had finished. The therapy sessions were timetabled, as far as possible, to be carried out

daily at the same time. This was largely managed in physiotherapy, but in occupational therapy *daily* sessions were often not achieved and were also carried out at different times of the day. The treating therapists continued their normal therapy sessions with GP following the plans and aims of therapy that they had devised prior to the start of the research project. The only difference in the therapy programme was the five minutes spent at the beginning and end of each timetabled session taking the described outcome measures. This enabled the therapists to have feedback on the *immediate* effect of their given intervention (once the project was completed) as well as the comparison with the 'experimental' sensory stimulation intervention. Examples of therapeutic activities carried out in this baseline phase were grasp and release exercises, exercises for isolated wrist and finger movements, activities using a large ball, static cycling, and practice in functional self care activities using task break-down strategies (as suggested by Wilson 1988). Other general strategies used during this period involved verbal and visual mediation of movement performance as suggested by previous research (Croce 1993, Pilgrim & Humphreys 1994, Riddock et al 1995). During the baseline phase, the treating therapists were 'blind' to the nature of the proposed 'experimental' intervention in order that no compromise was made with their already chosen intervention protocols. The baseline phase lasted for one month in order to gather enough data to establish the consistency of GP's performance. These data collection points were somewhat intermittent as days were 'missed' through long weekend leave, other therapist commitments, GP being unwell, and hospital and other appointments.

The intervention period also lasted for one month. The treating therapists involved in GP's rehabilitation and management were instructed in the same sensory stimulation procedure described and used in the pilot study. During this learning process, and as they practiced on one another, the therapists were observed by the researcher. This was to ensure that consistency in the intervention could, as far as possible, be achieved. Each therapist had a checklist to act both as an *aid memoir* and a record sheet of each completed part of the intervention. Periodic observation of the intervention sessions monitored the consistency and compliance with the intervention protocol given. The outcome measures remained unchanged, with assessments carried out before each sensory stimulation intervention and immediately afterwards. Whilst every attempt was made to carry out the intervention daily and at the same time, as with the baseline phase this was not wholly achieved. In addition to the sensory stimulation sessions during this intervention phase of the project GP was also participating in other activities in the rehabilitation centre; swimming, a leisure skills programme including fitness training and painting, daily living activities training, speech and language therapy, and a social and community skills programme. At the end of this intervention phase GP took a two-week holiday.

A post-intervention phase was set up to mirror the baseline phase using the same outcome measures and similar interventions devised, using normal clinical reasoning skills, by the treating therapists. This post-intervention phase was curtailed when GP discharged herself from the rehabilitation centre. Few data points were obtained from this phase.

For the three month duration of this case study project, running parallel to the procedure described above, kinematic recordings were made of GP's abilities to perform the drinking task in three modality conditions. The pantomimed gestures HAMMER, CARVE and WIND, previously described in case study 2, were also recorded using the left (non dominant) arm. These recordings were less frequent than had been planned as GP had started to feel over-burdened by the research project requirements. Two recordings were made in each of the baseline and intervention phases, and one recording was possible during the shortened post-intervention phase. In addition, an apraxia assessment was made monthly using the tests described in appendices D1-D4.

8.4(ii) Results

8.4(ii) a] Intervention outcome measures

Descriptive analysis of the outcome measures across each phase of the experiment showed a distinct change in central tendency between the baseline and intervention phases (Tables 8.4 and 8.5). Morley and Adams (1989 & 1991) suggested that the use of a broadened median (taking the average of five central numbers in a data set that has been rank ordered) in considering the central tendency of a data set was more appropriate than the mean score in clinical research. The broadened median, Morley and Adams suggested, was resistant to outliers in the data. They argued that such outliers might be a consequence of the conditions of data collection which cannot always be precisely controlled in clinical situations. Thus it was concluded that the broadened median summarised the data in a way which was sensitive to a reasonable proportion of the data, yet was also resistant to outliers (Morley & Adams (1991). It was considered

appropriate, therefore, to use such a broadened median in examining this case study data, as well as the standard deviation and inter-quartile ranges to determine the variability within each experimental phase.

Table 8.4 gives details of the data for GP's right hand performance across the ABA trial. Table 8.5 shows the left hand data set. A notable feature is the ***range*** of scores across all measures taken for both hands especially in the baseline period. This variability in performance was demonstrated throughout the period of investigation, but the descriptive statistics suggested that this might have lessened in the intervention phase when compared with the baseline phase (i.e. the reduction in standard deviation scores and interquartile ranges) across the main bulk of the outcome measures used. The only measures which did not demonstrate such a reduction in variance, if indeed that is what it was, was radial deviation in both hands and ulnar deviation in the left hand.

The data presented in Tables 8.4 and 8.5 also provided evidence which tentatively suggested an *improvement* in performance across the broad range of measures, as indicated by the mean and broadened median scores. Smaller or larger scores (broadened median and mean) in the intervention phase, according to the individual measure, could be viewed as an indication of improvement in performance (e.g. Timed task and opposition: *smaller* scores = improvement. Wrist extension, radial and ulnar deviation, maximum web space: *larger* scores = improved performance). GP's ability to oppose her thumb with her little finger, by looking at broadened median and mean scores, could be judged to have improved during the intervention phase (Tables 8.4 and 8.5) with reduced variability in performance shown in both hands by the reduction in standard

Table 8.4 - GP Right hand. Synopsis of data from each ABA phase.

RIGHT HAND	Baseline	Intervention	Post-Intervention
Timed Task (secs.)			
Mean score	81.5	58.33	49
Range	103 - 50	95 - 43	
Broadened median	**91.33**	**53.67**	
Standard deviation	16.84	15.81	
Interquartile range	22	17	
No. of data points	8	9	2
Wrist extension (degrees)			
Mean score	55	67.0	60
Range	40 - 64	66 - 76	
Broadened median	**56.11**	**67.0**	
Standard deviation	9.36	7.57	
Interquartile range	17.25	15	
No. of data points	12	9	2
Ulnar deviation (degrees)			
Mean score	24.42	26.89	66.5
Range	9 - 42	21 - 32	
Broadened median	**24.9**	**26.33**	
Standard deviation	10.14	3.33	
Interquartile range	10.5	5	
No. of data points	12	9	2
Radial deviation (degrees)			
Mean score	8.42	15.22	16.5
Range	1 - 16	8 - 21	
Broadened median	**7.14**	**16.0**	
Standard deviation	3.91	4.68	
Interquartile range	8.5	9	
No. of data points	12	9	2
Opposition (mm)			
Mean score	45.71	28.27	0.0
Range	75 - 15	46 - 0	
Broadened median	**43.1**	**30.0**	**0.0**
Standard deviation	16.14	14.22	0
Interquartile range	21.25	21	0
No. of data points	14	15	9
Web Space (mm)			
Mean score	11.73	19.53	24
Range	4 - 25	17 - 35	22 - 32
Broadened median	**12.67**	**20.6**	**24.33**
Standard deviation	6.92	5.75	3.94
Interquartile range	13	9	6.5
No. of data points	11	15	9

Table 8.5 - GP Left hand. Synopsis of data from each ABA phase.

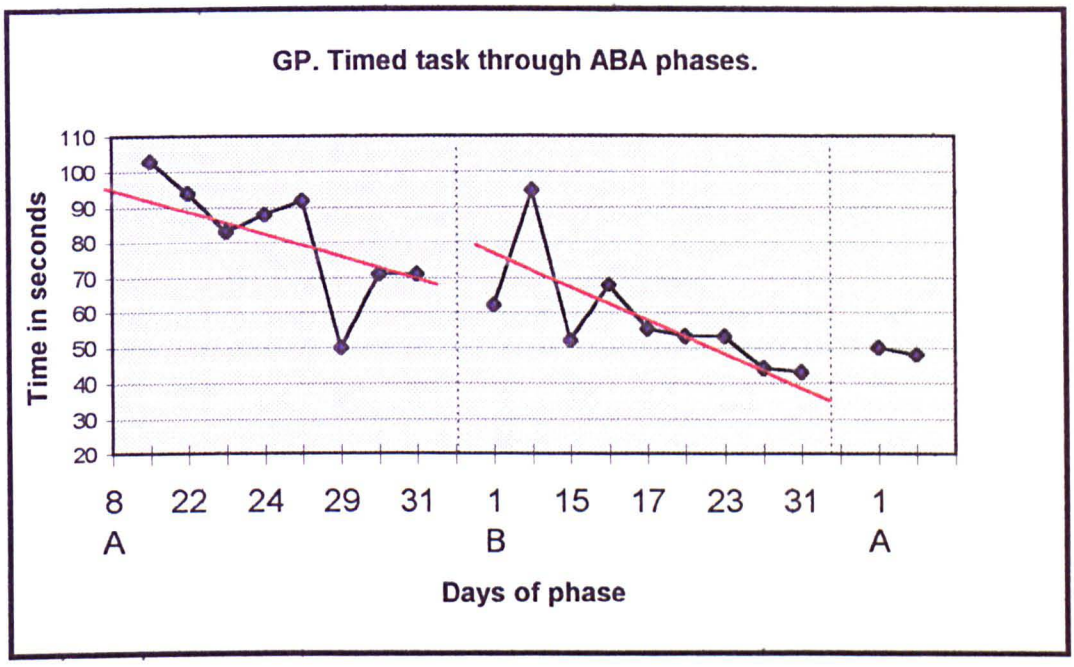
LEFT HAND	Baseline	Intervention	Post-Intervention
Timed Task (secs.)			
Mean score	62.0	42.78	38.0
Range	116 - 43	60 - 32	
Broadened median	**67.0**	**41.0**	
Standard deviation	25.18	9.15	
Interquartile range	31.25	15	
No. of data points	9	9	2
Wrist extension (degrees)			
Mean score	54.58	71.33	66.5
Range	41 - 66	66 - 76	
Broadened median	**55.61**	**70.67**	
Standard deviation	8.26	3.39	
Interquartile range	17.5	6	
No. of data points	12	9	2
Ulnar deviation (degrees)			
Mean score	19.33	24.78	12.0
Range	12 - 29	16 - 36	
Broadened median	**19.26**	**24.67**	
Standard deviation	6.24	6.6	
Interquartile range	6.75	8.5	
No. of data points	12	9	2
Radial deviation (degrees)			
Mean score	20.75	30.56	28.0
Range	5 - 39	0 - 46	
Broadened median	**20.09**	**30.33**	
Standard deviation	6.67	9.11	
Interquartile range	7	15	
No. of data points	12	9	2
Opposition (mm)			
Mean score	9.14	1.33	0.0
Range	26 - 0	20 - 0	
Broadened median	**9.7**	**0.0**	
Standard deviation	7.75	5.16	
Interquartile range	14.25	0	
No. of data points	14	15	9
Web Space (mm)			
Mean score	21.55	27.53	27.89
Range	8 - 38	17 - 35	22 - 32
Broadened median	**23.67**	**27.8**	**28.0**
Standard deviation	10.31	4.36	4.17
Interquartile range	18	6	7
No. of data points	11	15	9

deviation scores and interquartile ranges. Full opposition was achieved in both hands during the intervention phase (0 mm distance between thumb and little finger) and this was consistently maintained in the post-intervention phase. Ability to make a rounded cylinder shape with the hand (maximum web space measurement) and thus reduce the hyper-extension of the metacarpo-phalangeal joints might also have been judged to have improved during the intervention phase if the descriptive statistics were relied upon (Tables 8.4 and 8.5), though again, as with the other measures, any attribution of intervention effect to these changes could not be made by these descriptive data alone. Whilst variability in performance of this hand shape movement remained a feature throughout the time of investigation, a more consistent performance might be interpreted from the reduced range, standard deviation and interquartile ranges of web space measures found in both the intervention phase and the post-intervention phases (Tables 8.4 and 8.5). Reliance upon the descriptive data alone, however, is insufficient and a visual analysis of the various data sets might suggest a somewhat different and more complex story.

The data sets were plotted for visual analysis across the ABA phases to determine whether the 'improvements' seen in the intervention phase could actually be attributed to the intervention itself or a whether, from looking at the baseline data, they were a manifestation of an ongoing recovery process. In addition, the changes in variability of performance in each phase as suggested by the descriptive data could be usefully judged by visual inspection of the graphs. Graphical representation of the data indicated that, in fact, GP's right hand performance of the timed task showed a trend of improvement throughout the baseline phase despite the variability (Figure 8.11) and that this improvement continued in the intervention phase at about the same rate, indicated by the

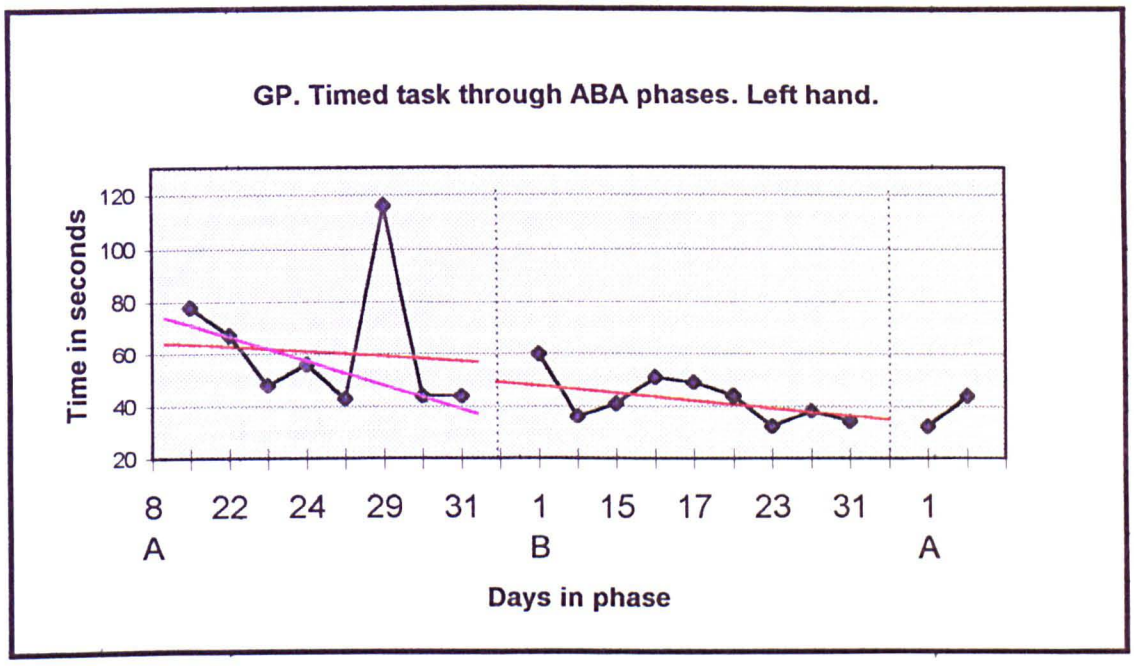
slope of the regression lines (Baseline r = .548 F(1,6) = 7.289 p< .05. Intervention r = .623 F(1,7) = 11.589 p< .05).

Figure 8.11 GP - RIGHT hand timed task performance across ABA phases



The left hand performance in the timed task showed a less dramatic graphical profile, but a slight trend of improvement could be seen in both baseline and intervention phases, though variability was still a feature (Figure 8.12). The regression lines for the left hand performanceaccounted for little of the variance seen [Baseline r= .0147 F(1,6) = 0.089 *NS*. Intervention r = .293 F(1,7) = 2.89 *NS*]. If the one extreme data point in the baseline phase were disregarded, then the regression line for that phase (shown in pink Figure 8.12) would suggest a steeper trend of improved speed of performance. Unfortunately for both these left and right hand measures only two data points were collected in the post-intervention phase, but some loss of performance ability might be interpreted from the data.

Figure 8.12 GP - LEFT hand timed task performance across ABA phases



The data for wrist extension, when plotted onto a graph, illustrated clearly the variability of performance in producing isolated movements, compared to the task performance data. In the right hand, so extreme was the variability in the baseline phase that the regression line was essentially non-meaningful ($r=.001$ $F(1,10) = 0.01$ *NS*), whilst the intervention phase indicated that the regression line only accounted for 24.8% of the variance [$F(1,7) = 2.32$ *NS*]. Visual inspection of the data (Figure 8.13) might suggest some degree of enhanced performance in the intervention phase, though variability was clearly a major factor.

Left hand performance of the wrist extension movement indicated a more consistent performance, but some gradual reduction in ability, during the intervention phase of the

Figure 8.13 GP - Wrist extension, RIGHT hand, through ABA phases.

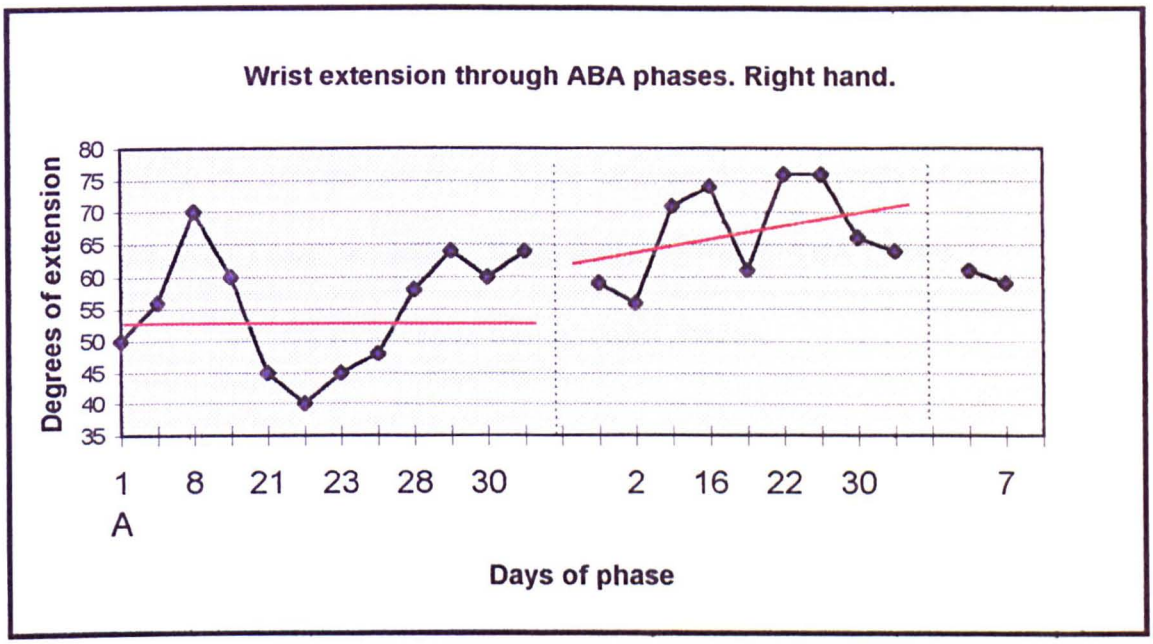
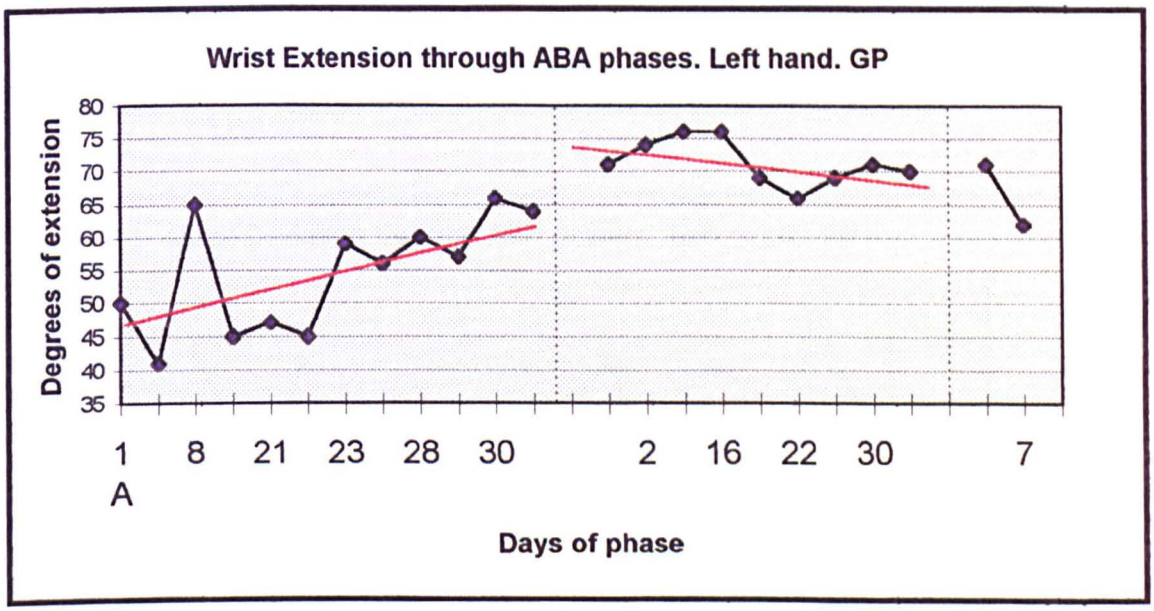


Figure 8.14 GP - Wrist extension, LEFT hand, through ABA phases.



research (Figure 8.14). Again, regression lines were not predictive of the data points, accounting for little of the variance [Baseline r =.256 F(1,10) = 3.44 *NS*. Intervention r =

.15 F(1,7) = 1.24 *NS*]. For both hands, the two data points that were collected in the post-intervention phase showed a deterioration in performance ability.

Ulnar deviation measurements showed variability in the left hand performance, but a better output in the intervention phase. Right hand performance (with the exception of

Figure 8.15 GP - Ulnar deviation (Right hand) through ABA phases.

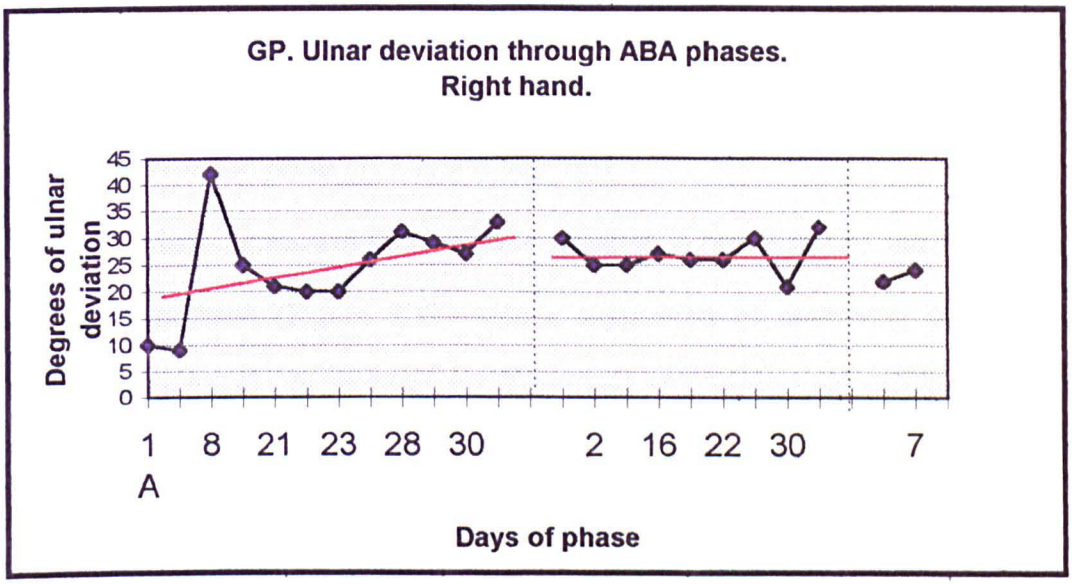
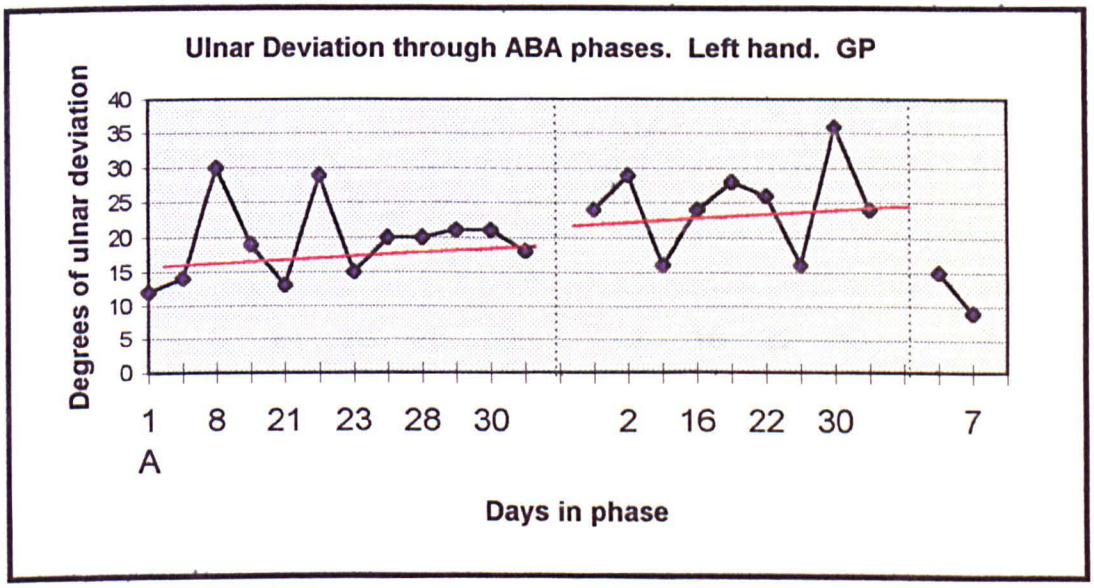


Figure 8.16 GP - Ulnar deviation (Left hand) through ABA phases.



one 'outlier' data point) indicated a steady trend of improvement in the baseline phase and a levelling off in the intervention phase (Figure 8.15 and 8.16). Again the regression lines, due to the variability in GP's performance, could not accurately predict the behaviour being measured with left hand baseline regression analysis accounting for only 3.8% of the varience and the intervention analysis only 1.1% [Baseline - $F(1,10) = 0.39$ *NS*. Intervention - $F(1,7) = 0.078$ *NS*]. The right hand ulnar deviation regression lines accounted for 23.3% of the variance in the baseline phase [$F(1,10) = 3.01$ *NS*] and 0.09% in the intervention phase [$F(1,7) = 0.006$ *NS*].

Radial deviation measures showed wide variation in both baseline and intervention phases in the right hand (Figure 8.17) with a suggestion of improving performance in the intervention phase. In contrast, a a clear trend of improvement was seen in the left hand during the baseline phase which was continued into the intervention phase (Figure 8.18).

Figure 8.17 GP. Radial deviation (Right Hand) through ABA phases

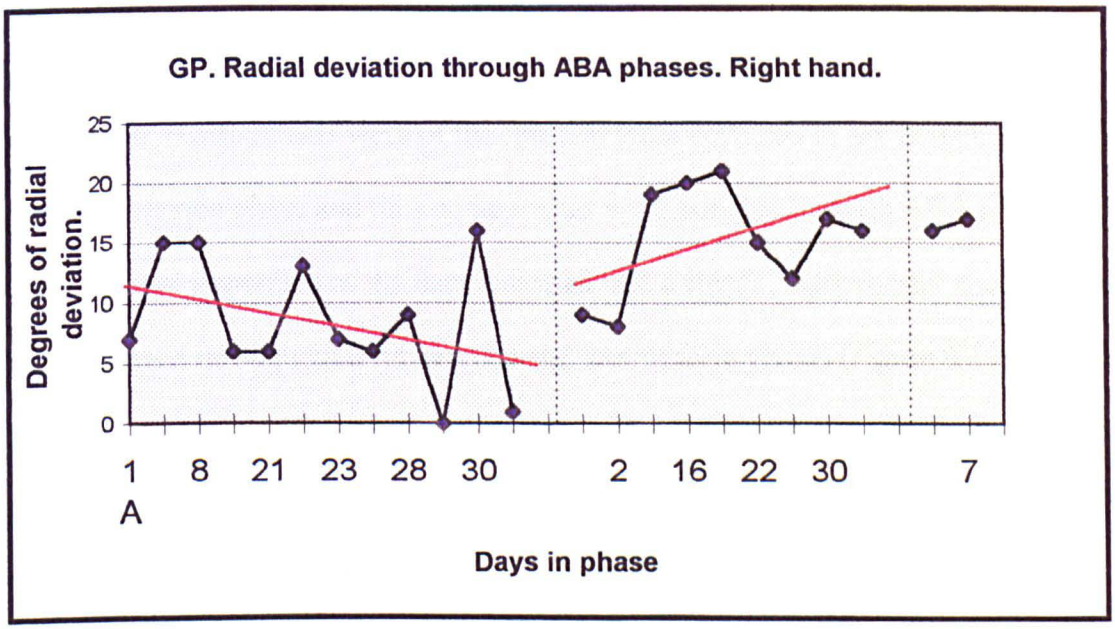
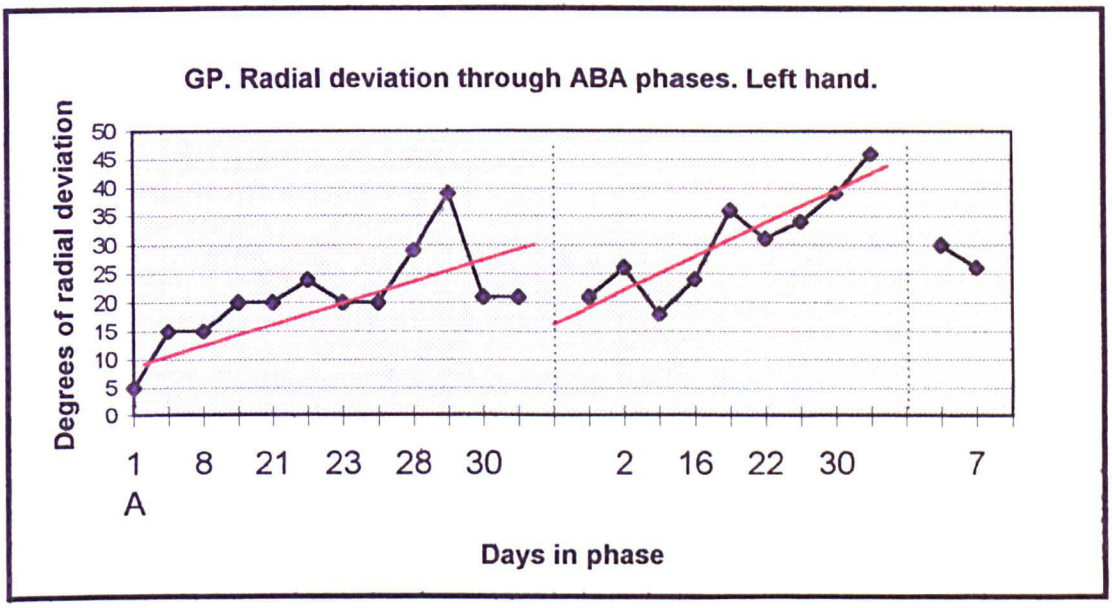


Figure 8.18 GP. Radial deviation (Left Hand) through ABA phases



The regression lines were poorly predictive of scores in the right hand [Baseline r = .15 F(1,10) = 1.77 NS. Intervention r = .27 F(1,7) = 2.64 NS], but were good predictors of performance in the left hand accounting for 56.7% of the variance in the baseline phase [F(,1,10) = 13.07 $p < .01$] and 59.9% of the variance in the intervention phase [F(1,7) = 10.46 $p < .05$].

Opposition measurements were of the few that were collected in any number during the post-intervention phase and thus comparisons were possible for each ABA phase of the study. Visual inspection of the data indicated that a steady improvement could be seen in the *right* hand throughout both baseline and intervention phases (Figure 8.19), and that the achievement of full opposition at the end of the intervention phase was likely to be the consequence of the continued recovery profile rather than to the intervention itself. Full opposition was maintained in the post-intervention phase. In contrast, the left hand acquired full opposition in the baseline phase of the study and maintained that ability throughout the subsequent two phases (Figure 8.20).

Figure 8.19 GP. Opposition (Right Hand) through ABA phases

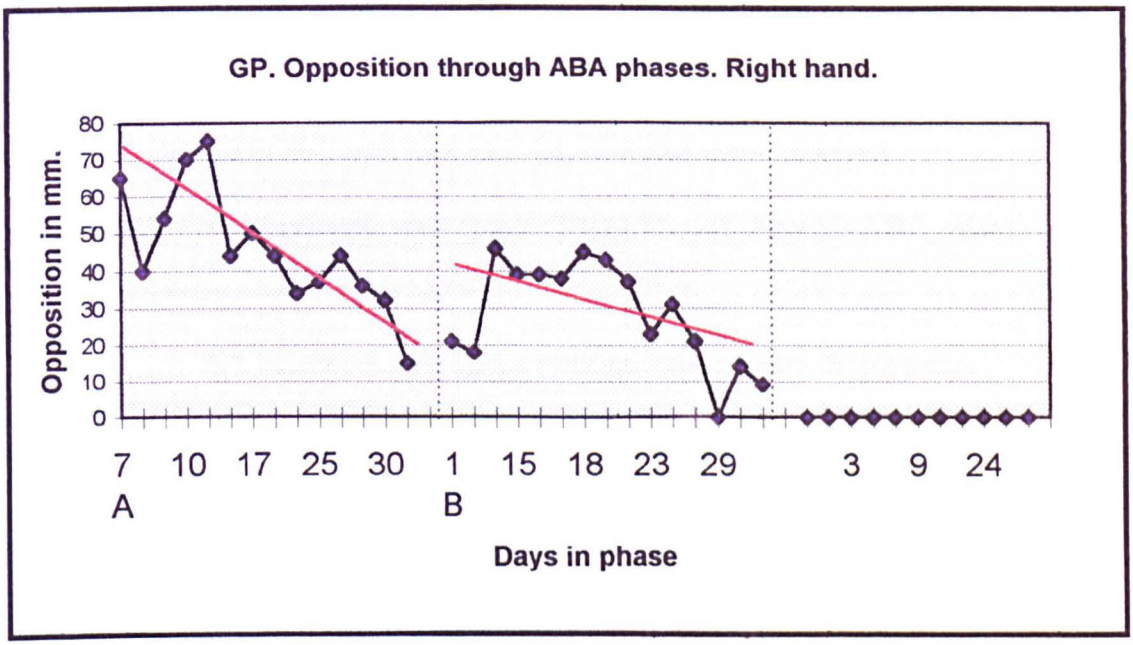
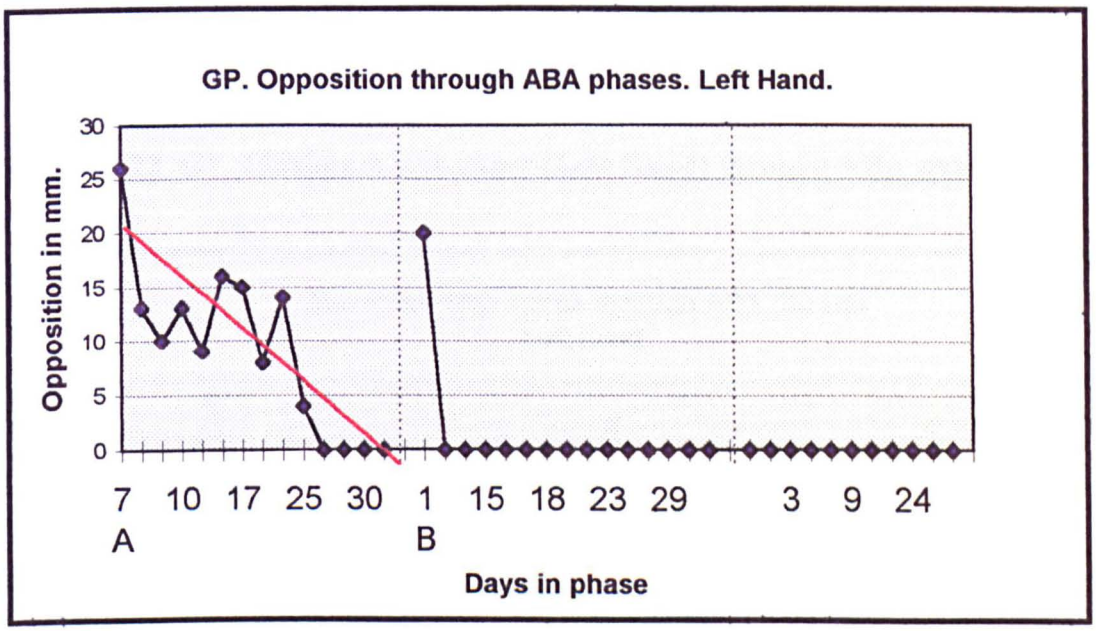


Figure 8.20 GP. Opposition (Left Hand) through ABA phases



The maximum web space measurements, which were an indicator of GP's ability to form a rounded cylinder shape with her hand (necessary for grasping cups, beakers, glasses etc) were taken in all three ABA phases. In the right hand, the baseline saw wide

variability of performance but a general trend could be observed of improvement. This was continued into the intervention phase with little difference in slope seen in the regression lines between the two phases (Figure 8.21). The post-intervention phase indicated a decline in ability to form the full, rounded shape required.

Figure 8.21 GP. Maximum web space (Right Hand) through ABA phases

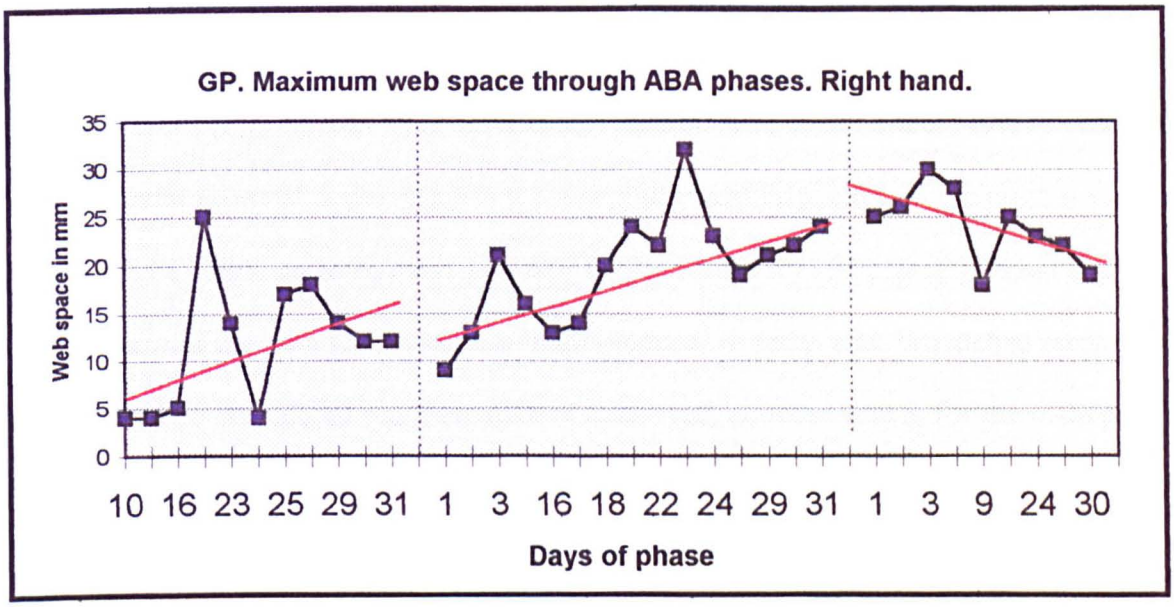
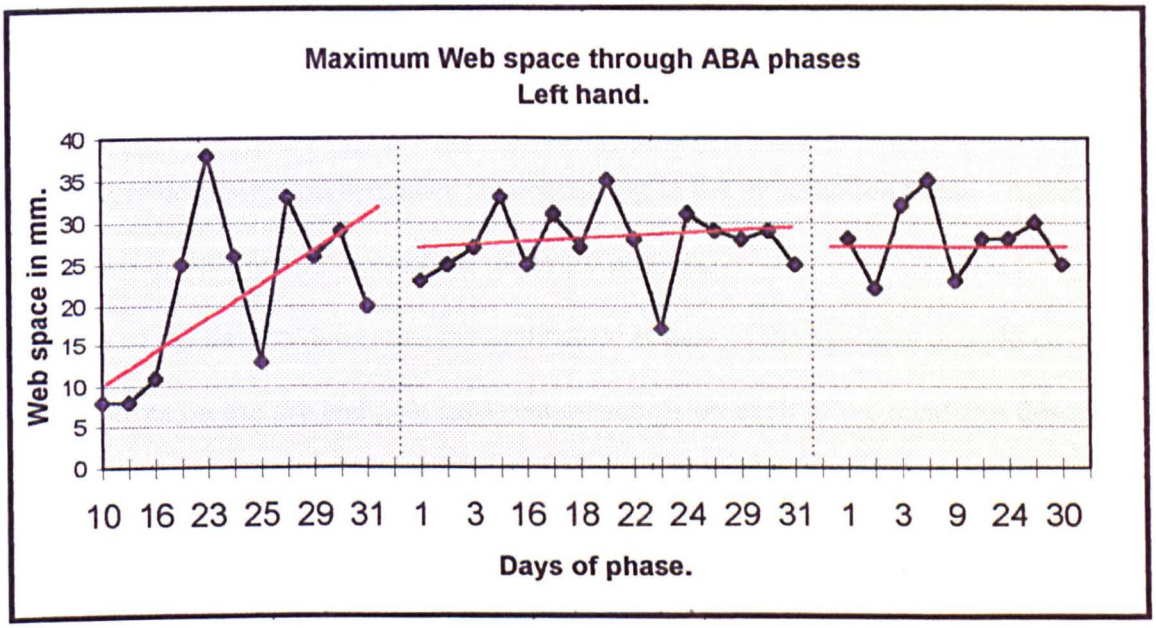


Figure 8.22 GP. Maximum web space (Left Hand) through ABA phases



The maximum web space measurements in the left hand can be seen, through visual inspection of the plotted data (Figure 8.22), to improve in the baseline phase and remain fairly stable in both the intervention and post-intervention periods though with continuing fluctuations and variation in the performance ability. Again, the regression lines shown on the graphs may be somewhat misleading as their predictive ability was generally low especially in the intervention phase ($r=.02$ $F(1,13) = 0.29$ *NS*] and the post-intervention phase ($r=.003$ $F(1,7) = 0.023$ *NS*]. Due to this variability in performance, autocorrelation analysis found no serial dependency in the data, but visual inspection of the plotted scores has allowed a judgment to be made that despite the variability some general trend can be seen in many of the data sets. Thus the 'improvement' in mean and broadened median scores could be considered representative of a recovery process which, for the main part, remained fairly constant in both baseline and intervention periods *regardless* of the type of therapeutic input being given (normal therapy or sensory stimulation). This was particularly noticeable in the timed task for both left and right hands, ulnar deviation, opposition and maximum web space measurements. The reduction in variability of performance in the 'B' intervention phase of the study (as demonstrated by the reduced standard deviation scores and interquartile ranges) remained a notable result, suggesting that a more consistent performance was occurring in the intervention phase. Again this might have been a natural part of the recovery process.

Analysis of the data to determine the *immediate* effects of therapy was completed using paired t-tests on the pre and post intervention scores on each of the measures described. During the baseline phase of the experiment only two of the twelve total measures taken showed significant differences in scores immediately following the intervention given; left hand radial deviation [$t(10) = 2.31$ $p< .025$] and left hand maximum web space

measurement [t(10) = 1.90 p< .05]. Figures 8.23 and 8.24 illustrate the trend of improving performance between the 'start' and the 'end' measures for most of the days

Figure 8.23 Immediate effects of intervention. Baseline phase. Left hand. Radial deviation.

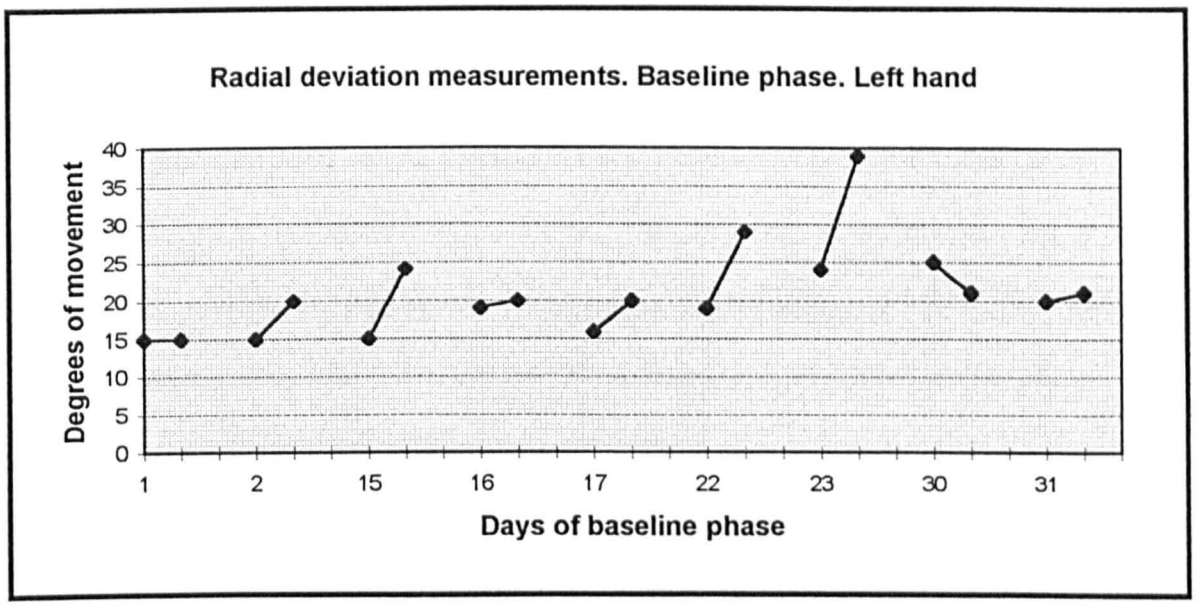
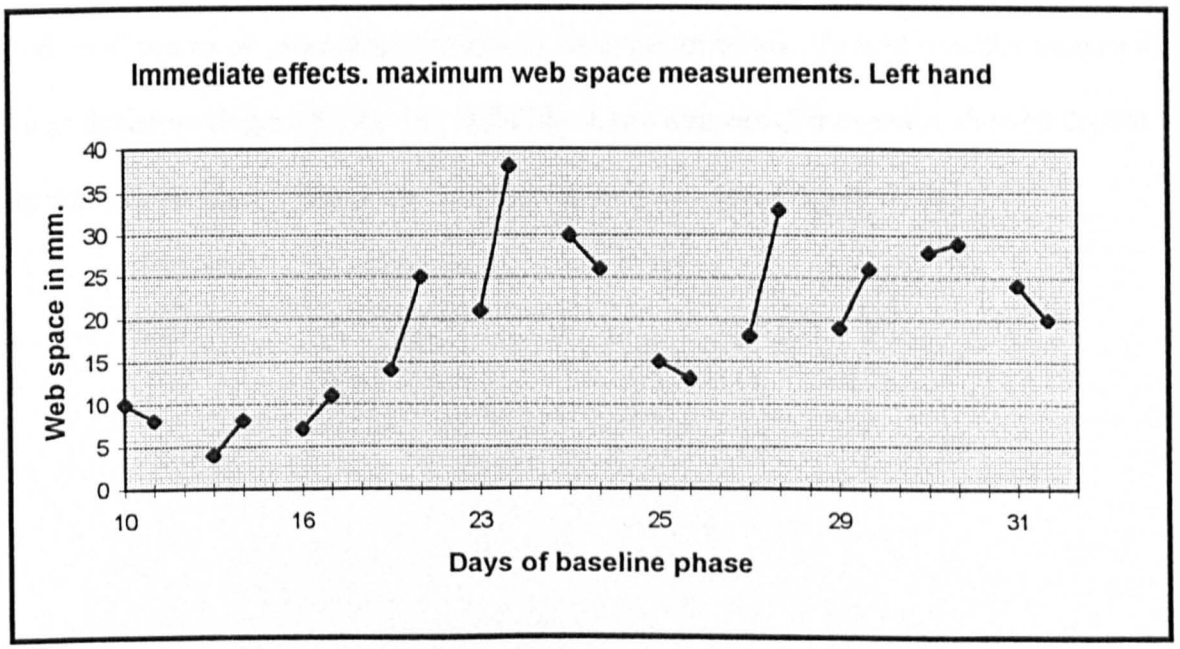


Figure 8.24 Immediate effects of intervention. Baseline phase. Left hand. Maximum web space.



of therapy input, with the radial deviation plots showing a more consistent pattern of improvement, whilst the web space measurements showed several days when improvement immediately following a therapy session was *not* apparent.

During the intervention phase, five of the twelve measures showed a significant difference immediately after the sensory stimulation procedure; left hand timed task [$t(8) = 3.28$ $p < .01$], left hand wrist extension [$t(8) = 2.03$ $p < .05$], left hand ulnar deviation [$t(8) = 3.42$ $p < .01$], right hand ulnar deviation [$t(8) = 1.9$ $p < .05$], and right hand radial deviation [$t(8) = 4.31$ $p < .01$] (Figures 8.25 to 8.29). This indicated an increase in efficacy of the sensory stimulation intervention over the baseline phase therapy of 150%. However, none of the measures which showed immediate improvement in the baseline phase repeated that improvement after sensory stimulation.

The plotting of the 'start' and 'end' measurements to show the immediate effect of sensory stimulation procedures indicated the quicker time taken for the timed task (Figure 8.25), and increased degree of wrist extension able to be performed after the therapy input (Figure 8.26). The degrees of ulnar deviation performed showed some days with dramatic improvements immediately following sensory stimulation, but very similar 'start' and 'end' scores on other days. The right hand performance showed a similar pattern for ulnar deviation (Figure 8.28), but radial deviation degrees of movement showed dramatic increases immediately following intervention on most days (Figure 8.29).

Figure 8.25 Immediate effect of intervention. Timed task. Left hand. 'B' intervention phase.

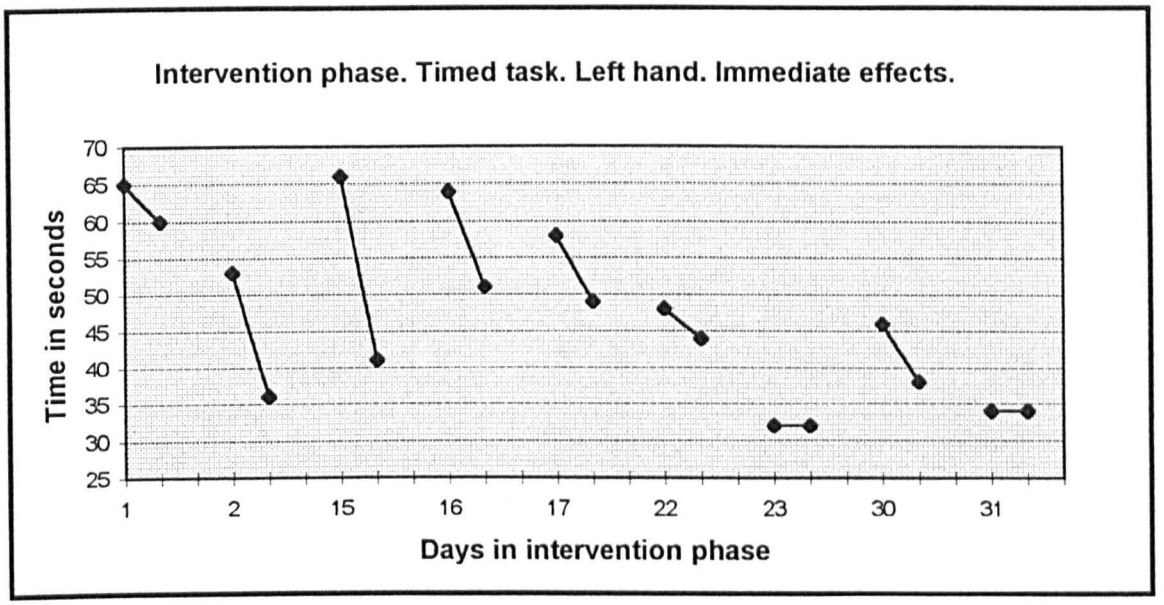


Figure 8.26 Immediate effect of intervention. Wrist extension. Left hand. 'B' intervention phase.

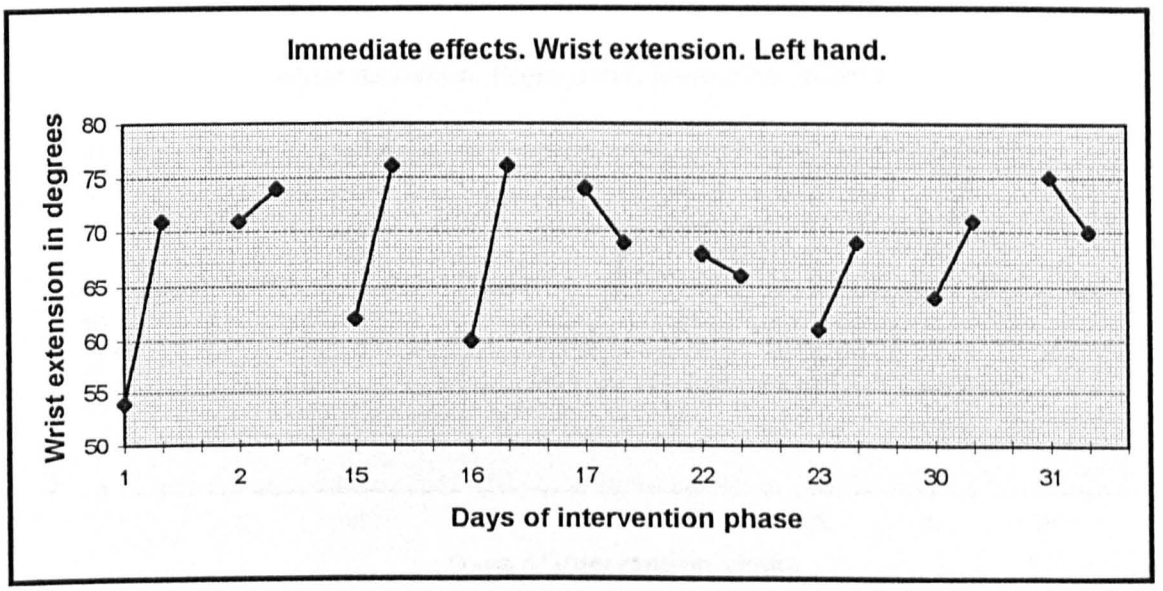


Figure 8.27 Immediate effect of intervention. Ulnar deviation. Left hand.

'B' intervention phase.

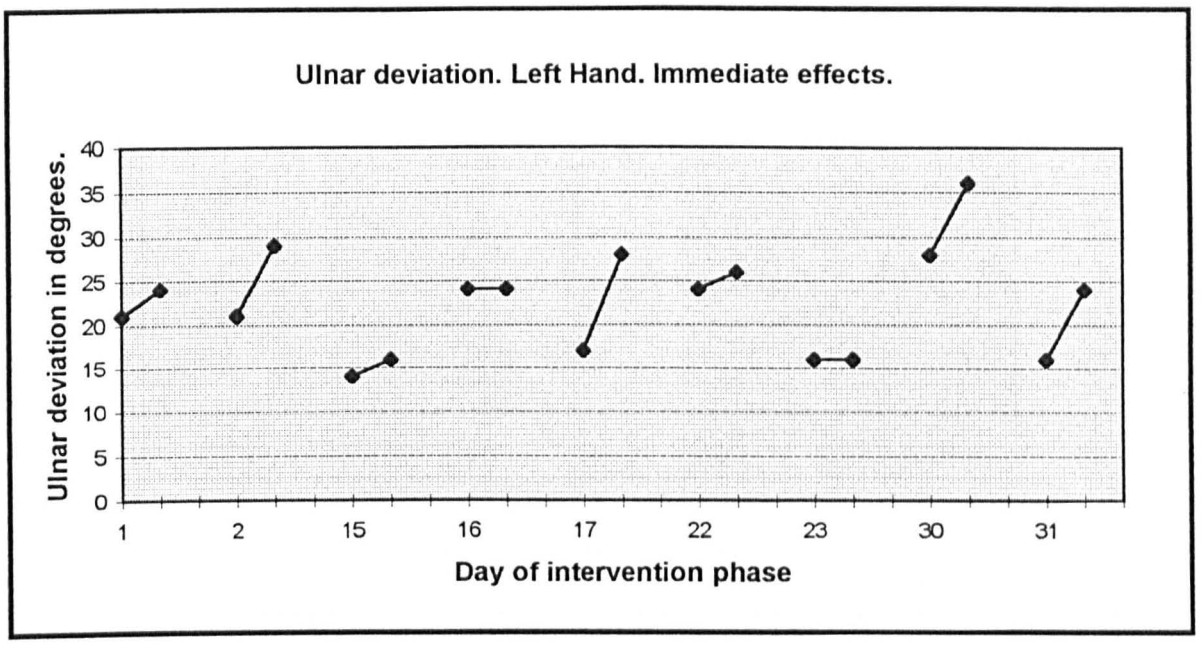


Figure 8.28 Immediate effect of intervention. Ulnar deviation. Right hand.

'B' intervention phase.

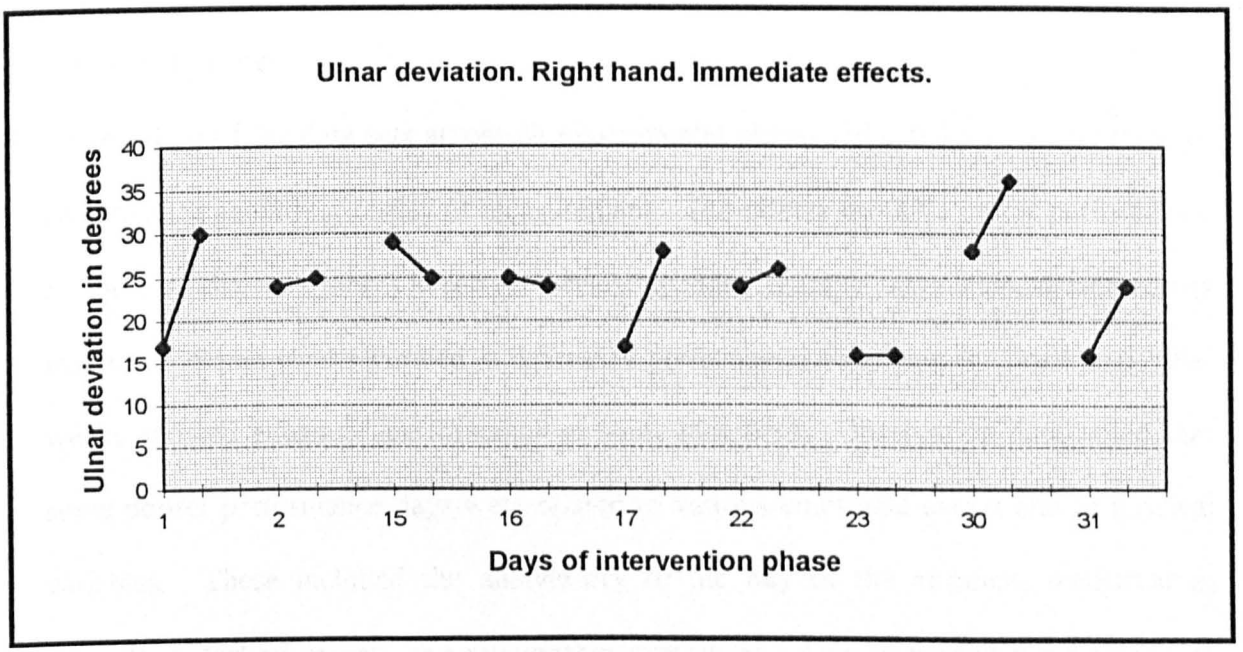
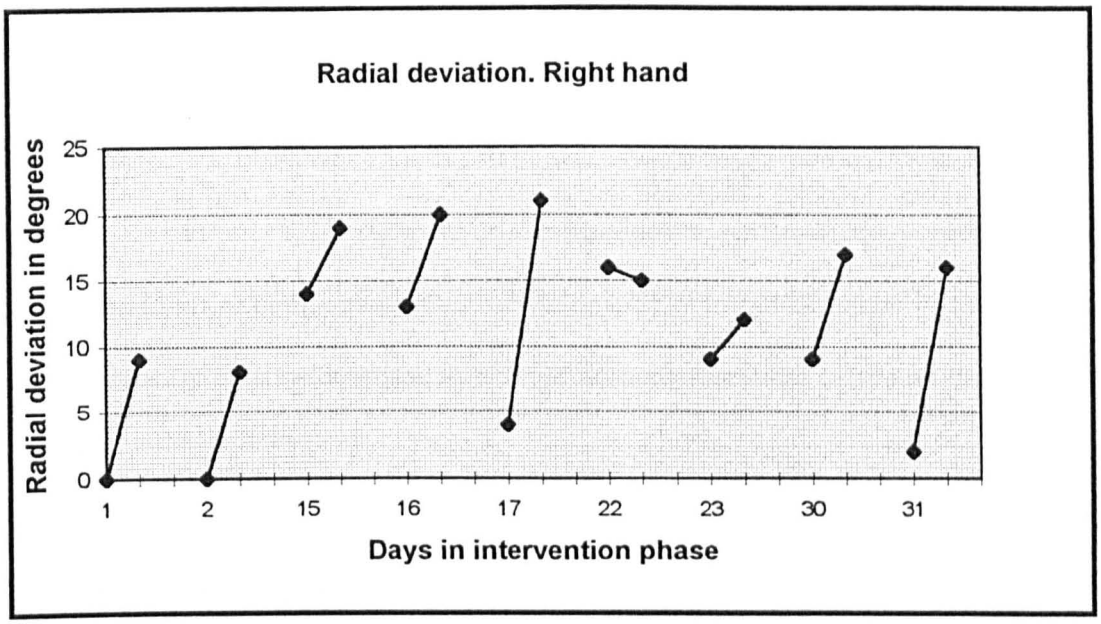


Figure 8.29 Immediate effect of intervention. Radial deviation. Right hand.

'B' intervention phase.



In the post-intervention phase only the maximum web space measurements could be analysed for immediate effects of therapy. No significant differences were found. Opposition in both hands remained at 0 mm, and inadequate data were available for other outcome measures.

Examination of the data sets across all experimental phases indicated no notable trend in performance according to day of measurement. The effects on GP's motor performance of the two-day (or more) weekend breaks without therapy were inconsistent across measures. Some weeks showed a decreased performance following the break and other weeks showed enhanced performance in some measures. Observation data noted that some poorer performance days were related to various emotional events and to physical variables. These included the anniversary of the day of the accident, menstruation difficulties, feeling unwell, and relationship difficulties. This supported the evidence of the pilot study.

Given the patterns that could be detected from visual analysis of the data across the ABA phases, the use of statistics to look for differences across the phases could be deemed inappropriate as any emergent differences would be reflecting the natural recovery trend. Throughout a wide range of outcome measures, therefore, a general pattern of improvement was observable in the data with the sensory intervention protocol being shown to be no more effective in improving motor performance compared to other therapeutic input although the reduction in variability in performance in the intervention period is worthy of consideration, as are the immediate effects of the intervention shown in the data.

8.4(ii) b] Apraxia assessment scores

The baseline phase apraxia assessments for this research were carried out five months after the original assessment when GP first attended the rehabilitation centre (Table 8.1). During this period GP had made progress in her rehabilitation but she remained severely apraxic with little change evident in the apraxia assessment scores. This was largely due to the nature of the scoring system with the large and inclusive 'impaired performance' category. The majority of GP's attempts at gesture or movement fell into this category throughout the period of investigation. GP concentrated hard during assessments in order to control her shoulder and elbow positions during performance of the requested hand and arm postures or pantomimed movements. Assessment was carried out at the *start* of the baseline phase, the *end* of the intervention phase and at the *end* of the post-intervention phase. Small changes in scores during the three phases of the investigation (Table 8.6) were achieved through improvement in GP's ability to control whole arm movements and in manipulation of some objects. GP remained severely impaired in her

ability to perform complex finger and hand gestures, and the observed motor output during testing procedures remained mostly in the impaired category.

Table 8.6 - GP apraxia assessment scores over time.

	Initial test		Baseline 'A'		Intervention 'B'		Post-intervention 'A'	
	Right	Left	Right	Left	Right	Left	Right	Left
Test 1 - gesture copying (max. = 40) (appendix D2)	17	16	19	19	19	18	25	23
Test 2 - object use test (max. = 36) (appendix D3)	14	18	12	18	19	20	21	19
Test 3 -ideational apraxia (max. = 12) (appendix D4)		0		0		0		6
Test 5 - De Renzi test. - gesture copying (max.= 72) (appendix D1)	6	6	9	9	12	12	18	16
Agnosia test (max.= 20) (appendix E)		19		20		20		20

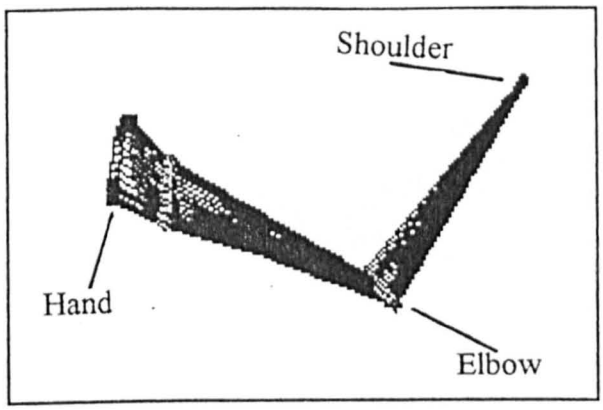
8.4(ii) c] Kinematic analysis

Due to the restrictions of time and the concern for not over-stretching GP with too many testing and measurement sessions, fewer recordings of movement were made during the ABA study than would have been seen as ideal. These recordings were taken during the same session as the clinical tests for apraxia at the start of the baseline phase, the end of the intervention phase and at the end of the post-intervention phase. Some results could be elicited, however, from this small data set. Stroboscopic reproductions of the requested gestures (HAMMER, CARVE, WIND) illustrated the slow, small and effortful controlled movements which GP produced. This is shown by the denseness of the

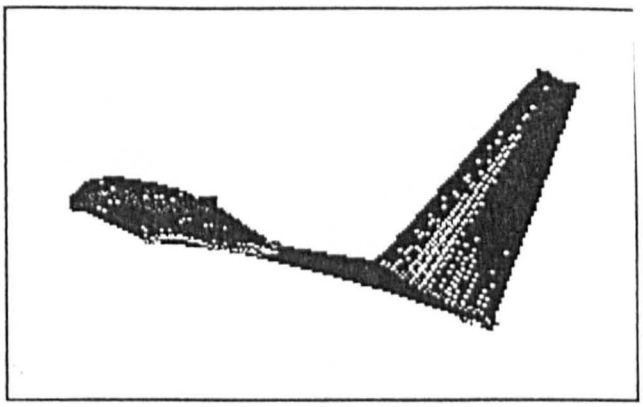
reproductions and the small amount of movement made in the baseline phase (Figure 8.30). GP managed to control her shoulder movement well, particularly during the HAMMER gesture. The speed and fluidity of movement were increased and improved slightly during the intervention phase of the investigation and more markedly so at the post-intervention phase recording (Figure 8.30). This is evident by the larger movements and the reduced density of the stroboscopic reproductions. GP managed these fluent movements without loss of shoulder control or alteration in the planes of movement. Observations made at the time of recording noted, however, that all movements were effortful and required focused attention. There was no sense of these being performed easily or automatically.

Figure 8.30 GP - gestural performance during the ABA phases.

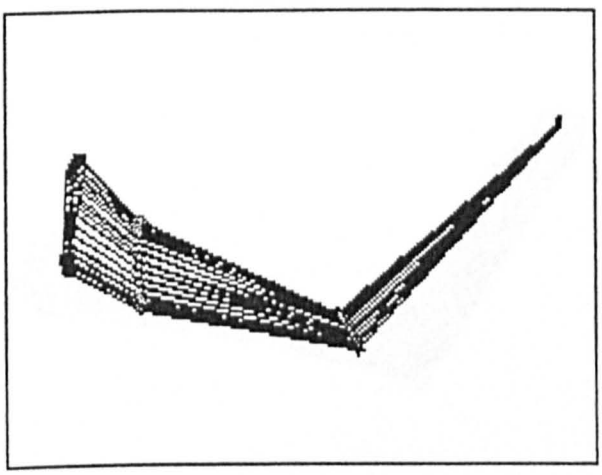
ai] HAMMER (baseline)



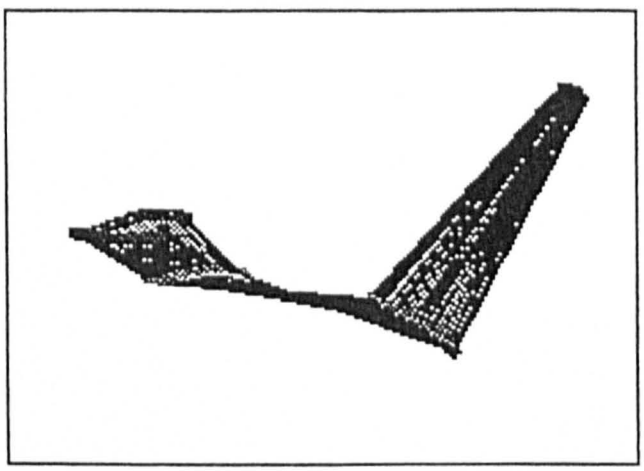
bi] CARVE (baseline)



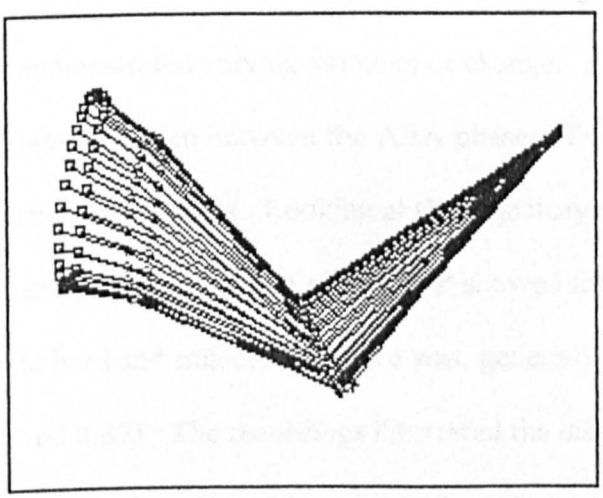
aii] HAMMER (intervention)



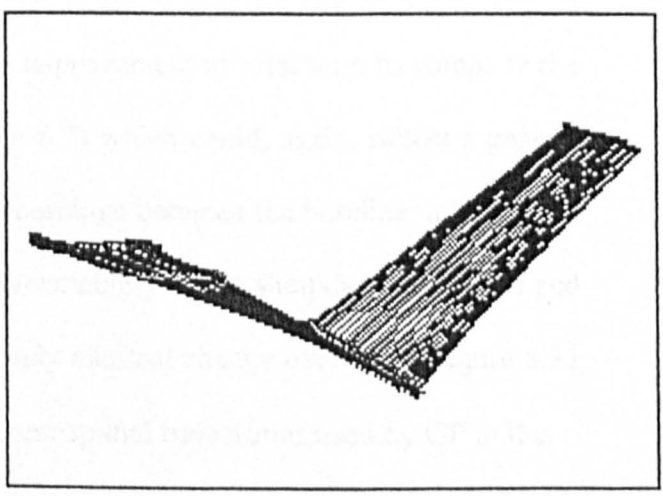
bii] CARVE (intervention)



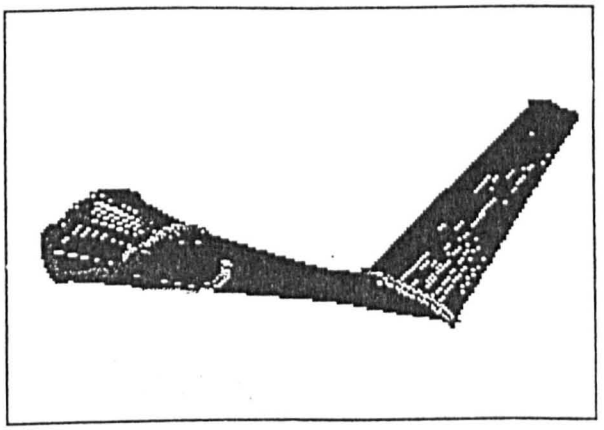
aiii] HAMMER (post-intervention)



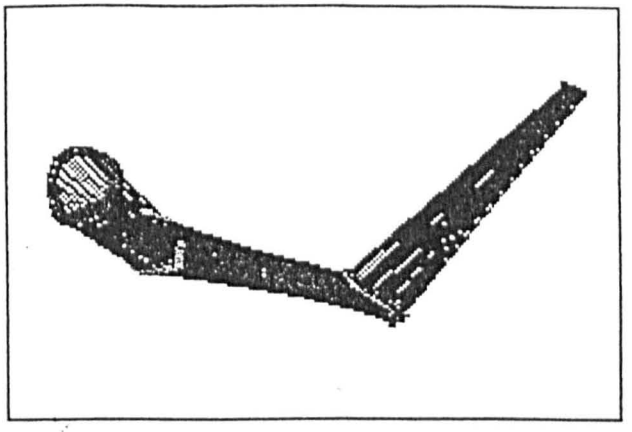
biii] CARVE (post-intervention)



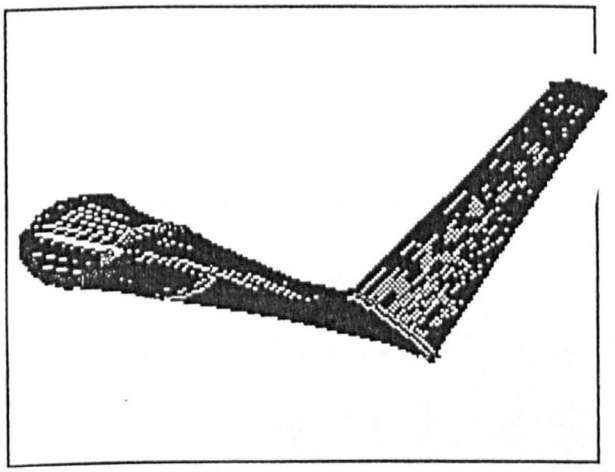
ci] WIND (baseline)



cii] WIND (intervention)



ciii] WIND (post-intervention)



Analysis of the drinking task recordings between the three experimental phases demonstrated varying amounts of change. An improvement in total time to complete the task was seen between the ABA phases (Table 8.7) which could, again, reflect a general recovery process. Looking at the trajectory recordings between the baseline, intervention and post-intervention phases, GP showed little variability in her shoulder movements and in her hand trajectory. There was, generally, only minimal change over time (Figure 8.31 and 8.32). The recordings illustrated the different spatial trajectories used by GP in the

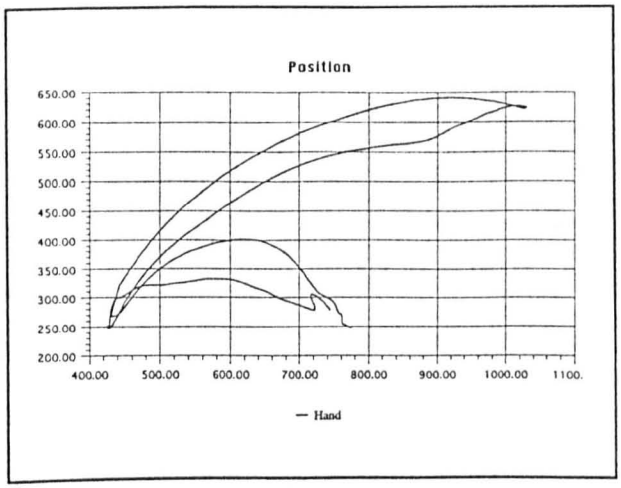
Table 8.7 GP -Total time taken to complete the drinking task through ABA phases.

	Baseline 'A'	Intervention 'B'	Post-intervention 'A'
Verbal condition (secs.)	14.5	9.4	7.74
Copy condition (secs.)	10.86	8.54	7.42
Real object condition (secs.)	14.84	11.9	10.88

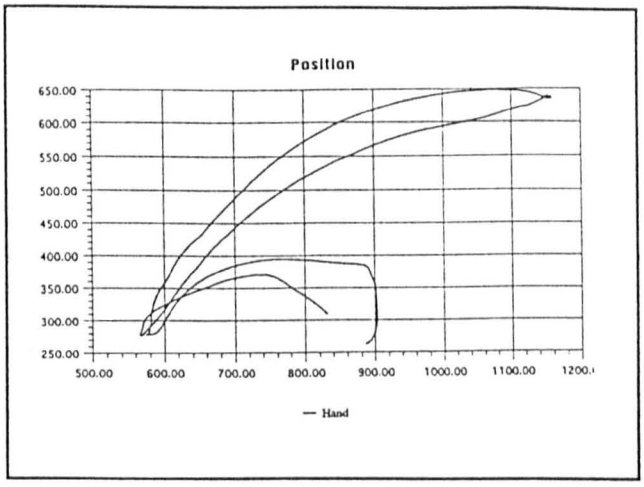
reach and return elements of the task, and also between the lift and down phases. The group study (chapter 5) provided evidence that *normal* movement in this task used even and consistent spatial pathways for these outward and return phases of the drinking task which GP did not show. All recordings were not demonstrating *grossly* abnormal patterns though, especially given the severity of the apraxia as shown by the clinical tests and GP's functional performance. This could be taken as more evidence of the dissociation between kinematics of movement and the clinical assessment and that GP's strategy for coping with the unpredictability of her movement performance was successful in the spatial elements of the task. The strategy might not be considered to be quite so successful in other kinematic elements of movement, however.

Figure 8.31 GP - Hand trajectories across ABA phases

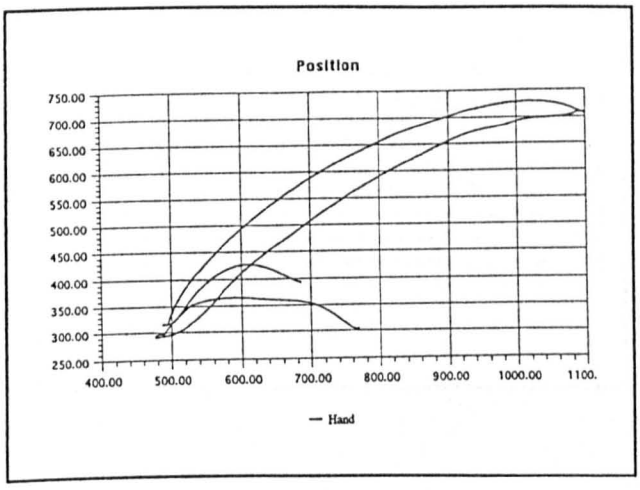
a] Hand trajectory. Baseline phase.



b] Hand trajectory. Intervention phase.



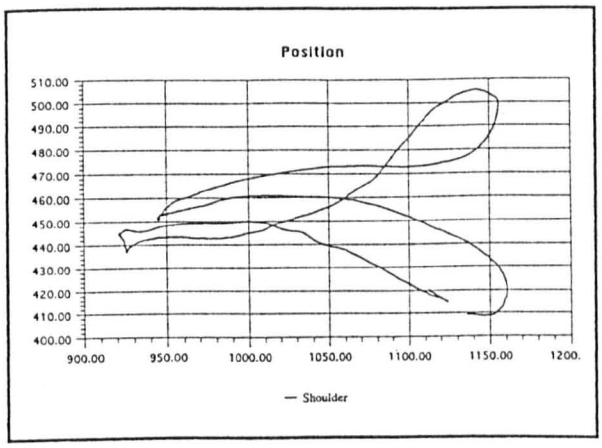
c] Hand trajectory. Post-intervention phase.



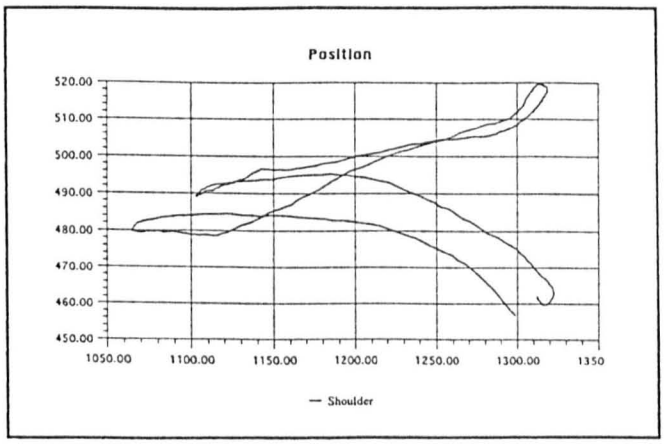
The shoulder trajectory recordings (Figure 8.32) also showed little convincing evidence of change. The shoulder trajectories remained abnormal throughout the three phases of the experiment when compared with those of the control group in Chapter 5, with some irregularity of shoulder use shown (Figure 8.32) though, again, not *grossly* exaggerated or abnormal compared with other apraxic profiles.

Figure 8.32 GP - Shoulder trajectories across ABA phases

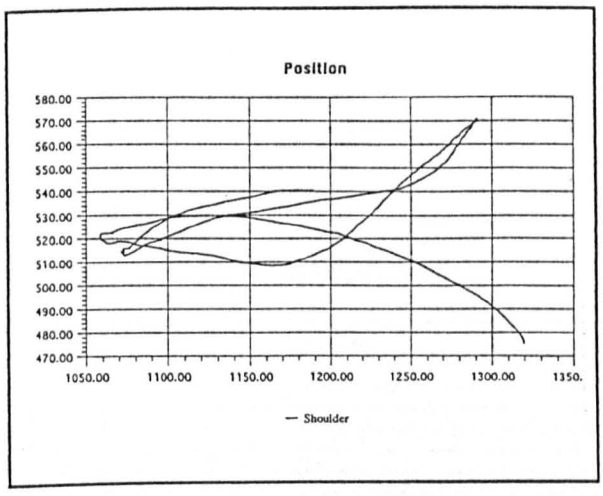
a] Shoulder trajectory. Baseline phase.



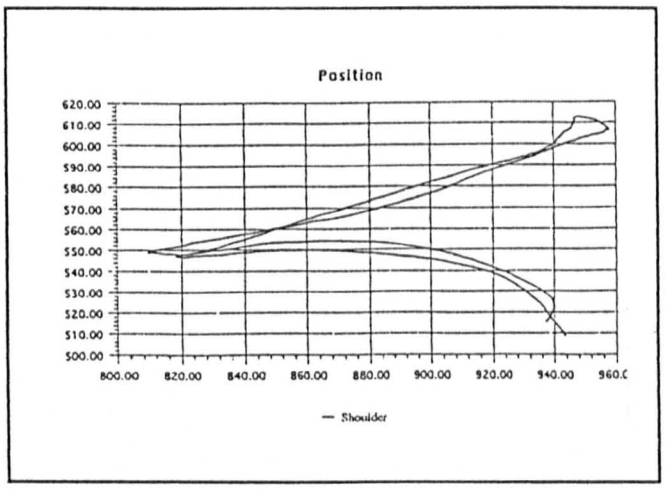
b] Shoulder trajectory. Intervention phase.



c] Shoulder. Post-intervention phase.



d] Example of normal shoulder trajectory.

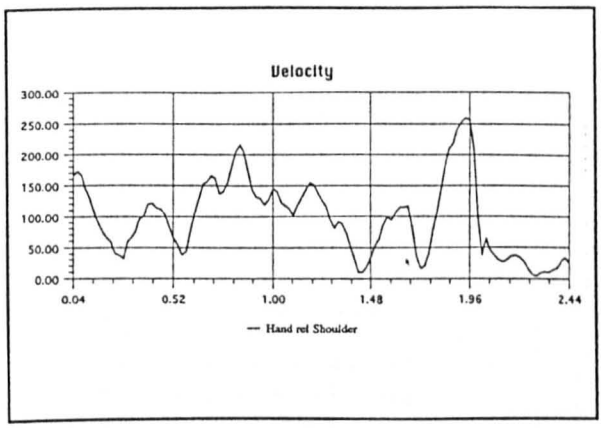


Other kinematic elements of GP's movement *did* show some abnormalities. The baseline recordings showed abnormalities in the velocity curve patterns, particularly in the reach phase of the task with multi-peak recordings being evident. These were somewhat modified in the intervention and post-intervention phase recordings (Figure 8.33). The multi-peak pattern seen in the baseline phase was less evident in the subsequent two phases and indicated that GP produced more fluid movement as time progressed, though

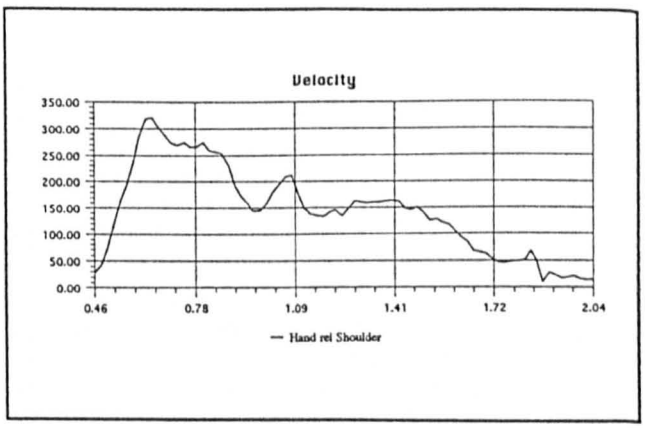
not so much as to be regarded as 'normal'. Observation of GP indicated that her attempts at movement were governed by a slow and effortful concentration to produce a 'good' result. The multi-peak pattern seen would support the argument of Hermsdorfer et al (1996) in suggesting that a slow, controlled strategy of movement output would produce such a velocity profile.

Figure 8.33 GP - Velocity curves in the 'reach' element of drinking task. ABA phases.

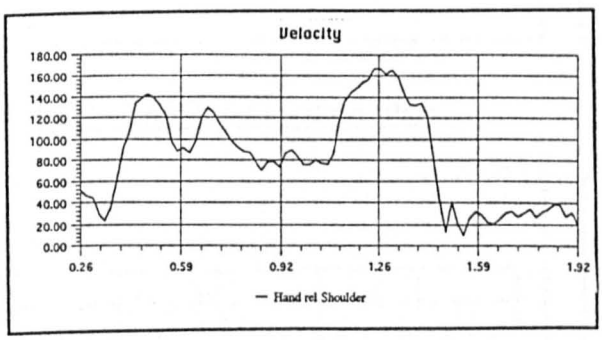
a] Baseline phase (A)



b] Intervention phase (B)



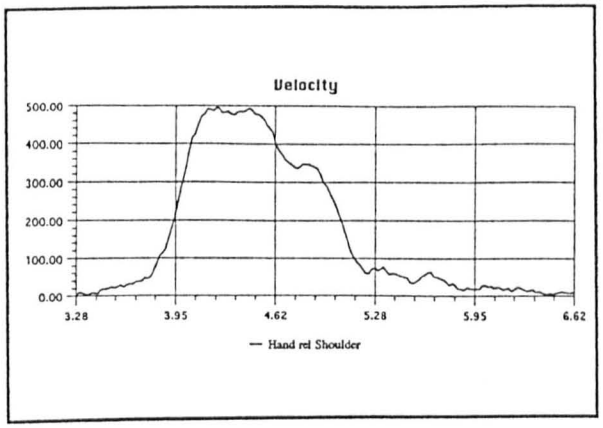
c] Post-intervention phase (A)



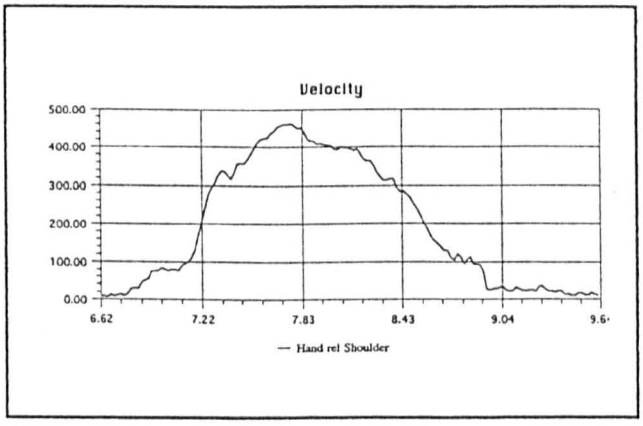
There was no evidence of a normal bell-shaped velocity curve pattern in the 'reach' phase of the task although these *were* apparent in the 'lift' and 'down' elements throughout the ABA phases of the study (Figure 8.34). Whilst this again provided some evidence that

Figure 8.34 GP - velocity curves in the 'lift' and 'down' elements of the drinking task .

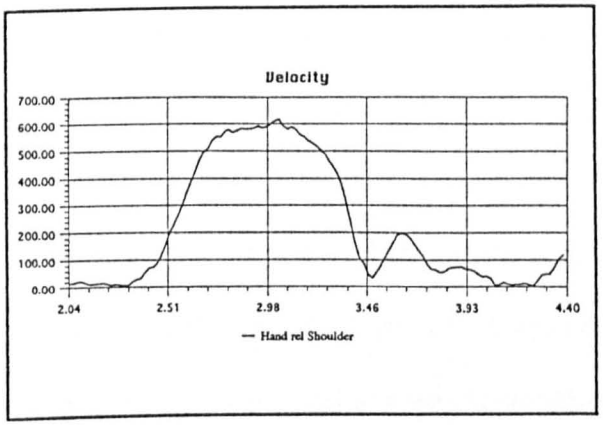
ai] 'Lift' - Baseline phase.



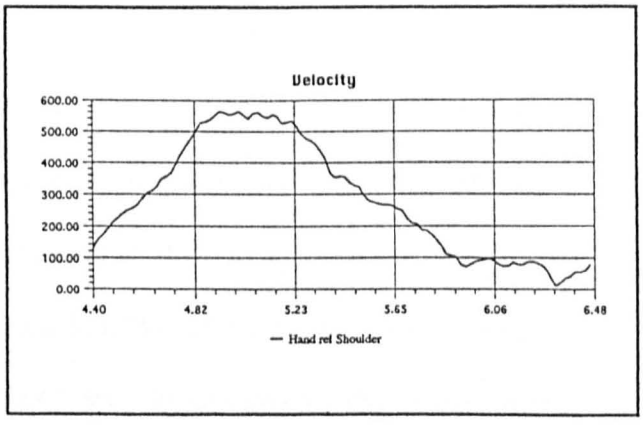
bi] 'Down' - Baseline phase.



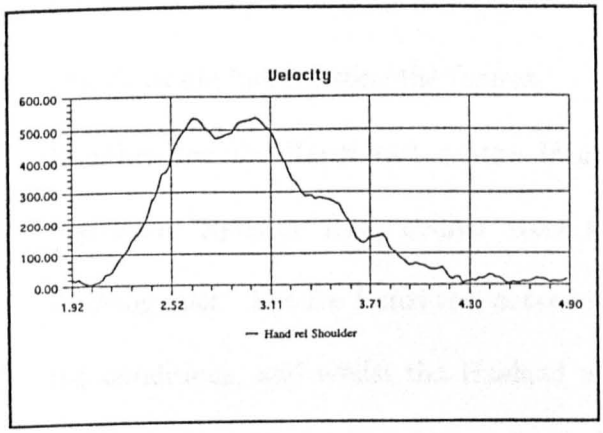
aii] 'Lift' - Intervention phase.



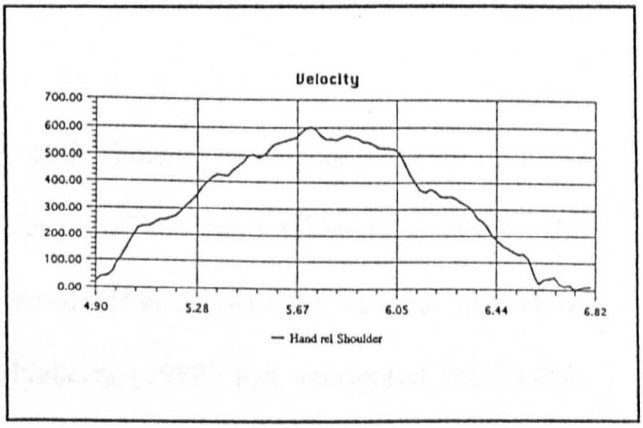
bii] 'Down' - Intervention phase.



aiii] 'Lift' - Post-intervention phase.



biii] 'Down' - Post-intervention phase.



GP's kinematic profile was essentially normal in many respects, there was noted to be a reduction in the long, low-velocity periods between task phases which emerged in the

intervention and post-intervention phases of the experiment. This could be taken to indicate a greater control over movement performance in managing transition stages of movement and changes of direction.

Analysis of the relationship between the clinical tests and the kinematics of movement indicated a modest correlation between the De Renzi gesture copying test and the total time taken in all three conditions in the drinking task [verbal r= -.58 *NS*, copy r= -.46 *NS*, real object r= -.*66 NS*]. A stronger relationship, though still not amounting to statistical significance was found with the apraxia test in appendix D2 (Haaland & Flaherty 1982) [Verbal r=-.72 *NS*, copy r= -.69 *NS*, Real object r =-.66 *NS*). The agnosia test, however, showed a strong relationship with all three conditions of the drinking task [Verbal r=-.97 $p < .05$, copy r= -.94 p= .059, Real object r =-.96 $p < .05$]. When a regression analysis was completed on the small data set available, the agnosia test was once more showing to be predictive of performance on the drinking task in relation to completion time [Verbal condition $F(1,2) = 28.19$ $p < .05$, Copy condition $F(1,2) = 15.56$ p= .058 *NS*, Real object $F(1,2) = 25.74$ $p < .05$] and was accounting for 93.4%, 88.6% and 91.8% of the variance for the different conditions. All these results should be taken with caution, however, as GP was scoring 19 and 20 of a possible 20 in the agnosia test and therefore the ceiling effects would be distorting the figures.

If either the De Renzi test or the Haaland and Flaherty test were entered into the regression equation first, neither were predictive of the time taken to complete the drinking task. The De Renzi test accounted for less than 25% of the variance in each of the conditions, and whilst the Haaland and Flaherty (1982) test accounted for 73.6%, 78.5% and 74.3% of the variance for each of the three verbal command, copy and real-object conditions this was not amounting to statistical significance [verb $F(1,2)$= 5.58 *NS*,

copy F(1,2) = 7.31 *NS*, real-object F(1,2) = 5.79 *NS*]. When compared with the previous information in this thesis, this strong showing by the Haaland and Flaherty test is surprising as usually the De Renzi test has emerged as the more sensitive one. The small data set might be the reason for this. Alternatively, GP could be of a 'sub-type' where the verbal command performance required by the Haaland and Flaherty test was the crucial element in eliciting apraxic errors.

8.4(iii) Discussion

This intervention study to determine the efficacy of a sensory stimulation intervention on motor performance in a person with ideomotor apraxia provided some evidence that a recovery process was taking place, though it was twelve months since the trauma when the baseline phase was begun. This information, in itself, is useful in understanding the long-term nature of recovery after head-injury and of improvements in apraxic performance that can occur. The experimental sensory stimulation procedure was shown to be no more effective than therapy intervention *without* sensory stimulation when looking at a wide range of outcome measures. However a reduction in the variability of motor performance in the intervention phase, as shown by the standard deviation scores and interquartile ranges across all the outcome measures except ulnar and radial deviation in the left hand (Tables 8.4 and 8.5), indicated a more consistent and reliable performance. Such improvement in consistency, though, might well be attributed to the natural recovery rather than the specific sensory stimulation protocol. Such reduction in variability, it could be argued, might be a useful outcome measure in itself in looking for improvement or recovery. In rehabilitation the provision of a care package for people

with apraxia has, ultimately, to accommodate the worst possible performance within the variable range shown by the individual. If, therefore, therapeutic intervention could be shown to reduce that range of variability then more confidence might be placed upon the person's daily performance levels, though such intervention effect has not been convincingly shown here.

The results from the outcome measures which demonstrated a gradual improvement over time in both the baseline and intervention phases were compared with those of the apraxia assessment scores. Some disparity was found here, with little change noted in the apraxia scores between experimental phases. This was largely attributed to the inability of the apraxia tests to adequately monitor **change** in performance unless of a gross nature. Small and subtle changes in ability to perform the requested gestures, movements and pantomimes could not be picked up by the categorical system of scoring. General observations made during testing procedures were that GP produced a better performance of the requested elements of the test over time. However, most performances continued to fall within the 'impaired performance' category and this resulted in little change in the scores of each test.

The kinematic recordings were carried out in order to overcome the inadequacies of the apraxia tests in measuring change in performance with a greater accuracy and reliability. The constraints of the investigation, including not over-taxing GP in measurement and testing, resulted in fewer computergraphic recordings than might have been seen as ideal. Results of those recordings demonstrated small elements of change and improvement over time. Speed and fluidity of movement were seen to be increased in the HAMMER, CARVE and WIND recordings, especially in the post-intervention phase. Other elements

of the kinematic analysis which demonstrated an improvement in motor performance were that the velocity curves lost the long, low-velocity patterns which had indicated uncertainty in changing movement direction, and some reduction in the multi-peak velocity pattern seen in the 'reach' element of the drinking task was evident. These results might be interpreted in the light of the Hermsdorfer et al (1996) argument which suggested that it was the strategy adopted by the individual patient which produced these differing kinematic profiles, and the success of those strategies. In GP, the slow control-process approach to moving in order to produce a 'best possible' result could have resulted in the multi-peak pattern and the long, low-velocity period in the baseline phase as she sought to correctly perform the movement. As time passed, this strategy might have become more efficient and so produced the changes seen in the kinematic analysis. In addition, it is interesting to note that GP's 'strategy' (if Hermsdorfer is correct) was successful in achieving a good performance outcome in the spatial elements of the movement as demonstrated by the trajectory recordings for both the hand and the shoulder. The strategy was not so successful in the temporal elements of the task, however, as demonstrated by these multi-peak patterns and the low-velocity periods though it was evident that some improvement in these temporal aspects did occur over time.

Analysis of the relationship between the kinematics of movement and the clinical tests revealed interesting results. The clinical tests could demonstrate only minor changes over time due to the categorical system of scoring, but observation notes indicated that whilst GP improved her praxic function in large arm and hand movements and demonstrated an ability to control the plane of movement, orientation of her hand, and general speed and fluidity of movement, she remained severely impaired in her fine finger movement and

dexterous hand function. This impairment remained throughout the period of investigation with little evidence of change. The clinical tests were not shown to correlate significantly with the total time taken to complete the drinking task, nor to predict performance. As with previous analysis of this relationship, in GP the agnosia test was shown to significantly relate to and predict completion time in the drinking task. Given that GP scored fully on this particular test, however, this result cannot be taken as supportive evidence for the power of the test. An interesting feature that did emerge was the strength (comparatively) of the Haaland and Flaherty (1982) test over the De Renzi test. This might be considered to indicate that GP fell into a 'sub-type' of apraxia where the verbal command element was critical in picking up performance errors rather than just the copy command inherent within the De Renzi test. This might signify the relative intact nature of the vision-to-action routes in GP but an interruption of the semantic-action routes, even though GP had no language deficit other than the bucco-facial apraxia which made her mute.

The outcome measures showed that significant *immediate* effects of therapy in the baseline phase were minimal (only two of the twelve measures). It might be expected that therapeutic intervention would have *some* immediate effect on that which it is aiming to improve. This, however, was not so in the baseline phase. It could be argued that tiredness following an arduous therapy session might be a factor to consider. If this were so, then immediate effects of therapy might not be seen; only cumulative effects. When compared with the sensory stimulation intervention, however, significant immediate effects of that intervention were seen in five of the twelve measures taken. This perhaps represented a greater efficacy of the intervention, at least in the short term.

It was interesting to note that none of the measures which indicated significant efficacy of therapy in the baseline phase showed significant changes in the sensory stimulation phase of the experiment. This might be taken to indicate a need for *both* types of therapeutic intervention in order to maximise the chances of improving motor performance in the apraxic patient. A further study using a methodology which allowed comparison of interventions might provide evidence to support this supposition. Such a project could compare verbal and visual mediation strategies in performing hand and arm exercises *with,* and *without,* sensory stimulation.

During this current investigation, the effects of the sensory stimulation intervention on functional performance in self-care and everyday activities was not monitored. The practicalities of reliably recording GP's performance in activities of self-care, using observation sheets, was considered beyond the scope of this project. Whilst any change in praxic function only really has relevance to the individual if it is transferred to a meaningful functional activity, the problems of recording and monitoring such change reliably and objectively were many. It was considered that the drinking task recordings would provide some evidence which might *simulate* functional change, and be sufficiently robust in reliability and objectivity terms. Future research might investigate how self-care activities could be observed and recorded in a practical but scientific manner, though lengthy check-lists and observation sheets used in naturalistic settings have been shown by other researchers to be problematic (Carr & Shepherd 1980, Miller 1986, Mayer et al 1990, Arnadottir 1990, Schwartz et al 1993, McDonald et al 1994, and Foundas et al 1995). In this investigation with GP a separate project was used to explore functional change using task break-down strategies.

8.5 Task breakdown in self-care activities

When GP was admitted to the rehabilitation centre seven months post-injury, she was dependent for all her self-care activities. Observation data indicated the extent of the difficulty she had. For example, in drinking GP was unable to position her hands correctly in relation to a beaker of liquid prior to gripping and lifting the beaker. If a beaker was passively placed in her hands she was able to sustain a grip but unable to lift and position the beaker in correct orientation to her mouth for drinking, resulting in spilling the drink. GP usually used a straw to drink, lowering her head towards the table to suck at the straw. In eating, GP needed help to position her right hand around a large-handled spoon but was then able to sustain a grip whilst bending her elbow. She was not able to position and orientate the spoon in relation to a dish of food, nor to retrieve food from the dish. When food was placed on the spoon for her, she could not adjust her movement to achieve the correct position of the spoon in relation to her mouth resulting in spillage. She was, though, able to lift some finger food from a plate and move it towards her mouth. This finger feeding was haphazard, messy, and unacceptable to GP who preferred to be passively fed in such circumstances.

A task-breakdown strategy was used on these self-care activities, coupled with verbal and visual mediation strategies (i.e. GP was asked to repeat the task break-down descriptions sub-vocally as she carried out the activity, and also asked to look at her shoulder, elbow and hand positions to check they were correct). This combination was selected in order to achieve the most effective remediation intervention as suggested by other researchers (Ayres 1985, Wilson 1988, Goodgold-Edwards & Cermak 1990, Croce 1993, Pilgrim & Humphreys 1994). For the activity of drinking, the movement instructions were given

while GP repeated them sub-vocally. She then attempted to carry out the individual movement described, while the therapist facilitated or adjusted the movement as required. The following sequence was used with a tall, empty beaker without handles:-

(Note: asterisk denotes later additions to the sequence.)

- I put my hands flat on the table.
- I slide my hands forward at a level with the cup.
- I look and see that my elbows are on the table top.
- I turn my hands onto their sides.
- I slide my hands towards each other until they touch the cup.
- I close my hands together tightly around the cup.
- I look and see my hands are in the right place.*
- I keep my elbows on the table.*
- I bend my elbows slightly, slowly lifting the cup halfway to my mouth.
- I stop my arms moving but still grasp the cup.*
- I bend my head down to the cup.
- I put my mouth on the cup.
- I use my head to tip the cup and drink.
- I lift my head.
- I straighten my elbows.
- I put the cup onto the table.
- I open my hands and straighten my fingers.
- I slide my hands sideways away from the cup.
- I slide my hands towards me and off the table.*

The first movement component was taken in isolation and practiced. When achieved, the next component was added to it and both completed together. When these were achieved, the third component was added and so on. Later additions were made to the sequence (as indicated by asterisks) in the light of errors made at certain points during trials. GP achieved each movement component within 3 attempts and only minor physical adjustments to positions were required by the therapist. The whole sequence was learnt very quickly and could be reliably carried out with verbal instructions after two therapy sessions.

After successful completion of the task to verbal instructions, the written instructions of the drinking sequence were introduced. GP was asked to read each item in the sequence before carrying out the instruction. The items in the sequence were not progressed unless the movement or position was correct. Minor adjustments were necessary with verbal prompting (e.g. "Look again at where your hands are" or "Are your elbows in the right position?"). A fourth therapy session, with six repetitions of the complete sequence, consolidated the successful drinking task using written instructions. GP eventually learnt the sequence off by heart, and eventually only checked at 'danger points' that her hands were in the correct position, or that her elbows were placed correctly. Full recitation of the instructions then became redundant.

This task break-down strategy had shown itself to be very effective for GP and so similar procedures and strategies were carried out for eating and all other self care tasks. Each task, and each item of clothing in dressing and undressing needed a detailed and complex break-down into small component parts to be practiced until completed. Tasks were selected for practice in the order in which GP felt to be most important for her. For

example, early in the rehabilitation she wanted to independently put on her own moisturising cream. This was broken down into the following phases :

- put right hand on the pump-action top.
- put left hand under the nozzle.
- push pump with right hand.
- put left elbow on the table.
- bring left cheek down to raised left hand containing cream.
- Do 'nodding' action.
- do 'no' action.

Repeat with other hand :

- put left hand on the pump-action top.
- put right hand under the nozzle.
- push pump with left hand.
- put right elbow on the table.
- bring right cheek down to raised right hand containing cream.
- Do 'nodding' action.
- do 'no' action.

For forehead and chin :

- put right hand on the pump-action top.
- put left hand under the nozzle.
- push pump with right hand.
- put left elbow on the table.
- bring forehead down to raised left hand containing cream.
- Do 'nodding' action.

- do 'no' action.

- put right hand on the pump-action top.

- put left hand under the nozzle.

- push pump with right hand.

- put left elbow on the table.

- bring chin down to raised left hand containing cream.

- Do 'nodding' action.

- do 'no' action.

Each phase of each activity had to be specified for GP, but once described and practiced she was quickly able to learn the sequence which would enable her to complete tasks. This applied to eating, washing, undressing and dressing (apart from fastening and unfastening her bra which proved to be a very difficult manoeuver), managing the toilet, brushing her hair, cleaning her teeth, showering, putting on deodorant, washing her hair and so on. Planning and practicing these tasks was lengthy and time-consuming, as little carryover was demonstrated from task to task. These self-care tasks were carried out with GP throughout the rehabilitation period and were running parallel to the pilot and intervention studies previously described. During the sensory stimulation intervention periods in both the pilot study and the main ABA intervention study, no notable interaction was seen between the sensory stimulation and the task breakdown, though this was not studied systematically. The treating therapists did not comment on any change in task-breakdown performance during those sensory stimulation periods compared with the other phases or periods of rehabilitation.

Further research to determine whether sensory stimulation intervention procedures influenced task break-down strategies by speeding up the learning process in self-care

activities or facilitating generalisation between tasks would be useful in this field of rehabilitation. The proponents of sensory stimulation procedures in children with developmental dyspraxia (Ayres 1985, Cermak 1985, Croce 1993) have argued that enhancing the sensory-motor integration of the individual tackles the underlying impairment in praxis dysfunction, whereas the task break-down strategy works only on the resulting dysfunction. If tactile stimulation had a significant effect on praxis ability, then independence in self-care and functional tasks should in consequence be achieved more quickly and with generalisation across activities. Further research is suggested to examine this proposition. In addition, an investigation into the effectiveness of task break-down strategies in *ideational* apraxia, with and without sensory stimulation, would also be useful to rehabilitationists.

8.6 Conclusion

This single case investigation provided the evidence for continuing recovery in apraxia over a year post-head injury, and suggested that using a sensory stimulation protocol as a therapeutic intervention in apraxia was no more effective than other therapeutic input. Changes noted over time in both kinematic profiles, timed tasks and ability to selectively produce individual hand movements was shown likely to be a product of a natural recovery process rather than a therapeutic effect. Some evidence of a reduction in variability in performance was shown in the intervention phase of the study, but this also could be attributed to the recovery process rather than the intervention itself. The sensory stimulation procedure *was* shown to have an effect on praxic ability *immediately* after the intervention, though no carry-over was noted between one day and the next. Research to determine whether performance outcome could be enhanced by using different

intervention techniques together, or singly, would be a useful addition to the rehabilitation literature.

This single case provided evidence of variability in a person with ideomotor apraxia across a wide range of measures. More research to verify this phenomenon as a potentially unique feature of the apraxic condition is needed. If it were found to be a particular feature of apraxia, then it might be suggested that a *reduction* in variability, as shown in the intervention phase of this study (reduced ranges, standard deviation and inter-quartile range scores), would be an indicator of improvement. This might be a useful outcome measure in cases of apraxia. Observation data suggested some potential reasons for performance variability. This included emotional factors relating to anniversary of the accident/injury, relationship difficulties, feelings of dependency and loss of dignity in self-care. Physical factors relating to minor ailments, tiredness and the weather also seemed to have performance effects in this particular case. If further investigations supported this finding and indicated a wide range of performance variability, then it could be argued that in rehabilitation settings a ***single*** assessment of the apraxic person's performance would not be an adequate or reliable indicator of general performance level. Multiple assessments would be required to give a more accurate picture of the person's ***range*** of performance.

A dissociation between the clinical tests for apraxia and the kinematics of movement was found, supporting published research and previous data from this current study. The Hermsdorfer et al (1996) suggestion that strategies for coping with apraxia could produce the different kinematic profiles was also supported in this study with GP being 'successful' in the spatial elements of the kinematics but less so in the temporal elements.

Exploration of a task break-down strategy in self-care activities provided *prime face* evidence of effectiveness. However, no generalisation between tasks and activities was noted. Further research could be suggested to determine whether sensory stimulation intervention would enhance task performance in cases of apraxia, especially given the immediate effects of the intervention shown by this study. If sensory stimulation were applied and *then* self-care activities performed immediately afterwards, perhaps an enhanced performance might be an outcome. In addition, evidence as to whether this technique was effective in cases of *ideational* apraxia would be a useful addition to this field of developing knowledge.

1

[1] Public output from chapter data :

Butler, J.A (in press) The reasoning behind assessment and treatment of apraxia. In Unsworth C. (Ed.) Cognitive and perceptual dysfunction: A clinical reasoning approach to assessment and treatment. Philadelphia : F.A. Davis & Co.

Butler, J.A. (1997). Variability of motor performance in apraxia. SOBROX meeting, Oxford . April 1997. (Invited speaker)

Butler, J.A. (1997). Intervention in a case of ideomotor apraxia : evidence of efficacy. National Association of Neurological Occupational Therapists meeting. Feb 1997. (Invited speaker).

Butler J.A. (1996) Evaluation of Intervention in a case of Ideomotor Apraxia. Eastern Motor Group meeting. Addenbrookes Hospital, Cambridge. March 1996. (Invited Speaker)

Butler J.A. (1996) Does Sensory Input Influence Recovery in Ideomotor Apraxia? Brain Research Association Abstrs. 13, pp 66. (ISSN 1354-8301)

Butler J.A. (1996) Intervention in a Case of Ideomotor Apraxia. Proc. British Psychological Society. London Conference. 4 (1). pp 55.

Chapter 9. CASE 4: *DC. Left Cerebro Vascular Accident. Ideomotor apraxia.*

9.1 Case background.

DC, a 48 year old right handed man with a left Middle Cerebral Artery infarction which resulted in mild right hemiparesis, had severe expressive aphasia with a sole utterance of "twenty two". This vocal output was well modulated, however, with good prosody and facial expression. Severe verbal comprehension difficulties were identified by the speech and language therapist but DC demonstrated good ability in understanding *non-verbal* instructions and gestural communications. His social skills remained high despite his impaired communication abilities. DC had a severely arthritic hip which caused him considerable pain at times. This curtailed some of his rehabilitation sessions in both physiotherapy and occupational therapy.

The first assessment for the presence of apraxia was carried out shortly after DC's arrival at the rehabilitation centre, approximately three and a half months post-stroke. He was unable to complete any of the verbal command items in Test 1 for gestures (Appendix D2) though was able to produce some accurate performances in the 'copy' condition (Table 9.1). Errors made included perseveration of gesture and using the hand in the wrong plane of movement. During the object use test (Test 2. Appendix D3) DC was able to attempt the verbal command items and produced incorrect but recognisable pantomimes. Again the main errors were perseverative in nature with some timing errors and incorrect hand postures. No errors were produced during the 'real object use' condition of the test. The DeRenzi et al test (Test 5. Appendix D1)

elicited particular difficulties for DC in copying complex static finger and hand gestures (Table 9.1). Errors were made in the orientation of the hand and inaccuracy of finger positions. Complex sequencing was also impossible for DC to perform correctly, and other movement errors included incorrect plane and timing of movements. He continually vocalised at each gesture but was able to correctly produce some of the simple movements at the first presentation. Indeed, there were many vocalisations of the "twenty two" utterance throughout the testing procedure. No evidence for ideational apraxia was elicited nor for visual agnosia. The accumulation of evidence at this first assessment suggested the presence of an ideomotor apraxia in DC with no accompanying ideational component.

Table 9.1 DC - Clinical assessment scores (raw and %) at first examination.

Clinical Tests	Left Hand
Test 1- gesture copying (max. = 40) (Appendix D2)	14 *(35%)*
Test 2 - object use test (max. = 36) (Appendix D3)	23 *(63.8%)*
Test 5 - DeRenzi et al test, gesture copying (max. = 72) (Appendix D1)	42 *(58.33%)*
Mean % scores for Tests 1, 2 & 5.	56.1%
Test 3 - ideational apraxia (max. = 12) (Appendix D4)	12 *(100%)*
Test 4 - agnosia (max. = 20) (Appendix E)	19 *(95%)*

Informed consent procedures were carried out with modifications made to allow for DC's language impairment. Illustrations and demonstrations were made to inform DC of the research project procedures and demands. DC indicated his consent with nods and facial expressions accompanied by 'thumbs up' signs. Confirmation was gained from the rehabilitation consultant and professionals involved in DC's treatment that it was appropriate to enroll him in the study.

9.2 Aims of the study

Using this single case of mild ideomotor apraxia following cerebro-vascular accident (CVA), this study aimed to :

- replicate the previous intervention study to evaluate the effectiveness of a sensory stimulation protocol on motor performance.
- determine the variability of motor performance in a milder manifestation of the apraxic condition than seen in Case 3 (GP).
- evaluate changes in kinematic data across experimental phases.

9.3 Procedure

Using an ABA design, a baseline period of four weeks was planned to establish the degree of variability of performance produced by DC. This baseline period involved therapy sessions of self-care training, domestic skills, computer training and social activities. These therapy sessions ran parallel to the other rehabilitation activities of

speech and language therapy, and physiotherapy sessions which concentrated on balance and walking activities.

An intervention period of six weeks followed the baseline phase during which DC participated in the sensory stimulation protocol previously described (Chapter 8). These sensory stimulation sessions in occupational therapy, during the intervention period, were interspersed with other sessions involving self-care activities, domestic activities and home visit preparation. The physiotherapy involvement with the project was withdrawn at the start of the intervention phase of the investigation. This was due to lack of staff time to devote the individual attention required; other patients and priorities had to take precedence. A post-intervention period of four weeks was planned but this was curtailed due to DC's admission into hospital for a hip replacement operation.

9.3 i] Outcome measures

The outcome measures devised for this investigation in consultation with the treating therapists were chosen to be manageable at the start and end of each therapy session. They needed to be quick, simple and reliable. The Nine Hole Peg Test (NHPT) (Mathiowetz et al, 1985) was chosen as a standardised test to measure motor performance. Also a paper cutting task was devised where DC was asked to cut around a star shape as quickly and accurately as possible. A motor task devised for the GP pilot case study was also used. Here DC was requested to place a mark with a

pen in each of a series of ten circles on a page returning to a 'base circle' at the nearest edge of the paper each time ('circles task'). All three tasks used the time taken to complete the activity as an outcome measure. A standard calibrated stopwatch was used for this, with the countdown protocol used to remove any researcher reaction-time variable from the data (see Section 4.5). All tasks used the left non-hemiparetic (non-dominant) upper limb for the testing process. Physiotherapy measures chosen for this investigation were: time taken to complete a paper folding task and a sequence of eight specified hand movements judged for both accuracy and completion time. These measures were not ultimately employed in the data analysis as only the baseline measurements were available following withdrawal of the physiotherapy involvement with the project.

Therapy sessions and data collection were intended to be at the same time daily in order to keep one potential variable constant, but this proved to be beyond the management of the timetable requirements, therapists' constraints, other rehabilitation sessions and differing priorities. Whilst time of day might not be seen to matter, and from the previous cases in this study this would appear to be so, it is a potential source of influence on outcomes in that the patient may be more tired at the end of the day for example, or less able to concentrate immediately following lunch etc. The therapy sessions were, therefore, spread as evenly as possible about the whole range of timetabled sessions both morning and afternoon in both baseline and intervention phases in order to reduce any timing or fatigue effect. Seven completed data points

for both pre and post therapy sessions were collected from the baseline phase (physiotherapy measures being discounted) and seventeen data collection points were obtained during the six week period of the intervention phase. The apraxia assessments used in this single case investigation consisted of the standard selection described in previous chapters (Appendices D1-D4). Data available in the post-intervention period were from the circles task, the apraxia tests and the kinematic recordings only.

9.3 ii] Kinematic recordings

During this investigation, kinematic recordings were made twice weekly (where possible) of DC performing the drinking task in each of the modality conditions. Recordings were made at similar times on each occasion (1.30pm after lunch-time rest) and in the same environment. Gestural performances of HAMMER, CARVE and WIND, described in Case 2, were also recorded (using the 'copy' condition only due to DC's language difficulties) to monitor motor performance changes over time. Nine recordings were made in the baseline phase, seven in the intervention phase, and one was possible in the 'post-intervention' period before DC had his hip replacement. All recordings used the limb ipsilateral to the lesion (left, non-hemiparetic, non-dominant upper limb) to eliminate any hemiparetic variables on motor performance.

9.4 Results

9.4 i] Outcome measures

Descriptive analysis of the data was completed for the baseline and intervention phases. No correlation in scores was found between the paper cutting, NHPT and 'circles' motor tasks in either baseline phase [circles & NHPT r= .027 *NS.*, circles & paper cutting r=.1 *NS*, NHPT & paper cutting r= .47 *NS*] nor intervention phases [circles & NHPT r= -.22 *NS.*, circles & paper cutting r=-.01 *NS*, NHPT & paper cutting r= -.09 *NS*]. It was considered, therefore, that each of the tasks was measuring independent attributes of motor performance. The range of scores was wide in the baseline phase, though this was reduced in the intervention phase. The standard deviation and interquartile ranges (Table 9.2) were also reduced in the intervention phase and could be interpreted as evidence for a greater consistency in performance. An overall reduction in time taken to complete each of the tasks in the intervention phase, indicated by the broadened median scores and smaller standard deviations (Table 9.2), might also be considered an indication of improvement in DC's motor performance. Plotting of the data across phases would help with interpretation and determine whether these were true effects or, as with GP, more a function of a natural recovery curve.

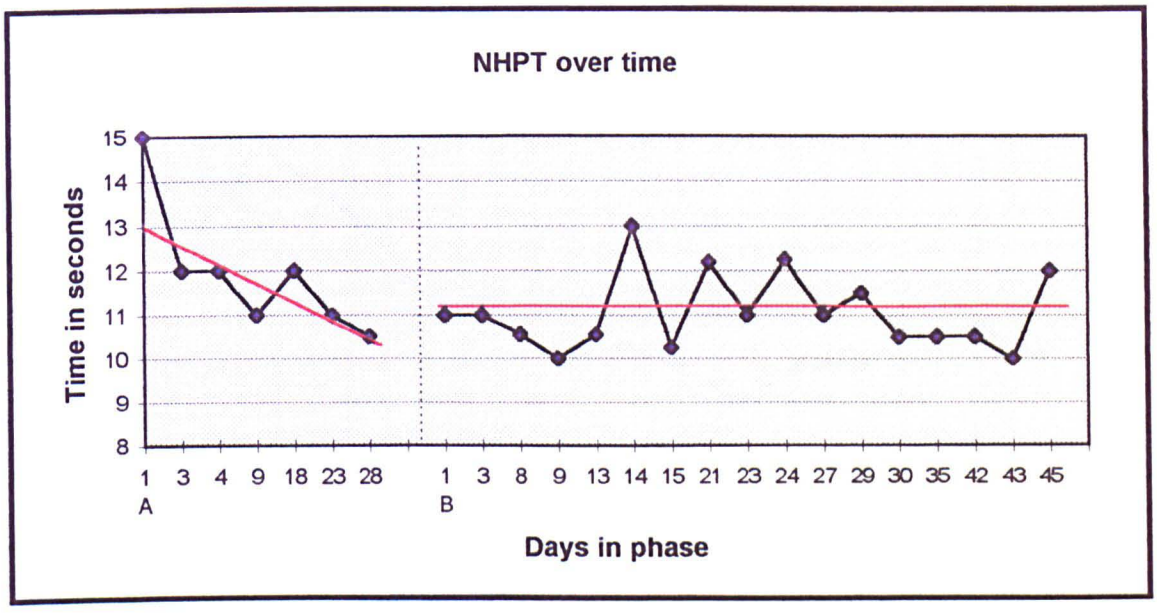
Table 9.2 DC - Synopsis of outcome measures data. Baseline and intervention phases.

(All scores given in seconds.)

	Baseline	Intervention
NHPT *(norm = 18 secs.)* Left hand.		
Mean score	11.93	11.05
Range	10.5 - 15.0	10.0 - 13.0
Broadened median	**11.55**	**10.91**
Standard deviation	**1.48**	**0.86**
Interquartile range	**1.00**	**1.25**
No. of data points	7	17
Paper cutting task Left Hand		
Mean score	120.29	90.91
Range	83.0 - 170.0	74.0 - 133.0
Broadened median	**115.17**	**89.4**
Standard deviation	**33.9**	**13.67**
Interquartile range	**56**	**14.5**
No. of data points	7	17
Circles task. Left hand.		
Mean score	18.41	15.65
Range	16.5 - 19.83	14.84 - 16.32
Broadened median	**18.03**	**15.17**
Standard deviation	**1.11**	**0.42**
Interquartile range	**1.52**	**0.38**
No. of data points	7	9

Visual inspection of the data across baseline phases indicated a strong trend of improving performance over time in the baseline phase (Figures 9.1, 9.2 and 9.3). The graphs also clearly show that by the time of the intervention phase, this improvement had leveled out.

Figure 9.1 DC - NHPT over time



The regression lines were quite good predictors during the baseline phase for each of the tasks (NHPT 46% of variance, circles 48.7%, paper-cutting 58%) confirming the trend seen in the graphs. These improvements could be taken as indicators of *learning* by DC rather than recovery processes as no continuation of improvement was seen in the intervention phase which might otherwise have been expected. The intervention phase showed a different pattern in the data with regression lines not predictive for NHPT (r= .00006) and highlighting the variability of performance on that task, seen in Figure 9.1.

Figure 9.2 DC -'Circles' task over time

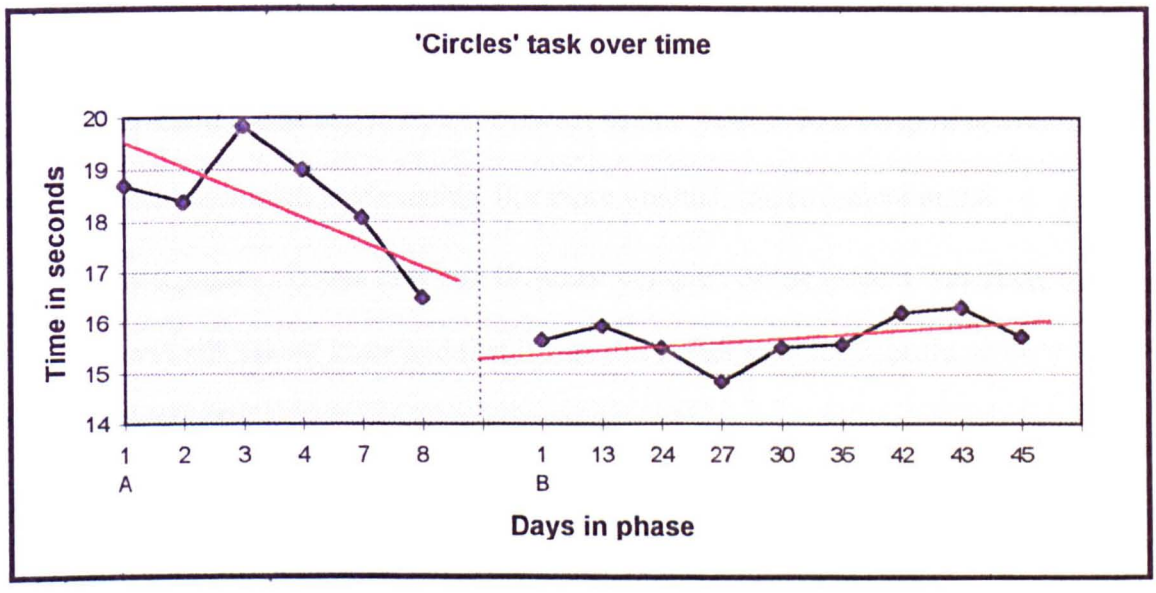
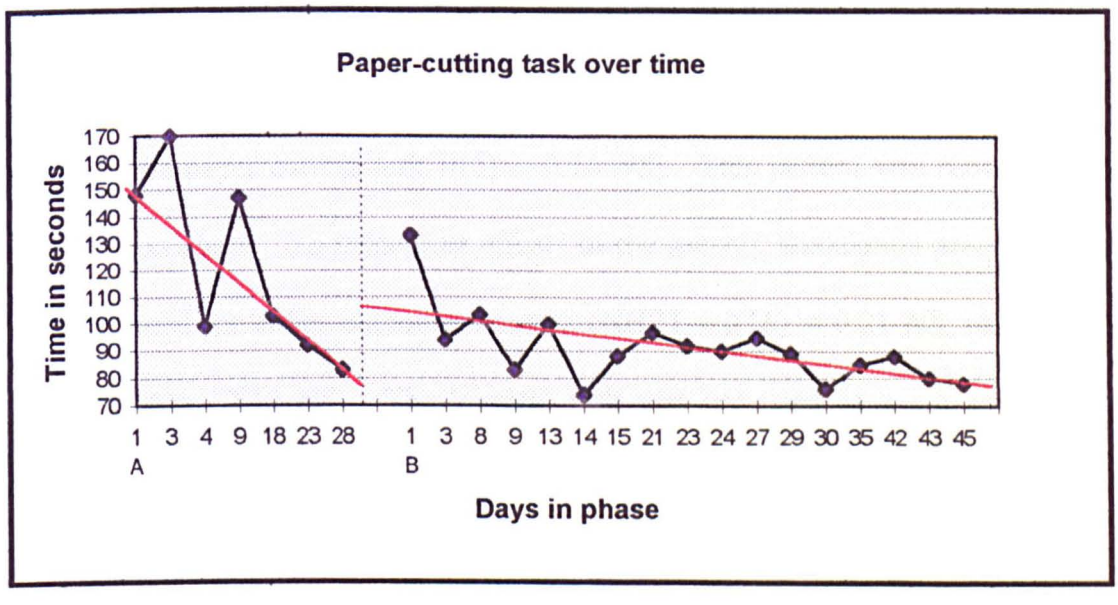


Figure 9.3 DC -Paper-cutting task over time



The 'circles' task also showed improved performance in the baseline phase with the regression line accounting for 48.7% of the variance, whilst in the intervention phase the variability of performance left the regression line only accounting for 0.17% of the

variance. This confirmed the visual analysis of the plotted data where the strong trend seen in the baseline phase was replaced by an uneven performance in the intervention phase.

The paper-cutting task followed a somewhat similar pattern of a steep improvement in the baseline phase with a continuing, but more gradual, improvement in the intervention phase. As this task was the most complex of the three it was likely that learning was still taking place and that it was this rather than the specific sensory intervention which could account for the change. The regression line for the baseline phase accounted for 59% of the variance but only 32.6% in the intervention phase, supporting the visual analysis of a less dramatic learning curve in the latter phase allowing the feature of variability in performance to show.

Analysis of the *immediate* effects of therapy during the baseline phase (using paired t-test statistics) indicated no significant differences for either the NHPT [$t(6) = 0.74$ *NS*] nor the paper cutting task [$t(6) = 0.16$ *NS*]. This pattern was repeated in the intervention phase. No *immediate* effects of the sensory stimulation procedure were demonstrated for either outcome measure (NHPT - $t(16) = 0.21$ *NS*, paper cutting task - $t(16) = 0.40$ *NS*). The 'circles' task was not used as a pre and post-test measure. So unlike the previous case (GP) sensory stimulation did not appear to have either an immediate effect following intervention nor be convincingly producing any effect over the natural learning process of the experimental tasks.

9.4 ii] Apraxia assessment scores

Apraxia assessments were completed every four weeks during the investigation although, after the initial screening process, only two of the tests were used to avoid fatigue in DC. Apraxia tests were carried out at the initial assessment of this patient before the research protocol was begun, at the start of the baseline phase, the start (time 1) and midway point (time 2: - beginning of week 4 of the 6 week intervention) of the intervention phase and on one occasion at the start of the post-intervention period before it was curtailed. The changes in scoring over time (Table 9.3)

Table 9.3 DC - Apraxia assessment scores (raw and %) in ABA phases.

	Initial assessment	Baseline 'A'	Intervention 'B'		Post-intervention
			Time 1	Time 2	
Test 1- gesture copying (max. = 40) (app. D2)	14 *(35%)*	18 *(45%)*	18 *(45%)*	26 *(65%)*	26 *(65%)*
Test 2 - object use test (max. = 36) (app. D3)	23 *(63.8%)*				
Test 3 - ideational apraxia (max. = 12) (app. D4)	12 *(100%)*				
Test 5 - DeRenzi test, gesture copying (max.= 72) (appendix D1)	42 *(58.33%)*	62 *(86.1%)*	65 *(90.3%)*	54 *(75%)*	63 *(87.5%)*

demonstrated DC's increasing praxic ability although the notably poorer score in the De Renzi et al test, at Time 2 in the intervention phase, was the exception. At the time of this particular examination it was noted that DC was experiencing some considerable pain from his hip, though he indicated he still wanted to participate in the

assessment. During the baseline assessment DC was unable to complete any of the items in the verbal command condition of Test 1, and in the copy condition demonstrated perseverative actions during the gesture performance. The De Renzi et al test performance was improved at the baseline examination when compared to the initial assessment. Considerable difficulty was still demonstrated by DC, though, in copying the static hand positions and in the sequencing of movement. These scores remained fairly constant, with similar patterns of performance shown at Time 1 of the intervention phase (Table 9.3). At Time 2 of the intervention phase, however, DC was able to attempt the verbal command condition of Test 1 (Appendix D2) and was successful in three of the five items. This improvement in the test score was attributed to improved language comprehension rather than praxic function. Performance in the De Renzi et al test was poorer at Time 2 in the intervention phase. It is possible that this might have been attributable to the hip pain DC experienced. Errors made during this assessment included wrong plane of movement, incorrect orientation of the hand positions, perseveration errors and inaccurate sequencing of movement. The static hand positions continued to provide the most challenging aspects of the assessment. DC was unable to recognise the errors he had made. After the intervention phase was completed (in the 'post-intervention' period before DC's hip operation) the apraxia assessment indicated fewer errors in the De Renzi et al test. This improvement related to the movement sequence items which

were correctly copied at the first or second presentation. DC continued to show difficulties in copying static hand postures with errors and inaccuracies evident.

During the intervention phase of the project, further investigation was made of DC's inability to identify errors in hand positioning. It was hypothesised that DC might have a finger agnosia which would include loss of ability to differentiate, recognise, identify, name, select and orientate the individual fingers of either hand. This would apply to his own fingers as well as those of others. The Rivermead Perceptual Assessment Battery was completed with DC who achieved a full score on all items and demonstrated no loss of perceptual function or disorder of body image. The De Renzi et al test was carried out using the right hand, with similar difficulties and errors shown in the completion of static hand postures. DC was unable to see where his errors lay and could not distinguish between the 'model' presented by the researcher and his own attempt.

Pictures of a right and left hand were placed on the table in front of DC. Four tasks were completed. DC was asked to :

1. identify (on the picture) the corresponding finger to that being held up by the researcher.
2. hold up the corresponding digit to that being pointed to in the picture by the researcher.
3. repeat task 1 but with two or three digits being held up for identification.
4. repeat task 2 but with multiple rather than single digits.

These tasks were devised to elicit DC's abilities in identification, differentiation and selection of fingers without the use of language. All four tasks were completed without difficulty. It was considered, therefore, that DC's difficulties were attributable to his ideomotor apraxia rather than to any finger agnosia.

9.4 iii] Kinematic recording

Descriptive analysis of the drinking task recordings suggested small improvements in timing in the intervention phase of the study when compared to the baseline phase in each of the three conditions (Table 9.4) shown by the reduced mean and broadened median scores. The variability of performance could be said to have been reduced in the intervention phase indicated by the smaller standard deviation and interquartile range scores (Table 9.4) though only plotting of the data would reveal whether this was a reasonable conclusion.

The total time taken to complete the drinking task was plotted for the three experimental conditions across ABA phases, although only one data point was gathered for the post-intervention phase. Visual analysis revealed that, as with the other outcome measures used for DC, there was a large effect of learning in the baseline phase as shown by the steep slope in the regression lines. The intervention phase data then either evened-out or the variability increased once the learning effect had been completed (Figures 9.4, 9.5 and 9.6).

Table 9.4 DC - Synopsis of kinematic recording data in ABA phases.

(All scores given in seconds)

Drinking Task.	Baseline	Intervention	Post-intervention
Total Time Verbal condition			
Mean score	6.64	6.47	6.44
Range	5.22 - 7.92	5.5 - 7.2	
Broadened median	**6.75**	**6.49**	
Standard deviation	0.86	0.68	
Interquartile range	1.4	1.42	
No. of data points	9	7	1
Total Time Copy condition			
Mean score	6.69	6.43	6.26
Range	5.82 - 7.64	5.82 - 7.06	
Broadened median	**6.61**	**6.42**	
Standard deviation	0.77	0.41	
Interquartile range	1.59	0.58	
No. of data points	9	7	1
Total Time Real object condition			
Mean score	7.22	7.05	6.96
Range	6.44 - 7.84	6.4 - 7.58	
Broadened median	**7.28**	**7.00**	
Standard deviation	0.52	0.42	
Interquartile range	0.96	0.76	
No. of data points	9	7	1

Figure 9.4 Drinking task, verbal condition. Total time across ABA phases.

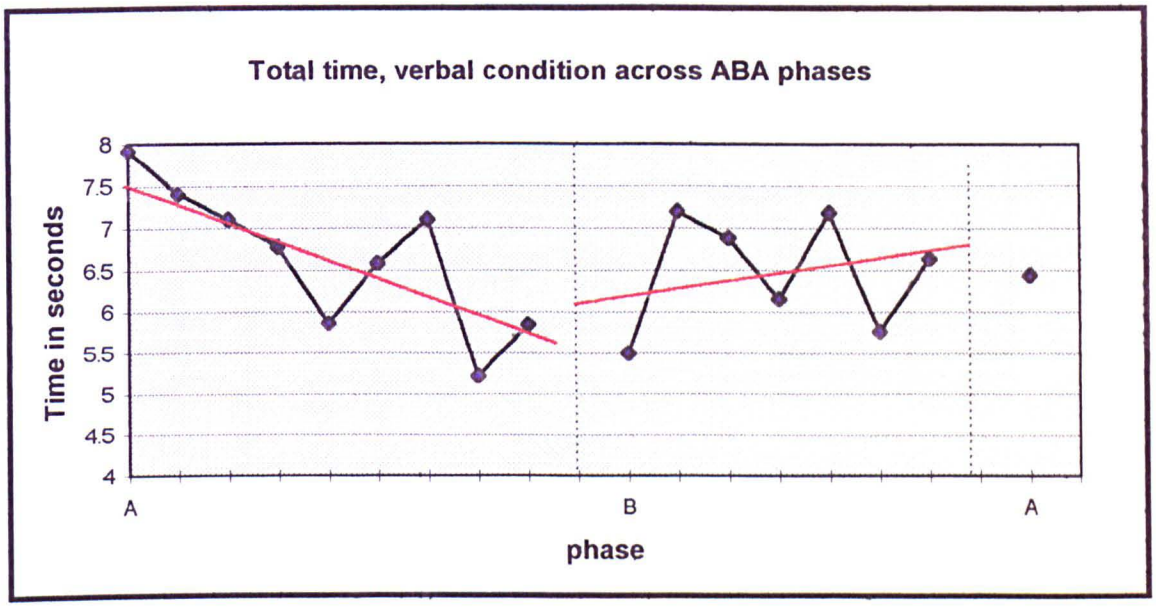


Figure 9.5 Drinking task, copy condition. Total time across ABA phases.

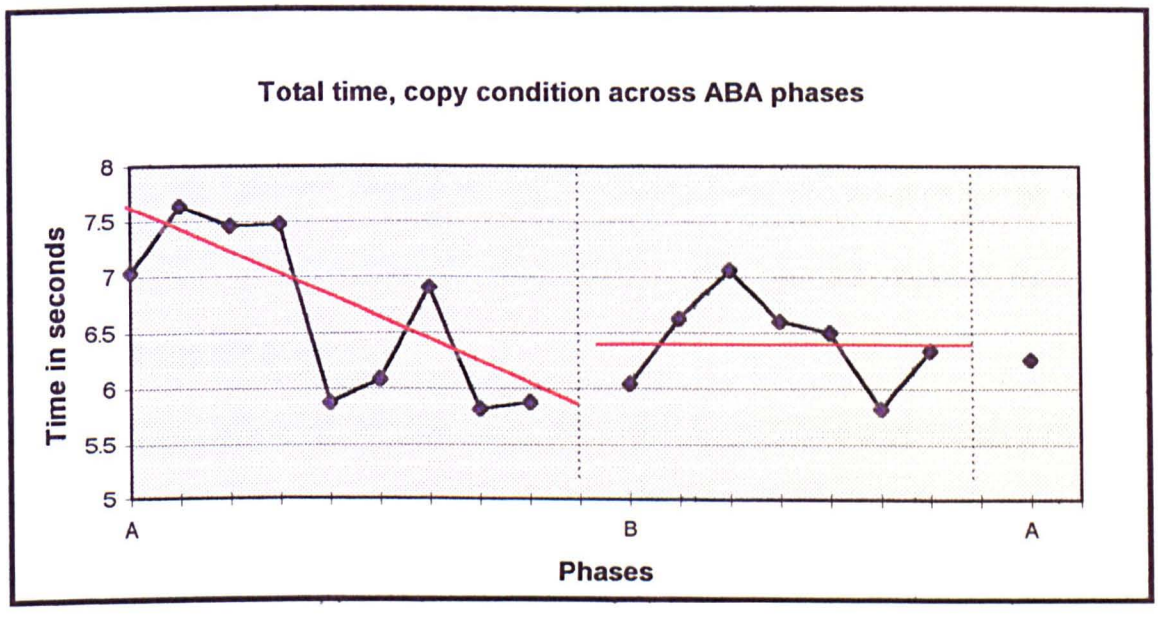
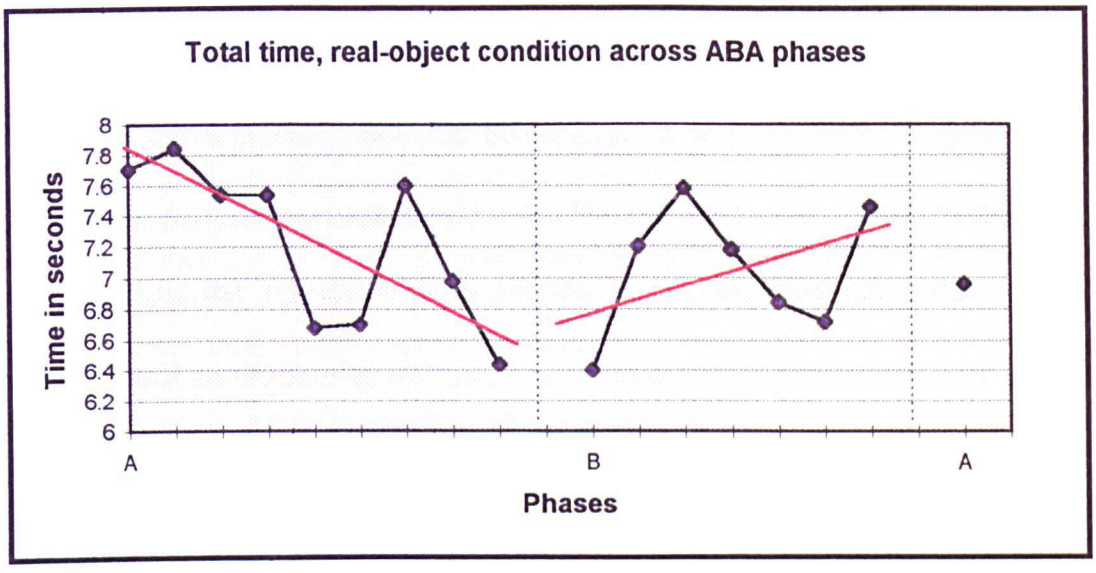


Figure 9.6 Drinking task, real-object condition. Total time across ABA phases.



The variability in performance, shown by the plotted data, was actually more notable in the intervention phase of the study with the regression lines only weakly predicting any trend in the data [verbal condition r= .06 $F(1,5) = 0.32$ *NS*, copy condition r=.0015 $F(1,5) = 0.0076$ *NS*, real-object condition r=.177 $F(1,5) = 1.08$ *NS*]. The baseline regression lines showed a statistically significant prediction of trend [verbal condition r= .61 $F(1,7) = 11.1$ $p < .05$, copy condition r=.54 $F(1,7) = 8.25$ $p < .05$, real-object condition r=.51 $F(1,7) = 7.23$ $p < .05$]. This was not apparent from the descriptive data on its own.

An alternative analysis of the data, however, might suggest that once learning effects had ceased then DC's performance range was the same for both baseline and intervention phases. If, for example, in the verbal condition (Figure 9.4) the first four data points were taken from the analysis then the subsequent performance followed much the same variable pattern as could be seen in the intervention phase recordings. This was not so clear in the copy condition of the drinking task where no real learning

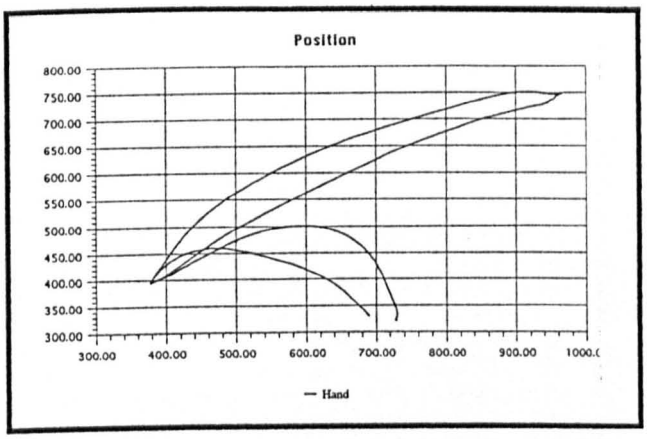
effect could be seen in the first four trials, though thereafter the variable pattern could be interpreted as similar to that seen in the intervention phase. In the real-object condition the first four trials *did* demonstrate a learning effect (Figure 9.6) and subsequent performance, again, was much like that seen in the following study phase. So, perhaps, the regression lines plotted in the experimental phases could be considered to be misleading.

The relationship between the temporal component of the kinematic data and the clinical tests was investigated. As the number of data points in each phase of the study were small for the clinical tests, all experimental phases were used as a single data set. Significant relationships were found between the scores achieved by DC on the De Renzi et al test for apraxia and on the total time taken in the drinking task in each condition [Verbal condition $r(5) = -.87$ $p = .054$ Copy condition $r(5) = -.97$ $p < .01$ Real object condition $r(5) = -.92$ $p < .05$]. The negative correlations indicated that the improvements in apraxia test scores were associated with a faster time taken to complete the drinking task. This relationship was not replicated in the other verbal command and gesture copying test for apraxia (Haaland & Flaherty 1982. Appendix D2) with $r = -.35$, $-.41$ and $.003$ for the verbal, copy and real-object conditions respectively. Unlike the data from previous cases, in this study DC did *not* show a dissociation between the clinical tests and the completion time in the drinking task. Regression analysis indicated that the De Renzi test accounted for 76% of the variance in the verbal condition of the drinking task [$F(1,3) = 9.52$ *NS*], 93.2% in the copy condition [$F(1,3) = 41.43$ $p < .01$], and 84.2% [$F(1,3) = 15.97$ $p < .05$] in the real-object condition.

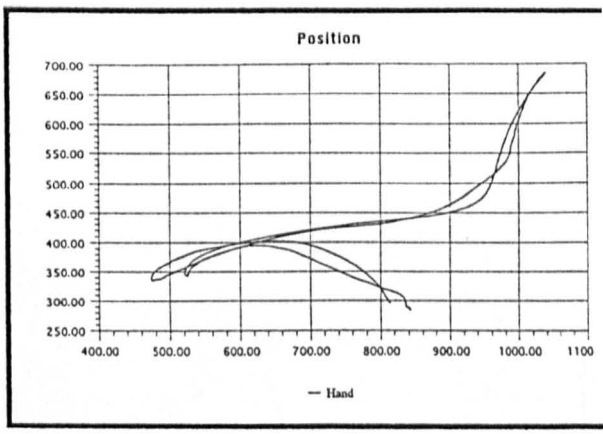
Analysis of the hand trajectories of the drinking task indicated some loss of spatial consistency in the early baseline phase recordings in the copy condition (Figure 9.1). This performance of the task rapidly became rather stylised, with embellished movements, and remained the dominant pattern of performance throughout the baseline phase (Figure 9.7). This pattern of performance was not noticeably changed during the early stages of the intervention phase, although later on more correct gestures were performed (Figure 9.8).

Figure 9.7 DC - Hand trajectory recordings in the drinking task.. Baseline phase.

a] First recording - Copy condition



b] Recording at Time 3 (copy).



c] Recording at Time 9 (copy).

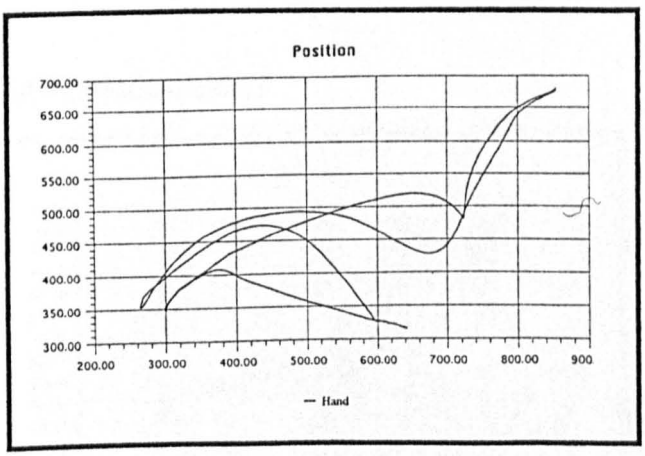
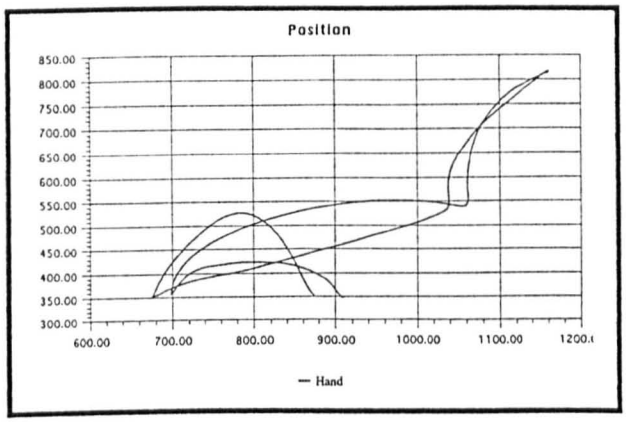


Figure 9.8 DC - examples of hand trajectory recordings. Intervention phase.

a] Early recording



b] Later recording

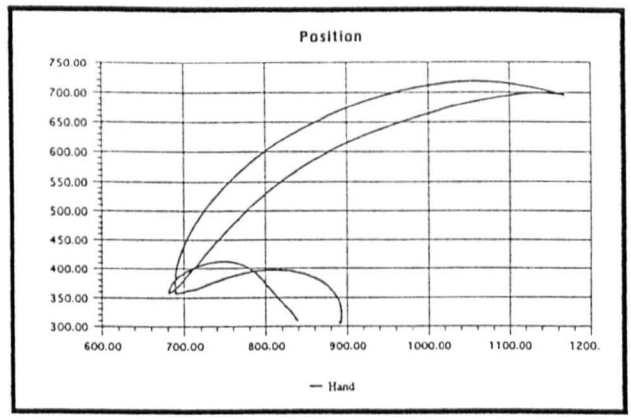
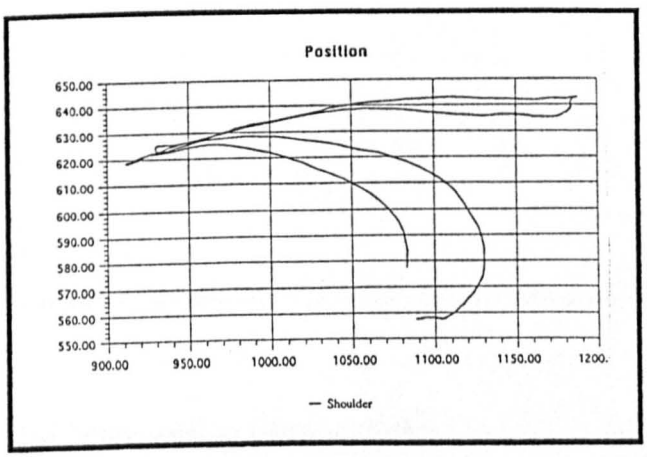
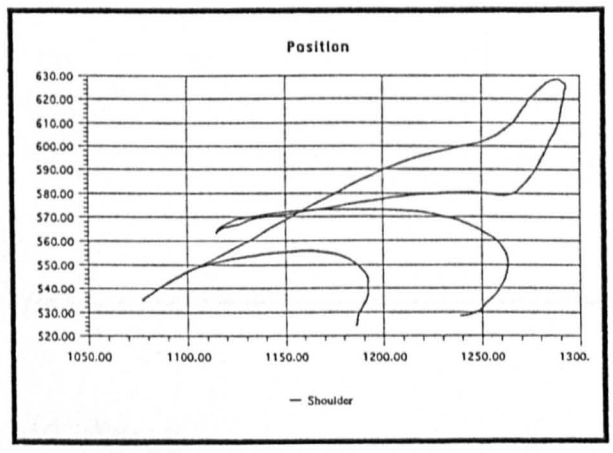


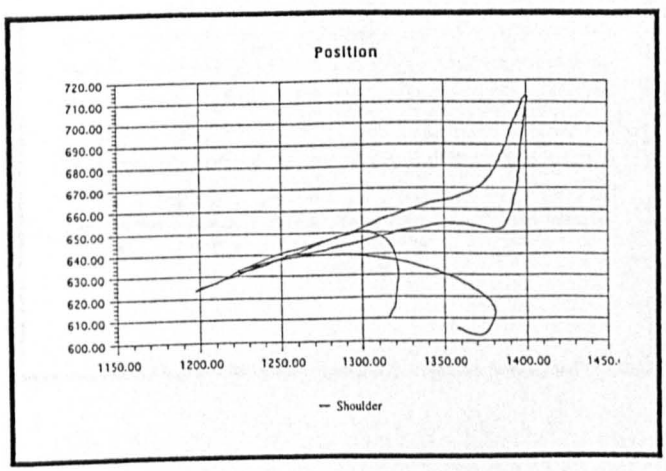
Figure 9.9 DC - Examples of shoulder trajectory. Baseline and Intervention phases.

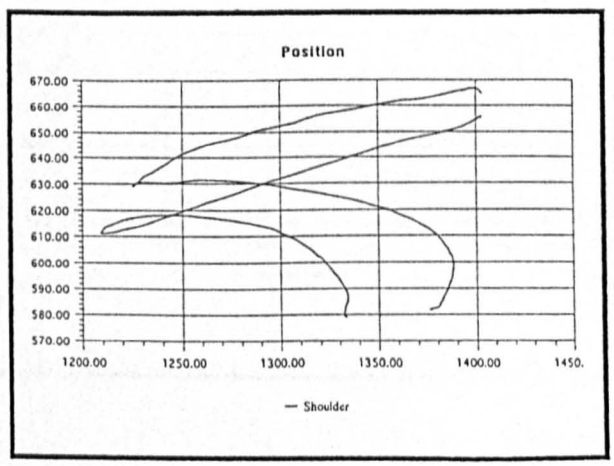
a] baseline phase.





b] Intervention phase



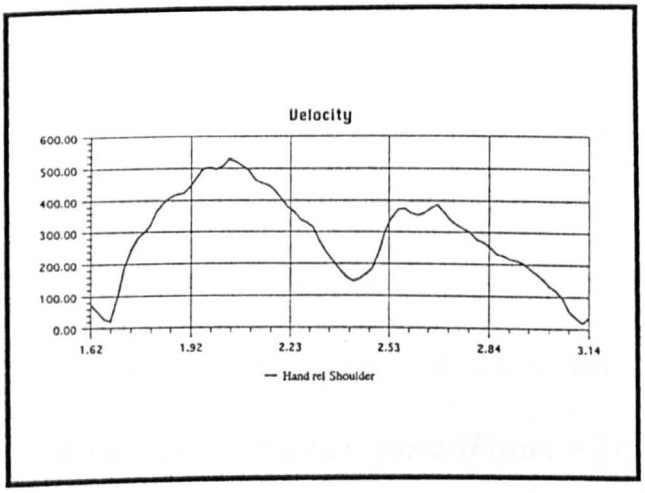


Shoulder movement was variable across performances during both the baseline and intervention phases (Figure 9.9) but was seen to have some mild exaggeration and over-use of the joint. These patterns were consistent with those associated with apraxia in the group study.

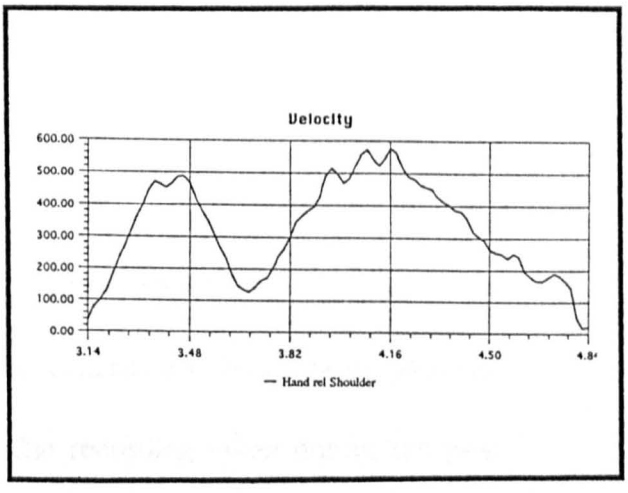
Velocity curves retained a fairly smooth rather than a multi-peaked profile, but the

Figure 9.10 DC - Examples of velocity curves. Baseline and Intervention phases.

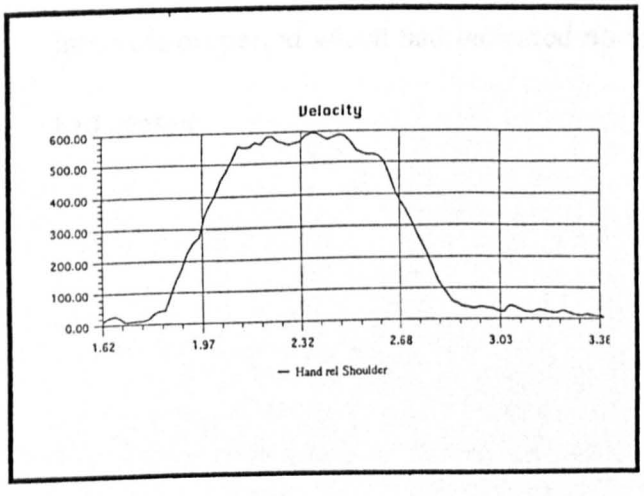
ai] Baseline phase - 'Lift'.



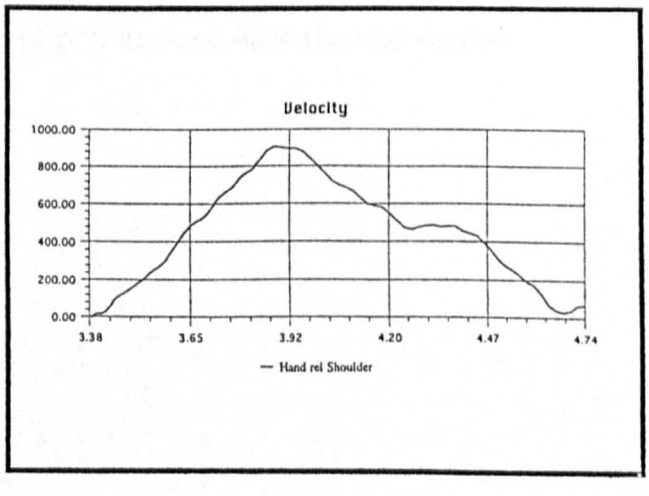
aii] 'Down'.



bi] Intervention phase - 'Lift'.



bii] 'Down'.

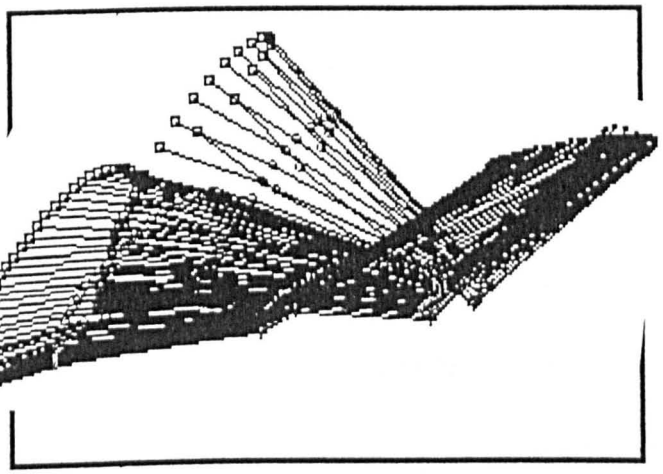


stylised performance caused a dual-peak appearance in the 'lift' and 'down' sections of the task (Figure 9.10). This pattern was modified by the end of the intervention phase towards a more normal bell-shaped curve associated with the normal controls in the group study.

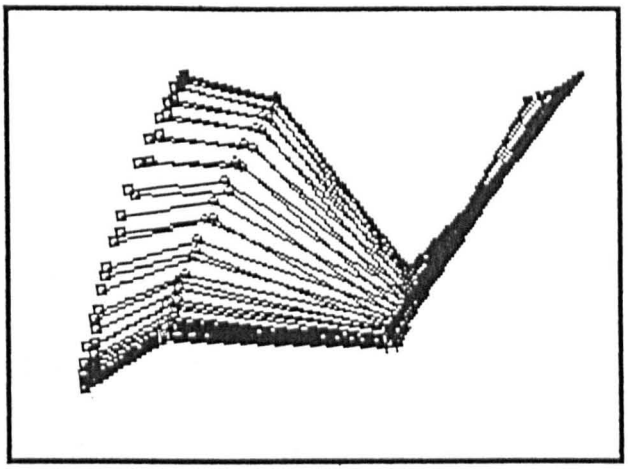
Analysis of the gesture recordings revealed some inconsistent use of spatial pathways to complete the repetitive movements. This applied to each of the three gestures (HAMMER, CARVE and WIND) with an over-use of the shoulder and elbow seen in all recordings in the baseline phase (Figure 9.11). DC demonstrated a 'searching' strategy in performance of the gestures with inaccurate movements at the start of the cycle with an increasing control and accuracy as the performance progressed. During the intervention phase of the project this 'searching' was eliminated, with good performance and control elicited from the start of each movement. The recordings demonstrated reduced use of the shoulder and consistently held spatial pathways during the intervention phase (Figure 9.11). The recording taken during the post-intervention period indicated that DC retained this movement control. This evidence added support to the De Renzi et al apraxia test score obtained in the post-intervention period which had indicated no loss of performance once the intervention had ceased.

Figure 9.11 DC - Examples of gestural recordings, baseline and intervention phases.

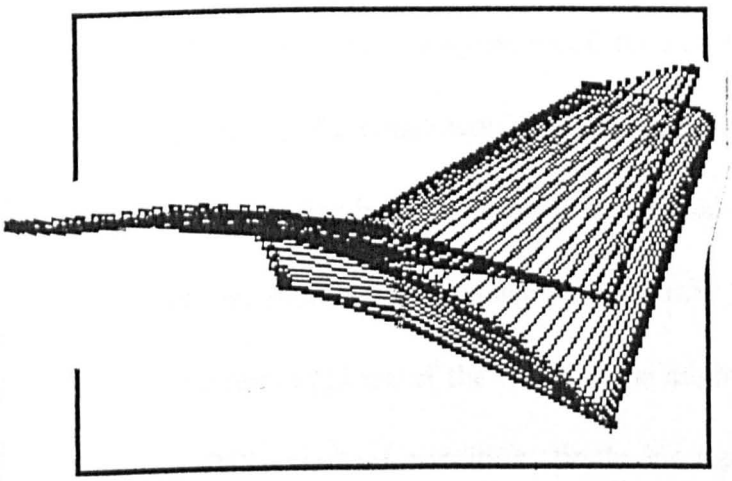
ai] Baseline - HAMMER



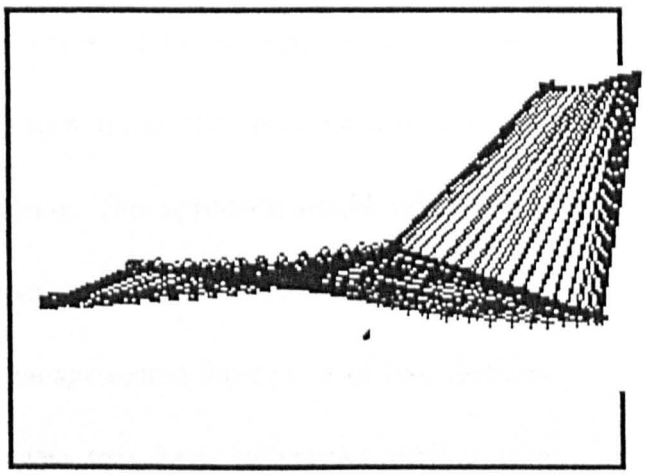
bi] Intervention - HAMMER



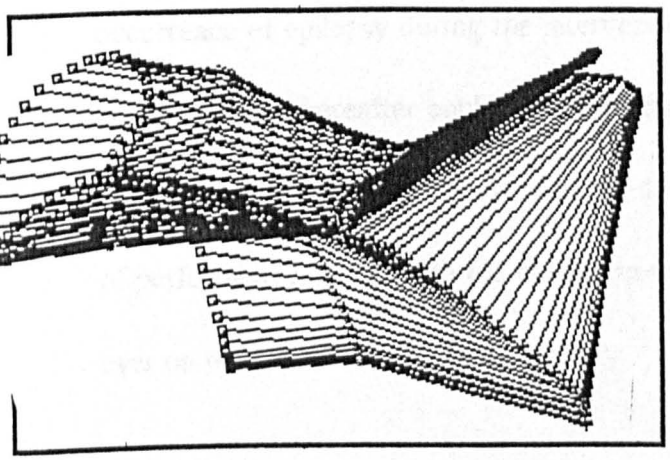
aii] Baseline - CARVE



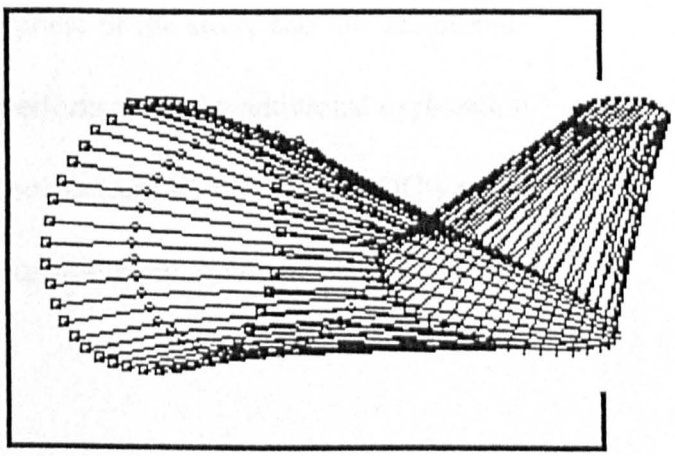
bii] Intervention - CARVE



aiii] Baseline - WIND



biii] Intervention - WIND



9.5 Discussion

This study aimed to evaluate intervention effectiveness of a sensory stimulation procedure. The outcome measures used in this single case were limited due to the withdrawal of physiotherapy involvement in the project. This, together with the limited data collection points, reduced the robustness of the conclusions which might be made. Those measures that were used with DC were devised to challenge his motor skills and be capable of monitoring change. It could be argued that DC was performing at ceiling level for the NHPT and so it was surprising that a learning effect could be interpreted from the baseline phase of the study. Examination of the plotted data, though, might suggest that if the first trial was eliminated from the baseline data then the performance would be much as that seen in the intervention phase, with the regression line being in a similar horizontal plane. This approach would indicate that the variance in performance was actually increased, rather than diminished, in the intervention phase of the study. This might be accounted for by one of two features in DC's *medical* condition: firstly his hip pain may have influenced performance according to its severity on particular days of measurement, or alternatively the occurrence of epilepsy during the intervention phase of the study and the use of anti-epilepsy drugs thereafter could have affected performance. An additional explanation could suggest that after the first trial, the data points were all indicative of DC's *range* of performance levels, with the change in therapeutic input having no effect what-so-ever on the outcome measures.

The 'circles' and the paper cutting tasks demonstrated a clear trend of improvement during the baseline phase with a general leveling off of performance by the time the intervention phase was established. In these two tasks the variability *was* seen to be less in the intervention phase which supported the evidence of the previous case (GP), though whether such reduction could be attributed to the sensory stimulation procedure rather than to a consolidation of a skilled task following learning has not been convincingly shown by this study. These results were supported by the apraxia assessment scores where little change was noted across the different phases of the study, apart from the 2nd assessment in the intervention phase where a reduction in clinical test scores was noted. The presence of severe hip pain during this assessment might have affected DC's concentration and performance ability as the time taken to complete the drinking task on that same day of assessment was greater than on previous occasions. This, again, might be indicative of pain influencing performance. If this conjecture was correct, then explanations for performance variability suggested in section 8.6 might include physical factors of all kinds, pain levels as well as such chemical influences as pain-relief medication.

The kinematic recordings of the drinking task showed minimal improvements in performance ability between the baseline and intervention phases in the time taken to complete the task. Visual analysis of the plotted data suggested that a learning effect had occurred in the baseline phase though the variability of performance day-to-day was great, especially after the first four trials. This variability continued into the

intervention phase of the study with no obvious change or reduction apparent. In conclusion, no convincing effect could be interpreted from the data concerning the sensory stimulation intervention either immediately after the procedure, or over time. The sensory stimulation procedures appeared not to have had any effect on DC's praxic abilities.

The adoption and maintenance of a stylised movement by DC was an unexpected feature of the results. It could be argued that this might be an indication of perseveration, given that no modification of the movement was made despite the correct 'model' being demonstrated on each occasion. The production of the inaccurate, internally-generated movement pattern at the verbal command may have been strengthened by the repeated (perseverative) movement and so would be more likely to be produced at each subsequent assessment. It could be hypothesised that a 'hands-on' physical training of the correct gesture and movement performance might have eliminated this error. This was not attempted in this project, but could indicate the need for such strategies in a rehabilitation programme. Such strategies would prevent errorful movements from becoming established and entrenched.

The kinematic data provided by the gestural performance recordings revealed some change in movement consistency and control during the intervention phase. DC was eventually able to rapidly determine and execute requested movement patterns. Whether such improvements could be considered a consequence of, and attributable

to, the sensory stimulation protocol could be considered unlikely given the evidence from the other outcome measures.

In this single case of mild ideomotor apraxia an association was found between the De Renzi assessment scores and the kinematics of the drinking task as represented by the completion time in each modality condition. Not only did the test correlate highly with the total time taken to complete the task, it also was significantly predictive of the performance in both the copy and the real-object conditions. As this relationship was not seen in the other cases previously described, nor in the group study of this thesis, and published work by Hermsdorfer et al (1996) suggested a *dissociation* between clinical tests and kinematics of movement, an explanation must be sought. It is possible that the hypothesised finger agnosia in DC relating to his inability to recognise performance errors, has some bearing on the case. The person with *ideomotor* apraxia is normally aware of performance deficits, and no *ideational* component was elicited in DC in either formal assessment or in self-care activities. No finger agnosia was identified from the limited examination given (due to DC's language dysfunction) nor body image disturbance nor perceptual dysfunction yet perhaps some vision-to-action processes had been disrupted which could account for the association. Alternatively there was an mild ideational component to DC's condition which might have influenced aspects of the clinical test and the movement kinematics. If such an ideational component were present, then Hermsdorfer's 'strategy' argument would suggest that this would lead to normal temporal kinematics

in movement but disrupted spatial elements which were picked up in the complex static gestures of the De Renzi test. If this were so, then an exploration of the relationship between clinical tests and kinematics of movement with those with a more severe ideational apraxia would be of interest. In addition, this association found with DC could, again, represent further evidence of 'sub-categories' found within the apraxic population. More research to tease out these 'sub-types' would be a useful avenue to explore also.

In terms of rehabilitation, DC's inability to recognise his errors in performance could have been targeted in intervention. A 'hands-on' rehabilitation strategy to physically correct the movement or gesture could be considered important in facilitating awareness and learning in people with such apraxic features and a future case study with an individual with ideational apraxia, using such an intervention, would be useful to complete.

9.6 Conclusion

This case investigation with DC was beset by practical difficulties and was a potent illustration of the problems facing clinical research. Despite the difficulties, the data analysis has indicated some areas worthy of further exploration. The need for quick and reliable outcome measures which are meaningful and pertinent was demonstrated. Whilst the use of non-standardised motor tasks demonstrated some value, the ideal would be to make use of standardised measures for all the reasons associated with

reliability and validity. In addition to the NHPT, it might be considered worthwhile to explore the use of the Gibson Spiral Maze from the CAPE (Clifton Assessment Procedure for the Elderly. Pattie & Gilleard, 1979) in the single case projects as it complies with all the criteria for a simple, quick, standardised measure for motor performance and one which is readily recognised and available within most therapy departments.

The effectiveness of the experimental sensory stimulation procedure was not demonstrated in this case. Motor learning was considered to account for much of the change seen in performance across outcome measures, and thereafter the data could be seen to reflect the range of performance through which DC operated. It is possible that due to all the practical difficulties experienced with this study, the stimulation procedure was not given sufficient opportunity to show its full effectiveness, though evidence from the previous case (GP) would not support such conjecture. Proponents of sensory integration procedures (Ayres 1985, Cermak 1985, Croce 1993) argue, however, for the need for *daily* therapy sessions over an even longer period than the six weeks covered by this case study investigation for the effects to become obvious. Further studies with enhanced control of regular intervention sessions, multiple data points, and a complete ABA methodology should help to tease out these issues.

The kinematic data in this single case of ideomotor apraxia provided objective data of motor performance and details of spatial and movement errors. The experimental task

was consistent with previous data (Hermsdorfer et al 1996) concerning the presence of relatively intact kinematic profiles which can be found in apraxia. In this particular case, however, an association was found between the clinical assessments and the kinematic components of movement which was not found either in the Hermsdorfer study nor in the data presented previously in this thesis. Possible explanations have been suggested relating to the loss of vision-to-action routes in DC (though his score on the agnosia test was high) and to the potential of a mild ideational component to his condition.

This case has provided further evidence of the performance variability in ideomotor apraxia. This became apparent once the effect of learning had ceased across a wide range of measures. Whether such variability is the province of apraxia or might be demonstrated across a variety of neuropsychological conditions remains to be determined. Further investigation of the nature of variability of performance would be a useful rehabilitation guide. In addition, further research concerning the use of physical prompts to guide movement and gesture might be a useful area to explore in terms of rehabilitation effectiveness for apraxia.

Chapter 10. CASE 5:

EW. Hypoglycaemic brain damage. Ideational and ideomotor apraxia.

10.1 Case Background.

EW, a 48 year old left-handed woman with diffuse and widespread hypoglycaemic brain damage following an insulin overdose, was noted as having a Glasgow Coma Scale score of 3 (lowest possible score from 15) on emergency admission to hospital. The deep coma lasted for three days. Four weeks later, EW was recorded in the medical notes as having started to walk, had recognised people and had begun to talk. An MRI scan was taken eight months post-incident. The radiographic report described dilated ventricles and prominent cerebral sulci which indicated generalised atrophy of the cerebrum. Some changes were reported in the posterior parietal region of the left hemisphere, and also in the sub-cortical white matter of both hemispheres. EW was assessed for the presence of apraxia shortly after admission to the rehabilitation centre, approximately five months after the incident. On initial presentation EW was ambulant, though somewhat unsteady on her feet. No significant motor or sensory impairments were found in her upper limbs. She had good social skills and was able to make appropriate verbal responses to social contact, though the content of her speech to *specific* questioning was poor. The psychologist's assessment reported the presence of a profound amnesia with little insight into her condition. EW was also noted to have poor problem-solving skills.

Apraxia assessment was completed (appendices D1-D4) for both upper limbs and provided evidence of severe ideational *and* ideomotor apraxias (Table 10.1). During the testing procedure EW exhibited behaviours which were indicative of a motor disconnection disorder. This meant that reproduction of the requested hand gestures or movements were sometimes completed using the left hand when the right hand was being tested, and vice versa. At times EW was unable to use the correct limb for testing unless the other was restrained. Once started on the movement, however, she was able to continue. No callosal damage was noted on the MRI scan.

The types of errors made by EW at the first assessment and the resulting apraxia scores (Table 10.1) were indicative of underlying dysfunctions in both motor planning

Table 10.1 EW - Clinical assessment scores (raw and %) at first examination.

Clinical assessments	Right Hand	Left Hand
Test 1- gesture copying (max. = 40) (Appendix D2)	27 (67.5%)	23 (57.5%)
Test 2 - object use test (max. = 36) (Appendix D3)	16 (44.4%)	16 (44.4%)
Test 5 - DeRenzi et al test, gesture copying (max. = 72) (Appendix D1)	27 (37.5%)	28 (38.8%)
Mean % scores for Tests 1, 2 & 5.	49.8%	46.9%
Test 3 - ideational apraxia (max. = 12) (Appendix D4)	0	
Test 4 - agnosia (max. = 20) (Appendix E)	12 (60 %)	

and execution, and in the *idea* of object use and performance. In the gesture copying test (appendix D2) she made errors in the plane of movement as well as using

incorrect hand positions both to verbal command and in copying gestures. Perseveration of gesture was a particular feature of EW's performance and was noted especially in the assessment of the left hand. The object-use test (appendix D3) provided evidence of errors associated with apraxia; timing and speed of gestures were inaccurate, the plane of movement was incorrect for some object-use pantomimes, incorrect orientation of the *imagined* object and the *real* object was observed. This was particularly clear in the 'pen' and 'key' items. Some vocalisation was produced by EW during the assessment process, particularly in the 'razor' and 'toothbrush' items. The DeRenzi et al test evoked major difficulties in reproducing static complex finger and hand positions. EW had more success in the movement items but demonstrated a tendency for perseveration. Errors noted during this test included inaccurate plane of movement or hand posture, inaccurate timing of movements with jerky and non-fluent movements being common, and severe sequencing errors. All these provided evidence for the presence of a severe *ideomotor* apraxia exhibited in both upper limbs.

In the assessment used to determine the presence of *ideational apraxia* (appendix D4) EW was unable to complete any part of the test successfully. On questioning, she recognised each object placed in front of her, could name each object and state its use. She was unable, however, to use the objects together or sequence their use meaningfully. These performance data provided evidence to indicate a severe *ideational* apraxia being present in EW.

10.2 Aims of the study

Using a single case design, this study aimed to replicate the previous single case investigations and to :

- evaluate the effectiveness of a sensory stimulation intervention protocol on movement performance in a person with severe ideomotor *and* ideational apraxia.
- evaluate the effectiveness of task breakdown on self care independence in a person with severe ideomotor *and* ideational apraxia.
- examine kinematic recording data of a person with both ideomotor *and* ideational apraxia in relation to the data gathered from those with ideomotor apraxia only.
- explore variability of motor performance in a person with ideomotor *and* ideational apraxia.

10.3 Procedure

To replicate the previous intervention case studies an ABA design was used. A baseline period of four weeks involved therapy sessions of domestic activities, leisure skills activities and personal self-care training using task break-down strategies. This was followed by an intervention phase of four weeks during which the sensory stimulation protocol described in Chapter 8 (Case 3) was carried out. The post-intervention phase was interrupted by an extended Christmas break for EW, but on her return to the rehabilitation centre further data collections were made. The therapeutic input for this post-intervention period involved mainly leisure skills

activities including dance music therapy, quizzes and games but also a continuation of personal self-care training and some domestic activities.

During the period of investigation, therapy sessions in each experimental phase were spread across the whole available range of timetabled slots in both the morning and afternoon periods. Daily sessions were also not always possible during the working week due to therapist constraints, plus hospital and other appointments for EW. Extended weekend leave and other activities for EW also left gaps in the proposed daily data collection. Generally three or four therapy sessions a week were carried out.

10.3 i] Outcome measures

In consultation with the rehabilitation team, outcome measures were chosen which could be incorporated easily into the timetabled therapy sessions and which were standardised tests. These were the Nine Hole Peg Test (NHPT; Mathiowetz et al, 1985) and the Gibson Spiral Maze from the CAPE (Pattie & Gilleard, 1979). These tests had the advantage of being quick, reliable, available in most occupational therapy departments, reproducible, and giving quantitative scores. Pilot trials of the tests indicated that EW had some difficulty in completing them independently. Therefore it was decided that in addition to the timing element of the tests, a record would be kept of the number and types of prompts needed to complete the tasks. A score of three would be given for a physical prompt, two for a gestural prompt and a score of 1 for each verbal prompt. In this way, improvement in performance could be measured by a reduction in time taken for completion, and also in a reduction in the number and type of prompts required for successful completion of the task. The tests were

administered at the start and finish of each therapy session throughout all phases of the experiment.

A total of 22 data collection points was made in the baseline phase to establish the range of performance scores for EW. 14 data points were collected in the intervention phase of the study, and a total of seven data points in the post-intervention phase. There was a four week gap between the first and second post-intervention data point due to the extended Christmas vacation taken by EW.

10.3 ii] Kinematic recording

As with the group study and previous single case investigations, the drinking task was performed by EW in three modality conditions (verbal command, visual/copy condition, and real-object use). In addition, the gestures of HAMMER, CARVE and WIND were recorded (described in section 7.3). The non-dominant right hand was used for all recordings. Prior to the start of the investigation, pilot trials of the drinking task indicated that EW was unable to complete the movement in a manner sufficient for the computer software analysis. The main difficulty lay in the severe perseveration exhibited during all performances of the task. In addition, EW omitted the 'reach' and 'return' elements of the drinking task protocol. Weekly assessments were carried out to determine EW's ability to follow the research protocol. The investigation was thus delayed for one month until EW demonstrated an ability to complete the experimental task.

As with case 4, fewer recordings were made of EW's motor performance than was originally planned. The practical difficulties inherent in completing clinical research whilst not 'on-site' were largely responsible for this. Ultimately four recordings were

made in the baseline phase, two in the intervention phase, and three in the post-intervention phase. It was considered that some degree of comparison and analysis should be feasible from these limited data.

10.4 Results

10.4i] Outcome measures.

Descriptive analysis of the outcome data was completed and a feature, once more, was the variability in performance as shown by the range of scores within each phase (Table 10.2). Results indicated a reduction in mean time taken to complete the NHPT and Maze tasks for both hands in the intervention phase of the experiment. This was accompanied by a reduction in the standard deviation scores across all measures when compared with the baseline phase, although the inter-quartile range for the right hand measures showed an increase. The NHPT scores increased in the post-intervention period (denoting a worsening of performance) with larger mean scores, broader standard deviations and wider inter-quartile ranges. These results might suggest that there was some improvement seen in the intervention phase of the study, but that this improvement was lost in the post-intervention phase. The maze task timing, however, showed lower mean scores, a reduced range and smaller standard deviation scores during the post-intervention phase for both hands. No correlation was found between the maze timing and the NHPT timing in any of the phases (Right hand :- baseline r= .08 *NS*, intervention r= .57 *NS*, post-intervention r= .65 *NS*. Left hand :- Baseline r= .12 *NS*, intervention r= .06 *NS*, post-intervention r= .57 *NS*). Each of the tasks, therefore, could be considered independent measures of different elements in motor performance .

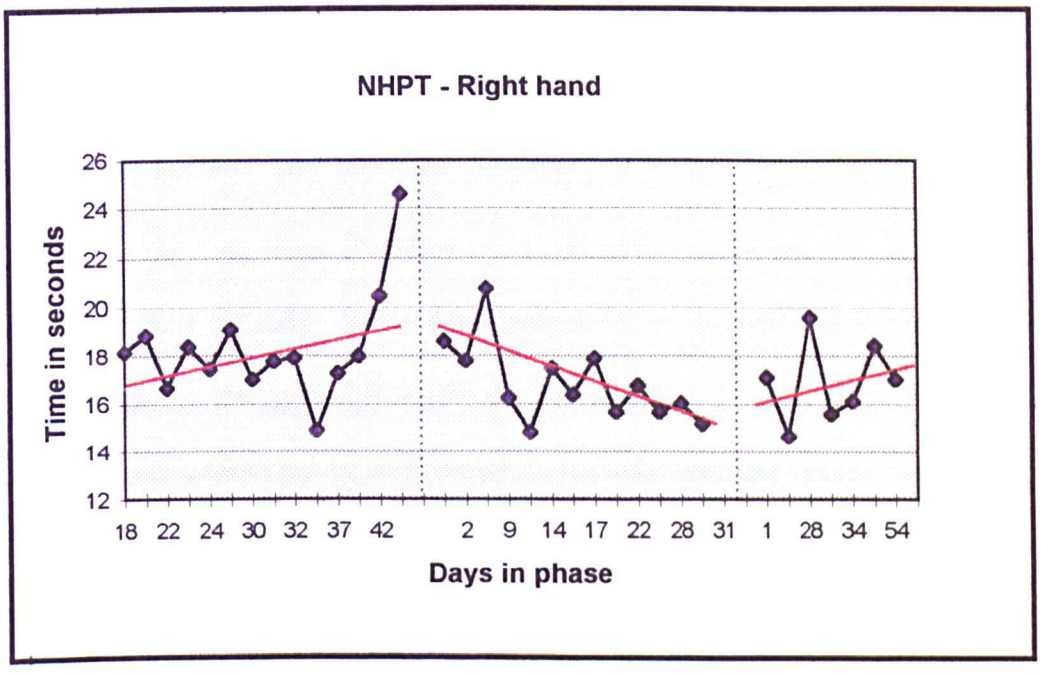
Table 10.2 EW - synopsis of outcome measures data from ABA phases

(All data given in seconds)

	Baseline	Intervention	Post-intervention
NHPT *(norm = 18 secs.)* **Right hand.**			
Mean score	18.01	16.86	17.17
Range	14.9 - 24.6	14.8 - 20.8	14.7 - 19.6
Broadened median	**17.91**	**16.58**	**17.43**
Standard deviation	1.93	1.63	1.69
Interquartile range	1.57	2.17	2.86
No. of data points	21	13	7
NHPT **Left hand.**			
Mean score	19.8	16.86	17.63
Range	16.5 - 30.6	14.5 - 22.0	15.4 - 19.4
Broadened median	**19.34**	**15.99**	**18.15**
Standard deviation	3.17	1.87	3.19
Interquartile range	3.58	1.94	2.62
No. of data points	21	13	8
Maze. *(norm = 106 secs using an elderly population.)* **Right hand.**			
Mean score	46.61	43.92	24.38
Range	29.1 - 70.8	25.5 - 57.0	15.0 - 35.8
Broadened median	**45.71**	**44.59**	**30.06**
Standard deviation	10.92	10.18	8.74
Interquartile range	16.83	18.47	16.99
No. of data points	21	13	7
Maze. **Left hand.**			
Mean score	49.25	31.94	26.83
Range	29.7 - 82.4	14.9 - 74.2	13.0 - 57.6
Broadened median	**46.12**	**38.12**	**21.96**
Standard deviation	16.03	15.59	7.5
Interquartile range	20.59	16.49	13.19
No. of data points	21	13	7

The data was plotted for each of the measures, both right and left hand. The right

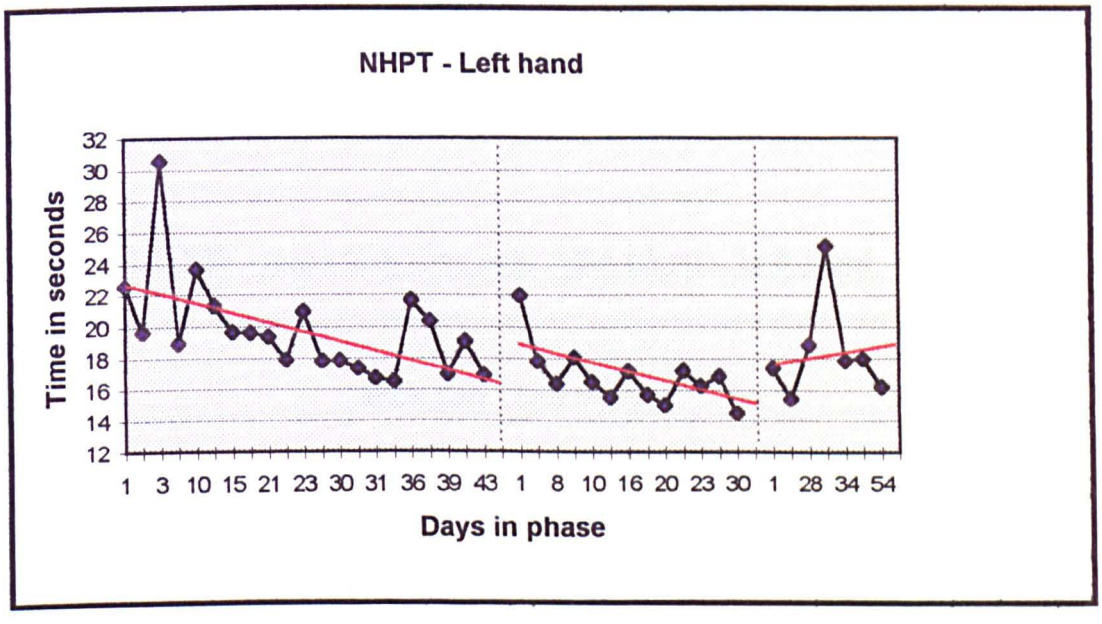
Figure 10.1 EW - NHPT time over ABA phases. Right hand



hand baseline performance in the NHPT suggested, if the regression line was viewed as meaningful, a gradual deterioration in time taken to complete the test, although the daily variation in performance is clearly visible (Figure 10.1). Regression analysis only acounted for 13.4% of the variation [r=.134 F(1,19)= 2.95 *NS*] so the slope of the regression line should be viewed with some caution in the visual analysis. In this baseline phase the last two data points could be considered abnormal performances ('outliers') judging by her previous timing scores, though no explanation could be offered for this. If these data points were removed, then a more even performance would have been seen. The execution of the NHPT in the *intervention* phase of the study showed variability again, but the regression line indicated a trend of improvement in speed of performance (r= 319 F(1,11) 5.17 p< .05]. The *post-intervention* phase suggested a deterioration in performance with the regression analysis only accounting for 10.6% of the variance. This might suggest that not only

did the performance with the right hand decrease generally in that post-intervention phase, but that the consistency of performance was also decreased. The left (dominant) hand data revealed a fairly steady trend of improved speed at the NHPT over both baseline and intervention phases with the regression lines significantly predicting the data slope [baseline r= .33 $F(1,19) = 9.64$ $p < .01$ Intervention r=.42 $F(1,11) = 8.01$ $p < .05$]. Some deterioration in performance in the post-intervention phase might be interpreted from the plotted data but the forth data point, if regarded as an 'outlier', distorted an otherwise less dramatic increase in task time.

Figure 10.2 EW - NHPT time over ABA phases. Left hand



Visual analysis of the right hand performance on the maze task (completion time) indicated the variable nature of EW's output (figure 10.3). The baseline phase showed wide variation in timing, though the intervention phase indicated a slight trend of improvement. The regression lines were non-representative of any trend in the data

due to this variability (Baseline r= .008, Intervention r=.17). The post-intervention phase, once again, illustrated a deterioration in performance, although this performance was generally *better* than in the baseline and intervention phases..

Figure 10.3 EW - maze task time over ABA phases. Right hand

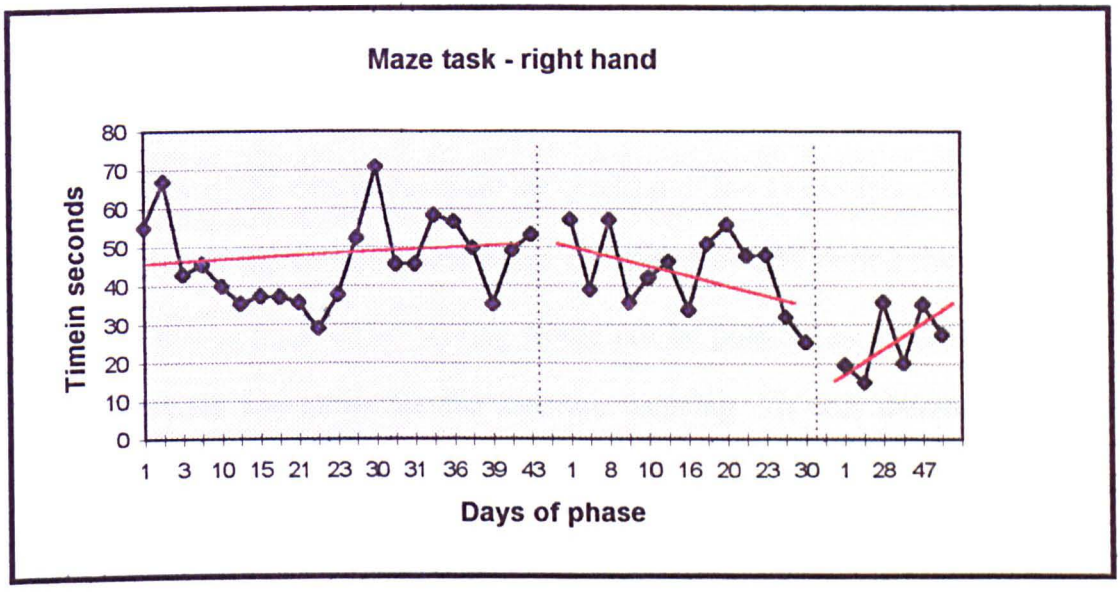
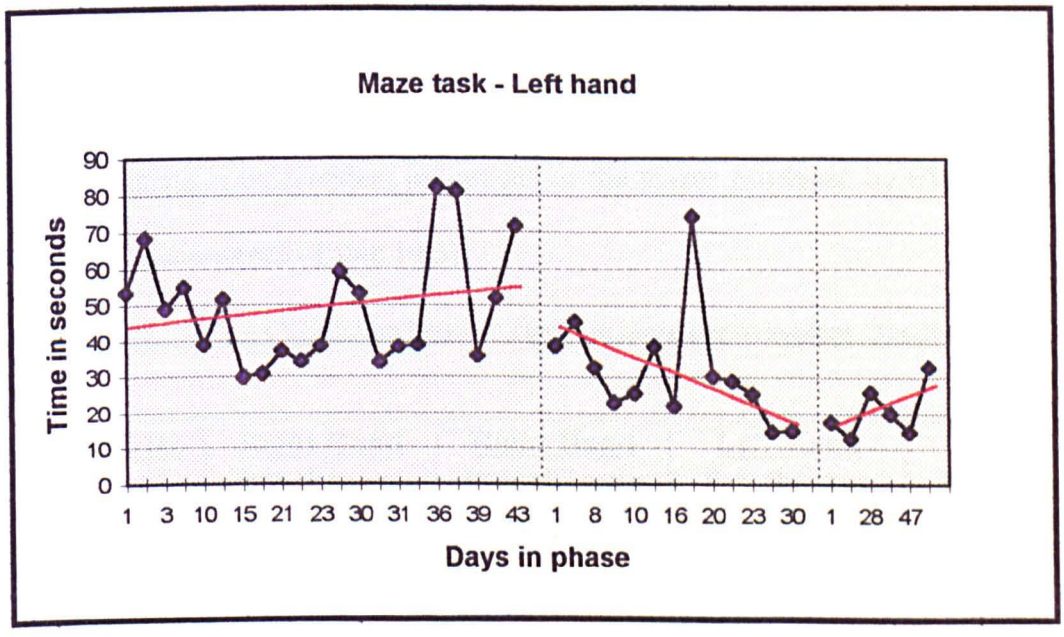


Figure 10.4 EW - maze task time over ABA phases. Left hand



This pattern was repeated fot the left hand maze task data (Figure 10.4) which could be seen, from visual inspection, to illustrate the performance variability in EW especially in the baseline phase. This variability was reduced in the intervention phase with a trend of improving performance also visible, despite the appearance of an 'outlier' data point (number 8) within the set. Again, the data showed a deteriorating performance in EW's task completion time in the post-intervention phase of the study though many of the data points were the quickest of the whole data set.

Examination of the Gibson Spiral Maze completed by EW during the baseline phase indicated that normal 'error' scoring would not be possible as the abundance of line crossings were too numerous for accurate counting. It was therefore decided to attempt a 3-category scoring system for the maze performance. 'Poor' = 1 point (criteria : an appearance of a very messy page with repetition errors and lines crossed many times), 'Medium' = 2 points (criteria : a less messy page with only one or two repetition errors and lines crossed), and 'Good' = 3 points (criteria : a neat page appearance and the pencil track showing mostly spiral configuration). Examples of these categories are given in appendix F. Correlations between the researcher and two therapists in independent judgments of the mazes produced by EW showed high levels of concordance (Table 10.3).

Table 10.3 EW - Correlation between raters in maze performances. Baseline phase.

Correlations r =	Right Hand Pre-therapy maze (N=21)	Right Hand Post-therapy maze (N=21)	Left Hand Pre-therapy maze (N=21)	Left Hand Post-therapy maze (N=21)
Raters 1 & 2	0.86	0.91	0.55	0.83
Raters 1 & 3	1.00	0.72	0.80	0.82
Raters 2 & 3	0.84	0.77	0.70	0.74

It was considered that an increase in numbers of mazes within the 'good' category, and a decrease in 'poor' category mazes, could be taken as an indication of improvement in performance ability. Analysis of the mazes completed across all experimental phases demonstrated a moderate shift towards the 'medium' and 'good' performance categories, and away from the 'poor' performance category, as an *immediate* effect of therapy in all phases of the experiment and over time (Table 10.4).

Table 10.4 EW Maze performance data (raw and %) across ABA phases.

Categories	**Baseline** (N=21)		**Intervention** (N=13)		**Post-intervention** (N=7)	
	Pre	**Post**	**Pre**	**Post**	**Pre**	**Post**
Right Hand						
'good'	5 *(23.8%)*	6 *(28.6%)*	1 *(7.6%)*	1 *(7.6%)*	1 *(14.3%)*	5 *(71.4%)*
'medium'	5 *(23.8%)*	8 *(38.1%)*	2 *(15.4%)*	4 *(30.8%)*	5 *(71.4%)*	2 *(28.6%)*
'poor'	11 *(52.4%)*	7 *(33.3%)*	10 *(76.9%)*	8 *(61.6%)*	1 *(14.3%)*	0 *(0%)*
Left Hand						
'good'	5 *(23.8%)*	7 *(33.3%)*	5 *(38.5%)*	4 *(30.8%)*	4 *(57.1%)*	5 *(71.4%)*
'medium'	7 *(33.3%)*	7 *(33.3%)*	2 *(15.4%)*	5 *(38.5%)*	2 *(28.6%)*	2 *(28.6%)*
'poor'	9 *(42.9%)*	7 *(33.3%)*	6 *(46.1%)*	4 *(30.8%)*	1 *(14.3%)*	0 *(0%)*

A significant relationship was found between the time taken to complete the maze task and the quality category of performance on that task. 'Good' category

performance mazes were significantly quicker in their timing scores compared to the 'medium' and 'poor' maze categories across all phases of the experiment in both pre-therapy and post-therapy measures for both hands [Right hand pre-therapy $F(2,40) = 4.08$ $p < .05$; Right hand post-therapy $F(2,38) = 16.21$ $p < .001$; Left hand pre-therapy $F(2,40) = 10.1$ $p < .001$; Left hand post-therapy $F(10.6$ $p < .001]$. Clearly the time taken to complete the maze task was a measure closely associated with performance errors. A speedy performance was not, as might have been expected, associated with increased errors but might have been more an indication of a good motor performance day in the variable range that EW operated in.

Examination of the prompts EW needed to complete the tasks revealed variable and intermittent performances in the baseline phase. No discernible pattern of prompting was evident, but was associated with increased time taken to complete the tasks. It might, therefore, be interpreted as a component part of the performance variability noted earlier. The maze task in particular required physical, gestural and verbal prompts in the baseline phase. Prompting needs had largely disappeared by the intervention phase. EW was able to self-correct errors by the intervention phase but remained distractible during assessments. Minimal numbers of prompts were recorded in the post-intervention phase for either task.

Analysis of the data to determine the *immediate* effects of therapy in each of the three phases was completed using paired t-test statistics. No significant differences were found between the pre and post test scores in the baseline phase in either the NHPT

(right hand t(20) = 1.1 *NS*, left hand t(20) = -0.85 *NS*) or the maze task (right hand t(20) = 0.97 *NS*, left hand t(20) = -0.28 *NS*). This was repeated for both the intervention phase [NHPT :- right hand t(12) = 1.13 *NS*, left hand t(12) = 1.22 *NS*. Maze task:- right hand t(12) = 0.42 *NS*, left hand t(12) = 1.56 *NS*] and the post-intervention phase [NHPT :- right hand t(6) = -1.45 *NS*, left hand t(6) = -1.3 *NS*. Maze task:- right hand t(5) = 4.37 P< .01, left hand t(5) = 0.44 *NS*]. The exception, as described, was the time taken to complete the maze task with the right hand in the post-intervention phase. This was assumed to be an anomaly in the study as no plausible explanation could be offered for this one significant effect.

10.4 ii] Apraxia assessment scores

Changes were noted in the apraxia assessment scores between the initial screening and the first assessment at the start of the investigation. This indicated some improvement had occurred in the apraxic condition which might have been a part of the natural recovery process (Table 10.5). Assessments were made at the *start* of the baseline phase, the *start* of the intervention phase and after EW's Christmas break *during* the post-intervention phase. During the baseline assessment for ideomotor apraxia EW was noted to have made errors in movement timing, plane of movement, perseverative errors and inaccurate hand positioning. In the test for ideational apraxia at the baseline assessment, EW was partially correct in her performance in manipulating objects in sequence in three of the test items (lighting a candle, sending a letter, polishing shoes).

During the intervention phase of the study, similar errors to those observed in the baseline phase were noted in EW's performance throughout the apraxia assessments with consequent minimal changes in scoring (Table 10.5). Post-intervention assessment indicated poor performance in the object-use test with greater numbers of 'body part as object' errors being made.

Table 10.5 EW : Apraxia test scores across ABA phases.

	Initial test		**Baseline 'A'**		**Intervention 'B'**		**Post-intervention 'A'**	
Time since onset	22 weeks		26 weeks		29 weeks		40 weeks	
	Right	Left	Right	Left	Right	Left	Right	Left
Test 1 - gesture copying (max. = 40) (appendix D2)	27	23	33	23	34	34	35	32
Test 2 - object use test (max. = 36) (appendix D3)	16	16	25	23	30	28	21	21
Test 3 -ideational apraxia (max. = 12) (appendix D4)		0		3		4		0
Test 4 - agnosia (max. 20)		12		13		13		11
Test 5 - De Renzi test, gesture copying (max.= 72) (appendix D1)	27	28	31	35	33	38	18	16

The De Renzi et al test of complex finger and hand gesture copying also showed a decline in performance ability, particularly in the static positions requested.

Inaccurate hand positions, wrong plane of movement, poor timing and fluidity of movements, wrong orientation of hand postures were all noted errors during the post-intervention phase assessment. Performance in the assessment for ideational apraxia was also severely impaired during the post-intervention examination. All items showed errors of use during the assessment (Table 10.6) and suggested a deterioration in praxic ability. These assessment results supported evidence of deterioration of performance found in the NHPT.

Table 10.6 EW - errors in ideational apraxia assessment, post-intervention phase.

	Activity	Comments /errors made
1	**Light a candle** (candle, candlestick, matches)	Matches in mouth. Wrong end of candle into holder. Matches 'played with'.
2	**Paper into a file** (sheet of paper, file, hole punch)	Hole-punch placed into file. No use of the paper. File opened and closed without purpose.
3	**Polish shoes** (shoes, polish, brush, cloth)	Picked up shoe without purpose. No polish used. Brush tapped onto shoe. Duster used appropriately.
4	**Send a letter** (paper, envelope, stamp)	Stamp placed into paper. Envelope placed on top of letter.
5	**Lock a box** (box, padlock and key)	Padlock placed into opened box. No attempt made to close box. Unable to manipulate padlock meaningfully.
6	**Make a cup of tea** (teapot, kettle, cup, saucer, spoon, teabag, milk jug, sugar bowl).	Used pouring motion with teapot over the side of the table. No other manipulation of objects.

10.4 iii] Kinematic recording

The right (non-dominant) hand only was used for the kinematic analysis as EW was judged to be unable to manage the lengthy time which *both* hand recordings would have entailed. Descriptive data from the drinking task (Table 10.7) suggested that the improvement noted in the outcome measures previously described might also be replicated in the kinematic analysis. Smaller mean scores in the total time taken to complete the drinking task, in each modality condition, might be taken to indicate an

Table 10.7 EW - Drinking task time over ABA phases.

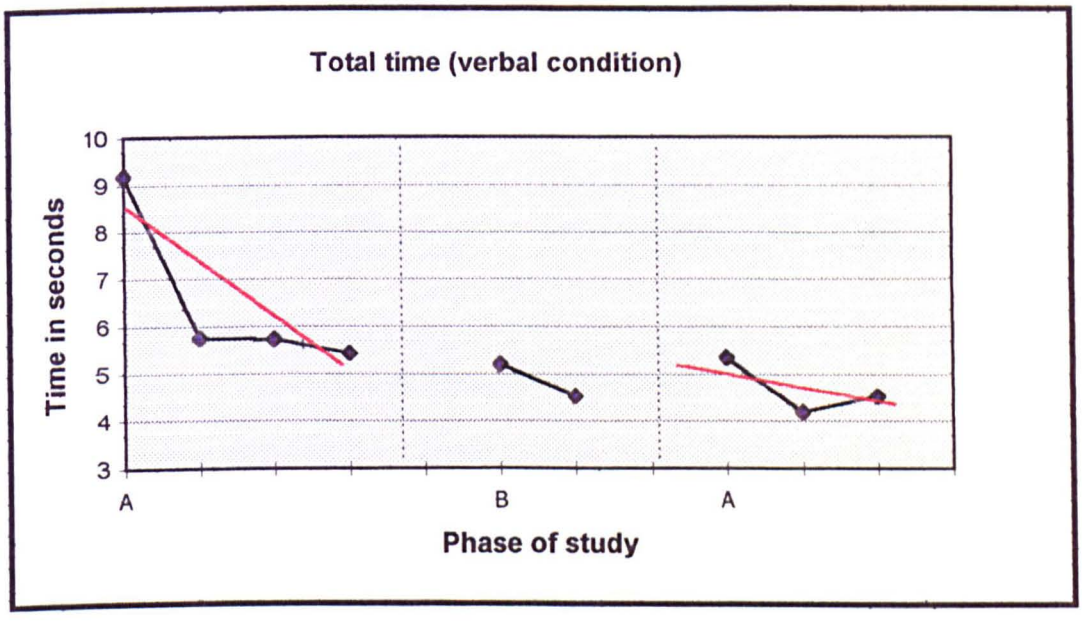
	Baseline	Intervention	Post-intervention
Total time - verbal condition			
Mean	6.53	4.87	4.68
Standard deviation	1.77	0.48	0.60
No. of data points	4	2	3
Total time - copy condition			
Mean	4.81	4.00	3.81
Standard deviation	0.75	0.24	0.67
No. of data points	4	2	3
Total time - real condition			
Mean	6.32	6.22	5.59
Standard deviation	0.62	0.03	0.67
No. of data points	4	2	3

improved speed of performance in the intervention phase of the study with the smaller standard deviation scores perhaps indicative of a more *consistent* performance. If so, then this consistency was lost in the post-intervention phase if the larger standard

deviation scores are meaningful. This was in contrast to the improvement in speed in the post-intervention period, suggested by the reduction in mean scores. The small data set meant that the interquartile range was not an appropriate statistic.

Visual inspection of the plotted data revealed a variable performance across the three modality conditions though the small number of data points has limited the conclusions which might be drawn. The verbal condition (Figure 10.5) suggested a relatively stable performance once the learning effect from the first trial has been taken into consideration.

Figure 10.5 EW - Total time (verbal condition) across ABA phases



The copy condition data (Figure 10.6) however, suggested a trend of deteriorating performance in the baseline phase, an improved performance in the intervention phase,

and an erratic performance in the post-intervention phase. With such small data sets, though, such impressions may not be true.

Figure 10.6 EW - Total time (copy condition) across ABA phases

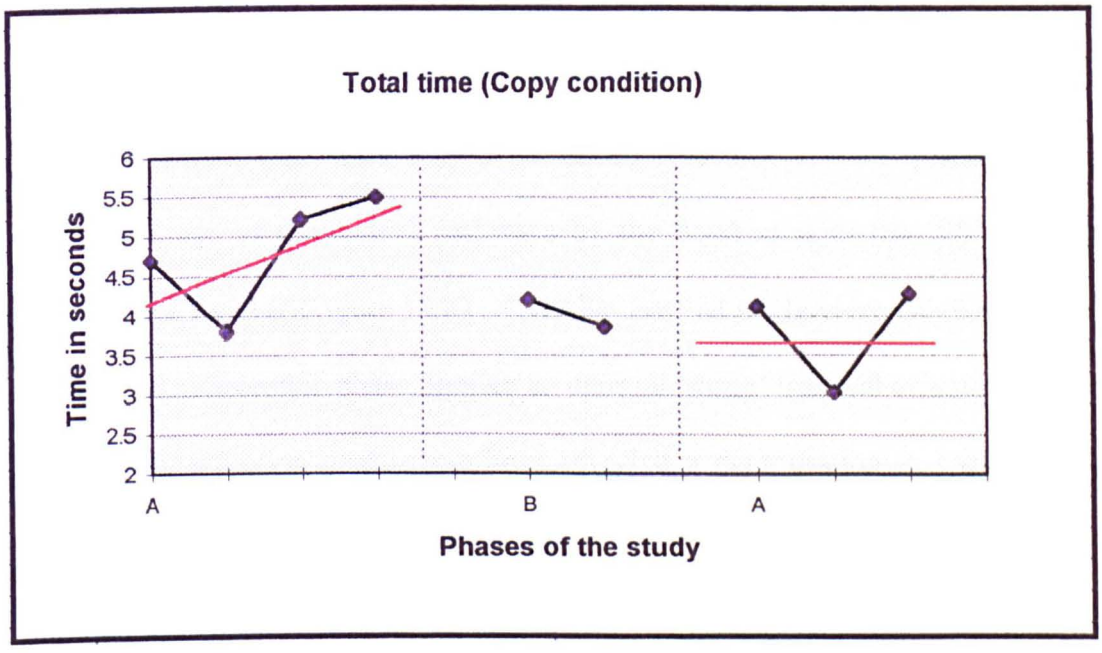
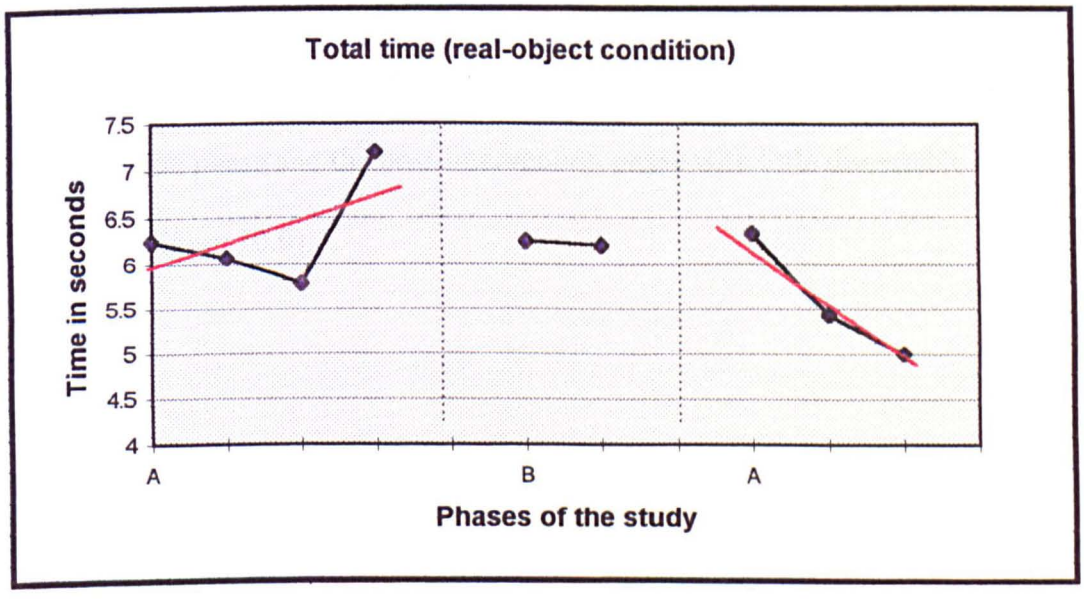


Figure 10.7 EW - Total time (real-object condition) across ABA phases



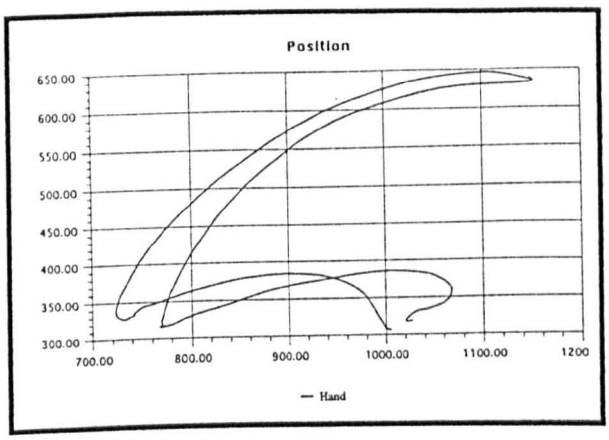
The most notable part of the real-object condition data was the striking improvement in the post-intervention phase.

The drinking task showed some inaccuracies in spatial trajectory particularly during the 'reach' and 'return' elements of the task in the baseline phase. There was an accompanying abnormality of the shoulder trajectory (Figure 10.8). These hand and shoulder trajectories were improved during the intervention phase with more consistent use of the spatial pathways by the hand seen in the 'lift' and 'down' elements of the task (Figure 10.8). A smaller amount of shoulder movement was seen during the intervention phase, though an unusual 'shrug' was still evident. Analysis of the post-intervention phase recordings revealed a deterioration in spatial trajectory patterns back to baseline status.

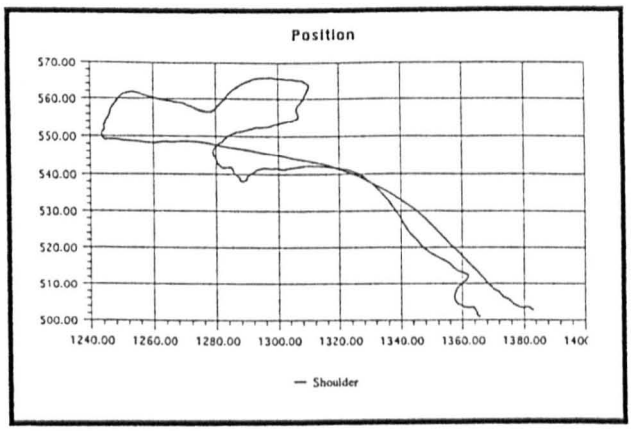
The most notable feature about EW's drinking task performances was the presence of the smooth bell-shaped velocity curves (Figure 10.9). There was no evidence of the multi- peaked velocity curves seen in other apraxic subjects, and is consistent with Hermsdorfer et al (1996) results which also showed apraxic subjects with normal kinematics of movement. These velocity patterns were maintained during the intervention phase and the post-intervention phase with little discernible change evident (Figure 10.9).

Figure 10.8 EW Examples of hand and shoulder trajectories in ABA phases.

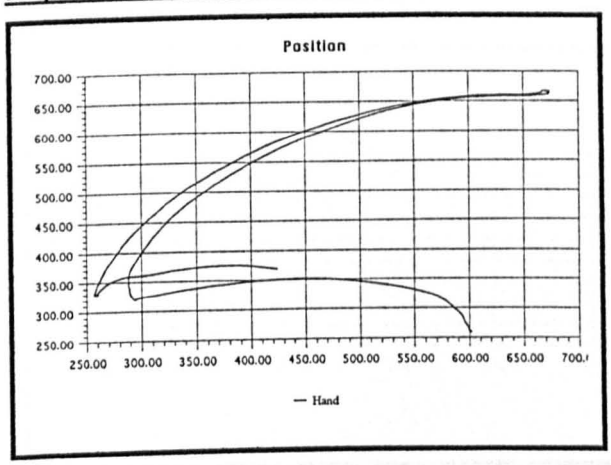
ai] Hand trajectory. Baseline



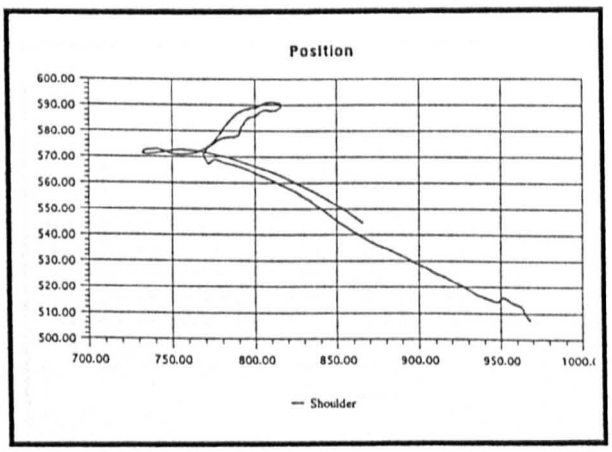
bi] Shoulder trajectory. Baseline



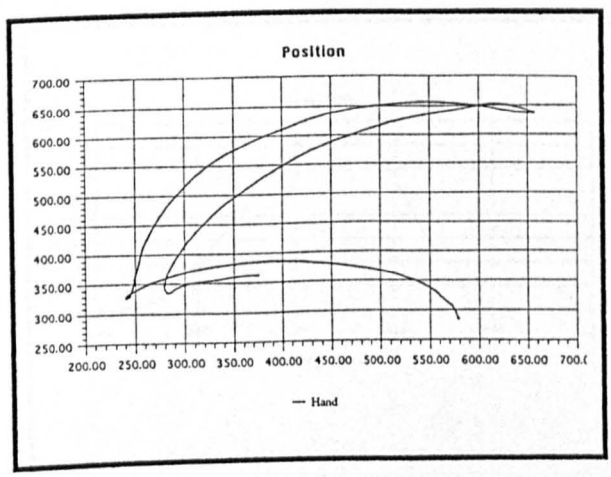
aii] Hand. Intervention phase.



bii] Shoulder. Intervention phase.



aiii] Hand. Post-intervention phase.



biii] Shoulder. Post-intervention phase.

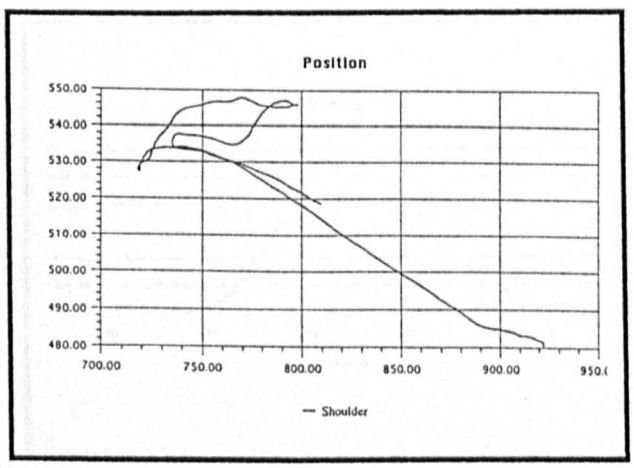
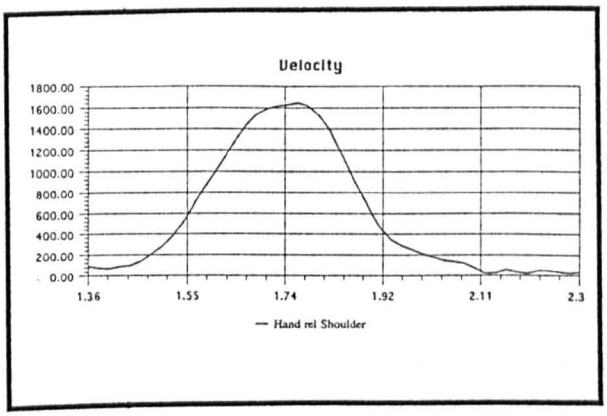
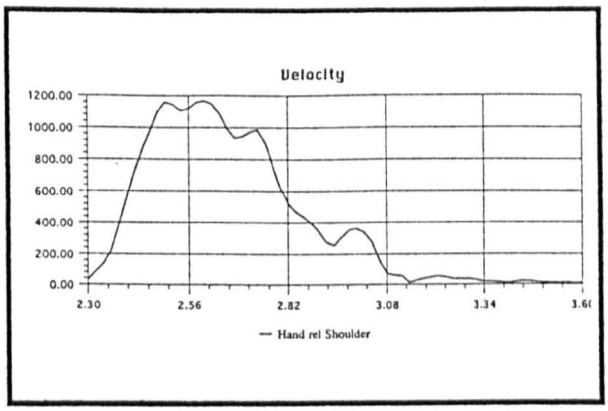


Figure 10.9 EW - Examples of velocity curves in ABA phases.

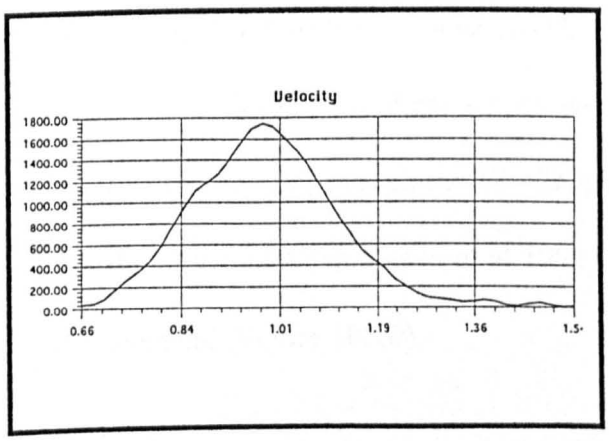
ai] 'Lift' - Baseline phase.



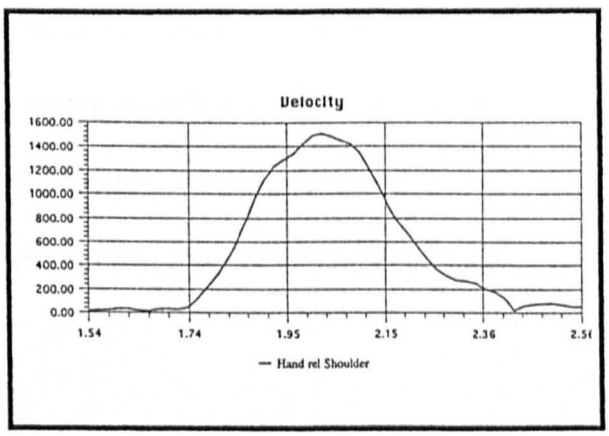
bi] 'Down' - Baseline phase.



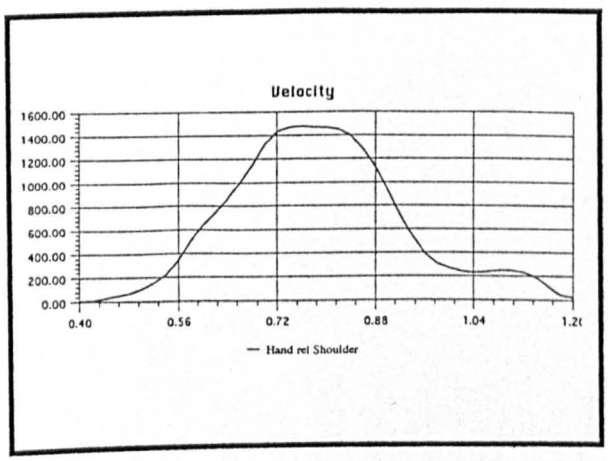
aii] 'Lift' - Intervention phase



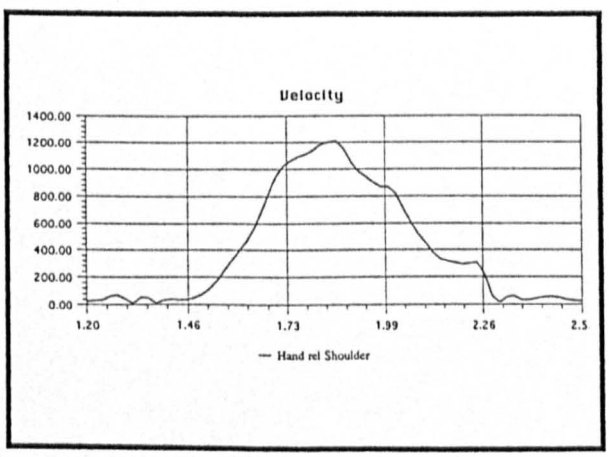
bii] 'Down' - Intervention phase



aiii] 'Lift' - Post-intervention phase



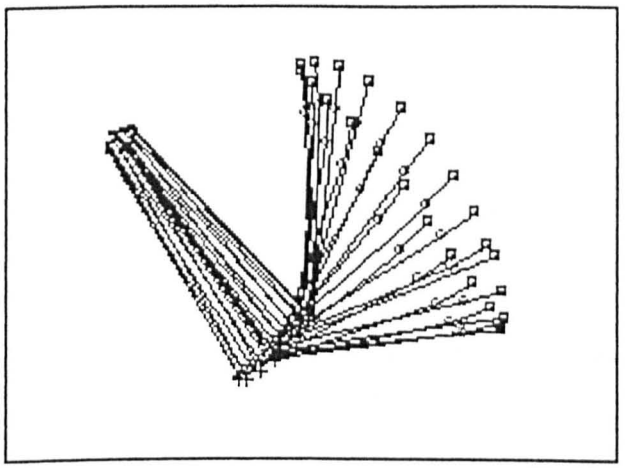
biii] 'Down' - Post-intervention phase



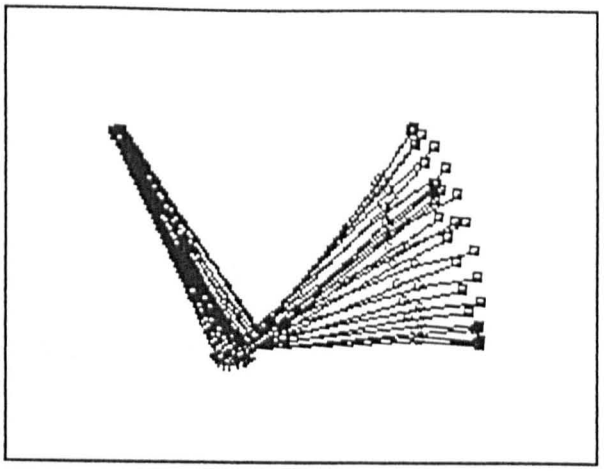
Analysis of the recordings made of the HAMMER, CARVE and WIND gestures indicated some performance errors in the baseline phase. EW did not demonstrate the abnormal shoulder movement and over-use of that joint seen in the previous cases described. Her movements, though, showed an inability to keep a consistent spatial trajectory in the HAMMER gesture. There was a tendency for forward displacement of the movement at each repetition (Figure 10.10). Similar spatial inaccuracies were noted in the CARVE gesture recordings where an ovoid motion was enacted rather than the expected horizontal forward and backward movement. In the WIND gesture, EW was unable to produce a well-formed circular motion and instead over-used the elbow to perform a large oblique movement (Figure 10.10). This movement had a progressive forward displacement in space with each repetition of the cycle of movement. These movement errors were somewhat modified in the intervention phase of the project, though the WIND gesture continued to be exaggerated and was classified (according to Raade et al 1991) as a spatial error with enhanced amplitude of movement (Figure 10.10).

Figure 10.10 EW - Examples of gestural performance. Baseline and intervention.

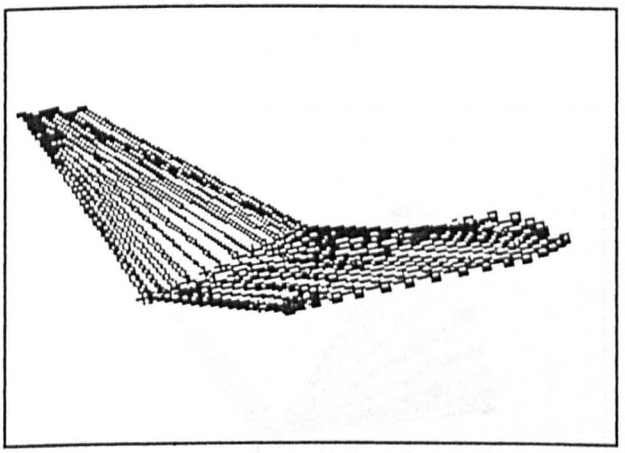
ai] Baseline - HAMMER



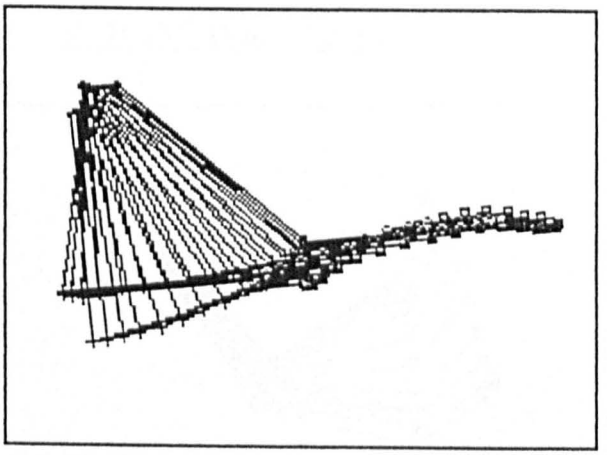
bi] Intervention - HAMMER



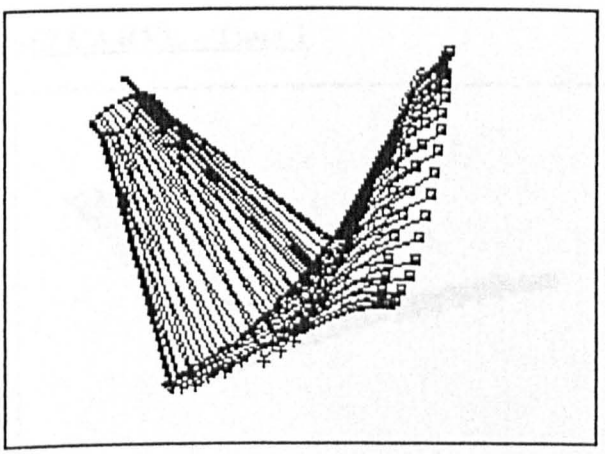
aii] Baseline - CARVE



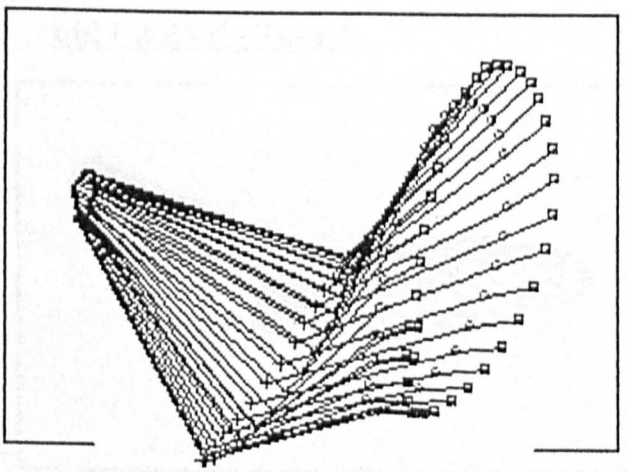
bii] Intervention - CARVE



aiii] Baseline - WIND



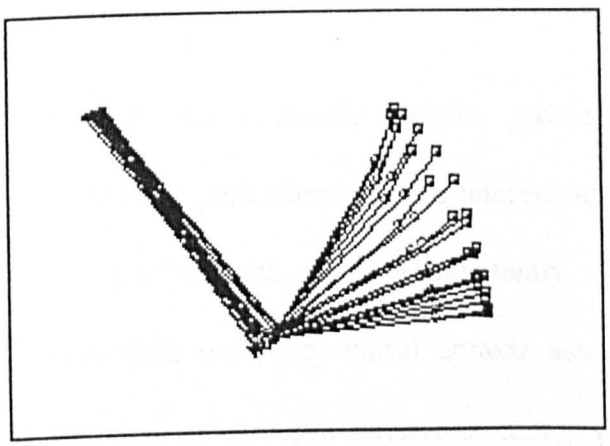
biii] Intervention - WIND



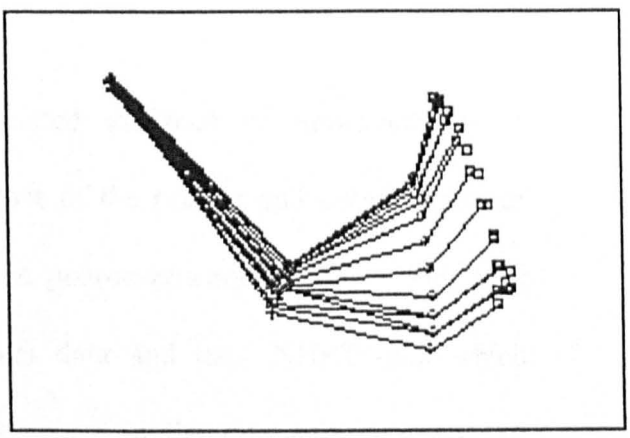
The post-intervention recordings illustrated the variability of performance by EW. Early in the post-intervention phase (Time 1) the recordings suggested a retention of the improved gesture ability shown by EW in the HAMMER and CARVE movements (Figure 10.11) although the WIND gesture continued to show evidence of movement errors in plane of movement and amplitude. The last recording of the post-intervention phase, however, (Time 5) suggested a deterioration in gestural performance in two of the gestures. Exaggeration of movement and poor consistency of spatial trajectory was

Figure 10.11 EW Post-intervention phase gesture recordings.

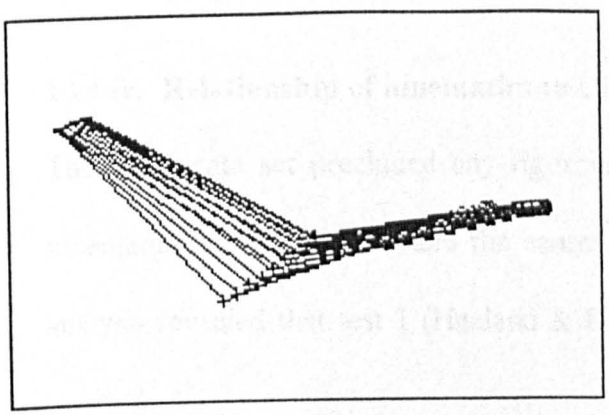
ai] HAMMER - Time 1.



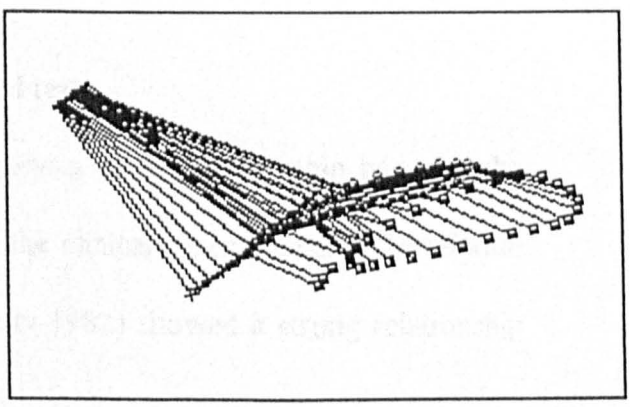
bi] HAMMER - Time 5



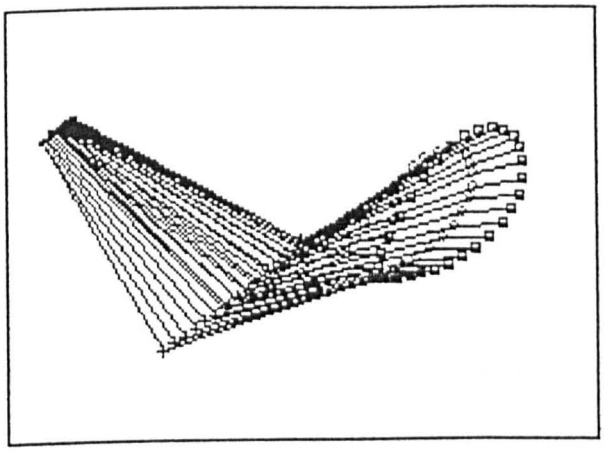
aii] CARVE - Time 1



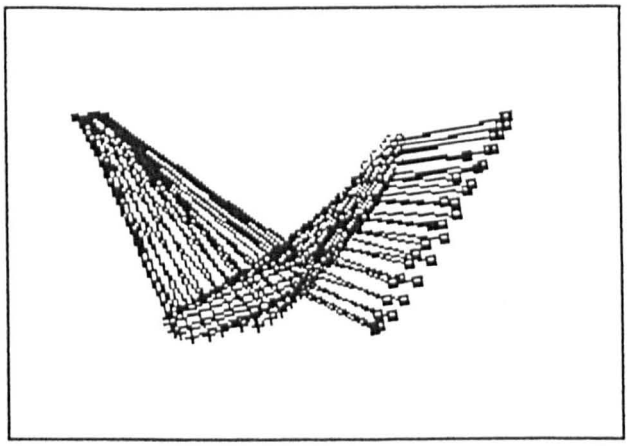
bii] CARVE - Time 5



aiii] WIND - Time 1



biii] WIND - Time 5



evident in the CARVE gesture and showed an inaccurate hand position together with a downward circular movement (Figure 10.11). The WIND gesture was produced in the wrong plane in front of the body, although the 2D recording cannot fully illustrate that feature.

Overall, the kinematic analysis provided limited evidence of improvements in movement performance in the intervention phase of the project and deterioration of that performance, albeit inconsistently, in the post-intervention phase. This was consistent with the clinical apraxia assessment data and the NHPT data which revealed small improvements in motor performance in the intervention phase but showed a declining performance in the post-intervention phase of the investigation.

10.4 iv. Relationship of kinematics to clinical tests

The small data set precluded any rigorous analysis of the relationship between the kinematics of performance and the scores on the clinical assessments. Correlation analysis revealed that test 1 (Haaland & Flaherty 1982) showed a strong relationship

with the total time taken to complete the drinking task in each modality condition [Verbal r(3)= - .85 Copy r(3) = - .92 Real r(3) = .87] whereas the De Renzi et al (1980) test only related strongly to the real-object condition [Verbal r (3) = .36 Copy r(3) = .50 Real-object r(3) = .99] though none reached a level of statistical significance. The test for agnosia related poorly to the verbal and copy conditions (r= -.03 and r= .13 respectively) but r= .87 for the real-object condition. None of the clinical tests reached statistical significance level in *predicting* performance in total time taken to complete the drinking task though, as the correlations indicated, the De Renzi test accounted for over 98% of the variance in the real-object condition and was the highest predictor [**De Renzi test** :- Verbal condition r=.13 F(1,1)= 0.15 *NS.* Copy condition r=.25 F(1,1) = 0.34 *NS.* Real-object condition r=.985 F(1,1)= 65.33 *NS.* **Haaland & Flaherty test 1** :- Verbal condition r=.73 F(1,1)= 2.63 *NS.* Copy condition r=.85 F(1,1) = 5.72 *NS.* Real-object condition r=.75 F(1,1)= 3.0 *NS.* **Agnosia test** :- Verbal condition r=.0008 F(1,1)= 0.0008 *NS.* Copy condition r=.016 F(1,1) = 0.016 *NS.* Real-object condition r=.75 F(1,1)= 3.0 *NS*]. With all these data, the small n is a factor to be considered in the light of EW's performance variability. These results should not, therefore, be *over*-interpreted.

10.5 Task break-down activities.

EW, as a lover of music, wanted to independently place tapes into a tape cassette recorder. This was capitalised upon and a task break-down was completed comprising nine stages with a devised scoring system;

1. Reach for the tape box.

2. Open box.

3. Take out tape.

4. Open cassette player.

5. Turn tape upside down (**additional instructions:* look for the brown tape / make sure the edge marked 'top' is at the top / turn tape so arrow is pointing downwards).

6. Move tape towards cassette player.

7. Put tape into slot (**additional instructions :* put tape between red marks).

8. Close the cassette player.

9. Press the 'play' button.

The scoring system consisted of: 3 = physical prompt (alone or with verbal prompt), 2= gestural prompt, 1 = verbal prompt, 0 = without prompting. Three or four repetitions of the task were completed at each therapy session. After the second therapy session an additional instruction was inserted into stage 5 when it became clear that EW was unable to turn the tape upside down before sliding it into the cassette player. She was asked to look for the brown tape visible in the cassette. When this did not seem to help, a label was placed on the cassette with 'TOP' written on it clearly at the start of the fourth therapy session. The instruction was changed to "make sure the edge marked 'top' is at the top". When it became clear that yet further help was needed in this stage of the task (in session 5) the cassette was modified again with a large yellow arrow and the instruction "turn tape so the arrow is pointing downwards". At the start of session four, modification was made to the

cassette player itself. Red marks were placed, as a visual prompt, to indicate where the tape had to be inserted. As can be seen in Table 10.8 steps 1-3 were shown to be redundant. The first therapy session provided the main learning with the second session (after a weekend) showing that the learning had been retained. EW rapidly

Table 10.8 EW - learning to place a tape in a cassette player.

(Scoring: 3 = physical prompt, 2= gestural prompt, 1 = verbal prompt, 0 = without prompting)

** additional instruction inserted.*

session		Steps								
Date	**Repeats**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**
4/10	1	0	0	0	3	3	0	3	2	0
Session 1	2	0	0	0	0	3	0	3	0	0
	3	1	0	0	1	3	0	3	0	0
	4	0	0	0	0	3	0	3	0	1
7/10	1	0	0	0	0	3	0	3	0	0
Session 2	2	0	0	0	0	3	0	0	0	0
	3	0	0	0	0	3	0	3	0	0
8/10	1	0	0	0	0	3*	0	3	0	0
Session 3	2	0	0	0	0	3*	0	3	0	0
	3	0	0	0	0	3*	0	2	0	0
	4	0	0	0	0	3*	0	3	0	0
11/10	1	0	0	0	0	3*	0	0*	0	0
Session 4	2	0	0	0	0	3*	0	0*	0	0
	3	0	0	0	0	3*	0	0*	0	0
	4	0	0	0	0	3*	0	0*	0	0
14/10	1	0	0	0	0	3*	0	0	0	0
Session 5	2	0	0	0	0	3*	0	0	0	0
	3	0	0	0	0	3*	0	0	0	0
23/10	1	0	0	0	0	3	0	0	0	0
Session 6	2	0	0	0	0	3	0	0	0	0
	3	0	0	0	0	3	0	0	0	0
	4	0	0	0	0	3	0	0	0	0
	5	0	0	0	0	3	0	0	0	0

improved in step seven after the change in instruction ('put tape between red marks') in session four. What remained unchanged, despite alterations to the instructions, was EW's ability to place the tape in the correct orientation to insert it into the slot of the cassette recorder.

This element of the task was, therefore, practiced in isolation (Table 10.8). Firstly, three steps (A) were practiced (using the same scoring system as before): 1. Take the cassette from the therapist's hand. 2. Turn the tape over. 3. Place the tape into the cassette player.

When these had been completed satisfactorily four or five times, then another three steps (B) were practiced: 1. Take tape from box. 2. Turn the tape over. 3. Place the tape into the cassette player. Table 10.9 indicated how, initially, the second task was more problematic than the first. Eventually a successful outcome was achieved by session five. For the following two weeks EW was observed using her tape cassette recorder independently. During re-assessment in session six, however, there was some evidence of deterioration in performance. This might be attributed to the assessment setting and constraints of the assessment procedure, or it might have been a manifestation of the anxiety provoked by assessment.

Table 10.9 EW - learning to correctly orientate the tape cassette.

(Scoring: 3 = physical prompt, 2= gestural prompt, 1 = verbal prompt, 0 = without prompting)

		Steps (A)			Steps (B)		
Date	**Repeat**	**1**	**2**	**3**	**1**	**2**	**3**
24/10	1	0	0	3	0	3	3
(Session 1)	2	0	0	3	0	3	2
	3	0	1	2	0	2	3
	4	0	0	0	0	2	0
	5	0	1	0	0	2	3
25/10	1	0	1	0	0	3	0
(session 2)	2	0	0	0	0	3	0
	3	0	0	0	0	2	0
	4	0	0	0	0	2	0
26/10	1	0	2	0	0	2	3
(session 3)	2	0	2	3	0	2	3
	3	0	0	0	0	2	3
	4				0	2	3
29/10	1	0	0	1	0	1	0
(session 4)	2	0	0	0	0	1	0
	3	0	0	1	0	0	0
	4	0	0	1	0	0	0
	5				0	0	0
	6				0	1	0
30/10	1	0	0	0	0	2	3
(session 5)	2	0	1	0	0	2	0
	3	0	1	0	0	0	0
	4	0	1	0	0	0	0
	5	0	0	3	0	0	0
14/11	1				0	0	0
(session 6)	2				0	0	2
	3				0	0	2
	4				0	0	0

A similar task break-down strategy was employed for a self-care task. The first task attempted was putting on pants. The same scoring system was used. Ten steps were devised :

1. Open drawer

2. Select pants.

3. Sit down, pants on lap.

4. Hands into pants.

5. Bend down to feet.

6. One leg in - pause.

7. Other leg in - check.

8. Pull pants up to knees.

9. Stand up.

10. Pull pants up to waist.

Sessions one to five (Table 10.10) were in the baseline phase of the investigation, sessions six to ten during the intervention phase, and sessions eleven to fifteen in the post-intervention phase. Table 10.10 showed that EW's performance ability for putting on her pants was variable, with 'good' performance days when minimal

Table 10.10 EW learning to put on her pants.

(Scoring: 3 = physical prompt, 2= gestural prompt, 1 = verbal prompt, 0 = without prompting)

	STEPS									
Session	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
1	1	0	3	0	0	0	0	0	0	0
2	0	0	3	0	0	1	1	0	0	0
3	2	0	3	0	0	1	0	0	0	0
4	0	0	0	0	0	1	0	0	0	0
5	0	0	0	0	0	0	3	0	0	0
6	0	1	0	0	0	0	0	0	0	0
7	3	2	0	0	0	1	1	0	0	0
8	0	0	3	2	1	3	3	0	0	0
9	0	0	1	0	0	0	0	0	0	0
10	0	1	1	0	0	0	1	0	0	0
11	1	1	1	0	0	0	0	2	0	0
12	0	0	1	0	0	0	2	0	0	0
13	1	2	3	0	0	1	1	0	0	0
14	1	2	2	2	0	3	3	0	1	1
15	0	0	1	0	0	0	1	0	0	0

verbal prompts were required and 'poor' days when many physical or gestural prompts were needed to complete the task successfully. Session eight in the intervention phase was noted to be a particularly 'poor performance' day. As was clear in Table 10.10 more prompts of all kinds were required in the post-intervention phase after EW's month-long Christmas break which suggested a deterioration in performance ability consistent with other data presented.

10.6 Discussion

This study aimed to corroborate previous single case results concerning the efficacy or otherwise of a sensory stimulation protocol in apraxia with a person who had both ideomotor and ideational apraxia. Evidence from the outcome measures used in this

single case (NHPT and maze task) suggested a degree of improved performance in the intervention phase of the experiment not only from an improved task time, but also a reduction in variability and a more accurate tracing around the spiral maze. EW was performing largely at, or quicker than, the standard norm for the NHPT and maze task, so differences between phases of the experiment might be considered surprising. However, EW was younger than the population used in standardisation procedures for the CAPE from which the maze task was extracted, and so the given norms were much slower than seemed to apply to her performance. In addition EW was rather impulsive in her actions and so her faster completion time for both tasks would have been interacting with this factor in her performance.

No *immediate* effect of the sensory stimulation procedure was found in EW, which supported the findings from DC (chapter 9) but not from GP (chapter 8). This result might be explained by the intact sensory system (tactile modalities) in both EW and DC, whereas GP had a sensory mis-location condition which indicted disruption to the normal sensory input. This factor could be fundamental in explaining the differences between the three cases.

The clinical apraxia assessments also demonstrated small improvement in the intervention period of the investigation. The change noted in the intervention period might be seen as particularly potent in the apraxia tests as they can only monitor large changes in gestural performance due to their category of scoring. A deterioration in performance was noted in the post-intervention phase in three of the four apraxia assessments and was similar to the NHPT data which also showed this decline. It should be noted, however, that only a single clinical assessment was completed for

each of the ABA phases of the investigation in order to avoid repetition and practice effects. Given the variability of performance in EW (and possibly in all people with apraxia) these clinical assessment scores, as a single measure of performance, must be viewed with caution. Their reliability in stating a ***general*** level of performance for each phase could be considered suspect.

The kinematic data provided evidence consistent with the other outcome measures. Some moderate improvement in the intervention phase of the study might be interpreted from the visual analysis of the completion time data, though the small data set makes any conclusion open to criticism. Small improvements might also be interpreted from the trajectory patterns demonstrated by EW in consistency of spatial pathways during the drinking task in the intervention phase, though these really were so slight as to be open to reasonable challenge. The gestural performance recordings (HAMMER, CARVE, WIND) indicated some modification and improvement in the intervention phase of the investigation. Some inaccuracies and errors were still evident however. An increase in performance errors was noted in the post-intervention phase, though this was variable. Thus the kinematic data added weight to the suggestion that some improved motor performance had been elicited during the intervention phase of the study, but whether this could be attributed to the sensory stimulation procedure is open to contention. What was clear, however, was the poorer performance across the majority of outcome measures in the post-intervention phase which seemed to be deteriorating as time went on. This might be partly attributable to the four-week holiday break taken shortly after starting the post-intervention phase. It was possible that during this period EW was not practicing her self-care and other activities, but

was more passively 'cared for' during her stay with relatives. Alternatively the deteriorating performance might have been a manifestation of an emotional depressive state at being back in the rehabilitation centre and away from her family. The extent to which the depressive state of EW contributed to the post-intervention phase deterioration in performance ability can only be a matter for speculation. Another alternative explanation might be that the sensory stimulation aided EW's attention and concentration in tasks, and helped to alert her to what she was doing thus producing a better performance in the intervention phase. This is the first data available from a post-intervention period obtained in the study and so the fall-off in motor performance when firstly no therapy was available (when EW was away on holiday) and then when general leisure activities and self-care training were the therapeutic input is of interest. If attention is a key component to ideational apraxia, then this aspect of the sensory stimulation procedure might be worth pursuing.

It was noted that EW showed a normal kinematic profile in her velocity curve analysis similar to the findings of Hermsdorfer et al (1996) and the group study data (chapter 5) which indicated that some people with apraxia maintained some normal elements of kinematic output. As EW had ideational apraxia as well as ideomotor components, it is possible that Hermsdorfer's suggestion of a strategy of compensation that allows a normal temporally-executed movement when there is an unawareness of errors (as in ideational apraxia) would explain EW's kinematic profile. Her impulsivity in action could also have resulted in the temporal components of the movement to retain their normal output. Some interruption in the *spatial* components of movement were seen and observed in the gestural recordings taken, and this mirrors the findings of the case

of DC where a normal temporal component with disrupted spatial movement components were found. Another similarity with DC is that, for EW, a relationship was found between clinical tests and timing of movement performance. Whilst in DC the De Renzi test showed an association with the drinking task completion time, in EW it was test 1 (Haaland & Flaherty 1982) that held the relationship. There was a question in the DC case of a possible mild ideational component to the apraxia and EW had a fairly severe ideational apraxia. These two cases, therefore could be reflecting some similar sub-type of apraxia that allows an *association* between the clinical tests and the temporal component of motor output. Such a relationship could be concerned with the strategy for coping adopted by different apraxic sub-types suggested by Hermsdorfer and colleagues. An interaction between this and the language components of each case's condition might be a possibility also, with disruption to semantic-action routes and well as vision-to-action routes.

Variability of performance was a feature in all the outcome measures used in this single case investigation. The data from the NHPT and maze tasks illustrated this most keenly, being measures which lent themselves to the multiple assessment points suggested in the previous chapter. Although the kinematic data did not provide as many data points as had been planned, variability within each of the experimental phases was evident. Similarly, in a naturalistic setting, the functional tasks monitored through task break-down strategies also indicated performance variability. No direct evidence was found to account for the daily performance changes, though EW was noted to be very distractible, had attentional deficits and appeared at times to be somewhat anxious. These factors could have contributed to performance variability

throughout the investigation. Mood was also noted to be variable. A psychiatric assessment carried out during the post-intervention phase concluded that EW had a depressive condition coupled with an increased awareness of her deficits. Such depression could account for the performance decline noted in most measures in the post-intervention phase of the investigation.

The task break-down strategies were used to moderate effect with EW. The data from the tape cassette player activity demonstrated the success of taking out an element of the task when no progress was evident, then practicing it in isolation before reintegrating it into the whole activity. The task break-down strategy with self-care training, however, showed mixed effectiveness. Performance variability was the major feature. This was in marked contrast to the data from Wilson (1988) where rapid improvement was noted with task break-down and no variability was seen. These differences might be accounted for by the presence of a profound ideational as well as ideomotor apraxia in the present case. The Wilson case study, whilst not naming the condition, suggested the presence only of ideomotor apraxia.

To determine whether the sensory stimulation intervention affected self-care activities, the task breakdown data were examined by experimental phase. No discernible difference was noted in the number of prompts needed by EW in the intervention phase to complete the 'putting on pants' task. The sensory stimulation intervention did not show any effectiveness in enhanced motor performance in this functional task. It is possible that as the stimulation procedure was not completed on a daily basis, any therapeutic effects would, necessarily, be lessened. Further studies involving daily sensory intervention procedures coupled with functional training tasks would provide

more robust evidence on this particular issue. It might be feasible to adopt this more rigorous stimulation strategy by using an electronic device like the TENS machine (Transcutaneous Electrical Neuromuscular Stimulation; Low & Reed 1994, Kitchen & Bazin 1995) to determine whether more regular and consistent sensory stimulation would have any effect on task performance. This electronic stimulator sends a medium frequency electrical current through the part of the body, though eutrophic stimulation at sub-contraction level could be considered a better strategy to provide the sensory bombardment required for functional benefits (Low & Reed 1994, Kitchen & Bazin 1995). The post-intervention data suggested a deterioration in self-care ability with an increased number of physical prompts needed to complete the 'putting on pants' task. This concurred with the post-intervention data from all the other outcome measures and suggested a general decline in overall performance ability.

10.7 Conclusion

This study provided some tentative evidence concerning the efficacy of a sensory stimulation protocol as a therapeutic intervention for apraxia. Small differences in motor performance between the baseline and the intervention phases could have been interpreted from the data collected from two timed tasks and also in kinematic recording data. Improvement was also seen in apraxia assessment scores during the intervention phase, though only a single clinical assessment was made in each experimental phase and therefore these results must be viewed with caution. However the improvements across the range of outcome measures were not sustained in the

post-intervention phase of the investigation. The presence of depression in EW, severe enough to require medication, was a factor which might have confounded results.

The sensory stimulation procedure in this case of ideational and ideomotor apraxia showed no *immediate* effect on praxic ability. This might be attributable to the presence of *ideational* as well as ideomotor apraxia in EW, providing a more intractable condition than that of ideomotor apraxia alone. Alternatively the presence of an attention deficit might have contributed to the performance results. The kinematic analysis confirmed some of the previous data regarding spatial configurations in apraxic movement and loss of consistency of movement performance. EW was noted to have maintained a generally normal velocity profile during movement which was consistent with previous research findings in the group study, the single case DC and by Hermsdorfer et al (1996). The explanation for normal temporal control of movement with loss of spatial control has been explained by the non-awareness of deficits in ideational apraxia leading to adopting a particular type of strategy in attempting to perform gestures and movements. EW, being unaware of her errors and deficits would impulsively perform movements to normal temporal standards but with loss of spatial components of the movement, and such evidence adds weight to support the Hermsdorfer hypothesis..

An association between clinical tests and timing of movement was found in this study, though the small data set precludes too much reliance being placed on such results. However, such associations when taken with the results from DC case (albeit with different clinical tests showing the association) might suggest the existence of a 'sub-type' of apraxia relating to ideational components of the condition interacting with

certain cognitive components. No such relationship was found in the group study, though, in terms of differences between those with ideational and ideomotor apraxia and performance in the drinking task. The presence of a language deficit in EW might also have been a contributing feature to the association between the clinical test requiring responses to verbal and visual instruction (Haaland & Flaherty 1982) and the kinematics of the drinking task though, again, such an interaction was not demonstrated in either the group study or the MT case.

This single case study provided additional evidence of variability of motor performance and added weight to the notion that this phenomenon may be a feature of the apraxic condition. The variability was evident in the range of measures used in this project; the timed tasks, the kinematic recordings and also in the functional tasks. This case also provided evidence of a limited effectiveness of a task break-down strategy in self-care and functional activities which was not, though, as dramatic as other research had indicated (Wilson 1988). Nevertheless, it suggests that a task break-down strategy has potential for rehabilitation and could be appropriate for people with both ideational and ideomotor apraxia. Further research is needed to substantiate the limited evidence to date.

No enhancement of functional performance, nor of generalisation between tasks and activities was noted in the intervention phase of the investigation. Sensory stimulation intervention did not appear to have the umbrella effect on all aspects of rehabilitation that has been predicted by sensory integration researchers (Ayres 1985, Cermak 1985, Croce 1993). Several methodological flaws have been noted in this investigation, however, which might have accounted for such findings. Extension of this experiment

would be useful, using single cases with different underlying pathologies and severity of apraxia, to gather further evidence regarding the effectiveness a sensory stimulation intervention in apraxia using a range of outcome measures including functional activities.

Chapter 11. CASE 6: *PH. Head injury. Ideational and ideomotor apraxia.*

11.1 Case Background.

PH, a 66 year old man, sustained a severe head injury after being knocked from his bicycle. He had a complex skull fracture and a left hemisphere subdural haematoma with cerebral contusion. After neurosurgery he required drainage of a mild hydrocephalus. His Post Traumatic Amnesia period was recorded as being between 6-8 weeks.

The first assessment for apraxia was carried out when PH was in a local cottage hospital four months post trauma. This was prior to his admission to the rehabilitation centre. When first assessed, PH was ambulant, had mild weakness of his right hand, and was mostly independent in self care. He had a fluent dysphasia with a virtually unstoppable flow of speech, plus a limited attention span. Many assessment requests had to be repeated as PH was unable to hold, or recall,

Table 11.1 PH - Clinical assessment scores at first examination.

Clinical Tests	Left Hand
Test 1- gesture copying (max. = 40) (Appendix D2)	32 *(80%)*
Test 2 - object use test (max. = 36) (Appendix D3)	20 *(55.5%)*
Test 5 - DeRenzi test, gesture copying (max. = 72) (Appendix D1)	30 *(41.67%)*
Mean % scores for Tests 1, 2 & 5.	59.06%
Test 3 - ideational apraxia (max. = 12) (Appendix D4)	0 *(0%)*
Test 4 - agnosia (max. = 20) (Appendix E)	17 *(85%)*

information. He displayed no insight into his difficulties. PH was assessed using his left, non-hemiparetic (non-dominant) upper limb. During the apraxia examination he displayed errors of positioning of the fingers and hand, incorrect plane of movement, verbalisation of movement rather than doing the action, poor sequencing and timing of movement, inappropriate object pantomimes and use of the body part as the object itself (BPO errors). Most errors in the 'object use' test (Appendix D3) were made in the verbal command and 'copy' conditions though some errors were also made in the handling of the actual objects themselves. These errors were consistent with the presence of ideomotor apraxia (Table 11.1). PH was unable to complete any items in the test for ***ideational*** apraxia. He displayed excessive and prolonged verbalisation of the tasks without actual performance. This inability to use objects appropriately in a sequence and the unawareness of errors displayed by PH indicated a severe *ideational* apraxia in addition to the ideomotor apraxia. There was no evidence for a visual agnosia. All the normal ethical guidelines for informed consent were followed and PH agreed to participate in the research project.

11.2 Aims of the study

Using this single case methodology the study aimed to :

- replicate the previous case studies and evaluate the effectiveness of a sensory stimulation intervention protocol on movement performance in a person with severe ideational *and* ideomotor apraxia.
- compare effectiveness of verbal and visual mediation strategies when used alone and when used *with* sensory stimulation procedures.

- compare kinematic recording data of a person with both ideational and ideomotor apraxia following head injury with previous single case data from people with apraxia from other underlying pathologies.
- examine the variability of motor performance in a person with ideational *and* ideomotor apraxia following head injury.

Following the previous single case investigations the following hypotheses were tested :

1. The addition of a sensory stimulation protocol to verbal and visual mediation strategies would enhance motor performance in the upper limbs as measured by specific timed tasks.
2. Variability of performance in given tasks would be demonstrated across experimental phases, but shown to be reduced in the intervention phase.
3. Kinematic analysis of movement during a drinking task and gestural performances would demonstrate loss of spatial consistency, over-use of shoulder movements and abnormal velocity patterns in a person with ideational and ideomotor apraxia. These features would be reduced in the sensory intervention phase of the investigation.

11.3 Procedure

Using an ABA design this single case project was carried out *in addition* to PH's normal daily physiotherapy and occupational therapy sessions. His occupational therapy intervention, outside the research project, concentrated on functional activities in the kitchen and workshop. Physiotherapy sessions involved balance and

stamina-building activities. PH also participated in speech and language therapy. The baseline and intervention periods of the project lasted three weeks each. A total of 13 data collection points were achieved for each phase. The post-intervention period lasted for two weeks with nine data collection points completed before PH was discharged home from the rehabilitation centre. Throughout the eleven week project, the research sessions were held at the same time daily, and in the same location in the rehabilitation centre. This was an attempt to eliminate timing and environmental variables from the investigation.

The baseline phase involved a protocol using verbal and visual mediation strategies for hand and arm movements. PH was seated at a table with the researcher opposite. The protocol involved three types of activity:

1] Touching targets

PH was asked to move either the left or right hand (randomly) to touch a draughts counter. The therapist moved the counter around on the table between each 'hit'. PH was asked to move as quickly and as accurately as possible to touch the target disc.

2] Gesture copying.

Simple, *single* gestures were demonstrated by the therapist (e.g. make a fist, point finger, 'thumbs up'). PH was asked to copy the gesture. Corrections and physical assistance were given when necessary. Bilateral movements were then used. PH was asked to copy the movement (e.g. clap hands, drum fingers together, make a 'cat's cradle'). Again, feedback and physical corrections were given as a part of the training procedure.

3] Verbalising hand position or movement.

Simple gestures and movements were used. PH was asked to verbalise his performance. (e.g. "I am pointing my finger", " I am clapping my hands", "I am stretching out my arm").

Outcome measures were taken immediately before and immediately after each session. The intervention phase of the project involved the identical activities given in the baseline phase *plus* the addition of the sensory stimulation protocol described previously (Chapter 8). The verbal and visual mediation activities had a duration of 15 minutes and the sensory stimulation procedures another 20 minutes. This was designed to fit into the rehabilitation centre timetable. Measurements (see 11.3i) were taken before and after each session. The post-intervention phase of the project repeated the baseline phase protocol. Parallel to these daily sessions, kinematic recordings were taken weekly. An apraxia assessment was completed at the start of each experimental phase and at the end of the project.

11.3 i] Outcome measures.

Measurements were taken pre and post each therapy session in all ABA phases using three tasks: Nine Hole Peg Test (NHPT; Mathiowetz et al 1985), Gibson Spiral Maze from the CAPE (Pattie & Gilleard, 1979) and a bilateral nut and bolt test (non-standardised). These tests replicated, in part, the previous single case investigations and were chosen for being quick, reliable, available in most occupational therapy departments, reproducible, and for providing quantitative scores. The nut and bolt test was devised as a bilateral measure of motor performance. PH was requested to

unscrew a wing nut from a long bolt as quickly as possible, then to replace the nut fully onto the bolt. The outcome measure for this test was the time taken to complete each part.

11.3 ii] Kinematic recordings

During this investigation, weekly kinematic recordings were made of the drinking task. Gestural performance (HAMMER, CARVE, WIND) was also recorded in two modality conditions; verbal command and visual/copy condition. These recording sessions were carried out on the same day and at the same time on a weekly basis, and in the same environment on each occasion. The left upper limb (ipsilateral to the lesion) was used for all kinematic measures.

11.4 Results

11.4 i] Outcome measures

Descriptive analysis was completed across ABA phases for each of the outcome measures (Table 11.2). Left upper limb motor performance (as measured by the NHPT, number of maze errors and the 'off' time for the nut and bolt task) showed a reduced mean time, lower standard deviation scores and lower inter-quartile range in the intervention period. These data might suggest improved performances in these areas. The left hand performance measured through the time taken to complete the maze task and the nut and bolt task 'on' phase, though, indicated larger standard deviation scores and interquartile ranges which could be indicative of a more erratic performance during the intervention phase of the study. The post-intervention phase

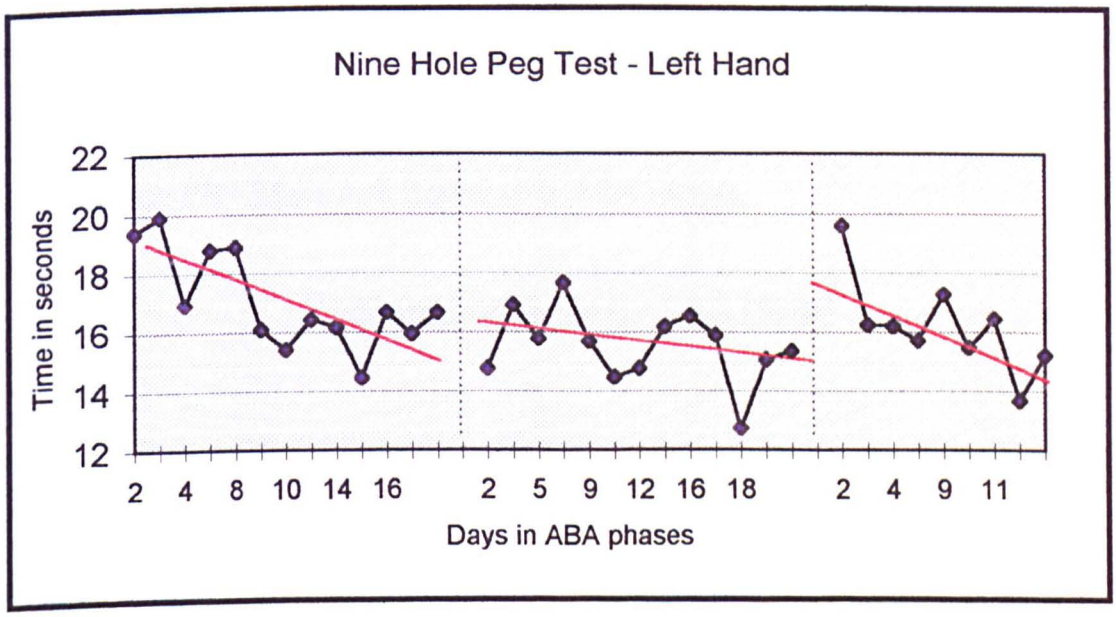
Table 11.2 PH - synopsis of outcome measures data from ABA phases

	Baseline	Intervention	Post-intervention
NHPT (in seconds) *(norm = 18 secs.)*			
Mean score	17.06	15.52	16.21
Range	14.47 - 19.91	12.75 - 17.66	13.69 - 19.59
Broadened median	**16.57**	**15.55**	**15.89**
Standard deviation	1.66	1.23	1.6
Inter-quartile range	2.84	1.58	1.52
No. of data points	13	13	9
Maze time. (in seconds) *(norm = 106 secs.)*			
Mean score	65.53	60.91	58.65
Range	56.5 - 71.72	53.28 - 85.75	50.66 - 69.28
Broadened median	**65.53**	**59.56**	**57.66**
Standard deviation	4.74	8.35	5.78
Inter-quartile range	7.53	7.56	8.42
No. of data points	13	13	9
Maze errors			
Mean score	23.46	16.85	15.11
Range	12 - 39	11 - 24	10 - 22
Broadened median	**23.8**	**17.20**	**14.19**
Standard deviation	7.28	4.12	4.4
Inter-quartile range	11	6.5	7.5
No. of data points	13	13	9
Bilateral nut & bolt task (in seconds) **Off**			
Mean score	19.38	17.8	16.69
Range	16.81 - 24.69	13.78 - 23.91	13.41 - 20.63
Broadened median	**18.47**	**17.47**	**16.60**
Standard deviation	2.43	2.48	2.28
Inter-quartile range	3.94	2.72	3.6
No. of data points	13	13	9
Bilateral nut & bolt task (in seconds) **On**			
Mean score	18.32	18.59	16.5
Range	14.83 - 22.16	13.56 - 23.35	10.59 - 21.43
Broadened median	**18.22**	**18.82**	**16.71**
Standard deviation	2.74	3.32	3.06
Inter-quartile range	5.25	6.02	3.55
No. of data points	13	13	9

data showed a mixed picture too, with a poorer performance in the NHPT as shown by the increased mean and standard deviation scores, yet improved average timing in the maze task and both parts of the nut and bolt task (Table 11.2). A reduction in the mean number of errors in the maze task was also noted in the post-intervention phase. Visual plotting of these data was completed.

Visual analysis of the NHPT data demonstrated wide daily variability in PH's performance across all phases of the study. A trend of improvement in speed of

Figure 11.1 PH - NHPT timing over ABA phases.

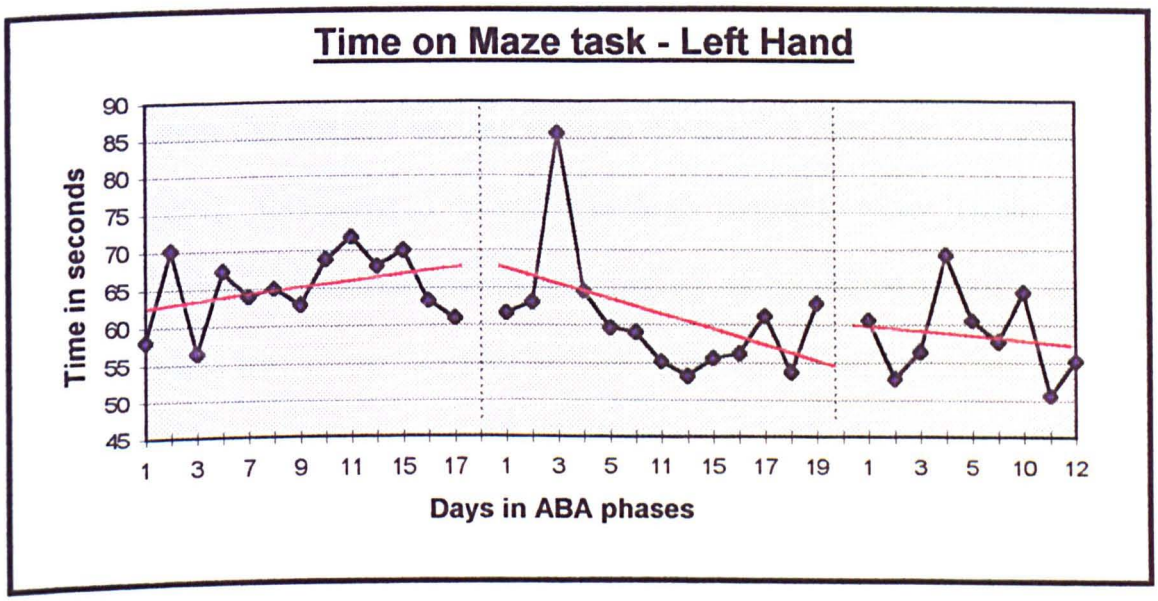


performance was noted in the baseline phase (Figure 11.1), with the regression line predicting 51.3% of the variance [$F(1,11) = 11.58$ $p < .01$]. This improvement was possibly attributable to a learning effect as PH became more familiar with the task. The intervention phase showed an erratic performance with the regression analysis only accounting for 12.3% of the variance [$F(1,11) = 1.54$ *NS*) so the slight trend seen

in the graph should be viewed with caution. The post-intervention phase could be interpreted as following along similar lines to the baseline phase in terms of the slope of the data, and regression analysis confirmed that 46.9% of the variance was accounted for [$F(1,7) = 6.19$ $p < .05$]. These data are the first which could suggest that the sensory stimulation procedure could actually have a detrimental effect on the *temporal* aspects of performance, though examination of the *spatial* aspects might reveal a different story.

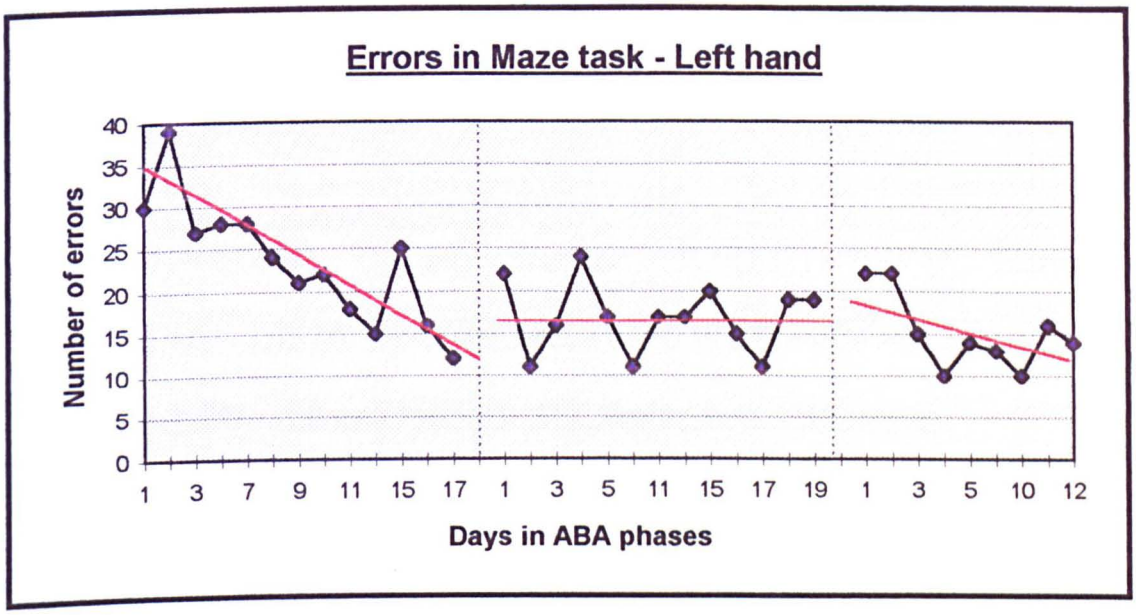
The time taken to complete the maze task (Figure 11.2) showed a trend of worsening performance in the baseline phase, though the slope of the regression line only accounted for 6.1% of the variance [$F(1,11) = 0.72$ *NS*]. The intervention phase, apart from the 'outlier' of the 3rd data point, showed a fairly stable performance day

Figure 11.2 PH - Maze task timing over ABA phases.



-by-day for the first part of the intervention phase with more erratic performance towards the end. The variable nature of the day-to-day performances returned in the post-intervention phase.

Figure 11.3 PH - Maze task errors over ABA phases.



The errors made by PH during completion of the maze task are illustrated in Figure 11.3 and show the dramatic learning effect in the baseline phase [r= .715 $F(1,11) = 27.66$ $p < .001$]. This aspect of the task was clearly subject to motor learning and an aspect of skilled performance which was unlikely to be related to PH's apraxia, although some part of the recovery process might have been interacting with the learning effect. As can been seen in the plotted data, once the learning effect had ceased, PH demonstrated great variability in his performance throughout the intervention phase [r= .005 $F(1,11) = 0.059$ *NS*]. This result mirrors that of DC (chapter 9) where variability of performance was made more apparent in the intervention phase of the experiment when learning effects had evened out. The post-

intervention phase suggested a similar pattern of performance to the intervention phase.

The nut and bolt task showed the most variability in performance of all the outcome measures. This applied to both 'off' and 'on' parts of the task. As can been seen in Figures 11.4 and 11.5 the regression lines have no real meaning to the data, accounting for minimal amounts of the variance seen ['Off' data :- Baseline r=.0004 $F(1,11) = 0.005$ *NS*. Intervention r=.005 $F(1,11) = 0.059$ *NS*. Post-intervention r= .005 $F(1,7) = 0.04$ *NS*. 'On' data :- Baseline r=.03 $F(1,11) = 0.35$ *NS*. Intervention r=.24 $F(1,11) = 3.56$ *NS*. Post-intervention r= .07 $F(1,7) = 0.54$ *NS*].

Figure 11.4 PH - Nut and bolt task 'Off' timing over ABA phases.

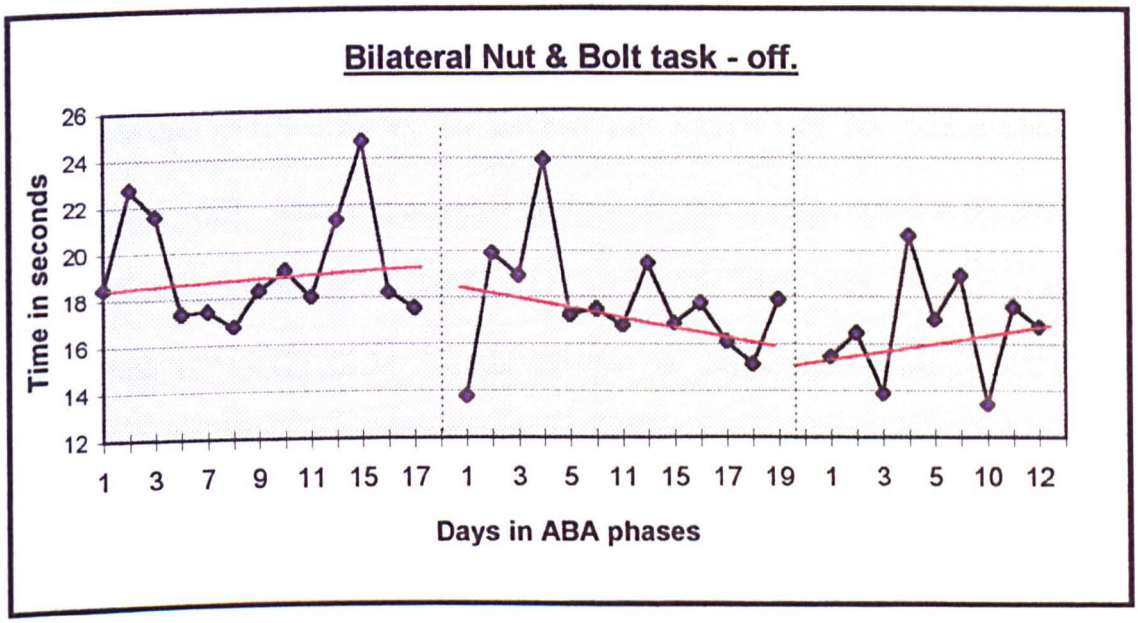
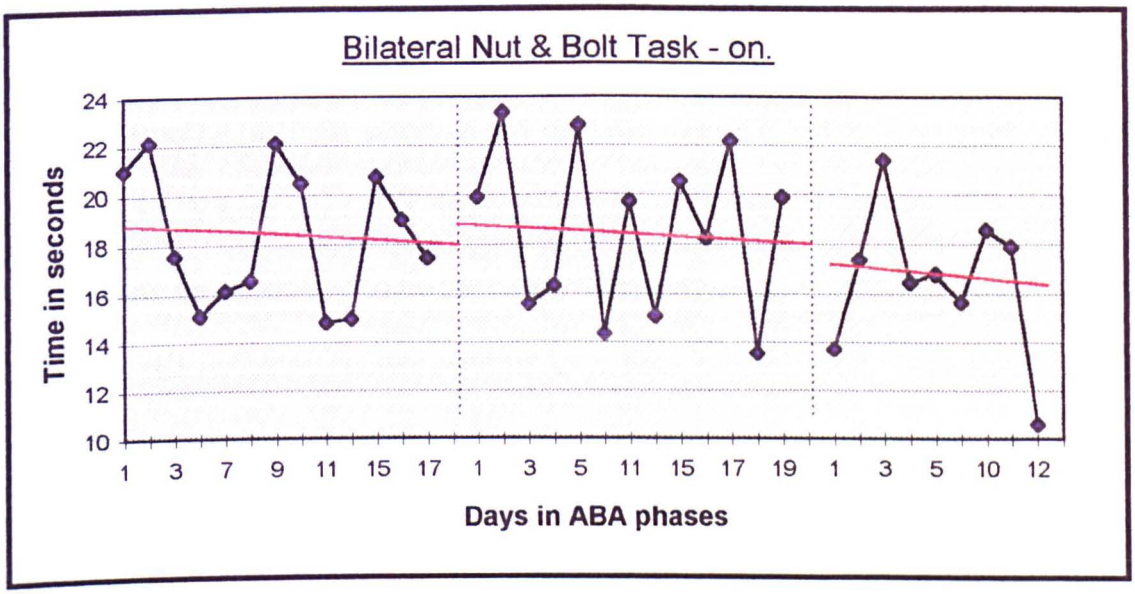


Figure 11.5 PH - Nut and bolt task 'On' timing over ABA phases.



Analysis of the data in the baseline phase to determine *immediate* effects of intervention (using paired t-test statistics) indicated no significant differences [NHPT $t(12) = -1.61$ *NS*, Maze time $t(12) = -0.64$ *NS*, nut and bolt 'off' $t(12) = 1.15$ *NS*, nut and bolt 'on' $t(12) = 2.11$ *NS*]. This was repeated in the intervention phase with no demonstrable effects of sensory stimulation [NHPT: $t(12) = 1.93$ *NS*, Maze time : $t(12) = -1.04$ *NS*, nut and bolt 'off' : $t(12) = 0.34$ *NS*, nut and bolt 'on' : $t(12) = 1.53$ *NS*]. This evidence demonstrated the lack of *immediate* treatment effect on motor performance of either the verbal and visual mediation strategy alone or with the addition of sensory stimulation.

11.4 ii] Apraxia test scores

During the experimental period of eleven weeks PH completed three apraxia batteries. The scores remained fairly constant throughout the investigation period (Table 11.3) although the percentage figures indicated a general deterioration in apraxic ability. The exception to this was in the test 5 scores (De Renzi et al 1980) which

demonstrated an improvement at the start of the intervention phase and again at the post-intervention phase. The De Renzi et al test was shown in previous analyses (chapter 4) to be the most sensitive test of those available and to have good internal consistency. Where there was disparity across test results, therefore, it is this test which might be considered to be the most reliable indicator of performance ability. In PH, then, this indicated an improvement over time in praxic skill but not necessarily related to the experimental phase. There was an increased use of the body part *as* the object (BPO errors) which resulted in a reduced score in the object-use test (appendix D3) in the post-intervention assessment. PH made movement and positioning errors, particularly in the plane of hand orientation, throughout the investigation. He also made many perseverative errors, and all assessments were accompanied by profuse

Table 11.3 PH - Apraxia assessment scores in ABA phases.

	Initial test	**Baseline 'A'**	**Intervention 'B'**	**Post- int. 'A'**
Test 1 - gesture copying (max. = 40) (appendix D2)	32 *(80%)*	31 *(77.5%)*	28 *(70%)*	27 *(67.5%)*
Test 2 - object use test (max. = 36) (appendix D3)	20 *(55.5%)*	29 *(80.5%)*	28 *(77.7%)*	15 *(41.6%)*
Test 3 -ideational apraxia (max. = 12) (appendix D4)	0 *(0%)*	0 *(0%)*	not tested	10 *(83.3%)*
Test 4 - agnosia (max 20. appendix E)	17 *(85%)*	18 *(90%)*	18 *(90%)*	16 *(80%)*
Test 5 - De Renzi test, gesture copy. (max.= 72) (appendix D1)	30 *(41.6%)*	28 *(38.8%)*	43 *(59.7%)*	47 *(65.3%)*

verbal commentary. The most noticeable difference in the apraxia assessments was in PH's ability to use the multiple objects in a meaningful sequence in the ideational

apraxia test. He changed from complete inability to perform any meaningful activity in the baseline phase to good and appropriate use of the objects in the post-intervention phase. This was accompanied by profuse vocal outflow, as at the start of the investigation. Only one apraxia assessment was made in each phase of the investigation. Given the performance variability seen in PH in other outcome measures, such single incidences of praxic performance might not reliably represent the *general* level of performance at each phase.

11.4 iii] Kinematic analysis

The drinking task requirements were sometimes difficult for PH to perform successfully. He made errors in omission of elements in the task and many perseverative errors. In consequence the computer software was not always able to analyse the performance efforts. A reduced number of recordings were processed, therefore, compared to those attempted. Four recordings were attempted in the baseline phase in three modality conditions; two were successful in the verbal command condition, three in the visual/copy condition, and two in the real-object-use condition. In the intervention phase a total of three recordings were made, this time successfully on all occasions for both the verbal command and visual/copy conditions and on two occasions for the real-object-use condition. This indicated an improvement in success rate of task completion during the intervention phase which was sustained in the post-intervention phase. Here, the two recordings attempted were successfully completed in the copy and 'real' conditions, with just one completed in the verbal command condition. Descriptive analysis of these small data

sets is set out in Table 11.4. The use of the broadened median, standard deviation and inter-quartile range scores with such small *n* was not appropriate in this instance. The small number of data points in this part of the study has reduced the veracity of the conclusions which might be drawn. Nevertheless, visual inspection of the plotted data has allowed some tentative suggestions regarding performance across ABA phases.

Table 11.4 PH - Synopsis of kinematic recording data in ABA phases.

(All scores given in seconds)

Drinking Task.	Baseline (N=4)	Intervention (N=3)	Post-intervention (N=2)
Total Time Verbal condition			
Mean score	7.11	9.25	7.90
Range	6.26 - 7.96	6.84 - 11.74	
No. of data points	2	3	1
Total Time Copy condition			
Mean score	7.17	8.13	8.18
Range	6.26 - 7.76	5.88 - 10.4	7.28 - 9.08
No. of data points	3	3	2
Total Time Real object condition			
Mean score	11.89	10.80	10.82
Range	10.46 - 13.32	9.78 - 11.82	9.8 - 11.84
No. of data points	2	2	2

The total time taken to complete the drinking task in the verbal condition indicated that, for the intervention phase, two of the three performances were slower than during the baseline (figure 11.6). The one data collection point in the post-intervention phase was at about the same speed of completion as the very first trial in the baseline. It is possible that the intervention was having an effect on the temporal aspect of movement performance perhaps by altering a 'strategy' adopted by PH in his movement output.

For the copy condition of the drinking task (Figure 11.7) again the intervention phase

Figure 11.6 PH - Total time (verbal condition) across ABA phases.

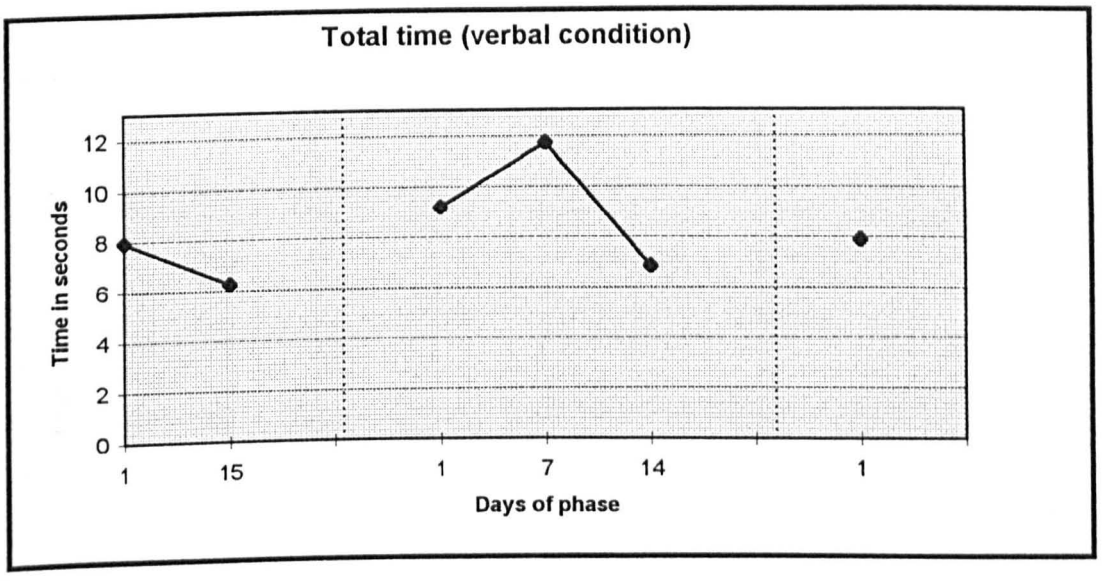
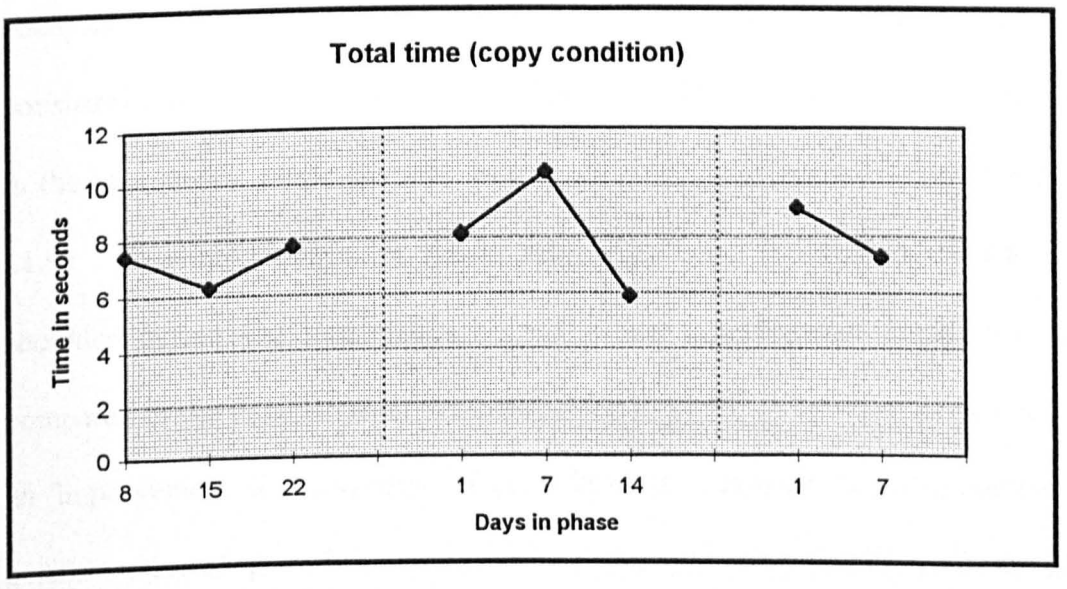


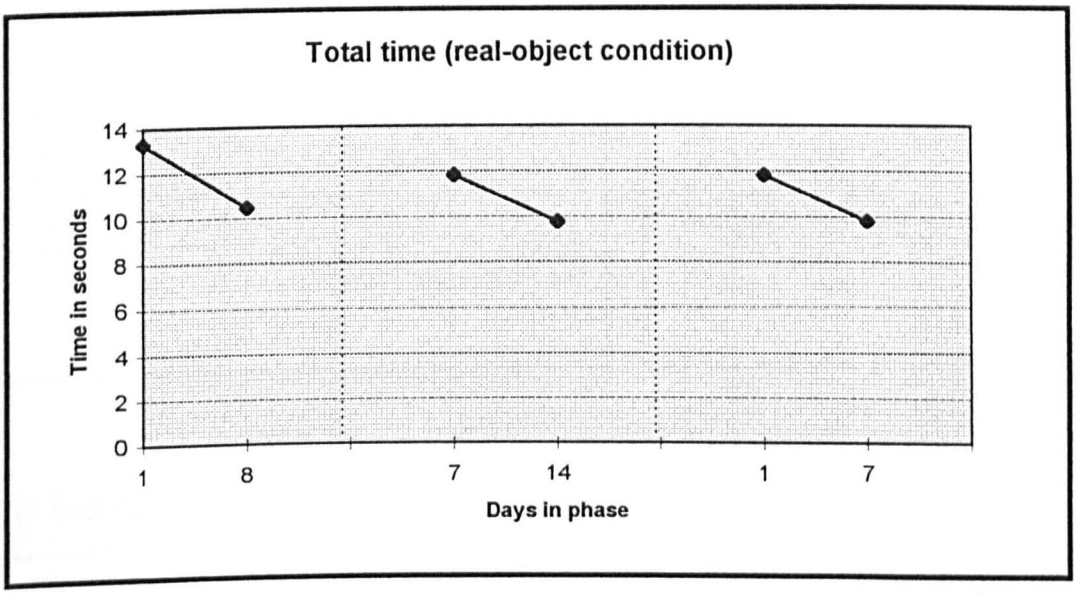
Figure 11.7 PH - Total time (copy condition) across ABA phases.



data appeared to hold a slower occasion of performance than at any other time of the study. No other trends could be seen from the data plots. This was also true of the real-object condition completion time in the drinking task (Figure 11.8) where only

two data points were available for each phase of the study and little, if anything, could be gleaned from them.

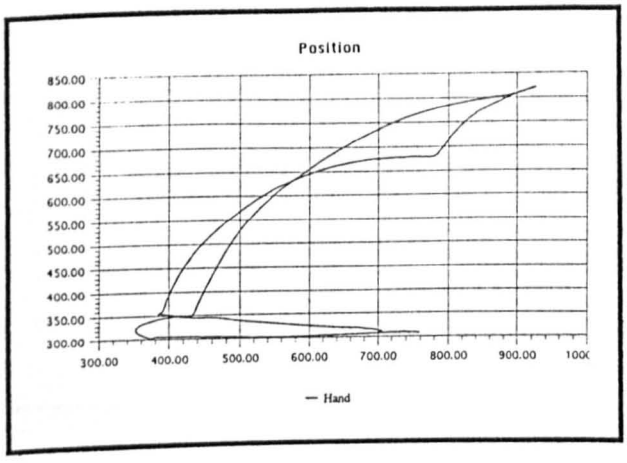
Figure 11.8 PH - Total time (real-object condition) across ABA phases.



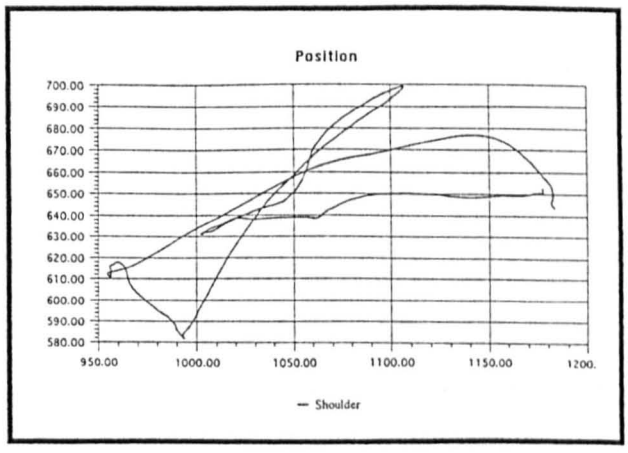
Analysis of the trajectories in the drinking task indicated some loss of spatial consistency in the hand trajectories in the baseline phase. This was shown to improve in the intervention phase and was better still in the post-intervention period (Figure 11.9). This improvement in spatial consistency was mirrored in the use of the shoulder during the intervention period where considerable change was noted compared to the baseline phase recording (Figure 11.9). These results demonstrated an improvement in movement performance, as measured by joint control and consistent use of space, but at the expense of (or as a result of?) longer time spent in completion of the task. Some perseverative features were noted in the verbal command condition throughout the investigation, but these were intermittent and appeared unrelated to experimental phase (Figure 11.10).

Figure 11.9 PH - Examples of hand and shoulder trajectories across ABA phases.

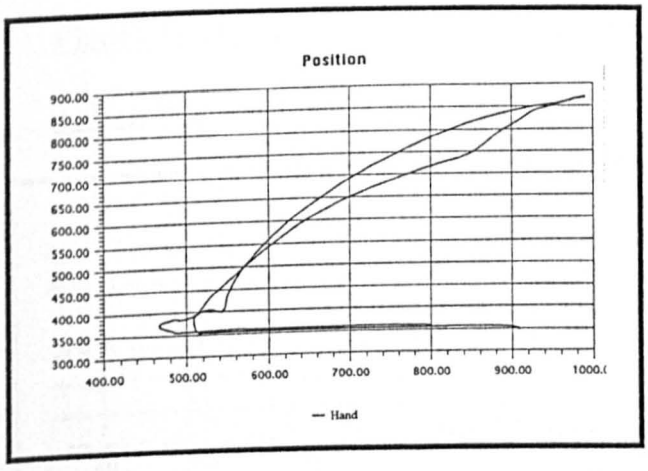
ai] Baseline - hand trajectory.



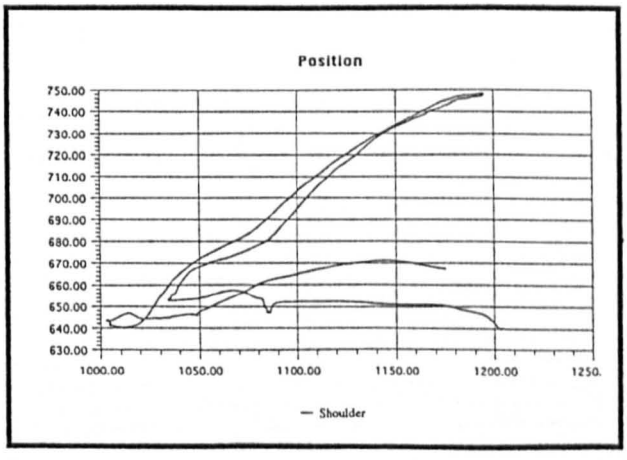
bi] Baseline - shoulder trajectory.



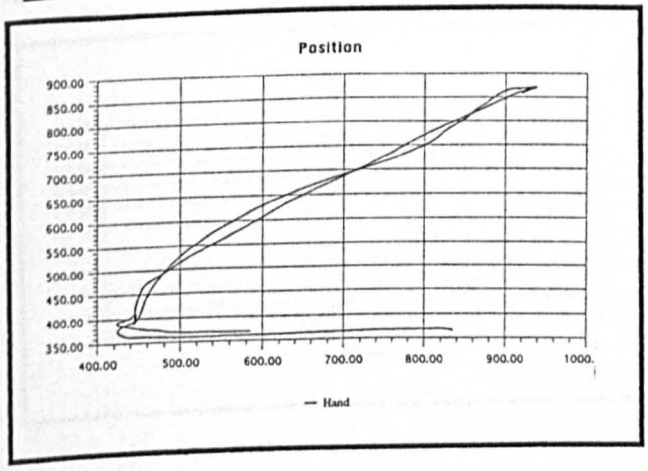
aii] Intervention - hand trajectory.



bii] Intervention - shoulder trajectory.



aiii] Post- Intervention - hand.



biii] Post-Intervention - shoulder

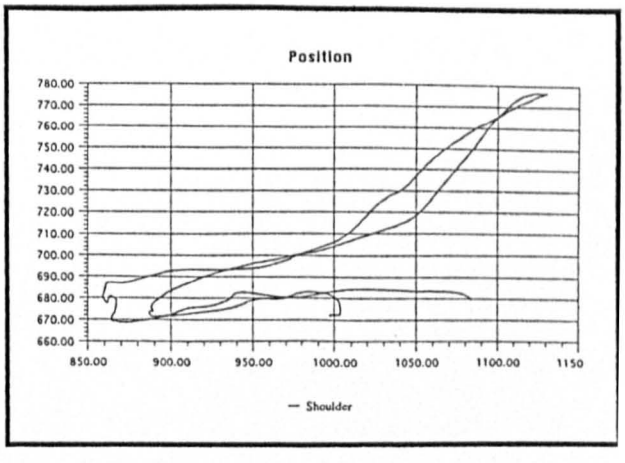
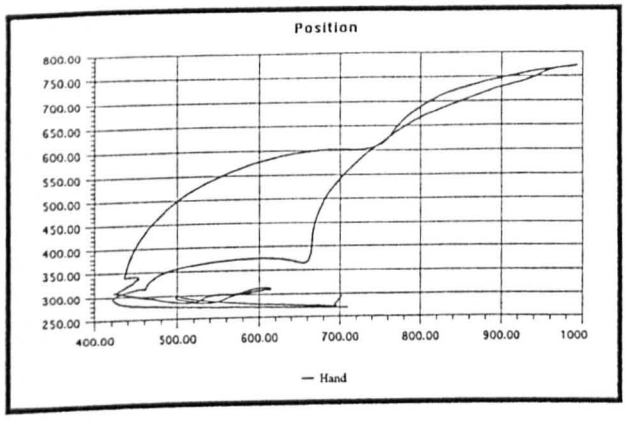
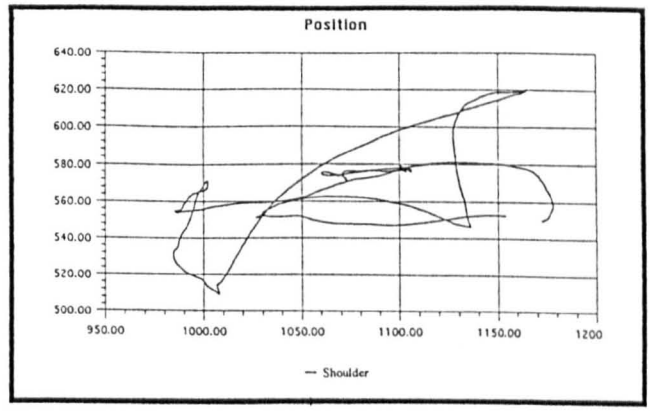


Figure 11.10 PH - Examples of perseverative errors in verbal command.

a] Hand trajectory



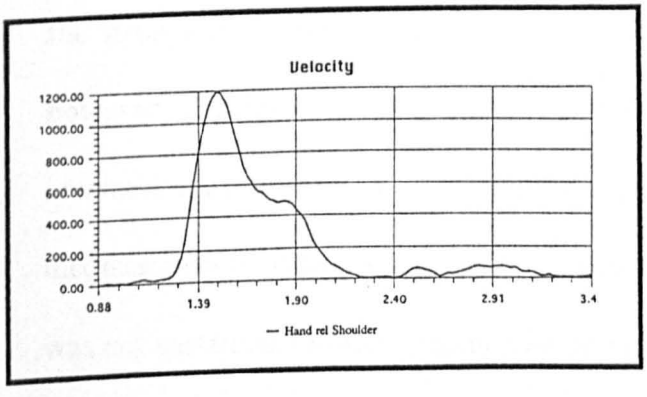
b] Shoulder trajectory



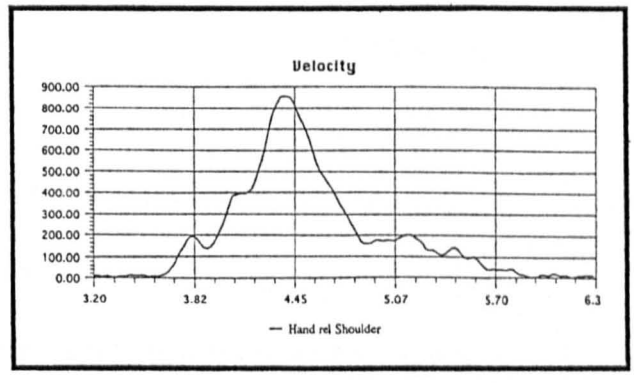
The velocity patterns exhibited by PH in the baseline phase were of sudden high peaks surrounded by very long low-velocity periods. Observations made at the time of the

Figure 11.11 PH - Examples of velocity peak recordings across ABA phases.

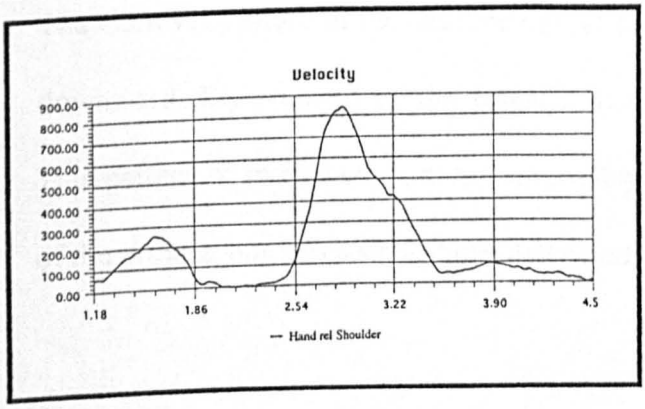
a] Baseline - 'lift' element.



b] Intervention phase - 'lift' element.



c] Post- Intervention phase - 'lift' element.



recordings noted the 'searching' behaviour and delay in starting movements at each element of the drinking task (reach, lift, down return). These velocity patterns were slightly modified in the intervention phase, as shown by the less extreme peaks, but still with long low-velocity periods (Figure 11.11). This was retained into the post-intervention phase. These changes in kinematic elements of movement performance would be consistent with a slower speed of movement performance, indicated by the total time taken in the drinking task, and might be attributable to a more controlled processing approach to achieving the desired movement in the intervention and post-intervention phases of the study.

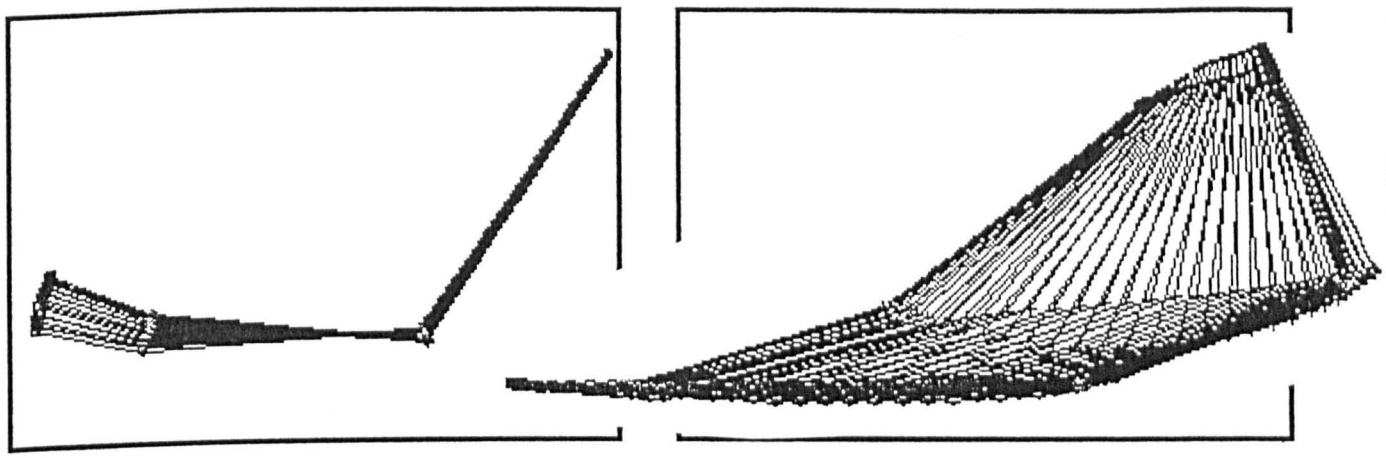
Gestural performance in the HAMMER, CARVE, and WIND recordings also showed change over time. The HAMMER gestures made in the baseline phase were very slow with minimal movement demonstrated. This was illustrated by the denseness of the stroboscopic representations (Figure 11.12). The intervention phase recordings, however, indicated a slightly quicker and more realistic gestural performance of a hammering action. A forward displacement of the repeated movement, as well as inconsistency of spatial alignment, was also apparent however (Figure 11.12). This was not sustained into the post-intervention phase where the recordings demonstrated a mixed/confused gesture half-way between HAMMER and CARVE.

The CARVE gesture in the baseline period showed an exaggerated movement with a downward displacement of the forearm in carrying out the action (Figure 11.12). This pattern of movement was not noticeably changed during the intervention phase of the project, but *was* seen to have deteriorated in the post-intervention recordings

Figure 11.12 PH - Examples of gestural performance in ABA phases.

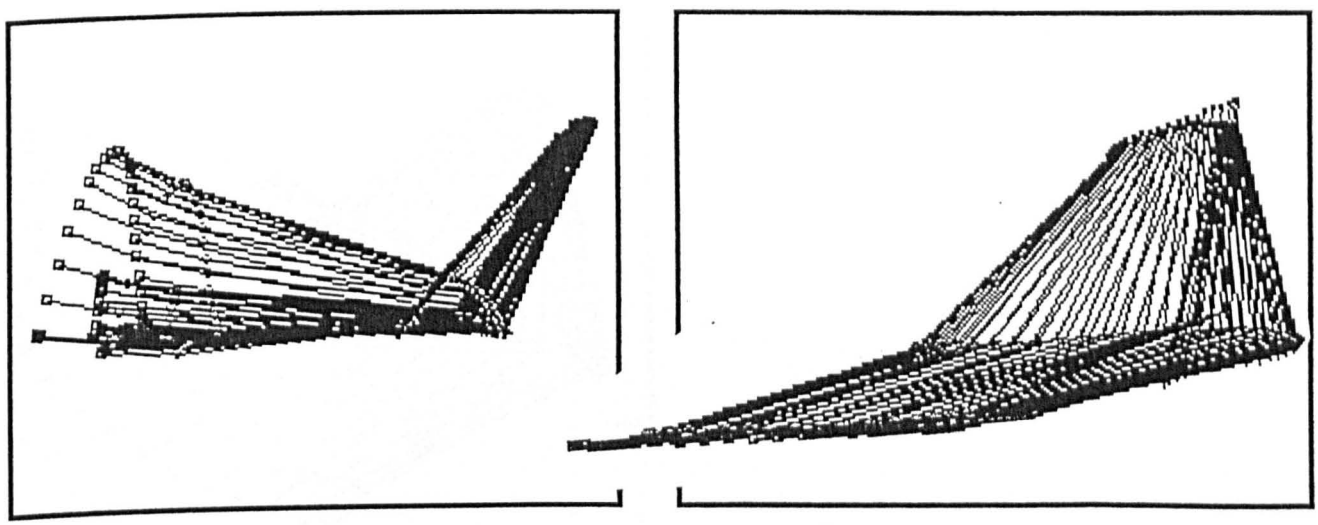
ai] Baseline - HAMMER.

bi] Baseline - CARVE.



aii] Intervention - HAMMER.

bii] Intervention - CARVE.



aiii] Post-intervention - HAMMER.

biii] Post-intervention - CARVE

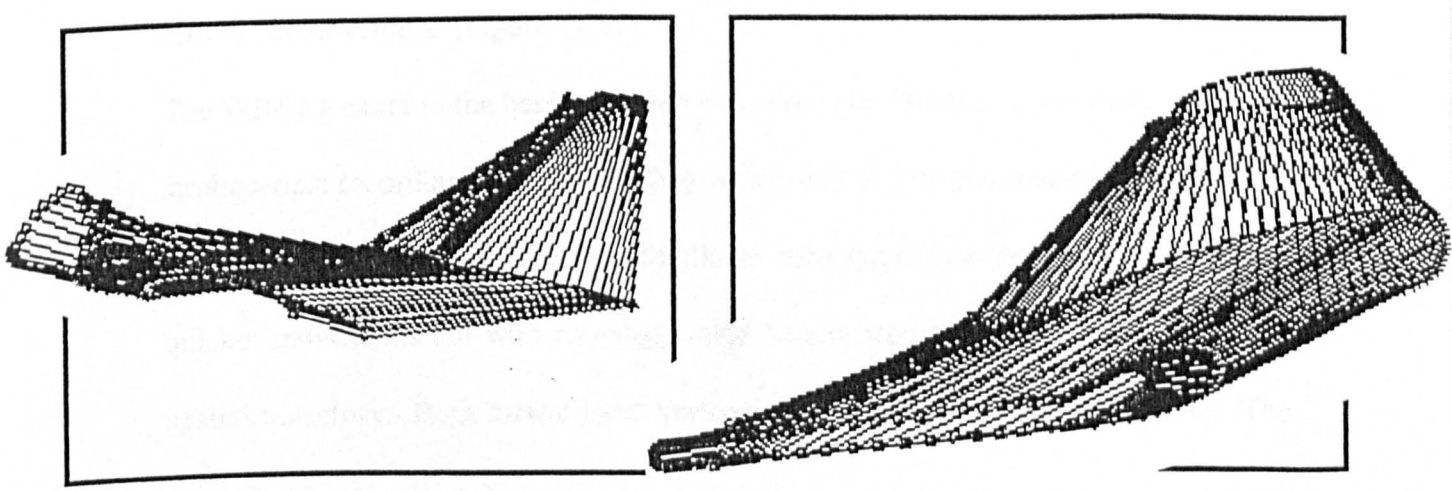
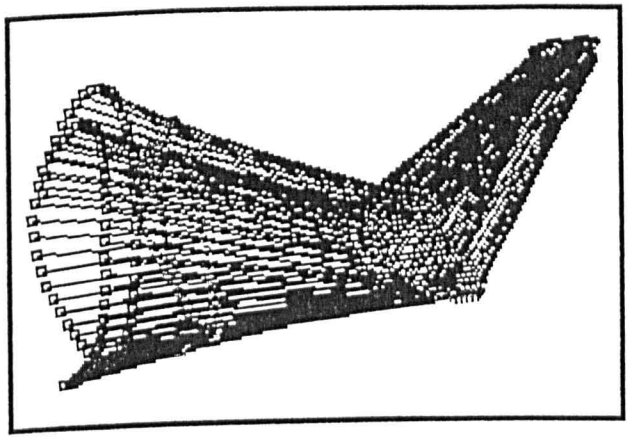
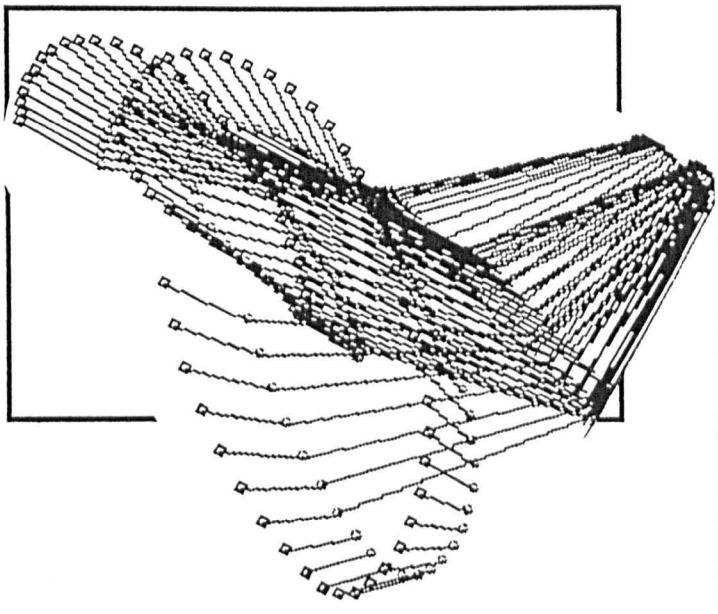


Figure 11.12 (cont.)

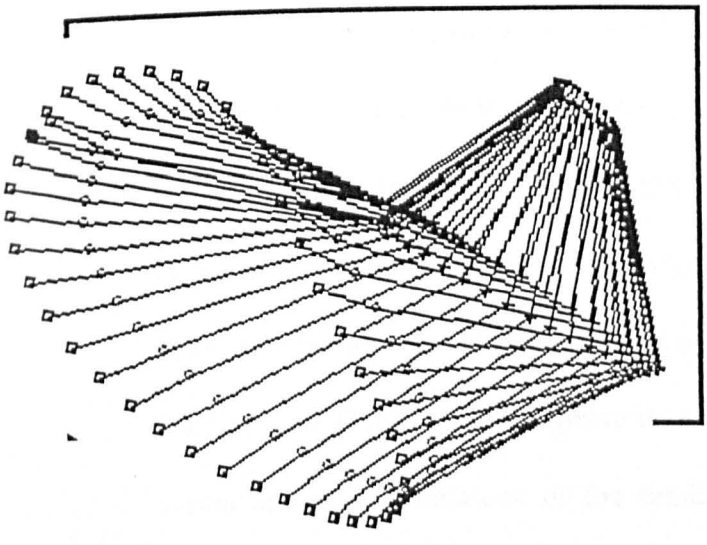
ci] Baseline - WIND



cii] Intervention - WIND



ciii] Post-intervention - WIND



with excessive use of the shoulder and elbow, coupled with a circular rather than horizontal movement (Figure 11.12).

The WIND gesture in the baseline phase was slow and effortful as shown by the dense stroboscopic recordings (Figure 11.12ci) with a forward displacement of the repeated movement action. The intervention phase recordings illustrated more fluid and quicker movements but with an exaggerated action accompanied by inconsistency in spatial trajectory. Both forward and backward action displacement was shown. The

spatial alignment improved in the post-intervention recordings, with a demonstrable quick and fluid action. The complete gesture was substantially exaggerated however (Figure 11.12ciii). These gestural recordings across experimental phases clearly demonstrated change in performance by PH, though the temporal and spatial aspects found in these repetitious gestures were somewhat inconsistent when compared with the drinking task. Given the variability of performance seen in other measures, the small data set of both gestural recordings and drinking task recordings might not be reliably representative of general performance in each phase.

11.4 iv] Relationship between clinical tests and kinematics of movement

An analysis of the correlation between the clinical test scores and the total time taken in the experimental drinking task revealed a statistically significant relationship only between the De Renzi et al (1980) gestural test and the copy condition, though only three data points were available. High negative correlations were found between the Haaland and Flaherty (1984) gestural test to verbal and copying commands and the verbal and copy conditions of the drinking task (lower the clinical test score the longer the task time), though this did not amount to statistical significance [**De Renzi test** : verbal command $r(3)= .89$ *NS.* Copy command $r(3)= .999$ $p< .05$, Real object $r(3)= .09$ *NS.* **Haaland & Flaherty test** : verbal command $r(3)= -.93$ *NS.* Copy command $r(3)= -.92$ *NS.* Real object $r(3)= .23$ *NS.* **Agnosia test** : verbal command $r(3)= .51$ *NS.* Copy command $r(3)=.45$ *NS.* Real object $r(3)= .43$ *NS*]. In this single case, the agnosia test was not seen to be correlated with completion time of the task in any condition. None of the clinical tests were predictive of the total time taken in

the drinking task in any condition, with the exception of the De Renzi test predicting performance in the copy condition [**De Renzi test** :- Verbal condition r=.799 $F(1,1)$= 3.97 *NS*. Copy condition r=.998 $F(1,1) = 633.72$ $p < .05$. Real-object condition r=.009 $F(1,1)= 0.018$ *NS*. **Haaland & Flaherty test 1** :- Verbal condition r=.87 $F(1,1)= 6.5$ *NS*. Copy condition r=.84 $F(1,1) = 5.37$ *NS*. Real-object condition r=.05 $F(1,1)= 0.11$ *NS*. **Agnosia test** :- Verbal condition r=.26 $F(1,1)= 0.36$ *NS*. Copy condition r=.20 $F(1,1) = 0.256$ *NS*. Real-object condition r=.18 $F(1,1)= 0.45$ *NS*]. All the clinical tests were extremely poor predictors of the total time taken in the real object-use condition of the task. The small data set for this analysis must, however, be taken into account when considering these results; they may not be generally representative of PH's performance given the variability in other data sets.

11.5 Discussion

This single case design attempted to overcome some of the methodological difficulties of the previous single case investigations. Daily sessions were (mostly) achieved with specific protocols given in each experimental phase. The timing of the research sessions was kept constant. Visual analysis of the data from the outcome measures revealed a distinct learning curve in the baseline phase of the study for the NHPT and the accuracy of maze task performance. Once this learning effect had ceased then the variability of day-to-day performance became apparent as with other cases (DC), with some erratic changes noted in both the maze task accuracy and the nut and bolt test performances. These might be explained by changing levels of concentration and attention in PH for the maze task, and by the adoption of a variety of strategies to

complete the nut and bolt task as no restrictions were placed on him in their execution. This variability of performance has been demonstrated across several cases, and might be judged to be a feature of the apraxic condition. No intervention effects were evident from the outcome measures data in this case, so sensory stimulation in addition to verbal and visual mediation strategies could not be judged to be effective in improving speed of performance in the chosen outcome measures.

No *immediate* effect of intervention was seen in any task in any experimental phase. This was in line with the findings of the previous case investigations (DC and EW) and lent support to the idea that those changes noted in GP were more a function of her particular case and dysfunctional sensory system (given the sensory mislocation noted) than aspects of effects on the praxic system *per se*. The use of sensory stimulation in patients *without* sensory dysfunction might, over time rather than immediately, act upon the attention and alerting mechanisms for successful motor output

Disparity between scores in the apraxia assessment battery was noted across experimental phases. A general deterioration was noted in two of the assessments for ideomotor apraxia (appendices D2 and D3), but an improvement in ability was noted in the test for ideational apraxia and the De Renzi et al gesture copying test. Since previous analyses had demonstrated the rigour of the De Renzi et al test in comparison with the other ideomotor apraxia tests, it might be considered a more reliable indicator of change in PH. Some change could be judged, therefore, to have occurred over time in both the ideational and ideomotor components of PH's praxic

ability, but this change could not be attributed to the experimental intervention procedure.

Kinematic performance both in the drinking task and the gesture performance (HAMMER, CARVE, WIND) showed some interesting relationships and dissociation's. The drinking task indicated a loss of speed of performance across all three modality conditions in the intervention phase of the study but accompanied by an improvement in consistency of spatial trajectory. An improved shoulder joint control was noted in the intervention phase, as well as an improved velocity curve showing more of the 'bell-shape' normal pattern and some degree of loss of the long, low-velocity periods at the transition of movements of each task phase. It is possible that the verbal and visual intervention strategy, perhaps together with the sensory stimulation procedures, led PH to adopt a less impulsive and more controlled-processing kind of strategy in his movement performance. The baseline phase where PH was unaware of his movements deficits would give way to an awareness of movement performance through the interventions, and thus to a controlled and focused effort as time progressed. Thus could be the explanation of the *slower* motor output but with improvement in the *spatial* aspects as demonstrated. Whilst such an explanation might hold true in the drinking task, a different pattern of performance was noted in the repetitive movement gestures recorded. Here slow, controlled and small gestures of HAMMER and WIND were seen in the baseline phase suggesting a deliberate and concentrated control-process strategy of movement output. The intervention phase recordings noted a more fluid and quicker performance in the gestures but with loss of spatial accuracy and constancy in the trajectory pathways,

which suggested an alternative strategy might have been adopted of the 'rough approximation' approach suggested by Hermsdorfer and colleagues (1996). If these results are meaningful (given the small number of recordings and the acknowledged variability in PH's performance) the PH might be said to have been adopting different strategies of motor output depending on the task demands. The single gesture of the drinking task focused on accuracy of reaching a target point on the table to 'pick up' the pretend mug or real object and thus the strategy of drawing PH's attention to his movement performance by verbal and visual mediation might have led him to adopt a more 'careful' strategy aiming for accuracy of output. The requirements of visual mediation in achieving the accuracy of target performance might have been an important factor here. The gestural performance using repetitive actions, on the other hand, required no 'target' accuracy and did not require continual visual mediation of the movement to achieve the goal output. The baseline attempts at the gestures could have been concentrated efforts to produce an ill-defined representation of the requested gesture in space, which gave way to a more confident and expansive gestural performance as time progressed. This progressed to an exaggerated movement in the post-intervention phase suggestive that PH had acquired a movement representation that he was confident and comfortable with, but his monitoring and visual mediation of the gesture was diminished. Given PH's lack of insight into his condition and errors, this lack of visual monitoring of movement when not dependent on a target for accurate output could be a compelling explanation of the differences between the task performances in the kinematic analysis. Task differences and resulting kinematic effects in performance would be an important

factor when looking at the results of other studies (Poizner et al 1990, Hermsdorfer et al 1996, Fisk & Goodale 1988) as the different task requirements for each study could have drawn out aspects of patient performance and movement strategy which could not be generalised to another task. Thus conclusions reached would only be applicable to the particular task 'type' used in the research, whether single gesture, repetitive gesture or pointing to target.

The lack of relationship between clinical test scores and kinematics of movement has been demonstrated in this case apart from the De Renzi test which correlated with, and was predictive of, time taken to complete the copy condition of the drinking task. This *general* dissociation confirmed the group study data and the GP case, though is contrary to that which emerged in EW and DC where a tentative hypothesis was being formed that those with ideational apraxia might show an association between clinical tests and kinematics of movement, different from the dissociation seen in patients with ideomotor apraxia (Hermsdorfer et al 1996). Several points are worthy of note here: an association *has* been found with PH in one test (De Renzi et al 1980) and the copy condition, and high correlations were seen with the Haaland and Flaherty (1984) test (though not amounting to statistical significance) in both the verbal and copy conditions. This suggestion of an association with the Haaland and Flaherty test which requires gestures to verbal and copying commands replicates similar findings in DC and EW. Both these cases had a language deficit as did PH; EW had ideational apraxia as did PH, though such a condition in DC was only very circumstantial. No relationship was found in the *group* study relating ideational apraxia to the Haaland and Flaherty test, but it is possible that these single cases have highlighted a potential

'sub-type' of apraxia where such an association *is* found when language and ideational components coexist. No association was found between the kinematics of movement and the agnosia test in this case, replicating findings for both DC and EW. These similarities in the three cases might reflect an disruption of semantic-action routes rather than vision-to-action routes (Riddock et al 1989) within the ideational apraxia condition. It is possible that the small data set restricted stronger associations being found, though this is conjecture. The De Renzi test and the copy condition were shown to have the strongest association in the DC case, and as it was also shown to be influential in PH *despite* the small data set, other associations might have emerged with a larger data base. It is also feasible that PH's variable performance could have affected the results both of clinical assessments and movement kinematics according to the day of testing so, again, the small data base has been a restriction in this analysis.

Given the data in all the outcome measures used in the study, the experimental hypothesis (no.1 page 313)relating to an intervention effect following a sensory stimulation protocol must be rejected. None of the measures taken provided sufficient evidence of change that could be incontrovertibly attributable to the sensory stimulation intervention. No difference in motor performance was shown between the verbal and visual mediation strategies alone or when paired with sensory stimulation. Variability of performance was seen throughout this single case study and added to the weight of evidence that this might be a feature of the apraxic condition. No reduction in variability was shown in the intervention phase of the investigation, so this hypothesis (no.2, page 313)was rejected also.

This single case investigation provided further evidence of kinematic features found in people with apraxia. The kinematic analysis demonstrated some improvement in drinking task performance in the intervention phase. PH executed a smoother, fluid movement, improved gestural action, more controlled shoulder use and changed velocity patterns in the intervention phase though this was not always maintained in the post-intervention period of the project. Different kinematics of movement performance were found in the repetitive gesture recordings. Some movement abnormalities were seen *throughout* the investigation. Thus the third experimental hypothesis (page 313) was partially supported by the evidence obtained.

11.6 Conclusions

This case study sought to provide a rigorously controlled ABA design to evaluate intervention effectiveness. Greater control of experimental variables *was* achieved, compared to the previous case studies, but the limited time scale and small numbers in some of the data sets reduced the reliability of conclusions which might be drawn. Learning effects were seen in several of the outcome measures used thus limiting the validity of any results relating to phases of the study. This methodological flaw could have been avoided by longer baseline periods where data could be collected *after* learning effects had ceased. This, however, had to be balanced by the time restrictions of the rehabilitation programme and likely time-projections as to PH's in-patient status when daily interventions could be carried out.

Variability of motor performance was observed throughout the investigation, particularly when learning effects had canceled out, and added to the evidence of previous case studies that this might be considered a phenomenon of the apraxic condition. In this single case, no reduction in variability was seen during the intervention phase which contrasted with previous findings. PH remained unpredictable and inconsistent in his motor performances and so the experimental hypothesis relating to variability of performance (no.2))was rejected. No *immediate* effect of any intervention was shown in any of the experimental phases, consistent with previous single case research (DC and EW). This might indicate that immediate, measurable results would be unlikely to be found following therapy and that change over time might be the only appropriate outcome to measure intervention effectiveness. No intervention effect was convincingly demonstrated by the evidence provided, and the experimental hypothesis (no.1) was rejected. No evaluation of changes in functional outcome was possible during this single case project though research of such a nature (relating intervention to self-care activities) would be valuable.

The kinematic data from this case study confirmed and supported previous study findings in spatial misalignment and inconsistency, over-use of the shoulder during activities, and abnormal velocity patterns. Different kinematics were shown in different tasks (drinking task vs gestural performances of HAMMER, CARVE and WIND) and led to the speculation that the task demands relating to visual mediation of accuracy to target in a single gesture might produce a different strategy of

performance, compared to repetitive gestures in space which do not require accuracy to a set point nor continual mediation of the movement in performance. This could go part way in explaining the differences found in published research where task 'types' differed from study to study. Some aspects of PH's spatial constancy and joint control improved during the intervention period thus partially supporting the 3rd experimental hypothesis.

Evidence for an association or dissociation between clinical tests and kinematics of movement were equivocal in PH. Some support was seen for a tentative notion that those with a combination of ideational apraxia and a language deficit might show an *association* between the temporal aspects of movement performance (completion time) and the Haaland and Flaherty test, and might thus be evidence of a 'sub-type' of apraxia relating to semantic-action route loss. Statistical analysis, though, revealed a general *dissociation* apart from one test (De Renzi et al 1980) with one (copy) condition in the drinking task. The lack of computer software to analyse, fully, data from the repetitive gestural performance in relation to the clinical assessments was a regret. The small data set was also limiting. Further data collection and greater use of the kinematic raw data might have allowed more powerful conclusions to be reached. Nevertheless, the data have pointed to interesting factors in apraxic movement relating to task demands as well as suggesting support for the notion of sub-types within the blanket 'apraxia' term.

Further research to establish the existence of sub-types of apraxia within the general diagnosis would be a useful avenue to explore, together with potential associations and dissociations with different clinical tests and movement kinematics. Further study to investigate and elucidate the suggestion of different task demands leading to adoption of different strategies of motor performance in the apraxic patient would also be pertinent to functional activities and rehabilitation strategies.

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[1] Public output from chapter data :

Butler, J.A. (1997). Intervention effectiveness : Evidence from a case study of ideomotor and ideational apraxia. British Journal of Occupational Therapy. 60 (11). pp 491-497.

Chapter 12. GENERAL DISCUSSION

This research project aimed to explore two main areas; the assessment of apraxia and the intervention/rehabilitation of the apraxic condition. This was achieved through a group study using apraxic, non-apraxic (but left hemisphere damaged), and normal participants to explore aspects of clinical assessment procedures and kinematic analysis of movement errors. This was followed up by a series of single cases investigating the natural history of recovery in apraxia and the effectiveness of intervention.

12.1 Assessment of apraxia.

12.1 i] Clinical tests.

An operational definition of apraxia used in this research involved behavioural observation in judgment of gestural errors and thus concentrated on the apraxic condition at the level of disability in terms of diagnosis and identification of apraxic patients for the study. This approach is adopted by other researchers in the field as observed behaviour during clinical tests is the most accessible route in determining the presence or absence of the condition, though opinion differs about the cognitive components which might best elicit manifestations of the apraxic behaviour. Most researchers would agree that confirmation of findings from formal apraxia tests should be made by observation of behaviour in real-life situations.

The differing opinions concerning apraxia tests and the cognitive components inherent within them reflects the debate concerning just what comprises the 'core' of apraxia. Different modalities of testing (to verbal command, or copying a model of movement) are suggested as important by some researchers (De Renzi et al 1982, Roy & Hall 1992) but not others (McDonald et al 1994). Similarly the assessment of apraxia involving pantomimed use of objects is considered essential by some researchers (Riddock et al 1989, Alexander et al 1992) though others (De Renzi et al 1980) have concentrated on just copying gestures as the means of testing. All of these different testing procedures have been used to elicit and identify ideomotor apraxia though each, by their construct, may have identified particular 'types' of the disorder. The assessment for ideational apraxia is less controversial and clinical tests have used sequencing of multiple objects to elicit the condition.

This project focused on ideational and ideomotor apraxia (as defined by De Renzi 1985, De Renzi & Lucchelli 1988, Kirshner 1991, Tate & McDonald 1995). In order to account for all types in the heterogeneous condition at the level of impairment, *several* assessments were employed involving different cognitive components. Identification of apraxia was achieved using the classification system of error types suggested by Raade et al (1991) to judge the movement performance (observed behaviour at the level of disability). The data from the group study and the single cases all demonstrated the separateness and dissociation of each of the clinical tests used one with another. No correlation between tests was found. This has suggested

that each of the apraxia tests *was* highlighting different aspects of the condition and might be considered evidence for 'sub-types' of apraxia. If this heterogeneity was accepted, and each of the tests was measuring something essentially different, then it might be considered that the use of a generic apraxia term to describe and 'group' people could be misleading at the least, and meaningless at worst. The present study, in using a *battery* of the tests and having the criterion for inclusion into an apraxic group dependent upon not only the De Renzi et al (1980) test but also cumulative scores on all the tests for apraxia, could be seen to include the range of 'sub-types' that there might be. This heterogeneous group, though, were shown to retain a uniqueness and statistically significant difference in all the individual clinical tests when compared to the non-apraxic and control groups ($p < .001$). It might be considered that the individual tests were capable of eliciting some 'sub-type' differences within the whole group, but that a unifying underlying impairment contrived to make an 'apraxia' diagnosis appropriate as a blanket term.

Analysis of the clinical tests themselves suggested that the De Renzi et al (1980) test was the most sensitive and reliable of the assessments employed and thus supported the work of Tate and McDonald (1995). The De Renzi et al (1980) test was also shown to be the best predictor of performance on the experimental drinking task, though it was demonstrated to account for little of the variance in motor performance completion times. Two of the apraxia assessments ((Haaland & Flaherty 1984, and single-object pantomime - appendices D2 and D3) were shown to have little internal

consistency and little predictive ability in task performance in the group study. These two tests, the group analysis suggested, were rather weak as psychometric tools. Interestingly, when the *single* cases were analysed, the Haaland and Flaherty (1984) test emerged as being potentially influential in cases with coexisting ideational apraxia and language dysfunction, though this might be an artifact of the small data sets in the study. This potential association attests to the power of using single cases when exploring a complex area, compared to larger n studies, and might be considered to identify a small sub-type of ideomotor apraxia with ideational elements and certain semantic-action route dysfunction.

In the clinical setting the use of a whole battery of available assessments would enable the clinician to observe and elicit a wide range of performance errors across the heterogeneous apraxic group and provide a full and thorough assessment. It would allow identification of what might be considered to be the 'core' of apraxia: the inability to perform actions to command without spatial, conceptual or temporal errors (in the absence of other movement disrupters like paralysis, sensory impairment, inattention etc.), and then to determine the 'sub-type' by identifying under which cognitive conditions the individual patient performed better or worse (verbal command, copying gesture, real-object use, complex sequencing, single gesture, peripersonal or extra-personal space etc.). It is argued, therefore, that a variety of tests should be used as a matter of principle in the identification of apraxia.

This research was dependent upon identifying the presence of apraxia from clinical assessments. Such diagnosis remains a matter of individual experience and clinical judgment, though inter-rater reliability was shown to be high in this study using two researchers. The identification process depends on extrapolation of apraxic signs from a sometimes extremely complex clinical presentation, taking into account difficulties with attention, primary motor difficulties and language deficits. The main judgments of errors is in the spatial domain (plane and angle of movement, hand configuration, orientation to self and extra-personal space) with little opportunity to judge any temporal component unless there are gross errors. The development of a standardised, reliable diagnostic tool, as suggested by Tate and McDonald (1995), would add reliability and validity to the diagnostic procedure and thus aid all researchers and clinicians.

The emergence of the agnosia test as an influencer and predictor of movement kinematics in the group study was an unexpected result. A similar influence was shown in the single case MT but not in the cases of DC, EW or PH. The group study highlighted the possibility that the agnosia test might be highlighting a cognitive-perceptual component of apraxia relating to visual representation and intact vision-to-action routes (as described by Riddock et al 1989, Pilgrim & Humphreys 1991). Further studies to refine and develop this test might be worthwhile. It would also be useful to consider why it was not influential in those single cases where language and ideational apraxia elements were found. This was not seen to be a factor in the group

study; no differences were found in the agnosia test between those in the apraxic group who had ideational apraxia compared with those only with ideomotor apraxia, and *all* subjects in the group had a language impairment. It is possible that the single cases were highlighting an important association, lost in the larger n analysis, though it might equally be an anomaly due to the small data set of the single cases. With these particular single cases, whilst the agnosia test was not seen to be influential in predicting motor performance, the Haaland and Flaherty (1984) test emerged as the one which *was* influential. Here it was hypothesised that the test, using verbal and visual modalities of testing gestural performance, could be focusing on impaired semantic-action routes in the patients rather than vision-to-action routes. This would not explain, though, why the severely aphasic patients with ideational apraxia did not show the association but only those with a relatively mild aphasia who were able to verbalise some meaningful vocal output. The inter-relationship between clinical tests and the kinematics of movement clearly warrants further investigation and the various factors extrapolated from the exploration.

12.1 ii] Kinematic analysis as an assessment tool

The lack of a standardised assessment tool for apraxia led specifically to the exploration of kinematic analysis of motor performance in people with different types of hemispheric lesions (Goodale et al 1990). If such analyses could provide a data base from which each movement disorder could be identified, it was hypothesised that

not only would this be a useful tool for identification of the apraxic condition, but it might also be a mechanism for achieving objective and reliable measures for assessment and evaluation of therapy. This project, therefore, specifically devised an experimental drinking task using three modality conditions to complement the conditions of some of the clinical assessment tests.

Despite the heterogeneity of the apraxic condition and the inclusion of a wide range of severity of apraxia within the diagnostic group, the kinematics of the group study showed a robustness of apraxic characteristics that were in keeping with previously published work and might support a notion of an underlying 'core' movement performance difficulty. The group study revealed differences in kinematic profiles between the apraxic and non-apraxic groups. The apraxic group showed a loss of fluidity of movement, inconsistency of spatial pathways, over-use of proximal joints in carrying out the experimental task, and the presence of multi-peaked velocity patterns with prolonged adjustment periods between directional changes of movement. Whilst such kinematic data provided supportive evidence for the clinical assessment tests in both the group study and in the subsequent single case projects, it was not seen to be unequivocal in the identification of apraxia. These 'apraxic' kinematic features were not found to be indicative of apraxia, nor present in all cases. Indeed, in both the group study and the single cases some normal kinematic profiles were evident in apraxic people, either in velocity curves showing a normal bell-shape, or in consistent trajectory pathways with normal spatial relationships evident from the recording

patterns. So while these objective data from the group comparisons provided some solid confirmation of movement deficits previously reported by research in one or two single case reports (Poizner et al 1990, Clark et al 1994) and supported the theoretical notion of apraxia involving a disruption of temporal-spatial elements of motor performance (Hermsdorfer 1996), they also confirmed the sometime dissociation between the kinematics of movement and the clinical tests.

Hermsdorfer et al (1996) suggested that kinematic profiles in apraxic people could be seen to differ according to different strategies employed by individuals in an attempt to compensate for their loss of visual representation of the target movement. A feature of one single case demonstrated different kinematic profiles according to the *task demands*. This single case, PH, might have been employing a different strategy for the drinking task (which required accuracy of a target position with continual monitoring and visual mediation of the accuracy of movement) leading to a profile of slower time (due to a focused and controlled processing strategy?) associated with improved spatial constancy and controlled joint position. This contrasted with the kinematic profile employed by PH during repetitive gestures where faster, more fluid movements (due to a 'rough approximation strategy without visual mediation?) were accompanied by loss of spatial constancy and abnormal shoulder joint positioning. This potential inter-relationship between strategies of movement performance and task demands would be an important consideration in the interpretation of published work in kinematics in apraxia, as each has been conducted on a different task-type

(repetitive gestures, meaningless single gestures, pointing to target). Results might be only pertinent to that specific task-type and not generalisible to overall movement performance.

The first experimental hypothesis of the thesis (page 48) was partly supported. A difference in movement performance, revealed by kinematic analysis, was seen to occur between the three participant groups of clinically diagnosed apraxic, non-apraxic but brain damaged, and normal controls. A rider to this should be, though, that such deficits in kinematic profile were not present in all individual cases of apraxia.

The computergraphic recordings *did* provided objective kinematic data, and also helped to identify aspects of movement difficulties for each individual patient. However, in using such kinematic tools, the clinician-researcher had to become an expert interpreter of the information in the same way as becoming an expert interpreter of observational data. The utility of such kinematic data in the everyday working life of a rehabilitation therapist is, therefore, questionable. It could be argued that the extra information concerning apraxic movement, reliable and objective though it might be, did not radically influence clinical judgment and that the extra time spent in collecting and analysing such data was not justified by changes in clinical evaluation and practice. There were also acknowledged software problems which inevitably occur with individual specifications in computer analysis. Bugs in the dedicated software, at times, meant that the raw data could not be retrieved. These difficulties

were largely attributable to the gross abnormalities in patients' movement which the software found uninterpretable. Without a dedicated budget or a specialist computer expert to sort out these difficulties, reprogramme the software and remove the system 'bugs', information was lost and data sets were incomplete. Any sophisticated IT system has, in practice, to have dedicated expertise available to ensure the smooth running of such a system and in a rehabilitation setting such practical difficulties as were experienced in this research project would render the system unworkable as it currently stands. Very simply, it could be argued that the movement data supplied by the computergraphic recordings did not add significantly to clinical effectiveness. The practical problems and time-consuming nature of its use, plus the need for specialist services to support the technology would argue against it being used in normal clinical situations. Its use at present might be largely consigned to research arenas.

An alternative view might be that the potential for discovering an individual's strategies of performance, via the kinematic performance analysis, and whether those strategies changed according to task demands would be a useful aid to the clinician in devising strategies for intervention. By such means the clinician might seek to maximise the use of a variety of strategies, so that the individual had a repertoire to choose from. Such developments would be an exciting prospect in this branch of neurorehabilitation.

12.1 iii] Relationship between clinical tests and kinematics of movement

The dissociation between clinical assessments and the kinematics of movement has been found both here and in the Hermsdorfer et al study (1996). The use of the temporal components of a task to compare with clinical assessments might be considered inappropriate because, as previously noted, the apraxia assessments are based mainly upon judgment of spatial errors made by the patient with temporal components only being capable of being seen when then were grossly distorted. Thus the completion time, or reaction time, or time in phases of a task would not generally be a part of diagnostic decision-making. Hermsdorfer et al, however, found no relationship between the *spatial* aspects of the kinematic profiles and the clinical tests, so it is arguable that some simple timing aspect of action performance might be as useful a way as any to measure motor output and performance. The Hermsdorfer hypothesis suggested that the dissociation could be attributable to different strategies employed by apraxic patients to compensate for a loss of mental representation of a target position, and the success or otherwise of those strategies. This is a compelling explanation for, in the group study with its heterogeneous apraxic population, different strategies and different levels of *successes* of those strategies would be found amongst the group leading to the clear dissociations found. Further compelling evidence for the Hermsdorfer argument arose from the single case data, where dissociations between each of the clinical tests and the kinematics of movement performance were confirmed, though not for all subjects. This suggested that the

dissociation, attributable to a coping strategy adopted by individual patients, could not be considered a universal occurrence.

An interesting result from some of the single subject analysis was the emergence of an *association* between clinical tests and the temporal components of the experimental drinking task. This was seen in three patients who had a language deficit and, in two of the three, an accompanying severe ideational component to their apraxia. Different clinical tests (Haaland & Flaherty 1984, De Renzi et al 1980) were seen to be part of that association in individual patients, suggesting they were not all conforming to a similar 'sub-type' crudely identified as ideational apraxia with language deficit. If that had been the case then MT should have also shown the association, as would those in the group study who formed an 'ideational' sub-group within the apraxia group. The Haaland and Flaherty (1984) test with verbal and visual commands for gestures was shown in two of the three single cases to be the test showing the association. It was suggested that this might be picking up some aspect of the dysfunctioning semantic-action routes in those patients. A connecting factor could be that EW and PH were not so severely aphasic as MT and DC, and were both able to verbalise which MT and DC were not. Perhaps this might be evidence for a 'sub-type' of apraxia. Certainly further exploration of the role of language within both the apraxia tests and motor performance in different experimental tasks (with different task-demands) would be of interest in determining potential sub-types.

An alternative explanation for the dissociation might be that this was evidence for, and indicative of, the 'sub-types' which could exist within the apraxia blanket diagnosis. Such heterogeneous subjects would perform according to very different underlying impairments on both the clinical tests and the kinematic tasks, and the different cognitive requirements for each task-type could manifest in different behavioural movement outcomes. A third possibility is the combination of both the 'coping strategy' explanation and the existence of 'sub-types'. Given the evidence from PH who potentially was adopting different strategies of coping with his movement deficiencies according to different task demands, then the sub-type groupings might also adopt different strategies for managing the differing task demands of the clinical tests and the kinematic movement tasks. This would show up in analysis as dissociations between the two. Until larger number studies with divisions of sub-types are carried out, or more single n studies which identify particular sub-types of apraxia, such speculation can only remain as such.

12.1 iv] Outcome measures.

Outcome measures to evaluate the effectiveness of intervention were problematic in this research project. The computergraphic recordings of the experimental drinking task provided one source of objective and reliable data from which interpretation of change might be made, though the process of data collection and analysis was lengthy and time-consuming. Whilst this might be considered appropriate for research

purposes, such time-intensive measures could not be considered viable in current everyday rehabilitation practice where clinicians have a heavy case load and time is very restricted. The research therefore attempted to use outcome measures which could be applicable in normal clinical practice. Measures were chosen both by the researcher and other therapists involved in particular patients' rehabilitation. The measures selected were chosen for being quick, manageable in a therapy session, appropriate to each particular patient's goals and giving quantitative, objective and reliable data. The selected hand movement measures (case 3, GP) and timed tasks such as NHPT and Gibson spiral maze (cases 4, 5 and 6 - DC, EW and PH) were, indeed, all manageable and applicable to the normal rehabilitation regime. They could quite reasonably be considered as relevant and generalisable to everyday rehabilitation practice. Some of these measures were seen to be subject to learning effects, however, and/or a part of the natural recovery process. Thus baseline data were often insufficiently stable to allow valid comparison of the intervention phase of the studies. A longer baseline phase was often not possible due to the timescale constraints of in-patient stay, though in the cases of GP and EW where four week ABA phases were designed the baseline data should have provided sufficient comparison.

It is possible that the outcome measures chosen for this apraxia research were inappropriate in targeting change in praxic ability. Using timing of a variety of tasks might be considered to be disassociated with apraxia, as shown by the kinematic analysis, though such measures would still be considered a part of motor action and

performance by the individual. One of the constraints of the research, and one which applies to all outcome measures in rehabilitation, was the difficulty in finding challenging and appropriate motor tasks that were also quick, repeatable and reliable. It could be postulated, therefore, that the design and standardisation of a range of motor performance tasks across a wide spectrum of difficulty would be a useful addition to the research and evaluation process in rehabilitation, and a future project worthy of consideration.

12.1 v] Modality differences.

Modality differences were not strongly evident in the kinematic analysis of movement performance in the group study. No major differences were seen, when the apraxic data was considered as a whole, between the trajectories and movement kinematics of apraxic patients in verbal, copying or real-object conditions. The only strong evidence in this project for modality differences in performance was shown in the single case LS where the 'copy' pantomime of HAMMER, CARVE and WIND were all seen to be performed more accurately than in the verbal command pantomime. The effects of modality *did* became apparent, however, when considering the relationship between clinical tests and movement kinematics.

The relationship between the agnosia test and the motor performance in the copy and real-object conditions in the group study and the single case GP had highlighted a potential connection concerning the intact vision-to-action routes in the apraxic

patients. In other single case analyses, DC and PH were shown to demonstrate an association between 'copy' performance in the drinking task (temporal component) and the De Renzi test, whilst EW demonstrated the same though in the 'real-object' condition. High correlations (though not amounting to statistical significance) were found in the single cases EW and PH between the Haaland and Flaherty test and the 'verbal' and 'copy' conditions of the drinking task, which had been tentatively suggested as highlighting a deficit in semantic-action routes. These single cases must be viewed with caution, however, due to the small data set involved with each. Such relationships that were found, though, might be preliminary evidence for 'sub-types' of apraxia where performance alters according to the cognitive components of different modality conditions.

For those patients with a language deficit, the verbal modality condition was obviously somewhat problematic and was an influencing variable in the assessment scores on those clinical tests employing verbal command (Haaland & Flaherty 1984, object-use test- appendix D3). The evidence from the group study and from Case 1 (MT), however, revealed that the inclusion of such tests enabled the identification of those patients whose motor performance of gestures was *better* to verbal command than to the copying/visual condition *despite* their language dysfunction and allowed for identification of possible sub-types of apraxia. This could be considered important information when identifying the rehabilitation strategies appropriate for each individual patient, though contrasted with the results from Alexander et al (1992) who

had found that verbal command performances were inferior to imitation performance *whether or not* the individual with apraxia had a language dysfunction.

The lack of modality differences in performance in the experimental drinking task, as shown by the kinematic analysis, could be attributed to the simplicity of that task. It might be argued that the drinking task was not sufficiently complex or challenging to the motor planning system to produce a differential effect in performance in the majority of patients. The second experimental hypothesis (page 48) that there is a difference in apraxic movement, shown in kinematic analysis, according to the modality of testing was, therefore, not supported by evidence.

12.1 vi] Anatomical basis for apraxia.

At the beginning of this research project it was hoped that MRI data would be available for both the group study and the single case investigations. It was intended that these data would be used to explore apraxic dysfunction in relation to focal areas of damage, determine whether intervention effectiveness was linked to lesion location, relate rehabilitation outcomes to lesion site, and contribute to the debate regarding the localisation of apraxia within the left hemisphere. Plans were made, initially, to recruit participants who had MRI scans in their medical notes already, and to approach others who might be suitable to seek their permission and cooperation in obtaining MRI data. Approval was sought, and given, by the local ethics committee for this extension to the research project. It was thus intended to relate the clinical

apraxic condition, assessment scores and kinematic profiles to the radiographic reports of lesion location and identification. Unfortunately, due to the constraints of researcher-participant dynamics this was not able to be followed up.

Participants in research, just as in clinical interventions, should be partners and colleagues with the clinician or researcher towards a given goal. This relationship must allow the patient at all times to be empowered to act according to his or her own wishes, and not feel coerced or pressured into agreeing to activities or actions which, in other circumstances, he might not otherwise agree to. This is the essence of ethics in clinical research. The devastation afforded to the individual by brain damage, from whatever cause, and the dysfunction resulting from apraxia is profound. All the participants in this research project had complex and multiple difficulties. As the relationship between the researcher and participants progressed over the weeks and months, especially during the single case research, it became very difficult to ask the participants to undergo the relatively unpleasant procedure of an MRI scan. It was felt that in addition to all their other experiences and difficulties, this would be one extra burden too much when the justification for such a procedure was largely intellectual curiosity and theoretical exploration. It could be argued that by not affording them even the opportunity to agree or not, this was a dis-empowerment of their abilities to contribute and 'give' to rehabilitation, when for much of their time they might consider themselves to be mainly 'takers'. Certainly in agreeing to the research in the first instance each individual had wanted to contribute and add what

he/she could, in the understanding that others might benefit. Despite these arguments, a hesitancy remained in approaching the participants. Accessibility to neuroanatomical information from scanning techniques was, therefore, limited to an opportunistic approach.

Another factor mitigating against relating elements of apraxia to site of lesion in this research concerned the dearth of suitable participants for the study. The numbers of apraxic people capable of coping with the rigours of a research protocol and suitable for approaching for participation in research projects were small. This was, to some extent, exacerbated by being limited to one rehabilitation centre for participant recruitment. A reactive approach to participant selection was therefore employed, being entirely dependent on whoever happened to be admitted to the rehabilitation centre with the apraxic condition. The group study project contained participants with a wide variety of lesion locations in the left hemisphere as far as could be gleaned from the CT scan reports and medical notes. In the single case research, only GP and EW had detailed MRI reports and both these people had widespread and diffuse damage across both hemispheres (cortically and sub-cortically), making it impossible to reliably relate apraxic errors and performance deficits to specific lesion sites. Interestingly though, both these cases had identified left hemisphere posterior parietal damage and thus might weakly support the literature which attests to this area being associated with the apraxic condition.

As a result of both philosophical and practical difficulties, the 'anatomical basis' aspect of the project had to be abandoned. The only feasible solution to participant recruitment would be the establishment of a multi-centre trial where large numbers of patients could be recruited. Only with such large numbers collected over a five to ten year period, and with MRI data, could these patients be then grouped according to lesion location.

12.2 Intervention/rehabilitation of apraxia.

Using the information gained from the group study project relating to the clinical assessments and the kinematic analysis, the intervention/rehabilitation phase of the research evolved.

12.2 i] 'Natural history' and recovery from apraxia.

The longitudinal data collection from MT (case 1) showed a 'natural history' progression of recovery over a period of eleven months. This case provided evidence of changes in kinematic profile indicative of improved motor performance abilities. There were supportive data of changes in timing, spatial trajectories and velocity profiles from patterns and features which were associated with apraxia compared with those associated with normal movement. The group study provided the essential comparison data source from which to interpret results. This provided the first evidence that improvements in praxic ability could be monitored and evaluated using *kinematic analysis.* It was also considered that such kinematic changes would be

outside practice manipulation, when compared to clinical assessment tasks, and therefore would be a truer, objective and reliable representation of praxic abilities.

LS (case 2), who over a six week period provided the second kinematic monitoring of recovery in apraxia, demonstrated an apparent phenomenon of spontaneous recovery from apraxia. Whilst such automatic recoveries have been seen in other neuropsychological deficits, particularly hemineglect, this case provided evidence to support the findings of Basso et al (1987) who noted such spontaneous recovery within five months of stroke, though LS is the first recorded case (according to published literature to date) in a head-injured subject. Whilst LS had some unusual elements in his background and presentation, particularly regarding his attentional difficulties, there was enough evidence to suggest the presence of apraxia at the time of first assessment, with little evidence of the condition six weeks later. The relationship between the attentional system and apraxia has not been fully explored in research to date, although the work of Posner and Petersen (1990) and Robertson (1995) both indicated connections between an overseeing attention system and performance in all neuropsychological functions. Evidence from three of the cases presented in this research (LS, EW and PH- cases 2, 5 & 6) suggested that attentional difficulties might have interfered with the apraxia assessments or that improvements in attention might have contributed to motor performance in outcome measures. In addition, the intervention protocol itself might be considered to be working on the attentional system as a means of improving motor performance. The patients were

asked to ***attend*** to their movements (by visual and verbal mediation strategies) and were also given sensory stimulation which could have acted as an attentional alerting mechanism. It might therefore be speculated that those patients who, without prompting, verbalised their actions and made verbal commentary were *spontaneously* attempting to provide additional stimulation to their motor system to achieve performance output. This, as a strategy, could be considered also to be acting on the attentional system of the brain. Exploration of this aspect of ideomotor apraxia would merit further investigation. It might further be argued that the role of the attentional system in *ideational* apraxia could be even more potent where awareness of (attention to?) the errors in action is deficient.

12.2 ii] Effectiveness of intervention.

The single case investigations attempted to establish *prime face* evidence regarding the effectiveness of interventions in apraxic rehabilitation. It was considered that the baseline phase of each single case would, in part, provide evidence of the effectiveness of the 'normal' rehabilitation strategies and procedures currently being provided for apraxic patients. The single cases demonstrated both recovery processes and learning curves in the baseline phase in the outcome measures used. The recovery process was clearly demonstrated in GP across virtually all measures with regression lines showing sharp degrees of slope despite variability in day-to-day performance. This baseline extended over a four week period and so could be seen to be an ongoing process of improvement. The change in therapeutic procedure to a sensory stimulation

intervention showed no true effect on these recovery patterns, suggesting that change was occurring no matter what the intervention procedure. The other single case baselines (DC, PH) demonstrated change attributed to a learning effect across outcome measures. Once the learning effect had ceased then the data plotting indicated a wide range of performance variance in which the patients operated. The sensory stimulation intervention was not shown to have any true effect on these data.

Little, if any, effect of therapy was demonstrated throughout this research that incontrovertibly was attributable to the intervention and not part of natural improvements and recovery over time. The sensory stimulation procedure was not shown to have an immediate effect on movement performance except in the case of GP. It was established that not only did GP have a sensory mislocation/dysfunction involving her upper limbs, but also that her own anecdotal evidence demonstrated an effect on motor performance with a severe sensory (pain) stimulus. It could be argued that in a case such as GP, there was a near absence of sensory stimulus to the sensori-motor system which resulted in apraxic performance. The provision, then, of a sensory enhancement procedure could logically have led to an improved integration within the sensori-motor system which in turn resulted in improved motor performance. It would have been interesting to see if GP had improved motor performance following the use of a TENS apparatus. This, unfortunately, was not attempted.

Other cases, such as DC (case 4), were already receiving sensory information and the provision of additional stimuli was, therefore, of limited benefit. Yet other cases, EW

and PH (cases 5 & 6), had both ideomotor and ideational apraxic components to their performance deficits. It was argued that the devastation of the loss of the *idea* of performance would over-ride any benefits of sensory input to the system. It might be hypothesized that for these cases, the prime aim should be to address the awareness/attentional/ideational component of the disorder. Without an awareness of the problem, without the individual being alerted to the performance deficit, then little progress could be achieved. Raising such awareness might be achieved through following the mental representations model of illness or through cognitive-neuropsychological theories of the attentional system. Future research exploring the interaction of the attentional system with ideational apraxia would appear beneficial to rehabilitation practice, especially determining whether attentional training strategies might improve the ideational/awareness aspect of the condition. Alternatively an approach which considered the *strategies* employed by individual patients to compensate for their loss of representation of target position (following Hermsdorfer et al 1996 suggestion) with an intervention which attempted to train the patient in a variety of strategies, would be an interesting rehabilitation avenue to explore. The relationship of intervention effectiveness (or not) and potential 'sub-types' of apraxia would also be worthwhile area of research.

The methodological difficulties which beset the single case protocols must be taken into account when considering the conclusions which might be reached from the data gathered, not only in the intermittent data collection and curtailed post-intervention phases. The majority of outcome measures used in the research could be criticised for tapping in to one particular aspect of performance (timing) and perhaps being

inappropriate for monitoring change in apraxia. Clearly more *overall* outcome measures of the interventions regarding the functional performance of individuals would be more meaningful.

The third research hypothesis (page 49) stated that the addition of a sensory stimulation programme would lead to an improvement in motor performance in apraxic patients compared to previous (baseline) interventions. This hypothesis could not be supported from the case study data. However, the cases studied have provided the beginnings of a data base in rehabilitation research and have indicated areas of future research to substantiate the data presented here. They have also provided a data series of recovery in apraxic patients using a wide range of outcome measures. This is the first such data source and could provide a useful comparison for future researchers in this field.

The need for a multi-centred trial is evident if sufficient patient data is to be collected to make meaningful suggestions regarding efficacy of interventions in apraxia. It would also be recommended that a randomised control design would be required in order to assess the outcome of different philosophies and strategies of intervention looking at the *overall* outcomes of rehabilitation in terms of everyday functioning, motor performance, independence levels and quality of life. Only by randomised control trials will such comparisons of interventions be achieved. The single case research also indicated possible differences in outcome according to the presence or absence of an ideational component to the apraxic condition, or of other sub-types of apraxia. Larger numbers in a multi-centred, randomised control trial would enable data to be obtained regarding the effectiveness of interventions according to the *type*

of apraxia present, and might be able to suggest which intervention would be more effective with which condition.

These single case data have provided a rich resource of information concerning the apraxic condition to both the clinician and the researcher. The importance of the individual case in research should not be minimised. Often it is the *individual* idiosyncrasy, or 'failure' in neuropsychological research and rehabilitation that provides the additional insights into the condition being studied. It is advocated, therefore, that in large-number research trials, single case data are also collected especially in attempting to establish the presence of 'sub-types' of the condition. This would provide a richness and depth to the information which might be otherwise lost in the leveling out process of statistical analysis of large number data collection. Further research to look at the recovery profile of apraxia, along with evidence for effectiveness of interventions is critically needed in the current evidence-based health care climate. This should be a priority in the rehabilitation field with large multi-centred trials used to verify this study's evidence.

12.2 iii] Variability of motor performance.

All the single case data illustrated an inconsistency of performance. This supported therapists' clinical impressions and experience of patient day-to-day variability with apraxia, and confirms Mayer et al (1990) and Cicerone and Tupper (1991) who reported variability in apraxic subjects performances. How far this variability in performance is unique to the apraxic condition is unknown. In some of the single case

analyses it was noted that only once the learning effect had leveled out was the variability of performance noticeable. Thus the intervention phases of DC and PH were judged, through visual analysis of the plotted data, to show *more* day-to-day variability than the baseline phase where learning was occurring. Certainly variability in human performance is a well-known difficulty in single case research (Barlow & Hersen 1984), but whether this inconsistency is more profound in apraxia than in other neurological conditions or than in the normal person is yet to be established.

What might be the reasons for this variability in apraxia? If it is accepted that apraxia is caused by a disruption of the sensory-motor integration within the brain and that people with apraxia are attempting to perform actions despite this loss of integration, then it might also be reasonable to consider that any additional external or internal stressor on the system will further disrupt performance. People with apraxia are performing to the limits of their physical abilities, struggling to produce a motor performance that matches with their internal vision and expectation of performance. The case study data provided indicators of the kinds of additional stressors which could have been affecting performance of day to day activities and motor tasks, and supported the reports of Mayer et al (1990) who suggested that emotional status of brain damaged patients affected performance on a wide range of measures. Emotional factors, such as relationship difficulties, were seen in case 1 (GP) to be affecting performance. Mood state was also identified as being influential on performance abilities, like the depression diagnosed in EW, as were significant life events and anniversaries like that of the traumatic injury. Some physical factors like feeling

tired or cold, the menstrual cycle, pain levels and medication (case DC for example) were seen in the case study analyses as having possible effects on performance.

An alternative explanation for the variability might be taken from Hermsdorfer's suggestion of coping strategies employed by apraxic patients in their movement performance. The variability in output might reflect a daily successful/unsuccessful application of such strategies to different task demands. If so, then practicing such strategies or training in a repertoire of strategies might refine the process and lead to a reduction in variability of performance.

The consistency of apraxic performance variability (consistently inconsistent) was a notable feature in the single cases studied even through those baseline phases showing natural recovery processes or learning curves. It could be argued that looking at the variability of performance in everyday activities would be a useful outcome measure of intervention. A reduction in variability (i.e. a more consistent performance) could be a useful indicator of change in the person's abilities. This has implications for practice settings where care plans and discharge recommendations have to take into account the lowest level of performance demonstrated by the individual in order that safety is assured. If the variability of performance could be reduced by a specific intervention, so that the patient would perform more reliably and consistently across a range of areas, then this would be a useful and meaningful outcome.

12.2 iv] Task break-down effectiveness.

Task breakdown strategies were particularly successful in case 3 (GP) across a variety of self-care tasks and activities. These findings supported Wilson (1988) not only in

the success of the strategy, but also in that each task showed little or no generalisation effect. GP did manage to learn some general principles necessary for movement success (e.g. checking that her elbow was stabilized) but all tasks had to be meticulously taught in step-by-step fashion. This strategy was also attempted in case 5 (EW) who had both ideational and ideomotor apraxia. The effectiveness of the strategy was more limited here than with GP, and great variability was observed in performance of self-care activities. It could be argued that task breakdown strategies might be more applicable in cases of ideomotor apraxia only, though more data would need to be gathered to make such an assertion. The fourth experimental hypothesis (page 49) stated that task breakdown combined with verbal and visual mediation strategies would lead to improvements in practiced functional tasks. This was only partially met, with due caution being given to the limited data available together with the acknowledged methodological flaws inherent in the experiments. Again the need for a multi-centred trial to examine the effectiveness of this aspect of rehabilitation intervention would be a useful and needed addition to evidence-based practice.

12.2 v] Practicalities of clinical evaluation.

The practical difficulties encountered in this research project have been discussed during each chapter evaluation. A recognised difficulty was in patient recruitment. Not every person with apraxia admitted to Rivermead rehabilitation centre was appropriate to approach for the research. Some had behavioural difficulties, others

psychological problems, severe language dysfunction, severe cognitive impairments or other neurological disease such that they did not fulfill the inclusion criteria. Thus numbers were inevitably limited. In addition, two patients who were started on the single case methodology were withdrawn due to medical complications after five and six weeks respectively. Consequently not all the patients initially recruited were able to complete the protocol and be included in the research outcomes.

Methodological difficulties were inherent, as with all clinical research. Data collection was intermittent at times due to hospital appointments, sickness, long weekend leave, therapy timetable clashes or mistakes, home visits or other appointments and a myriad of other reasons for disruption to the research protocol. In addition, fitting the researcher's timetable into that of the rehabilitation centre and the particular patient's own timetable compounded the difficulties to be overcome. Daily intervention procedures and data collection were not consistently obtainable. In some weeks, five data points were achieved but in other weeks only two or three might be possible. Knowing that consistency was unlikely to be achieved, a compromise strategy was adopted of *randomness* of time for intervention and data collection across each ABA phase in the single case design for three of the four cases. It was considered that if each phase was consistently random in regard to timing, then any intervention effects could be attributable more to the intervention itself than to an effect of time of day. Never-the-less, this lack of consistency was considered to be a flaw.

The difficulties encountered with the kinematic software have been highlighted. The loss of data from the information sets was a very small percentage of the whole, but represented a waste of patient time and effort in each recording lost. In addition, the disproportionate amount of time spent in trying to retrieve the interpreted data added to the research difficulties. A future project involving such computergraphic equipment would have to include a budget for specialist software support for it to be used more effectively.

Outcome measures appropriate to the clinical situation and relevant to the apraxic person were an unexpected challenge. The use of apraxia assessment batteries were limited in effectiveness in determining change both by the nature of the category system of scoring, but also by their limited repeatability. Attempts at devising shortened multiple versions of apraxia assessments were shown to be ineffective in the pilot study of case 3 (GP) in monitoring change. This element of outcome measurement is an area which might be explored and refined in the future, along with the development of a 'gold standard' assessment tool for the diagnosis of apraxia. Devising and/or finding appropriate motor tasks that could harness and monitor the person's praxic function was also a difficulty not fully resolved. It might be argued that some of the tasks used (e.g. NHPT) were not challenging enough for the patient and therefore not capable of reflecting change. This is a valid criticism. Future research protocols attempting to evaluate effectiveness of intervention on motor

performance would have to look more carefully at the selection of motor tasks used as outcome measures.

The involvement of therapists in the research project was essential to its success. Such involvement required interest and dedicated time from those therapists, though their busy clinical schedule and the time constraints of a rehabilitation timetable did not always allow for full harnessing of the enthusiasm and interest displayed. These difficulties have highlighted the need for dedicated research therapists who have allocated research time within their clinical practice. It also suggested the need for therapists to adopt a more stringent approach to assessment and intervention which would readily lend itself to a research evaluation as a part of normal audit. Such a culture of critical evaluation of intervention effectiveness could be seen as a priority and a necessary move in rehabilitation if evidence-based practice is to become the norm.

Chapter 13. CONCLUSIONS

This research project has examined two main areas in the apraxic arena : assessment and rehabilitation. The strengths of this study lie in several areas relating firstly to the clinical assessments used in apraxia diagnosis, the kinematics of apraxic movement and the relationship between the movement kinematics and clinical tests. The research has also provided some evidence for the presence of 'sub-types' of apraxia and considered the argument for different strategies of coping with the movement deficit employed by those sub-types. The study has then provided evidence for recovery processes in apraxia and explored the efficacy of interventions in the rehabilitation of the condition.

Analysis of group study data confirmed internal consistency problems with some of the tests for apraxia, but inter-rater reliability using those tests was shown to be high. The need for development of a standardised, reliable and objective assessment tool was highlighted. The study produced a test for 'agnosia'. Analysis indicated this tool might be eliciting a perceptual-cognitive component in apraxia similar to that required in movement performance to visual modality conditions. This non-standardised tool would seem to be worthy of development and refinement, particularly if it is capable of highlighting areas of underlying impairment in relation to observable behaviour, and potentially of identifying 'sub-types' of apraxia.

Kinematic analysis from the group study and the single case research provided evidence of disruption to the temporal-spatial aspects of movement in apraxia, but also confirmed previous published research in finding normal kinematic profiles in the apraxic population. The relationship between the clinical assessments (severity of

apraxia) and kinematics of movement verified dissociations found in other published work. The most sensitive of the clinical assessments (De Renzi et al 1980 - complex finger and hand gesture copying) was not associated with movement kinematics nor predictive of motor performance. These dissociations were considered in the light of Hermsdorfer et al's (1996) notion of different strategies being employed by apraxic patients to produce movement output. This idea was developed further in this study with evidence suggesting that patients could adopt different strategies according to varying task demands, and that a consistent performance strategy was not necessarily employed on all occasions. It was considered that a useful project might explore the training of apraxic patients in a variety of strategies for managing movement output to determine whether this improved performance. The best predictor of motor performance in the group study and in single cases was an agnosia test, which suggested it captured some perceptual-cognitive component in the impairment level of apraxia which might highlight intact vision-to-action routes suggested by Riddock et al (1989).

Other single cases with ideational apraxia and language deficit showed an *association,* rather than dissociation, between movement performance and clinical tests particularly with the Haaland and Flaherty (1984) test of gesturing to both verbal and copy command. This was hypothesised, again, to be highlighting potential 'sub-types' of the apraxic condition relating to semantic-action loss as well as conceptual loss of action representation. This association was not equivocal, though, as not all those with ideational apraxia and language deficit showed the same effect. The difficulties highlighted in the use of computergraphic technology for kinematic analysis has

suggested that this equipment might be more appropriately used in research projects rather than as a normal part of clinical practice.

The single case analyses demonstrated features of recovery in apraxia both in the longer term and in shorter term spontaneous recovery. Evidence was presented which showed quicker and more efficient performance on a variety of motor tasks as time progressed. Finding simple and practical outcome measures that could be used in single case research was identified as a difficulty. Devising and developing relevant, appropriate, reliable yet quick and practical measures that would monitor change in performance has been a suggested area for future research. Such developments would be relevant and useful to clinical settings and could encourage changes in procedures towards a more evidence-based philosophy of practice. Some aspects of the kinematics of movement performance were also demonstrated to have changed over time in single case analysis. These kinematic features were taken as evidence for recovery. This data base would be useful source material for future research in this area. The interface between the attentional system and apraxia has yet to be fully explored in clinical research but this project indicated that it might be a pertinent factor in apraxia. Evaluation of attentional training in relation to praxic ability, especially in ideational apraxia, would be a useful clinical project for the future.

This research explored the effectiveness of intervention strategies in apraxia. No evidence from the studies provided convincing arguments that therapeutic procedures were effective in changing motor performance; either of 'normal' therapy, or verbal and visual mediation strategies or sensory stimulation protocols. Improvements that

were witnessed were deemed to have been part of the natural recovery process in those with stroke or head injury, with no evidence suggestive that change was the direct effect of intervention. Some effects, it was suggested, might be case-specific and relate to the severity and 'sub-type' of apraxia present in each individual. This teasing out of intervention effectiveness relating to sub-types of apraxia is an avenue needing to be investigated in apraxia rehabilitation. The sensory stimulation procedures were not shown to have an overall 'umbrella' effect on functional activities as suggested by previous researchers in the field of sensory integration research (Ayres 1985, Cermak 1985, Croce 1993) although the single case used to test this hypothesis might be an exception. Further research with larger numbers would confirm or refute such findings.

Another intervention strategy, that of task break-down, was demonstrated to be effective in one case of ideomotor apraxia, but with little demonstrable generalisation of performance across tasks. The strategy was less successful with a case involving both ideomotor and ideational components of apraxia. Variability of performance was shown to be a consistent feature in all cases studied in this project. The uniqueness, or otherwise, of this feature to the apraxic condition has not been established. Its importance in rehabilitation, though, is critical with a *reduction* in performance variability (i.e. the establishment of a more reliable and consistent motor output) being identified as a meaningful outcome measure and one which would provide more assurance of safety for someone facing discharge home.

The difficulties encountered during the research have been explored, together with the identification of methodological flaws. Future research suggestions have been made

including that of a multi-centred, randomised control trial to determine the effectiveness of interventions in apraxia involving functional outcome measures as well as quality of life indices. Only with the provision of large numbers can major recommendations be made regarding the targeting of specific intervention strategies. The continued use of single case data has also been advocated to provide a complete, rich and rounded understanding of both ideomotor and ideational apraxia, as well as establishing the existence, or otherwise, of other sub-types of apraxia.

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Oxfordshire Health Authority

Central Oxford Research Ethics Committee
Manor House, Headley Way
Headington, Oxford OX3 9DZ
Tel: Oxford (0865) 222547

Please reply to Ellen Hearth

Our Ref: RMW/EH/SC/2795
Your Ref:

8th February 1993

Mrs J A Butler, Senior Lecturer
Oxford Brookes University
School of Occupational Therapy
Dorset House
58 London Road
HEADINGTON
Oxford

Dear Mrs Butler,

RE: COREC: 2795 - Deficits in limb and axial proxis following brain damage: analysis and treatment

Thank you for letting me have the further details on this project. I am happy to confirm ethical approval, and wish you every success with the study.

Yours sincerely,

Dick mayon-White.

Dr R Mayon-White
Vice Chairman
Central Oxford Research Ethics Committee

cc Dr T Jordan
Dr D Wade

Appendix B : Patient information and consent letter.

School of Occupational Therapy

OXFORD
BROOKES
UNIVERSITY

Dorset House
58 London Road Headington Oxford OX3 7PE

Tel: 01865 62831
Fax: 01865 69679

Head: Mrs E M Burrows BSc DipCot CertEd(FE)

email : jabutler@brookes.ac.uk

COREC No. 2795

OBUEC No. 93/OT/36

STUDY OF MOVEMENT PROBLEMS FOLLOWING STROKE OR HEAD INJURY

I am doing this study to try and find ways of helping people who have certain movement problems to overcome their difficulties.

I am asking if you would help me in this study which involves being asked to mime the use of several everyday objects, and to copy some hand movements.
A camera would take pictures of you when you do some of these movements, but these are recorded as 'stick figure' diagrams, NOT photographs.
You would not be individually identifiable from these pictures, nor from the recordings. The time needed with you would be about 45 minutes.

The tests would all take place at Rivermead Rehabilitation Centre, Oxford.

You need not take part in the study. You may leave it at any time without it affecting your normal care in any way.

If you have any questions or problems relating to the study, please contact me.
Jennifer A. Butler. Telephone 01865-485274.

Appendix C : Normal control information and consent letter.

School of Occupational Therapy

OXFORD
BROOKES
UNIVERSITY

COREC No. 2795

OBUEC No. 93/OT/36

Dorset House
58 London Road Headington Oxford OX3 7PE

Tel: 01865 62831
Fax: 01865 69679

Head: Mrs E M Burrows BSc DipCot CertEd(FE)

email : jabutler@brookes.ac.uk

STUDY OF MOVEMENT PROBLEMS FOLLOWING STROKE OR HEAD INJURY

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Jennifer A. Butler. Telephone 01865-485274.

Appendix D : Tests for apraxia examination.

Appendix D1: Ideomotor apraxia test items. Gesture and movement copying. (De Renzi et al, 1980)

Note : Put a tick in the correct column to score for accurate performance :
3= correct performance at first trial, 2= correct at second trial, 1= correct at third trial,
0 = incorrect performance.
Maximum score = 72

		Trials			
	Finger Movements	**3**	**2**	**1**	**0**
1	Forefinger and middle finger extended and abducted (sign of victory).				
2	Thumb and forefinger together in a ring (sign of O.K.).				
3	Forefinger and little finger extended, other fingers flexed (sign of cuckold).				
4	Forefinger extended, other fingers flexed.				
5	Middle finger arched over the dorsal aspect of top joint of forefinger, other fingers flexed.				
6	Thumb imprisoned between the flexed forefinger and middle finger, other fingers flexed.				
7	Flick middle finger 3 times over top of thumb.				
8	Click middle finger 3 times with the thumb.				
9	"Walk" forefinger and middle finger on the table.				
10	Make scissor movement with forefinger and middle finger, other fingers flexed.				
11	Tap the table top with 4 fingers in succession starting with forefinger (drum fingers).				
12	Back of the hand on the table top. Forefinger and middle finger extended, other fingers flexed. Flex the forefinger and straighten, flex the middle finger and straighten. In turn, 3 times.				

Appendix D1 (cont.)

		Trials			
	Hand and arm movements	**3**	**2**	**1**	**0**
1	Open palm of the hand onto the opposite shoulder.				
2	Open palm of the hand onto the back of the neck.				
3	Open hand on the chin.				
4	Salute.				
5	Hand circled as a tube, place to mouth and blow.				
6	Halt somebody - arm forward, elbow extended, open hand vertical.				
7	Sequence: Closed fist - thump sideways on the table top. Open hand - slap palm on table top. In turn, 3 times.				
8	Sequence: Fist on forehead, open palm to mouth. In turn, 3 times.				
9	Movement: Open hand forwards on one side, perpendicular to body. Sweep slowly across front of body to opposite shoulder, extended fingers moving from spaced apart (abducted) to touching each other (adducted) as the hand moves.				
10	Make the sign of the cross (touch forehead, chest, opposite shoulder, other shoulder).				
11	Extended fingers, closed together. Touch forehead 3 times with fingernails (palmar surface of hand facing forward).				
12	Send a kiss. (Fingertips and thumb together. Touch mouth, extend and abduct arm, spacing the fingers as you do so. 3 times.				

Appendix D2 : Ideomotor apraxia test. Meaningful and meaningless movements (Haaland and Flaherty, 1984).

Note: Items to be tested to verbal command, then repeated by copying tester gesture. Tick the correct box for : 2= accurate performance, 1= inaccurate but recognisable performance,
0 = unrecognisable performance.
Maximum score = 40

		Verbal			Copy		
	Meaningful gestures	**2**	**1**	**0**	**2**	**1**	**0**
1	Salute like a soldier.						
2	Wave good-bye.						
3	Scratch your head.						
4	Throw/blow a kiss.						
5	Snap/click your fingers.						
	Meaningless gestures						
1	Hand on your nose.						
2	Hand under your chin.						
3	Index finger on your ear.						
4	Thumb on your forehead.						
5	Hand behind your head.						

Appendix D3 : Pantomimed use of objects

(De Renzi et al 1980, Haaland and Flaherty 1984, Alexander et al 1992, Riddock et al 1989).

Note : Place tick in correct box for scoring. 2= correct performance, 1 = incorrect but identifiable performance, 0 = unidentifiable performance or body part used as object. Maximum score = 36.

		verbal			visual			real		
		2	1	0	2	1	0	2	1	0
1	hammer									
2	razor									
3	comb									
4	pen									
5	toothbrush									
6	key									

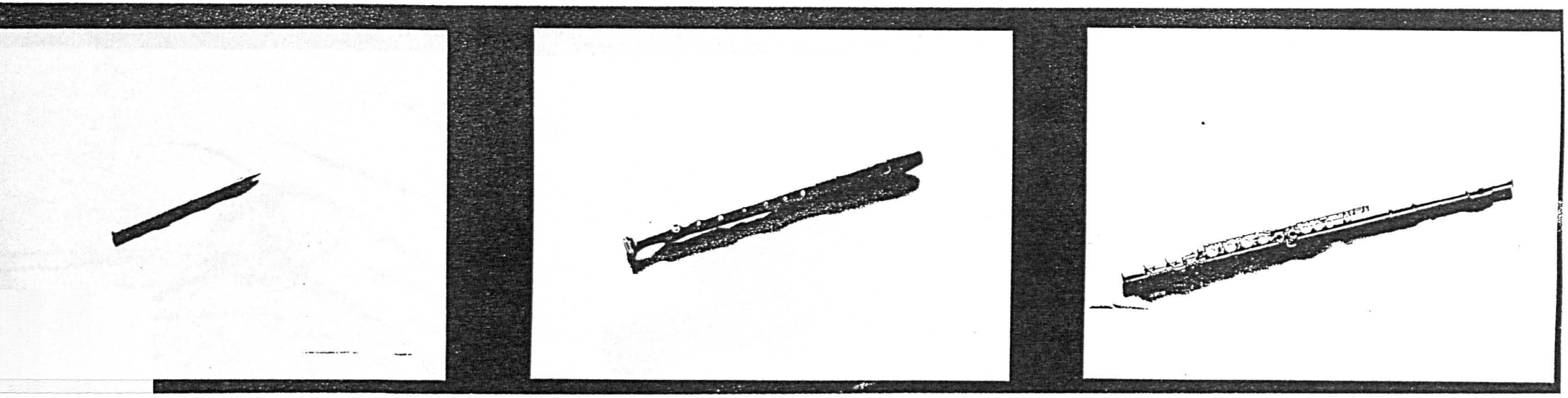
Appendix D4 : Ideational apraxia scoring sheet

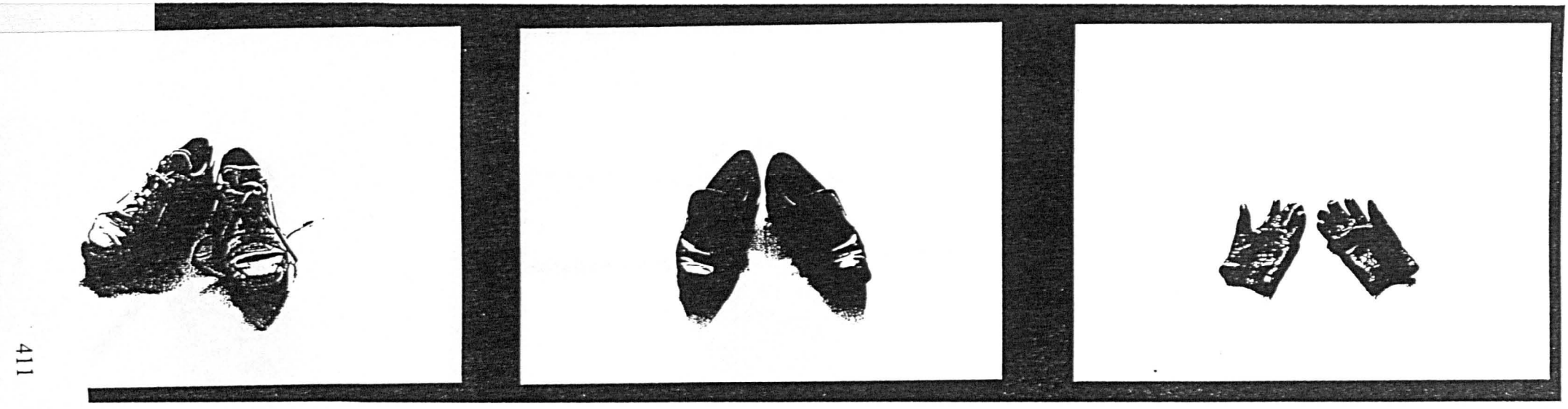
(De Renzi & Lucchelli 1988)

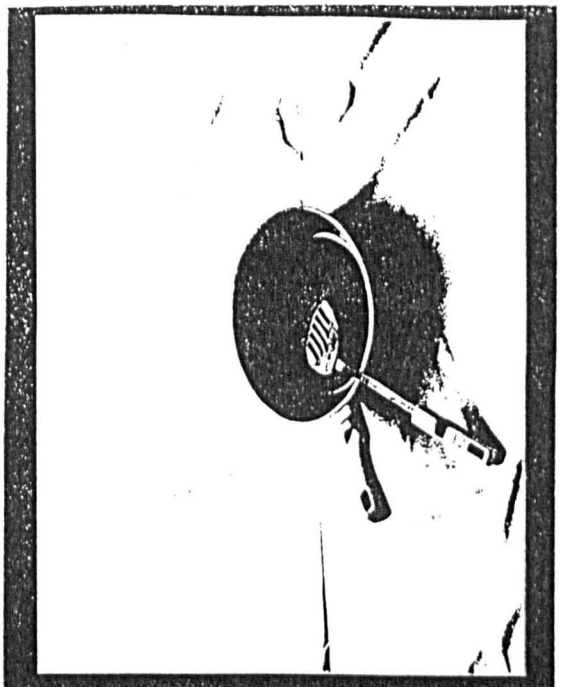
Note : put a tick in the box to score performance. 2 = correct execution of task sequence, 1 = incorrect but identifiable completion of task goal, 0 = incorrect and unidentifiable use of objects to complete task.

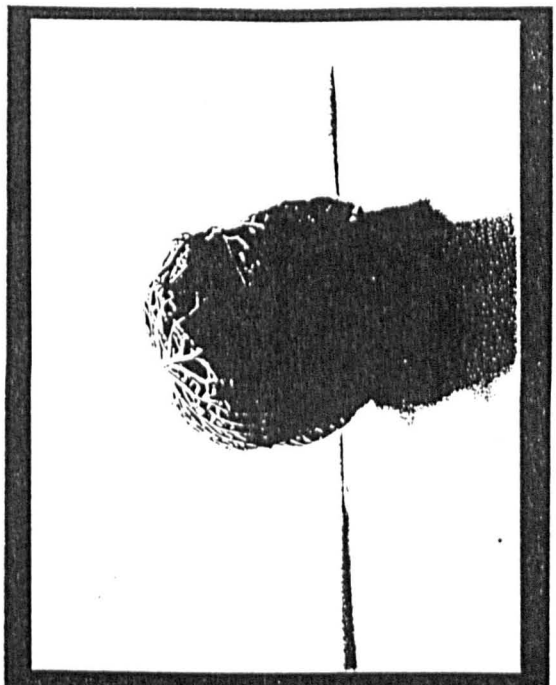
	Activity	Score			Comments /errors made
		2	1	0	
1	**Lock a box** (box, padlock and key)				
2	**Paper into a file** (sheet of paper, file, hole punch)				
3	**Polish shoes** (shoes, polish, brush, cloth)				
4	**Send a letter** (paper, envelope, stamp)				
5	**Light a candle** (candle, candlestick, matches)				
6	**Make a cup of tea** (teapot, kettle, cup, saucer, spoon, teabag, milk jug, sugar bowl).				

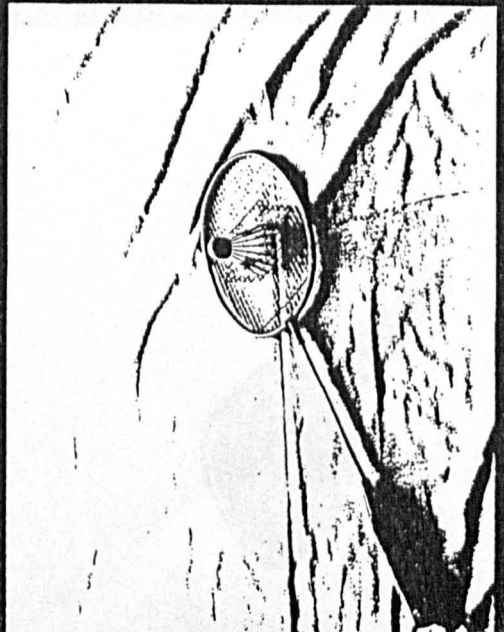
Appendix E. Agnosia screening test examples.

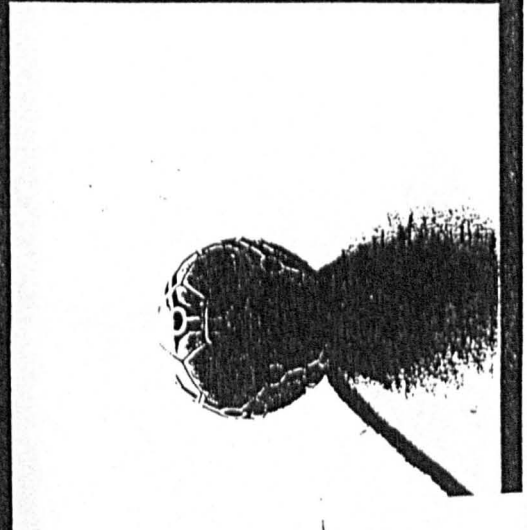




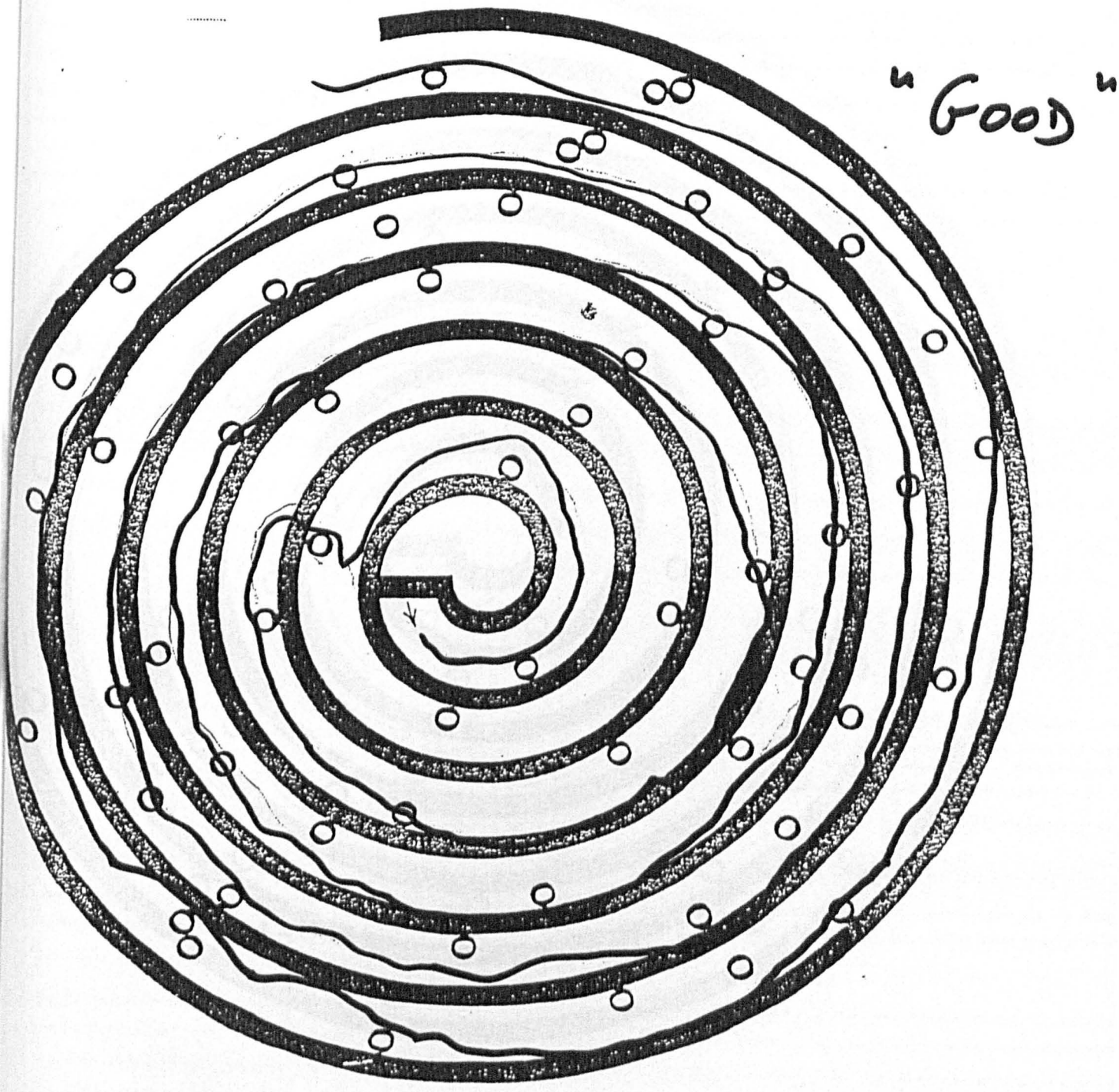


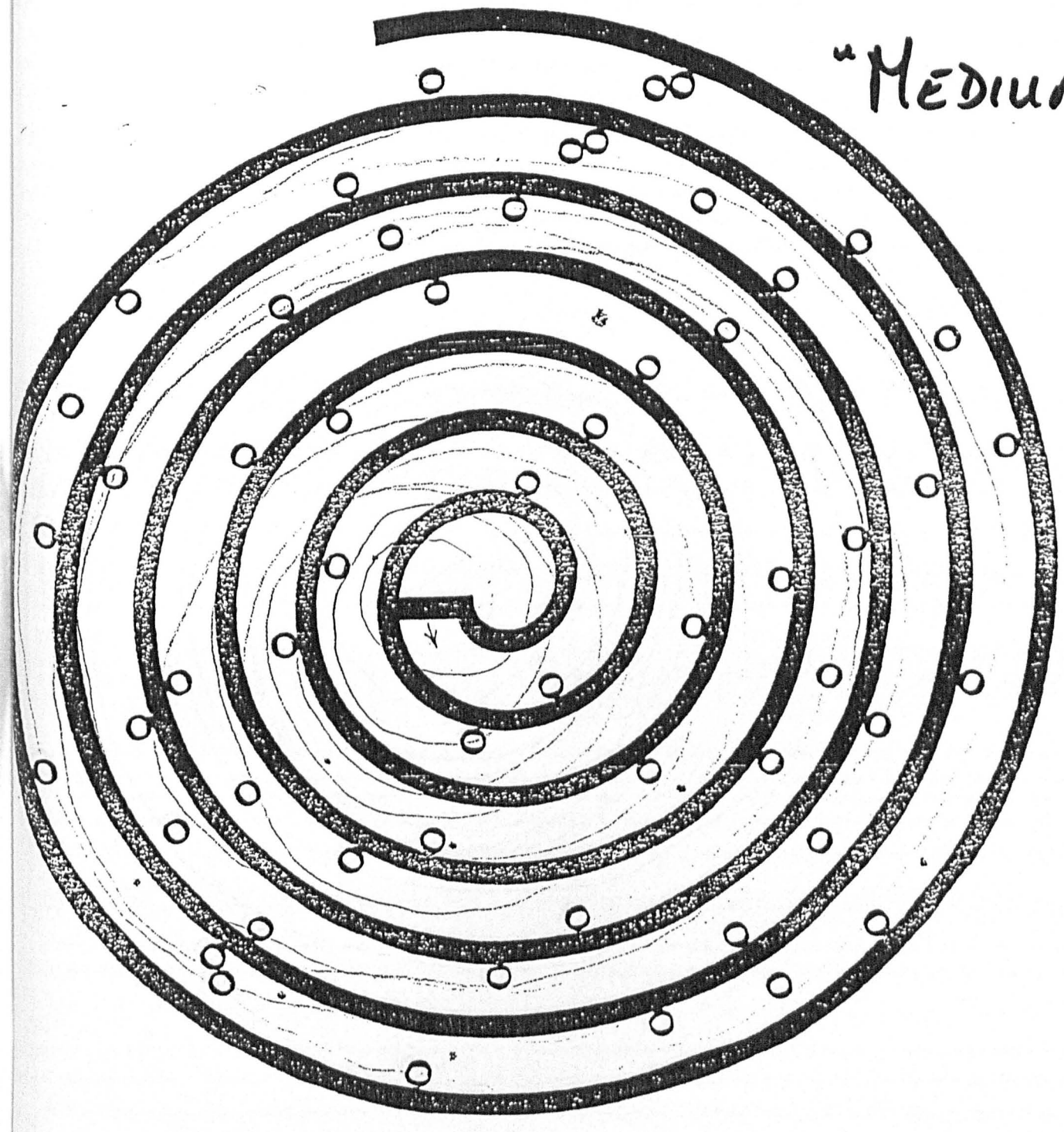


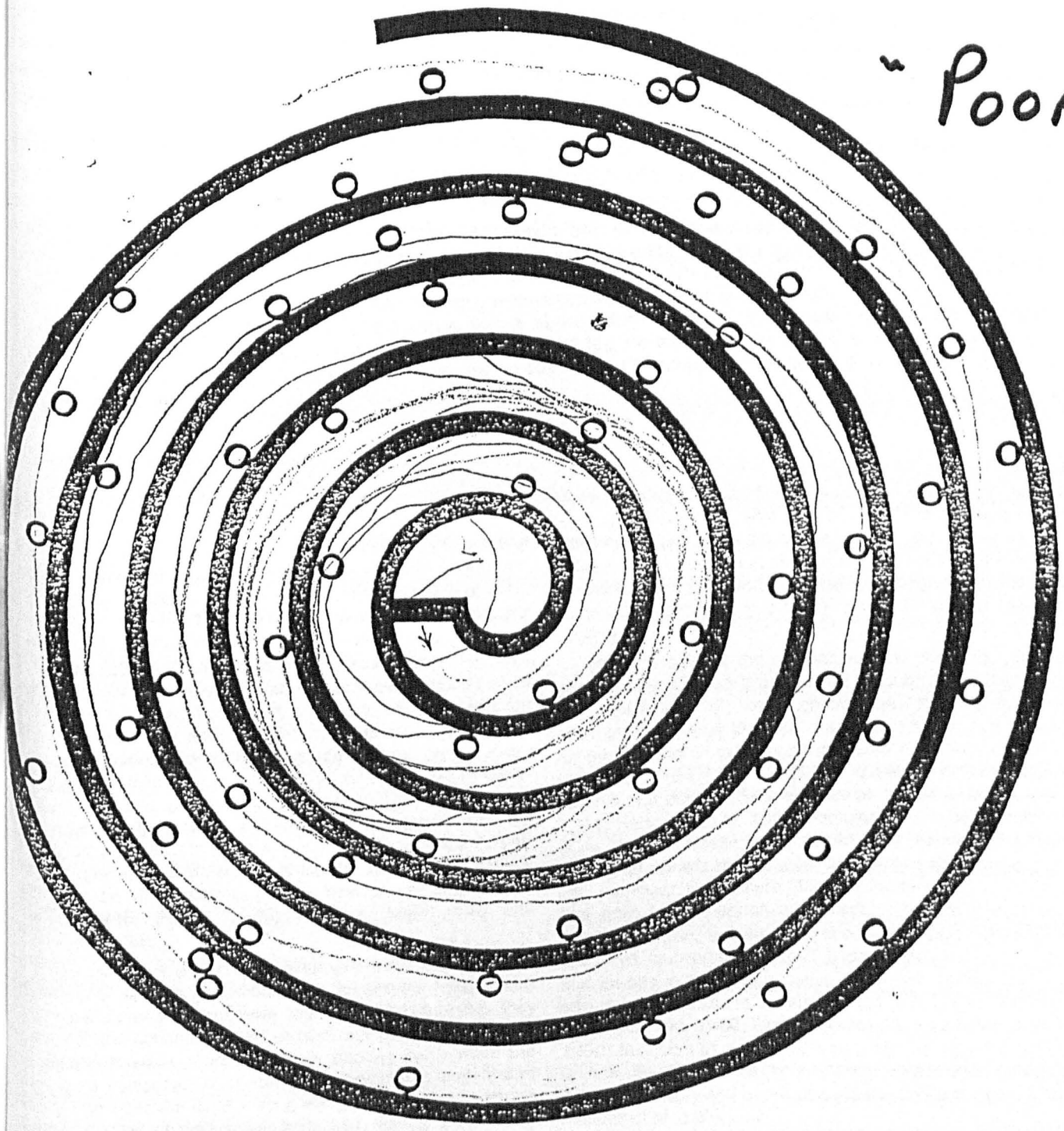




Appendix F : Examples of Gibson Spiral maze completed by EW.




"Medium"


"POOR"

Intervention Effectiveness: Evidence from a Case Study of Ideomotor and Ideational Apraxia

Jenny Butler

This paper considers the characteristics of apraxia and presents a case study which explores the effectiveness of tactile and kinaesthetic stimulation as an intervention strategy, in addition to visual and verbal mediation input, in the rehabilitation of a man with ideomotor and ideational apraxia following a head injury. Using an ABA design, quantitative measures were used to monitor change in motor performance. The results indicated some significant differences in measured performances between baseline and intervention phases and provided limited evidence of the effectiveness of additional sensory input. The implications for occupational therapy are discussed as are the limitations of the study.

Introduction

Adult-onset apraxia* is an impairment of the ability to perform skilled, learned or purposeful movement *not* due to any primary motor or sensory deficit, nor attributable to lack of comprehension, attention or willingness to perform the movement (Geschwind, 1975; Kirshner, 1991; Croce, 1993; Tate and McDonald, 1995). Researchers and clinicians agree that such a definition is problematic in that it describes the condition by exclusion: by saying what it is not. Also, such a definition does not take into account that many people with apraxia have, in addition, *coexisting* language deficits and/or motor and sensory deficits. It is the clinician who must try to determine the extent to which such impairments contribute to the dysfunction. Indeed, authors have suggested that the diagnosis of apraxia is often a function of the clinician's personal experience and 'expert' ability in being able to judge what can be attributable to primary deficits and what cannot (Miller, 1986; Poeck, 1986; Kirshner, 1991; Tate and McDonald, 1995).

Most clinicians and clinical researchers identify two main varieties of apraxia: ideomotor and ideational. People with *ideational apraxia* do not have the *idea* of what to do. They have lost the concept of how to carry out the required series of actions towards a set goal. They perform badly when the activity is composed of a series of movements and bizarre errors can often be seen during everyday activities, such as putting clothes on the wrong body part, eating food with a knife, buttering hot coffee or putting toothpaste on a razor (Mayer et al, 1990). The types of error made by these patients include the misuse or mislocation of objects, sequence errors and omission errors (DeRenzi and Lucchelli, 1988).

Ideomotor apraxia involves incorrect execution of the movement required. The person *does* know what to do, but is unable to implement the required action successfully. The kinds of error observed in clients with ideomotor apraxia have been detailed by Raade et al (1991) but, generally, the movements are observed to be clumsy and awkward with impairment in the timing, sequencing and spatial organisation of movement (Tate and McDonald, 1995). Ideational and ideomotor apraxia may present singly in an individual or together.

The exploration of the neuroanatomical correlates and the mechanisms underlying apraxia, as a route to understanding the organisation of motor control, has been a focus of research for several decades. Particular interest has centred on the location of the lesions that lead to apraxia (Basso et al, 1980; Agostoni et al, 1983; Kertesz and Ferro, 1984; Faglioni and Basso, 1985; Berthier et al, 1987; Selnes et al, 1991; Halsband et al, 1993; Papagno et al, 1993; Buxbaum et al, 1995; Classen et al, 1995). The majority of authors agree that apraxia results most frequently from lesions in the left, dominant hemisphere. There is evidence from research that both frontal lesions and posterior parietal lesions can result in apraxia, as can disruption of pathways between the two lobes (subcortical lesions). Some research has indicated that lesions in the corpus callosum or right hemisphere may also result in apraxia in certain cases, but these are less common (Archibald, 1987; Graff-Radford et al, 1987; Mozaz et al, 1990; Buxbaum et al, 1995). A theoretical concept of apraxia is now established as a multistep, multimodal model of impaired sensory and motor processing (De Renzi et al, 1982; Riddock et al, 1989).

Other research has explored the relationship between aphasia and apraxia which frequently coexist (Selnes et al, 1991; Papagno et al, 1993). There has been some interest recently in using kinematic analysis of limb actions (for example, acceleration and velocity patterns, trajectory of movement, joint angles and hand position) to determine differences with apraxia rather than relying on observer judgement (Poizner et al, 1990; Hermsdorfer et al, 1996). Such research

Jenny Butler, BSc, DipCOT, SROT, TCert(HE), Principal Lecturer, Rehabilitation and Disability Studies, School of Health Care, Oxford Brookes University, Dorset House, 58 London Road, Headington, Oxford OX3 7PE, and Honorary Research Assistant, Rivermead Rehabilitation Centre, Oxford.

*The term 'apraxia' is used throughout this article in keeping with neuropsychological research literature rather than 'dyspraxia' which is used more appropriately in the paediatric field concerning developmental dyspraxia in children.

projects have shown impairments in the timing of movements, disruption to the spatial-temporal dimensions of movement, and the over-use of proximal musculature to compensate for impairment of control in distal musculature in people with apraxia.

Research concerning the effectiveness of *rehabilitation* in apraxia is sparse, but has suggested that compensatory strategies may be the most effective approach (Wilson, 1988; Pilgrim and Humphreys, 1994). Others have proposed that a visual and verbal mediation strategy combined with appropriate tactile and kinaesthetic input provides the multimodal stimulation needed to facilitate movement in apraxia (Ayres, 1985; Fraser and Turton, 1986; Goodgold-Edwards and Cermak, 1990; Cermak, 1991; Cicerone and Tupper, 1991; Croce, 1993). In practice, this involves cueing people with apraxia to *look* at what they are doing and where they are going, as well as demonstrating activities or movements to give a visual model of how the activity or movement is to be performed. This strategy is accompanied by encouraging the person to *verbalise* what he or she wants to do and what he or she has done (for example, 'I want to open my hand', 'I want to reach for a cup', 'I am stretching my fingers out' and 'I am closing my fingers around a cup'). Croce (1993) suggested that such visual and verbal mediations should be linked with additional tactile and proprioceptive stimulation in order that the person with apraxia has opportunities to use a maximum amount of available information for planning and producing motor actions despite his or her limitations in sensory-motor processing and integration. Butler (1996a, b) has shown some evidence that such a sensory stimulation programme improves aspects of upper limb movement and activity in a case of ideomotor apraxia following severe head injury.

This paper presents a single case experiment which tested the hypothesis that the addition of sensory stimulation to a programme involving verbal and visual mediation of upper limb movement would improve motor performance in a man with ideomotor and ideational apraxia due to head injury.

Method

Case background

Mr W, a 66-year-old man, sustained a severe head injury after being knocked from his bicycle. He had a complex skull fracture and a left hemisphere subdural haematoma with cerebral contusion. After neurosurgery, he required drainage of some mild hydrocephalus. His post-traumatic amnesia was between 6 and 8 weeks.

When seen 4 months post-trauma, Mr W was ambulant, had mild weakness of his right hand and was mostly independent in self-care. He had a fluent dysphasia with a virtually unstoppable flow of speech, a limited attention span, an inability to hold information if task demands were high and no insight into his difficulties.

Tests used to assess for apraxia comprised a series taken from the research literature:

1. *Imitating gestures* (De Renzi et al, 1980). Twenty-four movements are carried out in front of the patient who is asked to reproduce these movements immediately afterwards from memory using the unaffected limb (ipsilateral to the lesion) to avoid the confounding variables in hemiparetic limb performance. Mr W scored 30 out of a possible 72, indicating a severe deficit.
2. *Gestures* (meaningful and non-meaningful) to both verbal command and a copying instruction (Haaland and Flaherty, 1984). Mr W scored 32 out of a possible 40.
3. *Pantomimed use of objects* (Haaland and Flaherty, 1984; Riddock et al, 1989; Alexander et al, 1992). Objects are presented in three conditions: (a) verbal instruction, 'Pretend to hold a ... and show me how you would use it'; (b) visual presentation of the object to the patient without touching it, 'Show me how you would hold and use one of these'; and (c) giving the object to the patient, 'Show me how you would use this'. Examples of objects used in these tests are a hammer, razor, comb, pen, key and toothbrush. Mr W scored 20 out of a possible 36. Errors were seen in all three conditions, but mostly in the verbal and visual sections.
4. Test for *ideational* apraxia using several objects in a sequence (De Renzi and Lucchelli, 1988). Mr W scored 0 out of a possible 12, indicating marked impairment. He was unable to choose or use any of the objects in an appropriate manner.
5. Screening test for visual agnosia to ensure that any difficulties with objects were not attributable to problems in recognising the objects themselves. Mr W scored 17 out of a possible 20, indicating good awareness and identification of objects and their use.

In addition to these tests, analysis of performance in functional tasks in a naturalistic setting (Arnadottir, 1990) was a critical part of the diagnostic process.

Observed errors in movement performance during these assessments were typical of those made by people with ideomotor apraxia (Raade et al, 1991) and included incorrect positioning of fingers and hand, wrong plane of movement, verbalisation of movement rather than doing the action, poor sequencing and timing of movement, inappropriate object pantomimes and use of the body part as the object itself. Mr W's inability to use objects in a sequence and his incomprehension of any bizarreness in his use and placing of objects indicated a severe ideational apraxia in addition to the ideomotor apraxia.

This research had the approval of the local health trust ethics committee. All the normal ethical guidelines for informed consent were followed and Mr W agreed to participate in the research project.

Experimental design

Using an ABA design, the study was carried out *in addition* to Mr W's normal daily physiotherapy and occupational therapy sessions. His occupational therapy outside the research project concentrated on functional activities in the kitchen and workshop.

Baseline phase (A): Verbal and visual mediation strategies with hand and arm movements for both upper limbs (see Appendix 1 for details). Measurements were taken pre and post each therapy session using three tasks: Nine Hole Peg Test (NHPT, Mathiowetz et al, 1985); Gibson Spiral Maze from the CAPE (Pattie and Gilleard, 1979); and a bilateral nut and bolt test (non-standardised). These tests were chosen for being quick, reliable, available in most occupational therapy departments, reproducible, and giving quantitative scores.

Intervention phase (B): The baseline programme *plus* a sensory stimulation protocol (tactile and proprioceptive input using different sensory pathways applied to both upper limbs; see Appendix 1 for details). Measurements were again taken pre and post each therapy session using the same tasks as for the baseline phase.

Post-intervention phase (A): A return to the baseline protocol (that is, without sensory input). Measurements were taken pre and post each therapy session as before.

These sessions were carried out as far as possible at the same time and place daily by the same therapist. Baseline and intervention phases were each over a 3-week period, collecting 13 data points for each phase. The post-intervention phase was curtailed, lasting just 2 weeks with 9 data points, because Mr W was discharged home.

Results

Statistical rationale

Visual inspection is the most commonly used method of examining single case data, looking at graphical displays across the different phases of the experiment. Barlow and Hersen (1984) suggested that reliance on such methods leads to a greater likelihood of making a type II error (that is, concluding that the intervention did not produce a veridical effect) than if reliance is placed upon statistical analysis.

In order to determine a treatment effect, Prada and Tallis (1995) calculated the difference between the slopes of regression lines in each phase of the case study design they used. Such statistical methods are only appropriate if the regression lines are meaningful as a predictor of one variable, given the value of another variable (Munro and Page, 1993; Bryman and Cramer, 1994). In the case of Mr W, the variability in performance from one day to the next was great. This meant that the variance accounted for by linear regression analysis was low or minimal across most scores and in all the phases of the experiment (r values ranging from 0.005 to 0.51, with most correlations being lower than 0.32). The only exceptions to this were the errors made in the Gibson spiral maze in the baseline period where $r = 0.92$ for the right hand and $r = 0.71$ for the left hand. The likely explanation of this phenomenon is that it represented an element of motor learning by the patient on this task. Given such variability in the scores, linear regression was not deemed an appropriate method of analysis for these case data.

The data were next assessed, therefore, for serial dependency using autocorrelation statistics. The extreme variability in the scores from day to day resulted in no serial dependency being found in the data set in the majority of scores, the only exception being the error scores on the Gibson spiral maze for both right and left hand in the baseline period only. Barlow and Hersen (1984, p288) stated that data revealing no serial dependency 'can be treated as independent observations and can be subjected to conventional statistical analyses'. Thus, in this case analysis, conventional F tests were used to analyse differences between the experimental phases and to determine intervention effects. Post hoc tests used in this analysis were the Bonferroni and Student-Newman-Keuls.

Findings

No differences were found in any of the measures in any phases immediately after intervention. This meant that there was no immediate treatment effect of either the verbal and visual mediation strategy alone, or with the addition of sensory stimulation, on measured motor performance. The variability of motor performance in this case study is illustrated by examples of results given in Fig. 1 showing time taken to complete the Gibson spiral maze (right hand), Figs 2 and 3 showing time taken on the bilateral nut and bolt task, and Fig. 4 showing time taken in the NHPT (left hand). Because there

Fig. 1. Time taken for maze task – right hand.

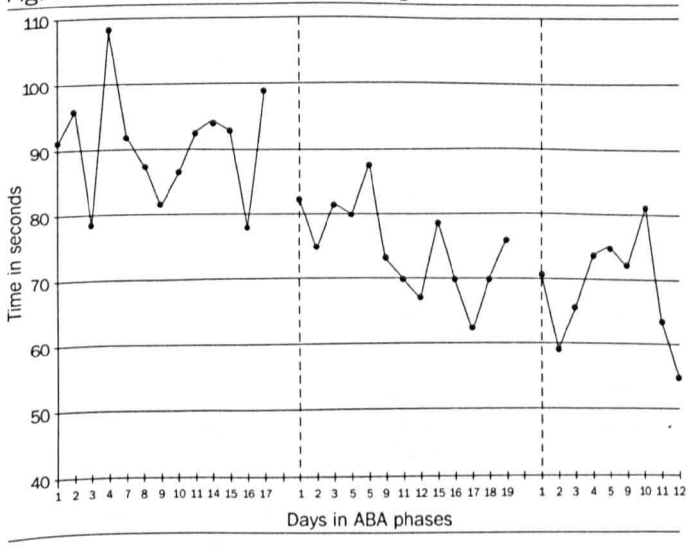


Fig. 2. Time taken for bilateral nut and bolt task – on phase.

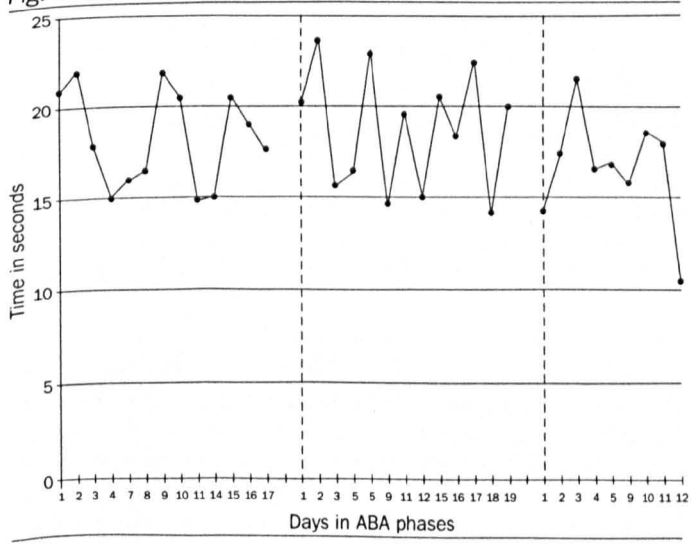


Fig. 3. Time taken for bilateral nut and bolt task – off phase.

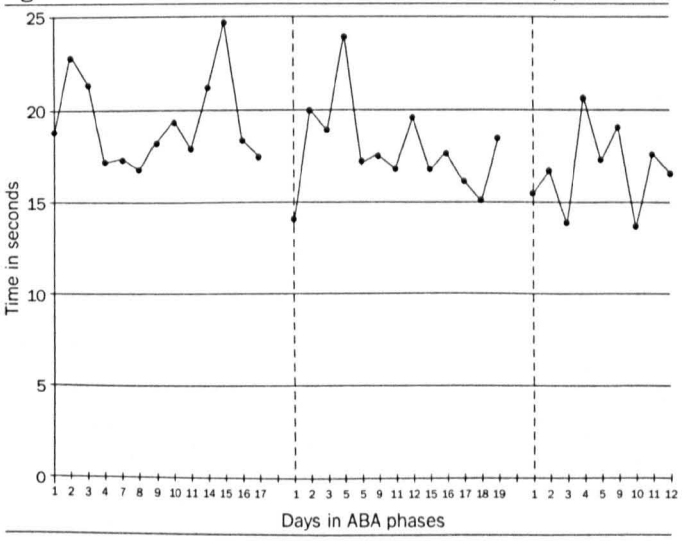
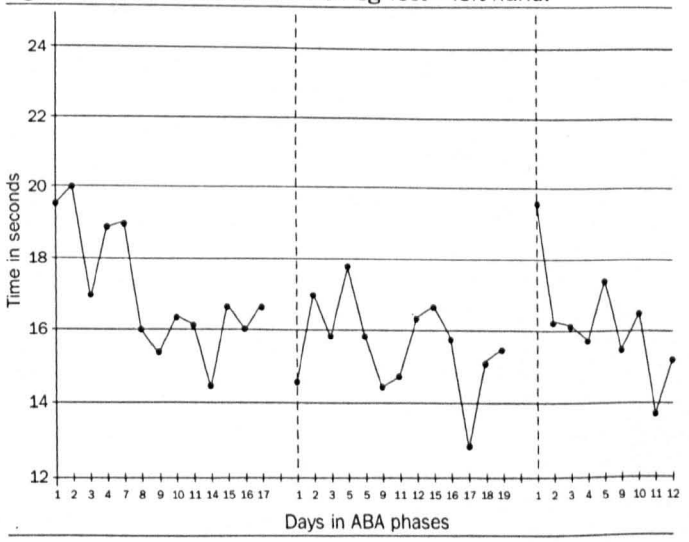


Fig. 4. Time taken for Nine Hole Peg Test – left hand.



were no significant differences between pre and post therapy scores, those used in Figs 1-4 were the ones taken at the end of each therapy session. These particular scores were also used in an analysis of variance to determine the effects of intervention.

Details of descriptive analysis of the data are given in Table 1. Motor performance in the **right hand** measured by the NHPT showed a reduction in mean scores in the intervention phase compared with the baseline, with reduced variability of performance as demonstrated by smaller standard deviation scores and reduced range. This reduction in mean scores and performance variability continued into the post-intervention phase. A pattern of improving motor performance over time was also demonstrated by the time taken to complete the spiral maze, the exception being that variability in performance increased slightly in the post-intervention phase of the experiment. As this right hand had a mild hemiparesis arising from the head injury, such changes in scores could be attributable in part to motor recovery. The normal timing for the spiral maze in a community elderly population is given as 106 seconds, and Mr W was scoring well below that from the outset.

Performance with the **left hand** (ipsilateral to the lesion) also showed a reduction in mean score in the NHPT with a reduction in variability of performance in the intervention phase, but this improvement in performance was not sustained into the post-intervention phase. As the ipsilateral limb is the one showing the 'purest' apraxic performance (that is, without the interfering variable of hemiparesis and motor recovery), these scores may be considered to be the most indicative of intervention effects. The time taken to complete the spiral maze in the left hand was also reduced during the intervention phase but there was an increase in variability of performance. Improvement was sustained into the post-intervention period with reduced variability. Given that the normal score for the NHPT is 18 seconds, Mr W was scoring well below that in his left hand from the outset of the experiment so had very few opportunities for 'improvement'. Such improvement as was demonstrated might be evidence for reduced apraxic problems because any motor learning is likely to have peaked in the baseline period.

Little change was seen in the time taken for the bilateral task of screwing a wing nut onto a bolt and removing it in any of the experimental phases. Mr W used a variety of strategies to complete this task, each hand performing different roles, with no restriction being placed on him during the performance. This may account, in part, for no difference being observed. Alternatively, the task might not have been challenging enough to demonstrate any changes in motor abilities due to ceiling effects.

Statistical analysis provided evidence that there was an intervention effect in some, but not all, of the motor performance scores obtained. The **right hand** motor performance showed no significant intervention effect with the NHPT, $F=2.88$ (2,32), *NS*. Time taken to complete the spiral maze with the right hand, however, did show intervention effects, $F=22.24$ (2,32), $p<0.001$, with the post hoc tests (Bonferroni and Student-Newman-Keuls) indicating that a difference occurred between the baseline phase and the subsequent intervention and post-intervention phases. Errors made with the right hand on the spiral maze task showed a significant difference between the baseline phase and the subsequent intervention and post-intervention phases, $F=7.03$ (2,32), $p<0.01$. This result has to be interpreted with caution, though, given the serial dependency shown in these data in the baseline phase which could bias the F tests and lead to type I and type II errors (Barlow and Hersen, 1984).

The **left hand** motor performance also showed a mixed effect. The NHPT demonstrated significant effects of intervention, $F=3.39$ (2,32), $p<0.05$, with the post hoc tests indicating that the difference occurred between the baseline phase and the intervention phase. Time taken to complete the spiral maze with the left hand showed no intervention effect however, $F=2.83$ (2,32), *NS*, contrasting with the time taken with

Table 1. Synopsis of results

		NHPT (secs) (norm = 18 secs)		Maze time (secs) (norm = 106 secs)		Maze errors	
		Right hand	Left hand	Right hand	Left hand	Right hand	Left hand
Baseline phase	Mean	26.64	17.06	90.22	65.13	24.15	23.46
	SD	10.08	1.66	8.65	4.74	8.53	7.28
	Range	20.47–59.06	14.47–19.91	76.03–107.19	56.5–71.72	14–40	12–39
Intervention phase	Mean	22.44	15.52	74.88	60.91	17.77	16.85
	SD	2.48	1.23	7.13	8.35	3.63	4.12
	Range	18.25–26.25	12.75–17.66	62.63–87.25	53.28–85.75	,10–26	11–24
Post-intervention phase	Mean	20.26	16.21	68.46	58.65	14.66	15.11
	SD	1.8	1.6	8.26	5.78	4.66	4.4
	Range	17.59–23.22	13.69–19.59	55.25–81.28	50.66–69.28	9–22	10–22

Bilateral nut and bolt task		On (secs) (norm = 15.5 secs)	Off (secs) (norm = 16 secs)
Baseline phase	Mean	18.32	19.38
	SD	2.74	2.43
	Range	14.83–22.16	16.81–24.69
Intervention phase	Mean	18.59	17.8
	SD	3.32	2.48
	Range	13.56–23.35	13.78–23.91
Post-intervention phase	Mean	16.5	16.69
	SD	3.06	2.28
	Range	10.59–21.43	13.41–20.63

the right hand. Errors made with the left hand on the spiral maze, as with the right hand, were significantly different between the baseline phase and the subsequent intervention and post-intervention phases, $F=7.3$ (2,32), $p<0.01$, but, for the same reasons as before, the results must be viewed with caution.

For both hands, Mr W may have been performing at ceiling level on both the NHPT and the timing of the spiral maze. Motor learning may be one explanation of the improvement in performance but does not explain, however, the differences in the phases of the experiment and the possible 'intervention effect' unless such differences were an artefact of the experiment.

The bilateral nut and bolt task 'on' phase demonstrated no differences between phases, $F=1.4$ (2,32), *NS*, but the 'off' phase showed a significant difference between the baseline and post-intervention phases (as indicated by the post hoc tests), $F=3.45$ (2,32), $p<0.05$. No plausible explanation can be offered for this result.

During the experimental period of 8 weeks, Mr W completed three repeated apraxia batteries. Some improvements were demonstrated after the intervention was completed (see Table 2), but there was an increase in use of the body part *as* the object in test three resulting in a reduced score for Mr W. The most noticeable difference in the diagnostic tests was in Mr W's ability to manage the use of multiple objects in a meaningful sequence in the ideational apraxia test, moving from a complete inability to perform any meaningful activity before the experimental (sensory) intervention to a good and appropriate use of the objects after the intervention. However, this was still accompanied by profuse vocal outflow as at the start of the experiment.

Table 2. Apraxia tests: synopsis of results

	Time 1 (week 1 – baseline)	Time 2 (week 4 – before intervention)	Time 3 (week 8 – after intervention)
1. Imitating gestures (score out of possible 72) (De Renzi et al, 1980)	30	28	47
2. Gestures, meaningful and non-meaningful (score out of possible 40) (Haaland and Flaherty, 1984)	32	29	27
3. Pantomimed use of objects (score out of possible 36) (Haaland and Flaherty, 1984)	20	Not tested	15
4. Test for **ideational** apraxia (score out of possible 12) (De Renzi and Lucchelli, 1988)	0	0	10

During the period of the experiment, Mr W's Barthel Index score remained constant at 18 out of a possible 20, needing help in both stair climbing and bathing. By the time he was discharged home, however, he was competent in his self-care and in simple domestic tasks, if each task was broken down into small manageable parts and some verbal prompting was given.

Discussion

Evaluation of motor performance in this case study showed mixed results related to an intervention effect. There was some support for the original hypothesis that the *addition* of sensory stimulation to an intervention protocol, involving verbal and visual mediation of motor activities in the upper limb, would increase motor performance in a man with ideomotor and ideational apraxia due to head injury. Motor recovery could explain the improvements in performance in the right hand, but those improvements seen in the performance in the left hand might indicate the effectiveness of intervention.

The use of tests to measure changes and to determine an intervention effect was problematic because Mr W was performing at quite a high level from the outset. This meant that ceiling effects may have been affecting results, and that more challenging and sensitive tasks might have given evidence of a greater degree of intervention effect. This has to be counterbalanced by the rationale for choosing the particular tests in the first place: quick, reliable, available in most occupational therapy departments, reproducible, and giving quantitative scores in task completion. Whilst it could be argued that timing (as used in the NHPT and the Gibson spiral maze) is a very crude measure of movement performance, it can certainly indicate at least a level of efficiency of movement production and therefore be an indicator of change or improvement. Systematic and careful observation of performance errors in everyday tasks might always be considered the more attractive and realistic measure of change in apraxia, but the difficulties of such methodologies have been well documented in the literature (Mayer et al, 1990; McDonald et al, 1994) with researchers agreeing that the reliability of observed behaviour is problematic. To overcome this, more specialised equipment for kinematic analysis of motor performance was used as a part of this research project to measure change in this patient during a naturalistic task, and will be reported in a later paper.

Variability of motor performance was a feature of this case study. This had implications for statistical analysis but also supports previous evidence (Butler, 1996a, b) that apraxic movement is unpredictable, unreliable from day to day, and very variable. If such variability is acknowledged to be the rule rather than the exception in apraxia, then this should be manifest in occupational therapy practice. A single assessment of clients with this condition would seem to be inappropriate as a basis for making judgements of performance (for example, in skill and safety) and in recommending care plans. It is suggested that multiple assessments, to discover the *range* of performance in which the client operates, would be a better indicator of function for people with apraxia. In addition, the outcome measures in evaluating apraxia intervention should include reports of the variability of performance, both functional ADL and specific standardised motor tasks. A reduction in variability can be considered to indicate improvement: the patient is shown to be performing more reliably and predictably.

The rationale for the intervention was based on previous research and published papers on the rehabilitation of apraxia (Ayres, 1985; Fraser and Turton, 1986; Goodgold-Edwards and Cermak, 1990; Cermak, 1991; Cicerone and Tupper, 1991; Croce, 1993) and provided some support for the argument that multimodal stimulation enhanced movement performance in people with apraxia. The intervention protocol described here was designed to take a total of about 30 minutes for each daily session and was judged to be a feasible and reproducible regime for most other rehabilitation departments or specialist brain injury units.

In determining neuroanatomical correlates with the apraxic condition, details of this particular case were similar to those explored in other research in that Mr W did have a left hemisphere lesion. No magnetic resonance imaging (MRI) scan was available, however, to give detailed information of the precise location and extent of this lesion. Clearly, it would have been useful to comment on how this particular case adds to the body of knowledge in determining the origins of disruption to praxis but, in the absence of a detailed MRI scan, this was not possible. Future case studies should, if

possible, include such information. Mr W also had an aphasic condition which, again, replicated details of previous studies looking at the relationship between apraxia and aphasia.

Some differences were shown in the clinical diagnostic test scores following the sensory intervention phase of the experiment. These diagnostic tests have a rather crude classification system with the middle category of 'impaired performance' found in most of the tests being so broad as to be fairly useless in monitoring *subtle* changes in an individual. The tests will reflect larger changes though (for example, from 'unable' to 'recognisable performance') and this is what was seen in the case of Mr W. Whether such changes could be attributable to this experimental procedure, to 'natural recovery', to other therapeutic interventions occurring concurrently in the rehabilitation programme or to a combination of all three, it is not possible to say.

Difficulties encountered in this case study research included not being able to obtain data and carry out the intervention every single day for a variety of reasons. Patient fatigue, home visit, hospital or other unexpected appointments, and time constraints of other therapist duties took priority. These are normal everyday occurrences in the rehabilitation setting and a part of the reality of practice life. It was considered that any evaluation of intervention had to cope with, and be robust enough to withstand, these realities of clinical life in order to be perceived as meaningful and relevant to other rehabilitation settings.

Conclusion

There is some evidence presented here, in one clinical case of ideomotor and ideational apraxia, that the addition of a sensory stimulation protocol had an effect on motor performance above that of an intervention using verbal and visual mediation strategies on their own. The original hypothesis was partially supported, although the relative insensitivity of the measures used was problematic. Further analysis and more case studies need to be undertaken to replicate or refute these results, and evaluate whether changes in *functional* outcome are achieved through this process, in a variety of patients with both ideational and ideomotor apraxic conditions of varying severities.

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Appendix 1. Intervention details

Testing (NHPT, Gibson spiral maze, nut and bolt test) with both hands was done before and after each session in all experimental phases.

A – Baseline phase (20 – 30 minute intervention)
Explanation to the patient that the session will involve moving his hands to try to help him concentrate in getting good and accurate movements. Patient to be seated at a table of the correct height. The therapist sits opposite the patient. With each hand, three types of activity are performed:

(i) Touching targets

- Hands should be positioned on the edge of the table where person is sitting.
- Ask patient to move either the left or right hand (randomly) to touch a disc (for example, draughts counter).
- Occupational therapist moves the counter around on the table between each 'hit'.
- Try to get patient to move quickly and accurately.
- Instruct patient not to talk whilst he is doing this, but to concentrate on looking and moving.

(ii) Gesture copying

- Using simple, single gestures, ask patient to copy the occupational therapist's actions, for example:
  - Make a fist
  - Point finger
  - 'Thumbs up'
  - Spread fingers
  - Little finger straight, rest of hand curled.
- Using bilateral movements, ask patient to copy the occupational therapist's actions, for example:
  - Clap hands (in different orientations)
  - Drum fingers together
  - Link thumbs and pull
  - Make a 'cat's cradle'.

(iii) Verbalising hand position or movement

- With simple gestures and movements ask patient to SAY what he is doing AS he is doing it. The occupational therapist can repeat some of the gestures and movements as before, for example:
  - 'I am pointing my finger'
  - 'I am clapping my hands'
  - 'I am bending my elbow'
  - 'I am tapping the table'
  - 'I am bending my thumb'
  - 'I am stretching out my arm'.

B – Intervention phase (20 – 30 minute intervention)

(i) Hand and arm movements

- Do a short session on the three types of hand/arm activities and movements, as in baseline phase.
  (a) Touching targets
  (b) Gesture copying
  (c) Verbalising hand position or movement.

(ii) Sensory input

- Spend 20 minutes on sensory input to forearms, hands and fingers using all modalities:
  - Deep pressure massage using oil or cream
  - Sharp touch using nail brush in rotating movement on skin
  - Proprioception, putting weight through arms and wrists, pushing/pulling activities
  - Soft touch using soft cloth with long strokes
  - Self-touch; patient stroking and smoothing hands over forearms and hands.

A – Post-intervention phase (20 – 30 minute intervention)
A return to exactly the same protocol as for the baseline phase.

Book Reviews

EVIDENCE-BASED HEALTHCARE: HOW TO MAKE HEALTH POLICY AND MANAGEMENT DECISIONS. *J A Muir Gray. Churchill Livingstone, 1997. 270pp. £16.95. ISBN 0 443 05721 4.*

This practical text is aimed primarily at people who have to make decisions about the best use of resources for patient groups. However, the book demystifies the language of evidence-based health care and the process of evidence-based decision making, so making it accessible to a wider audience that might include practitioners, lecturers, students and would-be researchers.

The book is unlikely to be read from cover to cover but it serves as a well-referenced source of information on evidence-based everything, including evidence-based organisations, evidence-based chief executives and evidence-based litigation, as well as evidence-based policy making and purchasing.

Muir Gray takes the view that decision making in health care is currently based on opinion informed by values and available resources. A shift is necessary to a more scientific approach to decision making in the current economic and political climate. He addresses this by not only analysing and explaining the components of evidence-based decision making, but also exploring the skills, techniques and media needed to carry it out.

The book addresses issues such as the nature of decision making in health care, and how to search for evidence, appraise the quality of research and use the evidence in practice. It examines outcomes and effectiveness and dimensions of patient satisfaction and explores the evidence-driven audit cycle. Systematic reviews, decision analysis and randomised controlled trials all come under scrutiny, although the breadth of coverage of evidence-based health care limits depth of exploration into the different facets. Nevertheless, this is a comprehensive text that touches on many components of evidence-based health care to aid the development of what Muir Gray refers to as the 'compleat' health care manager.

The book avoids an academic style of writing, using everyday language that is readily understood. However, an overuse of abbreviations without a glossary can be a little frustrating. The book is presented as a source of information, is well-illustrated and draws on many examples, albeit from medicine. The material is presented in bite-sized pieces which make it accessible and digestible. The book is not without a subtle humour and light-heartedness that adds to its readability.

This book is recommended to all those trying to come to terms with yet another fashionable phrase now pervading practice and care delivery. Muir Gray recognises that change to a more scientific approach to decision making might not be a comfortable one, so he urges professionals to view it not as a management imperative but as an intellectual challenge.

Auldeen Alsop,
Course Director, BSc(Hons) Occupational Therapy, South Bank University, London.

Computer-Assisted versus Teacher-Directed Teaching of Community Living Skills in People with a Mild Learning Disability

M M Nicol and A Anderson

An experiment was carried out in which adults with a learning disability of the mild type undertook either computer-assisted or teacher-implemented instruction in community living skills, with a third group acting as a control group. All groups were pre-tested on a standardised psychometric measure of community living skills (the HANC 2 subscale of the Hampshire Assessment for Living with Others test), after which the experimental groups received one half-day per week training in such skills as money handling and budgeting for 3 months, when all groups were reassessed. It was found that both the teacher-led and computer-assisted instruction groups appeared to gain more than the control group, but only the teacher-led group differed significantly from the control group. The computer-assisted group improved in their scores by an intermediate amount, with their gains being non-significantly different from either the control or the teacher-led groups. The strengths and weaknesses of the software used in the study are discussed.

Introduction

Computer-assisted instruction and learning disability

Connors et al (1986) defined computer-assisted instruction (CAI) as instruction implemented by a computer. This article uses the term computer-assisted instruction as a generic description for computer programs to support learning; another popular term is computer-assisted learning. The former term is preferred by the authors since it relates closely to the computer-led, drill-and-practice software in widespread use in schools and adult training centres, whereas the latter term is better applied to more user-led exploratory software which tends to be used less often with the population that is under study in the present work. Gerard (1967) postulated that CAI had a number of benefits compared with conventional teacher-led instruction, including (a) greater possibilities for students to learn at their own pace, (b) personalised tutoring and (c) more accurate appraisal and documenting of student progress. Whilst these claims have occasionally been met with scepticism (for example, Sewell, 1990) and the quality of educational software has come in for criticism (for example, Self, 1987), it is nevertheless the case that CAI has found widespread use throughout the education sector.

The effectiveness of CAI has been a controversial issue over the last decade. Kulik et al (1983) and Kulik and Kulik (1987, 1989, 1991) conducted meta-analyses of CAI in mainstream education and claimed that computer-based learning raised students' scores significantly in formal examinations, that this gain was maintained, and that students held positive attitudes to CAI. Clark (1991, 1992) criticised these findings, arguing that if the same teacher is used for the control and experimental groups and the duration of the study is longer term, then the effect size would be reduced. Fletcher-Flinn and Gravatt (1995) carried out a subsequent meta-analysis that paid heed to the methodological issues raised by Clark. Their results confirmed Kulik and Kulik's (1987) earlier study in terms of the sizes of effects. However, in the few studies that controlled for duration, teaching materials and the teacher, there was no advantage for CAI. Fletcher-Flinn and Gravatt (1995) contended that the apparent gain in proficiency came about because of the superior quality of CAI materials, rather than some intrinsic aspect of computers *per se* as vehicles of instruction.

CAI is very widely used within the field of learning disability. Overall, there is a reasonable body of evidence that suggests that CAI has its uses in the field of learning disability, although there are concerns about which learning method is most effective, the cost of the hardware and the lack of specialised and/or good quality software (Braun, 1981; Coburn et al, 1982; Hawkridge, 1983; Maddison, 1983; Powers, 1987; Self, 1987).

Training in community living skills

There is a considerable body of literature on the training of people with a learning disability in community living skills using conventional teaching programmes. The community living skills have been very varied and include cooking (for example, Martin et al, 1982), grocery shopping (for example, Matson and Long, 1986), banking (for example, Zencius et al, 1990), telephone training (Leff, 1975), pedestrian skills (for example, Matson, 1980) and using public transport (for example, McInerney and McInerney, 1992). Studies vary in whether the training is primarily classroom based (for example, Matson, 1980), in vivo (for example, Rudrad et al, 1984) or a combination of both (for example, Nietupski et al, 1983). Because of the time-intensive and labour-intensive nature of the training programmes involved, the number of participants

M M Nicol, PhD, MPhil, DipCOT, SROT, Senior Lecturer in Occupational Therapy, Department of Occupational Therapy, Queen Margaret College, Leith Campus, Duke Street, Edinburgh EH6 8HF.
A Anderson, BSc, PhD, Lecturer, Centre for Research into Interactive Learning, Department of Psychology, University of Strathclyde, Graham Hills Building, 40 George Street, Glasgow G1 1QE.