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Bilingual aphasia: Explanations in population encoding

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A B S T R A C T

The study of bilingual aphasia has particular power to determine the neural basis of language function and can advance the treatment of aphasia. No theory of bilingual language has provided a coherent and comprehensive account for research results. I begin with a précis of a population encoding model of language function that assumes that representations are based upon the patterns of activity of large populations of neurons. I then consider how regularities in specific domains of language function (e.g. in semantics and phonologic sequence knowledge), frequency effects, and age of acquisition effects (strongest in lexical-semantics) might account for observed behavior in polyglots with aphasia. Finally, I review the literature on bilingual aphasia and its treatment to determine how well these hypotheses account for observed behavior. This analysis reveals that the population encoding theory can provide a coherent and granular account for this behavior.

1. Introduction

Achieving a better understanding of the bilingual brain can enable not just greater insight into how a brain supports multiple languages: it can provide a unique window into how the brain supports language in general. Language function provides us the clearest, broadest window into how the brain functions. Studies of aphasia in bilinguals can be particularly valuable as each patient serves as her own control, thereby enabling direct contrast of the impact of differential language characteristics on aphasia recovery. Studies of bilingual aphasia can also enable a clearer understanding of mechanisms of generalization achieved during language therapy — from one language to another but also within a single language.

The purpose of this paper is to advance this field by taking a hypothesis-based approach, one based most fundamentally on the concept of population-encoding of neural representations and its related science, parallel distributed processing (PDP) (McClelland, Rumelhart & PDP Research Group, 1986), bolstered by the extensive discoveries in the science of language that have been made over the past 50 years, most particularly those stemming from studies of aphasia (Nadeau, 2012). Population encoded representations correspond to activity patterns of millions or billions of neurons distributed over variously extensive regions of the brain. Neuroscientific studies have repeatedly validated the concept of population encoding (Churchland & Sejnowski, 1992; Georgopoulos, Kalaska, Caminiti, & Massey, 1982; O'Keefe & Nadel, 1979; Rolls & Deco, 2002, 2015; Rolls & Treves, 1998; Zhang, Ginzburg, McNaughton, & Sejnowski, 1998; Zhang & Sejnowski, 1999; Lebedev & Nicolelis, 2017).

The field of PDP is a broad one, encompassing scientific investigations of models that are truly population encoded, as well as connectionist models, which employ local representations but incorporate mechanisms that enable spread of activation through the units in the network. Connectionist models introduced the key concept that brain states that yield thought or behavior are the culmination of a dynamic network settling process. They have also been of enormous value in demonstrating the impact of top-down/bottom up interactions. Work done by Gary Dell and his group exemplifies connectionist models at their best (Dell, Schwartz, Martin, Saffran, & Gagnon, 1997). Connectionist models, however heuristically valuable, are not neurally plausible because knowledge in the brain is distributed throughout large domains of synaptic connectivity and they are not capable of capturing the statistical regularities

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of knowledge as it is acquired, a matter of critical importance in understanding bilingual aphasia. For this reason, models built on local representations will not be considered in this paper. The term "population encoded" will be used in lieu of the term "distributed" because "distributed" is presently often used to refer simply to the fact that different regions of the brain are simultaneously engaged in a particular function; "distributed," used in this way, does not make inferences about how knowledge is represented in neural connectivity or how representations are generated as patterns of neural activity. Finally, it should be noted that this paper will not make reference to other comprehensive models of language function employing population encoded representations simply because, to my knowledge, they do not exist.

Forty years of research in the field of parallel distributed processing have substantially defined the unique capabilities naturally conferred by networks supporting population encoded representations (e.g., simultaneous support of processing and long-term memory, content addressable memory, working memory, representation of frequency and age of acquisition effects, the power of regularities wired into network connectivity, graceful degradation, and capacity for parallel constraint satisfaction) and the constraints they impose. These powerful properties are highly germane to understanding how the brain supports language function. Networks supporting population-encoded representations have a remarkable capacity to emulate the behavior of normal and brain-injured human subjects. In fact, the aphasia literature can be understood in a cogent fashion when seen through the lens of PDP (Nadeau, 2012). More generally, the phenomenology of neural representations based upon population encoding reflects principles of that domain of chaos theory (Gleick, 1987) that applies to all phenomena deriving from large numbers of relatively simple units interacting with each other according to their innate principles of function. Thus, PDP models represent not simply another heuristic approach to understanding brain function — they represent an attempt to apply a fundamental organizing principle of life forms, even as any one PDP model represents but a hypothesis.

Most models that incorporate population encoding in simulations employ rather simple unit activation functions, e.g., a sigmoid ($\int$) function over the range of 0–1, rather artificial network training algorithms, and a processing routine that involves serial adjustment of the activity of each unit, perhaps the adjustment of each connection weight, unit by unit, through the entire network, at which point the process starts over and repeats until steady state is achieved. However, there is extensive research on "integrate-and-fire" models that operate in real time and incorporate many of the known details of cortical architecture, including excitatory and inhibitory "neurons," veridical representations of axodendritic connectivity, multiple neurotransmitters and detailed representations of their actions on receptors, and multiple ion currents (Rolls, 2016; Rolls & Deco, 2015). These models are rapidly closing the gap between largely conceptual population encoded models, as discussed in this paper, and neural function as demonstrated in neuroscientific research. However, as sophisticated as they are, and as important as this sophistication is in enabling us to understand brain function, the foundational principles of these models, one that defines how they operate, are population encoding and knowledge representation as strengths of inter-unit connections, corresponding to synaptic strengths.

Whereas the ultimate goal of this paper is to improve our understanding of the bilingual (or multilingual) brain, my principal focus will be on bilingual aphasia. The study of cognitive behavior in individuals with brain injury achieves its enormous power to elucidate the how and the why of brain function because of the phenomenon of graceful degradation that is intrinsic to population encoding neural networks, in which knowledge is instantiated in the strength of connections (e.g., synapses) throughout the network. Damage to such networks does not bring them to a halt (as would occur with digital networks). Rather, it increases the probability of error in a systematic way that reveals the degree to which various functions are wired into neural connectivity, for example, because of differences in atypicality or frequency (contrast platypus with dog in these respects), or variability in consistency of use (e.g., article use in English and German). Because of the phenomenon of graceful degradation, we can, in a real sense, reverse engineer the brain through cognitive neuropsychological study of individuals with brain damage, delving deep into questions of how and why.

I will begin with a very brief introduction (Section 2) to a conceptualization of language and aphasia based upon the assumption of population encoding. This model has been shown to account in an orderly way for the findings of a very large body of studies on disorders of phonology, semantics, and grammar in normal people and in patients with aphasia without the need for ad hoc adaptations or algorithmic components. The reader is referred to a published monograph for further detail (Nadeau, 2012). In section 3, I briefly discuss the results of two fields of investigation, electrocortical stimulation studies and functional magnetic resonance imaging (fMRI), that have attempted to answer a question that has challenged language scientists for over a century: whether different languages are, at least to some extent, located in different regions of the brain. In section 4, I propose a number of hypotheses on the function of the bilingual brain in normal people and patients with aphasia that follow naturally from the model. The final section (Section 5) is devoted to review of the bilingual aphasia literature in an effort to test the value of the hypotheses developed in Section 3 in accounting for the empirical observations that have been made.

2. An introduction to language function from a PDP perspective

2.1. Core functions: phonology, semantics, and lexical-semantics

The domains of knowledge underlying language function that will be the subject of this section are outlined in Table 1. Although certain aspects of my conceptualization may appear to be at odds with many conventional views, the table derives from an extensive scientific literature (reviewed in detail in Nadeau, 2012).

Although the Wernicke-Lichtheim model continues to be a target of criticism, primarily because it remains underspecified (Tremblay & Dick, 2016), its fundamental validity is supported by an enormous body of scientific evidence and it represents a useful place to begin. The topography of the Wernicke-Lichtheim model (Roth, Nadeau, Hollingsworth, Cimino-Knight, & Heilman, 2006) is presented in Fig. 1. Each oval signifies a very large number of individual units. Every unit in a given oval is connected to every unit in

Table 1
Neural Foundations of knowledge underlying grammatic function.

Knowledge Base/function	Brain Location	Grammatic Function
1. Semantic knowledge underlying concept representations	Synaptic strengths in association cortices throughout the brain	Noun representations that are the substrate for modifications produced by adjectives, prepositional phrases, clauses, and verbs (which confer predicative features and thematic role features). Adjective semantic representations. Verb semantic representations. Adverb semantic representations that modify verb semantic representations
2. Concept manipulation	Predominantly left prefrontal cortex and its projections to sensory association cortices and polymodal cortices	Concept priorities, defined by prefrontal systems, dorsolateral and orbitofrontal-limb, and constrained by sentence-level sequence knowledge incorporating, at least in part, verb argument structure, and subcategorization rules
3. Sentence-level sequence knowledge	Predominantly left prefrontal cortex and its connections with other association cortices	Language-specific habits of sequencing of concepts and concept modifications corresponding to word class sequence at the sentence level.
4. Phonologic sequence	Predominantly left perisylvian acoustic-articulatory motor representations pathway	Phrase structure rules, free and bound grammatic morphology as phonotactic sequence knowledge.
5. Morphologic sequence	Perisylvian cortices, left > right	Phrase structure rules, free and bound grammatic morphology as morphotactic sequence knowledge
6. Semantic-phonologic (lexical)	White matter fan arrays linking all types of association cortex with perisylvian phonologic networks	Lexical access: conversion of modified concept representations and concept configurations (superdistributed concept representations) into words and vice versa: the phonologic input and output lexicons and the basis for phonologic neighborhood effects
7. Semantic-morphologic (lexical)	White matter fan arrays linking all types of association cortex and perisylvian morphologic networks	Morphologic access: influence of concepts, concept modifications, and concept configurations on engagement of representations of free and bound grammatic morphemes, and vice versa: the morphologic input and output lexicons
8. Syntactic-morphologic (lexical)	Portions of white matter fan arrays linking prefrontal cortex to perisylvian morphologic networks	Production of words with almost purely syntactic function, for example, complementizers
9. Working memory capacity — maintenance of selective engagement of one or more distributed concept representations and representations of recent conversation	Prefrontal cortex and its projections to sensory association cortices and polymodal cortices	Syntax, meaning in relation to immediately prior conversation, reference (e.g., pronouns, articles)
10. Episodic memory capacity — maintenance of capacity for reengagement of representations of recent conversation. Complements working memory in proportion to length of time over which selective engagement must be sustained (see (Shrager, Levy, Hopkins, & Squire, 2008))	Hippocampus	Syntax, meaning in relation to immediately prior conversation, reference (e.g., pronouns, articles)

From Nadeau SE. The Neural Architecture of Grammar. Cambridge, MA: MIT Press; 2012, with permission.

the adjacent ovals. All connections are 2-way, thereby emulating the brain. Information is represented in the model as the strengths of connections between units (in analogy to synapses in the brain). The activity of units is defined as a nonlinear function of their input (in many simulations as a sigmoid ($\int$) function over the range of 0–1). The output of units is another nonlinear function, typically incorporating a firing threshold. The units in hidden unit domains support representations that cannot be defined in behavioral terms. These domains, coupled with nonlinear unit functions, are essential to the computational capabilities of the network and endow the network with the capacity for translating representations in one domain into substantially unrelated (orthogonal) representations, e.g., concept representations (semantics/word meanings) into phonological representations (word sounds), and representing sequence knowledge (which instantiates implicit rules). The small circles appended to the three major linguistic domains signify that every unit within a domain is connected to every other unit in that domain. This endows these domains with an auto-associator property: the propensity for the activity pattern within the domain to automatically settle into a stable state—an attractor state—that represents an optimal response to the pattern of input. Input to the network leads to spreading activation throughout the network such that, in the course of repeated bottom-up/top-down interactions, the entire network eventually settles into an optimal state, which defines output. Fig. 1 also shows how this network might be plausibly mapped onto the brain surface and it captures that fact that, although language is predominantly supported by the left hemisphere in right handers and most left handers, there is some redundant representation of language knowledge in the right hemisphere (see (Nadeau, 2012) for detail). All of this will become clearer in subsequent sections.

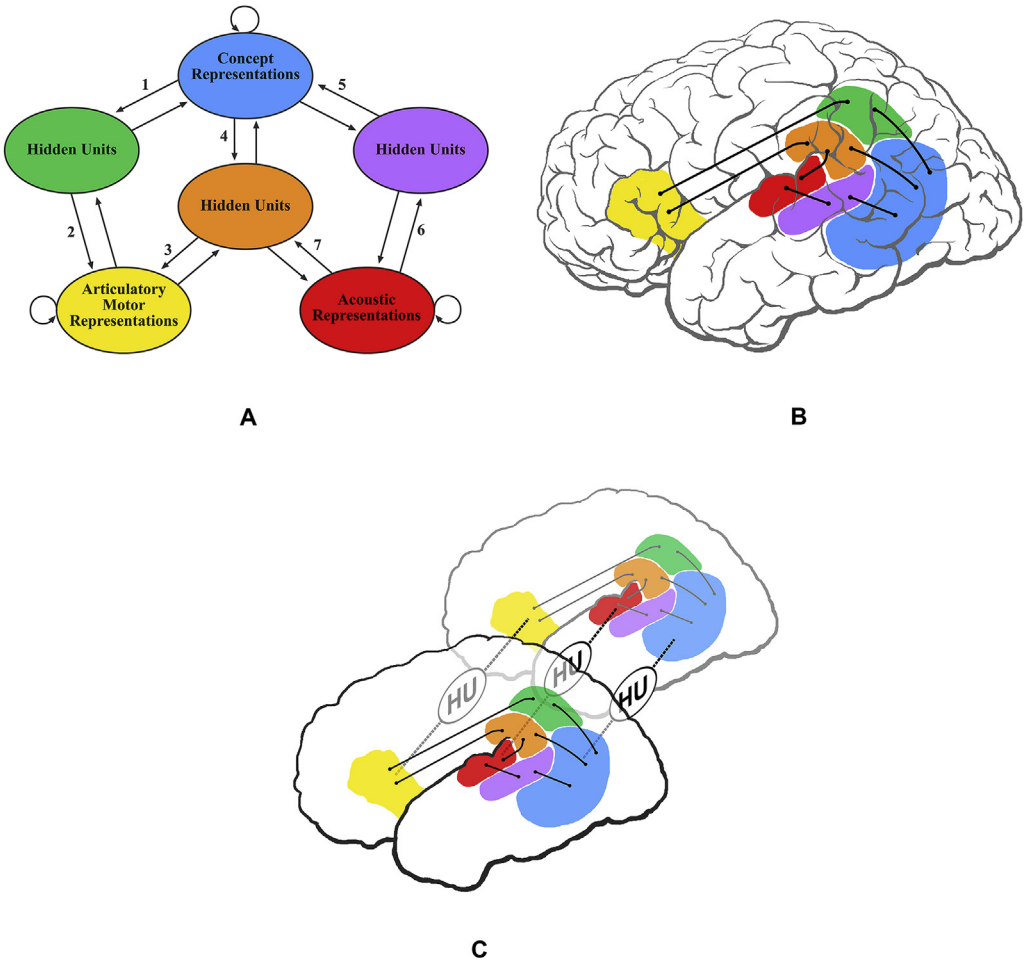

Fig. 1. Parallel distributed processing model of language (1a) with plausible mapping to the brain (1b). There is abundant evidence that, although in most people the left hemisphere is dominant for language, there is substantial redundant language knowledge in the right hemisphere (1c). Domains are shaded similarly in each figure. HU, hidden units. From Roth HL, Nadeau SE, Hollingsworth AL, Cimino-Knight AM, Heilman KM. Naming concepts: evidence of two routes. Neurocase. 2006; 12:61–70, with permission.

2.2. Concept representations: semantics

I will begin with the domain of concept representations because its function provides the best insight into the meaning of a population encoded representation. The operation of this domain can be best illustrated by a model developed by Rumelhart and his colleagues (Rumelhart, Smolensky, McClelland, & Hinton, 1986). This "rooms in a house" model was comprised of 40 "feature" units, each corresponding to an article typically found in particular rooms or an aspect of particular rooms. Connection strengths were defined by the likelihood that any two features might appear in conjunction in a typical house. When one or more units was clamped into the "on" state, activation spread throughout the model and the model eventually settled into a steady state — an attractor state — that implicitly defined a particular room in a house. Thus, clamping "oven" ultimately resulted in activation of all the items one would expect to find in a kitchen and thereby *implicitly* defined, via a *distributed or population encoded representation*, the concept of a kitchen. The model could also generate distributed representations of other rooms in a house (e.g., bathroom, bedroom, living room, study) and blends of rooms that were not anticipated in the programming of the model (e.g., clamping both bed and sofa led to a distributed representation of a large, fancy bedroom replete with a fireplace, television, and sofa). This auto-associator model, simple though it is, has the essential attributes of a network that might instantiate semantic knowledge and be capable of generating distributed representations corresponding to concepts.

Let us now scale this up to a concepts network that could plausibly support the knowledge in the human brain. Knowledge is accumulated throughout the life span on the basis of individual experiences and incorporated into cerebral cortex as changes in synaptic connection strengths, either directly (as in procedural and implicit memory acquisition) or via the hippocampal system (episodic memory acquisition). This encoding of knowledge in neural connectivity enables the generation of concept representations, which are population encoded (distributed), as patterns of features, but ultimately, as patterns of neural activity involving very large numbers of cortical micro-columns and billions of neurons (Nadeau, 2012). The mathematical function of cortical activity is a surface

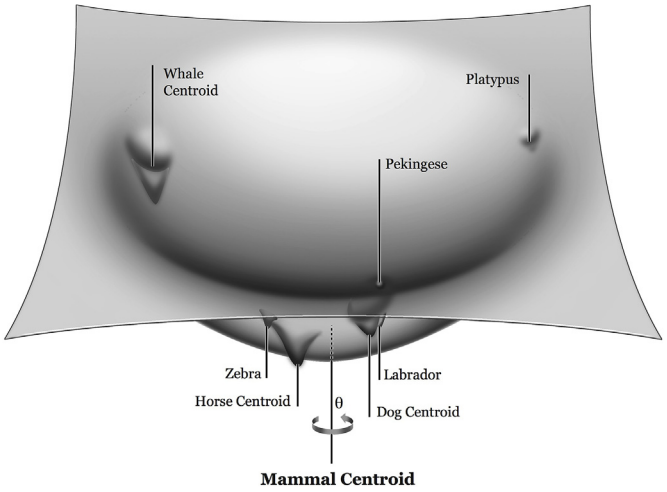

Fig. 2. The topography of the semantic network energy function in the vicinity of the mammal attractor basin. Each point corresponds to an energy level of all features in an N-dimensional feature hyperspace. The point of maximal typicality is represented by the centroid of a basin/sub-basin. Distance from the centroid reflects degree of atypicality. The value of θ defines the manner in which atypicality is defined. For example, whales and platypuses are both atypical but in very different ways. From Nadeau SE. The Neural Architecture of Grammar. Cambridge, MA: MIT Press; 2012, with permission.

in an N-dimensional hyperspace. To understand how this might look, we can do a thought experiment, taking a 3-dimensional slice of the corresponding energy landscape, just as we might take a 2-dimensional slice of a 3-dimensional loaf of bread, for example, a slab in the vicinity of mammal knowledge (Fig. 2). The central, lowest "energy" point — the "centroid" of mammal knowledge — corresponds to the representation of a creature that best defines our sense of mammalness. Within the mammal basin, there are innumerable attractor sub-basins corresponding to specific mammals. Very close to the centroid are sub-basins corresponding to mammals likely to be very close to the centroid representation, e.g., dogs, cats, cows, and horses. Distance from the centroid is defined by the degree of atypicality, which reflects feature and feature combination frequency within the mammal domain. Highly atypical animals, such as whales and platypuses, are represented near the periphery of the mammal attractor basin. Within any given sub-basin, there may be sub-sub-basins, for example, corresponding to types of dogs. The depth of the mammal basin and its sub-basins (the z-axis in Fig. 2) is determined by the strength of encoding of knowledge in neural connectivity. This in turn is determined by the degree to which a given exemplar shares features with other exemplars in the domain, the frequency of the exemplar, and age of acquisition. The network's settled activity state is most strongly influenced by the specific input features, which in most circumstances will absolutely define the sub-basin or sub-sub-basin into which the network settles, all the other factors exerting their major influence either on response latencies or the occasional errors. Errors will consist of slips into nearby sub-basins. This settling in response to input features instantiates *content-addressable memory*. This capacity for content addressable memory, which is intrinsic to auto-associator networks supporting distributed representations, automatically accounts for the fact that, with exposure to but scant features of a memory, we can instantly realize the full memory.

The effect of lesions (focal or diffuse) will be to produce *graceful degradation* of network performance. Network function does not simply cease. Instead, it becomes less reliable and more errorful, even as it continues to reflect the statistical regularities of remaining knowledge encoded in the network. With network damage, deep basins will become shallower and sub-basins, particularly those that are shallower and more distant from the centroid — corresponding to more atypical exemplars—will disappear. As sub-basins become shallow or disappear, responses will reflect the settling of the network into surviving neighbors located nearer the centroid — neighbors of higher typicality (yielding coordinate errors, e.g., horse in lieu of donkey), the parent basin (yielding superordinate errors, e.g., animal in lieu of donkey), or failure to settle at all, yielding omission errors. This is precisely what has been observed in studies of semantic dementia. This conceptualization of the neural basis for semantic knowledge is not at odds with recent formulations (Lambon Ralph, Jefferies, & Patterson, 2017), although I would argue that the behavior of patients with semantic dementia is adequately accounted for on the basis of loss of more detailed knowledge, by virtue of loss of neural connectivity, and one does not need to invoke, as Lambon Ralph et al. did, a topographical explanation that prioritizes the temporal pole.

Implicit in the discussion thus far are some fundamental principles, understanding of which is essential to understanding the function of a brain that supports population encoded representations (Fig. 3) and the development of aphasia therapies that have the potential for generalization. In our discussion of concept representations, we considered a mathematical activity function in N-dimensional hyperspace. Depending on the particular pattern of input, this function would yield a distributed representation of any entity we know about. The mammal attractor basin (Fig. 2) is the product of a thought experiment in which we took a 3-dimensional slice through the energy landscape corresponding to an N-dimensional activity function in the vicinity of mammal knowledge. The shape of this complex surface reflects the strength of knowledge regularities wired into neural connectivity and shared, to varying degree, by all mammals. The mammal attractor basin corresponds in psychological terms to a quasi-regular domain. It is regular to

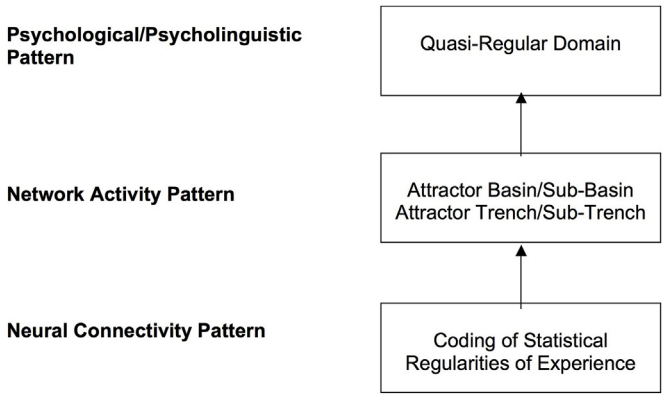

Fig. 3. The relationship between neural network connectivity, attractor basins and trenches, and patterns of behavior (Sections 2.2 and 2.3). From Nadeau S.E. (2012) The Neural Architecture of Grammar. Cambridge, MA: MIT Press; with permission.

the extent that all mammals share many features. However, it is quasi-regular because no two mammals are exactly alike.

There is good evidence that in the brain, the meaning of a given word is distributed not over one network, as we have been discussing, but over a number of networks. This idea, which owes to Lissauer (Lissauer, 1988) and Wernicke (as cited in Eggert, 1977), has recently been resurrected as the Embodied Cognition Framework (Buxbaum & Kalénine, 2010; Fernandino & Iacoboni, 2010; Spivey, 2007) and neural assemblies (Pulvermüller, 2013). The fundamental idea is that the distributed representation of the concept "dog," (for example) has major components in visual association cortices (incorporating knowledge of the visual appearance of dogs in general, as well as particular dogs); auditory association cortices (sounds that dogs characteristically make); the limbic system (one's feelings about dogs); somatosensory cortex; olfactory cortex; frontal cortex (supporting a predicative component corresponding to our knowledge of what dogs do or have done to them — a component of the semantic representation of a verb); and perisylvian language cortex, which enables us to translate the semantic representation of dog into an articulatory motor representation (Fig. 4). The multi-component representation of concepts provides the basis for category specific naming and recognition deficits (Forde & Humphreys, 1999; Warrington & Shallice, 1984).

2.3. Phonology

We return now to Fig. 1 to consider the acoustic representations-articulatory motor representations pathway. This is referred to as a pattern associator network because it translates representations in one form into corresponding representations of a different form. Acoustic and articulatory motor representations correspond to the acoustic and articulatory forms of phonemes, respectively. As conceptualized in the Wernicke-Lichtheim model, this is the pathway that supports repetition. In addition, because this network has acquired, through language experience, knowledge of the systematic relationships between acoustic and articulatory sequences, it has learned the statistical sound *sequence* regularities of the language: the phonemic sequences of joint phonemes (e.g., st, str), rhymes, syllables, affixes, morphemes and words characteristic of the language (Nadeau, 2001). I will use a reading model to illustrate the process (Plaut, McClelland, Seidenberg, & Patterson, 1996; Seidenberg & McClelland, 1989). It fundamentally recapitulates the acoustic-articulatory motor pathway of Fig. 1, the major difference (inconsequential to this discussion) being that in place of acoustic representations, it employed orthographic representations. The 3-layer pattern associator network was equipped with a learning algorithm and it was trained by presenting the orthographic representations of 3000 English single-syllable words and their corresponding phonologic forms.

One of the most striking things about the trained model was that it was able to produce correct pronunciations of plausible English nonwords (i.e., orthographic sequences it had never encountered before). This was possible because the model had learned the statistical relationships between *sequences* of graphemes and *sequences* of phonemes that are characteristic of the English language. To the extent that there is a limited repertoire of sequence types, the model was able to learn it and apply it. Certain sequences, those most commonly found in English single syllable words, were more thoroughly etched in network connectivity. Thus, it was very fast with high frequency words. It was also very fast with words with an absolutely consistent orthographic-phonologic sequence

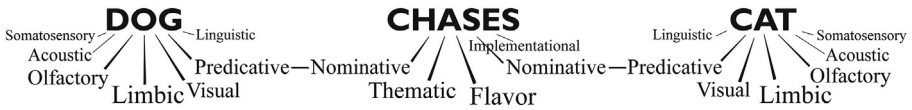

Fig. 4. The multifocal distributed representation of a sentence. The multi-regional distribution of noun knowledge is discussed in Section 2.2. Verbs have an analogous multi-regional distributed representation, including frontal components involved in the incorporation of thematic role(s), post-central components instantiating verb flavor (manner, path, and limbic representation), an implementational component in motor cortex instantiating movement, and a nominal component corresponding to linked noun representations. From Nadeau SE. The Neural Architecture of Grammar. Cambridge, MA: MIT Press; 2012, with permission.

relationship, for example, words ending in "ust", which are always pronounced/*ʌst*/(*must, bust, trust, lust, crust*, etc.). The model encountered difficulty (reflected in prolonged reading latency) only with low frequency words, and only to the extent that it had learned different, competing pronunciations of the same orthographic sequence. Thus, it was slow to read *pint* because in every case but *pint*, the sequence "int" is pronounced/*Int*/(e.g., *mint, tint, flint, lint*). It was very slow with words that are unique in their orthographic-phonologic sequence relationship (e.g., *aisle, guide* and *fugue*). These behaviors precisely recapitulate the behavior of normal human subjects given reading tasks (Plaut et al., 1996; Seidenberg & McClelland, 1989). This particular model was equipped to acquire the very limited set of sequences involving syllabic onset, nucleus, and coda. However, it provided proof of the principle that sequence knowledge in general can be acquired by a simple pattern associator neural network employing a hidden unit domain and units with nonlinear functions.

The fundamental concepts discussed in Section 2.2 (semantics) — the correspondence between patterns of knowledge encoded in neural network connectivity, attractor basins in an energy landscape of neural activity, and quasi-regular domains of knowledge — can be applied to sequence knowledge in pattern associator networks, e.g., acoustic-articulatory or orthographic-articulatory. Because pattern associator networks support a pathway from one representation to another, the regularities encoded in their neural connectivity support activity patterns that I have termed attractor trenches (Nadeau, 2012). These attractor trenches also correspond to psychological quasi-regular domains. The neural activity patterns generated by phonologic sequence knowledge supporting words with the rhyme "int" correspond to one such domain, this one containing two sub-trenches, one corresponding to *lint, mint, stint,* and *tint*, and one corresponding to *pint*. Fig. 3 illustrates the fundamental principle that knowledge is represented in patterns of neural connectivity, these patterns define the shapes of attractor basins and attractor trenches, and these basins and trenches correspond to the psychological form of quasi-regular domains.

2.4. Lexicons: lexical-semantic knowledge

The phonologic input lexicon is represented as the connections between the substrate for acoustic representations and the substrate for concept representations (Fig. 1: pathway 6-5). The phonologic output lexicon is represented as the two pathways between the substrate for concept representations and articulatory motor representations (pathways 1–2 and 4-3). As a reminder of this instantiation of lexicons as connections, I will henceforth use the term semantic-phonologic (lexical) knowledge. Evidence of the existence of pathway 1–2 derives from patients with repetition conduction aphasia who demonstrate the ability to repeat by the semantic route but have no capacity for repeating nonwords (Nadeau, 2001; Roth et al., 2006). Evidence of the existence of pathway 4-3 comes from normal subjects, who make phonologic slips of the tongue, and from patients with reproduction conduction aphasia, who make phonemic paraphasic errors in both repetition and naming. We have no good evidence that lexicons in different languages, however early- or late-acquired, are based upon fundamentally different neural connectivity.

2.5. Grammatic sequence knowledge

Grammatic sequence knowledge is evident at two levels: 1) grammatic morphology, which subsumes roots, grammatic inflections (e.g., affixes), and phrase structure rules; and 2) sentence level sequence, that is, syntax. Simulations involving a simple pattern associator network, such as the reading model of Plaut and colleagues (Plaut et al., 1996; Seidenberg & McClelland, 1989), provide proof of principle of the ability of simple PDP models to acquire sequence knowledge, albeit limited to onset-nucleus-coda of single syllable words. More powerful PDP architectures have been devised, tested, and proven capable of acquiring sequence knowledge at the sentence level (Joanisse & Seidenberg, 2003). Studies of macaques have provided neurophysiological evidence of population encoding of acquired sequence knowledge (Carpenter, Georgopoulos, & Pellizer, 1999).

Grammatic morphology, as defined here, involves sequence knowledge at the lexical and sublexical level. This knowledge could be represented in the acoustic-articulatory motor pathway of our model but there is evidence in the aphasia literature of dissociations between disorders of phonologic and morphologic sequence, suggesting that a second pathway exists (Table 1, #5). In a diffusion tensor tractography study, we have recently confirmed the existence of this pathway (first discovered by Frey, Campbell, Pike, & Petrides, 2008), originating in the transverse gyrus, connecting to mid-superior temporal gyrus, and thence, via the extreme capsule, to pars opercularis of Broca's area (Bohsali et al., 2015).

Syntax involves the sequential engagement and modification of one or more distributed concept representations (Nadeau, 2012). In language production, constraint is imposed by the intentions of the speaker, the speaker's acquired knowledge of sentence level sequence (Table 1, #2 and #3), and the effects on distributed noun concept representations of their association with specific adjective, verb, and embedded clause representations. These distributed representations are supported by association cortices throughout the brain. It is only the automatic engagement of the lexical-phonological core by these representations (Fig. 1) that yields actual spoken language.

2.6. Population encoding: a look backward

Many have been inclined to dismiss population encoding models out of hand because of the perception that they connote a vast, undifferentiated cortical network that, through vastly underspecified neurodynamical mechanisms, somehow achieves specialization of functions in language and other domains. Thus, it has seemed to many to be inconceivable that population encoding could be reconciled with traditional models of language function, derived from anatomy, psychology, neuropsychology, cognitive neuropsychology, and psycholinguistics, now supported by vast empirical evidence. In fact, PDP models employing population encoding

are fully compatible with these findings because they can easily be reconciled with the network heterogeneity of the brain. Domains of specialized knowledge, e.g., semantic knowledge and phonologic sequence knowledge, are isolated but linked by major white matter connectivity, which is now being defined fairly precisely through diffusion tensor tractography. Two domains so-connected comprise a pattern associator network (McClelland et al., 1986). Knowledge within domains, e.g., semantics or phonologic sequence, is "compartmentalized" to one degree or another by virtue of 1) variously sparse or dense coding and 2) differential connectivity to other regions of the brain. For example, there is relatively dense coding throughout the domain of mammals — units, cortical microcolumns, and neurons are highly interconnected with each other. This reflects the enormous body of knowledge common to all mammals and various subgroups of mammals (as between the features supporting the kitchen representation in the "rooms in house" model of section 2.2). On the other hand, there is relatively sparse coding between mammal and tool domains — relatively little interconnectivity between the units in the two domains, hence relatively little activation in one domain by activity in the other domain (as between features supporting the kitchen and bathroom representations). Second, mammal and tool domains have very different connectivity to other brain regions that provide the substrates for important parts of their semantic representations. For example, mammals have predicative representations that reflect the behaviors to be expected of them, as well as limbic representations that reflect our emotional feelings about them. Tools, on the other hand, have representations that correspond to the particular uses to which they are put, the manner of their use, and the motor instantiation (embodiment) of their use. Heterogeneity in density of coding and in connectivity with other brain regions, together, provide the basis for separation of domains within the neural substrates for semantic knowledge.

Phonologic sequence knowledge is very different in nature from semantic knowledge. The orthogonal relationship of semantic and phonologic sequence knowledge means that different domains of phonologic sequence knowledge are minimally distinguished by their connectivity to other brain regions. However, the domain of phonologic sequence knowledge corresponds to a very large attractor trench populated by a large number of subtrenches and surrounded by other, unrelated trenches, some corresponding to but a single exemplar ("hermits") (Vitevitch & Castro, 2015; Vitevitch & Luce, 2016). Network connectivity supporting trenches is relatively dense. Connectivity between domains supporting different trenches is sparser.

Thus, PDP, population encoding, and the complexities of the cortical mapping of language function, as revealed by a century and a half of research, are fully compatible. Furthermore, this largely conceptual "compartmentalized" population encoding model is entirely compatible with emerging neurodynamical accounts (Gerstner, Sprekeler, & Deco, 2012; Rolls, 2016; Rolls & Deco, 2015).

PDP models incorporating population encoding assume that the brain's response to input and the production of output representations are realized as the culmination of a dynamic process involving rapid intercommunication between all of the neural networks engaged by the process as their energy landscapes settle into attractor basins and trenches and finally, into an attractor state. There is unlikely to be an attractor state that is perfect for all involved networks. Rather, the settling process is shaped by the constraints imposed in parallel by all of the engaged networks (parallel constraint satisfaction) to arrive at a quasi-optimal state that represents the best fit. This occurs in both a normal brain and a damaged brain but is further perturbed by neural network noise (hence stochastic errors), which is greater in the damaged brain. The process is rapid indeed. A 1000 ms response latency provides the opportunity for 150 back and forth transmissions between the occipital pole and the frontal pole, assuming a central conduction velocity of 50 m/s. This concept of dynamic cerebral network settling is dramatically different from traditional serial processing models, e.g., that of Levelt (Indefrey & Levelt, 2004; Levelt, Roelofs, & Meyer, 1999), even those that incorporate top down/bottom up processes (Strijkers & Costa, 2016). Dynamic settling in the motor system was well established as far back as the early 1990s (Porter & Lemon, 1993). In this new conceptualization, latencies (Indefrey & Levelt, 2004), as defined by electrophysiologic studies, reflect not sequential processing steps but stages in the evolution of the settling process.

One final point: recognition that representations are population encoded in the brain has enabled a major advance in our understanding of language function and aphasia, unilingual (e.g, Nadeau, 2012) and bilingual (this paper). However, this is only a first step. To fully understand the connections between brain function and language, unit activity functions and attractor basin/attractor trench neurodynamics will have to be understood in all of their complex detail. This will include an understanding of unit firing patterns in the various types of neurons involved, how they evolve over the milliseconds of settling into attractor basins and attractor states, and the various ion currents involved. We will need to achieve a far better understanding of learning processes (synaptic modification) in the cerebral cortex. The sigmoid activation functions and backpropagation learning methodology that are commonly used in simulations of PDP models ultimately will not suffice. Extensive research along these lines is underway, exemplified by the work of Edmund Rolls (2016). I have been able to invoke these neurophysiologic advances as a possible explanation for the anomia that becomes nearly ubiquitous as we age (Nadeau, 2018) but we are not yet prepared to apply this knowledge to understand bilingual aphasia.

3. Differential language localization in the brain

One of the questions that has been at the forefront of research on bilingualism, dating back to the earliest studies in the nineteenth century, has been whether, in a bilingual speaker, knowledge of different languages is located in different regions of the brain. In this section, I will review the results of two major technologies, electrocortical stimulation mapping and functional imaging, that have had a substantial influence on thinking about the bilingual brain and bilingual aphasia. Electrocortical stimulation studies, whether of the monolingual brain or the bilingual brain, are beset with a number of very serious methodological problems that seriously undermine their validity even as these problems are generally unrecognized. Functional imaging studies have not made an important contribution to our knowledge about the bilingual brain but, because of their influence, some discussion of the major findings seemed essential to this paper.

3.1. Electrocortical stimulation studies

There have been many electrocortical stimulation studies that have reported language-specific regions in bilinguals (Bello et al., 2006; Giussani, Roux, Lubrano, Gaini, & Bello, 2007; Lucas, McKhann, & Ojemann, 2004; Ojemann, Ojemann, Lettich, & Berger, 1989; Roux et al., 2004; Roux & Trémoulet, 2002; Walker, Quiñones-Hinojosa, & Berger, 2004). The limitations of these studies are substantial and bear careful consideration:

1. Electrocortical stimulation to localize language function preparatory to surgical resection is constrained on the one hand by the need to use sufficient current to induce local cortical dysfunction and on the other by the risk of inducing after-discharge that could spread to neighboring or connected regions, reducing accuracy and putting the patient at risk for an intra-operative seizure. To further complicate matters, current threshold for inducing local dysfunction may exceed the threshold for inducing after-discharge and there is high threshold variability within and between patients (Pouratian, Cannestra, Bookheimer, Martin, & Toga, 2004). It is furthermore possible that threshold for inducing dysfunction varies over the course of multiple stimulations and that it is different for different languages as a reflection of differential depth of neural encoding. In some centers, stimulation is done routinely at a current level below that producing after-discharge and the same stimulation intensity is used at all sites (Lucas et al., 2004; Ojemann et al., 1989). Under these circumstances, stimulation could have been below threshold at many sites.
2. Reproducibility through the course of several stimulations is often a criterion for implication of a site in language function (Pouratian et al., 2004; Roux et al., 2004; Roux & Trémoulet, 2002; Walker et al., 2004). However, if stimulation of a given site elicits language impairment only 1/3 times, is it reasonable to conclude that this site is not involved in language function? Both factors 1 and 2 could account for the apparently sparse and non-contiguous pattern of language representation and high variability between individuals reported by Ojemann (Ojemann, 1991).
3. Evidence of language specific regions derived from stimulation of a given small cortical region has to be reconciled with the basic neuroanatomy, the fact that representations are distributed over substantial areas, and the fact that the neural networks that support them have the intrinsic property of graceful degradation. Stimulation maps are typically striking for the low percentages of patients who experience language impairment (typically impairment in naming to confrontation) at multiple sites within well-accepted language regions. (e.g., Ojemann et al., 1989; Rapport, Tan, & Whitaker, 1983; Roux et al., 2004). Ojemann et al. in fact noted that in only 65% of 117 patients were there sites, stimulation of which caused anomia — an observation fundamentally at odds with the anatomy. This could in part reflect the effects of graceful degradation. It could also reflect insufficiently intense stimulation at many sites (see 2 above). Finally, the failure to interfere with language function by stimulating at a given site could also reflect greater resilience of that particular language because of redundancy of neural network encoding.
4. Sustained electrocortical stimulation at one location could induce fast Hebbian learning in directly connected regions. This could alter the responsivity of these regions, or even temporarily suppress responsivity, thereby potentially leading to apparent differences in loci of language localization that are related to artifacts borne of stimulation order, location, and duration.
5. Control of language set may not have been adequate. If the patient entrains one set during stimulation of one site and a different set during stimulation of another, this could mislead investigators into inferring language-specific regions, particularly so as correct responses in the wrong language are commonly scored as errors. Set may be susceptible to influence by the predominant language spoken in the hospital in which the study was done and the native language or appearance of the tester.
6. To the extent that regional language specificity is demonstrated in the brain, it is far less likely to be defined by specific languages per se and more likely to be related to differences in mechanisms engaged by different languages, however limited they may be. For example, evidence of better-developed redundant morphologic sequence knowledge in the right hemisphere in subjects who speak richly inflected language (Nadeau, 2012) might predict less disruption in grammatic morphology with stimulation of left hemisphere perisylvian cortex when bilinguals speak these languages than when they speak English.

These various caveats in mind, there are no electrocortical stimulation studies that provide compelling evidence of differential cortical language representation.

3.2. Functional imaging

Functional imaging has not contributed significantly to the elucidation of function in the bilingual brain. However, there are also serious methodological concerns. Most fundamentally, even as we have abundant evidence that representations are population-encoded in the brain, functional imaging paradigms, by virtue of the mathematics underlying statistical parametric mapping and individual differences in cytoarchitectonic mapping, tend to localize areas of engagement to relatively small regions. In effect, we see only the peaks of the mountains of neural activity. Hyperlocalization is compounded by limitations of sensitivity, such that only the most strongly engaged regions or the regions that represent the greatest degree of differential engagement between tasks of interest appear on the composite image. This is a problem that besets functional imaging studies in general but it is particularly challenging when one is seeking differential localization of specific processes in two languages.

Second, it is very difficult to determine if the way that one or more brains are engaged by a particular task is the way that the experimenters intended in designing the task. This problem is compounded by the challenge of determining whether a specific region of heightened synaptic activity is essential or incidental to task performance, and whether, in the case of language studies, it reflects differential engagement of particular regions by the two languages or whether it reflects the differential engagement of ancillary processes while speaking one of the languages. For example, in their meta-analysis, Sebastian, Laird, and Kiran (2011) reported

similar patterns of cortical engagement by L1 and L2 in participants highly proficient in both languages, a finding quite congruent with other functional imaging studies (Abutalebi, 2008; Hernandez, Dapretto, Mazziotta, & Bookheimer, 2001; Vingerhoets et al., 2003). However, they found additional activity in supragenual anterior cingulate and left prefrontal cortex in association with L2 use in participants with low proficiency in L2. Many studies suggest that the supragenual anterior cingulate cortex is best understood as an intentional gate — the final processing region at which one of several plans in play is actualized as movement or thought (Nadeau et al., 2002). Thus, enhanced activity could reflect greater competition between two language-specific plans. Enhanced left prefrontal activity could reflect the greater need to suppress potential responses in the more proficient language while actually responding with the less proficient language. Both of these interpretations are compatible with those of the authors (Sebastian et al., 2011; Sebastian, Kiran, & Sandberg, 2012). Yang, Tan, and Li (2011) found similar loci of engagement with a lexical decision task for Chinese and English nouns and verbs in Chinese-English bilinguals with considerably less L2 proficiency. However, the extent of engagement was larger for L2, a pattern commonly encountered with increased task difficulty.

4. Hypotheses on aphasia in the bilingual brain

It is highly unlikely that the substrates for different languages are separately partitioned in the brain of a bilingual speaker because the business of individual neurons is primarily defined by their patterns of connectivity. Different language functions are localized to different regions of the brain because these regions, by virtue of their connectivity patterns, were equipped to acquire their specific knowledge (instantiated in synaptic connection strengths) *through the experience of verbal communication*. The regional connectivity patterns are the same, regardless of language spoken. Thus, auditory cortices are engaged by hearing spoken words and patterns of auditory-articulatory connectivity become established between auditory association cortices and various subregions of Broca's area as language is learned (establishing the basis for phonological sequence knowledge and the ability to speak words), no matter what the language. Through experience, association cortices throughout the brain acquire knowledge of the world and the objects within it, again regardless of language spoken (though, as we shall see, there may be some language-specific differences in the details connectivity underlying this knowledge). Association cortices are linked to dominant perisylvian cortex by broad white matter pathways; the precise pattern of strengths of connections will differ because two languages employ different words but the neuroanatomy that is engaged will be the same. Syntax involves manipulation of concept representations, constrained in language-specific patterns by sequence habits acquired during learning; nevertheless, frontal cortices (predominantly left) and their connectivity with post-central cortices will be engaged regardless of language spoken. In short, the principle that different languages in a bilingual speaker derive from the same anatomic substrate is so fundamental and there is so much empirical evidence from studies of bilingual subjects that support it (Edmonds & Kiran, 2004; Green & Abutalebi, 2008; Miozzo, Costa, Hernández, & Rapp, 2010) that any evidence against it should be scrutinized with particular rigor.

Some have argued that different patterns of language recovery might be related to differences in the processes of learning of first and second languages rather than to differences related to proficiency and regularity. Specifically, it has been proposed that, whereas procedural memory systems (basal ganglia and cerebellum are often cited as key structures) are engaged in the acquisition and use of a person's first language, declarative memory systems (cerebral cortex and hippocampal structures) are engaged in second language learning (M Paradis, 1994, 2009; Ullman, 2001a, 2001b, 2005). Linguistic knowledge encoded as procedural memory could, it is argued, provide the basis for greater automaticity of L1, and, by virtue of the structures ostensibly providing the substrate for this procedural knowledge, the basis for relative sparing by vascular lesions. There are many problems with this proposal.

First and foremost, language is rules and symbols. Population encoding theories of brain function easily accommodate these (implicit) rules and symbols through nonlinear mathematical functions and auto-associator networks, which enable network activity to settle into attractor basins and attractor trenches. Rules and symbols bear the hippocampal signature — they are learned all at once, if only through repeated exposures, and not the accumulation of successively better approximations — the signature of procedural learning. This is obviously true for semantic knowledge and semantic phonological (lexical) knowledge, but it is also true for syntax and grammatic morphology. Each language has a limited portfolio of implicit rules bearing on morphologic sequence and involves the use of various permutations and combinations of grammatically acceptable sequences. The grammatic morphology of any given language allows for only certain discrete possibilities. For example, a child learning the past tense of “come” may say “come,” “came,” “comed,” or “camed” but not a series of progressively better articulatory approximations to “came” (Rumelhart & McClelland, 1986) — still rules and symbols even if not necessarily the correct ones. Actual articulation of language sounds is characterized by successive approximations (the signature of procedural knowledge acquisition) but the respective role of episodic (declarative) and procedural learning systems in the acquisition of phonologic sequence knowledge and phonetic knowledge remains unclear.

Second, declarative and procedural mechanisms relate primarily to how knowledge and skills are acquired and only secondarily to the location of the substrates for this knowledge. Declarative memories are acquired all at once, as episodic memories, via the hippocampal system, and over time, are gradually consolidated in cerebral cortex to the extent that they share features with other cortically stored knowledge (Alvarez & Squire, 1994; McClelland, McNaughton, & O'Reilly, 1995; Rolls & Treves, 1998; Squire & Zola-Morgan, 1991). To the extent that memories do not share features, e.g., to the extent that they are linked to specific places and times, they remain hippocampally dependent, as in the case of autobiographical memory (Vargha-Khadem et al., 1997). Procedural memories are acquired incrementally through gradual adjustment of synaptic strengths in the neural networks that support them, completely independently of hippocampal systems. Thus, even if phonological, semantic-phonologic (lexical), semantic and grammatic knowledge were acquired via different learning mechanisms in L1 and L2, this would have no implications for the location of these knowledge stores in cerebral cortex or in any way imply that these knowledge stores would be different in the two languages.

Likewise, for metalinguistic approaches that might be employed by new language learners, e.g., naming objects in native language, then translating, except to the extent that such metalinguistic approaches require additional executive function to support the processes.

Third, as I have noted, the business of neurons primarily reflects their pattern of connectivity with other regions of the brain — which also defines the nature of the data that are encoded in this connectivity in the process of language learning, regardless of language learned.

Fourth, functional imaging studies (Section 4.2) have provided no compelling evidence of any differences in language localization (including frontal cortices particularly implicated in grammatic function), much less the dramatic differences that would be observed were the declarative-procedural hypothesis valid.

Fifth, close consideration of the scientific evidence unequivocally demonstrates that the neostriatum is not involved in language function, with the single exception of the implementational component of movement verb representations (Nadeau, 2012; Nadeau & Crosson, 1997). Although there is a lively ongoing scientific debate on the role of the cerebellum in higher neural function, aphasia, as seen with left cerebral hemisphere lesions, is not observed with cerebellar lesions.

Finally, the declarative/procedural, cortical/subcortical hypothesis fails the empirical test: if true, the type of aphasia in L2 and later acquired languages would be the same and it would consistently be fundamentally different from the type of aphasia in L1. However, no convincing cases have been reported.

In contrast to the declarative/procedural theory just discussed, differences in language structure and experience, as well as culture, understood through the lens of the population encoding model I have introduced, provide the basis for strong hypotheses about patterns of language breakdown in aphasia in monolingual and multilingual people, hypotheses that, as will be shown, have received strong empirical support. Three major factors contribute to the strength of encoding of knowledge in the brain: shared features encoded as regularities in neural network connectivity, word/sequence frequency, and age of acquisition (Nadeau, 2012). These factors will be detailed in Sections 4.1-4.3.

The topic of bilingual aphasia poses additional challenges. First, different languages are learned in different contexts and therefore, may reflect somewhat different semantic vocabularies. Two languages may be learned in different ways: one in the traditional way through verbal intercourse during infancy and childhood, the other involving major recourse to written language and writing with later refinement of speaking knowledge and skills (as is often the case with late-acquired languages). This is the topic of section 4.4, Context Effects.

Languages may differ substantially in their grammar in ways that make certain errors impossible in some, an extreme example being the nearly complete absence of grammatic morphology in Chinese, hence absence of grammatic morphologic errors in Chinese speakers with aphasia (noun classifiers excepted). These differences, the subject of Section 4.5, may substantially affect how one assesses degree of recovery from brain damage.

Language set, that is intentionality to speak, may be disrupted in some monolingual patients with aphasia, resulting in a variety of errors, including non-responses, perseverative responses, or responses that are grossly inappropriate to the set the examiner is trying to maintain but may be appropriate to the (perhaps occult) set that the patient is stuck in. Language set acquires an entirely new dimension in patients with bilingual aphasia because, in this circumstance, it also involves selection by the patient of the language to be spoken. Pathologic switching may occur between the two (or more) languages and, in some cases, via mechanisms that are starting to come to light, such switching may worsen aphasia. This will be the focus of Section 4.6.

Research on treatment of aphasia necessarily requires some consideration of timing of treatment as language improvement due to spontaneous recovery is often conflated with improvement that can be credited to treatment. This is the topic of Section 4.7.1. The holy grail of aphasia therapy is generalization to untrained exemplars and daily verbal communication. Therefore, it is necessary to consider mechanisms of generalization and the extent to which they might enable generalization of treatment in one language to performance in a different language — the topic of Section 4.7.2.

4.1. Regularities encoded in neural network connectivity

Language proficiency corresponds to the depth of encoding of linguistic knowledge, which reflects regularities of experience, and the effects of personal frequency of exposure to concepts, words, and phonologic/grammatic morphologic/phrase/sentence level sequences in a given language (akin to familiarity). Relative depth of encoding of linguistic knowledge of two different languages in the brain is the major determinant of their differential trajectory of recovery after stroke and their trajectory of decline in dementia. The most powerful contributor to the strength of knowledge representations in the brain is the sharing of features encoded as regularities in neural network connectivity. The best example is semantic knowledge, in which, for example, our knowledge of dogs to a great extent reflects our knowledge of animals in general, accumulated over the years from encoding of memories deriving from brief experiences with innumerable individual animals. However, regularities encoded in neural network connectivity also powerfully constrain phonological sequence knowledge (e.g., reading “ust” words), grammatical morphology (e.g., regular verb past tense forms), and syntax (e.g., the subject-verb-object order of English). One linguistic domain derives almost no benefit from regularities: semantic-phonologic (lexical) knowledge (the connectivity between substrates for semantics and phonology), simply because word meaning and word sound have almost no relationship to each other except for onomatopoeic words and derivational forms (e.g., governor, government).

4.1.1. Semantics

Semantic knowledge constitutes knowledge at the conceptual level. It seems unlikely that the neural substrate for such knowledge

differs in major ways between two or more acquired languages. However, there are important caveats. The dimensionality and scope of seemingly transparent concepts may differ substantially between languages (Majid, Boster, & Bowerman, 2008; Marian & Kaushanskaya, 2007). Many words in one language may connote shades of meaning that are difficult to capture in another. Cross-linguistic naming is more similar for concrete nouns than for abstract nouns; for cognates than for non-cognates; and for nouns than for verbs (Van Hell & De Groot, 1998) (cognates are words with the same meaning and the same or similar phonologic form in different languages). Word meaning may differ between individuals because it reflects the integration of a lifetime of single experiences, commonalities of which PDP networks are singularly adept at capturing. Personal experience may have been substantially different during the times that each of two languages were spoken (see context effects, below).

All this said, on the whole, empirical studies involving lexical ambiguity resolution (Altarriba & Gianico, 2003), language switching (Costa & Santesteban, 2004), lexical decision, progressive de-masking of printed words (allowing progressively longer stimulus exposure times) (Dijkstra, Grainger, & van Heuven, 1999) and semantic priming (Kiran & Lebel, 2007) in normal bilingual subjects strongly support the idea of a common pool of semantic knowledge accessible to all languages spoken (Faroqi-Shah & Waked, 2010). Priming effects are greater for responses given in the less proficient language (Kiran & Lebel, 2007), presumably because words in this language have more to gain. Because of the intrinsically greater performance variability in participants with aphasia, such effects have been harder to demonstrate in these individuals. Siyambalapitiya, Chenery, and Copland (2013) did demonstrate semantic priming in an Italian-English bilingual with nonfluent aphasia.

4.1.2. Phonology and cognate effects

The pervasive regularities in phonological sequence knowledge tend to be language specific. However, to the extent that they are shared, they may be more robust in the face of left hemisphere lesions causing aphasia, either because of deeper representation in left hemisphere neural connectivity or greater redundant right hemisphere encoding. Many languages share extensive regularities in phonological sequence, e.g., Spanish and Italian. This is particularly true in the case of cognates. Cognates can be produced more quickly and with fewer errors than non-cognates and can prime the phonologic form in the other languages (Costa, Caramazza, & Sebastián-Gallés, 2000; Gollan & Acenas, 2004) because phonological forms of cognates engaged from the same semantic representation share extensive sequences.

Many investigators have reported evidence of cognate effects in lexical decision, word reading, translation, repetition priming, and picture naming tasks in normal bilingual participants (Colomé & Miozzo, 2010; Costa et al., 2000; de Bot, Cox, Ralston, Schaufeli, & Weltens, 1995; De Groot, Borgwaldt, Bos, & van den Eijinden, 2002; Dijkstra et al., 1999; Lalor & Kirsner, 2000; Lemhöfer & Dijkstra, 2004; Verreyt, De Letter, Hemelsoet, Santens, & Duyck, 2013), as predicted by our theory. Cognate effects are frequency dependent: their effect is greatest when the word frequency in the target language is low relative to that in the non-target language (Dijkstra et al., 1999) and they tend to be greater for the second language (Lalor & Kirsner, 2000; Lemhöfer & Dijkstra, 2004). Cognate effects in production relate to shared semantic knowledge and phonologic-sequence knowledge. In reading-and lexical decision, they relate to shared semantic knowledge and orthographic-phonologic sequence knowledge. More generally, these studies demonstrate that, in accord with the model, acoustic, orthographic, and phonological substrates in different languages are simultaneously engaged. Language output is additionally influenced by language set (see below). Response to input, e.g., lexical decision, depends upon relative cross-linguistic frequencies and neighborhood effects and will be facilitated by cognates, for which semantic representations will be elicited in both languages (Dijkstra, 2003).

It is clear enough from priming studies of normal bilingual participants that there is no direct interaction between phonological sequences in two languages that share few if any sequence regularities (Hernandez & Reyes, 2002). The data on cognate effects noted above tell us that there can be strong interaction effects between two languages on a particular word when commonality in phonemic sequence is extensive or complete. If cognates facilitate production performance by enhancing the engagement of target phonologic sequences and inhibit performance by enhancing the engagement of competing phonologic sequences, then similar, albeit perhaps weaker, facilitatory/inhibitory effects should be seen when words in two language share less extensive phonology. This has been shown in a cognitive psychological experiment in which, on average, 53% of phonemes were shared (Colomé & Miozzo, 2010) (see also Sánchez-Casas & García-Albea, 2005) ($\geq$70% is a commonly accepted empirical definition of a cognate (Roberts & Deslauriers, 1999)). Furthermore, just as cognate facilitation effects occur with fragmentary phonological sequence overlap, they can be observed with incomplete overlap of semantic features, as with semantic associates (Sánchez-Casas & García-Albea, 2005).

Limited data from aphasia provide evidence of cognate effects (Detry, Pillon, & de Partz, 2005; Lalor & Kirsner, 2001; Roberts & Deslauriers, 1999). Goral, Levy, Obler, and Cohen (2006) reported a Hebrew-English-French patient with chronic aphasia whose first and most proficient language was Hebrew. Predictably, the patient made the fewest language mixing errors when speaking Hebrew. However, when speaking English, 89% of his mixing errors were French words (only 6% involving cognates), and when speaking French, 93% of his mixing errors were English words (48% involving cognates). The extensive commonality of phonemic sequence knowledge between English and French, and the nearly complete lack of such commonalities between these languages and Hebrew, appears to offer the best explanation for what Goral and colleagues observed. More generally, the realization of the phonological sequence form corresponding to a particular semantic representation is likely to reflect the culmination of a dynamic top down/bottom up process that continues until the state of neural activation settles into a quasi-optimal semantic attractor basin and phonologic sequence trench. Consequently, in monolinguals, phonologic errors reflect both semantic and phonologic influence (Nadeau, 2001). The situation in bilinguals is the same in principle but more complex in the realization. Semantic representations in two different languages may not precisely correspond, as I have noted, and any given semantic representation lives in a neighborhood of semantic representations that share a variable number of semantic features (near and distant neighbors), each neighbor reflecting frequency and age of acquisition effects wired into neural connectivity. Phonologic sequence representations may be highly similar

(e.g., cognates), share some sub-word sequences, or be largely orthogonal. Languages that share extensive vocabulary (e.g., English and French) are likely to share many phonological sequences at the word (cognate) and sub-word levels. Very different languages, e.g., English and Hebrew, are likely to share very few phonological sequences. In the patient with bilingual aphasia, the complex top-down/bottom-up process that eventuates in phonologic sequence selection is further influenced by relative proficiency, age of acquisition effects, differential frequency effects between the two languages, and the enhancement of stochastic effects by aphasia — the probability of successful engagement of the correct phonological sequence representation or even a quasi-optimal representation is reduced. Finally, further confounding this is the potential reduction in prefrontal influence on language set that contributes to switching and mixing errors (see below).

4.1.3. Reading

Assessment of reading aloud and of verb past tense formation may be among the most straightforward means of testing relative regularity effects in two languages. I will assume a two-pathway reading model (Plaut et al., 1996; Seidenberg & McClelland, 1989) incorporating a direct orthographic-articulatory motor pathway that encodes the relationships between orthographic and phonologic sequence knowledge, and an indirect orthographic-semantic-articulatory motor pathway that also supports comprehension of read material. There is no compelling reason to doubt that the two reading pathways exist in every language. Languages with transparent (shallow) orthography (e.g., Turkish, Spanish) incorporate many regularities in orthographic-phonologic connectivity. Extent of encoded regularities declines with increasing depth of orthography (e.g., Spanish > German > French > English) (B. Weekes, 2005). For languages employing non-alphabetic scripts (e.g., Japanese, Cantonese, and Mandarin), orthographic and phonologic sequence representations are essentially orthogonal and thus, this connectivity between their substrates encodes no regularities. Semitic languages (Hebrew, Arabic) present a complication in that most vowels are featured as diacritic markers adorning a poly-consonantal root. When the diacritic markers are present, orthography is shallow. However, written material commonly does not include such markers (so-called unpointed script) — something children learn to manage early in their schooling. The absence of diacritic markers creates many homographs that must be disambiguated on the basis of frequency and semantic effects. Whether the intrinsic shallowness of the orthography of these languages survives the loss of diacritic markers is an empiric question. However, the general prediction from these considerations is that, in bilinguals with aphasia, reading aloud, particularly of word-like nonwords, will be better preserved in the language with the more transparent orthography.

4.1.4. Grammatic morphology

The production of the past tense of English verbs involves a spectrum of regularities, ranging from complete (e.g., jump → jumped) to quasi-regular (e.g., swim → swam; hit → hit), to arbitrary (e.g., go → went) (Rumelhart & McClelland, 1986). There is strong evidence of two pathways supporting verb past tense formation, one incorporating phonologic sequence knowledge and thus best equipped to encode regularities, and one a semantic route for which frequency is the dominant encoding parameter and there is no capacity for encoding sequence regularities (Nadeau, 2012). The prediction from these considerations is that, in bilinguals with aphasia, verb past tense formation will be better preserved in the language in which the sequences involved tend to be more regular.

The contribution of regularities in neural connectivity to grammatic morphology in general varies somewhat from language to language. In the context of left hemisphere lesions, this could confer advantages for certain languages. From studies of Broca's aphasia, we know that in English, with its substantially degenerate and irregular grammatic morphology, patients exhibit a strong tendency to simply omit words of substantially grammatical significance (e.g., articles, prepositions, auxiliary verbs). However, in highly inflected languages (essentially all the major languages of the world except English and Chinese), patients with Broca's aphasia uncommonly omit grammatical morphemes but are prone to morphemic paraphasias (Nadeau, 2012). Given the size of the lesions in many patients with Broca's aphasia, these observations also suggest that with highly inflected languages, there is substantial redundant knowledge of grammatic morphologic sequence in the right hemisphere. Thus, one might predict that in a patient who speaks English and a richly inflected language and who has been rendered aphasic by a left hemisphere stroke, there might be better preservation of grammatic morphology for the richly inflected language.

4.1.5. Syntax

If knowledge of word order at the sentence level (syntax) reflects knowledge of the sequencing of concepts and their modifications, as I have posited (Nadeau, 2012), then regularities in word order characteristic of one language will either synergize or compete with regularities in word order characteristic of the other languages an individual speaks. For example, there is evidence from a number of studies of bilinguals, with or without aphasia, of priming of sentence structure in one language by a similar structure in a different language (Hartsuiker & Pickering, 2008; Hartsuiker, Pickering, & Veltkamp, 2004; Verreyt, Bogaerts, et al., 2013; Weber & Indefrey, 2009). Competition may result in the inappropriate use of the grammatic form from one language while speaking another (Marian & Kaushanskaya, 2007), the direction of the transfer reflecting the relative degree of constraint provided by regularities in the two languages. There is particularly extensive evidence of syntactic priming of dative constructions (Hartsuiker & Pickering, 2008), even for completely unrelated languages (e.g., English and Korean) (Shin & Christianson, 2009). Finally, priming has been observed for sequences best considered under the rubric of phrase structure rules, the repertoire for which is likely defined substantially by morphologic sequence knowledge (Nadeau, 2012), e.g., noun-adjective order and whether adjectival modification of a noun is achieved through pre- or post-positioning of the adjective or post-positioning of a relative clause.

With left hemisphere damage, the attractor trenches of syntactic representations will effectively become shallower and more atypical/less frequent sequence representations will be attenuated or disappear entirely. In the pooled syntactic knowledge of the two languages, the least atypical/most frequent sequence representations should dominate, regardless of language spoken.

4.2. Word frequency and sequence frequency

Frequency of exposure to a given exemplar provides both the opportunity to achieve stronger connectivity in its neural substrate through Hebbian learning and the opportunity for encoding of regularities in neural network connectivity reflecting similarities between this exemplar and other, already learned, exemplars. This is true whether the exemplar is a perceptual experience that builds or modifies semantic knowledge, a phonologic sequence, a grammatic morphologic sequence, a phrase structure, or knowledge of a particular language-specific habit of concept sequencing and modification (the major basis for syntax). Greater proficiency achieved through greater experience speaking any given language will be associated with enhanced frequency effects for all linguistic forms characteristic of that language. The neural instantiation of proficiency effects provides the basis for Pitres' rule that recovery tends to be better for the language that was most familiar before brain injury (Pitres, 1895).

Two factors relevant to differential linguistic performance in both normal bilingual subjects and bilingual subjects with left brain strokes causing aphasia may be related to frequency effects: the duration of time over which a given language was or has been spoken and the time elapsed since a language was last spoken. For example, if a patient with aphasia spent a greater part of her life speaking a Romance language, the prediction would be that she would show an enhanced tendency to post-position adjectives.

4.3. Age of acquisition

Controlling for frequency, words acquired earlier in life are relatively spared in aphasia (Hinton & Shallice, 1991; Hodges, Graham, & Patterson, 1995; Rogers et al., 2004). This age of acquisition effect corresponds approximately to Ribot's rule (Ribot, 1882), that more recently acquired memories are more likely to be the lost than remote memories.

The mechanisms underlying age of acquisition effects remain uncertain. One important hypothesis, formulated by Ellis and Lambon Ralph (2000), is that the age of acquisition effect reflects the evolution in the magnitude of change in neural network connectivity that occurs with the learning of items over the course of the training lifespan of the network — that is, the evolution of its plasticity. They tested this hypothesis in an extensive series of training simulations employing a three-layer PDP network. Connection weights were initially set to small random values and adjusted during training in the direction of a maximum of $+1$ or a minimum of -1. A large set of abstract representations was fed to the input units and the network had to make a specific change in each input pattern, which was expressed as a pattern across the output units. The network learned how to make this change through a training technique known as back propagation. Back propagation involves comparison of actual network output with target output, followed by slight adjustment of network connection strengths in proportion to their contribution to the error – the discrepancy between actual and targeted output pattern. In the course of repeatedly cycling through a training set, the network asymptotically approached optimal production of the desired output patterns for the entire training set. "Early" representation sets were trained during the initial 250 epochs (cycles through the set being trained), at which point (or later), training of "late" representation sets was initiated and sustained for at least 500 epochs (in association with continued training of the "early" set). The investigators found that the accuracy of production achieved with training of late representations was always less than that achieved with early sets, no matter how long training was sustained (out to 100,000 epochs). The later the introduction of a training set, the lower the ultimate accuracy achieved. The larger the training set (the "vocabulary" of the network), the worse the ultimate performance, but disproportionately so for late sets. Late-trained sets were more vulnerable to network damage and, by inference, noise.

The authors sought to determine why this occurred. What they discovered is that the magnitude of the error measure that drove learning declined over time as the activity of units in the intermediate layer of the network evolved, through the course of training, from an average of 0.5 (on a scale of 0–1, at which point error signal was maximal at 0.25) toward 0 or 1, at which points the error signal was zero. That is, the more items previously trained, whether because of the size of the trained vocabulary or the point in time at which a given training set was introduced, the less the "neuroplasticity" of the network.

It might fairly be argued that this dynamic was an artifact of the back-propagation algorithm. This criticism is hard to address because, whereas back propagation is a simple and powerful computer algorithm for training networks in simulations, we have yet to achieve a computer-realizable algorithm that captures the complexities involved in encoding of episodic memory by the hippocampal system and subsequent consolidation of these memories, to the extent that they can be consolidated, as declarative memory in cerebral cortex. On the other hand, the fundamental insight achieved by Ellis and Lambon Ralph has found extraordinary confirmation in empirical studies of human subjects (Lambon Ralph & Ehsan, 2006). It is also congruent with the emerging concept of synaptic homeostasis (Tononi & Cirelli, 2014), which is now supported by almost innumerable experimental studies. Learning during wakefulness corresponds to increases or decreases in synaptic strengths within neural systems implicated in learning experiences. Eventually, this will lead to saturation of neural connectivity as, over time, synaptic strengths are driven to maximal or minimal values (as in the Ellis and Lambon Ralph simulations). Not only does this steadily reduce learning capacity but it also decreases the ability to selectively encode more important memories. The synaptic homeostasis hypothesis is that during wakefulness, there is, in aggregate, an overall strengthening of synaptic connectivity, while during non-REM sleep, there occurs a "normalization" of synaptic connectivity defined by comprehensive downgrading of synaptic connections strengths, constrained by a "survival of the fittest" process in which neural connectivity that is most implicated in the day's knowledge acquisition *and* implicated in existing long-term memory will be least weakened, or even strengthened, while neural connectivity that does not share these attributes will be differentially weakened. Thus, both capacity for further learning (neuroplasticity) and capacity for prioritization of knowledge to be retained are preserved. In this conceptualization, age of acquisition effects could reflect the fact that, given a sustained capacity for prioritization of memories based on reconciliation of existing memories with new experience, early acquired memories, by virtue of lifelong repeated re-engagement and by virtue of being least affected by uncompensated saturation of neural connectivity, will be at

an advantage.

Simulations of network models, reinforced by data from studies of human subjects, strongly suggest that age of acquisition effects tend to be attenuated by the development of regularities within a given knowledge domain that are wired into neural connectivity (Lambon Ralph & Ehsan, 2006). Thus, they have not been readily detectable in semantic knowledge, which is redolent with regularities (e.g., if you know about dogs, you know a great deal about mammals and vertebrates in general). In a study of Spanish-English bilinguals, age of acquisition of knowledge of objects achieved during early Spanish-only speaking years had no impact on lexical decision latency in English (Izura & Ellis, 2002), which likely depends substantially on semantic knowledge. Age of acquisition effects are detectable only in certain niches of orthographic-phonologic sequence knowledge (see below), which is also redolent with regularities, and less so in languages with highly transparent orthographies.

Age of acquisition effects are most apparent in semantic-phonologic (lexical) knowledge, which, as I have noted, is marked by very few regularities. They can also be observed in domains such as orthographic-phonologic sequence knowledge whenever mapping tends to be arbitrary. Thus, they are more prominent in the naming of English words with irregular or inconsistent spelling-sound correspondences (Monaghan & Ellis, 2002) and they are negligible in the naming of Italian words, which are characterized by very regular spelling-sound correspondences (Barca, Birani, & Arduino, 2002; Bates, Burani, D'Amico, & Barca, 2001; Colombo & Burani, 2002). On this evidence, age of acquisition effects will tend to be more apparent to the extent that there are irregularities (e.g., Hungarian and Turkish share the same elaborate grammatic morphologic scheme but Hungarian has considerably more irregularities than Turkish, which is almost completely regular (Nadeau, 2012)). Age of acquisition effects are more likely to be apparent in irregular or quasi-regular domains, e.g., in English irregular past tense marking, than in fully regular domains of a given linguistic function, e.g., English regular past tense marking (Nadeau, 2012).

The computational work of Ellis, Lambon Ralph, Ehsan, and others does not exclude the possibility that the brain, during childhood, is characterized by unique plasticity related to mechanisms so far undefined. However, empirical studies have not succeeded in establishing an inflection point reflecting the end of a maturational critical period, much less distinguish an apparent inflectional point from a non-linear age of acquisition effect (accent excepted) (Birdsong & Molis, 2001).

4.4. Context effects

To the extent that life was different during the acquisition of a first language, the large cohort of semantic knowledge acquired during that time may be less applicable than the body of knowledge gained during the time of acquisition of a second language. Exemplars that have a high frequency in the second language may have a much lower frequency in the first language, with the result that patients may perform worse when trying to use their first language on tests deriving from the linguistic ecology of the second language. Even in advanced countries, the use of certain domains of semantic knowledge, and associated lexical forms, may be differentially associated with the discussion of particular topics (e.g., professional), in which case language spoken during such discussion will exhibit its own unique regularity, frequency, and age of acquisition attributes. In testing language after brain injury, limited commonality between the test items and the vocabulary of L1 or L2, as routinely used by the individual prior to the injury, would be expected to negatively impact test performance.

The manner in which a language is learned may also have important ramifications (Kotik-Friedgut, 2001). The learning of early-acquired language normally involves extensive development of acoustic-articulatory motor connectivity underlying phonologic sequence knowledge together with the development of connectivity between substrates for semantic knowledge and phonologic sequence knowledge that enable spoken language comprehension and production. Later, orthographic-phonologic sequence knowledge, orthographic-inscriptional sequence knowledge, orthographic-semantic knowledge, and semantic–inscriptional knowledge are added to this core scaffold as reading and writing are learned. However, the learning of a late-acquired language may initially depend much more heavily on the development of orthographic-phonologic and orthographic-semantic knowledge, leaving knowledge domains involving acoustic connectivity relatively under-developed, hence more susceptible to lesion effects.

4.5. The challenge of comparing language performance

Differential recovery of one language in a bilingual with aphasia is of particular theoretical interest. However, comparing performances within a given domain in two languages may not be straightforward. For example, as Fabbro (2001) points out in a study of Friulian-Italian bilinguals, in Friulian, the subject noun phrase must obligatorily include a pronoun (e.g., “Il frut al bef” — “The boy he drinks”), whereas in Italian, a pronoun is not needed and the entire noun phrase can be omitted (“The child drinks”, “drinks”). Thus, the Friulian language provides much greater opportunity for errors in this particular domain. Deficits in grammatic morphology can scarcely be measured in Chinese, which is almost bereft of grammatical morphology, except for the highly idiosyncratic noun classifiers. Patients who speak languages with on-average longer words and/or lower biphone frequencies (lower phonotactic constraints) will be more susceptible to phonological slips in the context of damage to the substrate for phonologic sequence knowledge.

4.6. Maintenance of language set

Language breakdown, graceful degradation notwithstanding, is characterized by an increased propensity for error, which may include non-responses (e.g., anomia and morphological agrammatism), simplified responses (e.g., syntax in Broca's aphasia), or substitutions (e.g., phonemic, grammatic, and semantic paraphasias). In the bilingual person with aphasia, substitution errors may involve inappropriate use of exemplars from other known language(s). This may be characterized by the intermingling of two

languages within a single utterance, referred to as pathological mixing. However, mixing is not invariably pathologic. In bilingual communities, it may be culturally accepted and observed in everyday conversation (Fabbro, 2001). Patients may also alternate between languages across different utterances — pathological switching (M Paradis, 1977).

Our theory predicts that substitution (mixing) errors should occur most frequently in the generation of word forms of major lexical items from concept representations because the semantic-phonologic sequence pattern associator network incorporates the fewest regularities and because of the high prevalence of anomia in aphasia. The selection of a given lexical form by a bilingual person reflects competition between the two alternative phonological sequence representations (one for each language) that correspond to the concept representation, strongly influenced by a distributed representation of a frontally mediated plan or intention to use a particular language — a linguistic intentional set — that is effectuated via frontal-postcentral perisylvian connectivity. This general concept can be credited to L'Hermitte and colleagues (L'Hermitte, Hécaen, Dubois, Culioli, & Tabouret-Keller, 1966) and Stengel and Zelmanowitz (1933). It has been explicitly incorporated into a fairly successful unified computational model, employing distributed representations, that mapped word orthography to word meaning in two languages (Thomas, 1997). The physiologic mechanisms underlying the concept were demonstrated years ago in attentional systems (Moran & Desimone, 1985). Functional imaging studies have implicated left dorsolateral prefrontal cortex in language switching (Hernandez et al., 2001). The concept has been well explored in studies of lexical ambiguity resolution (Altarriba & Gianico, 2003) and language switching (Costa & Santesteban, 2004; Meuter & Allport, 1999) in normal bilingual subjects (see also review by Kroll, Bobb, Misra, & Guo, 2008). Notably, these studies demonstrate the Stroop-like effects that are to be expected when frontal systems mediate competing postcentral distributed representations of differential salience.

The mechanisms underlying prefrontal determination of linguistic set can properly be viewed as a form of selective engagement (Nadeau & Crosson, 1997). By selective engagement, I mean the bringing online of selected representations in selected neural networks, either by eliciting alterations in the pattern of neural activity, alterations in the likelihood of neural firing, or selection of inputs that will induce or modify neural firing (Desimone & Duncan, 1995; Moran & Desimone, 1985). In neurodynamical terms, this corresponds to deepening of attractor basins and attractor trenches (Rolls & Deco, 2015).

The mechanisms described in the foregoing predict two classes of error in the context of brain lesions. One class would reflect pathological inability to maintain appropriate language set, that is, pathological switching. It might be expected to occur with purely frontal lesions and could be seen in the absence of a disorder of language per se. The second class would reflect selection of the phonological sequence corresponding to the wrong language — pathological mixing. This would be expected to occur in the context of lesions damaging the substrates for semantics, phonological sequence knowledge, or the connections between the two, that is, with lesions of postcentral perisylvian cortex. Such lesions would also be expected to damage frontal-postcentral perisylvian connections that maintain language set. In such circumstances, there will be a still higher probability of selection of the phonological sequence from the wrong language. That is, damage to frontal-posterior connectivity that maintains language set could, in the context of perisylvian damage, lead to worsening of aphasia. It is even possible that two or more phonological sequences, each corresponding to a different language, might be brought above threshold for word expression, leading to cross-linguistic blends (Marién, Abutalebi, Engelborghs, & De Deyn, 2005) in direct analogy to the generation of blends that have been observed in single languages (Nadeau, 2001). Although production of the word forms of major lexical items should be most susceptible to language mixing effects, in principle, any domain of language function could be affected.

4.7. Aphasia rehabilitation

Our model speaks strongly to the subject of aphasia rehabilitation. However, there are two issues that confound interpretation of the aphasia treatment literature (monolingual and bilingual): 1) the potential conflation of treatment effects with the results of spontaneous recovery; and 2) insufficiently precise understanding of mechanisms of generalization, and in particular, what types of therapy might intrinsically generalize and what types likely engage other, nonlinguistic mechanisms that could promote generalization. The means for avoiding conflation of treatment and spontaneous recovery effects are now fairly well defined but are worth brief discussion (Section 4.7.1). Principles of generalization are not so well defined and remain a topic of very active research. Section 4.7.2 provides a summary that covers both mechanisms of intrinsic generalization, which derive directly from the model, and nonlinguistic mechanisms, which should be viewed as adjuncts to the model.

4.7.1. Timing

The rapid improvement in language function in the months immediately following stroke has led to the widely held belief that this early period represents a time of particular opportunity for treatment. However, spontaneous recovery during this early period reflects a host of reactive neuroplastic mechanisms (reactive neuroplasticity) (Nadeau, 2015); there is no scientific reason to think that experience-dependent neuroplastic mechanisms (which are engaged in aphasia treatment) interact with reactive neuroplastic mechanisms; and there is no evidence from clinical or preclinical studies that early treatment yields superior long-term outcomes, even as outcomes of early treatment risk being attributed to therapy rather than to reactive neuroplasticity. Linguistic function, however measured, more or less doubles in the first six months after stroke and there is some evidence of continued spontaneous recovery during the second six months and beyond (Nadeau, 2015). For these reasons, treatment studies initiated within one year of stroke will not be considered unless stability of baseline has been demonstrated for a period at least as long as the time elapsed between initiation of treatment and final outcome assessment. In general, during the first year, the conflation of effects of reactive and experience-dependent neuroplastic changes can best be definitively addressed through large randomized, controlled, parallel group studies. In bilingual aphasia, experience dependent neuroplasticity could be leveraged to achieve gains in one language and,

absent crosslinguistic generalization, the other language could serve as the control. For example, it is well-established that naming therapy is effective and that treatment results are sustained, even as the treatment does not generalize to untrained items in the treated language (Wisenburn & Mahoney, 2009), let alone in the untreated language (see below: generalization between languages).

4.7.2. Generalization between languages

The effects of treatment of one language in a patient with aphasia will generalize to a second, untreated language to the extent that there are regularities in knowledge encoded in neural networks that are common to both. This mechanism is most transparent for semantic knowledge, for which regularities will be the same for both languages because our knowledge of the world is largely independent of language (barring significant context effects and interlingual differences in the dimensionality and scope of many languages (Majid et al., 2008; Marian & Kaushanskaya, 2007)). However, there may be shared regularities in other domains, most notably phonologic sequence and syntax, as discussed above in the section on regularities encoded in neural network connectivity (Section 4.1).

Anomia is both the most common and the most disabling component of aphasia. It may stem from semantic impairment, semantic-phonological (lexical) impairment (disconnection of the substrates for semantic knowledge and phonologic sequence knowledge), or loss of phonologic sequence knowledge. Because semantic knowledge and phonologic sequence knowledge are substantially orthogonal, there are few in the way of regularities in this domain and little potential for generalization of training even within a given language, let alone between two different languages. However, theoretically, treatment of either semantic knowledge or phonological sequence knowledge (both intrinsically generalizing treatments) can improve object naming, and we now have empiric support for this hypothesis (Edmonds, Mammino, & Ojeda, 2014; Edmonds, Nadeau, & Kiran, 2009; Kendall et al., 2008; Kendall, Oelke, Brookshire, & Nadeau, 2015). Thus, to the extent that treatments of anomia enhance semantic or phonological sequence knowledge, there is the potential for generalization of treatment of one language to another, untreated language. This is particularly the case with semantic treatment because semantic knowledge is likely to be extensively shared across languages, but to some extent, it will also be the case with phonologic treatment to the extent that there are shared phonological sequences, and even more so, cognates. Semantic training of cognates in one language provides an opportunity for promoting Hebbian learning of semantic-phonologic (lexical) connections in the other language because the semantic representation (common to the two languages) and the phonological representation in the untrained language (which, in the case of cognates, shares phonological sequence features with that of the trained language) will be simultaneously engaged.

Treatments of aphasia that engage non-linguistic mechanisms would also be expected to generalize from one language to another. These have been discussed in detail elsewhere (Nadeau, 2015; Nadeau & Kendall, 2006; Nadeau, Rothi, & Rosenbek, 2008), but may be briefly summarized:

Extrinsic. Development during therapy of a knowledge acquisition/skill learning technique that patients with motivation, and capable of engaging motivation to employ the technique, can use during and outside of therapy to rebuild language function (e.g., semantic therapy, phonological sequence therapy, syntactic therapy).

Mechanistic. Training of a key brain resource, essential to language processing but not fundamentally linguistic, that enables improvement in language function. Two subtypes can be identified:

A. Development of working memory capacity needed for language.
B. Development of a new intentional bias that favors language use over either nonuse or gestural communication, the cardinal example being constraint induced language therapy (CILT) — or, as its originators now prefer to call it, intensive language action therapy (ILAT) (Difrancesco, Pulvermüller, & Bohr, 2012; Faroqi-Shah & Virion, 2009; Maher et al., 2003; Meinzer, Djundja, Barthel, Elbert, & Rockstroh, 2005; Meinzer, Streiftau, & Rockstroh, 2007; Pulvermüller et al., 2001).

Socially mediated. Change in the perception of the subject and her family regarding her role in the family unit, with the adoption of a new/revised role, i.e., a major change in the ambient therapeutic environment, that subsumes more expectation of speech, more pressure to speak, and greater language production (Blonder, 2000).

5. Bilingual aphasia — empirical tests of the model

In this section, I will take the hypotheses developed in Section 4 and attempt to apply them to the results of empirical studies of aphasia in bilingual and multilingual patients with focal brain injury due to stroke, trauma, or resection of tumors, or more diffuse and progressive lesions associated with dementia. A model, however elegant and neutrally plausible, is of little value if it cannot provide an orderly account for behavioral observations. As I noted earlier, one of the cardinal attributes of population encoding models is graceful degradation. Because knowledge is represented in connectivity (synapses) throughout networks, neither focal nor diffuse lesions eliminate that knowledge. Rather, they reduce the reliability of network output. There may be unreliability in achieving the threshold for production of responses, hence, for example, the prevalence of anomia in aphasia. Lesions increase the probability of near-miss errors, hence semantic, phonologic, and grammatic paraphasias. Lesions differentially degrade knowledge that was not deeply encoded in the first place, hence frequency and age of acquisition effects on performance and the tendency to greater use of more typical forms, e.g., subject-verb-object order in patients with Broca's aphasia, particularly English speakers. Because graceful degradation yields orderly patterns of knowledge degradation, cognitive neuropsychological studies of patients with aphasia has been able to elucidate language structure and function in the normal brain with extraordinary granularity.

I have sought to maintain as much parallelism as possible between this section and Section 4. In the first subsection (5.1), patterns

of recovery, the evidence bearing on the issue of parallel versus divergent change in the recovery of two or more languages after stroke or decline with dementia is reviewed. In section 5.2, I focus on the major theme of Section 4.1, regularity effects. Word frequency and sequence frequency are obviously linked to how much one speaks a language. However, although much studied in normal individuals, they have not received much explicit attention in the bilingual aphasia literature so there is no counterpart in this section to Section 4.2. Section 5.3, age of acquisition effects, corresponds to Section 4.3. Context effects (Section 4.4) and challenges of comparing language performance because of idiosyncrasies of language structure (Section 4.5) have received only glancing attention in the bilingual aphasia literature and therefore there are no corresponding subsections on these topics in Section 5. Section 5.4, maintenance of language set, finds its direct counterpart in Section 6.6. Section 5.5, aphasia treatment, has its counterpart in section 4.7.

5.1. Patterns of recovery from aphasia or deterioration with dementia and factors influencing them: parallel versus divergent change

As predicted by the model, nearly all recent studies have reported parallel recovery of language in bilingual patients following stroke, *allowing for the expected effects of proficiency (pre-stroke strength of encoding related to regularities and frequency effects) and age of acquisition* (Abuom, Shah, & Bastiaanse, 2013; Amberber, 2012; Croft, Marshall, Pring, & Hardwick, 2011; Dai, Kong, & Weekes, 2012; Diéguez-Vide, Gich-Fulla, Puig-Alcántara, Sánchez-Benavides, & Peña-Casanova, 2012; Fabbro & Paradis, 1995a, 1995b; Fabbro, Peru, & Skrap, 1997; Galloway, 1978; Gil & Goral, 2004; Goral et al., 2006; Green et al., 2010; Green et al., 2011; Hinckley, 2003; Junqué, Vendrell, & Vendrell, 1995; Junqué, Vendrell, & Vendrell-Brucey, 1989; Kambanaros, 2010; Kambanaros & Grohmann, 2011, 2012; Kambanaros & van Steenbrugge, 2006; D.; Kendall et al., 2015; Kiran & Lebel, 2007; M. I. K.; Knoph, 2011; Kremin & De Agostini, 1995; Langanaro & Venet, 2001; Marangolo, Rizzi, Peran, Piras, & Sabatini, 2009; McCann, Lee, Purdy, & Paulin, 2012; Miozzo et al., 2010; Muñoz & Marquardt, 2003; Nilipour & Ashayeri, 1989; Roberts & Le Dorze, 1998; Sasanuma & Park, 1995; Sebastian et al., 2012; Siyambalapitiya et al., 2013; Stadie, Springer, De Bleser, & Bürk, 1995; Voinescu, Vish, Sirian, & Maretsis, 1977; Watamori & Sasanuma, 1976; B. S.; Weekes & Raman, 2008). These findings are entirely in accord with the views of Freud in 1891 (Freud, 1953) and Pitres in 1895 (Pitres, 1895).

Dementia constitutes a fundamentally different lesion model, one characterized by insidious progression rather than abrupt decline followed by some degree of recovery, as in stroke. In fact, data exist for two very different types of dementia, Alzheimer's disease and primary progressive aphasia. However, the predictions of the population encoding model detailed early in this paper should apply, regardless of the pathology and the pattern of its development. Studies of dementia therefore provide another way of testing the model. Language function consistently declines in parallel in both languages in patients with Alzheimer's disease (Costa et al., 2012; Salvatierra, Rosselli, Acevedo, & Duara, 2007; Gollan, Salmon, Montoya, & da Pena, 2010) or primary progressive aphasia (Druks & Weekes, 2013; Filley et al., 2006; Hernández et al., 2008; Mendez, Sachafi, & Clark, 2004; Zanini, Angeli, & Tavano, 2011). Costa et al., 2012 found that performance was slightly worse in the self-reported non-dominant language, consistent with the greater susceptibility of less redundantly encoded knowledge to the dementing process, which is associated with widespread loss of synapses.

In summary, the evidence is overwhelming that parallel decline and, in the case of acute injury, subsequent parallel recovery, is the rule. The result is that patients consistently demonstrate better language function when using the language that pre-morbidly was characterized by greater proficiency and earlier age of acquisition. A number of factors may account for apparent divergence from parallel recovery. Performance may vary considerably between testing sessions and test-retest reliability is seldom taken into account (Roberts & Le Dorze, 1998). Furthermore, estimates of proficiency are based largely on self-report, which is not always accurate (Gollan, Weissberger, Runnqvist, Montoya, & Cera, 2012; Kohnert, Hernandez, & Bates, 1998). Parts A and B of the BAT include many questions bearing on language acquisition and speaking history but recent efforts to refine self-report measures and to obtain multiple measures in each participant, enabling detection of discrepancies and assessment of correlations, are noteworthy (Gray & Kiran, 2013; Sebastian et al., 2012). Interpretation of comparative performance in two different languages in relationship to premorbid language use and proficiency may be aided by norms derived from control bilingual populations (Muñoz & Marquardt, 2003). Finally, language acquisition histories are often complex, making it difficult to determine likely age of acquisition effects.

5.2. Regularity effects

The study results that predominantly reflect proficiency effects usually conflate frequency and regularity effects. As noted previously, assessment of reading aloud and verb past tense formation may provide relatively ideal tests of regularity effects. The cardinal hypothesis to be tested is that in bilingual patients with aphasia, performance will be superior in the language in which more regularities are incorporated within the knowledge in a particular domain.

Béland and Mimouni (2001) conducted an extensive psycholinguistic study that included probes of reading aloud by an Arabic-French bilingual with deep dyslexia. In word reading, the proportion of phonological errors in French, 17.36%, was far higher than in Arabic with (7.58%) or without (5.9%) diacritic markers ($p < .0001$). The proportion of phonological/visual errors in nonword reading in French (66.65%) was also far higher than in Arabic with (27.27%) or without (34.76%) diacritic markers ($p < .0002$). Thus, the results of this study provide suggestive evidence that, in bilingual aphasia, reading aloud in the language with more extensive regularities (shallow orthography—Arabic) is less affected. They could also reflect the effects of proficiency and age of acquisition favoring Arabic.

Druks, Aydelott, Genethliou, Jacobs, and Weekes (2012) reported a Hungarian-English bilingual patient who developed nonfluent primary progressive aphasia. English had been his dominant language (even though L1 was Hungarian). His lexical access was better

in Hungarian, suggesting an age of acquisition effect. However, there was also evidence of better reading in Hungarian. Because of the extent of regularities in neural networks supporting reading, age of acquisition effects should be largely extinguished in this network. On the other hand, Hungarian has transparent orthography with few irregularities in spelling to sound correspondences, whereas English spelling to sound correspondences are rife with irregularities (Plaut et al., 1996; Seidenberg & McClelland, 1989). Thus, the findings of this study are also consistent with the hypothesis that performance in a given language domain will be relatively preserved in the language with greater regularities.

Verb past tense formation has been extensively studied by Abuom and colleagues in English + Swahili patients (Abuom & Bastiaanse, 2012; Abuom, Obler, & Bastiaanse, 2011). In Kenya, the two languages are learned in parallel during early language development and throughout schooling and both are used extensively in daily life. Thus, in general, there is no basis for frequency and age of acquisition effects. In their first study (Abuom et al., 2011), involving two English + Swahili patients with agrammatic aphasia, the investigators employed a close test to prompt for future or past tense forms. Participants performed normally in Swahili. However, while performance with the English future tense (signaled by the auxiliary "will") was at ceiling, production of English past tense forms was severe impaired. In a second study (Abuom & Bastiaanse, 2012), the investigators reported an analysis of samples of spontaneous language in six English + Swahili speakers with chronic agrammatic aphasia. The performance of these six participants in English and Swahili was quite comparable on nearly all language measures. However, during spontaneous language, there was far less use of tense or agreement inflected verb forms in English (10% versus 26% in controls). Again, the most noteworthy interlinguistic difference was in tense marking. In English, tense marking by patients with aphasia was 34% that in controls, whereas in Swahili, it was 66%. In a subsequent study of 13 English + Swahili speakers with agrammatic aphasia, employing the Test for Assessing Reference to Time, which includes a test of past, present continuous, and future tense comprehension, and a close test assessing production of these forms, the authors again found that participants were significantly more impaired in English past-tense processing (Abuom & Bastiaanse, 2013). Two basic neural principles can be invoked to account for these observations. First, while the English past tense is regular for most verbs, a variety of quasi-regular or irregular forms are employed for the approximately 160 verbs that are the most commonly used (hence competing regularity and frequency effects) (Nadeau, 2012). In contrast, Swahili employs only one entirely regular past tense form (an infix). Second, Swahili is a very richly inflected language in which verbs are marked with prefixes, infixes, and suffixes denoting person (subject and object), tense, aspect, mood, and negation. An extensive body of crosslinguistic aphasia data strongly suggests that in richly inflected languages, there is greater bilaterality of grammatic morphologic sequence knowledge, hence a far greater tendency for patients with agrammatism to make grammatic morphologic substitutions (reflecting inadequacy of right hemisphere knowledge stores), rather than omissions (as in English) (Nadeau, 2012). Thus, both knowledge regularity effects and likely degree of bilaterality of knowledge favor preservation of past tense forms in Swahili.

There is some evidence of regularity effects in derivational morphology. Knoph reported a Farsi-Norwegian bilingual patient who demonstrated roughly comparable performance in the two languages on the BAT (M. I. K. Knoph, 2011). However, on the derivational morphology subtest (turning nouns into adjectives), his accuracy in Farsi (80%) was twice that in Norwegian (40%). Derivational morphology is considerably more regular in Farsi than in Norwegian.

5.3. Age of acquisition effects

Druks and Weekes (2013) reported a compelling case suggesting age of acquisition effects. Their patient, a Hungarian-English bilingual, first began to acquire English at age 14 but ultimately, by age 66, English likely became his language of greatest proficiency. He presented with a 5-year history of progressive language decline and met diagnostic criteria for nonfluent primary progressive aphasia. Despite scant use of Hungarian over the course of 52 years, he scored 50% correct on the Hungarian version of the BNT and 15% correct on the English version. On the Object and Action Naming Test (Druks & Masterson, 2000), he correctly named 91% of objects and 74% of actions in the Hungarian version, but only 44% of objects and 36% of actions on the English version. On the other hand, on two tests of syntactic comprehension, one developed by the investigators, the other the Test for Reception of Grammar (TROG; Bishop, 1989), his performances in Hungarian and English were identical. Because syntax is redolent with regularities, one would expect age of acquisition effects to eventually be extinguished. This is exactly what Druks and Weekes found.

Diéguez-Vide et al. (2012) reported a Mandarin-Spanish-Catalan-English speaking patient who started learning Spanish at age nine and Catalan at age 16. Family members rated his proficiency in Mandarin as excellent and in Catalan, good. The patient felt he was equally proficient in the two languages before the stroke. Nevertheless, his performance on the BAT in Mandarin was nearly normal, whereas it was significantly impaired in Catalan, most strikingly so for lexical access. One cannot rule out proficiency effects but this report appears to make a strong case for age of acquisition effects.

5.4. Maintenance of language set

As predicted by the model, language mixing has been well described in polyglots with aphasia; it is most often observed in the generation of word forms but may be seen in phonological, morphological and syntactic domains; and it may be associated with the generation of cross-linguistic phonologic or other blends (Abutalebi, Miozzo, & Cappa, 2000; Fabbro, 1999; Marién et al., 2005; Perecman, 1984). Mixing errors involving lexical forms are typically associated with temporo-parietal lesions (Fabbro, 1999). Cases exhibiting both mixing and switching have been reported but isolated pathological switching, a less common phenomenon, has been reported in a bilingual patient with a malignant glioma of the left frontal lobe in the absence of impairment of language per se (Fabbro, Skrap, & Aglioti, 2000), as also predicted by the model and consistent with the evidence that maintenance of set in language depends on frontal mechanisms that are not necessarily specific to language (Meuter & Allport, 1999).

Other cases, almost certainly reflecting pathological switching, have been reported, often as evidence of selective aphasia, differential recovery of one language, alternating antagonism, or "seesaw" (periodic alternation in degree of language impairment in different languages) (Fabbro, 1999; Nilipour & Ashayeri, 1989; M.; Paradis & Goldblum, 1989; M; Paradis, Goldblum, & Abidi, 1982). In some cases, e.g., Paradis et al. (1982), the switching from one language to the other and back again was sufficiently rapid to raise the possibility of influence of language set by intercurrent seizure activity. More generally, however, the mechanisms underlying oscillatory pathologic switching remain poorly understood.

There is an important distinction to be made between the case of Fabbro et al. (2000) and other reports best interpreted as evidence of pathological switching: the patient reported by Fabbro and colleagues, who had a left frontal glioblastoma, exhibited diminished ability to maintain language set appropriate to his surroundings but did not exhibit aphasic errors in either language. In other cases, however, the use of the language that was set-incongruent was associated with an at times dramatic increase in aphasic errors. It seems most likely that patient choice of a pathological set will be associated with an increase in aphasic errors only when there is postcentral hemispheric damage implicating language networks. This is because choice of the correct phonological representation depends on the integrity of semantic knowledge, phonologic sequence knowledge, connections between the substrates for semantics and phonology, and frontal input to these postcentral substrates. Impairment of frontal input will potentiate errors borne of postcentral damage. Put differently, successful treatment aimed at increasing intention to speak a particular language might also result in reduction of aphasic errors by virtue of sharpening of distributed representations (deepening of postcentral attractor basins) of various language elements (see Rolls & Deco, 2015).

5.5. Aphasia treatment

My focus in this section will be on studies of treatments that either have the potential for generalization (impacting performance with untrained exemplars and/or improving daily verbal communication) or have been demonstrated to generalize. We currently have a very large number of published studies that have demonstrated that speech-language therapy can improve performance with trained items, often dramatically (Wisenburn & Mahoney, 2009). However, only generalizing treatments can improve the verbal communicative lives of patients with aphasia. In this review of treatment studies, both the choice of studies for inclusion and interpretation of results were influenced by the plausibility of generalization given known mechanisms of generalization (Nadeau, 2015), the magnitude and consistency of treatments effects, and population statistics (when they were available). Studies in which treatment impacted only trained exemplars, that is, in which there was no evidence of within or cross-linguistic generalization, were excluded. Treatment studies initiated before six months post-stroke have been categorically excluded because treatment effects are inevitably conflated with spontaneous recovery during this time period (Nadeau, 2015).

5.5.1. Language mixing and switching

Ansaldo, Saidi, and Ruiz (2010) reported successful treatment of a Spanish-English bilingual patient with post stroke aphasia who was plagued by language mixing errors. They trained the patient in Spanish to gradually internalize a strategy of explicit translation as he was about to produce a word in the non-target language. Mixing errors, which had comprised 80% of his naming errors in Spanish, completely disappeared and were not replaced by anomic or other errors — a marked improvement in Spanish lexical access. Treatment effects generalized to English, but unfortunately, at a cost of a corresponding large increase in anomic errors. Because English had become this patient's everyday language, improvement in Spanish came at considerable cost. Nonetheless, this study is proof of concept that a pathological propensity for mixing errors is susceptible to treatment.

5.5.2. Generalization

A number of different mechanisms of generalization have been identified (Nadeau, 2015) and were briefly discussed in Section 4.7.2. Of these, our model bears only on intrinsic generalization, the topic of the next section. However, mechanisms of generalization are frequently ill-defined in the aphasia treatment literature. When generalization is observed in a particular study, there is typically little consideration given to whether it was related to spontaneous recovery, the development of intentionality to speak verbally (the CILT effect), intrinsic generalization, or generalization related to some other potential mechanism. Thus, in reviewing the bilingual aphasia treatment literature, it is essential to take a broad view, one not strictly limited to implications of the model, in order to tease apart the sources of generalization that might have accounted for the results, particularly when a more superficial assessment might suggest that the findings of a study are at odds with the model.

5.5.2.1. Intrinsic generalization. To the extent that different languages share knowledge within particular domains, semantics being the best example, treatment of that domain in one language has the potential for generalization to the other, untreated language.

Croft et al. (2011) reported the results of naming therapy coupled with adjuvant semantic or phonologic therapy in five Bengali-English bilingual participants with left hemisphere strokes. They found that performance on trained items improved in the treated language, regardless of which language was treated, with no differential effect for L1 or L2. There was no significant generalization to untreated items. Crosslinguistic generalization was minimal and, to the extent that it occurred, it was with semantic therapy and tended to occur with treatment of L1. The occurrence of crosslinguistic generalization uniquely after therapy with a semantic component is consistent with the thesis that training in either language can enhance neural connectivity in the substrate for semantics, which would in turn benefit both languages. Crosslinguistic generalization would not have been expected to occur with phonological treatment because Bengali and English have little phonologic sequence knowledge in common.

Kiran and her colleagues (Edmonds & Kiran, 2006; Kiran & Iakupova, 2011; Kiran & Roberts, 2010; Kiran, Sandberg, Gray,

Ascenso, & Kester, 2013) have led the field in the study of within and cross-linguistic generalization in the course of naming therapy with adjuvant semantic feature analysis. Their detailed report of treatment outcomes in 17 Spanish-English bilingual participants with aphasia due to acute brain injury (stroke in all but one) provides the clearest and most comprehensive data available on treatment effects (direct, within language generalization, and cross-linguistic generalization) involving a treatment with a potential for intrinsic generalization (Kiran et al., 2013). Eight participants were trained in Spanish, nine in English. Two participants exhibited no treatment effects whatever, and one showed a significant effect size for the trained word set but nothing else. Of the 14 participants who showed some generalization effect, ten exhibited evidence of generalization to untrained items in the trained language; five to untrained translations of items trained (e.g., Spanish translations of English-trained items); and six to items in the untrained language that were semantically related to items in the trained language. Two participants showed generalization only to translations in the untrained language, suggesting that training succeeded in strengthening the neural substrate for semantic representations of the trained items but did not succeed in enhancing performance with untrained items that shared semantic features. Taken together, the results of these studies provide strong support for the concept that training a domain of language function shared by two languages (semantics) and characterized by an intrinsic potential for generalization to untrained exemplars that share features, can indeed achieve within and between language generalization effects. Although these effects were not observed in all participants, expectably so given variability in patient backgrounds, circumstances, lesion locus and aphasia severity, as well as the vicissitudes of aphasia therapy, they are nonetheless compelling.

As discussed above, one would expect crosslinguistic generalization of treatment to be strongest for cognates, words of the same meaning and substantially similar phonology. This has been demonstrated (Goral, Rosas, Conner, Maul, & Obler, 2012).

5.5.2.2. Extrinsic. Goral, Levy, and Kastle (2010) treated a Hebrew-English-French physicist, director of a computer animation company, who had mild chronic aphasia (grammatic impairment plus anomia) stemming from a left hemisphere stroke, using a CILT-like paradigm. The treatment, conducted in English only, consisted of nine hours focused on grammatic morphologic impairment and nine hours focused on improving speech rate through better approaches to anomia. Following treatment, there was evidence of improvement in noun-verb agreement in English and in use of prepositions and tense consistency in French. Grammatic function in Hebrew was at ceiling levels at baseline. Given the completely different domains of knowledge underlying noun-verb agreement, preposition use, and tense consistency and the paucity of shared regularities between English and French in these domains, it is difficult to argue for *intrinsic* generalization. However, one can speculate as to whether the intense focus on grammatic morphology could have achieved generalization via an *extrinsic* effect — providing this brilliant and highly motivated patient a technique that he could use outside therapy to improve grammatic morphologic function.

5.5.2.3. Mechanistic. The results of some multi-lingual aphasia treatment studies seem to be best interpreted as reflecting the successful engagement of a fundamentally non-linguistic mechanism, as in CILT, e.g. Miertsch, Meisel, and Isel (2009). That is, formal treatment and/or ambient treatment (McClung, Rothi, & Nadeau, 2010) led to development of a new intentional bias — a language set — that favored increased use of language in general or of non-preferred language(s). The neural mechanisms engaged by such therapy are likely to be precisely those involved in bilingual language impairment marked by switching. The best marker of this effect may be evidence of marked efficacy of a modest dose of an intrinsically non-generalizing treatment, such as one directed at the lexicon. Goral et al. (2012) contrasted the impact of speech-language therapy, first in Spanish (language of greatest proficiency) and subsequently in English (one of three languages, including French and German, in which the participant was less proficient) in a patient with a chronic left hemisphere stroke. The patient received five years of speech language therapy in Spanish and Catalan (he was a native of Barcelona), during which time he lived in Spain. He subsequently underwent naming plus adjuvant semantic feature analysis therapy delivered in English while visiting New York. Treatment in Spanish had little effect on performance in any language on BAT subtests tapping semantic-phonologic (lexical) function. Treatment in English improved object-action naming of trained items in English but not of untrained items or any items in the other languages. However, in a task in which the patient was instructed to tell a short story on the basis of six wordless line drawings from the BAT, he demonstrated a dramatic increase in number of clauses and words generated in English after English therapy. The results of this study fail to provide evidence of intrinsic generalization but they do demonstrate an increase in propensity for using a language of lesser proficiency in which both therapeutic and ambient environments may have conduced to this effect. Goral et al. (Goral, Naghibolhosseini, & Conner, 2013) reported a similar phenomenon using an explicit version of CILT of Broca's aphasia in a Persian-English bilingual for whom English, after 13 years of absence from Iran, had become her most proficient language. Treatment in English had a very modest impact on English language production in action naming, picture description and a self-narrative task. However, treatment in Farsi had a fairly dramatic effect on Farsi production on these tasks. These results suggest that intentionality to use English, which was essentially the patient's only language for 13 years, could not be enhanced, even with 35 h of treatment in English. However, intentionality to speak Farsi could be significantly enhanced with 35 h of treatment in Farsi.

Keane and Kiran (2015) reported the effects of therapy employing semantic feature analysis, first in French, subsequently in English, in an Amharic-English-French patient with chronic aphasia following an extensive surgical resection of the left frontal and temporal lobes for treatment of an oligoastrocytoma. Treatment in both languages improved performance with trained items but no within-language or crosslinguistic generalization to translations or to semantically related items. Treatment in one language led to a substantial increase in mixing errors in the untreated language. The absence of semantic generalization suggests that the major cause of the patient's severe anomia was either disconnection of the substrates for semantics and phonology or damage to the substrate for phonology. The marked increase in mixing errors in the untreated language suggests that the treatment did impact intentionality to speak, favoring the language being treated. As discussed in Section 4, an object to be named would elicit sub-threshold phonologic

sequence representations in both languages. Input from prefrontal cortex, instantiating intention to speak a given language, would then favor the generation of the phonological sequence representation of that language.

Meinzer, Obleser, Flaisch, Eulitz, and Rockstroh (2007) used traditional CILT with a German-French patient with chronic aphasia marked by anomia and grammatic impairment. The patient demonstrated substantial improvement on untrained material but only in the language treated — German. This study suggests that improvement of intention to speak is language treatment specific. However, Altman, Goral, and Levy (2012) employed CILT focused on discourse production conducted exclusively in English with a Hebrew-English-French trilingual who had a mild nonfluent aphasia with some impairment in grammar (the physicist described in Section 5.5.2.2). They noted generalization of treatment effects to all three languages. In a comparable study of a Dutch-English-French-German-Italian-Spanish-Norwegian polyglot one year after left brain stroke, Conner et al. (2018) conducted 40 h of treatment in his first language, Dutch, focused on discourse but not explicitly defined as CILT. They observed improvement on discourse measures for Dutch and his four other languages of highest proficiency. The reason for the different results obtained in these three studies is not certain but it is worth noting that Altman et al. and Conner et al. focused strongly on narrative development, hence generation and manipulation of concepts, processes that, according to our model, should be language nonspecific, except to the extent that they are influenced by sentence level sequence knowledge, i.e., syntactic knowledge. Of course, particularly in the patient of Conner et al., who had transcortical motor aphasia, an effect on intentionality to speak must also be considered.

M. I. N. Knoph, Simonsen, and Lind (2017) employed both CILT and semantic feature analysis (20–25 h each) in two patients with chronic aphasia after stroke, one a Portuguese-Ronga-Norwegian trilingual and the other an English-Norwegian bilingual. Ronga is a Bantu language. Treatment was in Norwegian, the weaker language. Outcomes were assessed only in the European languages. The CILT focused on production of complete sentences and selection of an acceptable verb in response to pictures. The semantic feature analysis focused on verbs and trained argument structure and semantic roles, including agent/experiencer, theme/patient, purpose, locations, instrument and association. Semantic feature analysis training but not CILT produced an improvement in naming of trained verbs that was sustained over follow-up in one participant but neither treatment generalized to untrained verbs in either language. On a discourse measure derived from a narrative about a movie, book, happy moment, or vacation, obtained after completion of both treatments, the participants demonstrated an increase in verb types and tokens in both languages following treatment of one. The number of correct information units also improved in both participants in both languages post-treatment. Maintenance of these various gains at long-term follow-up was inconsistent. The complexity of the treatment program precludes confident conclusions as to the mechanism of the treatment effect. Clearly the improved production of correct information units in both languages after training limited to Norwegian could reflect an intentionality effect. However, the increase in verb types and tokens suggests that something additional may have been going on. Verbs prime commonly associated nouns, whether agents or patients (Ferretti, McRae, & Hatherell, 2001), and nouns prime commonly associated verbs (McRae, Hare, Elman, & Ferretti, 2005). Thus, verb arguments provide support for the production of verbs and vice versa. Therapy focused on verbs provides practice in the modification of argument representations implicit in their association with the verb representation in a way that is congruent with the perceptual stimulus or the narrative stream. All of this occurs at the concept level and is therefore language nonspecific. The therapies used in this study, whether alone or in combination, could plausibly have improved the participant's concept manipulation abilities, hence their ability to produce correct information units. This idea derives support from the success of verb-centered therapy for monolingual participants (Edmonds et al., 2014).

There may be a variety of ways that intentionality to speak may be promoted. Sebastian et al. (2012) reported their experience with a 32-year old Marathi-English patient who had experienced severe left-brain damage 16 years earlier in a train accident. The history provided suggests that, pre-morbidly, the patient was most proficient in speaking Marathi but more proficient in reading and writing in English. He presented with agrammatic aphasia in which he was scarcely able to speak at all in English but was less impaired in reading and writing English than Marathi — that is, parallel impairment that reflected the balance of age of acquisition and proficiency effects in different language domains. In the course of 14 h of a complex, bilingual, pragmatic therapy tailored to the patient that included melodic intonation therapy (MIT), the patient demonstrated clinically meaningful improvement in all language domains in both languages. However, his language remained agrammatic and his balance of skills — better spoken Marathi and better reading and writing in English — remained the same. The therapy program was relatively brief and did not focus on intrinsically generalizing treatments, hence my conclusion that the treatment effect was mechanistic in origin. The possibility that MIT might usefully be included in treatments like CILT that promote intentionality to speak warrants further consideration.

6. Conclusions

As this analytic review of the literature has shown, approaching the problem of bilingual aphasia from the powerful perspective of population encoding neural networks provides a substantially coherent account for experimental observations. The major weaknesses of the analysis reflect the fact that, to a substantial degree, the highly granular predictions of a population encoding model have not been adequately tested by the often broad-brush observations of patients with aphasia.

The primary appeal of the population encoding perspective lies in the fact that it is neurally plausible and provides a direct mechanistic link between neural populations and cognitive function. Representations in the brain *are* highly distributed. Population encoding neural networks also contribute powerful processing advantages to the functions they support (e.g., content addressable memory, the ability to learn from experience, and graceful degradation). Finally, they impose important constraints on the functions they support, e.g., the enormous power of encoded regularities acquired during learning experience and the attenuation of age of acquisition effects by encoded regularities. Careful consideration of the ramifications of these constraints enables a highly granular understanding of language function and language breakdown in monolingual and bilingual patients with aphasia (Nadeau, 2012).

Understanding the brain as a machine that provides the neural basis for population encoding also directly addresses one of the questions that has most pre-occupied investigators of bilingual aphasia — whether different languages are represented in different regions of the brain. Because what is encoded in neural connectivity in any given region of the brain reflects input to the region from connected regions, the population encoding perspective makes clear that the differences in encoding different languages are matters of how, not where. No empirical data have challenged this most fundamental principle.

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References

Abuom, T. O., & Bastiaanse, R. (2012). Characteristics of Swahili-English bilingual agrammatic spontaneous speech and the consequences for understanding agrammatic aphasia. *Journal of Neurolinguistics, 25*, 276–293.
Abuom, T. O., & Bastiaanse, R. (2013). Production and comprehension of reference of time in Swahili-English bilingual speakers. *Aphasiology, 27*, 157–177.
Abuom, T. O., Obler, L. K., & Bastiaanse, R. (2011). Using Swahili and English to test explanations for agrammatism. *Aphasiology, 25*, 559–575.
Abuom, T. O., Shah, E., & Bastiaanse, R. (2013). Sentence comprehension in Swahili-English bilingual agrammatic speakers. *Clinical Linguistics and Phonetics, 27*, 355–370.
Abutalebi, J. (2008). Neural processing of second language representation and control. *Acta Psychologica, 128*, 466–478.
Abutalebi, J., Miozzo, M., & Cappa, S. F. (2000). Do subcortical structures control 'language selection' in polyglots? Evidence from pathological language mixing. *Neurocase, 6*, 51–56.
Altarriba, J., & Gianico, J. L. (2003). Lexical ambiguity resolution across languages: A theoretical and empirical review. *Experimental Psychology, 50*, 159–170.
Altman, C., Goral, M., & Levy, E. S. (2012). Integrated narrative analysis in multilingual aphasia: The relationship among narrative structure, grammaticality and fluency. *Aphasiology, 26*, 1029–1052.
Alvarez, P., & Squire, L. R. (1994). Memory consolidation and the medial temporal lobe: A simple network model. *Proceedings of the National Academy of Sciences USA, 91*, 7041–7045.
Amberber, A. M. (2012). Language intervention in French-English bilingual aphasia: Evidence of limited therapy transfer. *Journal of Neurolinguistics, 25*, 588–614.
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*, 309–324.
Barca, L., Birani, C., & Arduino, L. S. (2002). Word naming times and psycholinguistic norms for Italian nouns. *Behavioral Research Methods Instruments & Computers, 34*, 424–434.
Bates, E., Burani, C., D'Amico, S., & Barca, L. (2001). Word reading and picture naming in Italian. *Memory & Cognition, 29*, 986–999.
Béland, R., & Mimouni, Z. (2001). Deep dyslexia in the two languages of an Arabic/French bilingual patient. *Cognition, 82*, 77–126.
Bello, L., Acerbi, F., Giussani, C., Baratta, P., Taccone, P., & Songa, V. (2006). Intraoperative language localization in multilingual patients with gliomas. *Neurosurgery, 58*, 115–125.
Birdsong, D., & Molis, M. (2001). On the evidence for maturational constraints in second language acquisition. *Journal of Memory and Language, 44*, 235–249.
Bishop, D. V. M. (1989). *Test for the reception of grammer: (TROG)* (2nd ed.). London: Medical Research Council.
Blonder, L. X. (2000). Language use. In S. E. Nadeau, L. J. Gonzalez Rothi, & B. Crosson (Eds.). *Aphasia and language. Theory to practice* (pp. 284–295). New York: Guilford Press.
Bohsali, A. A., Gullett, J. M., Mareci, T., Crosson, B., Fitzgerald, D. B., White, K., et al. (2015). Structural connectivity of Broca's region. *Neurology, 86*(16 Supplement) I7. 004.
de Bot, K., Cox, A., Ralston, S., Schaufeli, A., & Weltens, B. (1995). Lexical processing in bilinguals. *Second Language Research, 11*, 1–19.
Buxbaum, L. J., & Kalénine, S. (2010). Action knowledge, visuomotor activation, and embodiment in the two action systems. *Annals of the New York Academy of Sciences, 1191*, 201–218.
Carpenter, A. F., Georgopoulos, A. P., & Pellizer, G. (1999). Motor cortical encoding of serial order in a context recall task. *Science, 283*, 1752–1757.
Churchland, P. S., & Sejnowski, T. J. (1992). *The computational brain.* Cambridge, Massachusetts: MIT Press.
Colombo, L., & Burani, C. (2002). The influence of age of acquisition, root frequency and context availability in processing nounds and verbs. *Brain & Language, 81*, 398–411.
Colomé, A., & Miozzo, M. (2010). Which words are activated during bilingual word production? *Journal of Experimental Psychology: Learning, Memory, and Cognition, 36*, 96–109.
Conner, P. S., Goral, M., Anema, I., Borodkin, K., Heaendler, Y., Knoph, M., et al. (2018). The role of language proficiency and linguistic distance in cross-linguistic treatment effects in aphasia. *Clinical Linguistics and Phonetics,* 1–19. https://doi.org/10.1080/02699206.2018.1435723.
Costa, A., Calabria, M., Marne, P., Hernández, M., Juncadella, M., Gascón-Bayarri, J., ... Reñé, R. (2012). On the parallel deterioration of lexico-semantic processes in the bilinguals' two languages: Evidence from Alzheimer's disease. *Neuropsychologia, 50*, 740–753.
Costa, A., Caramazza, A., & Sebastián-Gallés, N. (2000). The cognate facilitation effect: Implications for models of lexical access. *Journal of Experimental Psychology: Learning, Memory and Cognition, 26*, 1283–1296.
Costa, A., & Santesteban, M. (2004). Lexical access in bilingual speech production: Evidence from language switching in highly proficient bilinguals and L2 learners. *Journal of Memory & Language, 50*, 494–511.
Croft, S., Marshall, J., Pring, T., & Hardwick, M. (2011). Therapy for naming difficulties in bilingual aphasia: Which language benefits. *International Journal of Language & Communication Disorders, 46*, 48–62.
Dai, E. Y. L., Kong, A. P. H., & Weekes, B. S. (2012). Recovery of naming and discourse production: A bilingual anomic case study. *Aphasiology, 26*, 737–756.
De Groot, A., Borgwaldt, A., Bos, M., & van den Eijinden, E. (2002). Lexical decision and word naming in bilinguals: Language effects and task effects. *Journal of Memory & Language, 47*, 91–124.
Dell, G. S., Schwartz, M. F., Martin, N., Saffran, E. M., & Gagnon, D. A. (1997). Lexical access in normal and aphasic speakers. *Psychological Review, 104*, 801–838.
Desimone, R., & Duncan, D. (1995). Neural mechanisms of selective visual attention. *Annual Review of Neuroscience, 18*, 193–222.
Detry, C., Pillon, A., & de Partz, M.-P. (2005). A direct processing route to translate words from the first to the second language: Evidence from a case of a bilingual aphasic. *Brain & Language, 95*, 40–41.
Diéguez-Vide, F., Gich-Fulla, J., Puig-Alcántara, J., Sánchez-Benavides, G., & Peña-Casanova, J. (2012). Chinese-Spanish-Catalan trilingual aphasia: A case study. *Journal of Neurolinguistics, 25*, 630–641.
Difrancesco, S., Pulvermüller, F., & Bohr, B. (2012). Intensive language-action therapy (ILAT): The methods. *Aphasiology, 26*, 1317–1351.
Dijkstra, T. (2003). Lexical processing and multilinguals: The word selection problem. In J. Cenoz, B. Hufeisen, & U. Jessner (Eds.). *The multilingual lexicon* (pp. 11–26). Dordrecht/Boston/London: kluwer Academic Publishers.

Dijkstra, T., Grainger, J., & van Heuven, W. J. B. (1999). Recognition of cognates and interlingual homographs: The neglected role of phonology. *Journal of Memory & Language, 41*, 496–518.
Druks, J., Aydelott, J., Genethliou, M., Jacobs, H., & Weekes, B. (2012). Progressive dyslexia: Evidence from Hungarian and English. *Behavioral Neurology, 25*, 185–191.
Druks, J., & Masterson, J. (2000). *An object and action naming battery*. East Sussex, UK: Psychology Press.
Druks, J., & Weekes, B. S. (2013). Parallel deterioration to language processing in a bilingual speaker. *Cognitive Neuropsychology, 30*, 578–596.
Edmonds, L. A., & Kiran, S. (2004). Confrontation naming and semantic relatedness judgements in Spanish/English bilinguals. *Aphasiology, 18*, 567–579.
Edmonds, L. A., & Kiran, S. (2006). Effect of semantic naming treatment on crosslinguistic generalization in bilingual aphasia. *Journal of Speech, Language & Hearing Research, 49*, 729–748.
Edmonds, L. A., Mammino, K., & Ojeda, J. (2014). Effect of verb network strengthening treatment (VNeST) in persons with aphasia: Extension and replication of previous findings. *Amererical Journal of Speech-Language Pathogy, 23*, S312–S329.
Edmonds, L. A., Nadeau, S. E., & Kiran, S. (2009). Effect of verb network strengthening treatment (VNeST) on lexical retrieval of content words in sentences in persons with aphasia. *Aphasiology, 23*, 402–424.
Eggert, G. H. (1977). *Wernicke's works in aphasia: A sourcebook and review*, *Vol. 1*. The Hague, Netherlands: Mouton.
Ellis, A., & Lambon Ralph, M. A. (2000). Age of acquisition effects in adult lexical processing reflect loss of plasticity in maturing systems: Insights from connectionist networks. *Journal of Experimental Psychology: Learnearning, Memory & Cognition, 26*, 1103–1123.
Fabbro, F. (1999). *The neurolinguistics of bilingualism.* Hove: Psychology Press.
Fabbro, F. (2001). The bilingual brain: Bilingual aphasia. *Brain & Language, 79*, 201–210.
Fabbro, F., & Paradis, M. (1995a). Acquired aphasia in a bilinguistic child. In M. Paradis (Ed.). *Aspects of bilingual aphasia* (pp. 67–83). Oxford: Elsevier Science Ltd.
Fabbro, F., & Paradis, M. (1995b). Differential impairments in four multilingual patients with subcortical lesions. In M. Paradis (Ed.). *Aspects of bilingual aphasia* (pp. 139–176). Oxford: Elsevier Science Ltd.
Fabbro, F., Peru, A., & Skrap, M. (1997). Language disorders in bilingual patients after thalamic lesions. *Journal of Neurolinguistics, 10*, 347–367.
Fabbro, F., Skrap, M., & Aglioti, S. (2000). Pathological switching between languages after frontal lesions in a bilingual patient. *Journal of Neurology, Neurosurgery & Psychiatry, 68*, 650–652.
Faroqi-Shah, Y., & Virion, C. R. (2009). Constraint-induced language therapy for agrammatism: Role of grammaticality constraints. *Aphasiology, 23*, 977–988.
Faroqi-Shah, Y., & Waked, A. N. (2010). Grammatical category dissociation in multilingual aphasia. *Cognitive Neuropsychology, 27*, 181–203.
Fernandino, L., & Iacoboni, M. (2010). Are cortical motor maps based on body parts of coordinated actions? Implications for embodied semantics. *Brain & Language, 112*, 44–53.
Ferretti, T. R., McRae, K., & Hatherell, A. (2001). Integrating verbs, situation schemas, and thematic role concepts. *Journal of Memory & Language, 44*, 516–547.
Filley, C. M., Ramsberger, G., Menn, L., Wu, J., Reid, B. Y., & Reid, A. L. (2006). Primary progressive aphasia in a bilingual woman. *Neurocase, 12*, 296–299.
Forde, E. M. E., & Humphreys, G. W. (1999). Category specific recognition impairments: A review of important case studies and influential theories. *Aphasiology, 13*, 169–193.
Freud, S. (1953). Zur Auffassung der Aphasie. Leipzig: Wien: Deuticke, 1891. Translated in. In E. Stengel (Ed.). *On aphasia, a critical study*. New York: International University Press.
Frey, S., Campbell, J. S., Pike, G. B., & Petrides, M. (2008). Dissociation of the human language pathways with high angular resolution diffusion tensor tractography. *Journal of Neuroscience, 28*, 11435–11444.
Galloway, L. (1978). Language impairment and recovery in polyglot aphasia. A case study of a heptlingual. In M. Paradis (Ed.). *Aspects of bilingualism* (pp. 139–148). Columbia, South Carolina: Hornbeam Press.
Georgopoulos, A. P., Kalaska, J. F., Caminiti, R., & Massey, J. T. (1982). On the relations between the direction of two-dimensional arm movements and cell discharge in primate motor cortex. *Journal of Neuroscience, 2*, 1527–1537.
Gerstner, W., Sprekeler, H., & Deco, G. (2012). Theory and simulation in neuroscience. *Science, 338*, 60–65.
Gil, M., & Goral, M. (2004). Nonparallel recovery in bilingual aphasia: Effects of language choice, language proficiency, and treatment. *International Journal of Bilingualism, 8*, 191–219.
Giussani, C., Roux, F.-E., Lubrano, V., Gaini, S. M., & Bello, L. (2007). Review of language organisation in bilingual patients: What can we learn from direct brain mapping. *Acta Neurochirurgica, 149*, 1109–1116.
Gleick, J. (1987). *Chaos: Making a new science.* New York: Viking.
Gollan, T. H., & Acenas, L. R. (2004). What is a TOT? Cognate and translation effects on tip-of-the-tongue states in Spanish-English and Tagalog-English bilinguals. *Journal of Experimental Psychology: Learning, Memory & Cognition, 30*, 246–269.
Gollan, T. H., Salmon, D. P., Montoya, R. I., & da Pena, E. (2010). Accessibility of the nondominant language in picture naming: A counterintuitive effect of dementia on bilingual language production. *Neuropsychologia, 48*, 1356–1366.
Gollan, T. H., Weissberger, G. H., Runnqvist, E., Montoya, R. I., & Cera, C. M. (2012). Self-ratings of spoken language dominance: A multilingual naming test (MINT) and preliminary norms for young and aging Spanish-English bilinguals. *Bilingualism: Language and Cognition, 15*, 594–615.
Goral, M., Levy, E. S., & Kastle, R. (2010). Cross-language treatment generalization: A case of trilingual aphasia. *Aphasiology, 24*, 170–187.
Goral, M., Levy, E. S., Obler, L. K., & Cohen, E. (2006). Cross-language lexical connections in the mental lexicon: Evidence from a case of trilingual aphasia. *Brain & Language, 98*, 235–247.
Goral, M., Naghibolhosseini, M., & Conner, P. S. (2013). Asymmetric inhibitory treatment effects in multilingual aphasia. *Cognitive Neuropsychology, 30*, 564–577.
Goral, M., Rosas, J., Conner, P. S., Maul, K. K., & Obler, L. R. (2012). Effects of language proficiency and language of the environment on aphasia therapy in a multilingual. *Journal of Neurolinguistics, 25*, 538–551.
Gray, T., & Kiran, S. (2013). A theoretical account of lexical and semantic naming deficits in bilingual aphasia. *Journal of Speech, Language & Hearing Research, 56*, 1314–1327.
Green, D. W., & Abutalebi, J. (2008). Understanding the link between bilingual aphasia and language control. *Journal of Neurolinguistics, 21*, 558–576.
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*, 188–209.
Green, D. W., Ruffle, L., Grogan, A., Ali, N., Ramsden, S., Schofield, T., ... Price, C. J. (2011). Parallel recovery in a trilingual speaker: The use of the bilingual aphasia test as a diagnostic complement to the comprehensive aphasia test. *Clinical Linguistics & Phonetics, 25*, 499–512.
Hartsuiker, R. J., & Pickering, M. T. (2008). Language integration in bilingual sentence production. *Acta Psychologica, 128*, 479–489.
Hartsuiker, R. J., Pickering, M. T., & Veltkamp, E. (2004). Is syntax separate or shared between languages? Corss-linguistic syntactic priming in Spanish-English bilinguals. *Psychological Science, 15*, 409–414.
Hernández, M., Caño, A., Costa, A., Sebastián-Galles, N., Juncadella, M., & Gascón-Bayarri, J. (2008). Grammatical category-specific deficits in bilingual aphasia. *Brain & Language, 107*, 68–80.
Hernandez, A. E., Dapretto, M., Mazziotta, J., & Bookheimer, S. Y. (2001). Language switching and language representation in Spanish-English bilinguals: An fMRI study. *NeuroImage, 14*, 510–520.
Hernandez, A. E., & Reyes, I. (2002). Within- and between-language priming differ: Evidence from repetition of pictures in Spanish-English bilinguals. *Journal of Experimental Psychology: Learning, Memory & Cognition, 28*, 726–734.
Hinckley, J. J. (2003). Picture naming treatment in aphasia yields greater improvement in L1. *Brain & Language, 87*, 171–172.
Hinton, G. E., & Shallice, T. (1991). Lesioning an attractor network: Investigations of acquired dyslexia. *Psychological Review, 98*, 74–95.
Hodges, J. R., Graham, N., & Patterson, K. (1995). Charting the progression in semantic dementia: Implications for the organization of semantic memory. *Memory, 3*, 463–495.
Indefrey, P., & Levelt, W. J. M. (2004). The spatial an temporal signatures of word production components. *Cognition, 92*, 101–144.

Izura, C., & Ellis, A. W. (2002). Age of acquisition effects in word recognition and production in first and second languages. *Psicológica, 23*, 245–281.
Joanisse, M. F., & Seidenberg, M. S. (2003). Phonology and syntax in specific language impairment: Evidence from a connectionist model. *Brain & Language, 86*, 40–56.
Junqué, C., Vendrell, P., & Vendrell, J. (1995). Differential impairments and specific phenomena in 50 Catalan-Spanish bilingual aphasic patients. In M. Paradis (Ed.). *Aspects of bilingual aphasia* (pp. 177–209). Oxford: Elsevier Science Ltd.
Junqué, C., Vendrell, P., & Vendrell-Brucey, J. M. (1989). Differential recovery in naming in bilingual aphasia. *Brain & Language, 36*, 16–22.
Kambanaros, M. (2010). Action and object naming versus verb and noun retrieval in connected speech: Comparisons in late bilingual Greek-English anomic speakers. *Aphasiology, 24*, 210–230.
Kambanaros, M., & Grohmann, K. K. (2011). Profiling performance in L1 and L2 observed in Greek-English bilingual aphasia using the bilingual aphasia test: A case study from Cyprus. *Clinical Linguistics & Phonetics, 25*, 513–529.
Kambanaros, M., & Grohmann, K. K. (2012). BATing multilingual primary progressive aphasia for Greek, English, and Czech. *Journal of Neurolinguistics, 25*, 520–537.
Kambanaros, M., & van Steenbrugge, W. (2006). Noun and verb processing in Greek-English bilingual individuals with anomic aphasia and the effect of instrumentality and verb-noun name relation. *Brain & Language, 97*, 162–177.
Keane, C., & Kiran, S. (2015). The nature of facilitation and interference in the multilingual language system: Insights from treatment in a case of trilingual aphasia. *Cognitive Neuropsychology, 32*, 169–194.
Kendall, D., Edmonds, L. A., van Zyl, A., Odendaal, I., Stein, M., & van der Merwe, A. (2015a). What can speech production errors tell us about crosslinguistic processing in bilingual aphasia? Evidence from four English/Afrikaans bilingual individuals with aphasia. *South African Journal of Communicative Disorders, 62*(1), 1–10. https://doi.org/10.4102/sajcd.v62i1.111 (Art #111).
Kendall, D. L., Oelke, M., Brookshire, C. E., & Nadeau, S. E. (2015b). The influence of phonomotor treatment on word retrieval abilities in 26 patients with chronic aphasia: An open trial. *Journal of Speech, Language, & Hearing Research, 58*, 798–812.
Kendall, D. L., Rosenbek, J. C., Heilman, K. M., Conway, T. W., Klenberg, K., Gonzalez Rothi, L. J., et al. (2008). Phoneme-based rehabilitation of anomia in aphasia. *Brain & Language, 105*, 1–17.
Kiran, S., & Iakupova, R. (2011). Understanding the relationship between language proficiency, language impairment and rehabilitation: Evidence from a case study. *Clinical Linguistics & Phonetics, 25*, 565–583.
Kiran, S., & Lebel, K. R. (2007). Crosslinguistic semantic and translational priming in normal bilingual individuals and bilingual aphasia. *Clinical Linguists & Phonetics, 21*, 277–303.
Kiran, S., & Roberts, P. M. (2010). Semantic feature analysis treatment in Spanish-English and French-English bilingual aphasia. *Aphasiology, 24*, 231–261.
Kiran, S., Sandberg, C., Gray, T., Ascenso, E., & Kester, E. (2013). Rehabilitation in bilingual aphasia: Evidence for within- and between-language generalization. *Americal Journal of Speech-Language Pathology, 22*, S298–S309.
Knoph, M. I. K. (2011). Language assessment of a Farsi-Norwegian bilingual speaker with aphasia. *Clinical Linguistics & Phonetics, 25*, 530–539.
Knoph, M. I. N., Simonsen, H. G., & Lind, M. (2017). Cross-linguistic transfer effects of verb-production therapy in two cases of multilingual aphasia. *Aphasiology, 31*, 1482–1509.
Kohnert, K. J., Hernandez, A. E., & Bates, E. (1998). Bilingual performance on the Boston naming test: Preliminary norms in Spanish and English. *Brain & Language, 65*, 422–440.
Kotik-Friedgut, B. (2001). A systemic-dynamic Lurian approach to aphasia in bilingual speakers. *Communicative Disorders Quarterly, 22*, 100–109.
Kremin, H., & De Agostini, M. (1995). Impaired and preserved picture naming in two bilingual patients with brain damage. In M. Paradis (Ed.). *Aspects of bilingual aphasia* (pp. 101–110). Oxford: Elsevier Science Ltd.
Kroll, J. F., Bobb, S. C., Misra, M., & Guo, T. (2008). Language selection in bilingual speech: Evidence for inhibitory processes. *Acta Psychologica, 128*, 416–430.
L'Hermitte, R., Hécaen, H., Dubois, J., Culioli, A., & Tabouret-Keller, A. (1966). Le probleme de l'aphasie des polyglottes: Remarques sur quelques observations. *Neuropsychologia, 4*, 315–329.
Lalor, E., & Kirsner, K. (2000). Cross-lingual transfer effects between English and Italian cognates and noncognates. *International Journal of Bilingualism, 4*, 385–398.
Lalor, E., & Kirsner, K. (2001). The role of cognates in bilingual aphasia: Implications for assessment and treatment. *Aphasiology, 15*, 1047–1056.
Lambon Ralph, M. A., & Ehsan, S. (2006). Age of acquisition effects depend on the mapping between representations and the frequency of occurrence: Empirical and computational evidence. *Visual Cognition, 13*, 928–948.
Lambon Ralph, M. A., Jefferies, E., & Patterson, K. (2017). The neural and computational bases of semantic cognition. *Nature Reviews/Neuroscience, 18*, 42–55.
Langanaro, M., & Venet, M. O. (2001). Acquired alexia in multilingual aphasia and computer-assisted treatment in both languages: Issues of generalization and transfer. *Folia Phoniatrica et Logopaedica, 53*, 135–144.
Lebedev, M. A., & Nicolelis, M. A. (2017). Brain-machine interfaces: From basic science to neuroprostheses and neurorehabilitation. *Physiological Reviews, 97*, 767–837.
Lemhöfer, K., & Dijkstra, T. (2004). Recognizing cognates and interlingual homographs: Effects of code similarity in language-specific and generalized lexical decision. *Memory & Cognition, 32*, 533–550.
Levelt, W. J. M., Roelofs, A., & Meyer, A. S. (1999). A theory of lexical access in speech production. *Behavioral Brain Sciences, 22*, 1–75.
Lissauer, H. (1988). Ein fall von seelenblindheit nebst einem beitrag sur theorie derselven. *Cognitive Neuropsychology, 5*, 157–192.
Lucas, T. H., McKhann, G. M., & Ojemann, G. A. (2004). Functional separation of languages in the bilingual brain: A comparison of electrical stimulation language mapping in 25 bilingual patients and 117 monolingual control patients. *Journal of Neurosurgery, 101*, 449–457.
Maher, L. M., Kendall, D., Swearengin, J. A., Pingel, K., Holland, A., & Roth, L. J. G. (2003). Constraint induced language therapy for chronic aphasia: Preliminary findings. *Journal of the International Neuropsychological Society, 9*, 192.
Majid, A., Boster, J. S., & Bowerman, M. (2008). The cross-linguistic categorization of everyday events: A study of cutting and breaking. *Cognition, 109*, 235–250.
Marangolo, P., Rizzi, C., Peran, P., Piras, F., & Sabatini, U. (2009). Parallel reovery in a bilingual aphasic: A neurolinguistic and fMRI study. *Neuropsychology, 23*, 405–409.
Marian, V., & Kaushanskaya, M. (2007). Cross-linguistic transfer and borrowing in bilinguals. *Applied Psycholinguistics, 28*, 369–390.
Marién, P., Abutalebi, J., Engelborghs, S., & De Deyn, P. P. (2005). Pathophysiology of language switching and mixing in an early bilingual child with subcortical aphasia. *Neurocase, 11*, 385–398.
McCann, C., Lee, T., Purdy, S. C., & Paulin, A. K. (2012). The use of the Bilingual Aphasia Test with a bilingual Mandarin-New Zealand English speaker with aphasia. *Journal of Neurolinguistics, 25*, 579–587.
McClelland, J. L., McNaughton, B. L., & O'Reilly, R. C. (1995). Why there are complementary learning systems in the hippocampus and neocortex: Insights from the successes and failures of connectionist models of learning and memory. *Psychological Review, 102*, 419–457.
McClelland, J. L., & Rumelhart, D. E. PDP Research Group. (1986). *Parallel distributed processing.* Cambridge, MA: MIT Press.
McClung, J. S., Rothi, L. J. G., & Nadeau, S. E. (2010). Ambient experience in restitutive treatment of aphasia. *Frontiers in Human Neuroscience, 4*, 1–19.
McRae, K., Hare, M., Elman, J. L., & Ferretti, T. R. (2005). A basis for generating expectancies for verbs from nouns. *Memory & Cognition, 33*, 1174–1184.
Meinzer, M., Djundja, D., Barthel, G., Elbert, T. R., & Rockstroh, B. (2005). Long-term stability of improved language functions in chronic aphasia after constraint-induced aphasia therapy. *Stroke, 36*, 1462–1466.
Meinzer, M., Obleser, J., Flaisch, T., Eulitz, C., & Rockstroh, B. (2007a). Recovery from aphasia as a function of language therapy in an early bilingual patient demonstrated by fMRI. *Neuropsychologia, 45*, 1247–1256.
Meinzer, M., Streiftau, S., & Rockstroh, B. (2007b). Intensive language training in the rehabilitation of chronic aphasia: Efficient training by laypersons. *Journal of the International Neuropsychological Society, 13*, 846–853.
Mendez, M. F., Sachafi, S., & Clark, D. G. (2004). Semantic dementia in multilingual patients. *Journal of Neuropsychiatry & Clinical Neuroscience, 16*, 381.
Meuter, R. F. I., & Allport, A. (1999). Bilingual language switching in naming: Asymmetrical costs of language selection. *Journal of Memory & Language, 40*, 25–40.
Miertsch, B., Meisel, J. M., & Isel, F. (2009). Non-treated languages in aphasia therapy of polyglots benefit from improvement in the treated language. *Journal of Neurolinguistics, 22*, 135–150.
Miozzo, M., Costa, A., Hernández, M., & Rapp, B. (2010). Lexical processing in the bilingual brain: Evidence from grammatical/morphological deficits. *Aphasiology, 24*,

262–287.
Monaghan, J., & Ellis, A. W. (2002). What , exactly, interacts with spelling-sound consistency in word naming? *Journal of Experimental Psychology: Learning, Memory & Cognition, 28*, 183–206.
Moran, J., & Desimone, R. (1985). Selective attention gates visual processing in extrastriate cortex. *Science, 229*, 782–784.
Muñoz, M. L., & Marquardt, T. P. (2003). Picure naming and identification in bilingual speakers of Spanish and English with and without aphasia. *Aphasiology, 17*, 1115–1132.
Nadeau, S. E. (2001). Phonology: A review and proposals from a connectionist perspective. *Brain Language, 79*, 511–579.
Nadeau, S. E. (2012). *The neural architecture of grammar.* Cambridge, MA: MIT Press.
Nadeau, S. E. (2015). Neuroplastic mechanisms of language recovery after stroke. In J. E. Tracy, B. M. Hampstead, & K. Sathian (Eds.). *Cognitive plasticity in neurologic disorders* (pp. 61–84). Oxford: Oxford University Press.
Nadeau, S. E. (2018). Aging-related alterations in language. In K. M. Heilman, & S. E. Nadeau (Eds.). *Cognitive changes and the aging brain*Cambridge, U.K: Cambridge University Press (In Press).
Nadeau, S. E., & Crosson, B. (1997). Subcortical aphasia. *Brain Language, 58*(355–402), 436–458.
Nadeau, S. E., & Kendall, D. L. (2006). Significance and possible mechanisms underlying generalization in aphasia therapy: Semantic treatment of anomia. *Brain Language, 99*, 10–11.
Nadeau, S. E., McCoy, K. J. M., Crucian, G. P., Greer, R. A., Rossi, F., Bowers, D., ... Triggs, W. J. (2002). Cerebral blood flow changes in depressed patients after treatment with repetitive transcranial magnetic stimulation. *Evidence of individual variability. Neuropsychiatry, Neuropsychology & Behavioral Neurology, 15*, 159–175.
Nadeau, S. E., Rothi, L. J. G., & Rosenbek, J. C. (2008). Language rehabilitation from a neural perspective. In R. Chapey (Ed.). *Language intervention strategies in aphasia and related neurogenic communication disorders* (pp. 689–734). (5th ed.). Philadelphia, PA: Lippincot Williams & Wilkins.
Nilipour, R., & Ashayeri, H. (1989). Alternating antagonism between two languages with successive recovery of a third in a trilingual aphasic patient. *Brain & Language, 36*, 23–48.
O'Keefe, J., & Nadel, L. (1979). The hippocampus as a cognitive map. *Behavioral and Brain Sciences, 2*, 487–533.
Ojemann, G. A. (1991). Cortical organization of language. *Journal of Neuroscience, 11*, 2281–2287.
Ojemann, G. A., Ojemann, J., Lettich, E., & Berger, M. (1989). Cortical language localization in left, dominant hemisphere. An electrical stimulation mapping investigation in 117 patients. *Journal of Neurosurgery, 71*, 316–326.
Paradis, M. (1977). Bilingualism and aphasia. In H. Whitaker, & H. A. Whitaker (Vol. Eds.), *Studies in neurolinguistics: Vol. 3*, (pp. 65–121). New York: Academic Press.
Paradis, M. (1994). Neurolinguistic aspects of implicit and explicit memory: Implications for blingualism. In N. Ellis (Ed.). *Implicit and explicit learning of second language* (pp. 393–419). London: Academic Press.
Paradis, M. (2009). *Declarative and procedural determinants of second languages.* Amsterdam/Philadelphia: John Benjamins.
Paradis, M., & Goldblum, M.-C. (1989). Selective crossed aphasia in a trilingual aphasic patient followed by reciprocal antagonism. *Brain & Language, 36*, 62–75.
Paradis, M., Goldblum, M.-C., & Abidi, R. (1982). Alternate antagonism with paradoxical translation behavior in two bilingual aphasic patients. *Brain & Language, 15*, 55–69.
Perecman, E. (1984). Spontaneous translation and language mixing in a polyglot aphasic. *Brain & Language, 23*, 43–63.
Pitres, A. (1895). Etude sur l'aphasia chez les polyglottes. [Readings on Aphasia in Bilinguals and Polyglots, Montreal:. Montreal: Marcel-Didier. 1983:26-49]. *Revue de Médicine, 15*, 873–899.
Plaut, D. C., McClelland, J. L., Seidenberg, M. S., & Patterson, K. (1996). Understanding normal and impaired word reading: Computational principles in quasi-regular domains. *Psychological Review, 103*, 56–115.
Porter, R., & Lemon, R. (1993). *Corticospinal function and voluntary movement.* Oxford: Clarendon Press.
Pouratian, N., Cannestra, A. f., Bookheimer, S. Y., Martin, N. A., & Toga, A. W. (2004). Variability of intraoperative electrocortical stimulation mapping parameters across and within individuals. *Journal of Neurosurgery, 101*, 458–466.
Pulvermüller, F. (2013). Semantic embodiment, disembodiment, or misembodiment? I search of meanilng in modules and neuron circuits. *Brain & Language, 127*, 86–103.
Pulvermüller, F., Neininger, B., Elbert, T., Mohr, B., Rockstroh, B., Koebbel, P., et al. (2001). Constraint-induced therapy of chronic aphasia after stroke. *Stroke, 32*, 1621–1626.
Rapport, R. L., Tan, C. T., & Whitaker, H. A. (1983). Language function and dysfunction among Chinese- and English-speaking polyglots: Cortical stimulation, Wada testing, and clinical studies. *Brain & Language, 18*, 342–366.
Ribot, T. (1882). *Disease of memory.* London: Kegan Paul, Trench and Co.
Roberts, P. M., & Deslauriers, L. (1999). Picture naming of cognate and non-cognate nouns in bilingual aphasia. *Journal of Communication Disorders, 32*, 1–23.
Roberts, P. M., & Le Dorze, G. (1998). Bilingual aphasia: Semantic organization, strategy use, and productivity in semantic verbal fluency. *Brain & Language, 65*, 287–312.
Rogers, T. T., Lambon Ralph, M. A., Garrard, P., Bozeat, S., McClelland, J. L., Hodges, J. R., et al. (2004). Structure and deterioration of semantic memory: A neuropsychological and computational investigation. *Psychological Review, 111*, 205–235.
Rolls, E. T. (2016). *Cerebral cortex: Principles of operation.* Oxford: Oxford University Press.
Rolls, E. T., & Deco, G. (2002). *Computational neuroscience of vision.* Oxford: Oxford University Press.
Rolls, E. T., & Deco, G. (2015). Stochastic cortical neurodynamics underlying the memory and cognitive changes in aging. *Neurobiology of Learning & Memory, 118*, 150–161.
Rolls, E. T., & Treves, A. (1998). *Neural networks and brain function.* New York: Oxford University Press.
Roth, H. L., Nadeau, S. E., Hollingsworth, A. L., Cimino-Knight, A. M., & Heilman, K. M. (2006). Naming concepts: Evidence of two routes. *Neurocase, 12*, 61–70.
Roux, F.-E., Lubrano, V., Lauwers-Cances, V., Trémoulet, M., Mascott, C. R., & Démonet, J.-F. (2004). Intra-operative mapping of cortical areas involved in reading in mono- and biligual patients. *Brain, 127*, 1796–1810.
Roux, F.-E., & Trémoulet, M. (2002). Organization of language areas in bilingual patients: A cortical stimulation study. *Journal of Neurosurgery, 97*, 857–864.
Rumelhart, D. E., & McClelland, J. L. (1986). On learning the past tenses of English verbs. In J. L. McClelland, D. E. Rumelhart, & the PDP Research Group (Vol. Eds.), *Parallel distributed processing: Vol. 2*, (pp. 216–271). Cambridge, MA: MIT Press.
Rumelhart, D. E., Smolensky, P., McClelland, J. L., & Hinton, G. E. (1986). Schemata and sequential thought processes in PDP models. In J. L. McClelland, D. E. Rumelhart, & the PDP Research Group (Vol. Eds.), *Parallel distributed processing: Vol. 2*, (pp. 7–57). Cambridge, MA: MIT Press.
Salvatierra, J., Rosselli, M., Acevedo, A., & Duara, R. (2007). Verbal fluency in bilingual Spanish/English Alzheimer's disease patients. *American Journal of Alzheimer's Disease and Other Dementias, 22*, 190–201.
Sánchez-Casas, R., & García-Albea, J. E. (2005). The representation of cognate and noncognate words in bilingual memory. In J. F. Kroll, & A. De Groot (Eds.). *Handbook of bilingualism. Psycholinguistic approaches* (pp. 226–250). Oxford: Oxford University Press.
Sasanuma, S., & Park, H. S. (1995). Patterns of language deficits in two Korean-Japanese bilingual aphasic patients. In M. Paradis (Ed.). *Aspects of bilingual aphasia* (pp. 111–122). Oxford: Elsevier Science Ltd.
Sebastian, R., Kiran, S., & Sandberg, C. (2012). Semantic processing in Spanish-English bilinguals with aphasia. *Journal of Neurolinguistics, 25*, 240–262.
Sebastian, R., Laird, A., & Kiran, S. (2011). Meta-analysis of the neural representation of first language and second language. *Applied Psycholinguistics, 32*, 799–819.
Seidenberg, M. S., & McClelland, J. L. (1989). A distributed, developmental model of word recognition and naming. *Psychological Review, 96*, 523–568.
Shin, J.-A., & Christianson, K. (2009). Syntactic processing in Korean-English bilingual production: Evidence from cross-linguistic priming. *Cognition, 112*, 175–180.
Shrager, Y., Levy, D. A., Hopkins, R. O., & Squire, L. R. (2008). Working memory and the organization of brain systems. *Journal of Neuroscience, 28*, 4818–4822.
Siyambalapitiya, S., Chenery, H. J., & Copland, D. A. (2013). Lexical-semantic rerpresentation in bilingual aphasia: Findings from semantic priming and cognate repetition priming. *Aphasiology, 27*, 1302–1321.

Spivey, M. (2007). *The continuity of mind.* Oxford: Oxford University Press.
Squire, L. R., & Zola-Morgan, S. (1991). The medial temporal lobe memory system. *Science, 253*, 1380–1386.
Stadie, N., Springer, L., De Bleser, R., & Bürk, F. (1995). Oral and written naming in a multilingual aphasic patient. In M. Paradis (Ed.). *Aspects of bilingual aphasia* (pp. 85–99). Oxford: Elsevier Science Ltd.
Stengel, E., & Zelmanowitz, J. (1933). Über polyglotte motorische Aphasie. *Zeitschrift fuer die Gesamte Neurologie und Psychiatrie, 149*, 291–301.
Strijkers, K., & Costa, A. (2016). The cortical dynamics of speaking: Present shortcomings and future avenues. *Language, Cognition & Neuroscience, 31*, 484–503.
Thomas, M. S. C. (1997). Distributed representations and the bilingual lexicon: One store or two? In J. Bullinaria, D. Glasspool, & G. Houghton (Eds.). *Proceeedings of the fourth annual neural computation and psychology workshop* (pp. 240–253). London: Springer.
Tononi, G., & Cirelli, C. (2014). Sleep and the price of plasticity; from synaptic and cellular homeostasis to memory consolidation and integration. *Neuron, 81*, 12–34.
Tremblay, P., & Dick, A. D. (2016). Broca and Wernicke are dead, or moving past the classic model of language neurobiology. *Brain & Language, 162*, 60–71.
Ullman, M. T. (2001a). The neural basis of lexicon and grammar in first and second language: The declarative/proceural model. *Bilingualism: Language and Cognition, 4*, 105–122.
Ullman, M. T. (2001b). A neurocognitive perspective on language: The declarative/procedural model. *Nature Reviews/Neuroscience, 2*, 717–726.
Ullman, M. T. (2005). A cognitive neuroscience perspective on second language acquisition: The declarative/procedural model. In C. Sanz (Ed.). *Mind and context in adult second language acquisition: Methods, theory and practice* (pp. 141–178). Washington, DC: Georgetown University Press.
Van Hell, J. G., & De Groot, A. M. B. (1998). Conceptual representation in bilingual memory: Effects of concreteness and cognate status in word association. *Bilingualism: Language and Cognition, 1*, 193–211.
Vargha-Khadem, F., Gadian, D. G., Watkins, K. E., Connelly, A., Van Paesschen, W., & Mishkin, M. (1997). Differential effects of early hippocampal pathology on episodic and semantic memory. *Science, 277*, 376–380.
Verreyt, N., Bogaerts, L., Cop, U., Bernolet, S., De Letter, M., Hemelsoet, D., ... Duyck, W. (2013a). Syntactic priming in bilingual patients with parallel and differential aphasia. *Aphasiology, 27*, 867–887.
Verreyt, N., De Letter, M., Hemelsoet, D., Santens, P., & Duyck, W. (2013b). Cognate effects and executive control in a patient with differential bilingual aphasia. *Applied Neuropsychology: Adult, 20*, 221–230.
Vingerhoets, G., Van Borsel, J., Tesink, C., van den Noort, M., Deblaere, K., Seruinck, R., et al. (2003). Multilingualism: An fMRI study. *NeuroImage, 20*, 2181–2196.
Vitevitch, M. S., & Castro, N. (2015). Using network science in the language sciences and clinic. *International Journal of Speech language Pathology, 17*, 13–25.
Vitevitch, M. S., & Luce, P. A. (2016). Phonological neighborhood effects in spoken word perception and production. *Annual Review of Linguistics, 2*, 75–94.
Voinescu, I., Vish, E., Sirian, S., & Maretsis, M. (1977). Aphasia in a polyglot. *Brain & Language, 4*, 165–176.
Walker, J. A., Quiñones-Hinojosa, A., & Berger, M. S. (2004). Intraoperative speech mapping in 17 bilingual patients undergoing resection of a mass lesion. *Neurosurgery, 54*, 113–118.
Warrington, E. K., & Shallice, T. (1984). Category specific semantic impairments. *Brain, 107*, 829–854.
Watamori, T. S., & Sasanuma, S. (1976). The recovery process of a bilingual aphasic. *Journal of Communication Disorders, 9*, 157–166.
Weber, K., & Indefrey, P. (2009). Syntactic priming in German-English bilinguals during sentence comprehension. *NeuroImage, 46*, 1164–1172.
Weekes, B. (2005). Acquired disorders of reading and writing: Cross-script comparisons. *Behavioral Neurology, 16*, 51–57.
Weekes, B. S., & Raman, I. (2008). Bilingual deep dysphasia. *Cognitive Neuropsychology, 25*, 411–436.
Wisenburn, B., & Mahoney, K. (2009). A meta-analysis of word-finding treatments for aphasia. *Aphasiology, 23*, 1338–1352.
Yang, J., Tan, L. H., & Li, P. (2011). Lexical representation of nouns and verbs in the late bilingual brain. *Journal of Neurolinguistics, 24*, 674–682.
Zanini, S., Angeli, V., & Tavano, A. (2011). Primary progressive aphasia in a bilingual speaker: A single case study. *Clinical Linguistics & Phonetics, 25*, 553–564.
Zhang, K., Ginzburg, I., McNaughton, B. L., & Sejnowski, T. J. (1998). Interpreting neuronal population activity by reconstruction: Unified framework with application to hippocampal place cells. *Journal of Neurophysiology, 79*, 1017–1044.
Zhang, K., & Sejnowski, T. J. (1999). Neuronal tuning: To sharpen or broaden? *Neural Computation, 11*, 75–84.