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Cognitive Neuropsychology twenty years on

Alfonso Caramazza
Cognitive Neuropsychology Laboratory, Harvard University, Cambridge, MA, USA
Max Coltheart
Macquarie Centre for Cognitive Science, Macquarie University, Sydney, NSW, Australia

Cognitive neuropsychology began in the second half of the nineteenth century when neurologists such as Lichtheim, Wernicke, Bastian, and others began to make inferences about the cognitive architecture of the intact language-processing system from studying the different ways in which spoken or written language abilities broke down after brain damage. They even began to express their proposals about this architecture by means of explicit box-and-arrow diagrams: hence the term "the diagram-makers" that was applied to them.

These cognitive neuropsychologists were also cognitive neuroscientists: They were interested not only in the functional architecture of cognition, but also in how the components of such an architecture were localized in the brain. Their cognitive neuropsychology was successful (their diagrams of the language-processing system are simplified versions of diagrams that enjoy contemporary support; see Coltheart, Rastle, Perry, Langdon, & Ziegler, 2001, for examples) but their cognitive neuroscience was not. They did not succeed in localizing in the brain any of the hypothesized components of a functional architecture of cognition, and this failure exposed the whole enterprise to damning criticisms from noncognitively-oriented neurologists such as Head (1926). This, plus the demise of cognitive psychology itself consequent upon of the rise of behaviourism at the beginning of the twentieth century, saw cognitive neuropsychology practically vanish from the scientific scene for the first half of the twentieth century.

However, after the advent of the so-called "Cognitive Revolution" in the middle of the twentieth century (Broadbent, 1956; Chomsky, 1959; Miller, Galanter, & Pribram, 1960), cognitive neuropsychology awoke from its slumbers, aroused by seminal papers from Marshall and Newcombe (1966, 1973) on the cognitive neuropsychology of reading and from Shallice and Warrington (1970) on the cognitive neuropsychology of memory. Also important were developments in the area of sentence processing in aphasia where linguistic and psycholinguistic theory played a crucial role in guiding the analysis of aphasic symptoms (Caramazza & Zurif, 1976; Marin, Saffran, & Schwartz, 1976). The first conference solely devoted to cognitive neuropsychology was held at Oxford in 1979 (the conference was on deep dyslexia, one of the three forms of acquired dyslexia defined by Marshall & Newcombe, 1973), and its proceedings were published as a book in the following year (Coltheart, Patterson, & Marshall, 1980). The field was burgeoning rapidly; it needed its own journal, and *Cognitive Neuropsychology* began publication in 1984. The field also needed an undergraduate text, and *Human Cognitive Neuropsychology* (Ellis &

Correspondence should be addressed to Alfonso Caramazza, Cognitive Neuropsychology Laboratory, Harvard University, Cambridge, MA 02138, USA (Email: caramazz@fas.harvard.edu).

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Young, 1986) was published shortly after the journal first appeared. Shortly after that, an advanced text (Shallice, 1988) appeared.

The 20th birthday of the journal was celebrated by a symposium at the 22nd European Workshop on Cognitive Neuropsychology at Bressanone, Italy, in February 2004, and from that symposium came the papers collected together here.

Cognitive neuropsychology and cognitive neuroscience

It is useful, if one wants to make some remarks about the past twenty years of cognitive neuropsychology, to begin by discussing the distinction, already alluded to above, between cognitive neuropsychology and cognitive neuroscience. Cognitive neuroscience is the study of those neural systems of the brain that subserve cognition, and so it is a branch of neuroscience. Cognitive neuropsychology is the use of investigations of people with impairments of cognition (acquired or developmental) to learn more about normal cognitive processes, and so it is a branch of cognitive psychology, just as Rapp and Goldrick (2006) say. This view of cognitive neuropsychology as a branch of cognitive psychology and as distinct from cognitive neuroscience is widely accepted: "The term cognitive neuropsychology often connotes a purely functional approach to patients with cognitive deficits that does not make use of, or encourage interest in, evidence and ideas about brain systems and processes" (Schacter, 1992, p. 560); or for a more nuanced position: "Cognitive Neuropsychology's domain of inquiry concerns the structure of normal perceptual, motor, and cognitive processes. As such, it constitutes a branch of cognitive science. What distinguishes cognitive neuropsychology from other branches of cognitive science is the type of observations that it uses in developing and evaluating theories of normal cognition. The data used in cognitive neuropsychology are the patterns of performance produced by brain-damaged subjects. Because the basic data used in cognitive neuropsychology are the result of a biological manipulation—a brain lesion—these data will be directly relevant to claims about the functional organization of the brain. Hence cognitive neuropsychology may also be considered to be a branch of cognitive neuroscience. However, depending on the particular proclivities of individual cognitive neuropsychologists, there is considerable variation in the specific weight given by any one investigator to the cognitive or the neural part of the brain/cognition equation" (Caramazza, 1992, pp. 80–81).

The distinction between cognitive neuroscience and cognitive neuropsychology is an important one, but it is sometimes overlooked or neglected. There seem to be several reasons for this.

First, cognitive neuropsychologists are usually studying people with brain disorders (even though not studying those disorders).

Second, some cognitive neuropsychologists are also cognitive neuroscientists (just as some mathematicians are also physicists, and some psychologists are also economists) whereas others are not. For example, the papers in this collection by Rapp and Goldrick, Martin, Coltheart, and Miceli and Capasso are solely cognitive-neuropsychological; they have nothing at all to say about the brain system subserving cognition. In contrast the papers by Caramazza and Mahon, Humphreys and Riddoch, Buxbaum, Vallar, and Schwartz are not only cognitive-neuropsychological (seeking to reach conclusions about the nature of normal cognitive mechanisms from studying people in whom these mechanisms have broken down) but also cognitive-neuroscientific (seeking to reach conclusions about the neural organization of those mechanisms).

Third, there often seem to be (generally unstated) assumptions that conclusions about the functional architecture of cognition have implications for what the brain must be like, and conclusions about what the brain is like have implications for theories of the functional architecture of cognition (see Caramazza, 1992, for discussion of this issue). If these assumptions were correct, then anyone doing cognitive neuropsychology would also ipso facto be doing cognitive neuroscience. But are these assumptions correct? Do facts about the mind constrain possible

theories about what the brain could be like? One of the present authors has expressed doubts about that (Coltheart, 1982, 2004, in press), and so did one of the papers in the very first issue of *Cognitive Neuropsychology* (Mehler, Morton, & Jusczyk, 1984). Consider, for example, such claims about cognitive architecture as those made in Figure 5 of Rapp and Goldrick (2006, which depicts a detailed model of how speech production is achieved), Figure 3 of Coltheart (2006, which depicts a detailed model of how visual word recognition and reading aloud are achieved), or Figure 5 of Miceli and Capasso (2006, which depicts a detailed model of the structure of the orthographic representations used in spelling). Here we have strong, explicit, and detailed claims about certain cognitive architectures. Suppose we were convinced that the claims were true in all three cases; would anything about the brain follow, and, if so, what? And what do we currently know about the brain that importantly constrains theories about cognitive architecture? It is clear that many people do believe that there are such constraints in both directions—for example, "because I believe that neural constraints can be important for cognitive theorizing, I use the term cognitive neuroscience rather than cognitive neuropsychology" (Schacter, 1992, p. 560). However, whether there are actually such constraints is currently still a matter of controversy: See, for example, the symposium on "What has cognitive neuroimaging told us about the mind (so far)?" in the journal *Cortex* this year (see Coltheart, in press, and the commentaries upon that paper). So there is no general agreement as to how cognitive neuropsychology is related to cognitive neuroscience.

Nonetheless, as already noted, it is equally clear that neuropsychological data are by their very nature function–brain pairings, and therefore they offer a potential window into the neural organization of cognitive systems (Caramazza, 1992). Neuropsychological data have been used to formulate hypotheses about the relationship between particular brain areas and processing components in cognitive theories. For example, the elegant and detailed studies of patient DF (Goodale, Milner, Jakobson, & Carey, 1991) have been used to distinguish between the perception of form within the object recognition process and the "perception" of form used to guide motor behaviour. However, this work has also led to the proposal that a specific part of lateral occipital cortex is crucial for a perceptual process tied to the conscious recognition of form but that these processes are not needed for guiding motor behaviour (see Humphreys & Riddoch, 2006, for further discussion of these issues).

Syndromes, symptoms, and single case studies: The patient as a snowflake

It is deeply characteristic of cognitive neuropsychology that it studies symptoms rather than syndromes and carries out single case studies rather than group studies. These issues surface in many of the papers collected here; For example, it is precisely these issues that underlie the comment, made in relation to hemispatial neglect by Buxbaum (2006), who observes that "nearly every possible fractionation of the disorder has been reported, raising the possibility that each patient may be as unique as a snowflake".

This is not true just of neglect: It is true of every disorder that has been studied in any detail by cognitive neuropsychologists. Broca's aphasia is a classic example, as is very clearly documented by Martin (2006). Early work (Caramazza & Zurif, 1976) began with a hypothesis that attributed this disorder to a single cause: a defect of a syntactic processing system that is used both for understanding sentences and for constructing them. Soon, however, it became clear that agrammatic comprehension and agrammatic production do not always co-occur; indeed, they doubly dissociate (see, e.g., Caramazza & Berndt, 1985). Given this double dissociation, the disorder of language seen in Broca's aphasia cannot always be ascribed to damage to a single syntactic system used for both sentence comprehension and sentence construction. Distinct explanations are thus needed for agrammatic sentence comprehension and agrammatic sentence construction, and so it

cannot be right to seek *the* explanation of the syndrome of Broca's aphasia.

But perhaps a more restricted aphasic syndrome—agrammatic sentence construction, say—might be a suitable subject of scientific study? This soon turned out not to be so either, because the various symptoms of even this more restricted syndrome doubly dissociate too, to a remarkably refined degree. For example, some patients with agrammatic sentence constructions are impaired at the use of function words but not at the use of affixes, whereas others show the opposite dissociation (Berndt, 1987; Miceli, Silveri, Romani, & Caramazza, 1989; Parisi, 1987).

This is one reason why cognitive neuropsychologists study symptoms, not syndromes. What, they might say here, can we infer about the architecture of the sentence construction system from the fact that it can be damaged in such a way that function words suffer but affixes do not, and also can be damaged in such a way that function words do not suffer but affixes do? Which theories about this architecture are ruled out by this double dissociation, and which are compatible with it? Questions like this can be asked if the data from which inferences are to be drawn are data about single symptoms. Such questions cannot be asked if the data are data about syndromes such as Broca's aphasia (Caramazza, 1984).

The focus on studying single symptoms rather than syndromes (groups of symptoms) goes hand-in-hand with the strategy of carrying out single case studies rather than group studies. It is easy to collect together a group of people all diagnosed as exhibiting Broca's aphasia; it is very unlikely that one could collect together a group of people with intact sentence comprehension and impaired ability to generate affixes correctly but otherwise intact sentence construction (including intact use of function words).

To pursue Buxbaum's (2006) analogy, that would be like trying to collect together a group of snowflakes that all had exactly the same morphology. The number of possible different shapes for snowflakes is so large that the likelihood of obtaining two snowflakes with the same morphology is quite small, which makes the prospect of group studies of snowflake morphology an impractical one. Marshall (1984), Coltheart (1984), and Howard and Franklin (1988) make exactly the same point about the impracticality of group studies in cognitive neuropsychology. Suppose the model of some cognitive domain that one wishes to investigate contains n processing components and m pathways of communication between them. If each component or pathway can be independently impaired by brain damage, then the number of different patterns of impairment of the system that can arise is $2^{(m+n)}$. The values of m and n do not have to be very large for $2^{(m+n)}$ to become astronomically large. Since the probability of seeing two consecutive patients with the same pattern of impairment (which is the only justification for treating them as a group) is $1/2^{(m+n)}$, this probability is infinitesimal for values of m and n that are typical of current models of cognition. That is why it is appropriate to think of patients as being as unique as snowflakes.

If, for practical purposes, every patient is unique, how can we amass knowledge that generalizes across people? This requires what Caramazza (1986) referred to as the "universality assumption": the assumption that there is no qualitative variation across neurologically intact people in the architecture of the cognitive system that these people use to perform in a certain cognitive domain. This allows us to infer that, although patient X and patient Y currently have very different systems as a consequence of their brain damage, they had the same system premorbidly, and it is about that system that we want to make inferences from studying patients X and Y. Cognitive neuropsychology cannot be practised unless this universality assumption is made, but nor can cognitive psychology. This is how, even though every patient is essentially unique, we can seek generalizable knowledge from studying them (Caramazza, 1986).

The same is true for snowflakes. There is a falsifiable theory of snowflake generation that

makes the following predictions (and no doubt many others):

1. All snowflakes are snow crystals, or are composed of snow crystals.
2. Any snow crystal can have 3 or 6 or 12 sides, but none can have 4 or 5 or 8 sides.

Just as a single patient can refute some hypothesis about cognitive architecture by yielding a pattern of data that according to that hypothesis could never occur, so a single snow crystal can refute this theory of snowflake generation—a crystal with 4 or 5 or 8 sides is all that is needed (see Caramazza, 1986, for detailed discussion).

None of this is meant to be a claim that syndromes and group studies have no role to play at all in cognitive neuropsychology. Indeed, the first studies of the cognitive neuropsychology of reading in the modern era were studies of syndromes of acquired dyslexia: surface dyslexia, deep dyslexia, and visual dyslexia (Marshall & Newcombe, 1966, 1973). What this work showed us was that there are subtypes of acquired dyslexia (Castles & Coltheart, 1993, did the same thing with reference to developmental dyslexia). That allowed work on dyslexia to focus on the subtypes rather than on some undifferentiated entity "acquired dyslexia". Work on each subtype then revealed subtypes of the subtypes: different subtypes of deep dyslexia and different subtypes of surface dyslexia, for example. So the data compelled finer and finer fractionations of the syndromes, until eventually what is being studied is not a small group of symptoms (a small syndrome) but a single symptom.

The moral is clear: In any field of cognition where cognitive neuropsychology is underdeveloped, starting with small group studies of symptom collections (syndromes) might prove to be a useful ground-clearing exercise. Mature development of the cognitive neuropsychology of that domain of cognition is signalled by the replacement of this approach in favour of research in which inferences about the intact cognitive system are made on the basis of data from studies of individual symptoms. In all bar one of the papers in this volume, this is the approach that is taken. The one exception is the work on the cognitive neuropsychology of everyday action discussed by Myrna Schwartz. She discusses explicitly the use of group studies. However, the aim of the research that she discusses is not to infer something about the cognitive architecture of the intact action-planning system from studying people with acquired deficits of action planning: It is to investigate "hypotheses about brain–behaviour or deficit–behaviour correlations" (Schwartz, 2006).

Modularity

With his *The Modularity of Mind* (1983), Jerry Fodor did cognitive neuropsychology a great service by elucidating a concept that has played an important role in the development of the subject—namely, modularity. It should be no surprise, then, to find an extended review of Fodor's book in the first issue of the journal (Schwartz & Schwartz, 1984).

Some cognitive neuropsychologists (e.g., Coltheart, 1999) are completely committed to the view that the mind is modular (in the Fodorian sense). Others (e.g., Caramazza, 1992) assume only a weak form of modularity—that is, that the mind is componentially structured (in the sense used by Simon, 1969, and Marr, 1982). In all the papers in this volume, theories about the architecture of a cognitive system are postulated in which that system is considered to be composed of information-processing components each responsible for one of the information-processing jobs that need doing if cognition is to run smoothly. It is this property of cognitive systems that makes them amenable to neuropsychological investigation. In other words, it is because cognitive systems are composed of relatively autonomous processing components that "local" brain damage can result in dissociation of functions.

That we do not need the strong modularity assumption in order to make progress in cognitive neuropsychology is well illustrated by the case of belief formation. On a Fodorian account of modularity, belief formation is supported by

nonmodular central cognitive processes, and Fodor claims that these nonmodular central processes are not amenable to scientific study. Yet the cognitive neuropsychology of belief formation actually seems to be progressing rather well (see, e.g., Coltheart, 2005; Coltheart & Davies, 2000).

Computational cognitive neuropsychology

A computational model of cognition is a computer program that is capable of performing the cognitive task in question and, more importantly, performs the task in exactly the same way as, according to the theory that is instantiated by the model, people perform that task. There are major virtues associated with computational modelling:

1. Attempting to implement any theory of cognition as a working computer program always identifies a host of hitherto unsuspected ways in which the theory was underspecified or incomplete—problems that have to be fixed if the theory is to claim viability.
2. Once the theory has been made "fully complete", and the program is executable, one can see immediately whether the theory does in fact offer an adequate account of this domain of cognition: Can the program actually do the task?
3. Theory testing can be done with great rigour: Is the behaviour of the programs affected by all and only those stimulus properties that affect human performance in this cognitive domain, and in the same way?
4. Even if the answer to the question above is "Yes" in relation to a particular computational model, there may be other computational models in that cognitive domain, implementations of competing theories, which are equally successful in simulating the relevant facts. So theory adjudication is needed. It is much easier to discover experimental outcomes about which competing theories make different predictions if these theories are expressed as computer programs.

The papers in this volume show that computational modelling is rapidly becoming important in cognitive neuropsychology. If the theory of which the model is an instantiation is correct, that theory ought to be able to offer an explanation of abnormal as well as of normal cognition: When the theory has been translated into computational terms, it should be possible to "lesion" the computational model so that it shows symptoms that are also shown by patients. Whenever this is achieved, further support for the underlying theory has been obtained. This is computational cognitive neuropsychology; and this kind of work is reported in a number of the papers in this volume.

A widely used model of speech productions is that of Gary Dell and his associates (see, e.g., Dell, 1986). This has been used not only to offer an account of normal speech production, but also to model speech production in aphasia (Dell, Schwartz, Martin, Saffran, & Gagnon, 1997), and data from aphasia have been essential in development of the model. Rapp and Goldrick (2006) discuss in detail the implications of data from aphasic speech production for fundamental computational properties of the speech production system such as whether there is feedback in the system and whether processing is cascaded or thresholded. Coltheart (2006) argues in his chapter that data from acquired dyslexia have played a crucial role in evaluating competing computational models of reading. Schwartz (2006) discusses in detail in her chapter acquired disorders of action and how they can be accounted for in relation to an explicit computational model of everyday action and planning, the CS model, and in the chapter by Miceli and Capasso (2006) we see data on acquired dysgraphia beginning to exert constraints on an explicit computational model of spelling.

Although it is indisputable that computational modelling provides an especially useful extra tool in the toolbox of cognitive neuropsychologists, this is not to say that the interpretation of modelling results is any less problematic than the interpretation of other experimental results. Thus, for example, there are open and difficult

issues concerning how one determines whether a computational model can generate the patterns of results seen in brain-damaged subjects. Some theorists are content with a general qualitative fit of the data (e.g., Dell et al., 1997) while others consider it crucial that the fit be quantitatively appropriate (e.g., Coltheart et al., 2001; Ruml & Caramazza, 2000; Ruml, Caramazza, Capasso, & Miceli, 2005). Thus, for example, Ruml et al. (2005) have argued that the strongly interactive model of lexical access proposed by Dell and colleagues is undermined by the fact that it fails to account for the detailed distribution of naming error types in aphasic patients. Independently of how this issue is resolved, the important point here is that increasingly precise theoretical proposals are possible in the context of computational models.

From boxes and arrows to the structure of representations

Much of the early development in modern cognitive neuropsychology was concerned with the articulation of the functional architecture of specific cognitive systems (e.g., the spelling system). These theories were formulated in terms of the components of processing implicated in a task and their organization—the so-called box-and-arrow models. Although often denigrated for their relatively general nature, these models played (and continue to play) an important role in formulating hypotheses about the general architecture of cognitive systems (see Coltheart et al., 2001). In fact, such cognitive architectures are inescapable features of all cognitive theories (even of those proposed by denigrators of the so-called box-and-arrow theories) for the simple reason that any nontrivial aspect of cognition will involve a number of processing components and their associated representations. Be this as it may, the crucial question is whether cognitive neuropsychological data can be used to inform cognitive theory beyond the general level of functional architecture.

Some theorists (e.g., Shallice, 1988) have suggested that cognitive neuropsychological data are too "noisy" for use beyond the level of functional architecture. Others (McCloskey & Caramazza, 1991) have argued instead that there is no a priori restriction on the usefulness of such data for the purpose of developing cognitive theory at any arbitrary level of detail. They offered as existence proof for this position the case of spelling, where significant progress has been made in characterizing the structure of the orthographic representations computed at various levels of the spelling process. Crucially, McCloskey and Caramazza noted that the kind of data that were used for the latter purpose consisted of the detailed analyses of error distributions and not simply the patterns of dissociations across tasks (the more common type of data reported in neuropsychological investigations). Caramazza and Miceli (1990) reported that there were precise constraints on the occurrence and distribution of error types in the spelling performance of their dysgraphic subject, LB. For example, they noted that LB's letter substitution and transposition errors were strictly constrained by their consonant–vowel (CV) status: Consonants were exchanged/transposed only with consonants, and vowels were exchanged/transposed only with vowels. This constraint, together with other converging evidence, was taken as indicating that the orthographic representation used at the level of the graphemic buffer specified not only the identity and order of graphemes but also their CV structure. This conclusion has since received wide confirmation (for review, see Miceli & Capasso, 2006; Tainturier & Rapp, 2001), validating the claim that cognitive neuropsychological data can be used to constrain theories beyond the level of functional architecture to inform the types of representation used at various levels of processing. Indeed, there is a growing body of literature focusing on the implications for the structure and content of cognitive representations from the patterns of deficits in brain-damaged individuals (see, e.g., Nickels, 2001; Rapp & Goldrick, 2006).

By way of conclusion: More on cognition and the brain

As already noted, cognitive neuropsychology can be considered a branch of cognitive psychology where subjects' performance is used to inform theories of normal cognition. However, as also already noted, there is increasing interest in relating cognitive neuropsychological investigations to developments in cognitive neuroscience (e.g., papers by Buxbaum, 2006, Caramazza & Mahon, 2006, Humphreys & Riddoch, 2006; and Vallar, 2006). In fact, *Cognitive Neuropsychology* has recently published a good number of papers that focus on the interface of cognitive neuropsychology and neuroscience (see, e.g., papers in two special issues edited by Martin & Caramazza, 2003, *The organization of conceptual knowledge in the brain: Neuropsychological and neuroimaging perspectives*, and by Rumiati & Caramazza, 2005, *The multiple functions of sensory-motor representations*), and the composition of the Editorial Board increasingly reflects this slight repositioning of the journal vis-à-vis strictly cognitive versus neuroscience accounts of cognitive processes. This is a healthy development, and we think it reflects the recognition that cognitive neuropsychological data play a central role not only in developing theories of normal cognition but also in validating conclusions reached on the basis of neuroimaging and other neuropsychological data. This development in no way represents a rejection or even a dilution of the original motivation for the creation of a journal devoted to classical cognitive neuropsychology. As can be seen from the papers included in this volume, the principal objective of cognitive neuropsychology remains the formulation and evaluation of cognitive theories. The data from cognitive neuropsychology are extremely rich in terms of the constraints that they provide for cognitive theory but are rather weak as the basis for constraining theories of the functional organization of the brain. Still, it is important that a mature cognitive neuropsychology should reach out to cognitive neuroscience in their common effort to understand the mind-brain.

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REFERENCES

Berndt, R. S. (1987). Symptom co-occurrence and dissociation in the interpretation of agrammatism. In M. Coltheart, G. Sartori, & R. Job (Eds.), *The cognitive neuropsychology of language.* Hove, UK: Lawrence Erlbaum Associates Ltd.

Broadbent, D. E. (1956). *Perception and communication.* London: Pergamon Press.

Buxbaum, L. J. (2006). On the right (and left) track: Twenty years of progress in studying hemispatial neglect. *Cognitive Neuropsychology, 23*, 156–173.

Caramazza, A. (1984). The logic of neuropsychological research and the problem of patient classification in aphasia. *Brain & Language*, *21*, 9–20.

Caramazza, A. (1986). On drawing inferences about the structure of normal cognitive systems from the analysis of patterns of impaired performance: The case for single-patient studies. *Brain & Cognition, 5*, 41–66.

Caramazza, A. (1992). Is cognitive neuropsychology possible? *Journal of Cognitive Neuroscience*, *4*, 80–95.

Caramazza, A., & Berndt, R. S. (1985). A multicomponent deficit view of agrammatic Broca's aphasia. In M.-L. Kean (Ed.), *Agrammatism.* New York: Academic Press.

Caramazza, A., & Mahon, B. Z. (2006). The organization of conceptual knowledge in the brain: The future's past and some future directions. *Cognitive Neuropsychology, 23*, 13–38.

Caramazza, A., & Miceli, G. (1990). The structure of graphemic representations. *Cognition, 37*, 243–297.

Caramazza, A., & Zurif, E. B. (1976). Dissociation of algorithmic and heuristic processes in language comprehension: Evidence from aphasia. *Brain & Language, 3*, 572–582.

Castles, A., & Coltheart, M. (1993). Varieties of developmental dyslexia. *Cognition, 47*, 149–180.

Chomsky, N. (1959). Review of B. F. Skinner's *Verbal Behavior. Language*, *35*, 26–58.

Coltheart, M. (1982). The psycholinguistic analysis of acquired dyslexias: Some illustrations. *Philosophical Transactions of the Royal Society, B298*, 151–164.

Coltheart, M. (1984). Acquired dyslexias and normal reading. In R. N. Malatesha & H. A. Whitaker (Eds.), *Dyslexia: A global issue*. The Hague, The Netherlands: Martinus Nijhoff.

Coltheart, M. (1999). Modularity and cognition. *Trends in Cognitive Sciences*, *3*, 115–120.

Coltheart, M. (2004). Brain imaging, connectionism, and cognitive neuropsychology. *Cognitive Neuropsychology, 21*, 21–25.

Coltheart, M. (2005). Delusional belief. *Australian Journal of Psychology, 57*, 72–76.

Coltheart, M. (2006). Acquired dyslexias and the computational modelling of reading. *Cognitive Neuropsychology, 23*, 96–109.

Coltheart, M. (in press). What has cognitive neuroimaging told us about the mind (so far)? *Cortex*.

Coltheart, M., & Davies, M. (Eds). (2000). *Pathologies of belief.* Oxford, UK: Blackwells.

Coltheart, M., Patterson, K., & Marshall, J. C. (Eds.). (1980). *Deep dyslexia*. London: Routledge and Kegan Paul.

Coltheart, M., Rastle, K., Perry, C., Langdon, R., & Ziegler, J. (2001). DRC: A dual route cascaded model of visual word recognition and reading aloud. *Psychological Review, 108*, 204–256.

Dell, G. S. (1986). A spreading-activation theory of retrieval in sentence production. *Psychological Review*, *93*, 283–321.

Dell, G. S., Schwartz, M. F., Martin, N., Saffran, E. M., & Gagnon, D. A. (1997). Lexical access in aphasic and nonaphasic speakers. *Psychological Review*, *104*, 801–838.

Ellis, A. W., & Young, A. W. (1986). *Human cognitive neuropsychology.* Hove, UK: Lawrence Erlbaum Associates Ltd.

Fodor, J. A. (1983). *The modularity of mind.* Cambridge, MA: Bradford Books.

Goodale, M. A., Milner, A. D., Jakobson, I. S., & Carey, D. P. (1991). A neurological dissociation between perceiving objects and grasping them. *Nature*, *349*, 154–156.

Head, H. (1926). *Aphasia and kindred disorders of speech*. London: Cambridge University Press.

Howard, D., & Franklin, S. (1988). *Missing the meaning? A cognitive neuropsychological study of processing of words by an aphasic patient*. Cambridge, MA: MIT Press.

Humphreys, G. W., & Riddoch, M. J. (2006). Features, objects, action: The cognitive neuropsychology of visual object processing, 1984–2004. *Cognitive Neuropsychology, 23*, 174–201.

Marin, O. S. M., Saffran, E. M., & Schwartz, M. F. (1976). Dissociations of language in aphasia: Implications for normal function. *Annals of the New York Academy of Sciences*, *280*, 868–884.

Marr, D. (1982). *Vision*. New York: W.H. Freeman and Company.

Marshall, J. C. (1984). Toward a rational taxonomy of the developmental dyslexias. In R. N. Malatesha & H. A. Whitaker (Eds.), *Dyslexia: A global issue.* The Hague, The Netherlands: Martinus Nijhoff Publishers.

Marshall, J. C., & Newcombe, F. (1966). Syntactic and semantic errors in paralexia. *Neuropsychologia, 4*, 169–176.

Marshall, J. C., & Newcombe, F. (1973). Patterns of paralexia: A psycholinguistic approach. *Journal of Psycholinguistic Research*, *2*, 175–199.

Martin, R. C. (2006). The neuropsychology of sentence processing: Where do we stand? *Cognitive Neuropsychology, 23*, 74–95.

Martin, A., & Caramazza, A. (Eds.). (2003). The organisation of conceptual knowledge in the brain: Neuropsychological and neuroimaging perspectives [Special issue]. *Cognitive Neuropsychology*, *20*, 195–592.

McCloskey, M., & Caramazza, A. (1991). On crude data and impoverished theory. *Behavioral and Brain Sciences*, *14*, 453–454.

Mehler, J., Morton, J., & Jusczyk, P. W. (1984). On reducing language to biology. *Cognitive Neuropsychology, 1*, 83–116.

Miceli, G., & Capasso, R. (2006). Spelling and dysgraphia. *Cognitive Neuropsychology, 23*, 110–134.

Miceli, G., Silveri, M. C., Romani, C., & Caramazza, A. (1989). Variation in the pattern of omissions and substitutions of grammatical morphemes in the spontaneous speech of so-called agrammatic patients. *Brain and Language*, *36*, 447–492.

Miller, G. A., Galanter, E., & Pribram, K. H. (1960). *Plans and the structure of behavior.* New York: Holt, Rinehart & Winston.

Nickels, L. (2001). Spoken word production. In B. Rapp (Ed.), *The handbook of cognitive neuropsychology: What deficits reveal about the human mind*. Philadelphia: Psychology Press.

Parisi, D. (1987). Grammatical disturbances of speech production. In M. Coltheart, G. Sartori, & R. Job (Eds.), *The cognitive neuropsychology of language.* Hove, UK: Lawrence Erlbaum Associates Ltd.

Rapp, B., & Goldrick, M. (2006). Speaking words: Contributions of cognitive neuropsychological research. *Cognitive Neuropsychology, 23*, 39–73.

Rumiati, R.I., & Caramazza, A. (Eds.). (2005). The multiple functions of sensory-motor representations [Special issue]. *Cognitive Neuropsychology*, *22*, 259–496.

Ruml, W., Caramazza, A., Capasso, R., & Miceli, G. (2005). A test of models of naming using Italian fluent aphasics. *Cognitive Neuropsychology*, *22*, 131–168.

Ruml, W., & Caramazza, A. (2000). An evaluation of a computational model of lexical access: Comments on Dell et al. *Psychological Review*, *107*, 609–634.

Schacter, D. L. (1992). Understanding implicit memory: A cognitive neuroscience approach. *American Psychologist, 47*, 559–569.

Schwartz, M. F. (2006). The cognitive neuropsychology of everyday action planning. *Cognitive Neuropsychology, 23*, 202–221.

Schwartz, M. F., & Schwartz, B. (1984). In defence of organology. *Cognitive Neuropsychology, 1*, 25–42.

Shallice, T. (1988). *From neuropsychology to mental structure.* Cambridge, UK: Cambridge University Press.

Shallice, T., & Warrington, E. K. (1970). Independent functioning of verbal memory stores. *Quarterly Journal of Experimental Psychology, 22*, 261–273.

Simon, H. A. (1969). *The sciences of the artificial*. Cambridge, MA: The MIT Press.

Tainturier, M.-J., & Rapp, B. (2001). The spelling process. In B. Rapp (Ed.), *The handbook of cognitive neuropsychology: What deficits reveal about the human mind*. Philadelphia: Psychology Press.

Vallar, G. (2006). Memory systems: The case of phonological short-term memory. A festschrift for *Cognitive Neuropsychology*. *Cognitive Neuropsychology, 23*, 135–155.