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Cognitive Neuropsychology Has Been, Is, And Will Be Significant To Aphasiology

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Abstract

Background—In recent years, some critical voices have been raised in regard to the significance of cognitive neuropsychology (CNP) to the study of brain and mind. Given the central role of language disorders in CNP research, it is time to consider the relevance of this research approach in aphasiology.

Aims—We analyze the main points of criticism raised against the CNP research approach, evaluate the significance of this approach to the study of acquired language disorders, and make some suggestions concerning further development of the field.

Main Contribution—The main points of criticism against CNP (reliance on single-case studies; single-minded hunt for dissociations; emptiness of theorizing) have been important long-term concerns but do not take into account the fact that during its history of circa four decades, the CNP approach has diversified. There are thus CNP studies that rely on case series analyses, focus on error analyses rather than mere dissociations, or employ computational modeling rather than the “boxes-and-arrows” models of the mental architecture. The CNP approach to cognition and its disorders is thus applicable to different research questions and theoretical stances, providing experimental rigor to single-case patient studies. With regard to clinical applications in aphasia diagnostics and treatment, the CNP approach provides a richer view on the strengths and weaknesses of a patient’s cognitive-linguistic abilities.

Conclusions—We believe that CNP case studies continue to be an important source of information for generating hypotheses and providing converging evidence for research on the mind and on the brain. There is however a need for further research development especially in computational modeling of language processes, their impairments, and recovery. This research is expected to provide further benefit to clinical diagnostics and treatment of aphasia.

Keywords

cognitive neuropsychology; single-case study; aphasiology

Introduction

The present target paper was prompted by recent discussions of the scientific import of cognitive neuropsychology (CNP) (Harley, 2004a, b; Patterson & Plaut, 2009, 2010; Coltheart, 2010) as well as general trends in brain and mind sciences that may create the

impression that the CNP approach is somewhat obsolete. The CNP approach, as its name implies, embraces both cognition and the brain by linking the two via associations of neural damage and residual patterns of spared and impaired cognitive abilities. However, current paradigms in cognitive neuroscience depart from the original patient study approach advanced by CNP in two directions. At the methodological level, advances in neuroimaging technology have led to an explosion of studies on the neural substrates of cognitive processes in the normal brain. At the theoretical level, emphasis on non-symbolic (distributed connectionist) models of cognition deviates from the reliance on “boxes-and-arrows” models that typically have been associated with the CNP approach. In light of these developments, it appears timely to discuss the role of CNP in the field of aphasiology, as the cognitive architecture of language has been a prime target for the CNP approach.

What is CNP? In a review chapter, Max Coltheart (2001) defines CNP as a study of the nature of cognitive processes through data from people who suffer from either developmental or acquired disorders of cognition. As in cognitive psychology, CNP attempts to unveil the inner workings of functions such as memory, perception, language, and action control. A necessary assumption here is that the data from the study of impaired cognitive functions are useful to support or falsify theories of normal cognition. As compared with mainstream cognitive psychology, the defining feature of the CNP approach is thus its primary data source, people with disordered brains. As compared to neuropsychology and behavioral neurology, CNP differs in its focus: the former disciplines are primarily interested in brain-behavior relationships while CNP uses study outcomes to test and to build models of cognition. Likewise, CNP is not necessarily concerned with treatment of cognitive deficits, albeit evidence from this approach can be relevant for treatment purposes. Coltheart's definition thus positions CNP as a subfield of cognitive psychology, apart from the study of the neural underpinnings of cognition. This characterization of the CNP as separate from issues related to brain structure and function is also known as “ultra-CNP” and has not been shared by all researchers in the field (e.g., Shallice, 1988). Nevertheless, one can broadly define the CNP approach with three key features: its domain of study (inner workings of cognition), sources of data (people with developmental or acquired disorders of cognition), and methodology (systematic implementation of quantitative case study methods).

The CNP approach has its roots in the long history of neurology and psychology, but it did not emerge as an autonomous field until the 1970s and 1980s when its pioneers (e.g., Marshall & Newcombe, 1973; Warrington & Shallice, 1979; Saffran, 1982; Caramazza, 1986; Schwartz, 1987), often with a background in experimental psychology, neuropsychology, and/or psycholinguistics, began to systematically use single-patient behavioral data to test and develop models of the functional architecture of normal cognition (Selnes, 2001). The emergence of CNP paralleled more general trends in cognitive research, such as the development of information-processing models. Right from the start, CNP research focused on the study of the functional architecture of language through its disorders, with particular emphasis on (single-word) reading (e.g., Marshall & Newcombe, 1973; Saffran & Marin 1977; Warrington & Shallice 1979; Coltheart, Patterson & Marshall, 1980). The CNP approach has subsequently been applied to other cognitive functions such as spoken language (still predominantly at the single-word level), memory (especially semantic memory and working memory), visuospatial processing (object and face recognition, in particular), praxis, and attention. Even certain neuropsychiatric conditions have been tackled by this approach, and there is a dedicated journal (Cognitive Neuropsychiatry) for such studies. However, language remains a key research area in CNP, probably because it provides such a rich knowledge base in terms of available models and findings. In addition, language is the uniquely human capacity that offers an explicit means

for assessing a variety of related cognitive processes, including perception and semantic memory.

Albeit lesion studies in general are cited much less often than functional neuroimaging studies (Fellows et al., 2005), CNP research has developed visibility in the last few decades through the prominent role of theoretically relevant case studies in some cognitive psychology textbooks (e.g., Groome, 1999, 2006; Baddeley et al., 2009) and via the publication of CNP case reports in general science journals (e.g., Marslen-Wilson & Tyler, 1997; Caramazza et al., 2000). In addition, the CNP approach to the study of language disorders has also made its way into the clinic through diagnostic test resources such as the PALPA (Kay, Lesser & Coltheart, 1992) that seeks to identify the functionality of various information-processing units of the language system in a language-impaired patient. Moreover, it has been applied to aphasia treatment studies (see Whitworth, Webster & Howard, 2005, for review). Being a seemingly well-established field of study, it may come as a surprise that CNP has recently attracted strong criticism questioning its impact and significance to the study of cognition. As regards aphasiology, the critical questions are as follows: concerning the past, present, and future, is the CNP approach actually making any significant contribution to understanding language impairment, diagnosing language disorders and developing appropriate treatment methods?

The present paper is organized as follows. First, we briefly describe the recent criticisms leveled against CNP research and our own take on these issues. This is followed by some methodological considerations that pertain to the single-case study approach in CNP. Then we provide the reader with one concrete example on the use of the CNP approach in the study of language and memory systems and discuss the relevance of this approach to clinical work in aphasiology. Finally, we draw conclusions on the theoretical and clinical significance of CNP and consider some future directions for this approach.

Challenges to CNP research

In a section coined as *Visions in Cognitive Science*, the journal *Topics in Cognitive Science* recently published a provocative paper by Patterson and Plaut (2009) that questioned the relevance of CNP. They argued that misguided choices in data collection, methodology, and theorizing have led CNP into a sidetrack that has reduced its relevance to mainstream cognitive science. The key points of their criticisms are summarized below, in order of importance from least to most as defined by Patterson and Plaut (op.cit.). They also point out that many aspects of this criticism have been raised earlier (see e.g. Seidenberg, 1988).

(i) An almost complete reliance on single-case studies

Patterson and Plaut (2009) argue that the reliance on single cases in CNP is problematic because it cannot be assumed that the functional organization in all brains is identical. This limits the conclusions that can be drawn from single case studies about the organization of the cognitive system in the general population. Moreover, they note that individuals with similar underlying deficits could employ very different response strategies (speed-accuracy tradeoffs) that would complicate the interpretation of the results if only a single case is available. Patterson and Plaut (2009) also point out that in other disciplines employing very small n's (e.g., neuroscientific experiments on animals, psychophysical experiments in humans), the data set typically includes at least 2–3 participants. Accordingly, Patterson & Plaut (2009) call for the use of the case-series approach where several patients with the same core deficit are analyzed and reported.

(ii) An almost universal goal of establishing dissociations

The second concern raised by Patterson and Plaut (2009) deals with the emphasis on double dissociations in CNP. Finding two patients who show contrasting single dissociations in two functions (patient 1: function A impaired but function B spared; patient 2: function A spared but function B impaired) would imply that somewhere along the processing chain, these two functions rely on separate mechanisms. As a consequence of the strong reliance on double dissociations in CNP, "A hundred or a thousand patients showing an association [...] would not offset even one double dissociation" (Patterson & Plaut, 2009, p. 44). Patterson and Plaut question the validity of a double dissociation as evidence about the organization of cognition and cite other researchers who have raised concerns about the validity of this method (Seidenberg, 1988; Goldberg, 1995). They also note that the plasticity of the human brain following damage may be such that the performance pattern of a single patient may be partly based on qualitatively different processes that would not be informative about the mental organization of normal function.

(iii) Emptiness of theorizing

In the words of Patterson and Plaut (2009), this means "theorizing that consists almost entirely of descriptive, box-and-arrow diagrams with little, indeed sometimes no attempt to specify actual mechanisms of the putative processes" (p. 42). Again, they cite Seidenberg (1988) who voiced this concern over 20 years ago. Seidenberg (1988) considered the primary goal of the CNP approach, localizing a deficit within a (weakly defined) functional architecture, as misguided. Rather, one should strive towards specifying the knowledge representations and processing mechanisms involved. Of the three points of criticism presented by Patterson and Plaut (2009), they consider this as the most serious one.

In what follows, we offer our own take on these three points of criticism. For further discussion, the reader is referred to Patterson and Plaut (2009), Coltheart's commentary (Coltheart, 2010), and the reply (Patterson & Plaut, 2010).

To comment on the first point, it is true that for several reasons, drawing inferences about cognitive function from a single case is not totally straightforward. Some cases with acquired deficits may by chance exhibit some unusual features in premorbid functional brain organization that the researcher is unaware of but that can affect their post-injury performance profile. Additionally, an individual may employ unique compensatory mechanisms to overcome cognitive deficits, particularly at a chronic stage following brain injury. The complexities of interpreting data from single case studies underscore the need for converging evidence from other data sources (additional patients, normal controls)ⁱ. Reporting case series is not always possible either, as patients showing a certain performance pattern may be rare. As an example, during his career as a clinical neuropsychologist, the first author administered diagnostic tests on roughly 3,000 adult neurological patients and found only three cases with deep dyslexia over the years. The applicability of the case series approach that indeed has been used in CNP (e.g., Martin, Shelton & Yaffee, 1994; Martin & Saffran, 1997, 1999; Lambon Ralph, Patterson, Garrard, & Hodges, 2003) is related to the level of specificity in defining the core deficit. For

ⁱThe weight of a single case is of course related to the strength of the empirical findings and to the potentially groundbreaking value of the theoretical implications the results carry. As was noted in the Editorial of *Nature Neuroscience* (2004), "One talking pig is sufficient to prove that pigs can talk". Even if the genetic and neural features of such a pig (if it would exist) would turn out to be somewhat different from ordinary pigs and thus not generalizable to the whole population of pigs, there is no doubt that the existence of such an animal would be a truly revolutionary finding. To take more realistic examples, there are single cases of brain-damaged humans such as Phineas Gage who suffered a severe frontal lesion, Paul Broca's aphasia patient Leborgne (subsequently confirmed by Broca with another case, Lelong), and the amnesic patient H.M. that have had a lasting impact in science. See Code, Wallesch, Lecours, & Joannette (1996; 2003) for discussion of these cases.

example, it would be easier to compile a case series of patients with a semantic deficit than of patients with a deficit in a specific semantic category. Nevertheless, the incremental nature of scientific inquiry should lead to the accumulation of studies of many rare disorders, enabling meta-analyses and evaluation of the coherence of the reported findings. Meanwhile, the inability to replicate the findings of a rare case study by other researchers places special demands on the quality of single case study reports. For example, it is necessary to include standard background information (demographic, neurological, neuroradiological, neuropsychological), provide primary data on the main tasks, and include within-case replications of the critical behavioral phenomena whenever possible.

As regards the second point, the dissociation method has been instrumental in the creation of modular models of the functional architecture of the mind in CNP, but it is true that as such it reveals little about the dynamics of these modules (e.g., McCloskey, 2001). It seems to us that the potential of strong dissociations to challenge existing models of cognition (not only of the boxes-and-arrows type) and thus advance our knowledge on the mental architecture of the underlying cognitive machinery has not vanished, but there are a number of issues that need to be considered. First, performance dissociations must be carefully verified. The critical tasks used to reveal a dissociation should be reliable and sensitive enough, and the dissociation should be statistically supported. It would also be preferable to replicate the dissociation within the case. Second, dissociations must be explicitly related to relevant current empirical and theoretical work. In other words, one must carefully consider the convergence of the results with previous evidence and the extent to which the results conform or challenge current models of cognition and potentially lead to new insights and interesting testable hypotheses that could be applied to normal individuals. Third, it is important to consider alternative accounts for the dissociations. These include dissociations related to compensatory changes in processing strategies, different performance/resource curves for the critical tasks that could account for a double dissociation (see Shallice, 1988, fig. 10.5., p. 234), and connectionist accounts where a double dissociation could arise even from a single distributed network (e.g., Plaut, 1995; Plunkett & Bandelow, 2006). This is a long list of issues to consider, highlighting the fact that the logic of double dissociations, as any method of inference, is open to several interpretations (see also Davies, 2010). Whilst the significance of dissociations has been emphasized in the context of modular models of the mind, it is important to note that they provide an opportunity to set any relevant model to test. Moreover, we agree with Patterson and Plaut (2009) that *associations* between different task performances in patients also give potentially valuable data, albeit mere task associations do not necessarily evidence a functional relationship because of possible spurious correlations where the relationship between tasks A and B within patients depends on a third, confounding factor such as anatomical proximity of the neural substrates needed for these tasks. However, experimental setups where a patient's performance on a specific language task is shown to be sensitive to parametric manipulations of certain stimulus-or task-related factors can provide evidence for a functional relationship between the independent and the dependent variable, inform about the inner dynamics of the cognitive system, and lead to further testable hypotheses.

Concerning the emptiness of theorizing in CNP, it is interesting to note that Patterson and Plaut (2009) extend this critique to much of the current research using functional neuroimaging in normal participants. At the same time, they remind us that it is ill advised to generalize the faults of a research approach to the entire CNP research community. There are high-quality studies that are carefully conducted and exert caution in their conclusions. We share the concern expressed by Patterson and Plaut (2009) and others (Harley, 2004a) for the need to implement computational models in CNP studies to a larger extent than has been done thus far. Importantly, the CNP approach should not be linked exclusively with cognitive 'boxology' that is becoming somewhat outdated (see below for an example of

more dynamic computational single-case studies in aphasia). We would also like to highlight the methodological import of the CNP approach that provides experimental rigor to single-case patient studies that address the inner workings of human cognition. These include features familiar from true experiments: a principled theory-driven examination of the patient's relevant cognitive abilities, close attention to stimulus selection (e.g., number of items and their psycholinguistic features), systematic manipulation of stimulus or task features (e.g., frequency, length, phonological structure, semantic contents, working memory load), adequate stimulus presentation (e.g., controlling for stimulus order effects), careful consideration of possible confounds, and the use of statistical methods in the analysis of results. However, this does not mean that we are attempting to reduce CNP simply to a methodologically sound case study: the goal of CNP is to advance our understanding of the structure and dynamics of the cognitive system, its disorders, and (depending on the researcher's focus) to shed light on the neural underpinnings of cognition. Instead, what we want to emphasize is that empirical evidence on human cognition provided by methodologically sound CNP studies is valuable for fundamentally different theoretical enterprises, not just for a single view on mental architecture that, e.g., the boxes-and-arrows models represent.

An important aspect of methodological rigor in quantitative research is the careful use of the statistical toolbox for analyzing and interpreting empirical findings. Unfortunately, the development of approaches to statistical analysis of single-case studies is still in its early stages: even a cursory look at statistics textbooks in psychology and social sciences shows that they almost solely deal with group studies. Accordingly, the statistical methods commonly used to determine deficits and dissociations in single patients have been far from optimal. As a case in point, Laws, Gale, Leeson & Crawford (2005) recently demonstrated how some commonly used methods (z-score comparison to controls; within-patient comparison by the Chi-square test) provide misleading results in the analysis of category-specific semantic disorders in patients with Alzheimer's disease. Thanks to recent work mainly by John Crawford and his colleagues, better statistical methods are now available. Among others, these include a modified t-test to determine the existence of a performance deficit in a patient as compared to a control sample (Crawford & Howell, 1998) and the Revised Standardized Difference Test for testing the difference between a patient's performance on two tasks (Crawford & Garthwaite, 2005). When put together, these methods provide clearly defined criteria for the existence of performance dissociation in a single patient while keeping Type I error rates under control. Furthermore, methods for statistical inference for comparing scores from two cases have been developed (Crawford, Garthwaite & Wood, 2010). For further information, the reader is referred to the following website: <http://www.abdn.ac.uk/~psy086/dept/SingleCaseMethodology.htm>.

CNP case study as a heuristic tool: an example

Drawing upon the research of the second author and her colleagues, we take up one concrete example of a CNP single-case study. We think that this case has furthered the understanding of the mental structure and dynamics of specific language functions and their impairment, and thus serves to highlight the value of the CNP approach. This case exemplifies computational modeling that we consider as an important tool in developing models of language processing beyond boxes and arrows.

Martin and colleagues conducted a longitudinal study of an individual NC with deep dysphasia (Martin, Dell, Saffran & Schwartz, 1994; Martin, Saffran & Dell, 1996). This case study, along with another from this group (FL; Schwartz, Saffran, Bloch & Dell, 1994) was instrumental in establishing a paradigm for testing computational connectionist models of language with data from individuals with aphasia. The chief data of interest in NC's case

study were the error patterns produced in picture naming and word repetition. The term “error patterns” refers to the relative rates of each error type when naming pictures or repeating words. In NC’s case, errors included word substitutions that were semantically related, phonologically related (formal paraphasias) or unrelated to the intended word, as well as phonological paraphasias and neologisms. His diagnosis of deep dysphasia was due to the occurrence of semantic errors in repeating single words. More will be said about this below, but first a few words about how a computational model of word processing is tested using error patterns in aphasia.

The model tested in this study was Dell and O’Seaghdha’s (1992) interactive spreading activation model of word production and repetition. A prediction of this localist connectionist model was that word processing deficits in aphasia could be characterized as alteration of two model parameters that control processing of words. The first parameter is connection weight, or the rate at which activation spreads throughout the lexical network. When a word is to be spoken, activation spreads to the semantic, lexical and phonological representations of that word and at the same time activates other words in the lexicon that are related semantically or phonologically to the intended word. The second parameter is the rate at which activation decays. The two parameters work together to keep activation levels of the target word and other related words balanced such that the intended word is most active when the word is to be spoken and the other words are not retrieved in error. Alterations of each model parameter result in different error patterns when the model simulates word production.

To test the model’s assumptions about the relation of connection weight and decay rate to error patterns in aphasia, its parameters were first set to values that simulated error patterns produced by speakers without aphasia (based on studies of normal speech errors, e.g., Fromkin, 1971; Garrett, 1982; Harley, 1984). The model was then “lesioned” by altering one or both processing parameters in a way that should match NC’s error patterns. This was done by keeping connection weight at its normal strength and increasing the decay rate. Thus, although a word’s semantic and phonological representations were activated normally, they decayed too quickly. This increased the probability that other words activated by spreading activation would be retrieved in error. The distribution of error types produced by the model matched that of NC in the early stages of his recovery from a stroke. Simulating the error pattern was a notable accomplishment, but a stronger test would be whether the model could predict how that error pattern might change with recovery.

When NC was initially tested early in the course of his recovery from a stroke, he presented with the following pattern: formal, semantic and phonemic paraphasias in picture naming and in single word repetition. Additionally, he presented with a severely restricted span (1 item in repetition or pointing span). As noted earlier, it was his production of semantic errors (e.g., cactus → desert, unicorn → horse) in single word repetition that led to the diagnosis of deep dysphasia. This type of error is rare in aphasia and not produced at all by normal speakers. To determine what changes in error pattern would be predicted by the model, the decay parameter, which was increased significantly to simulate a pattern that included semantic errors in repetition (along with the other features of his error pattern) was reduced towards the ‘normal’ values. This resulted in no semantic errors and a predominance of formal and phonological paraphasias in repetition, and in naming, predominantly semantic errors and phonological errors.

NC’s language abilities were studied over the course of roughly two years. As he recovered, his span increased to 2–3 items and his error pattern changed in several ways. He no longer made semantic errors in single word repetition, but continued to make formal and phonological paraphasias. Thus, by diagnosis, he would be classified as having phonological

dysphasia (see Wilshire & Fisher, 2004). This pattern was accomplished in the model by reducing the decay rate towards the “normal” rate, resulting in an absence of semantic errors in repetition of single words.

Martin and colleagues did not end the study with a conclusion that with recovery, deep dysphasia evolves into phonological dysphasia. Rather, they offered another hypothesis to explain this shift from one syndrome to another. They proposed that NC’s restricted verbal STM span and error pattern in repetition were related by a common impairment of the ability to maintain activation of word representations (the “too-fast” decay rate) sufficiently to support performance on word processing tasks. This hypothesis comes with the prediction that his error pattern in single word repetition when his verbal STM span was only a single item should re-emerge in repetition of two words, which would tax his verbal span capacity. That is, semantic errors should be present in repetition of two words. This is exactly the pattern that was observed in NC’s repetition of word pairs at later stages of recovery (e.g., officer liquor → officer alcohol). This pattern was also produced by the interactive activation model when the decay rate was held constant (at a rate closer to the normal rate) and the number of time steps prior to retrieval of a word was varied to simulate retrieval of each word in the word pair (word 1 retrieved after 8 arbitrary time steps, word 2 after 9 time steps). The empirical and computational data from this recovery study provide support for the hypothesis that a common impairment affecting maintenance of activated word representations contributes to the language impairment and reduced verbal STM deficit typically observed in aphasia.

Our CNP case study example was specifically chosen to represent a “success story”. The results obtained from this patient led to the generation of testable hypotheses that influenced further research. Importantly, the study conducted with the patient NC departed from the traditional boxes-and-arrows approach in several ways. It focused on impaired *process* rather than identification of a profile of symptoms fitted into a functional architecture. This enabled a dynamic account of the patient’s deficit, as well as hypothesizing a continuum between two “syndromes”, deep and phonological dysphasia, based on the severity of a single processing impairment, maintenance of activation. Thus, this case study illustrates that CNP need not be linked to a boxes-and-arrows model. Addressing the dynamics of language processing in aphasiology does not disqualify it as a cognitive neuropsychology study. As in what might be thought to be traditional CNP studies, connectionist models like the one employed with NC can be used to guide the study of language and other cognitive impairments without appealing to the neural substrates of the behaviors being studied.

A second contribution of this study was to stimulate interest in a perspective on the relation of language and other cognitive processes, specifically short-term memory. Rather than focusing on identifying dissociations between the language and verbal STM impairments in aphasia (e.g., Shallice, 1988), NC’s case study aimed to identify associations between impairment of each ability. The severity continuum linking NC’s repetition and verbal span impairment led to further investigation of this association in several case series studies (Martin & Saffran, 1997; Martin & Ayala, 2004). Finally, the approach used in the studies of NC’s repetition, naming and verbal STM performance provided important insights into the cognitive plasticity that comes with recovery from a neurological impairment. The investigations of NC’s language and verbal STM influenced later studies that used the interactive activation model with large patient samples, bearing relevance for both diagnostics and understanding recovery from aphasia (Dell, Schwartz, Martin, Saffran & Gagnon, 1997; Schwartz, Dell, Martin, Gahl, & Sobel, 2006; Dell, Martin & Schwartz, 2007).

Finally, we would like to make a note on symptoms vs. syndromes in CNP. The reader may have noticed that the case study of NC was characterized both in terms of symptoms (e.g., production of semantic errors) and syndromes (deep dysphasia, phonological dysphasia). These syndromes differ significantly from the clinical ones such as Broca's, Wernicke's, or conduction aphasia. The clinical syndromes represent symptom clusters that can be caused merely by anatomical proximity of different systems and thus vary considerably in terms of the specific constellation of symptoms an individual patient has. Albeit patients with syndromes like deep dysphasia are not identical, their symptom constellations are tied together functionally in a theoretical model and thus expected to be consistent (at least until some case is convincingly shown to deviate from the pattern to the extent that the underlying model needs to be revised).

The relevance of cognitive neuropsychology to clinical aphasiology

The discussion above has focused on research issues, as the CNP approach has recently been criticized on those grounds. Less attention has been paid to the relevance of CNP to clinical work. In this chapter, we shortly consider the clinical import of CNP in aphasiology.

As regards aphasia diagnostics, the CNP approach has proved to be highly relevant to clinical work as it provides concepts and tools to evaluate individual patients' cognitive behavior and to gain a better understanding of the patient's spared and impaired language and cognitive abilities than traditional clinical assessment instruments can provide. One limitation has been the lack of accessible literature for clinicians concerning a principled selection and interpretation of assessment instruments in a CNP framework, but there are recent contributions in the literature to remedy this (Whitworth et al., 2005). Additionally, in recent years, there is an increasing interest in using advanced statistical methods to validate assessments for aphasia (e.g., Gutman, DeDe, Michaud, Liu & Caplan, 2010) and treatment protocols, many of which were developed in the context of CNP.

CNP-based diagnostic assessments can be time-consuming, and thus need to be applied in a sensible way in clinical settings. As the boxes-and-arrows information-processing models became established and more detailed, they guided development of diagnostic tools which could provide finer descriptions of the impairment. A prime example is the Psycholinguistic Assessments of Language Processing in Aphasia (PALPA; Kay et al., 1992), a comprehensive assessment resource that has been well received in the field. For further discussion on the strengths and limitations of the PALPA, the reader is referred to the Aphasiology Special Issue "PALPA: 10 years after" (Code, 2004). PALPA is used widely in clinical and research endeavors around the world (Bate, Kay, Code, Haslam, & Hallowell, 2010). A more recent aphasia test battery that is based on information-processing models of language and systematic manipulation of several important psycholinguistic variables is the Comprehensive Aphasia Test (CAT; Swinburn, Porter, & Howard, 2005; see also the Forum paper by Howard, Swinburn & Porter, 2010, and ensuing commentaries).

Concerning treatment, an often cited limitation of the boxes-and-arrows models in clinical use is that they can be used to identify the targets of treatment, but cannot tell us *how* to treat an impairment (e.g., Holland, 1994; Hillis, 2005). There are numerous aspects of the "how" that need to be addressed. Treatment involves interaction between the treatment activity and the activity of a somewhat uniquely damaged language system as it responds to the treatmentⁱⁱ. It is difficult to predict the outcome of that interaction without a better understanding of the processing components of the language system. Connectionist models

ⁱⁱIssues relevant to aphasia therapy naturally enough extend beyond the scope of CNP to pragmatics and social interaction, but we limit ourselves to cognitive issues.

can provide the needed framework to guide investigations of processes that support treatment and recovery of language abilities. They provide constructs that reflect dynamics of processing (e.g., strength and endurance of activation) and are capable of demonstrating effects of impaired processing on language function.

In addition to the potential of connectionist models to account for dynamic aspects of language processing and its impairment in aphasia, those connectionist models with distributed architectures possess a mechanism for learning and re-learning. This feature enables researchers and clinicians to probe the role of learning processes in language rehabilitation. Some recent efforts along this line exemplify the potential of this enterprise. For example, Kiran & Thompson (2003) applied the principles of a connectionist account of re-learning a semantic category after brain damage (Plaut, 1996) to therapy for word retrieval disorders; training on atypical members of a category generalized to learning of typical members, but not the reverse. Although their approach was couched in terms of which *items* to treat, the resulting generalization pattern reflects dynamic processes of learning.

More recently, Abel, Wilmes & Huber (2007) designed a treatment for word retrieval impairments based directly on the hypothesis that some impairments stem from reduced connection strength and others from increased decay rate (the processing parameters in Dell's interactive activation model discussed earlier). The treatment involved use of two types of cueing hierarchies, the increasing (or ascending) cue method and the vanishing (descending) cue method. Abel et al.'s (2007) predictions were confirmed by the results of treatment. These two treatment studies are good examples of the potential for two-way traffic between theory and treatment work: not only can models guide therapeutic attempts, but therapy results can also be used to set models to test.

The application of connectionist models to development of treatment approaches is thus a potentially fruitful direction of CNP (for a review, see Martin, Laine & Harley, 2002) but it is also necessary to achieve a greater understanding of fundamental processes of learning and relearning in brain-lesioned patients through empirical studies. Here again, there are some studies of learning processes in aphasia that are beginning to shed light on these abilities (Kelly & Armstrong, 2008; Tuomiranta, Gronholm-Nyman, Kohen, Rautakoski, Laine, & Martin, 2011). As we have discussed throughout this article, traditional boxes-and-arrows models cannot address questions about dynamic processes of learning and relearning. To answer these questions in a principled way, CNP needs to embrace tools such as connectionist models in a wider extent. They can do this without abandoning the primary principle of CNP that the study of the nature of cognitive processes can be informed by studying the breakdown of cognitive processes in people who suffer from either developmental or acquired disorders of cognition.

Conclusions

We firmly believe that the CNP approach is a fruitful one for understanding aphasic deficits as well as normal language function when used with up-to-date theoretical/modeling approaches and by employing the necessary methodological stringency (for which there are now also adequate statistical tools). To our minds, a particularly useful methodological import of CNP is in bringing experimental (methodological and statistical) rigor to the case studies that seek to shed light on the inner workings of human cognition through disorders. In this way, CNP also carries further the long tradition of detailed patient studies in neurology and psychology. The CNP approach can be applied to different research questions and theoretical frameworks, and it is critical that it is not linked with a single fixed model of cognitive processing that reflects early stages of appreciation of the subcomponents of a

particular ability, for example, the information-processing models of language. Additionally, although CNP in its ‘purest’ form does not depend on linking function and neuroanatomy/physiology, CNP models and research can be used with studies of neuroanatomy and physiology to map cognitive models onto neural models. As Caramazza and Coltheart (2006) recently noted, researchers exercise CNP in different ways: a cognitive neuropsychologist may or may not attempt to link behavioral and neural evidence, may or may not entertain the idea of a strong modularity of the cognitive system, and may emphasize dissociations or put weight on, e.g., detailed error analyses. In the same vein, Turnbull (2001) commented that there has been a definite shift in CNP towards higher “tolerance” and more varied approaches.

As regards the neural level, it is difficult to see why one should actively drive the “ultra-CNP” approach and isolate CNP from brain studies. By the nature of its data source (brain-damaged individuals), CNP is inextricably linked with brain function. The mind-brain gap is still there due to the extreme complexity of brain-behavior relationships, but we should use various ways to try to cross this gap, and CNP is one of the available approaches. Together with mainstream cognitive psychology, it tries to unveil the organizational principles of human cognition that may relate better to the workings of the brain than more diffuse psychological concepts.

With regard to the clinical relevance of the CNP approach, researchers and clinicians alike have made full use of CNP models to guide treatment approaches in aphasia rehabilitation. It soon became apparent that contributions of traditional boxes-and-arrows models to aphasia rehabilitation were limited to a thorough study and report of what was impaired vs. preserved in the language system of an individual patient, mostly at the single-word level. Although a thorough diagnosis is the starting point for planning aphasia treatment, the question of *how* to treat language impairment has remained unanswered in any satisfying way. Connectionist models made it clear that aphasia rehabilitation science needed to attend to the dynamics of language processing (i.e., what the arrows do and how information is actually processed within the boxes), in order to develop effective treatment procedures. Additionally, boxes-and-arrows models are not capable of accounting for either the dynamics of changes in language patterns that occur with recovery of language function without treatment intervention, or the dynamics of re-learning during treatment. These needs are beginning to be addressed as is evidenced by some applications of connectionist models to the development of treatments for aphasia (Abel et al., 2007) and studies of language changes over the course of recovery (Martin et al., 1996) and it is highly likely that these studies represent the beginning of a new path for CNP as it is applied to aphasia rehabilitation.

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