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Chapter 6
Semantic Processing in Transcortical Sensory Aphasia

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Abstract

Transcortical sensory aphasia (TCSA) has historically been regarded as a disconnection syndrome characterized by impaired access between words and otherwise intact core object knowledge. Yet, an extensive body of research has also demonstrated a range of associated non-verbal semantic deficits in TCSA, suggestive of a multi-modal semantic impairment that transcends representational modality (i.e., language). Here we delineate the semantic impairment incurred in TCSA within a neurologically constrained model of semantic memory premised upon dynamic interactivity between stored knowledge (e.g., semantic features) and integrative processes that serve to bind this knowledge into cohesive object representations. We discuss practical implications for clinical aphasiology and outline considerations for the broader fields of cognitive neuropsychology and neurolinguistics.

Introduction

In a 1992 review of aphasia for the *New England Journal of Medicine*, Antonio Damasio remarked that since the latter half of the 19th century, "little new has been uncovered regarding the transcortical aphasias"(pg. 535). The same cannot be said for today's view of the unique neurological disorder known as *Transcortical Sensory Aphasia* (hereafter abbreviated TCSA). During the last decade, there has been a great resurgence of interest in TCSA and its associated semantic impairment. TCSA has since emerged as a controversial lesion model for parsing the organization of word and object knowledge, and much of this controversy is derived from recent studies that have revealed cracks in the assumption that the semantic impairment in TCSA, and more broadly in classical cortical aphasias, reflects a pure disconnection syndrome (Baldo et al., 2005; Bay, 1962; Caramazza, Berndt, & Brownell, 1982; Chertkow, Bub, Deaudon, & Whitehead, 1997; De Renzi, Faglioni, Scotti, & Spinnler, 1972; Gardner et al., 2012; Hamsher, 1998; Jefferies & Lambon Ralph, 2006; Noonan, Jefferies, Eshana, Garrard, & Lambon Ralph, 2013; Sandberg, Sebastian, & Kiran, 2012; Vallila-Rohter & Kiran, 2013).

Many neurolinguistic models hold in common the assumption that TCSA reflects a disruption in mapping between arbitrary symbols (i.e., words) and their associated object concepts (Lichtheim, 1885; Wernicke, 1874). An aphasiologist steeped in the associationist tradition expects to observe deficits in naming and auditory comprehension in TCSA. However, the associationist also operates under the assumption that this linguistic impairment masks intact object knowledge and that such knowledge can often be accessed through alternative representational modalities (e.g., tactile handling, odor, visual presentation) (for theory regarding the visual modality as providing 'privileged access' to the semantic system see Caramazza & Shelton, 1998). For a concrete example of this linguistic access assumption, consider the situation where an *apple* is placed into the hand of an individual with TCSA. Virtually all aphasia models generate the prediction that the person will spontaneously demonstrate his preserved knowledge by appropriately taking a bite out of the apple rather than throwing it like a baseball. Moreover, this access assumption also forms the basis for many multi-modal treatment strategies and semantic cueing hierarchies for aphasias such as TCSA that incur a primary semantic component (i.e., collectively referred to as semantic aphasia) (Boyle, 2004; Head, 1926; Hoffman, Jefferies, Ehsan, Hopper, & Lambon Ralph, 2009).

Over the last decade, the tenability of the linguistic access assumption has slowly been eroded by a growing body of literature demonstrating that individuals with TCSA also present with a range of non-verbal semantic impairments. People with various forms of post stroke semantic aphasia have been reported to experience paradoxical dissociations in tasks not commonly viewed as heavily language-mediated. For example, De Renzi and colleagues (1972) reported impairment among persons with aphasia in reliably choosing an appropriate colored pencil to fill in the dominant color in a series of line drawings depicting common objects (e.g., selecting 'yellow' to fill the boundaries of a banana). A variety of other studies have reported impairments in TCSA on tasks that measure semantic association and categorization abilities for pictures (Martin, Schwartz, & Kohen, 2006; Noonan, Jefferies, Corbett, & Lambon Ralph, 2010). Semantic aphasias such as TCSA have also been associated with paradoxical impairment on a variety of other non-linguistic tasks such as pantomiming the gestures of actions, using tools appropriately, discriminating between real and artificial environmental sounds, and matching environmental sounds to referents.

Some have argued that many experimental tasks used to elicit non-verbal semantic deficits in TCSA are confounded by numerous factors such as verbally mediated instructions and high executive demands, a factor we will soon revisit. This thorny point is not meant to obscure our central premise but instead to highlight our hypothesis that the dominant semantic impairment in TCSA has roots that transcend language. In the chapter to follow, we will make our case for this claim of a supra-linguistic impairment within the constraints of a model of semantic memory that we have recently advanced through work in other semantically-impaired populations (Reilly, Cross, Troiani, & Grossman, 2007; Reilly & Peelle, 2008; Reilly, Peelle, Antonucci, & Grossman, 2011; Reilly, Rodriguez, Peelle, & Grossman, 2011; Reilly et al., 2012). It is important to acknowledge that this model-driven approach represents one view among many potential alternatives and also offers a point of divergence from many previous descriptions of the disorder (e.g., Wernicke-Lichtheim model). Disclaimers aside, we turn now to a description of semantic memory and our guiding theoretical framework.

Semantic Memory: The What and Where of It

Semantic memory encompasses knowledge of object and word meaning, including encyclopedic facts and general world knowledge (Saffran, Schwartz, Umiltà, & Moscovitch,

1994; Tulving, 1972). As one might predict, the integrity of semantic memory is essential for negotiating many of our most fundamental interactions with the world. Thus, impairment in either access to object representations or degradation of the object representations themselves will typically compromise many global aspects of daily functioning (e.g., following a recipe, interpreting a road sign), including basic language expression and comprehension (Warrington, 1975).

Cognitive psychologist, Endel Tulving (1972), is often attributed with the formal distinction between episodic and semantic memory. By Tulving's episodic-semantic dichotomy, episodic memory is constrained to a specific temporal and environmental context linked to a singular event (e.g., source detail about when I was lost at the mall) or exemplar (e.g., my dog, Felix). In contrast, semantic memory consists of a form of de-contextualized knowledge that has been generated often over long periods of time by forming 'central tendencies' (e.g., dogs, mammals, tools) upon one's exposure to many exemplars (see also Moscovitch, Nadel, Winocur, Gilboa, & Rosenbaum, 2006; Murphy, 2002). Since the early 1970's, our understanding of semantic memory as a dissociable system (or systems) has undergone a rapid evolution that has been fueled by a vast body of parallel empirical work from aphasiology, neuropsychology, functional neuroimaging and behavioral paradigms (e.g., psycholinguistic investigations of semantic priming). Paradoxically, with the advent of new and sophisticated imaging and cortical stimulation technologies, the field has seen even further divergence. Today, there exist several entrenched and also diametrically oppositional theories of semantic organization within the human brain (Antonucci & Reilly, 2008; Binder, Desai, Graves, & Conant, 2009; Patterson, Nestor, & Rogers, 2007).

It has long been theorized that object knowledge is represented within the human brain by a process of decomposition whereby objects are initially torn apart and then reconstructed ad hoc. For example, a Labrador retriever has a set of distinct visual, odor, auditory, and tactile properties in addition to a range of affective and encyclopedic associations. In addition, object knowledge is mediated by representational modality (e.g., verbally learned versus visually observed) (see Dual Coding Theory as a mutliple semantic systems hypothesis in Paivio, 1985, 1991, 1995). Many cognitive neuroscientists believe that these disparate bits of information about objects are stored in long-term memory across a widely distributed network of

neuroanatomically remote brain regions. Moreover, theories of semantic memory that have strong sensorimotor grounding emphasize the idea that semantic features are stored either within or proximal to brain structures that are also engaged during the actual processes of sensation, perception, and motor execution (Barsalou, Simmons, Barbey, & Wilson, 2003; Gallese & Lakoff, 2005; Martin, 2007a, 2007b).

Wernicke and several prominent contemporaries, including Freud and Lissauer, proposed various iterations of fully distributed models of conceptual knowledge (Eggert, 1977; Freud, 1891; Gage & Hickok, 2005; Lissauer, 1890/1988; Wernicke, 1874), an overarching approach to memory organization that has both endured and evolved into one of the dominant theories of semantic representation today. Wernicke and others (e.g., Lichtheim) also held that the putative concept center or 'semantic field' (i.e., *Begriffsfeld* in the original German) was so diffusively distributed as to be virtually undifferentiated cortically (see also later theories of equipotentiality and mass action propounded by Lashley, 1948). Today's consensus within cognitive neuropsychology is that the supporting architecture of semantic memory, although highly distributed, does have both specificity and localization. Nevertheless, extant theories of semantic memory still radically differ with respect to just how 'distributed' they view knowledge representation (Reilly et al., 2014).

A fully distributed view holds that knowledge is maintained across many different parts of the brain without the necessity for a central organizing structure (i.e., a binding site or convergence zone). In the parlance of neural network modeling, our knowledge of objects reflects an auto-associative pattern of activation across multi-regional cell assemblies. As complex as this statement sounds, we can reframe it within the simple Hebbian maxim that as clusters of neurons repeatedly fire together, they soon wire together. This means that patterns of neural co-activation (i.e., simultaneous activity) that are potentiated through repeated experience (e.g., seeing many different dogs) ultimately serve to ground a basic object concept as a distributed activation pattern (Gage & Hickok, 2005; Pulvermüller, 1999; Pulvermüller, 2001). Importantly, this fully distributed theory makes the explicit prediction that there is no single brain region dedicated to the binding and representation of semantic memory. Therefore, this view also predicts that only the most diffuse and catastrophic brain injury would be sufficient to produce a global impairment of semantic memory. Figure 1 depicts a schematic of the fully

distributed theory of semantic feature representation and connectivity with the language system advanced by Allport (1985).

-Figure 1-

One of the greatest challenges for a fully distributed model of semantic memory is ambiguity with respect to its mechanism of semantic binding. That is, it is unclear how the brain can pull together and interpret a plethora of data into cohesive object representations with such remarkable speed and accuracy in the absence of a dedicated 'center'. This fundamental question, known as the *binding problem*, remains highly charged across neuroscience, consciousness research, and the philosophy of mind (Kandel, 2006; McNorgan, Reid, & McRae, 2011).

The polar alternative to a fully distributed model of semantic organization holds that human conceptual knowledge is organized in an entirely abstract, propositional manner that does not rigidly honor an object's sensorimotor properties (Fodor, 2000). That is, modality neutral regions of the human cortex pull from primary sensory regions and bind features into higher level conceptual representations. An important distinction with respect to this class of theory is that object concepts (e.g., Labrador retriever) are far more complex than the simple sum of their distributed parts (e.g., color, odor, sound). This class of theories has collectively been described as *amodal* or *supramodal* with respect to a necessary role of grounding semantic features across regions of the cortex that are critical for sensorimotor processing, perceptual simulation, and mental imagery.

One of the primary criticisms of early amodal semantics theories is that mapping a construct such as 'amodality' to the brain is opaque. In contrast, fully distributed models of semantic organization make the explicit and testable assumption that knowledge of a specific feature is organized within or near the brain structure that is dedicated to actual perception of that feature. For example, functional imaging studies have demonstrated that when participants are asked to imagine the color of a tomato, they engage anterior projections of the visual cortex that are also engaged when seeing a tomato (Chao, Haxby, & Martin, 1999; Chao & Martin, 1999). Thus, many distributed theories predict that the 'redness' feature of a tomato lives in the inferior temporal lobe. From a hypothesis testing standpoint, distributed theories offer a remarkable

advantage relative to early amodal semantics theories that tended to be unapologetically mute on the following questions: 1) What exactly is an abstract propositional representation of an object concept?; and 2) Where are these putative abstract representations housed within the human brain?

Contemporary amodal semantic theories have undergone a radical evolution in terms of their capacity to map onto brain structure. Much of this neurological foundation can be attributed to a highly influential theory known as the *convergence zone framework* proposed by Damasio, Tranel, and colleagues (Damasio & Damasio, 1994; Tranel, Grabowski, Lyon, & Damasio, 2005). These authors have advanced the hypothesis that distributed semantic feature knowledge gradually converges upon regions of the brain that are dedicated to polymodal feature binding (e.g., barking + musky odor = dog). Neurally encoded information streams efferently from the various receptive fields of topographically organized sensory regions (e.g., retinotopic organization within the primary visual cortex) toward the various sensory association areas. An important part of this process is that as featural information streams away from the primary sensory areas and into polymodal convergence zones, such information grows incrementally less literal and more abstracted (for rejection of this view in toto see Gallese & Lakoff, 2005).

Single cell recording and functional imaging data provide compelling evidence for a variety of convergence zones in the human brain (Beauchamp, 2005; Beauchamp, Argall, Bodurka, Duyn, & Martin, 2004; Binder et al., 2009; Fuster, Bodner, & Kroger, 2000; Taylor, Moss, Stamatakis, & Tyler, 2006). In the most extensive meta-analysis of functional neuroimaging studies of semantic processing to date, Binder and colleagues (2009) reported that one of the most consistently activated polymodal convergence zones is the angular gyrus and adjacent regions of the temporo-parietal junction. As we will soon discuss, this finding has critical relevance because the TPJ is also a canonical site of lesion for TCSA (Damasio, 1998, 2001).

Convergence zone theories hold in common the assumption that particular regions of the brain are essential for the integrity of semantic memory. That is, unlike the redundancy offered by a fully distributed organization of object knowledge, this framework predicts that focal damage to a convergence zone will produce a global impairment in semantic knowledge that transcends access-related factors such representational modality (e.g., writing or speech) or

sensory domain (e.g., odor, visual form, environmental sound). Elizabeth Warrington (1975) was perhaps the first to show that such a selective impairment of semantic memory, although rare, is indeed possible in a disorder later termed *semantic dementia*.

Today's dominant model of semantic memory (i.e., Hub & Spoke Model) is largely fueled by the unique phenotype and corresponding distribution of brain morphology in semantic dementia, the temporal lobe variant of frontotemporal degeneration, whose ubiquitous semantic impairment has been linked to bilateral dysfunction within the anterolateral temporal lobes (Gorno-Tempini et al., 2011; Jefferies & Lambon Ralph, 2006; Lambon Ralph, Cipolotti, Manes, & Patterson, 2011; Lambon Ralph, Graham, Ellis, & Hodges, 1998; Lambon Ralph, Lowe, & Rogers, 2007; Lambon Ralph & Patterson, 2008). The Hub & Spoke Model holds that ventral and anterolateral portions of the temporal lobes (i.e., temporal poles) act as massive convergence zones wherein modality-specific knowledge is bound and differentiated into an amodal form (Lambon Ralph et al., 2011; Lambon Ralph, Sage, Jones, & Mayberry, 2010; Patterson et al., 2007; Rogers et al., 2006; Rogers et al., 2004). The function of this putative 'hub' is to organize the distributed bits of knowledge that radiate inward from a series of 'spokes' that constitute modality-specific regions of the cortex. Proponents of the Hub & Spoke Model have argued that the temporal poles are anatomically well-suited to this purpose due to both their centrality and underlying functional connectivity (i.e., massive interconnections to medial temporal and frontal lobes in addition to direct relays to primary sensory areas).

Hub & Spoke model proponents have described semantic memory as an amodal and dynamic system whose effectiveness relies on a precise orchestration between stored knowledge and cognitive control mechanisms (i.e., semantic control) that operate on such knowledge (Coccia, Bartolini, Luzzi, Provinciali, & Lambon Ralph, 2004).[1] In our own work, we have outlined a similar dichotomy between the content of stored representations (i.e., *content*) and the range of functions (i.e., *process*) that are crucial for acting upon knowledge and constructing meaning (Grossman, Koenig, Troiani, Work, & Moore, 2007; Koenig & Grossman, 2007; Koenig, Smith, & Grossman, 2010; Peelle, Troiani, & Grossman, 2009; Reilly, Rodriguez, et al., 2011; Reilly et al., 2014). Thus far, our discussion has focused on the organization of semantic content. It is our central premise, however, that many of the semantic difficulties in TCSA have a basis within *process*. Thus, we shift our discussion to the role of process in semantic memory.

Process in Semantic Memory

Much of our daily functioning demands rapid object recognition and flexible use of our vast repertoire of semantic knowledge. By no means is this a passive process. We construct our own understanding of the world through long-term experience and assimilate a great deal of incoming perceptual and semantic detail with previous knowledge. Much of our online semantic processing is conducted so quickly that we must employ efficient cognitive shortcuts in the form of active organizing principles. For example, it has long been believed that object knowledge is organized in a hierarchical manner such that specific exemplars (e.g., Labrador Retriever) are nested within successively broader category distinctions (e.g., dog, animal, thing) (Rosch, 1973). We derive a rapid understanding of word and object identity (e.g., a tiny unfamiliar dog) by assigning tentative category membership through an active evaluative process comparing 'goodness of fit' (i.e., typicality) and updating the tenability of this initial guess as incoming data arrives (e.g., the tiny "dog" unexpectedly meows) (Sandberg et al., 2012). Clearly these semantic processing heuristics are resource demanding. Yet, we are just beginning to understand the unique and often idiosyncratic role played by process in assigning structure to semantic knowledge.

One unanswered question in cognitive neuropsychology regards whether there exists a dedicated executive semantic system or whether human conceptual knowledge is mitigated by a "domain-general" executive process that supports more global cognitive functions. Randi Martin and others have argued for the existence of a semantic short-term memory (STM) store, which is one component of a multi-store verbal STM model along with lexical and phonological STM stores (Martin, 2009; Martin, Shelton, & Yaffee, 1994). According to Saffran (1990) these levels of short-term storage are extensions of the levels of word representation (e.g., phonological, lexical) postulated in interactive activation models of word processing (Dell, 1986; Dell & O'Seaghdha, 1992; Dell, Schwartz, Martin, Saffran, & Gagnon, 1997; Foygel & Dell, 2000). The common factor that defines this extension is the maintenance of activated word representations during language processing. The process of activating and maintaining activation of semantic, lexical and phonological representations of words is time-dependent whether the language system is generating a single word or multiple words. This implicates some sort of short-term memory process as integral to word processing. As noted earlier,

'process' differs from content in that it operates over content. In this model, maintenance of activation is one of those operations, but others have also been implicated (e.g., strength of activation, inhibition of other competing activated representations and response selection).

It is worth noting an important distinction made in many models of word processing between conceptual semantic and word-specific (sometimes called verbal) semantic processing. In the word processing models of Dell and colleagues, for example, word-specific semantic features are distinguished from a conceptual semantic level of representation from which they are generated (Dell et al., 1997). It is the word-specific semantic features that subsequently activate associated words in the lexicon, which in turn connect with phonemes that comprise those words. As in multi-store models of verbal STM, some have postulated an extension of lexical-semantic representations toward encompassing a wider domain-neutral conceptual STM (e.g., words, pictures, environmental sounds). Potter (1993), for example, hypothesized the existence of a very short-term conceptual memory dedicated to knowledge representation. This lexical vs. conceptual processing distinction is of particular importance when considering the established view that TCSA reflects a focal breakdown in accessing lexical representations. If one considers the existence of a short-term conceptual store, it is conceivable that reports of impaired object knowledge in TCSA may actually be related to an impaired conceptual STM that subserves both lexical and non-lexical aspects of semantic memory in a blanket fashion.

Potter's (1976, 1993) work on conceptual STM focuses primarily on its role in enabling consolidation of fleeting conceptual events evoked by perceptual experience into long-term memory. She proposed a model detailing the relation between a modality-independent conceptual STM in relation to a conventional working memory model (Baddeley, 1986) whereby a verbal sentence is perceived and processed by the phonological STM system with its articulatory system but simultaneously processed by conceptual STM in which the conceptual aspects of the utterance are briefly activated in this short-term store and integrated with long-term memory. This model schematic is reflected in Figure 2a.

-Figure 2a & 2b-

Potter's (1993) original model did not specify a mechanism by which conceptual STM interacts further with language comprehension or production systems. Neither does it pre-

suppose a mechanism of mapping components onto the human brain. However, if we consider a typical word production model such as Dell's (1986) interactive activation model (Figure 2b), it is possible to extrapolate some semantic components from Potter's model. The semantic feature network, for example, may be equated with Potter's conceptual STM. Semantic features of words to be spoken are briefly activated as they initiate a spread of activation through the word production system. They are the first to be activated in production and the last to be activated in repetition or comprehension of auditory input. The conceptual 'cloud' that is often depicted above this semantic feature system may be equated with long-term memory in Potter's model. It seems likely that disparate terminology has made it difficult to think about Potter's conceptual STM and Dell's semantic feature system similarly, but when focusing on descriptions of their functions, the overlap is more apparent.

The contribution of a conceptual STM to the execution of non-verbal semantic tasks is crucial to consider. The selective impairment of semantic STM is one potential factor among a constellation of other dysexecutive impairments that have been argued to qualitatively distinguish the semantic impairment in TCSA from that evident in semantic dementia (Corbett, Jefferies, Ehsan, & Lambon Ralph, 2009; Corbett, Jefferies, & Lambon Ralph, 2011; Jefferies & Lambon Ralph, 2006; Noonan et al., 2010). Proponents of the Hub & Spoke Model have referred to the range of executive functions that support semantic processing collectively under the rubric of *semantic control.* Hub & Spoke proponents have also argued that brain damage in TCSA compromises a diverse range of cognitive control mechanisms, resulting in deregulation of semantic memory.

Hub & Spoke proponents have hypothesized that semantic control constitutes a sub-domain of generalized executive functioning (Hoffman et al., 2009). That is, semantic control draws on the same executive resource pool that is also dedicated to various other EF demands. As such, s*emantic control* is vulnerable to many of the same criticisms that have been lodged against executive functioning (EF) regarding limited construct specificity. Consider, for example, the wide range of EF abilities that contribute to our online construction of meaning, of which only a small subset includes: 1) inhibitory control and suppression of plausible competitors; 2) attentional vigilance; 3) selective attention; 4) error and anomaly detection; 5) hierarchical semantic categorization; 6) working memory updating ability; 7) cognitive

flexibility and task switching; 8) response selection; 9) lexical retrieval; 10) filtering irrelevant or non-diagnostic detail; 11) self-monitoring and arousal; 12) sustained attention; 13) divided attention and mitigation of dual task interference.

We have identified at least thirteen distinct executive components, many of which are believed to be both neurologically and behaviorally dissociable. We see ample evidence for dissociable components of EF within clinical aphasiology. For example, a bilingual adult's ability to flexibly switch between languages (i.e., code switching) is sometimes compromised by damage to portions of the basal ganglia (e.g., left caudate nucleus) that are also engaged during more general EF-related switching behaviors (e.g., switching during verbal fluency, changing the subject during conversation) (Crinion et al., 2006). In contrast, patients with damage to more lateral aspects of the frontal cortex (e.g., left inferior frontal gyrus) tend to show a different pattern of executive impairment that is characterized by strong proactive interference effects and a weakened ability to inhibit competing alternatives (Barde, Schwartz, Chrysikou, & Thompson-Schill, 2009; Schnur, Schwartz, Brecher, & Hodgson, 2006; Schnur et al., 2009). Thus, the emerging picture of EF is that of an umbrella construct that unites numerous subcomponents, and one can readily imagine how a selective 'lesion' to any single one of these subcomponents of EF might perturb online semantic processing. Yet, there is a paradox with respect to the relation between EF dysfunction and semantic impairment. That is, many studies have reported either null or weak correlations between measures of EF and semantic abilities (Fine, Delis, Paul, & Filoteo, 2011; Gongvatana, Woods, Taylor, Vigil, & Grant, 2007; Laisney et al., 2009). We see evidence for this null relation in both healthy and neuropsychologically impaired populations. If indeed semantic memory loads on a domain-general EF, then healthy normal adults should also show dual task costs (i.e., interference) when juggling virtually any other resource-demanding task. Yet, our ability to process basic conceptual knowledge is not radically impacted by concurrent attentional demands. Moreover, a number of neurological disorders present with prominent dysexecutive impairment, including traumatic brain injury, frontal variant of frontotemporal dementia, HIV dementia complex, and non-demented Parkinson's disease. Yet, these patients do not typically experience the profound severity of multi-modal semantic impairment seen semantic dementia or TCSA (Reilly, Peelle, et al., 2011)

In summary, much remains unclear about the role of process in semantic memory and whether there exists a dedicated module to conceptual STM (e.g., Potter, 1993). However, we have identified several reasons why a domain-general EF impairment might fail to drive the semantic impairment in TCSA.

A Hybrid Model of Semantic Memory: Multiple Convergence Zones with Sparse Representation

We recently proposed a model of semantic organization that integrates many of the relative merits of amodal and modality-specific theories of knowledge representation (Reilly, Antonucci, Peelle, & Grossman, 2011; Reilly & Peelle, 2008; Reilly et al., 2011). We turn now to a description of the cognitive and neural substrates of this particular theory, starting from the premise that semantic organization is highly distributed but also relies on a series of convergence zones. Thus, we are aligned with approaches to the architecture of semantic memory that are best described as hybrid in nature (Hart et al., 2007; Hart & Kraut, 2007). However, we diverge from extant hybrid theories in 1) how we view the nature of the semantic representation within convergence zones; and 2) which convergence zones we view as being essential for semantic memory.

We hypothesize that within convergence zones, object knowledge is stripped of its original sensorimotor salience and maintained in a sparse form (Reilly et al., 2012; 2014). One can conceive of a sparse representation as analogous to the way that the brain stores detail about episodic memories. That is, memory does not behave in a photorealistic manner but instead much non-essential source detail is filtered. For example, when one recalls details of a specific salient event (e.g., the time one was lost at a mall), details such as what one was wearing at the time are more vulnerable to forgetting and/or distortion than more salient diagnostic details. Sparse representations, therefore, reflect the aggregate of many modality-specific semantic features.

In this sparse representation approach, we have emphasized a role of reciprocal activation between convergence zones and distributed features. That is, as task demands dictate, one must perceptually enrich a sparse representation by indexing modality-specific brain regions of the cortex. Thus, object knowledge is re-constructed on line through a process of perceptual simulation or enactment (Barsalou, 2008; Kosslyn, 2005; Kosslyn, Ganis, & Thompson, 2001).

We hypothesize that these imagery and mental simulation processes are mediated by a modular semantic working memory system that is at least partially dissociable from that of the neural system that supports more general EF (Martin, Dell, Saffran, & Schwartz, 1994; Martin & Saffran, 1990; Martin et al., 1994; Poldrack et al., 1999; Thompson-Schill, D'Esposito, Aguirre, & Farah, 1997). This interactive system is perhaps best illustrated by a real-world example. Consider the following:

a) Is a Labrador Retriever friendlier than a German Shepherd?
b) Is a Labrador Retriever larger than a German Shepherd?
c) Does a Labrador Retriever bark louder than a German Shepherd?
d) Does a Labrador Retriever smell muskier than a German Shepherd?

These are simple questions that one might encounter on any given day. Yet, they tap very different aspects of conceptual knowledge, including affective associations (e.g., *friendlier than*), relative size (e.g., *larger than*), environmental sound (e.g., *louder than*), and olfaction (e.g., *smell muskier than*). We hypothesize that each of these questions forces the reader to ultimately converge upon a common, sparse representation for Labrador Retrievers and German Shepherds. However, convergence upon a common representation is only half of the battle; one must also index various modality-specific regions in order to successfully evaluate the given proposition (see also Kellenbach, Brett, & Patterson, 2001). That is, in order to make a relative size judgment in the absence of a visual stimulus (i.e., assuming neither dog is physically present while you read this), one must invoke visual imagery and spatial working memory. This specific task demand, therefore, calls for communication between a sparse representation and a concrete set of visual features stored primarily in the visual association cortex. Importantly, we hypothesize that this communication between convergence zones and modality-specific regions of the cortex is both reciprocal (i.e., 2-way) and cognitive resource demanding.

We offer the following analogy for understanding this multiple component approach to semantic memory. Consider that one is tasked with completing a research project that requires visiting the stacks of a massive reference library. Successful retrieval is contingent upon both a comprehensive inventory of books (semantic features) in tandem with a skilled librarian or search engine. Thus, in semantic memory there is an inextricable link between the content of stored knowledge and processes that operate upon such knowledge. *Process* includes resources

that are dedicated to response selection, inhibitory control of competing alternatives, semantic categorization, error and novelty detection, vigilance, sustained attention, and semantic working memory. *Content* encompasses our vast array of knowledge about the features of objects, including sensorimotor properties (e.g., color, form) as well as abstract lexical associations and verbally learned encyclopedic facts. As the discussion of TCSA unfolds, we interpret semantic impairment within the context of this overarching, multiple component model of semantic memory.

A Brief History of Transcortical Sensory Aphasia

Although an extensive historical treatise is beyond the scope of this review, one must first contextualize the evolution of our understanding of TCSA within the zeitgeist under which the syndrome was conceived. TCSA emerged as a unique syndrome at a pivotal point during the classical period of aphasiology of the 19th century. One of the major lower branches of an expanding tree structure of aphasia was propounded by Paul Broca (1865) who linked the seat of articulate language to the third frontal convolution of the left hemisphere [for parallel work see Marc Dax as reviewed in Buckingham (2006), and for a critique of the logic of language localization see Luria (1974)]. Broca is also renowned for establishing a distinction between classes of aphasia marked by deficits in fluent articulatory production (i.e., aphemia) or abstract symbol manipulation. Broca's localization of a fluent-nonfluent aphasia distinction using the lesion model approach set the stage for a rapid series of advances.

The next major bifurcation in a rapidly evolving aphasia tree structure resulted from Carl Wernicke's (1974) monograph, *Der Aphasiche Symptomcomplex: Eine Psychologische Studie auf Anatomischer Basis*. In this work, Wernicke proposed the concept of a sensory aphasia, characterized by impaired auditory comprehension in the context of generally fluent but often highly paraphasic expressive language production (M. L. Berthier, 1999; Eggert, 1977; Wernicke, 1874). Using the mortem lesion correlation approach, Wernicke linked the neural substrate of sensory aphasia (now called Wernicke's Aphasia) to middle and posterior portions of the superior and middle temporal gyri, regions now collectively referred to as Wernicke's area. Wernicke's localization of sensory aphasia in tandem with Broca's earlier localization of motor aphasia offered a substantive leap forward in our understanding of the instantiation of language within the human brain. Yet, his contribution went far beyond the localization of

sensory aphasia. Neuroscience owes Wernicke such a great debt because he pioneered a formal computational mechanism for mapping the various components of language onto the human brain. From a philosophy of science standpoint, Wernicke's proposal was revolutionary in that it afforded systems level hypothesis testing on a grand scale.

TCSA originally emerged out of the early connectionist models proposed by Wernicke, Lichtheim, and other prominent contemporaries (e.g., Kussmaul) (Roth & Heilman, 2000). The clear power of these models is that they could derive aphasia subtypes based on a fixed taxonomy. Thus the connectionist modeling approach offered a new and highly deductive way of generating a set of concrete predictions about specific behavior(s) related to focal brain injuries. Put simply, it was now possible to 'lesion' a computational model and look for confirmatory evidence from patients in contrast to the extraordinary limitations imposed by the inductive approach of observing behavior and attempting to derive a unified theory of brain (for bitter dissent see Berthier, 1999b; Head, 1926; Luria, 1974).

Importantly, many of the early connectionist models that originally gave rise to the syndrome of TCSA incorporated an assumption that portended the concept of a *disconnection syndrome*, later formalized by Dejerine (1891) and Geschwind (1965). Namely, many of the connectionist models offered contingencies for lesions to either impact a dedicated language center (e.g., Broca's or Wernicke's Area) or the major fiber tracts that join these centers. Goldstein (1948) offered the formal nomenclature for this *cortical-transcortical* distinction as a means for discriminating classes of aphasia characterized by either the former (e.g., cortical) or the latter (transcortical) type of damage. Consequently, within the classical aphasia taxonomy, TCSA was conceived very much like a disconnection syndrome despite the fact the formal terminology for such a syndrome had not yet been proposed. Figure 3 represents how a hypothetical lesion to the combined Wernicke-Lichtheim-Geschwind connectionist model would produce the unique behavioral profile of TCSA in addition to seven other aphasia subtypes.

-Figure 3-

In summary, as its name implies, TCSA reflects the historical confluence of two overarching dichotomies: 1) cortical-transcortical; and 2) sensory-motor. The term, *transcortical,* itself invokes the Latin prefix *trans-* (meaning across), thus denoting an aphasia

syndrome that results from disrupted communication *across* dedicated language centers (i.e., a disconnection syndrome). The second half of the name TCSA denotes primary impairment to afferent s*ensory* rather than efferent motor functions. Since its inception, TCSA has evolved in terms of specificity, and many formal aphasia batteries today include TCSA among their diagnostic classifications (Goodglass & Kaplan, 1983; Kertesz, 1982).

Clinical Criteria for TCSA

TCSA is characterized by severe deficits in auditory comprehension and language production in the context of preserved repetition ability (Goodglass & Kaplan, 1983; Kertesz, 1982). Within the schema of the Boston Diagnostic Examination of Aphasia (BDAE), TCSA is marked by well-articulated speech with appropriate prosodic contours and melodic line. However, the output of spontaneous language production is peppered by a combination of irrelevant semantic paraphasias (i.e, real word substitution errors) and neologisms (i.e., nonword errors). Naming and spontaneous narrative speech production in TCSA are often profoundly impaired, and these individuals typically experience comparable impairment for reading and writing (i.e., alexia and agraphia) (but for a unique subtype of TCSA without disturbance in these domains see Heilman, Rothi, McFarling, & Rottmann, 1981).

One remarkable feature that distinguishes TCSA from Wernicke's Aphasia is its associated pattern of preserved auditory repetition. That is, patients with TCSA often show accurate repetition ability for both words and nonwords. Moreover, echophenomena (e.g., echolalia) are commonly reported in TCSA, as is the selective preservation of islands of rote memorized material such as prayers and songs (Berthier, 1999a; Goodglass, 1993; Goodglass & Kaplan, 1983). Patients with TCSA often talk excessively (i.e., logorrhea) and show 'press of speech', a phenomenon characterized by speaking over others with great pressure and at a rapid rate. Moreover, these characteristics are usually amplified by anosognosia and poor self-monitoring abilities. In Henry Head's (1926, pp. 257-260) description of this phenomenon he remarked, "These patients tend to talk rapidly as if afraid of forgetting what they wanted to say; at times this actually occurs and the conversation tails away aimlessly" [as noted by Berthier (1999b)]. Head's comment has special relevance for the hypothesis we will soon attempt to

advance; namely, that aspects of the cognitive-linguistic impairment in TCSA result from the impact of amnestic and/or dysexecutive impairments on the maintenance of a memory trace, regardless of the representational modality of the particular trace (e.g., language or non-verbal).

Despite remarkably preserved repetition ability, there is strong evidence to suggest that much of what a patient with TCSA repeats he does not understand. Experimental evidence for this claim is derived from several sources, including both receptive and expressive language tasks. For example, patients with TCSA typically fail to self-correct semantic anomalies when repeating sentences but do in fact show sensitivity to and will tend to spontaneously correct grammatical violations (Berthier et al., 1991; Cimino-Knight, Hollingsworth, & Gonzalez Rothi, 2005; Martin, Saffran, & Pate, October, 1984). In addition to these unique patterns of self-correction in TCSA, repetition accuracy is often relatively immune to the manipulation of lexical-semantic variables such as word concreteness, imageability, frequency, age of acquisition, or even lexicality (i.e., whether the target item is a real word or nonword), whereas these individuals continue to show normal effects of word length and other phonological variables (Martin & Saffran, 1990).

In terms of classic dual route models of reading and repetition, one might account for TCSA repetition in terms of phonology as a dominant encoding strategy. That is, in the context of an impaired semantic contribution to recall, individuals with TCSA tend to quickly parrot information back to their interlocutor through an exclusive reliance upon form. Moreover, when memory span is taxed by presenting many items, individuals with TCSA often revert to a pattern of recall that is characterized by rapid forgetting of earlier presented items (i.e., lack of primacy effects) in the context of more accurate recall of the most recently presented items (i.e., recency effects), further suggestive of a pervasive lack of semantic support for recall (Martin & Saffran, 1990) and a reliance on activated phonological representations to repeat.

The Puzzling Neurology of Transcortical Sensory Aphasia: Site(s) of Lesion

TCSA is a prominent aphasia subtype in terms of its theoretical relevance. However, in the daily course of one's clinical practice, pure TCSA is a relatively rare phenomenon. In a large and well-characterized aphasia sample, Berthier and colleagues (1991) reported a prevalence of approximately 18% for all of the transcortical aphasias combined (i.e, motor, sensory, and

mixed) and noted that this estimate is convergent with other aphasia classification studies (e.g., Kertesz, 1982). The relative infrequency of TCSA can be attributed to its somewhat atypical lesion distribution. That is, although numerous neurological disorders can produce TCSA (e.g., neoplasm, Alzheimer's disease), stroke is the most common reported cause.

The most common site of vascular occlusion in stroke is the middle cerebral artery (MCA), which perfuses the basal ganglia and much of the perisylvian cortex (i.e., Broca's area, inferior precentral gyrus, Heschl's gyrus, Wernicke's Area) (Berthier, 1999). As such, left MCA stroke commonly produces the classical perisylvian aphasia subtypes. However, in its chronic form TCSA is classified as an extrasylvian syndrome, unique for its preservation of the classical perisylvian language regions. Damasio (1998) noted that the one of the most distinctive neuropathological features of TCSA is that it is rarely induced by a lesion that obliterates the whole of Wernicke's area but instead typically affects more rostral structures such as the temporo-parietal junction. Today the most common reported site of lesion for chronic TCSA is the temporoparietal junction (TPJ), whose gross anatomy is illustrated in Figure 4 (Alexander, Hiltbrunner, & Fischer, 1989; Berthier, 1999; Boatman et al., 2000; Damasio, 1998; Nadeau, Gonzalez Rothi, & Crosson, 2000; Otsuki et al., 1998; Sarno, 1998).

--Figure 4—

TCSA most commonly results from damage to the supramarginal and angular gryi and the posterior terminating portion of the superior temporal gyrus, areas sometimes classified as 'watershed' regions in terms of their distal proximity to the major arterial vasculature supplying the cortex (Damasio, 1998; Geschwind, Quadfasel, & Segarra, 1968; Goodglass & Kaplan, 1983; Roth & Heilman, 2000). Often the TPJ is impacted by extensive left hemisphere strokes whose associated global aphasia during the acute stage only later resolves into the more chronic form of TCSA. In most cases of acute stroke, however, TCSA tends to manifest in a transient form. Although the TPJ is the most commonly reported site of lesion, TCSA also emerges from lesions to other temporal lobe regions. Boatman et al. (2000), for example, reported eliciting the auditory profile of TCSA through direct cortical stimulation of the middle temporal gyrus among a group of patients undergoing pre-surgical cortical mapping for intractable epilepsy. Alexander et al. (1989) and Berthier et al. (1991) both reported cases of TCSA that occurred in the context

of posterior cerebral artery (PCA) strokes that impacted more ventral and posterior temporo-occipital regions just superior to the inferior temporal gyrus (i.e., visual association cortex) (see also Chertkow et al., 1997). A variety of other lesion correlation studies have implicated more anterior frontal and mesial structures, including the left prefrontal cortex (Berthier, 1999; Berthier et al., 1991; Cimino-Knight et al., 2005; Otsuki et al., 1998; Zahn et al., 2002), left basal ganglia (Crosson, 1992; Yamadori, Ohira, Seriu, & Ogura, 1984), and portions of the left midbrain (i.e., thalamus) (Alexander et al., 1989; Cappa & Vignolo, 1979; McFarling, Rothi, & Heilman, 1982).

Anatomical variability underlying TCSA creates a paradox that at first glance thwarts any principled attempt at localization. How is it possible that damage to a range of distant brain structures can produce an identical TCSA phenotype? [2] Perhaps the best hope for an answer to this anatomical riddle lies within network hypotheses. That is, TCSA results from damage to one or more components of a distributed but highly functionally-connected network dedicated to a specific cognitive function (e.g., sensorimotor feature representation, executive functioning). Using such systems-level logic, Alexander and colleagues (1989) hypothesized that TCSA reflects damage to a distributed network dedicated to feature representation in semantic memory, a controversial claim that we will soon revisit.

Proponents of the Hub & Spoke model have recently offered perhaps the most cogent solution to the riddle of brain-behavior localization TCSA, arguing that the TPJ and the prefrontal cortex (PFC) constitute two highly interactive components of the cortical network supporting EF and semantic control (see earlier discussion of process in semantic memory). Accordingly, damage to either a frontal or temporo-parietal distribution might produce a similar profile of dysexecutive impairment that deregulates semantic memory.

Semantic Impairment in Transcortical Sensory Aphasia: Lexical and Beyond

The question of whether TCSA impacts semantic memory and other global aspects of cognition and intelligence remains highly controversial. At the heart of this debate lies one of the most fundamental questions about the relationship between language and consciousness.

That is, do we think in words, or does language simply act as a blanket upon which abstract propositional thought is draped? The Roman emperor and stoic philosopher Marcus Aurelius (*AD 121-180*) remarked, "Your life is an expression of all your thoughts." If one were to agree with Aurelius, one must pursue the conclusion to its logical end and assume that TCSA fundamentally compromises abstract thought. Although this was indeed the position espoused by a number of highly influential figures such as Hughlings Jackson (1878), Luria (1974), and Goldstein (1948), it represents a departure from the mainstream position that TCSA reflects an immutable divorce between language and thought.

Today's dominant view holds that the semantic impairment in TCSA qualitatively differs in many respects from that of semantic dementia or Alzheimer's disease. Jefferies and Lambon Ralph (2006) conducted the first case study formally pitting TCSA against semantic dementia, concluding that the verbal semantic impairment in TCSA is marked by the following distinct characteristics: [3]

1) Insensitivity to word frequency/familiarity: Individuals with TCSA tend to manifest similar degrees of impairment for low frequency relative to high frequency words; for example, a patient with TCSA might experience an equal likelihood of erring on *pterodactyl* versus *alligator.* As a result, individuals with TCSA often show similar patterns of impairment across split halves of naming tests that are graded in difficulty by familiarity or frequency [e.g., Boston Naming Test/BNT (Kaplan, Goodglass, & Weintraub, 1983)]
2) Response inconsistency and poor inter-item correlations: Individuals with TCSA tend to show high variability in their response accuracy both across and within sessions. For example, test-retest consistency with respect to item-level accuracy tends to be weak in TCSA. In contrast, patients with semantic dementia or Alzheimer's Disease tend to show a strong pattern of consistency in their anomia across repeated trials, as well as a strong correlation between 'naming and knowing' that manifests as reduced semantic knowledge for items they cannot name (Hodges, 2003; Hodges, Graham, & Patterson, 1995; Hodges & Patterson, 1995; Hodges, Salmon, & Butters, 1992; Lambon Ralph et al., 1998; Lambon Ralph, Graham, Patterson, & Hodges, 1999). In TCSA, this correlation between successful naming and the quality of underlying semantic knowledge

is thought to be weak. Such inconsistency is also apparent when contrasting performance in verbal versus nonverbal tests of semantic knowledge, whereas a patient with semantic dementia typically shows comparable performance across different representational formats (Reilly et al., 2011; Reilly & Peelle, 2008).

3) Receptivity to cueing and priming : Individuals with TCSA often remain receptive and stimulable to cueing. For example, this is evident in progressively gating the phonemes of a target word (e.g., d, da, dak….), incrementally providing semantic detail (e.g., the person who you see at the hospital when you are sick), or asking the person to complete a cloze fragment (e.g., When you are sick you see a……). The benefit of cueing suggests that these individuals experience difficulties that can be ameliorated by increasing the salience of the target item or otherwise shifting task demands.
4) Associative naming errors: Individuals with TCSA tend to produce many associative naming errors in spontaneous speech (e.g., scissors → paper), whereas this pattern is less common in semantic dementia where category coordinate (e.g., cat →dog) or superordinate (e.g., cat →animal) naming errors predominate.

In addition to a prominent language disturbance, TCSA is also associated with a range of non-verbal semantic impairments. A sampling of these deficits, summarized in Table 1, includes difficulties in categorization of picture stimuli, matching pictures of gestures to tools, matching environmental sounds to pictures, and detecting absurdities in pictures. In addition, a range of naturalistic action deficits have also been reported in aphasia, including inability to pantomime gestures or use objects appropriately. Finally, others have reported even more paradoxical deficits in aphasia such as matching color to global visual form (e.g., coloring a banana yellow) and categorical visual perception of the cup-bowl distinction.

-Table 1 here-

The presence of a multimodal (verbal + nonverbal) semantic impairment in TCSA suggests disturbance of a latent factor that affects both language and cognition (see also Jackson, 1878). One might argue that such non-verbal semantic impairments offer the proverbial “slam dunk” against the Wernicke-Lichtheim-Geschwind account of TCSA as a language disconnection syndrome. Yet, it has been argued that many of the tasks used to elicit non-verbal semantic impairment in aphasia lack ecological validity and are highly confounded by task

demands (e.g., high EF demands and verbally mediated instructions). Further complicating matters is the fact that many studies that have reported nonverbal conceptual impairment have categorized individuals not in terms of a specific syndrome (e.g., TCSA) but by broader distinctions such as fluent-or-nonfluent, anterior-or-posterior, or dominant semantic-or-phonological impairment, thus, limiting inference both across and within studies (for treatment of this quandary see Chertkow et al., 1997).

Heterogeneity introduced by variable sites of lesion and TCSA subtypes lends further murkiness. This is especially apparent when considering studies that have collapsed the profiles of patients with posterior relative to middle cerebral artery strokes. That is, PCA and massive MCA strokes can both produce TCSA-like symptoms in the context of damage to posterior, inferior temporal and temporo-occiptal cortex (BA 37). However, these regions of the brain that comprise the visual association cortex are more commonly implicated in the semantic impairments seen in semantic dementia and Alzheimer's disease (Grossman et al., 2004; Mummery et al., 2000; Rosen et al., 2002). Some have argued accordingly that the extent of damage along the dorsal-ventral axis of the brain is strongly predictive of the severity of non-verbal semantic impairment (i.e., ventral=worse) (Antonucci, Beeson, Labiner, & Rapcsak, 2007; Antonucci, Beeson, & Rapcsak, 2004; Chertkow et al., 1997). Chertkow and colleagues (1997), for example, found that individuals with fluent aphasia clustered into two groups with respect to verbal-nonverbal impairment. Those with damage to the TPJ (the dorsal group) showed an isolated verbal comprehension deficit with preserved performance on a picture variation of the same task (i.e., matching a picture of a lemon to teacup or coffee mug). In contrast, individuals with posterior and inferior temporal lobe damage (the ventral group) showed comparable impairment for both words and pictures (but see Jefferies & Lambon Ralph, 2006).

In summary, a gestalt of TCSA is emerging that paints the syndrome as only truly "semantic" (i.e., satisfies criteria for multimodal storage impairment) in the context of an inferior and posterior distribution of temporal lobe damage. On a related note, Berthier (1999b) has described semantic dementia as a form of TCSA due to its profile of preserved repetition and impaired comprehension. Although semantic dementia does technically satisfy broad criteria for TCSA, its designation as a form of TCSA has not met with widespread acceptance. Rather,

many emphasize that the canonical profile of TCSA results from a more superior distribution of TPJ and/or frontal subcortical involvement and that TCSA exclusively represents a form of stroke aphasia (Corbett et al., 2011; Geschwind, 1965; Geschwind et al., 1968; Hoffman et al., 2009; Noonan et al., 2010). Thus, most argue that TCSA and semantic dementia offer orthogonal semantic impairments that are subject to comparison but not combination/collapsing.

For the sake of argument, let's suspend disbelief for a moment and ignore the frontal, midbrain, and inferior temporal lobe manifestations of TCSA and assume that the TPJ is the primary site of lesion for TCSA. This assumption might at the very least yield a logical starting point for delineating the semantic impairment in TCSA. Yet, this reductionist approach of focusing on the TPJ is riddled with problems in that damage to the inferior parietal lobe, in addition to causing aphasia, is also commonly associated with a constellation of non-linguistic impairments that impact semantic memory (or at least tests thereof). Parietal dysfunction commonly produces difficulties in constructional praxis, spatial working memory, and the four classic symptoms of Gerstmann's Syndrome (i.e., dyscalculia, finger agnosia, dysgraphia, and right-left directional confusion) (Gold, Adair, Jacobs, & Heilman, 1995). One can imagine how a combination of aphasia and Gerstmann's syndrome can potentially interact to degrade performance on a variety of cognitive measures that assess both verbal and nonverbal semantic knowledge (and more general fluid intelligence) (Hamsher, 1998). Such effects were apparent in early work correlating TPJ damage with poor performance on the Army General Classification Test (AGCT), a measure of purported general intelligence whose three components include vocabulary, arithmetical reasoning, and block counting (Weinstein & Teuber, 1957).

Summary of Semantic Impairment in TCSA

We have conveyed a picture of great complexity with respect to semantic impairment in TCSA. Yet, we have also highlighted some recurrent themes and offered an interpretative theoretical framework. To reiterate, this hybrid theory of semantic memory is premised upon multiple convergence zones and sparse representation (see earlier discussion). Within the context of this model, we view TCSA as a primary impairment of semantic *process*. Neurological damage to either a high level semantic convergence zone within the TPJ (e.g., angular gyrus) or a lower level sensory convergence zone within midbrain (e.g., thalamus) compromises the ability to integrate and effectively package distributed feature knowledge into

cohesive wholes. Importantly, this loss of semantic convergence is likely to impact both online perception and remote reconstructive processes (i.e., perceptual enactment and mental imagery) that are critical for enriching object knowledge.

As aphasiologists, in our daily clinical practice we do not often associate frank semantic errors such as pouring bleach into one's spaghetti sauce as representative of TCSA. Yet, such errors are commonplace in semantic dementia and Alzheimer's Disease (Bozeat et al., 2003; Bozeat, Ralph, Patterson, & Hodges, 2002; Coccia et al., 2004; Funnell, 2001; Hodges, Bozeat, Lambon Ralph, Patterson, & Spatt, 2000; Patterson, 2007; Pulvermuller et al., 2009). One potential explanation for this aphasia-dementia discrepancy regards redundancy in the organization of convergence zones across the human brain. That is, a number of potential semantic convergence zones have been identified bilaterally across the cortex (e.g., temporopolar cortex, superior temporal sulcus, middle temporal gyrus, perirhinal cortex) (Binder et al., 2009; Damasio & Damasio, 1994; Damasio, Grabowski, Tranel, Hichwa, & Damasio, 1996; Davies, Graham, Xuereb, Williams, & Hodges, 2004; Ding, Van Hoesen, Cassell, & Poremba, 2009; Turken & Dronkers, 2011). It has also been hypothesized that semantic convergence occurs in a series of successive hierarchical steps, beginning with lower order sensory conjunctions ascending to higher order perceptual and linguistic crossmodal integration processes (see also conceptual structure account and related work by Bussey & Saksida, 2002; Tyler & Moss, 2001; Tyler, Moss, Durrant-Peatfield, & Levy, 2000). The TPJ in general and the angular gyrus in particular have been identified as sites of higher order semantic convergence (Binder et al., 2009; Ramachandran & Hubbard, 2001). Yet, the TPJ does not appear to be the only site of semantic convergence in the human brain, nor does it appear to be the most critical relative to more inferior temporal lobe regions compromised in semantic dementia.

When comparing the semantic impairment in TCSA to that of semantic dementia or Alzheimer's Disease there are additional considerations. The most common brain injury that produces TCSA is stroke, and with rare exceptions, manifests as unilateral brain injury. Thus, patients who incur TCSA in the context of a left hemisphere TPJ infarct typically retain their right hemisphere TPJ homologue. This affords the possibility of a right hemisphere contribution to semantic memory in addition to the possibility for post-stroke functional re-organization (see also M L Berthier, 2001). In addition, the nature of stroke is also such that beyond the acute

stage, cognition is typically either static or improving. The same cannot be said for neurocognitive disorders such as semantic dementia and Alzheimer's disease that both manifest in a progressive and bilateral form (Galton et al., 2001; Hodges et al., 1992; Lambon Ralph et al., 2011).

It is undeniable that some aspects of the semantic impairment in TCSA are related to executive resource limitations. However, the nature of the executive resource pool remains a question of intense debate. One possibility espoused by Hub & Spoke Model proponents is that the semantic impairment in TCSA is driven by a subset of domain-neutral EF capabilities (Hoffman et al., 2009; Hoffman, Jefferies, Ehsan, Jones, & Lambon Ralph, 2012; Hoffman, Jefferies, & Lambon Ralph, 2011; Jefferies & Lambon Ralph, 2006; Noonan et al., 2010). An alternative view holds that semantic memory is subserved by a modular and at least partially dissociable semantic working memory system from that of general EF (Martin & Saffran, 1990, 1992). This hypothesis is supported by the presence of weak or null correlations between impaired EF and semantic processing in many neuropsychologically impaired populations (e.g., traumatic brain injury, Parkinson's Disease) (but see Baldo et al., 2005), as well as the absence of prominent dual task EF-semantic costs in healthy normal adults.

We have offered a point of departure from the classical disconnection account of TCSA and posed the alternative hypothesis that many associated difficulties in TCSA are rooted in semantic process. The contention that a latent, supra-linguistic cognitive impairment lurks quietly within TCSA is by no means new (Goldstein, 1948; Head, 1926; Jackson, 1878; Luria, 1974). Yet, the specific mechanism of impairment that we have proposed here (i.e., multiple convergence zone + sparse representation) is in many respects novel. Although any incipient theory should be met with a healthy dose of skepticism, there are many reasons to consider a semantic process-based approach to TCSA. This perspective has the potential to elucidate our theoretical understanding of language and conceptual organization while also offering practical and innovative considerations for language rehabilitation.

Clinical Implications

In its chronic form, TCSA is a rare but severe and highly debilitating form of aphasia that is often resistant to behavioral treatment. Standard therapies for semantically-based language disorders such as TCSA tend to focus heavily on retraining semantic features and semantic category structure. Semantic feature analysis (SFA) is a prime example of a feature-based therapy that operates through a paired associate learning approach wherein individuals with aphasia repeatedly generate multimodal semantic features in an effort to link conceptual salience with an object's name (Antonucci, 2009; Boyle, 2004). Another popular approach to semantic treatment (i.e., typicality) involves capitalizing on the nature of semantic category structure through training atypical exemplars (e.g., *penguin*) with the goal of re-establishing cohesion, breadth, and central tendencies/prototypes (e.g. *robins*) within weakened semantic networks (Kiran & Thompson, 2003).

SFA and typicality treatments are heavily driven by semantic content. Although the purpose of these therapies may be to strengthen connections within a network of semantic content, they do this by manipulating content of treatment tasks. We have argued for a process account of TCSA that involves poor feature binding and retrieval. An approach to language treatment that shifts the focus from semantic content to process offers unique direction for aphasia therapy. That is, semantic impairment in TCSA might paradoxically be ameliorated by selective training on an EF component such as inhibitory control, or a perceptual process such audiovisual crossmodal integration (e.g., pairing tones with colors, or environmental sounds with images). One possible solution is to forego treatment specificity and employ a "kitchen sink" approach that simultaneously addresses many general domains of EF, whereas a more principled alternative might involve isolating and rehabilitating one or more specific component(s) of EF (e.g., inhibitory control but not sustained attention). Both of these EF-driven approaches pose individual strengths but also share a common pitfall. That is, if semantic memory is subserved by its own modular processing system, one would predict a minimal therapeutic benefit from training general EF. This remains an important unanswered empirical question with respect to structuring semantic treatments for both TCSA and many other forms of semantic aphasia.

A third approach that could potentially address semantic process impairments is to manipulate executive function demands in the context of treatment tasks. We have recently employed such an approach toward treatment of a person with conduction aphasia (Kalinyak-

Fliszar, Kohen, & Martin, 2011) and two others, one with Wernicke's aphasia (Martin, Kohen, McCluskey, Kalinyak-Fliszar, & Gruberg, May, 2009) and one with anomia (Kalinyak-Fliszar, Kohen & Martin, 2012). In each of these cases, the treatment task was repetition of words or nonwords under three conditions that gradually increased the memory and executive function load of the task: immediate repetition, repetition after 5 seconds and repetition after naming aloud numbers on a screen for 5 seconds. The second condition increased memory load on simple repetition and the third condition increased STM and executive load. It is the claim of Martin and colleagues that this treatment stimulates fundamental processes that drive access to and maintain activation of semantic and phonological representations of words. Consistent with this hypothesis, they observed in all three cases improvement on the training task (repetition) as well as improvement on other language tasks (e.g., phoneme discrimination, rhyming, verbal span tasks). To use this treatment approach in cases of TCSA, the treatment task would have to be changed to a task which stimulates pathways between lexical and semantic representations (input and output). For example, to promote comprehension of words, word-to-picture matching tasks could be used and STM and executive loads could be manipulated as described for repetition. Whether this treatment would be effective with TCSA remains to be seen. However, the preliminary success of these few treatments that incorporate STM/executive load on other aphasias are promising.

Concluding Remarks

TCSA has recently provided both a compelling and controversial lesion model for parsing the structure of semantic memory. Moreover, many theories of semantic organization now incorporate active processing components and recognize that semantic memory is a dynamic system. It is now apparent that we must reconsider Damasio's (1992) claim that little new has been uncovered regarding TCSA. Yet, it is also clear that much remains to be learned about the nature of this unique disorder and ways to address the considerable disability posed for these individuals with TCSA.

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Endnotes

1. When referring to *Proponents of the Hub & Spoke Model*, we are collectively describing the combined efforts of a highly talented group of affiliated scientists, including Karalyn Patterson, Matthew Lambon Ralph, Elizabeth Jefferies, Paul Hoffman, Timothy Rodgers, John Hodges, and their many collaborators.

2. This statement might not be entirely accurate. Many researchers have argued that sensitive testing can reveal distinct TCSA subtypes (Coslett, Roeltgen, Gonzalez Rothi, & Heilman, 1987; Heilman et al., 1981).

3. Warrington and Shallice have proposed formal criteria by which disorders of access versus storage could be discriminated (Shallice, 1988; Warrington & Crutch, 2004; Warrington & McCarthy, 1983). The criteria for a semantic access impairment are as follows: 1) preserved facilitative effects of priming and cueing; 2) response inconsistency across sessions; 3) insensitivity to word frequency; 4) facilitation from superordinate category exemplars; 5) sensitivity to rate of presentation (i.e., see discussions of semantic refractory access disorders. In contrast, the criteria for a semantic storage disorder include: 1) negligible priming and cueing effects; 2) consistency in task performance across repeated sessions; 3) sensitivity to word frequency and object familiarity; 4) retention of superordinate detail (e.g., ‘animal’ but not ‘Labrador’).

Table 1. A sample of studies reporting non-verbal semantic impairment in non-progressive manifestations of aphasia (e.g., stroke, focal neurological insult)

Impaired Nonverbal Domain	References

Semantic association ability for pictures • Picture Subtest: Pyramids and Palm Trees Test(3-choice) • Picture Subtest: Camels & Cactus Test (4-choice) • Judging best fit for picture triads (e.g., lemon: coffee pot or teapot)	Martin et al., (2006) †Jefferies & Lambon Ralph, (2006) Chertkow et al., (1997)
Categorization/category sorting of pictures • Sorting pictures by intersections of different perceptual features (e.g., canary=yellow+bird)	Gainotti et al. (1986)
Non-verbal abstract problem solving and EF • Raven's Progressive Matrices Test • Wisconsin Card Sorting Test	Baldo et al. (2005) Hoffman et al., (2009)
Naturalistic action deficits • Matching pictures along a common dimension of action, function, or use (e.g., <twisting> corkscrew-to-screwdriver; <cutting> knife-to-scissors; <canonical recipient of an action> hammer-to-nail • Spontaneous manipulation ability for actual tools during tactile handling [e.g., scissors → grasp, movement, and orientation] • Comprehension, pantomime, and matching of symbolic gestures to pictures (e.g., gestured guitar playing match to guitar)	†Corbett et al., 2009 †Corbett et al., 2009 Gainotti & Lemmo (1976)
Matching color to global visual form via spontaneous drawing • Coloring line drawings of common objects	De Renzi et al. (1972)
Figure drawing deficits • Figure reproduction upon delay • Reduced specificity/omission of features among freehand line drawings of natural	Gainotti (1983) Grossman (1993)
Higher level visual deficits in the absence of a frank apperceptive agnosia • Cup/Bowl Categorical Perception: As a cup flattens and its diameter	Caramazza et al. (1982)

simultaneously expands, there reaches a critical threshold where most people perceive it a bowl (Labov, 1973). This study reported a threshold shift in aphasia.	
Detection of "absurdities" in pictures via gesture/pointing	Bay (1962)
Environmental sound perception • Matching common environmental sounds to their respective picture referents via pointing	†Jefferies & Lambon Ralph (2006) Vignolo (1982) Saygin et al. (2003)

Note: † signifies that the particular study isolated TCSA; Pyramids and Palm Trees Test (Howard & Patterson, 1992); Camels & Cactus Test (Bozeat, Lambon Ralph, Patterson, Garrard, & Hodges, 2000); Raven's Coloured Progressive Matrices Test (Raven, 1962); Wisconsin Card Sorting Test (Heaton, Chelune, Talley, Kay, & Curtis, 1993)

Figure Headings and Captions

Figure 1. Schematic of Allport's (1985) distributed semantic memory model

Figure 2. Depictions of a) Potter's (1993) model of conceptual short-term memory (STM); and b) Dell and colleagues' interactive activation model of word processing

Note: In Figure 1a, auditory input (e.g., a spoken sentence) is perceived and simultaneously processed through phonological STM and also conceptual STM. It can be recalled verbatim via the phonological STM (with rehearsal support via the articulatory loop) and can also be recalled by regeneration via the interaction of conceptual STM and LTM. Figure 1b depicts Dell's interactive spreading activation model (e.g., Dell & O'Seaghdha, 1992). Word production begins the activation spreading from conceptual knowledge to specific semantic features of a concept (e.g., tactile, visual, auditory features) which in turn activates the target word form in the lexicon and to a lesser extent other word forms that share some of those features. Activation from the word forms spreads again to the phonemes that comprise the activated word forms. The model is "interactive" because the activation spreading forward through each stage of linguistic representation sends feedback to the previous stage. This interactivity continues until a word form is selected for production. In a second stage (not depicted), the selected lexical form is phonologically encoded and eventually articulated.

Figure 3. Depiction of Wernicke-Lichtheim-Geschwind Model and superposition on human brain

Note: Abbreviations retained from Lichtheim's orginal schema include: M (Motor Center corresponding to Broca's Area); B (Concept Center corresponding to what Gechwind and others loosely localized to the supramarginal and angular gyri); A (Auditory Word Form Center localized to Wernicke's area); a (auditory afferent pathway marking input to the language network); m (motor speech peripheral musculature, efferent output pathway). The model predicts the presence of seven distinct classical aphasia subtypes, numbered sequentially above and described to follow. 1) Damage m → M compromises the transmission of high level motor programs to the speech musculature resulting in a primary articulatory disorder; 2) Damage to M produces the classic profile of Broca's Aphasia; 3) Damage to the B → M pathway disconnects the putative concept and motor planning centers, resulting in the profile of transcortical motor aphasia (TCMA); 4) Damage to the major tract (i.e., arcuate fasiculus) joining Broca's and Wernicke's areas impairs bidirectional communication between A ↔ M resulting in conduction aphasia; 5) Damage to the pathway joining the putative concept center (B) with the center for auditory word forms (A) results in the disconnection of words from concepts that is the classic hallmark of TCSA; 6) Damage to A produces Wernicke's Aphasia; 7) Damage to the a→A pathway produces pure word deafness.

Figure 4. Gross Anatomy of the temporoparietal junction (TPJ)

Note: Landmarks and abbreviations were adapted from the neuroanatomical atlas of Hanna Damasio (2005) superimposed upon a lateral rendering of a single subject from the ch2better.nii

brain template from MRIcron (Rorden, 2007): Anatomical abbreviation key: **AG**=Angular Gyrus; **HG**= Heschl's Gyrus; **IPL**=Inferior Parietal Lobule; **LOG**=Lateral occipital gyrus; **postCG**= Post Central Gyrus; **pMTG**=Posterior Middle Temporal Gyrus; pSTG=Posterior Superior Temporal Gyrus; **SF=**Sylvian Fissure; **SMG=**Supramarginal Gyrus; **STS**=Superior Temporal Sulcus