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Impairment- and activity/participation-directed interventions in progressive language impairment: Clinical and theoretical issues

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**Background:** There is a broad constellation of clinical syndromes in which the most prominent initial and ongoing symptom is deterioration in spoken or receptive language processing, reading, writing, or semantic knowledge. Despite the core language impairments, people with these disorders are likely to be under-referred for speech pathology services (Taylor, Kingma, Croot, & Nickels, 2008 this issue), and there is limited published research on speech pathology interventions in these disorders.

**Aims:** This paper reviews the published impairment- and activity/participation-directed interventions in semantic dementia and progressive aphasia to determine the current evidence base for clinical decisions about client suitability and selection of treatment goals, methods, and measures. We also identify questions that need to be addressed in future therapy research.

**Methods & Procedures:** We reviewed 15 reports of impairment-directed behavioural, pharmacological, and repeated transcranial magnetic stimulation treatments that implemented some level of experimental control. We reviewed a further 10 reports of interventions generally carried out without experimental control, targeting impairments, activity/participation limitations, education, carer training, or a mixture of these approaches.

**Outcomes & Results:** In the impairment-directed studies almost all participants showed improvement on treated items immediately post-treatment. Improvement was specific to treated items in most word retrieval studies, but there is a possibility that greater generalisation to other items or other language tasks/contexts may have occurred in treatments not targeting naming, and/or for clients with articulatory impairments. Without ongoing practice of therapy activities, treatment gains declined for all...
participants over a period varying from 2 months to between 6 months and 1 year. There was only one experimentally controlled activity/participation-directed intervention reported for people with progressive language impairments, although other reports of activity/participation-directed interventions described increased communicativeness and communication effectiveness for participants following intervention. Consistent with the aims of activity/participation-interventions, these interventions appear to have a greater impact on everyday communication outside the clinic than impairment-directed interventions, but these impacts need to be investigated and documented.

**Conclusions:** These studies highlight the need for rigorous research design to identify treatment, generalisation, and maintenance effects (Rapp & Glucroft, 2008 this issue), and to identify outcomes that go beyond improvement in the clinic on targeted words, language structures, and behaviours. It is important to tailor interventions closely to individual client needs, involve the spouse/carer in intervention, and ensure that clients have appropriate expectations about therapy. The patterns of therapy gain, limited generalisation, and decline following cessation of therapy activities in impairment-directed studies raise questions about the learning mechanism(s) involved. They also have implications for client suitability, and for choice of therapy items, and therapy delivery and duration.

**Keywords:** Primary progressive aphasia; Frontotemporal dementia; Semantic dementia; Treatment; therapy.

The past decade has seen a rapid growth in understanding the speech, language, and communication changes associated with various types of dementia (Bayles & Tomoeda, 1997; Bourgeois, 1991; Clare, 2001; Lubinski, 1995; Ripich & Horner, 2004; Royal College of Speech and Language Therapists, 2005). Of particular interest to speech pathology practice is the fact that a number of neurodegenerative diseases may result in clinical syndromes in which the most prominent initial and ongoing symptom is deterioration in language processing. The best known of these syndromes are primary progressive aphasia as defined by Mesulam and colleagues (Mesulam, 2001, 2003; Mesulam, Grossman, Hillis, Kertesz, & Weintraub, 2003), semantic dementia, characterised by severe anomia, comprehension deficits, and impaired object recognition due to loss of semantic memory (Snowden, Goulding, & Neary, 1989), and nonfluent progressive aphasia (Gorno-Tempini et al., 2004), in which nonfluent spontaneous speech, anomia, and speech sound production errors are the typical presenting symptoms. A wide range of other syndrome labels have also been used for progressive language impairments, including pure progressive anomia, primary progressive conduction aphasia, primary progressive apraxia of speech, language- or temporal-variant frontotemporal dementia, etc. (see Croot, 2008 this issue, for more details about syndromes involving progressive language impairment).

Despite the central nature of the language symptoms, in the past some speech pathologists have not perceived that they have a role in the management of people with progressive language impairments. Even now, speech pathologists may remain uncertain about the best management pathway for these individuals (Taylor et al., 2008 this issue). However, as McNeil and Duffy (2001, p. 475) note, the “presenting speech and language complaints can be strikingly similar to those of people with stroke-induced aphasia” (their emphasis). They therefore argue that decisions about treatment in progressive language impairments can be based on the same philosophical, clinical, theoretical, and practical considerations that apply in stroke-related aphasia. Although the nature of the disease process requires that clinicians modify treatment goals and approaches over time in response to (or even in anticipation of) disease progression (Rogers, King, & Alarcon, 2000), this is hardly a new skill: modifying therapy in response to changing client ability or
circumstances is standard practice and is required for any speech pathology client group (McNeil & Duffy, 2001). Individuals with progressive language impairment often present with additional cognitive, behavioural, or motor disorders, or develop these with disease progression (see Croot, 2008 this issue, for an overview). Therefore, as in stroke-related aphasia, the speech pathologist needs to be alert for co-occurring disorders, taking them into account in therapy planning and referring the individual for other healthcare services as needed.

The challenge for the speech pathology profession in working with clients with progressive language impairment is to apply, and as necessary adapt, the knowledge-base derived mainly from stroke-related aphasia to this emerging area of practice. Moreover, people with progressive language impairments are almost certainly under-referred for speech pathology services (Taylor et al., 2008 this issue). Therefore speech pathologists also have a role in promoting the suitability and availability of services for these clients to referring medical practitioners.

The need to provide speech pathology services for clients with progressive language impairments has been a clinical reality for some years (e.g., Craenhals, Raison-Van-Ruymbeke, Rectem, Seron, & Laterre, 1990; Holland, McBurney, Moosy, & Reimnuth, 1985; McNeil, Small, Masterson, & Tepanta, 1995; Northen, Hopcutt, & Griffiths, 1990; Schneider, Thompson, & Luhring, 1996). Nevertheless, there are few published studies of speech pathology interventions in this population, although the rate at which new studies are being reported is accelerating (see Appendix Tables 1 to 3). Pressing clinical questions about client suitability for the various therapy options, and about therapy efficacy, timing, and dose, await evidence-based answers. In the absence of published evidence-based practice guidelines, individual clinicians must evaluate the evidence, together with the theoretical and philosophical rationales for the various alternatives, in order to make a reasonable choice about the intervention(s) they will offer (Alarcon, Duffy, McNeil, & Rogers, 2000).

There are three main sources of information available to inform treatment planning for clients with progressive language impairments. First, descriptions of previous interventions currently constitute the best empirical information on which to base clinical decisions about client suitability, and treatment goals, methods, materials, and measures. In addition, psycholinguistic and learning theories can inform about how a particular treatment may work and for whom it is/is not suitable. Finally, expert opinion papers are a rich resource that offer suggestions about best practice based on extensive clinical and research experience with these and allied disorders (McNeil & Duffy, 2001; Robinson, 2001; Rogers et al., 2000; Snowden & Griffiths, 2000; Thompson & Johnson, 2006).

The aim of this paper is to assist clinicians to make choices about speech pathology service provision. We do this by summarising published intervention studies and identifying the theoretical issues that influence therapy planning. Along the way we highlight some questions that need to be addressed in future therapy research. We have organised the review according to whether the reported interventions were primarily directed towards remediating the client’s “impairment”, or improving their activity/participation levels. The terms impairment and “activity/participation” are taken from the World Health Organisation health classification system (as described in the practice guidelines of the American Speech-Language-Hearing Association, 2001). Impairment-directed interventions are designed to rehabilitate psychological or physical functioning of body structures (e.g., retrieval of lexical items, processing of syntactic structures within the language system, motor
control of speech articulators, neuronal function in language areas, etc.). Activity/participation-directed interventions aim to improve an individual’s ability to perform a particular task or action or participate in a desired life situation (e.g., express basic needs, participate in a conversation or a game of cards).

**IMPAIRMENT-DIRECTED STUDIES**

We include in this review of impairment-directed studies only published treatment studies that implemented a level of experimental control. Some studies were carried out in the context of clinical speech pathology therapy provision, others as experimental studies in laboratory settings outside the clinical arena. Studies using behavioural therapies are summarised in Appendix Tables 1 and 2. One of these (McNeil et al., 1995) compared the effect of a behavioural treatment alone with behavioural plus pharmacological treatment in a single case. There is another study reporting a pharmacological treatment only with a group of seven participants (Reed, Johnson, Thompson, Weintraub, & Mesulam, 2004). Finally, one pioneering study targeted a person’s impaired verb production using repetitive transcranial magnetic stimulation (rTMS, Finocchiaro et al., 2006). Most of the impairment-based studies used single-case designs (Bier et al., 2008 this issue; Finocchiaro et al., 2006; Frattali, 2004; Funnell, 1995; Graham, Patterson, Pratt, & Hodges, 1999; Heredia, Sage, Ralph, & Berthier, 2008 this issue; Jokel, Rochon, & Leonard, 2002; McNeil et al., 1995; Rapp & Glucroft, 2008 this issue; Schneider et al., 1996), with two studies replicating the same therapy across more than one participant (Jokel, Cupit, Rochon, & Leonard, 2008 this issue; Louis et al., 2001).

**IMPAIRMENT-DIRECTED BEHAVIOURAL INTERVENTIONS**

The pattern of treatment-specific effects across impairment-directed behavioural interventions is unmistakeable. Despite the range of participants, interventions used, and therapy designs, all participants (except one man, discussed somewhat briefly by Graham et al., 1999; Graham, Patterson, Pratt, & Hodges, 2001) improved on treated items compared to baseline and/or untreated items immediately post-treatment. However, generalisation to untreated items or situations was more restricted. We will now examine the studies in more detail.

**Therapy design**

All the impairment-directed studies reported took at least some steps to ensure that change subsequent to intervention could be attributed to the intervention, rather than to some uncontrolled factor. The simplest form of experimental control was to include two sets of items (treatment and control sets) matched on factors thought to influence treatment, in an ABA design. Performance was initially measured on both sets, then one set was treated over some period, with performance measured again on both sets at the end of treatment (e.g., Funnell, 1995; Heredia et al., 2008 this issue).

The most common experimental control involved a cross-over or multiple baselines across behaviours or sets design (Frattali, 2004; Graham et al., 1999; Jokel et al., 2008 this issue; Jokel et al., 2002; Jokel, Rochon, & Leonard, 2006; Laurence, Manning, & Croot, 2002; McNeil et al., 1995; Schneider et al., 1996), in which treatment commenced at different times on different behaviours (e.g., use of gestures
to indicate future versus past tense in Schneider et al.) or on different sets of items (e.g., nouns versus verbs in Frattali, 2004). This design typically also involves several measures of the treatment targets prior to intervention, which are then used to identify the trajectory of change prior to intervention.

Rigorous intervention design is essential because ultimately the quality of evidence that the therapy was effective depends on the quality of the experimental control. Highly informative single-case and single-case series designs can be implemented in a clinical setting with careful planning (Wilson, 1987, 1997), and there are excellent options for statistical analysis of clinically based single-case studies (Gorman & Allison, 1996). Rapp and Glucroft (2008 this issue) provide an excellent discussion of issues regarding the design of treatment studies for people with neurodegenerative disease. Following treatment there may be improvement above baseline levels of accuracy. If this occurs it is considered to be evidence for the effectiveness of treatment, just as it is in the non-progressive population. However, in those individuals with progressive language impairments stable baseline performance may not be observed, instead there may be a decline in performance before treatment. Given the likelihood of slow decline without treatment, an outcome of no change, or a slowing of deterioration can also indicate that the therapy has had an effect. If treated and untreated items have similar pre-treatment accuracy (and hence difficulty), and following therapy treated items are more accurate than untreated items, then the treatment has had an effect. This is the case even if the treated items are less accurate than they were at baseline. If treated and untreated items are equally accurate following treatment (but below baseline levels) the results are more difficult to interpret. There could be no effects of treatment, or the treatment may have had a benefit that has generalised completely (treated and untreated items all benefit). While an analysis comparing the rate of decline before and during treatment may help clarify the situation, the evidence in this situation remains equivocal.

As therapy may result in slowing of deterioration, rather than a noticeable improvement, it is vital that the person with progressive language impairments and their communication partners understand this possibility. Similarly, it is important to ensure that clients and communication partners understand that treatment will not reverse the progression of disease (McNeil & Duffy, 2001).

One limitation of a number of the impairment-directed studies reviewed is that they report effects over a very small number of items (e.g., Bier et al., 2008 this issue, had only 4 words per condition; Funnell, 1995, had 6; and Rapp & Glucroft, 2008 this issue, had 10 per condition). This makes the results vulnerable to item-specific effects, and lacking in statistical power to discriminate “real” change from variability of performance. However, this aspect of therapy design typically reflects a compromise between an ideal study design allowing high statistical power and the clinical realities of limited clinical time and participant-related constraints. For example, the choice to use small numbers of items in the Bier et al. and Rapp and Glucroft studies was determined by the time it took the participant to become fatigued. In such cases, a direct replication of the therapy effects with different items and/or participant(s) would strengthen the conclusions of the study. Studies using larger numbers of items (e.g., approximately 60 items in Jokel et al., 2002, 2006) are more persuasive with regard to positive treatment effects.

The studies we reviewed varied in mode of therapy delivery, duration of individual sessions, duration of the overall intervention, frequency of therapy activities, and whether or not the participant carried out home practice. Studies also
varied on the extent to which and how they evaluated treatment effects, generalisation, and maintenance. All these aspects of therapy design await systematic investigation across numbers of participants with progressive language impairments.

What were the characteristics of the participants?

As shown in Appendix Table 1, the participants in the studies surveyed included both men and women (although more women), of a range of ages (49–77 years), at differing times since the onset of symptoms. All showed abnormal brain imaging for left temporal regions (structure and/or function), with a range of other speech and language regions affected. Some participants showed relative sparing of memory-related medial temporal structures but others did not.

Participants were reported under three syndrome categories: semantic dementia (eight clients), nonfluent progressive aphasia (five clients), and primary progressive aphasia (five clients). However, within these syndrome categories participants had heterogeneous presentations. For example, those described as having nonfluent progressive aphasia presented with speech and language symptoms including slow speech (P1 in Jokel et al., 2008 this issue), hesitant speech (P2 in Jokel et al., 2008 this issue), and grammatical impairments, pronunciation errors, and auditory phonetic/phonological processing deficits (Louis et al., 2001). As discussed below, the extent of semantic impairment varied for the people with semantic dementia. Similarly, although three of the five participants reported under the label of primary progressive aphasia (participant CD in Laurence et al., 2002; Rapp & Glucroft, 2008 this issue; Schneider et al., 1996) met the Mesulam (2001) criteria for this diagnosis, they had diverse symptoms. These included nonfluent progressive aphasia and slow speech (Schneider et al.), prominent early writing difficulties and accompanying naming, calculation, constructional, and attentional deficits (Rapp & Glucroft, 2008 this issue), and prominent anomia with fluent aphasia, subtle pragmatic changes, and semantic and pronunciation errors (participant CD in Laurence et al., 2002). The remaining two had a history of isolated language impairment and prominent anomia for less than 2 years.

Memory and naming abilities

The studies reported a wide range of neuropsychological and language assessments, but there were no tests in common across all studies that would allow direct comparison of all participants on any area of neuropsychological or language function. Most, however, reported participants’ performance on at least one measure of delayed recall, on the three-picture version of the Pyramids and Palm Trees test (a nonverbal test of semantic associations, Howard & Patterson, 1992), and on the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983). Where these latter measures are not reported, Appendix Table 1 gives results from other tests assessing similar abilities.

Only five participants were impaired on measures of delayed recall (Bier et al., 2008 this issue; Frattali, 2004; Heredia et al., 2008 this issue; Snowden & Neary, 2002) and all of these individuals had a diagnosis of semantic dementia. Poor delayed recall of recently presented material may be associated with poorer generalisation performance (see below under Generalisation).
Approximately half the participants for whom a semantic memory measure was reported were impaired on this measure (8 out of 14; of whom 7 had a diagnosis of semantic dementia and 1 of primary progressive aphasia). However, all but two of these (Snowden & Neary, 2002) were performing above chance levels. Some of the early treatment studies had suggested that perhaps participants with relatively well-preserved semantic processing were most likely to show improved naming following lexical retrieval therapy (Laurence et al., 2002). For example, in the study of Graham et al. (1999), DM relearned members of many semantic categories, while AM, who carried out somewhat similar learning activities, did not. One of the differences between these two men was their score on the three-picture version of the Pyramids and Palm Trees Test (Howard & Patterson, 1992). DM scored 49/52, just inside the normal range, whereas AM (scoring 39/52) was quite impaired relative to controls. Scores in the normal range were also reported for GP (McNeil et al., 1995), and AC and CD (Laurence et al.), and the woman reported by Schneider et al. (1996). However, subsequent studies reported participants whose semantic memory scores were impaired but who nevertheless showed an immediate treatment effect. It now appears, therefore, that AM’s comparatively low semantic memory scores do not explain the absence of treatment effect in his case. This may instead be due to an ineffective rehearsal strategy based on the initial letters of words without semantic elaboration (Graham et al., 1999).

With regard to naming performance, all but one (Schneider et al., 1996) of the 15 for whom data are provided were impaired on the reported naming test, with three at floor level (Frattali, 2004; Snowden & Neary, 2002). The participants reported by McNeil et al. (1995) and Rapp and Glucroft (2008 this issue) had relatively mild spoken naming impairments (scoring 46/60 and 48/60 respectively on the Boston Naming Test)—potentially one of the reasons spoken noun naming was not targeted for intervention in these studies. Thus, just as is the case in the literature with non-progressive language impairments, individuals with a variety of degrees of naming and semantic impairment can benefit from impairment-directed interventions targeting word retrieval. However, participants’ knowledge of specific words and concepts does seem to be relevant for selection of therapy targets (for further details, see below under Maintenance).

The characteristics of people with progressive language impairments who have participated in published impairment-directed therapies suggest that a wide range of individuals may be suitable, as long as they are able to understand the tasks required of them. Some individuals may require certain practical support (McNeil & Duffy, 2001) in terms of assistance in getting to therapy, or setting up or being motivated to carry out home practice activities. In some cases therapists may be requested to implement an impairment-directed therapy because of consumer preferences (as with participant CD in Laurence et al., 2002), or may suggest modifications to the way a client is already practising words using a self-devised strategy, as happened with DM (Graham et al., 1999). More precise information about participant factors that influence treatment, generalisation, and maintenance effects is yet to be established.

Selection of impairments targeted and therapy activities

In the above studies the impairments targeted were in each case either the most prominent, or one of the most prominent, of participants’ impairments. This is true even in the “proof of concept” studies (Rapp & Glucroft, 2008 this issue) that
primarily aimed to find out whether therapy had any effect in clients with progressive language impairments. A high proportion of investigations targeted word retrieval: two-thirds of the interventions targeted spoken noun retrieval, with other studies targeting written naming, and adjective and verb retrieval. A smaller number of studies targeted other impairments: knowledge of semantic attributes, use of tense markers (gestural and verbal), and auditory-phonological processing.

The focus on word retrieval is unsurprising. Word retrieval is always one of the earliest symptoms noticed by people with progressive language impairments (Mesulam, 2001), and has a marked impact on communication. In addition, in non-progressive aphasia, interventions for word retrieval impairments have been relatively well investigated compared with other language-impairment therapies (for review, see Laine & Martin, 2006; Nickels, 2002; Nickels & Best, 1996a, 1996b; Raymer & Gonzalez Rothi, 2002). Nevertheless, even given the small number of studies so far, there is a range of interventions directed to a range of progressive language impairments. This supports McNeil and Duffy’s (2001) claim that impairment-directed therapies developed for use in non-progressive aphasia can be readily utilised in progressive language impairments.

Decisions about whether to use an impairment-directed intervention depend on collaborative decisions by the speech pathologist, the client, and their communication partners (e.g., Jokel et al., 2006). Decisions are guided by the social and functional usefulness of the particular communicative activities that are affected by that impairment (see also below under Generalisation and Maintenance). For example, Bier et al. (2008 this issue) chose to target TBo’s knowledge of semantic attributes of objects as well as object names, on the basis that she would find it more useful to know core facts about objects rather than their names. They give the example that it might be more useful for someone with semantic dementia to know you can eat an apple than to know that it is called “apple”.

Once the decision is made to treat a particular impairment, selection of therapy activities and items is influenced by the clinician’s hypotheses about the impairment. These often include hypotheses about the level and type of cognitive impairment within a cognitive theory of language word production (e.g., Kay, Lesser, & Coltheart, 1992; Nickels, 2000). Rapp and Glucroft (2008 this issue) explicitly used a cognitive model of spelling to guide the assessments and therapy tasks selected for the participant in their study. Different levels of impairment are generally hypothesised to require different approaches to treatment (Hillis & Caramazza, 1994). For example, a word retrieval impairment that is underpinned by a semantic impairment may be most effectively treated by a task focusing on semantic distinctions between words (Hillis, 1991; Nickels, 2002). In contrast, poor word retrieval in the context of relatively preserved semantics but impaired retrieval of phonological forms from the lexicon is best treated using tasks that provide both semantic and phonological activation (e.g., word–picture matching; repetition of the word in the presence of the picture; Nickels, 2002). In addition to hypotheses about the level of impairment, a clinician may form hypotheses about the nature of the impairment. Representations may be hypothesised to be lost from long-term memory, or to be degraded, or to remain intact while access to those representations is impaired. In practice, distinguishing between these different types of impairment is extremely difficult (see Howard, 1995, for discussion) as severely impaired access may be virtually indistinguishable from loss of representations (but for further discussion see below under How do the behavioural therapies work?).
Finally, the structuring and frequency of therapy activities, as well as selection of words, language structures, or concepts to target in therapy, also depend on the clinician’s theory about how the therapies work: about how previous knowledge is reinstated and/or knowledge is learned or relearned. These issues are also discussed below under How do the behavioural therapies work?

Effects of treatment

As we noted above, all studies found that treated items improved compared to baseline and/or untreated items immediately post-treatment, except for one individual with semantic dementia (AM), discussed by Graham et al. (1999, 2001). Although these results are extremely positive and suggest that impairment-directed therapy with clients with a wide range of progressive language impairments can yield treatment gains, caution is required. First, it is possible that a publication bias has distorted the true proportion of cases in which improvement is likely to be seen (i.e., perhaps only studies that found a treatment effect have been published). Second, even if there is no publication bias, and treatment effects are positive for the majority of participants and interventions, decisions about whether impairment-directed therapies should be incorporated into speech pathology provision for individual clients must take account of the much-less-impressive effects found for generalisation and maintenance.

Generalisation

While treated items showed significant benefits from treatment, the effect of treatment on untreated items was far less impressive across studies. None of the seven people with semantic dementia who participated in word retrieval therapies, and for whom generalisation was evaluated, showed any improvement in naming of untreated items. This is entirely consistent with the literature on non-progressive aphasia, where effects of treatment for word retrieval are generally restricted to treated items (Nickels, 2002). One study, that of McNeil et al. (1995) with a man with a 9-month history of progressive anomia, used a word retrieval treatment (a cueing hierarchy for adjective retrieval) and did find some generalisation to untreated stimuli (adjectives, verbs, and prepositions). This is not without precedent and has generally been attributed to individuals using the cueing hierarchy to self-cue when word retrieval fails (Coelho, McHugh, & Boyle, 2000; Nickels & Best, 1996b). However, despite the generalisation to untreated items, McNeil et al. found no generalisation to connected speech or a range of standardised speech and language tasks.

Within treated items, some studies examined whether there was generalisation from the stimuli used in treatment to other stimuli designed to elicit the same items. One participant, CR, was able to name the treated items when they were presented in a different order or on differently coloured and formatted materials (Snowden & Neary, 2002), which has implications for the nature of the learning that took place. Similarly, CUB could name different exemplars of the items to those presented in training (Heredia et al., 2008 this issue), although in this latter case the more typical the exemplar (and perhaps therefore the more visually similar it was to the treated exemplar, e.g., two different pictures of a whole banana compared with a picture showing sliced banana), the more successful she was. CUB’s husband also reported
generalisation of therapy items to her spontaneous speech, a factor worth noting because it may account for CUB's atypically high maintenance of learning after 6 months without deliberate practice. Future studies could fruitfully address the extent to which improvements in word retrieval, as measured by single word retrieval, generalise to the use of these stimuli in communication. There is some evidence in the literature on non-progressive aphasia, that this can occur (Herbert, Best, Hickin, Howard, & Osborne, 2003). Given that this is critical for the functional utility of word retrieval interventions, it is vital that this should not be overlooked.

At present, therefore, evidence for generalisation from treatments aimed at word retrieval is extremely limited, for both progressive and non-progressive language impairments. For this reason, the stimuli treated must be highly functional items for the client (e.g., names of family and friends, emotions), chosen in collaboration with the client and their family/friends. Similarly, the client should be made aware that generalisation may not be a realistic objective of therapy (Bier et al., 2008 this issue), and that over time only a small number of words may be retained, with eventual loss of these also occurring with disease progression.

Only one study reported improvement across a range of untrained language tasks (Louis et al., 2001), for all three participants, but this was a brief report with few data provided and only the briefest information about how these measures were obtained. Interpretation of the generalisation effect in this study is unfortunately further limited because a measure of experimental control for the treatment effect was only reported for one of the three participants, while the actual treatment effect was reported for all three participants combined. Thus the data in the report do not adequately demonstrate that participants’ improvements on untrained tasks were related to improvements on the intervention. However, the limitations of this report aside, two other studies with greater experimental control and stronger data also found some evidence of generalisation; those of McNeil et al. (1995, described above) and Schneider et al. (1996). The woman described by Schneider generalised use of verbal and gestural future tense markers and verbal past tense markers to untrained sentences in the therapy task. Subsequent to therapy, she also produced a higher proportion of sentences with the trained structure in a narrative task than she had produced before therapy.

The studies of Louis et al. (2001) and Schneider et al. (1996) were similar in that both involved teaching techniques that could be directly applicable to language in many contexts. For example, the study of Louis et al. focused on receptive processing of formant information in spoken language comprehension, and the gestures taught by Schneider et al. were appropriate to mark future or past tense for any verbs. In contrast, the studies focusing on word retrieval typically focused on facilitating retrieval of individual lexical items (or relearning the association between the concept and/or picture and the phonological form). Hence, the type of therapy directly relates to the generalisation expected (and found). The word retrieval study that showed generalisation (McNeil et al., 1995) may also have involved a strategy that could be generalised across stimuli (most likely in addition to a specific facilitatory effect for untreated items).

A more speculative observation at this point in time is that four of the five people reported to show generalisation to untreated items (Louis et al., 2001; Schneider et al., 1996) had the type of articulation deficits that Josephs et al. (2006) suggested are likely to be apraxia of speech (although described in different qualitative terms by the different authors). Further, while the fifth participant who showed generalisation
(McNeil et al., 1995) did not have apraxia of speech, he did have an early and rapidly progressing spastic dysarthria, as evidenced by mild hypernasality, strained-strangled speech, and slow diadochokinetic rate. Prominent articulation deficits (e.g., apraxia of speech, voice changes) are likely to arise from abnormal brain function in different regions of the brain from those most affected in semantic dementia. It remains to be seen whether this different distribution of neuropathology, typically associated with episodic memory difficulties in the semantic dementia but not in the former group, also contributes to the different patterns of generalisation seen across the two groups.

Finally, it must be noted that there is a certain ambiguity in evaluating generalisation in neurodegenerative disease. There is always the possibility that items to which there is no apparent generalisation may actually have deteriorated more quickly without treatment (Rapp & Glucroft, 2008 this issue; Schneider et al., 1996).

**Maintenance**

Across the impairment-directed studies reviewed, treatment gains declined substantially for almost all participants over a period varying from 2 months (Participant CD, Laurence et al., 2002) to somewhere between 6 months and 1 year (Jokel et al., 2008 this issue; Rapp & Glucroft, 2008 this issue) following cessation of treatment activities. This occurred regardless of clinical syndrome or intervention type. The exceptions were CUB (Heredia et al., 2008 this issue) who retained 82% of treated items at 6 months, and CR (Snowden & Neary, 2002) who scored 65% correct on naming approximately 8 months after treatment ceased.

Heredia et al. (2008 this issue) discuss a number of possible reasons for CUB’s superior performance, including rapid learning of items she had been unable to name (she reached ceiling on 28/28 items within 3 days of commencing therapy) with consequent errorless rehearsal of these items for almost a month afterwards, discussed further below in relation to learning mechanisms. CUB also generalised use of treated items to spontaneous speech, suggesting at least some ongoing comprehension and rehearsal of them. Participant CR’s comparatively good maintenance was attributed to the concurrent learning of item names and definitions that were personally meaningful to her (linked to their location in her environment and her experience with them, e.g., “This is a duck. It’s like the one in your conservatory. You see them on the pond when you go to the park”; Snowden & Neary, 2002, p. 1722). A number of other studies also suggest a role for a “meaningfulness” factor in maintenance of therapy gains by people with semantic dementia. AK (Jokel et al., 2006) showed a stronger treatment effect in naming, and better maintenance of items she could still comprehend but not name, than items she could neither comprehend nor name. Bier et al. (2008 this issue) found better initial generation of general attributes than specific attributes by TBo, but better maintenance of specific attributes than general attributes. People with semantic dementia typically lose more specific knowledge about objects first, with more general knowledge preserved longer (Hodges, Graham, & Patterson, 1995), thus the specific attributes TBo was able to generate potentially reflected a greater level of comprehension than did the general attributes, which may explain why they were maintained longer.

Snowden and Griffiths (2000) therefore advocate that therapy for people with semantic dementia should take place in their own surroundings and using their own
belongings, not in a clinic using line drawings or photographs of objects. Use of a client’s own objects avoids some of the difficulties a person with semantic dementia is likely to face in generalising from one exemplar to another, and the familiar surroundings provide meaningful context which will potentially support maintenance of knowledge. For the same reasons, Snowden and Griffiths suggest that carers should be educated about the value of brand loyalty when shopping (so that the appearance of household products remains constant when they are replaced), of storing objects in the same place at home, and of carrying out routine activities, such as shopping, in well-known places. As well as enabling people with semantic dementia to carry out activities of daily living more easily, there are safety implications in trying to ensure that items and surroundings are maximally understandable. Snowden and Griffiths recount as a warning an episode in which one woman with semantic dementia mistakenly used bleach, rather than bath bubbles, when preparing a bath for herself.

Given the consistent finding of loss of therapy gains following cessation of treatment activities, one obvious clinical decision would be to plan for the client to simply continue rehearsing the treated words and being exposed to them in day-to-day language use, until the disease progresses to a point where they are no longer maintained even with rehearsal. Such a plan would again highlight the need to target highly functional, personally meaningful words, and to probe maintenance of knowledge on an ongoing basis. In practice, the set of words most likely to be functionally useful and the set of words most likely to be maintained tend to overlap, because personal relevance, frequent exposure to items in everyday life (including use in conversation), and level of existing semantic knowledge for target items are all hypothesised to be related (Heredia et al., 2008 this issue; Snowden & Neary, 2002). In clients with progressive language impairments, if continuing rehearsal is the best way for a participant to maintain highly functional items, studies should not aim to evaluate maintenance without rehearsal without extremely strong ethical justification.

Motivation to practice and personality change

The potential need for ongoing rehearsal raises a final important consideration when planning impairment-directed therapy. If therapy relies on the client carrying out homework tasks, the client needs to be sufficiently motivated to complete these tasks. However, DM (Graham et al., 1999), CD (Laurence et al., 2002), and the man reported by Northen et al. (1990) showed noticeable distress at rehearsing treatment words/carrying out impairment-based therapy in the face of ongoing decline in their abilities. Graham et al. (1999, p. 377) describe DM’s therapy as a “two-edged sword”, writing that “although this perpetual homework had a beneficial effect on DM’s anomia, it seems to have had the opposite effect on his psychological well-being”. Further, both AK (Jokel et al., 2006) and CUB (Heredia et al., 2008 this issue) were noted to be obsessed about relearning previously known words, although distress per se is not mentioned in these reports. The possibility of psycho-emotional distress must be discussed with clients and carers in advance and monitored during the course of treatment. It is difficult to know in these cases to what extent distress and obsessive practice may be associated with grief and anxiety in the face of neurodegenerative disease, or with personality change caused by pathology in frontal brain regions. Personality changes are often reported in association with progressive
language impairments, with estimates of 46–75% of clients becoming depressed, apathetic, or developing behavioural fixations (Chow, Miller, Boone, Mishkin, & Cummings, 2002; Kertesz, McMonagle, Blair, Davidson, & Munoz, 2005). Personality changes, and grief and insight, together with premorbid personality and motivational factors, will all influence a client’s response to therapy, so must be taken into account in treatment planning. Psychiatric assessment is a central part of diagnosis in some clinical settings (Hodges, Berrios, & Breen, 2000), and referral for psychiatric and/or counselling services must remain an option for client and carers with ongoing review.

How do these behavioural therapies work?

A number of the impairment-directed studies discuss the mechanisms by which the therapy is hypothesised to work. These discussions take into account not just theories about the structure and processing characteristics of the language system, but theories of memory function and learning.

Before we discuss specific accounts of the mechanisms for improvement, there is an interesting contrast between studies that focus on progressive versus non-progressive language impairments. In both sets of studies there is a substantial focus on word retrieval. However, the terms that are used to describe the effects of the same treatment techniques tend to be rather different. In the non-progressive literature the treatment is generally described as “improving word retrieval” (e.g., Hickin, Best, Herbert, Howard, & Osborne, 2002) or “priming” retrieval (e.g., Martin & Laine, 2000) of names (but see Fillingham, Sage, & Lambon Ralph, 2005, 2006). In contrast, in the progressive literature there is a tendency instead to talk about “learning” or “relearning” of names (Bier et al., 2008 this issue; Graham et al., 1999; Heredia et al., 2008 this issue; Snowden & Neary, 2002). This use of different terminology seems to imply that in non-progressive aphasia word forms remain “intact but unavailable”, whereas in progressive language impairments word forms are “lost” and have to be relearned. As discussed above, this is a difficult distinction to determine experimentally, and to date, for the majority of individuals who have been treated, no attempt has been made to determine whether there is loss or inaccessibility of word form representations. Thus, we would urge caution in over-interpreting these terms in the absence of a clear debate regarding whether one mechanism can account for the data better than the other.

Below we outline four of the mechanisms discussed in the studies reviewed here: a temporary memory system supported by medial temporal lobe structures, the role of multiple retrievals, errorless learning mechanisms, and the linking of target items to existing knowledge.

Graham et al. (1999, 2001), and Heredia et al. (2008 this issue) suggest that the ability to retrieve object names after therapy is due to the relearning of these names within a temporary memory system. This system is supported by medial temporal lobe structures that are relatively undamaged by the neurodegenerative disease compared with longer-term stores supported by other cortical regions that may have been almost decimated by disease. Failure to consolidate learning in long-term stores distributed in neocortex explains why treatment gains are not maintained after therapy activities stop. According to Heredia et al., these long-term stores are also hypothesised to support generalisation, explaining why limited generalisation occurs, at least in participants with semantic dementia.
Several studies discuss the hypothesised benefit of multiple retrievals of information in therapy. Bier et al. (2008 this issue) found no difference between spaced retrieval training (in which the interval between retrievals is carefully controlled and expands as the participant responds correctly) and a technique they called “simple repetition”, which utilised the same number of retrievals without the expanding retrieval interval. In both techniques, retrieval of information relevant to different items was interleaved, a procedure thought to support learning of multiple associations (McClelland, McNaughton, & O’Rielly, 1995). According to McClelland et al., interleaving the presentation of target items with that of similar items over many presentations allows information about individual items to be learned gradually. By contrast, participants DM and CUB were provided with a fairly rigid rehearsal order that did not involve interleaving (Graham et al., 1999; Heredia et al., 2008 this issue). The effect of number of retrievals (and the interaction between this and interleaving of target items) needs to be systematically investigated to establish whether there is further support for this hypothesis.

Errorless learning (discouraging the participant from guessing and thus making errors) is another factor thought to play a role in the success of spaced retrieval training in dementia of the Alzheimer type and vascular dementia (Brush & Camp, 1998), and in relearning of forgotten associations in dementia of the Alzheimer type (Clare, Wilson, Breen, & Hodges, 1999; Wilson, Baddeley, Evans, & Shiel, 1994). Frattali (2004) and Snowden and Neary (2002) deliberately utilised errorless learning in their intervention design. Graham et al. (1999) noted that the rigidly ordered rehearsal that DM carried out involved errorless learning. In addition, Heredia et al. (2008 this issue) reported that CUB was getting the names of the therapy items correct by day 3, then continued to rehearse them in what was effectively an errorless paradigm for almost a month. In fact, only two of the eight impairment-directed interventions reported for people with semantic dementia did not utilise errorless learning, both of which had very small sets of items (Bier et al., 2008 this issue: four items in spaced retrieval, four in simple repetition; Funnell, 1995: six vegetable names), requiring replication to rule out item-specific effects.

Although it initially appeared that people with semantic dementia had well-preserved episodic memory (Hodges, Patterson, Oxbury, & Funnell, 1992), subsequent research demonstrated impaired performance on episodic memory tests (Simons & Graham, 2000), and atrophy to medial temporal structures associated with episodic memory (Mummery et al., 1999). Lambon Ralph and Fillingham (2007), propose that there are two learning systems. One involves the strengthening of associations between frequently paired stimuli and responses (a simple Hebbian learning system), and another modulates this Hebbian learning depending on whether a response proves to be correct or incorrect. If a person’s ability to use this second modulatory system is impaired (because they cannot detect or encode errors), they are left only with the simple (unmodulated) Hebbian system that learns erroneous responses just as effectively as correct responses. In this situation, errorless learning is beneficial because it ensures that only associations between correct stimuli and responses are strengthened. The hypothesis that errorless learning supports (re)learning in semantic dementia requires systematic investigation.

Whether there is also a benefit in errorless learning for people with syndromes of non-fluent or primary progressive aphasia awaits investigation, because all the interventions to date with people diagnosed with these syndromes (Appendix
Table 2) have involved errorful learning (in which the client has the opportunity to
give a response, even if it is incorrect). Interestingly, however, for non-progressive
aphasia, treatment for word retrieval is not found to be more effective when errorless
learning is utilised (Fillingham et al., 2005, 2006). This could be because these
participants have sufficiently preserved memory and executive skills to support the
modulatory learning system proposed by Lambon Ralph and Fillingham (2007).

A final mechanism that potentially contributes to participants’ improvement on
naming tasks relates to linking target items to existing knowledge. Meaningfulness of
items to be learned, degree of item-specific knowledge retained by the participant,
and use of therapy activities that activate a wide semantic network including the
network supporting semantic features and lexical knowledge, are all factors
associated with item-specific improvement in the naming studies surveyed. Once
again, perhaps this relates to the distinction between relearning and re-accessing
representations. Those items for which there is retained knowledge (e.g., richer
semantics) are more likely to be retained themselves, and require facilitation of
access, rather than requiring relearning of semantic information and/or relearning of
the links between meaning and form. In the non-progressive literature there is a
preponderance of explanations for improvement in word retrieval that rely on
improving access through “priming” (strengthening links between representations or
increasing resting levels of activation of representations themselves, e.g., Biedermann
& Nickels, 2008; Howard, 2000). It is therefore plausible that improvement that is
dependent on improving access is easier to attain than improvement requiring
complete or substantial relearning, although both mechanisms may account for
improved word retrieval.

CUB’s results are particularly interesting because she showed longer maintenance
than the other participants with semantic dementia. Heredia et al. (2008 this issue)
consider whether any of the following factors might provide an explanation: amount
of therapy, number of items treated, therapy method and items selected, severity of
semantic memory impairment, degree of brain atrophy, and differences in
occupation or education prior to illness, but they conclude that the most likely
factor is generalisation to spontaneous speech (see above). The effects of all of these
factors await systematic investigation. Information about CUB’s generalisation of
treated items to spontaneous speech relied on her husband’s report; in fact, none of
the studies we reviewed included a measure of language use in an everyday setting:
clearly this is important for future research.

The theories discussed above to some extent make different predictions about
what manipulations in therapy activities will be most beneficial, thus future clinical
research studies need to tease these apart. Until the results are in, the best chance of
clinical success will be to combine as many of these factors as possible and
practicable, as in the example of the intervention with CUB.

**IMPAIRMENT-DIRECTED PHARMACOLOGICAL INTERVENTIONS**

To date there are only two papers reporting pharmacological interventions aimed to
improve language abilities in primary progressive aphasia, neither of which show
strong promise. McNeil et al. (1995), as discussed above, targeted adjective retrieval
in a single-case design that alternated a combined pharmacological–behavioural
intervention with behavioural intervention only and withdrawal phases.
Dextroamphetamine was administered on the basis of research showing greater
recovery of function following cerebral lesion with dextroamphetamine plus behavioural treatment than behavioural treatment alone (McNeil et al., 1995). There was no strong evidence for a benefit of the combined treatment above that of the behavioural treatment alone, although the researchers noted a trend for the combined treatment to be associated with increased generalisation.

The second study (Reed et al., 2004) trialled bromocriptine with six people with primary progressive aphasia diagnosed according to the criteria of Mesulam (2001). Bromocriptine, a dopamine agonist used in treating Parkinson’s disease, was selected because it has been associated with improved speech production in nonfluent aphasia caused by left hemisphere stroke (Raymer et al., 2001). The study had a double-blind, placebo-controlled, crossover design, and measured language abilities on naming, word fluency, and a narrative task. Most of the measures did not suggest any benefit from the bromocriptine. However, mean length of utterance was found to be greater in the bromocriptine condition compared with placebo, but nonetheless declined for the group over the 5-month course of the study. The researchers concluded that bromocriptine might have slightly slowed the participants’ language deterioration but that the effect was limited.

**IMPAIRMENT-DIRECTED rTMS INTERVENTION**

A small number of recent studies have begun to demonstrate benefits of repetitive transcranial magnetic stimulation (rTMS) on language function in people with acquired language impairments (Cotelli et al., 2006; Naeser et al., 2005). This technique has also shown some benefit for a 60-year-old man diagnosed with primary progressive aphasia according to the Mesulam (2001) criteria (Finocchiaro et al., 2006). Repetitive TMS is hypothesised to inhibit or excite neuronal function, depending on whether the stimulation is low versus high frequency, respectively. In the study of Finocchiaro et al., high-frequency rTMS was applied to a left inferior frontal region hypothesised to be involved in verb processing, in five sessions held over consecutive days. The man with primary progressive aphasia improved on a verb-naming task but not on two memory span tasks, with the benefits lasting approximately 46 days after the first round of TMS, and approximately 31 days after the second round of TMS. A “sham” treatment served as a control, in which, unknown to the participant, the magnet was angled away from the scalp, but all other aspects of treatment delivery were unchanged. Although the man improved substantially relative to baseline after the two rTMS treatments, there was no change after the sham treatment. These technologies are very much in their infancy, and all applications of therapeutic rTMS are at present highly experimental and limited in scope—but we should follow the progress of rTMS as a potential alternative or addition to behavioural approaches for impairment-based intervention.

**ACTIVITY/PARTICIPATION-DIRECTED INTERVENTION STUDIES**

There are 11 papers that describe activity/participation-directed interventions, education, and carer training in some detail (summarised in Appendix Table 3). A number of other reports (e.g., Béland & Ska, 1992; Hart, Beach, & Taylor, 1997; Thompson, Ballard, Tait, Weintraub, & Mesulam, 1997) mention speech therapy (including use of a communication book, articulatory-kinematic training, and pacing and contrastive stress exercises), but with insufficient detail to be included in
Appendix Table 3. Some of the interventions reviewed combined impairment-directed approaches with activity/participation-directed activities (Holland et al., 1985; L. L. Murray, 1998; Northen et al., 1990). One was a group therapy (four participants) in which the clinicians developed aphasia-friendly formats for viewing and discussing half-hour episodes of a television series dealing with the life events of contemporary Australians (Cartwright & Elliott, 2008 this issue).

The literature suggests that written and pictorial information (communication books, boards and cards, encouraging the person and/or their communication partner to write, or draw pictures or symbols), gesture and sign (pantomime and communicative gesture, Amerind and American Sign Language), and other augmentative and alternative (AAC) technologies (text-to-speech machines, emergency wrist devices) may all have the potential to help maximise the communication potential of a person with progressive language impairments. Most of the studies also mention speech pathology sessions dedicated to carer training or counselling, and/or group sessions in which the person with progressive language impairments has the opportunity to practise communication techniques, interact socially with others with aphasia or dementia, and receive feedback and peer support. Discourse analysis has been used to identify functional communication needs and sources of communication breakdown as the basis for training the person with progressive language impairments and their communication partner, and to measure the success of conversation training techniques (L. L. Murray, 1998; Wong, Anand, Chapman, Rackley, & Zientz, 2008 this issue).

The activity/participation-directed studies that we reviewed highlight the importance of tailoring the intervention to the individual client and context. Cress and King (1999) reported a number of benefits of the AAC approaches introduced with participant CE. Using a receptive communication board, CE was able to comprehend around 70% of the necessary routine messages his family wished to communicate. Significantly, CE still responded to messages communicated via the board 1 year later, when he no longer demonstrated comprehension of verbal material. CE also “tended to express more complex messages and more communicative turns with a wider variety of listeners when using communication boards than without them” (Cress & King, 1999, p. 256), and continued to use his expressive communication board at 1-year follow-up to manage conversation topics. However, a noteworthy feature of the intervention was the speech pathologist’s close social relationship with CE, which allowed her to spend intensive time with CE and his family over a month-long period to introduce the AAC techniques. She was also able to facilitate CE’s use of these techniques outside the home, at bowling, restaurants, parties and musical events. Similarly, Rogers et al. (2000) describe how FA, who had given up playing poker with close friends, was able to begin playing again with assistance from his wife and friends, using communication cards (e.g., types of poker game, bidding rules) also individually tailored for use with those partners in that situation. Again similarly, Cartwright and Elliot (2008 this issue) prepared in advance a glossary of key words that would be used in each group television-watching session, and a summary of the plot of each episode that the group watched together. In the latter two studies, enjoyment of the activity by all communication participants (person with aphasia, spouse, friends, speech pathologist) was reported as one major outcome of the intervention.

Rogers and colleagues (Rogers & Alarcon, 1998; Rogers et al., 2000) have written most extensively on activity/participation approaches to intervention in progressive
language impairments, and give a number of case examples. They stress the importance of three principles: (1) implement goals in anticipation of decline, (2) use dyad-focussed therapy, and (3) use augmentative techniques that rely on preserved abilities to increase participation. Rogers et al. provide suggestions for assessing impairments, activities, and participation in progressive language impairments, and discuss potentially useful content for inclusion in communication books and ways to address acceptance issues. They underscore the suggestion above that transfer of AAC approaches outside the clinic is maximised when the speech pathologist dedicates regular time to facilitating typical interactions between the client and his or her regular communication partners using AAC.

At this point in time the case reports and anecdotes summarised in Appendix Table 3 indicate that activity/participation approaches with people with progressive language impairments show great promise. To date, however, there are almost no published formal evaluations of these interventions that would allow future activity/participation therapy planning for these clients to be evidence based. Future activity/participation interventions must be designed to allow clinicians/researchers to show that apparent improvements in a client’s activity/participation levels are reliable, and can be unambiguously attributed to the intervention(s) implemented (e.g., using control groups/participants or control sets of items or behaviours). Wilson (1987, 1997) provides an excellent summary of research designs that can be readily used in clinical settings. In this context, the aphasia-friendly television-viewing intervention designed by Cartwright and Elliot (2008 this issue) is innovative and exemplary in describing the intervention methods and measures in detail, and in comparing participants’ performance before and after intervention within participant, within group, and to a control group, on several discourse measures.

Future activity/participation-directed interventions in this population must also meet the challenge of assessing whether clients show improvements in activity/participation levels outside the clinic. This is equally an issue in non-progressive aphasia (Turner & Whitworth, 2006) and quantitative and qualitative measures of functional communicative success developed for people with aphasia (e.g., Armstrong & Mortensen, 2006), dementia (e.g., Lubinski & Orange, 2000), and traumatic brain injury (Togher, McDonald, Code, & Grant, 2004) could all be adapted for use with people with progressive language impairment. Work is just beginning in this area with people with progressive language impairment. Rogers et al. (2000) discuss potential assessments for use with this population, and the discourse intervention with Bobby V (Wong et al., 2008 this issue) makes an important contribution by specifying clearly a framework within which communicative effectiveness can be evaluated.

Finally, many of the reports summarised in Appendix Table 3 also refer to additional benefits of communication intervention for the person with progressive language impairments and their communication partners. These include improved quality of life, a decreased sense of isolation (Thompson & Johnson, 2006), increased feeling of personal control (McNeil & Duffy, 2001; Robinson, 2001; Thompson & Johnson, 2006), enhanced sense of purpose and goal in life (McNeil & Duffy, 2001), participant and speech pathologist enjoyment (Cartwright & Elliott, 2008 this issue), and neuroprotective benefits of cognitive stimulation provided by therapy (Cartwright & Elliott, 2008 this issue). All of these potential positive outcomes of therapy await formal evaluation (e.g., use of standardised scales, questionnaires, and tailor-made outcome measures) and documentation in peer-reviewed articles.
ISSUES RELEVANT TO ALL INTERVENTIONS

Involvement of spouse/carer

A spouse/carer may have a number of roles in maximising health, social, and communication opportunities and speech therapy outcomes of the person with progressive language impairments, perhaps even taking on a case manager-type role (Robinson, 2001). Given the potential burden of caring for someone with progressive disease (J. Murray, Schneider, Banerjee, & Mann, 1999), education programmes for people with dementia typically include components that focus on caring for the carer (Northen et al., 1990; Ripich, 1994). The Primary Progressive Aphasia Program at the Cognitive Neurology and Alzheimer’s Disease Center at North Western University, Chicago, offers educational lectures and small group discussion with input from medical staff, psychologists, and social workers, followed up by monthly carer support meetings (Cognitive Neurology & Alzheimer’s Disease Center, 2006). As one of the healthcare team, the speech pathologist has a role in monitoring a carer’s coping abilities and referring to extra services (general practitioner, social work, occupational therapy, psychology, respite services, support groups etc.) as required.

A spouse/carer in good health is an invaluable informant about the functional relevance of potential therapy activities, and about key items to treat in impairment-directed interventions or include in a communication book. If changes in insight and personality occur with disease progression, the spouse/carer is likely to be the most sensitive observer of these, and one of the people most affected by them. A number of anecdotes reporting successful activity/participation-directed interventions (Appendix Table 3) highlight the role of the spouse/carer in transferring communication strategies from the clinical setting to everyday life. As mentioned above, Rogers et al. (2000) suggest that speech pathologists dedicate at least 5–10 minutes per client per week in the clinic to observing and facilitating communication between clients and their communication partners using augmentative and alternative communication tools, to assist transfer of these skills outside the clinic. However, primary carers (spouse, adult children) may see the speech pathology appointment time as an opportunity for respite (Cathleen Taylor & Rachel Miles Kingma, personal communication, 19 June 2007), so it may be important to discuss with them the potential benefits of their participation in speech pathology sessions.

Participants may be less likely to carry out home practice activities if they live alone or do not otherwise have a spouse or carer who is committed and able to help them with home therapy activities. In two contrasting examples, Rapp and Glucroft (2008 this issue) noted that CB, who was living alone, did not consistently complete her homework tasks, whereas CUB (Heredia et al., 2008 this issue) asked her husband for names and information about therapy items, and this may have assisted transfer of these items to her spontaneous speech. Older clients and their carers may also need assistance, instruction, and reassurance in learning to use computer-delivered therapy activities or other technologies such as those reported by Heredia et al. (2008 this issue), Jokel et al. (2008 this issue), and Cartwright and Elliot (2008 this issue). For example, to address these sorts of difficulties in their aphasia-friendly television viewing intervention, Cartwright and Elliot provided a glossary, and spoken and pantomimed explanations of terminology associated with using a DVD player.
Participant characteristics and co-occurring deficits

Future reports also need to document clients’ non-language characteristics in detail to establish which client factors are associated with particular therapy outcomes. The suitability of particular interventions (impairment-directed as well as activity/participation-directed) for particular clients will depend on the client’s and partners’ cognitive abilities, on personality and motivational factors, and on attitudes and access to particular communication modes and opportunities (Turner & Whitworth, 2006).

Individuals with co-occurring behaviour, motor, and/or mood disturbances will also require referral to neurology, psychiatry, psychology, physiotherapy, and occupational therapy services (Robinson, 2001). Additional behavioural and pharmacological interventions may be required, as well as interventions directed at enhancing the participants’ mobility and safety in their environment, including assessment of swallowing (Gregory & Lough, 2001; Rahman, Sahakian, Hodges, Rogers, & Robbins, 1999; Robinson, 2001; Royal College of Speech & Language Therapists, 2005).

Replication

Finally, replication for all the therapies reported is extremely important. First, a successful replication reduces the possibility that an effect reported in a single-case study (almost all the studies reviewed here) is peculiar to that study (clinician and/or participant effects). A limitation of the single-case design used in most of the studies that we reviewed is the impossibility of knowing to what extent the observed results can be generalised to other clients, whereas a series of replications would begin to indicate the client group(s) for whom a positive outcome from therapy might be expected. The currently limited replication of impairment-directed interventions with people with progressive language impairments therefore urges caution in assuming results for one participant will be observed in another. For example, in the Jokel et al. study in this issue, both participants received the same intervention, but P2 took far longer than P1 to reach criterion and the treatment effect and maintenance were not as strong. In the study of McNeil et al. (1995), a cueing hierarchy for adjective retrieval in an antonym task did not yield the same results as a similarly designed synonym task.

CONCLUDING COMMENTS

Taylor et al. (2008 this issue) highlight the need for a speech pathology management pathway for people with progressive language impairments. As part of a multidisciplinary dementia health and social care service, such a pathway will need to be eclectic, considering the relative merits of impairment-directed, activity/participation-directed, and combined approaches on an individual basis for each client. There is a pressing need for client and carer education, peer support, and for counselling about communication, disruptive behaviour, personality change, carer burden, and grief and end-of-life issues. Therapy options already in use in the context of stroke-related aphasia are appropriate for use with people with progressive language impairments. These should be selected on the basis of comprehensive initial assessment and ongoing review, and proactively implemented
in anticipation of decline, rather than solely matched to the client’s current needs (Rogers & Alarcon, 1998; Rogers et al., 2000).

In sum, it is clear that treatment for progressive language impairments can be of great benefit. However, intervention needs to encompass the individual’s communicative needs in everyday contexts, and to anticipate future needs. We hope that clinicians embrace this challenge and that services for this population become more widespread. Similarly, we hope that the clinical research base will increase, enabling future clinical decisions about therapy design to be both theoretically and empirically motivated.

REFERENCES


### APPENDIX TABLE 1
Summary of demographic characteristics and clinical presentations of participants in the reviewed impairment-directed studies

<table>
<thead>
<tr>
<th>Studies grouped by clinical syndrome</th>
<th>Participant</th>
<th>Age, Sex</th>
<th>Prominent symptoms</th>
<th>Time post-onset</th>
<th>Brain imaging</th>
<th>Neuropsychology</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Delayed recall</td>
</tr>
<tr>
<td>Semantic dementia</td>
<td>Mrs P</td>
<td>63, F</td>
<td>semantic dementia(^1)</td>
<td>3 yrs</td>
<td>CT: L temporal, SPECT: frontotemporal</td>
<td>Rey figure recall ✓, BPVT: at 5yrs;6 mo level (x)</td>
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<tr>
<td>Funnell, 1995</td>
<td></td>
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<td></td>
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<tr>
<td>Graham et al., 1999, 2001</td>
<td>DM</td>
<td>61, M</td>
<td>anomia, impaired comprehension; progressed to semantic dementia(^1)</td>
<td>4 yrs</td>
<td>MRI: L temporal</td>
<td>P&amp;PT: 49/52 pics ✓</td>
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<td></td>
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<tr>
<td>Jokel et al., 2002, 2006</td>
<td>AK</td>
<td>63, F</td>
<td>semantic dementia(^1)</td>
<td>7 yrs</td>
<td>SPECT: L temporal, MRI: bitemporal L &gt; R, L ventral frontal</td>
<td>nonverbal cognition ✓, PALPA synonym judgement: 56/60(^6) 3, P&amp;PT 37/52 pics(^3) (x)</td>
</tr>
<tr>
<td>Snowden &amp; Neary 2002</td>
<td>KB</td>
<td>64, F</td>
<td>semantic dementia(^1) especially visual knowledge</td>
<td>3 yrs</td>
<td>SPECT, MRI: bitemporal, R &gt; L, including medial temporal structures</td>
<td>RMT 30/50 words (x), 25/50 faces (x)</td>
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</tr>
<tr>
<td></td>
<td>CR</td>
<td>57, F</td>
<td>semantic dementia(^1)</td>
<td>3 yrs</td>
<td>SPECT, MRI: bitemporal, L &gt; R, relative preservation of hippocampus</td>
<td>RMT: 29/50 words (x), 29/50 faces (x)</td>
</tr>
<tr>
<td>Frattali, 2004</td>
<td>66, M</td>
<td></td>
<td>anomia, word-by-word reading, impaired auditory comprehension, word recognition &amp; auditory memory, behaviour change</td>
<td>n/a</td>
<td>PET: L fronto-temporal &amp; ant. insula, MRI: inf medial L temporal, mostly spared hippocampus</td>
<td>verbal memory on DRS and WMS ✓</td>
</tr>
</tbody>
</table>

\(^1\) Indicates relative preservation of structures.

\(^2\) Indicates the battery used.

\(^3\) Indicates the subtest used.

\(^6\) Indicates the number of items correct.

\(^7\) Indicates the number of items incorrect.
<table>
<thead>
<tr>
<th>Studies grouped by clinical syndrome</th>
<th>Participant</th>
<th>Age, Sex</th>
<th>Prominent symptoms</th>
<th>Time post-onset</th>
<th>Brain imaging</th>
<th>Neuropsychology</th>
<th>Delayed recall</th>
<th>Semantic processing</th>
<th>Naming</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bier et al., 2008 this issue</td>
<td>TBo</td>
<td>70, F</td>
<td>semantic dementia</td>
<td>5 yrs</td>
<td>SPECT, MRI: L sylvian fissure, L frontal</td>
<td>all tests ✓, except hard form of Doors Test ✓,</td>
<td>P&amp;PT: 36/52 words ✓, 37/52 pics ✓</td>
<td>76/127 ✓</td>
<td></td>
</tr>
<tr>
<td>Heredia et al., 2008 this issue</td>
<td>CUB</td>
<td>53, F</td>
<td>semantic dementia</td>
<td>2 yrs</td>
<td>MRI: bitemporal, focal on L, mild on R</td>
<td>visual memory ✓</td>
<td>P&amp;PT: 31/52 words ✓, 33/52 pics ✓</td>
<td>EPLA: 13/60 ✓</td>
<td></td>
</tr>
<tr>
<td>Nonfluent progressive aphasia</td>
<td>Louis et al., 2001</td>
<td></td>
<td>all 3: nonfluent aphasia (agrammatic, pronunciation errors, anomia), impaired auditory processing and auditory working memory</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>Jokel et al., 2008 this issue</td>
<td>P1</td>
<td>58, F</td>
<td>nonfluent aphasia, slow and anomic speech</td>
<td>3-4 yrs</td>
<td>SPECT: L temporal</td>
<td>story recall ✓</td>
<td>P&amp;PT: 51/52 pics ✓</td>
<td>BNT: 26/60 ✓</td>
<td></td>
</tr>
<tr>
<td>P2</td>
<td>75, F</td>
<td></td>
<td>nonfluent aphasia, hesitant and anomic speech</td>
<td>n/a</td>
<td>SPECT: bitemporal</td>
<td>story recall ✓</td>
<td>P&amp;PT: 50/52 pics ✓</td>
<td>BNT: 29/60 ✓</td>
<td></td>
</tr>
<tr>
<td>Primary progressive aphasia</td>
<td>McNeil et al., 1995</td>
<td>GP</td>
<td>anomia, mild spastic dysarthria, mild aphasia</td>
<td>9 mo</td>
<td>SPECT: L temporo-parietal, MRI: L temporal</td>
<td>short passage story recall ✓, complex figure story recall ✓</td>
<td>P&amp;PT: 49/52 pics ✓</td>
<td>BNT: 46/60 ✓</td>
<td></td>
</tr>
<tr>
<td>Schneider et al., 1996</td>
<td>62, F</td>
<td></td>
<td>nonfluent aphasia (agrammatic, pronunciation errors, anomia), slow reading &amp; spelling</td>
<td>2.5 yrs</td>
<td>SPECT, MRI: L perisylvian</td>
<td>WMS ✓</td>
<td>category naming ✓</td>
<td>BNT: 59/60 ✓</td>
<td></td>
</tr>
</tbody>
</table>
### APPENDIX TABLE 1
(Continued.)

<table>
<thead>
<tr>
<th>Studies grouped by clinical syndrome</th>
<th>Participant</th>
<th>Age, Sex</th>
<th>Prominent symptoms</th>
<th>Time post-onset</th>
<th>Brain imaging</th>
<th>Neuropsychology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laurence et al., 2002</td>
<td>CD</td>
<td>49, F</td>
<td>fluent aphasia, anomia, reading and writing difficulties, semantic &amp; pronunciation errors</td>
<td>30 mo</td>
<td>SPECT, MRI: L temporal</td>
<td>Delayed recall: words &amp; complex figure</td>
</tr>
<tr>
<td></td>
<td>AC</td>
<td>63, F</td>
<td>mild anomia, fluent aphasia, semantic errors, pragmatic changes</td>
<td>1 yr</td>
<td>MRI: L temporal</td>
<td>n/a</td>
</tr>
<tr>
<td>Rapp &amp; Glucroft, 2008 this issue</td>
<td>CB</td>
<td>60, F</td>
<td>dysgraphia, anomia, dyscalculia, attention deficits, mild constructional deficits</td>
<td>8 yrs</td>
<td>MRI: temporoparietal, L &gt; R</td>
<td>RAVLT and Rey copy ✔</td>
</tr>
</tbody>
</table>

**Abbreviations for tests:** BNT = Boston Naming Test (Kaplan & Goodglass, 1983); BPVS = British Picture Vocabulary Scale (Dunn, Dunn & Whetton, 1982); EPLA = Evaluación del Procesamiento Lingüístico en la Afasia (Coltheart, Kay & Lesser, 1995); PALPA = Psycholinguistic Assessment of Language Processing in Aphasia (Kay, Lesser & Coltheart); P&PT = Pyramids and Palm Trees test (Howard & Patterson, 1992); RAVLT = Rey Auditory Verbal Learning Test (Rey, 1964); RMT = Recognition Memory Test (Warrington, 1984); WMS = Wechsler Memory Scale (Wechsler, 1987)

**Other abbreviations:** ant. = anterior; CT = computerised tomography; F = female; inf. = inferior; L = left; M = male; mo = months; MRI = magnetic resonance imaging; n/a = not available; PET = positron emission tomography; pics = pictures; R = right; SPECT = single photon emission computed tomography; vol. = volume; yrs = years; %ile = percentile ✔ = within normal limits/judged to be unimpaired for this participant; ✗ = judged to be impaired for this participant

**Notes:** 1 As defined by the Lund criteria (Neary, 1998); 2 Semantic Battery (Hodges & Patterson, 1995); 3 Test of Adolescent Word Finding (German, 1990 in Schneider et al., 1996); 4 Baddeley (1994); 5 Locally developed naming test using pictures from Snodgrass & Vanderwart (1980); 6 Seven months before study; 7 Five months after study; 8 Deficits potentially described by other researchers as apraxia of speech.
<table>
<thead>
<tr>
<th>Studies grouped by clinical syndrome</th>
<th>Participant</th>
<th>Impairment treated</th>
<th>Description of intervention</th>
<th>Treatment effect</th>
<th>Generalisation</th>
<th>Maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Semantic dementia</strong></td>
<td>Funnell, 1995</td>
<td>Mrs P Vegetable naming</td>
<td>1 session pairing vegetable and name 10 times each. Home practice: self-test with vegetable, written name and description; EF</td>
<td>YES: 6/6 correct</td>
<td>NO, not to other less familiar vegetables</td>
<td>1 week: 6/6; 1-2 months: 5/6; 8 months: 3/6. Practice over this period not described but vegetables not in season</td>
</tr>
<tr>
<td>Graham et al., 1999, 2001</td>
<td>DM Noun retrieval</td>
<td>Rehearsing members of semantic categories (name + picture) in home practice, 30 mins/day × 2 weeks; EL</td>
<td>YES</td>
<td>NO</td>
<td>40% at 10 weeks</td>
<td></td>
</tr>
<tr>
<td>Jokel et al., 2002, 2006</td>
<td>AK Naming</td>
<td>Picture + reading name aloud + read personally relevant description. Home practice 30 mins/day × 6 days; EL</td>
<td>YES: -N + C 18/30 (60%); -N − C 11/30 (37%)</td>
<td>NO</td>
<td>4 weeks: −N + C43%*, −N − C 23%; 6 months: −N + C 30%, −N − C 13%. *statistically reliable effect</td>
<td></td>
</tr>
<tr>
<td>Snowden &amp; Neary 2002</td>
<td>KB Naming</td>
<td>errorless pairing of picture with spoken and written name, 3 times per set on 2 occasions; EL studied picture, written name and information relevant to personal experience in 2 treatment sessions. Home practice 20 mins/day × 3 weeks; EL</td>
<td>YES for items with partial knowledge. YES (marginal) for items about which KB had no preserved knowledge</td>
<td>Not reported. KB continued to learn name of new people and medicines in everyday life</td>
<td>2 weeks: YES; 4 months: NO</td>
<td></td>
</tr>
<tr>
<td>Studies grouped by clinical syndrome</td>
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<tr>
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</tr>
<tr>
<td>Frattali, 2004</td>
<td>Noun and verb retrieval</td>
<td>Conversation about semantic features and associates of photographed items. 12 × 2 hour sessions over 3 months; EL</td>
<td>YES on 20 treated nouns and 20 treated verbs</td>
<td>NO, not to untreated nouns and verbs, nor to performance on neuropsychological tests</td>
<td>3 months: NO</td>
<td></td>
</tr>
<tr>
<td>Bier et al., 2008 this issue</td>
<td>TBo</td>
<td>Naming &amp; learning semantic attributes</td>
<td>8 items, formal-semantic therapy using cueing hierarchy and semantic feedback. 3 sessions spaced retrieval compared with 3 sessions simple repetition; EF</td>
<td>YES: picture names YES: specific attributes NO: general attributes. Spaced retrieval equal to simple repetition.</td>
<td>NO, not on naming to within-category items or another naming test or letter fluency task</td>
<td>Naming: NO, loss of treatment gains over 5-week period. Better maintenance of specific attributes</td>
</tr>
<tr>
<td>Heredia et al., 2008 this issue</td>
<td>CUB</td>
<td>Naming</td>
<td>computer-based presentation of picture alone then picture + read aloud written name, home practice daily for 1 month; EL</td>
<td>YES: 28/28 treated items</td>
<td>YES: 92% correct on other exemplars of treated items. YES: to spontaneous speech (anecdotal report)</td>
<td>1 month: YES 27/28; 6 months: YES 23/28 with no deliberate practice</td>
</tr>
</tbody>
</table>

**Nonfluent progressive aphasia**

<p>| Louis et al., 2001 3 Ps auditory-phonological processing | Tapping syllables, phoneme discrimination and segmentation of tokens slowed by 166%. 15-20 mins/day × 42 days; EF | YES in data pooled across 3 Ps | YES: Improved performance for at least 1 of the 3 Ps on several French BDEA subtests. 1 reduced phoneme errors, 1 improved on reading and repetition. | One P was given a phase of treatment with tokens at normal speed; performance declined |</p>
<table>
<thead>
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<th>Generalisation</th>
<th>Maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P1</td>
<td>Naming</td>
<td>MossTalk Words® Cued Naming  of 3 lists of 14 words, 1 hour 3×/week for 4 weeks per list; EF</td>
<td>YES: all 3 lists</td>
<td>NO, not to Philadelphia Naming Test</td>
<td>1 month: YES; 6 months: NO; all 3 lists</td>
</tr>
<tr>
<td></td>
<td>P2</td>
<td>Naming</td>
<td>MossTalk Words® Cued Naming, 3 lists of 14-15 words, 1 hour 2×/week for 12 weeks per list; EF</td>
<td>YES: all 3 lists</td>
<td>NO, not to Philadelphia Naming Test</td>
<td>1 month: YES 2 lists, NO 1 list; 6 months: NO 2 lists, MARGINAL 1 list</td>
</tr>
<tr>
<td><strong>Primary progressive aphasia</strong></td>
<td>GP</td>
<td>Adjective retrieval</td>
<td>Cueing hierarchy + pharmacological treatment (dextroamphetamine); 31×2-hour sessions over 5 months; EF</td>
<td>YES, but pharmacological treatment + cueing hierarchy no clear advantage over cueing hierarchy alone</td>
<td>YES, some to untrained adjectives, and to untrained verbs and prepositions. NO, not to connected speech</td>
<td>declined at approximately 3 months without rehearsal</td>
</tr>
<tr>
<td></td>
<td>CD</td>
<td>Naming</td>
<td>Verbal+gesture training of target structures in sentences×18 sessions. Home practice with pictures of gestures; EF</td>
<td>YES</td>
<td>YES, to untrained verbs &amp; untrained sentences. NO, not to untrained tenses. Increased use of trained sentence form in story retell.</td>
<td>3 months: gesture maintained but verbal forms not maintained</td>
</tr>
<tr>
<td>McNeil et al., 1995</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Schneider et al., 1996</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Laurence et al., 2002</td>
<td></td>
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</tbody>
</table>
### APPENDIX TABLE 2
(Continued.)

<table>
<thead>
<tr>
<th>Studies grouped by clinical syndrome</th>
<th>Participant</th>
<th>Impairment</th>
<th>Description of intervention</th>
<th>Treatment effect</th>
<th>Generalisation</th>
<th>Maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laurence et al., 2002 (Cont.)</td>
<td>AC</td>
<td>Naming</td>
<td>Written word-picture</td>
<td>YES, on written and spoken naming for high and low frequency words</td>
<td>NO, not to picture description task</td>
<td>11 weeks: High frequency spoken naming still 100%; 12 weeks: low frequency spoken naming at baseline levels; 14 weeks: high frequency written naming at 42%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>matching with semantic</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>distractors. 40 high</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>frequency items × 6 weeks,</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>low frequency items × 4</td>
<td></td>
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<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>weeks. Daily home practice;</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>EF</td>
<td></td>
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</tr>
<tr>
<td>Rapp &amp; Glucroft, 2008 this issue</td>
<td>CB</td>
<td>Dysgraphia</td>
<td>4 sets: Trained (spell-</td>
<td>YES; 10% gain in Trained, Repeated and Homework stable, decline on Control</td>
<td>NO</td>
<td>6 months: YES, Trained, Repeated &amp; Homework better than Control; 12 months: NO</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>study-spell) biweekly 3</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>hour sessions over 15</td>
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<td></td>
<td></td>
<td></td>
<td>weeks, Repeated: spelled</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>in each session; Homework</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>(copy and self-test)</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>sporadic; Control (Not</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Trained); EH</td>
<td></td>
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</tr>
</tbody>
</table>

**EF** = errorful learning; **EL** = errorless learning; **mins** = minutes; **−N−C** not named and not comprehended; **−N+C** not named but partially comprehended; **P(s)** = participant(s); **BDEA** = Boston Diagnostic Aphasia Examination (French adaptation; Mazaux & Orgogozo, 1971)
## APPENDIX TABLE 3  
Summary of activity/participation-directed interventions in the reviewed studies

<table>
<thead>
<tr>
<th>Studies grouped by clinical syndrome</th>
<th>Participant</th>
<th>Age, Sex</th>
<th>Clinical presentation</th>
<th>Time post-onset</th>
<th>Duration of therapy</th>
<th>Brain imaging</th>
<th>Neuropsychology</th>
<th>Therapy components</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary progressive aphasia</strong></td>
<td>CE</td>
<td>60, M</td>
<td>BDAE impaired on commands, complex ideation, repetition, sentence comprehension, naming, verbal fluency; used gesture and vocalisations to supplement/substitute for speech</td>
<td>7 years</td>
<td>1 mo AAC development, 1 year follow-up</td>
<td>MRI: focal L temporal</td>
<td>Visual processing and learning, arithmetic, sensorimotor skills, complex attention, orientation</td>
<td>In context of close social relationship therapist developed communication boards, and spent 1 hour with CE 3-4 times/week, 3-6 hours with CE and family &amp; 1-3 occasions/week outside the home e.g. bowling</td>
</tr>
<tr>
<td>Cress &amp; King (1999)</td>
<td>MC</td>
<td>59, F</td>
<td>Mod-severe non-fluent aphasia on WAB, high level of fillers, repetitions &amp; restarts, used self-generated communicative strategies e.g. oral spelling, written information in purse, questions to repair breakdown</td>
<td>6 years</td>
<td>Approx 3 sessions</td>
<td>MRI: L temporal</td>
<td>Reading comprehension better than auditory comprehension; delayed verbal memory</td>
<td>(i) Words written when she couldn’t understand, (ii) communication book with maps, family tree, house layout, pets, relationships (iii) names and pictures of food items on file cards filed in categories, taken out for shopping list and matched to items in supermarket</td>
</tr>
<tr>
<td>Murray (1998)</td>
<td>DD</td>
<td>65, F</td>
<td>Stuttering, slurred, agrammatic speech, word-finding difficulties, auditory &amp; reading comprehension impairment</td>
<td>4 years</td>
<td>2.5 years</td>
<td>PET: L fronto-parietal</td>
<td>Complex figure delayed copy WNL; Auditory word recognition 57/60; BNT 19/20</td>
<td>(i) Activities involving listening to, reading, speaking and writing sentences and paragraphs; (ii) &quot;back to the drawing board&quot; communicative use of pictures; (iii) functional communication including spouse training, support group and Dyna Vox AAC device</td>
</tr>
</tbody>
</table>
### Studies grouped by clinical syndrome

<table>
<thead>
<tr>
<th>Participant</th>
<th>Age, Sex</th>
<th>Clinical presentation</th>
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<th>Neuropsychology</th>
<th>Therapy components</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pattee, Von Berg &amp; Ghezzi (2006)</td>
<td>57, F</td>
<td>PPA with AOS: &lt; 20% of speech intelligible, preserved receptive language</td>
<td>n/a</td>
<td>9 weeks</td>
<td>PET: L temporal</td>
<td>Preserved non-language cognition implied</td>
<td>Elicited agent-action-object sentences and Wh-questions using TTS and ASL. Utterances in both modes were similarly informative, but P preferred ASL because it felt more &quot;normal&quot;. Accepted AAC electronic wrist device for emergencies.</td>
</tr>
<tr>
<td>Rogers, Alarcon &amp; King (2000)</td>
<td>FA M</td>
<td>Fluent aphasia</td>
<td>4 years</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td>AAC (communication notebook, gestures, strategies for specific situations e.g. poker including wife’s assistance)</td>
</tr>
<tr>
<td>Thompson &amp; Johnson (2006)</td>
<td>RP M, 58</td>
<td>Difficulty understanding words and expressing his thoughts, independent ADLs, social withdrawal outside family, WAB: anomic aphasia, word-finding difficulties, semantic paraphasias</td>
<td>4 years</td>
<td>n/a</td>
<td>PET: bilateral temporal (L &gt; R), frontal and parietal</td>
<td>non-verbal cognition WNL</td>
<td>Communicative gesture, self-cueing strategies and ways of compensating for word-finding difficulties, emergency communication card, education in communication strategies for family</td>
</tr>
</tbody>
</table>

### Nonfluent primary progressive aphasia

<table>
<thead>
<tr>
<th>Participant</th>
<th>Age, Sex</th>
<th>Clinical presentation</th>
<th>Time post-onset</th>
<th>Therapy components</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rogers &amp; Alarcon (1998)</td>
<td>OD 71, M</td>
<td>Anomia, AOS, telegraphic speech, short phrases</td>
<td>2 years</td>
<td>4 years</td>
</tr>
<tr>
<td>Studies grouped by clinical syndrome</td>
<td>Participant</td>
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<td>Clinical presentation</td>
<td>Time post-onset</td>
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<tr>
<td>-------------------------------------</td>
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<td>----------------</td>
</tr>
<tr>
<td>Rogers, Alarcon &amp; King (2000)</td>
<td>NA M</td>
<td></td>
<td>Nonvocal communicator, required AAC for comprehension of even simple utterances</td>
<td>10 years</td>
</tr>
<tr>
<td>Pick's disease</td>
<td>Mr E M</td>
<td>68, M</td>
<td>Slow, deliberate speech, long pauses, anomia, phonological paraphasias</td>
<td>0 years</td>
</tr>
<tr>
<td>Holland, McBurney, Moossy, &amp; Reinmuth (1985)</td>
<td></td>
<td></td>
<td></td>
<td>4 years (4 yrs post)</td>
</tr>
<tr>
<td>Semantic dementia</td>
<td>Bobby V M</td>
<td>61, M</td>
<td>Anomia, fluent empty speech, semantic paraphasias. Phonology, syntax, repetition unimpaired</td>
<td>n/a</td>
</tr>
<tr>
<td>Slowly progressive aphasia without generalised dementia</td>
<td>M, 64 M</td>
<td></td>
<td>Effortful speech with fluent islands, effortful syllable repetition, right-sided tremor and rigidity</td>
<td>18 mo</td>
</tr>
<tr>
<td>Northen, Hopcutt &amp; Griffiths (1990); Goulding et al. (1990)</td>
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(Continued.)

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<th>Neuropsychology</th>
<th>Therapy components</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progressive language impairment</td>
<td>Cartwright P1 F, 65; &amp; Elliot P2 F, 59; (2008 this issue) P3 F, 66; P4 M, 62</td>
<td>Heterogeneous language, presentations: 3 with nonfluent speech, one semantic dementia</td>
<td>10 weeks n/a</td>
<td>n/a</td>
<td>CLQT language severity score: 2 severe, 1 moderate, 1 mild</td>
<td>Group programme with aphasia-friendly TV viewing, supported discussion of episode content and themes relevant to participants</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations for tests: BDAE Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1983); CLQT Cognitive Linguistic Quick Test (Helm-Estabrooks, 2001); MTDDA Minnesota Test for Differential Diagnosis of Aphasia (Scheull & Sefer, 1973); WAB Western Aphasia Battery (Kertesz, 1979); WAIS Wechsler Adult Intelligence Scale (Wechsler, 1981); WMT Wechsler Memory Scale (Wechsler, 1987)

Other abbreviations: AAC = augmentative and alternative communication; ASL = Americal Sign Language; EEG: electroencephalogram; F = female; L = left; M = male; mo = months; MRI = magnetic resonance imaging; n/a = not available; P = participant; PPA = primary progressive aphasia; perf = performance; PET = positron emission tomography; pics = pictures; R = right; SPECT = single photon emission computed tomography; TTS = Text to Speech; WNL = within normal limits; yr(s) = year(s); %ile = percentile