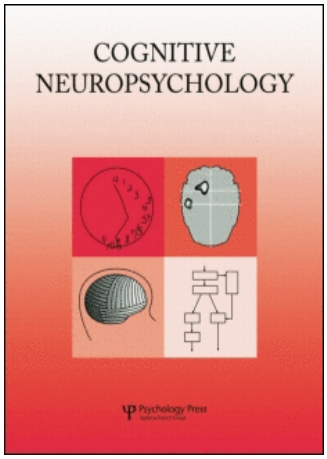

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Matthew A. Lambon Ralph [a]
[a] University of Manchester, UK

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RECONNECTING COGNITIVE NEUROPSYCHOLOGY: COMMENTARY ON HARLEY'S *DOES COGNITIVE NEUROPSYCHOLOGY HAVE A FUTURE?*

Matthew A. Lambon Ralph
University of Manchester, UK

There can be little doubt that cognitive neuropsychology has made major contributions to cognitive theorising and has become a part of mainstream psychology, at least in Britain and Europe. Not only do many undergraduate courses include dedicated modules on this field but cognitive neuropsychology has also been assimilated into many other related disciplines including cognition, perception, and developmental psychology (and is reflected in some of the most commonly used undergraduate textbooks: e.g., Eysenck & Keane, 1990). Harley's article provides a provocative enquiry of the assumptions underlying single-case cognitive neuropsychology and draws a rather negative conclusion about its future. This commentary will look again at those assumptions. Specifically, the role of computational models, case-series methodology, and neuroimaging are reviewed. When all three techniques are brought together to form a convergent perspective on the workings of brain and mind, a more optimistic case for the future of cognitive neuropsychology is revealed.

COMPUTATIONAL NEUROPSYCHOLOGY

As noted by Harley, much of the effort in cognitive neuropsychology has been aimed at unearthing the functional architecture of the cognitive system. Sometimes this leads to significant advances in cognitive theorising. The pioneering studies by Warrington and Shallice (1969) and Marshall and Newcombe (1973) led to major revisions in theories of memory and reading, respectively. Although clinicians and academics now treat deep dyslexia as an entirely normal pattern of acquired dyslexia, the ramifications of the first reported cases were so unexpected and surprising that it took several years for Marshall and Newcombe's classic paper to be published. With a focus on developing functional architecture, cognitive neuropsychology has tended to measure progress in terms of collecting fascinating individual patients and describing their patterns of dissociation with respect to increasingly detailed box-and-arrow diagrams (although there are important exceptions that date back to the Deep Dyslexia book: Morton & Patterson, 1980; see also Shallice, 1988). This approach raises two key questions: How exactly do the boxes and arrows function and, perhaps most importantly, if we knew more about the underlying computation, would all behavioural dissociations necessitate further divisions within a modular framework?

Computational models offer a form of theorising in which both architecture and function are explicitly defined. The act of engineering a model forces researchers to consider both aspects of cognitive systems. More often than not, consideration of a module's functioning means that the overall architecture does not need to be as complex as a box-and-arrow approach would imply. When applied to neuropsychological data, computational models in a variety of domains have found that important behavioural dissociations need not

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necessitate new modular divisions. For example, the Farah and McClelland (1991) model of category-specificity was able to explain a range of patient data without positing an equal number of separate modules. The demonstration of an emergent category-specific pattern from damage to visual or functional information is well known. In addition, Farah and McClelland illustrated the importance of interactivity within the semantic system: With severe lesions of visual information, for example, there was a knock-on effect on the activation of the representations coded in the undamaged function-knowledge system. In traditional cognitive neuropsychology this would imply deficits in both visual and function knowledge subsystems. The demonstration of physical damage producing functional lesions elsewhere in the system is not only important for neuropsychological theorising but also might explain similar phenomena from recent functional neuroimaging studies of patients (dynamic diaschisis: Mummery, Patterson, Wise, Price, & Hodges, 1999; Price, Warburton, Moore, Frackowiak, & Friston, 2001).

Computational models have been applied to many different domains in neuropsychology including deep dyslexia (Plaut & Shallice, 1993), surface dyslexia (Plaut, McClelland, Seidenberg, & Patterson, 1996), speech production (Dell, Schwartz, Martin, Saffran, & Gagnon, 1997), face recognition (Burton, Bruce, & Johnston, 1990), word recognition (Mayall & Humphreys, 1996), unitary vs. multiple semantic systems (Lambon Ralph & Howard, 2000), and correlational accounts of category-specific deficits (Devlin, Gonnerman, Anderson, & Seidenberg, 1998; Tyler, Moss, Durrant-Peatfield, & Levy, 2000). These models all share the same important characteristics noted above—they specify both the architecture and functioning of the domain they are attempting to describe and, as a consequence, explain a range of neuropsychological phenomena without necessarily positing modular dissociations for each one. These features make computational neuropsychology a rigorous form of theorising (Plaut & McClelland, 2000). Like any other psychological theory the model is tested against existing data and can make novel predictions, both architecture and processes have to be explicitly defined and, in addition, the model is given an engineering test, i.e., the model is actually implemented to see if it can produce the behaviour required. Two diametrically opposed criticisms are levelled at computational models, sometimes by the same reviewer. First, as noted by Harley, critics argue that these models have so much computational flexibility that they are able to explain any pattern of data. Anyone who has fought to build or train models knows from their own bitter experience that this is far from true. In fact, the succession of design changes needed to produce a model that captures the range of data required can reveal important psychological insights (e.g., the series of PDP reading models: Plaut et al., 1996; Seidenberg & McClelland, 1989). Second, models are also criticised because the researchers have failed to capture every finding from the domain. Setting aside the possibility that not all of these criticisms are valid, the fact that computational models can be scrutinised in this way is testament to the inherent transparency and rigour of the technique.

SINGLE VS. CASE-SERIES METHODOLOGY

As noted above, the study of single patients has produced important findings that have been used to advance theories in many areas of perception and cognition. There are, of course, a number of drawbacks to single-case methodology. It is not always clear if a behavioural difference in performance between two tasks or types of material is indicative of a modular dissociation or due to some other cause. Dissociations can arise because of different task sensitivities, premorbid individual differences, or variation in the underlying functions that relate severity to performance accuracy (cf. Shallice, 1988). Indeed, these factors have been used in computational modelling studies to provide alternative explanations for individual patients in the domains of surface dyslexia and category-specific deficits (Devlin et al., 1998; Plaut, 1997). It is notable that an increasing number of publications now include a case-series of individually-studied

patients. For example, the editor of a recent special issue on speech production in the journal *Aphasiology* noted with some surprise that all of the submitted articles reported more than one patient (Nickels, 2002, pp. 12–13). Although it is logistically harder to recruit and test a case-series of similar patients, this approach does avoid a number of the drawbacks of single-case methodology without losing the benefits of considering each patient's data (Lambon Ralph, Moriarty, & Sage, 2002). In particular, this approach allows the relationship between impairment severity and task performance to be plotted out—something that is impossible to do with single-case studies of chronic neurological diseases (it can be done with individual progressive patients, though such studies are still vulnerable to the drawbacks of single-case methodology: e.g., Moss & Tyler, 2000). Computational neuropsychology encourages case-series study because in addition to describing the architecture of a cognitive system, it also proposes the internal mechanisms. Various patterns of performance are produced by different degrees of damage to one or more of these internal systems. So in addition to positing architectural dissociations, computational models also predict various forms of association. These severity-performance functions or associations can only be tested empirically using a number of patients—i.e., using a case-series methodology.

NEURO*PSYCHOLOGY*

Harley's article calls into question the role of information about the brain in studies of human cognition and its impairments. The ultimate goal of neuropsychology, however, will not be achieved by studying neurological patients in isolation from information about the brain. Instead, convergent information from behavioural studies combined with functional and structural neuroimaging of intact subjects and patients will be required (Price, 2000; Price & Friston, 2002). An obvious and complex problem is that it is very difficult to relate brain structures to cognitive models, especially given the fact that there is no one-to-one correspondence. Computational neuropsychology could bridge this gap: The architecture and processes within the model could be constructed to mimic real neural substrates and, at the same time, the overt behaviour of the resultant neural network could then be compared directly against human data. This is, of course, easier said than done. Setting aside the practical problems of engineering such a model, more information about neural mechanisms, brain structures, and white matter connections are required. While this ultimate goal is a long way off, some recent studies demonstrate the potential power of combining neural and behavioural information. For example, the memory-amnesia literature contains many theories that use both neurology and psychology (Mayes & Roberts, 2001). A current debate in this literature revolves around the interaction between medial temporal lobe structures and the cortex for long- and short-term memories (e.g., Graham, 1999; Moscovitch & Nadel, 1999) including two neurally constrained computational models (McClelland, McNaughton, & O'Reilly, 1995; Murre, Graham, & Hodges, 2001). In addition, a successful computational model of executive task switching has been based directly on the architecture of subcortical to frontal lobe circuits (O'Reilly, Noelle, Braver, & Cohen, 2002).

Harley and many others have argued that knowing where structural or functional lesions are in the brain does little, if anything, to inform or constrain cognitive theories. A recent computational model of optic aphasia shows that the positioning of components can be critical (Plaut, 2002). Plaut notes that it is physically impossible to connect all the neurones fully within the space provided by the intercranial cavity and so the brain will tend to use local connections between neighbouring neural systems in preference to long links between distant substrates. Plaut encapsulated this notion within a computational model of the semantic system that mediated translations between surface representations (visual, action, phonology, and touch). As the connections between each modality and the semantic hidden units were weighted by their distance, the behaviour of the damaged model depended not only on

the degree but also on the location of the simulated lesion. The size and position of damage has also been shown to be important in a computational model of comprehension and naming (Lambon Ralph, McClelland, Patterson, Galton, & Hodges, 2001). This study is potentially interesting because by drawing together convergent information about patient behaviour, structural imaging, and computational modelling, the investigation was able to show that even basic information about the location of brain damage can change the form of cognitive theory required. Nearly all models of speech production assume that semantic impairment will lead to anomia. While empirical studies of patients with semantic dementia show this to be true, careful analysis across a case-series of patients shows that the degree of anomia can vary for the same level of semantic impairment. In traditional theorising, this would be taken as evidence for two separate forms of damage (semantic and post-semantic impairments). Lambon Ralph et al. were able to demonstrate that the variation in anomia was directly related to the distribution of temporal lobe damage. In a continuum of right- to left-sided temporal lobe atrophy, anomia was most pronounced for the left-sided patients. By including this basic neural distinction into a simple computational model of picture naming, Lambon Ralph et al. showed that an account based on a single semantic impairment was sufficient to explain the patients' results. In the future, a combination of more sophisticated computational neuropsychology and neuroimaging will make greater strides towards the ultimate goal of producing neurally constrained models of human behaviour.

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Matthew A. Lambon Ralph
University of Manchester, UK
Email: matt.lambon-ralph@man.ac.uk