Perspectives of the ASHA Special Interest Groups
SIG 2, Vol. 2(Part 3), 2017, Copyright © 2017 American Speech-Language-Hearing Association

Clinical Implications of Neurocognitive Control Deficits in Bilingual Adults With Aphasia

Tanya Dash

Ana Inés Ansaldo

Centre de recherche de l'Institut Universitaire de Gériatrie de Montréal,
Montreal, Quebec, Canada
École d'orthophonie et d'audiologie, Faculté de médecine, Université de Montréal,
Montreal, Quebec, Canada

Disclosures
Financial: Tanya Dash has no relevant financial interests to disclose. Ana Inés Ansaldo has no relevant financial interests to disclose.
Nonfinancial: Tanya Dash has no relevant nonfinancial interests to disclose. Ana Inés Ansaldo has no relevant nonfinancial interests to disclose.

Abstract

The purpose of the paper is to review the literature on the neurocognitive control in bilingual aphasia and extrapolate research findings into clinical guidelines. Neurocognitive control, as well as bilingualism, are multifaceted phenomena whose complex interaction is disrupted by stroke. Bilingualism is an added factor of complexity to aphasia assessment and rehabilitation. Rehabilitation specialists are more aware of the need to understand language and nonverbal cognitive abilities, for a better treatment outcome (Ansaldo, Saidi, Ruiz, 2010; Green, 2005; Helm-Estabrooks, 2002). Consequently, assessment and management of neurocognitive skills in bilingual aphasia are gradually gaining momentum. Applying principles from language-cognitive control interactions to the rehabilitation of bilingual populations with aphasia appears to be a valuable intervention strategy for this population.

Bilingual Aphasia: Journey So Far

In the last two decades, there has been an increase in studies on the neurocognitive control and bilingualism (Abutalebi & Green, 2008; Abutalebi, Della Rosa, Tettamanti, Green, & Cappa, 2009; Bialystok, Abutalebi, Bak, Burke, & Kroll, 2016), in healthy as well as bilingual adults with aphasia (BAA). Bilingual aphasia research has focused on single case and group studies, addressing a wide variety of issues. These include the clinical characteristics of bilingual aphasia (Paradis 2001), crosslinguistic effects of interventions (Ansaldo & Saidi, 2014), recovery patterns of language and cognitive skills (Marangolo, Rizzi, Peran, Piras, & Sabatini, 2009), the validation of assessment and management strategies (Ansaldo, Saidi, & Ruiz, 2010; Kohnert, 2004), and the identification of the pre-requisite skills to achieve efficient bilingual rehabilitation in BAA (Gray & Kiran, 2013, 2016).

Researchers have addressed a series of questions and the answers have evolved overtime with the advent of new knowledge in the field. Among these questions: Are neurocognitive control deficits present in all cases of bilingual aphasia? How can clinicians assess neurocognitive control deficits? Which tools are available to assess these deficits? How do neurocognitive deficits affect the bilingual aphasia profile? What clinical signs characterize neurocognitive control deficits in BAA? What is an efficient way of approaching these deficits in therapy? How can speech-language pathologists (SLPs) take into account these deficits while choosing the stimuli and therapy approach to treat BAA? These questions are of great interest and clinical relevance in the field of speech-language pathology since it has become very clear that the understanding of bilingual aphasia

cannot be achieved by the mere extrapolation of data from monolingual aphasia. Until recently, most of the studies on bilingual aphasia focused on the impact of brain damage on language representation and function, with a less direct focus on bilingual aphasia rehabilitation. In particular, the issue of the clinical implications of neurocognitive control deficits in bilingual aphasia still remains "terra incognita".

Hence, given that bilingualism is a global phenomenon characterized by a constant increase in the number of bi/multilingual individuals around the world (de Bot, 1992), there is as well an increase in bilingual clinical populations with speech and language disorders, such as BAA. There is a great need to develop adapted assessment and intervention tools for these populations; tools and interventions that tap on the disrupted processes, and provide valid solutions for the management of BAA. Most of the literature has focused on common and distinct features between monolingual and bilingual aphasia, in terms of clinical signs, and recovery patterns. Conversely, there is little research on the status of other cognitive functions than language, and limited information about neurocognitive deficits in bilingual adults with aphasia, and their appraisal. The purpose of this paper is to discuss the clinical implications of neurocognitive control deficits observed in bilingual aphasia, and how this understanding can substantiate the bilingual assessment and rehabilitation methods in this widely increasing clinical population.

The Concept of Neurocognitive Control and Its Implications in Bilingual Aphasia

The concept of neurocognitive control refers to the neural and cognitive mechanisms that sustain the ability to adapt and flexibly change behavior to novel conditions. Neurocognitive control consists of an array of cognitive processes, including attentional control, cognitive flexibility, abstraction, reasoning, concept formation, the use of strategies, problem solving, initiation, sequencing, monitoring, inhibition, impulse control, goal setting, and planning (Cahana-Amitay & Albert, 2015). It is also referred to as "executive function," "executive control," "central executive," or "cognitive control". In the context of this paper, we will use the term neurocognitive control to refer to the definition above.

Whether in a bilingual or unilingual mode of communication, the two languages of the bilingual person are in constant competition. This competition is managed by the cognitive control system, which selects or inhibits either language, depending on the constraints of the communicative situation. Hence, at the lexical, morphosyntactic, or discourse levels, and at the comprehension or production levels of oral and written language, neurocognitive control abilities play a key role in the achievement of efficient communication (Cahana-Amitay & Albert, 2015).

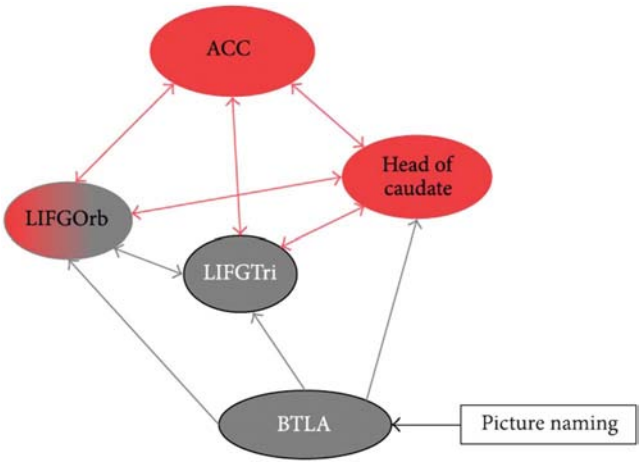
Deficits in neurocognitive control following stroke have been largely reported in persons with aphasia, and they have been related to a wide variety of aphasia signs and symptoms, including paraphasia (Ardila, Bernal, & Rosselli, 2016), anomia (Ansaldo et al., 2010), discourse comprehension (Novick, Trueswell, & Thompson-Schill, 2005), language production (Biegler, Crowther, & Martin, 2008), apraxia of speech (Ardila et al., 2016), and conversational discourse (Penn, Frankel, Watermeyer, & Russell, 2010). More specific to BAA, some neurocognitive advantages in bilinguals, as compared to monolinguals, have been reported (Penn et al., 2010). A thorough understanding of neurocognitive control abilities and deficits in the context of bilingual aphasia, will contribute to designing intervention approaches adapted to this population. To do so, it is important that SLPs are sensitized to the links between language and cognitive control, mechanisms, and how these two can be affected in the context of stroke (Abutalebi & Green, 2008; Radman et al., 2016).

A Model-Based Approach to Understand Neurocognitive Control-Language Interactions

Abutalebi's neurocognitive model of bilingual language switching provides a framework for the understanding of neurocognitive control deficits in bilingual aphasia (Abutalebi & Green,

2008; Abutalebi et al., 2009) while providing a neuroscience perspective that may guide clinical assessment. Thus, highlighting the importance of assessing cognitive skills in addition to language skills as well as their interaction in BAA. According to this model (Abutalebi et al., 2009) lexical selection is supported both by control (neurocognitive control; in red) and language (naming; in grey) processing networks, as shown in Figure 1.

Figure 1. Model of Language-Control Network (Proposed by Abutalebi et al. [2009], from Radman et al. [2016]).



Note. ACC = anterior cingulate cortex, BAs 24, 32, 33; BTLA = basal temporal language area, BAs 19 and 37; LIFGOrb = left inferior frontal gyrus pars orbitalis, BA47; LIFGTri, left inferior frontal gyrus pars triangularis, BA45.

The language network comprises semantic decoding areas (Broadmann Areas [BAs] 19, 37), a lexical production area (BA 45) and a lexical retrieval area (BA 47). As for the control network, it comprises the left inferior frontal pars orbitularis (LIFGOrb; BA 47), the head of the caudate, and the anterior cingulate cortex (ACC; BAs 24, 32, 33), all of which support conflict monitoring, and managing interference from the nontarget language. Damage to any of areas or their interconnection may result in typical clinical signs in aphasia. For example, damage to semantic decoding areas may result in semantic paraphasia, which occurs when the correspondence between the conceptual and lexical representation is disrupted. As a result, the person with aphasia produces words that are semantically related to the target within or across languages (i.e., saying “chair” instead of “sofa”) or uses a semantic alternative across the two spoken languages. The person could also select an inappropriate phonological representation, which results in a phonemic paraphasia (i.e., saying “spot” instead of “pot”). In addition, damage to these areas (BAs 37, 19, 45) may similarly lead to verbal fluency deficits with the BAA being unable to name items in a specific category. Damage to the control network results in an array of clinical signs, including pathological code-switching/-mixing, a common symptom in bilingual aphasia, and blending. While pathological code-switching consists of switching between the languages, in a single conversational context. Blending involves uncontrollable mixing of the two languages at any language level while attempting to speak in only one language. Brain damage may lead to selective language control deficits, namely impairments at the language level only, or nonlinguistic control deficits, affecting nonverbal auditory or visual stimuli processing or both linguistic and nonlinguistic control deficits, as reported in single case studies of bilingual aphasia (Dash & Kar, 2014; Green et al., 2010; Verreyt, De Letter, Hemelsoet, Santens, & Duyck, 2013).

A question of clinical interest concerns whether neurocognitive control deficits are present in all types of bilingual aphasia. What we know is that neurocognitive control deficits in BAA may show variable manifestations, ranging from very evident behavioral manifestations- such as pathological code-switching to subtle deficits at lexical, phonetic, or prosodic levels (Gray & Kiran, 2016; Green et al., 2010). The study of neurocognitive control deficits in BAA is limited and hampered by the caveats of a number of individual factors, including the age of L2 acquisition, mode of L2 acquisition, extent of use of either language, as well as language proficiency. All the previously named factors influence neurocognitive control and language deficits in the context of the assessment of bilingual aphasia. Thus, assessment of BAA can be compromised by the lack of consideration of the bilingual language experience, and neurocognitive control ability (including attention and working memory) in the interpretation of language test results. In this regard, it is to be noted that research provides important clues to be considered when working with this population. The next sections focus on a number of clinical questions, while adopting an evidence-based perspective. The reader is encouraged to keep in mind that our answers are incomplete, requiring further research to improve our understanding of these issues.

Neurocognitive Control Deficits and Bilingual Aphasia: Some Clinical Signs

Neurocognitive control deficits may be observed both in the acute and chronic stages of aphasia. The behavioral manifestations include lack of flexibility in moving from one language to the other, switching between communicative situation and difficulties in inhibiting the nontarget language, which are reflected by intrusions, blending, or mixing that are typical of pathological language switching. Specifically, it has been shown that repeated failure to inhibit the non-target language becomes a source of verbal disfluencies, aborted sentences, and disorganized discourse (see Penn et al., 2010 for more examples). Also, slow and laborious speech may suggest neurocognitive control deficits due to slowness in execution of the goal-directed behavior in language production.

Assessing Neurocognitive Control Deficits

In addition to standard language assessment strategies, assessment of neurocognitive control abilities in BAA will contribute to develop a broad diagnosis and prognosis profile of the patient. Knowledge of the patient's neurocognitive abilities will provide important information for the assessment process, including the analyses and interpretation of the BAA's interaction with the bilingual environment, the potential for recovery and cross-language transfer of language therapy effects, as well as the overall communication impairment. Language processing in bilinguals taxes the cognitive control mechanism in unique way, as bilinguals have to select the target language, inhibit the nontarget one, and be ready to switch from one to the other, depending on the requirements of the communicative situation. This calls for the assessment of neurocognitive deficits in bilinguals with aphasia. Although assessing neurocognitive control for monolingual speakers with aphasia is the best practice, it is especially important for the bilingual speakers.

In order to avoid subjective bias in neurocognitive assessment, it is necessary to adapt the testing environment by selecting tests that have a minimal load on linguistic skills. Also, choosing the language better understood by the PWA or caregiver in the context of assessment. It is also important to gather information about the premorbid language experience of the BAA (Centeno, 2005), to distinguish between premorbid proficiency and aphasia consequences. A bilingual language processing framework (Gray & Kiran, 2013) shows that the impact of pre-morbid language proficiency, language use, and history are fundamental elements for the diagnosis of bilingual aphasia. Specifically, the authors (Gray & Kiran, 2013) argue that pre-stroke language use patterns can predict lexical and semantic deficits as measured by standardized language tests. However interesting, the authors also provide caution while interpreting these results, as only a limited number of participants were examined.

Traditionally, assessment of neurocognitive control abilities relies upon neuropsychological tests, such as the Wisconsin Card Sorting Test (Heaton, Chelune, Talley, Kay, & Curtis, 1993), the Tower of Hanoi Test (Humes, Welsh, Retzlaff, & Cookson, 1997), the Tower of London Test (Shallice, 1982), and the Trail Making Test (Reitan & Wolfson, 1985), that are now sparsely used in clinical practice. However, because these tests were not developed to assess BAA, and are highly loaded on comprehension of instructions; thus, which limits their diagnostic validity and level of reliability for this population (Cahana-Amitay & Albert, 2015). However, although testing tools with high comprehension demands are not always suited to assess neurocognitive control deficits in BAA, these demands can be decreased by lifting time constraints, simplifying instructions, using gestures, and providing practice trials (Cahana-Amitay & Albert, 2015). In other words, if the clinician has experience with these tests and with bilingual aphasia, a parsimonious use of these assessment tools may provide important information about the neurocognitive profile of person with bilingual aphasia.

Two valuable tools for the assessment of cognitive-linguistic abilities in BAA are the Cognitive-Linguistic Quick Test-Plus (CLQT-Plus; Helm-Estabrooks, 2017) and the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; Randolph, 1998). Originally developed for monolingual populations with stroke, head injury and dementia, this tools can provide a general cognitive profile of the persons with aphasia. Further, neurocognitive control skills in daily life situations, such as cooking, driving, etc., can be tested with the Activity of Daily Living Profile (Dutil, Bottari, Vanier, & Gaudreault, 2005) or the Communication Effectiveness Index (Lomas et al., 1989). Finally, validated neuropsychological tests, used to assess attention-based cognitive control, maybe useful, as well. Examples of those tests include the Stroop task (MacLeod, 1991; Stroop, 1935), the Flanker task (Eriksen & Eriksen, 1974), and the negative priming task (Tipper, 1985). These tests generate important information about neurocognitive control abilities at the executive function level. However, as none of these instruments has been standardized with bilingual populations, the results should be considered with caution.

Neurocognitive Control Deficits in Bilingual Adults With Aphasia: Current and Future Directions

Bilingual aphasia rehabilitation encompasses therapeutic options, ranging from traditional symptomatic approaches focusing on the rebuilding of language skills, to compensatory approaches, focusing on an alternative modes of communication (Hallowell & Chapey, 2008; Hux, Weissling, & Wallace, 2008). More specifically to bilingual aphasia, intervention studies usually focus on language choice for therapy, cross-language transfer of therapy effects, and factors influencing recovery in the chronic phase. Furthermore, some authors have supported the idea of addressing impaired cognitive skills (i.e., attention, working memory, etc.) in addition to the language impairment. As for the clinical management of neurocognitive control deficits in bilingual aphasia, intervention traditionally aimed at selecting one of the two languages for therapy to enhance the chances of recovery (Green, 2005; Paradis, 2001). A key reason driving this approach is that, secondary to brain damage, the amount of available cognitive resources decreases. Thus, the rationale behind this approach is that limiting intervention to one language will minimize the load on neurocognitive abilities, and pathological mixing or switching (Green, 2005; Paradis, 2001). Given that brain damage causes the amount of available resources to decrease, limiting intervention to one language is expected to decrease the load on neurocognitive abilities, thus favoring recovery (Green, 2005; Paradis, 2001).

However, there is evidence that integrating code-switching in the therapy process may be beneficial to improve communication in BAA. In particular, Switch Back through Translation Therapy (SBTT) has been shown to be an efficient way to bypass anomia, access the target language, and maintain fluid communication, while preventing communication breaks (Ansaldo & Saidi, 2014). SBTT uses translation as a means of overcoming pathological code-switching. More precisely, when the person switches to the nontarget language, instead of trying to prevent the switch, the

SLP uses a cue to guide the person to translate the items from the nontarget to the target language (it can be a short cue sentence in the target language or a gesture). As translation restricts the choice in vocabulary selection, it makes it easier to access the word in the target language, while avoiding communication breakdown due to the pressure of producing a word in a specific language. Hence, SBTT provides top-down control by increasing awareness of the use of translation equivalents in the nontarget language, and by providing BAA with a strategy to overcome the impairment, by using the word that first comes to their minds, and if needed, translate that word to the target language (Ansaldo & Saidi, 2014).

Another perspective promotes combining interventions targeting general cognitive control abilities, in addition to speech-language therapy (SLT; Green et al., 2010). In this regard, the evidence suggests that therapy for nonlinguistic neurocognitive control abilities, including attention, working memory and inhibitory control, may contribute to better language recovery in BAA (Dash & Kar, 2014; Verreyt et al., 2013). Specifically, in a single-case study on a bilingual participant with severe aphasia, Kohnert (2004) applied a cognitive treatment, consisting of visual scanning, categorization, and simple arithmetic tasks. The participants showed therapy gains in both the languages. In addition, Gray and Kiran (2016) speculate that the training of nonverbal control skills, rather than language control skills should help improving language in BAA. However, this argument warrants further investigation.

A recent meta-analysis of the literature on bilingual aphasia therapy points to the importance of considering the potential for cross-language therapy effects (CLTE) by looking at similarities and differences between two languages used by the bilinguals (L1-L2; Ansaldo & Saidi; 2014). Specifically, cognates (i.e., words that share phonology and semantics across languages) show a higher potential for CLTE than noncognates (i.e., words that share semantics but not phonology across L1-L2). For cognates, a lack of inhibitory control is less harmful than when processing noncognates. The competition involved in processing noncognates requires a strong inhibition of the nontarget item. As a whole, the research reviewed suggests that the post-stroke functional status of the language-control network (Abutalebi et al., 2009, please refer to Figure 1) will have a strong impact on the recovery profile of the BAA. A description of the different recovery profiles in bilingual persons with aphasia is beyond the scope of the present paper (see Lorenzen & Murray, 2008; Paradis, 2001). However, when assessing either language of the BAA, it should be noted that neurocognitive control deficits may jeopardize observations regarding pure language or cognitive deficits, due to the difficulty in selecting or inhibiting the pertinent information at a given time.

Finally, it is also reported that neurocognitive control skills are a prerequisite for successful use of compensatory strategies (i.e., using gestures, or writing or self-corrections in wrong language). These compensatory strategies may help in facilitating overall communication. In cases of neurocognitive control deficits, therapy goals can be reframed based on research findings from the past. It should be noted that promoting a bilingual mode of communication may improve performance, and reduce stress related to the inability to inhibit a language and communication breakdown (Lorenzen & Murray, 2008).

Summary and Recommendations

There is no general rule for the assessment and intervention of neurocognitive control deficits in BAA. However, it is greatly acknowledged that the neurocognitive control-language interactions are particularly complex in a bilingual context. Thus, brain damage can have a serious impact on the delicate balance between the two of them. There is a great need for more research, to disentangle the behavioral signs and symptoms of neurocognitive control deficits in BAA. And find, the best practice in the context of speech-language clinical assessment and

interventions can be identified. There are pros and cons to the actual situation, and these can be summarized as follows:

(a) Caveats regarding the available evidence:

- Current literature is heavily influenced by the single case studies. Thus, a word of caution is needed while interpreting the information.
- Not all BAA will demonstrate similar cognitive-linguistic profile. Neurocognitive control deficits may vary as a function of individual factors regarding the type of pre-morbid bilingual language profile in a given person.
- Use of monolingual assessment tools with BAA is inappropriate, and may jeopardize neurocognitive deficits, and lead to poorly designed interventions.

(b) Recommendations for future research:

- More group studies comparing neurocognitive control and language patterns across bilinguals and monolinguals with aphasia, and by reference to healthy populations will increase the external validity of clinical entities described.
- Randomized control trials to study intervention impact in large cohorts of BAA would then follow.

(c) Recommendations for clinical assessment:

- Collecting a thorough case history on language background of the BAA is essential to determine premorbid proficiency and language use, to avoid over or underestimating the impact of aphasia.
- Assessing neurocognitive deficits in BAA should include both nonverbal and verbal tools, so that domain specific vs. domain general neurocognitive deficits can be disentangled, and considered when planning therapy.
- Recommendations for treatment:
- Take into consideration the pre-morbid and post-morbid language proficiency as well as the language-use patterns in both the languages (Gray & Kiran, 2016) for efficient chose of language of treatment according to premorbid competence.
- Target specific cognitive processes together with language intervention, for example by using cognitive treatment (Kohnert, 2004).
- Include SBTT (Ansaldo, Saidi, & Ruiz, 2010) when switching between languages can ease the communication instead of restricting the BAA to the use of one language.
- Favor a multimodal/multi-language approach, by combining speaking, writing, and reading in both languages to ease communication (Gil & Goral, 2004).

To sum up, the high neurocognitive demands that characterize bilingualism may be particularly hampered in the context of aphasia. This topic has been particularly neglected in the bilingual aphasia rehabilitation literature and research. Further research is required to better characterize these deficits. This will contribute to developing assessment and intervention procedures adapted to BAA. Until this happens, and given the exponential growth of bilingual population, clinicians need to be aware of at least two main things: first of all, it is important to acknowledge the possibility of neurocognitive control deficits in the context of BAA. Secondly, assessing this possibility in an adequate manner will provide key information to substantiate rehabilitation approaches and tools that can enhance the chances of recovery of BAA.

References

Abutalebi, J., & Green, D. W. (2008). Control mechanisms in bilingual language production: Neural evidence from language switching studies. *Language and Cognitive Processes, 23*(4), 557–582.

Abutalebi, J., Della Rosa, P. A., Tettamanti, M., Green, D. W., & Cappa, S. F. (2009). Bilingual aphasia and language control: a follow-up fMRI and intrinsic connectivity study. *Brain and Language, 109*(2), 141–156.

Ansaldo, A. I., Saidi, L. G., & Ruiz, A. (2010). Model-driven intervention in bilingual aphasia: Evidence from a case of pathological language mixing. *Aphasiology, 24*(2), 309–324.

Ansaldo, A. I., & Saidi, L. G. (2014). Aphasia therapy in the age of globalization: Cross-linguistic therapy effects in bilingual aphasia. *Behavioural Neurology, 2014,* 1–10.

Ardila, B., Bernal, B., & Rosselli, M. (2016). How localized are language brain areas? A review of Brodmann areas involvement in oral language. *Archives of Clinical Neuropsychology, 31*(1), 112–122.

Bialystok, E., Abutalebi, J., Bak, T. H., Burke, D. M., & Kroll, J. F. (2016). Aging in two languages: Implications for public health. *Ageing Research Reviews, 27,* 56–60.

Biegler, K. A., Crowther, J. E., & Martin, R. C. (2008). Consequences of an inhibition deficit for word production and comprehension: Evidence from the semantic blocking paradigm. *Cognitive Neuropsychology, 25*(4), 493–527.

Cahana-Amitay, D., & Albert, M. L. (2015). *Redefining recovery from aphasia*. Oxford University Press.

Centeno, J. G. (2005). Working with bilingual individuals with aphasia: The case of a Spanish-English bilingual client. *American speech-language-hearing-association division 14—Perspectives on Communication Disorders and Sciences in Culturally and Linguistically Diverse Populations, 12*(1), 2–7.

Dash, T., & Kar, B. R. (2014). Bilingual language control and general purpose cognitive control among individuals with bilingual aphasia: Evidence based on negative priming and flanker tasks. *Behavioural Neurology, 2014,* 1–20.

De Bot, K. (1992). A bilingual processing model: Levelt's 'speaking' model adapted. *Applied Linguistics, 13,* 1–24.

Dutil, E., Bottari, C., Vanier, M., & Gaudreault, C. (2005). ADL Profile: Description of the instrument (H. Scott & C. Bottari, Trans. 4th ed., Vol. 1). Montreal: Les Éditions Émersion.

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception and Psychophysics, 16*(1), 143–149.

Gil, M., & Goral, M. (2004). Nonparallel recovery in bilingual aphasia: Effects of language choice, language proficiency, and treatment. *International Journal of Bilingualism, 8*(2), 191–219.

Gray, T., & Kiran, S. (2013). A theoretical account of lexical and semantic naming deficits in bilingual aphasia. *Journal of Speech, Language, and Hearing Research, 56*(4), 1314–1327.

Gray, T., & Kiran, S. (2016). The relationship between language control and cognitive control in bilingual aphasia. *Bilingualism: Language and Cognition, 19*(03), 433–452.

Green, D. W. (2005). The neurocognition of recovery patterns in bilingual aphasics. In J. F. Kroll, & A. M. B. de Groot (Eds.), *Handbook of bilingualism: Psycholinguistic perspectives* (pp. 516–530). New York: Oxford University Press.

Green, D. W., Grogan, A., Crinion, J., Ali, N., Sutton, C., & Price, C. J. (2010). Language control and parallel recovery of language in individuals with aphasia. *Aphasiology, 24*(2), 188–209.

Hallowell, B. & Chapey, R. (2008). Introduction to language intervention strategies in adult aphasia. In R. Chapey (Ed.), *Language Intervention Strategies in Aphasia and Related Neurogenic Communication Disorders* (5th edition, pp. 3–19). Baltimore, Maryland: Lippincott, Williams, & Williams.

Heaton, R. K., Chelune, G. J., Talley, J. L., Kay, G. G., & Curtis, G. (1993). *Wisconsin Card Sorting Test manual: Revised and expanded*. Odessa, FL: Psychological Assessment Resources.

Helm-Estabrooks, N. (2002). Cognition and aphasia: A discussion and a study. *Journal of Communication Disorders, 35*(2), 171–186.

Helm-Estabrooks, N. (2017). Cognitive-linguistic quick test-Plus. San Antonio, TX: Pearson.

Humes, G. E., Welsh, M. C., Retzlaff, P., & Cookson, N. (1997). Towers of Hanoi and London: Reliability and validity of two executive function tasks. *Assessment, 4,* 249–257.

Hux, K., Weissling, K., & Wallace, S. (2008). Communication-based interventions: Augmented and alternative communication for people with aphasia. In R. Chapey (Ed.), *Language Intervention Strategies in Aphasia and Related Neurogenic Communication Disorders* (5th ed., pp. 814–836). Baltimore, MD: Lippincott, Williams, & Williams.

Kohnert, K. (2004). Cognitive and cognate-based treatments for bilingual aphasia: A case study. *Brain and Language, 91*(3), 294–302.

Lomas, J., Pickard, L., Bester, S., Elbard, H., Finlayson, A., & Zoghaib, C. (1989). The Communicative dffectiveness index development and psychometric evaluation of a functional communication measure for adult aphasia. *Journal of Speech and Hearing Disorders, 54*(1), 113–124.

Lorenzen, B., & Murray, L. L. (2008). Bilingual aphasia: A theoretical and clinical review. *American Journal of Speech-Language Pathology, 17*(3), 299–317.

Marangolo, P., Rizzi, C., Peran, P., Piras, F., & Sabatini, U. (2009). Parallel recovery in a bilingual aphasic: A neurolinguistic and fMRI study. *Neuropsychology, 23*(3), 405.

MacLeod, C. M. (1991). Half a century of research on the Stroop effect: An integrative review. *Psychological Bulletin, 109*(2), 163–203.

Novick, J. M., Trueswell, J. C., & Thompson-Schill, S. L. (2005). Cognitive control and parsing: Reexamining the role of Broca's area in sentence comprehension. *Cognitive, Affective, & Behavioral Neuroscience, 5*(3), 263–281.

Paradis, M. (2001). *Bilingual and polyglot aphasia. Handbook of neuropsychology* (2nd ed.). Oxford: Elsevier Science.

Penn, C., Frankel, T., Watermeyer, J., & Russell, N. (2010). Executive function and conversational strategies in bilingual aphasia. *Aphasiology, 24*(2), 288–308.

Radman, N., Mouthon, M., Di Pietro, M., Gaytanidis, C., Leemann, B., Abutalebi, J., & Annoni, J. M. (2016). The role of the cognitive control system in recovery from bilingual aphasia: A multiple single-case fMRI study. *Neural Plasticity, 2016,* 1–22.

Randolph, C. (1998). *Repeatable battery for the assessment of neuropsychological status* (RBANS). San Antonio, TX: Psychological Corporation.

Reitan, R. M., & Wolfson, D. (1985). *The Halstead–Reitan neuropsycholgical test battery: Therapy and clinical interpretation*. Tucson, AZ: Neuropsychological Press.

Shallice, T. (1982). Specific impairments of planning. *Philosophical Transactions of the Royal Society of London, 298,* 199–209.

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*(6), 643–662.

Tipper, S. P. (1985). The negative priming effect: Inhibitory priming with to be ignored objects. *The Quarterly Journal of Experimental Psychology, 37A,* 571–590.

Verreyt, N., De Letter, M., Hemelsoet, D., Santens, P., & Duyck, W. (2013). Cognate effects and executive control in a patient with differential bilingual aphasia. *Applied Neuropsychology: Adult, 20*(3), 221–230.

History:
Received April 12, 2017
Revised June 19, 2017
Accepted September 28, 2017
https://doi.org/10.1044/persp2.SIG2.117