Contributions of experimental psychology to neuropsychology

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Neuropsychological effects of brain damage on mental processes are varied and complex. Cognitive models from experimental psychology help us make sense of them. Such models now exist for many different domains of cognition, allowing us insight into how brain damage affects cognition in each of these domains—even higher-order domains of cognition such as belief formation. Work which applies experimental psychology to neuropsychology in this way also supports some very general conclusions about cognition, such as that the mind is highly modular, and that mental representations are typically local rather than distributed.

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Neuropsychology is the study of the relationship between the brain and psychological processes such as language, memory, object recognition, skilled action and thought. Neuropsychological research is frequently done by investigating ways in which such psychological processes can be disturbed by brain damage. Such investigations need data from experimental psychology which tell us how these psychological processes are normally carried out in people with intact brains. We cannot understand ways in which psychological processes are functioning abnormally after brain damage unless we know how they function normally, that is, in people without brain damage.

At the same time, we can learn more about theories of normal psychological functions by studying people in whom such functions have been impaired by brain damage.

This area of research is known as cognitive neuropsychology. It has two essential features:

- The use of data from people with abnormalities of some cognitive system to develop, test or extend theories about that system.
- The use of theories of some cognitive system to explain data from people with abnormalities in that cognitive domain.

Cognitive neuropsychology is thus the application of experimental cognitive psychology to neuropsychology.

Cognitive neuropsychology was much practiced in the second half of the Nineteenth Century, and quite sophisticated models of language processing were proposed by Wernicke (1874) and Lichtheim (1885), based on their research on aphasia. The rise of behaviourism at the beginning of the Twentieth Century, and the lack of success of the cognitive neuropsychologists of that era in their attempts to localize in the brain the components of their cognitive models of language-processing, led to the disappearance of cognitive neuropsychology for many decades. A seminal paper on reading disorders after brain damage—that is, on acquired dyslexia—published by Marshall & Newcombe (1973) led to the rebirth of the subject.

Acquired Dyslexia & the Dual Route Model of Reading

Marshall & Newcombe (1973) described three different forms of acquired dyslexia and proposed a simple model of how reading aloud occurs which they used to interpret the different symptoms seen in these acquired dyslexias. More sophisticated models of reading now exist, and so more sophisticated interpretations of acquired dyslexia are possible.

Current models of reading, whether connectionist
In the form of acquired dyslexia known as **phonological dyslexia** (Beauvois & Derouesné 1979; Funnell, 1983; for review see Coltheart, 1996), the ability to read aloud pronounceable nonwords is selectively impaired relative to the ability to read aloud words. Phonological dyslexia occurs not only in readers of alphabetic scripts, but also in readers of Japanese: Patterson, Suzuki, & Wydell (1996) reported a case of a Japanese reader who after a stroke could read real words written in hiragana very well (even when these were words which were normally written in kanji) but scored 0/90 in reading 2–3 character hiragana nonwords.

In phonological dyslexia, nonword reading may be completely abolished (Funnell, 1983) but more commonly some degree of nonword reading ability remains. In such cases, there are several properties of nonwords which influence the likelihood that the phonological dyslexia will succeed in reading them aloud. In some cases (Beauvois & Derouesné, 1979; Berndt, Haendiges, Mitchum, & Wayland, 1996), nonwords are read more successfully when they are pseudohomophonic (i.e. pronounced exactly like some real word—for example KOAT or PHOCKS) and this advantage is greater when the pseudohomophone is orthographically similar to its parent word (e.g. KOAT) than when it is not (e.g. PHOCKS). Both of these effects are seen in the behaviour of the DRC model when its ability to read nonwords is impaired by causing its nonlexical route to operate abnormally slowly (Coltheart et al., 1996, 2001). In other cases of phonological dyslexia (Beauvois & Derouesné 1979), nonword reading is worse for nonwords in which multiple letters map onto a single phoneme (e.g. OOV) than with nonwords which have one-to-one mappings of letters to phonemes (e.g. OLV). Such cases would seem to have an impairment in the graphemic parsing stage of the nonlexical reading route (Coltheart, 1985).

Application of models of reading to the study of phonological dyslexia thus shows us not only that this is a separate subtype of acquired dyslexia but even that it itself has different subtypes: the modeling work suggests that in some cases the poor non-

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*(Figure 1. The DRC model of visual word recognition and reading aloud.)*

(e.g. Plaut, McClelland, Seidenberg, & Patterson (1996) or nonconnectionist (e.g. Coltheart, Rastle, Perry, Langdon, & Zeigler, 2001) all incorporate the general idea that there are two computational pathways from print to speech in the human reading system. One of these pathways is capable of reading aloud pronounceable nonwords but has some or complete difficulty in reading aloud irregular or exception words (words which disobey standard spelling-to-sound rules); in the DRC model of Coltheart et al. (2001) this is the nonlexical reading route. The other pathway in these models is capable of reading aloud all words but incapable of reading aloud pronounceable nonwords; in the DRC model of Coltheart et al. (2001) this is the lexical reading route. Figure 1 shows the processing architecture of the DRC model.

Coltheart et al. (2001) described numerous results from studies of visual word recognition and reading aloud which are correctly simulated by this model. They also showed that simulations of various forms of acquired dyslexia can be achieved: one can artificially lesion the model and show that its now impaired reading shows symptoms that correspond to symptoms seen in people whose reading has been impaired by brain damage (see also Coltheart, Lang-
word reading arises because the nonlexical route is operating too slowly whereas in other cases it arises because of impairment of a specific component of this route, the graphemic parsing stage.

If phonological dyslexia is due to some form of impairment of the nonlexical route, it is worth considering what one might expect if the opposite impairment—an impairment of the lexical reading route—occurred. Since in the DRC model that route is required for the successful reading of irregular words (such as blood, are, move, yacht or gauge), what would be expected is selective impairment in the reading of irregular words with preservation of regular word reading and nonword reading. Marshall and Newcombe described a pattern like this in two patients with acquired dyslexia and named the pattern surface dyslexia; for a particularly pure case of surface dyslexia, see McCarthy & Warrington (1986) and for a review of this form of acquired dyslexia see Patterson, Marshall, & Coltheart (1985).

I have noted that phonological dyslexia occurs not only in readers of alphabetic scripts, but also in readers of Japanese. What about surface dyslexia? This is a complex question, because its answer depends upon the definition of "surface dyslexia". If the definition of surface dyslexia is worse reading of irregular than regular words, then this condition could not be identified in Japan because the terms "irregular word" and "regular word" are defined only with respect to alphabetic scripts. However, Japanese patients have been reported who, after brain injury, can still read nonwords written in kana, but make errors in reading kanji words, errors that could be treated as analogous to the kinds of regularization errors that surface dyslexic readers of alphabetic scripts make. Should this be called surface dyslexia in Japanese? For arguments that it should, see e.g. Patterson, Suzuki, Wydell, & Sasanuma (1995). Even if such arguments are not accepted, this subtype of acquired dyslexia in Japanese is clearly different from Japanese phonological dyslexia. Any model of the Japanese reading system would therefore have to be lesioned in different ways to simulate these two subtypes of Japanese acquired dyslexia.

Just as is usual in phonological dyslexia, the impairment of reading is not absolute in surface dyslexia i.e. the patient does not score 0% correct on reading aloud irregular words. A major determinant of the likelihood that an irregular word will be read correctly is its frequency, with accuracy being higher for high-frequency irregular words than for low-frequency irregular words. Coltheart et al. (1996; see also Coltheart et al., 2001) successfully simulated surface dyslexia, including the relationship of word frequency to reading success with irregular words, by impairing the operation of the lexical route of the DRC model. Specifically, this was done by reducing the sensitivity to input of the orthographic representations in the model's orthographic lexicon. This causes the model to misread many exception words (especially when they are low in frequency) while leaving untouched its accuracy in reading aloud regular words and nonwords. What is more, when irregular words are misread by the lesioned model, the reading errors are regularization errors (i.e. reading the irregular word according to the rules, such as reading blood to rhyme with "mood"), and regularization errors are characteristic of surface dyslexia on patients with this form of acquired dyslexia.

I noted earlier that there are different subtypes of phonological dyslexia; there are also different subtypes of surface dyslexia. In one subtype, there appears to be an impairment in or around the orthographic lexicon (as in the DRC simulation). But if one refers to the DRC model in Figure 1, one can see that there are various other loci at which an impairment would harm the reading of irregular words while sparing the reading of regular words and nonwords: for example, failure to access words in the phonological lexicon when reading aloud would compel reliance on the nonlexical reading for reading aloud and hence cause a surface dyslexia. This output form of surface dyslexia has been described (for further discussion of the possible subtypes of surface dyslexia and their interpretation in terms of a model like that shown in Figure 1, see Coltheart & Funnell, 1987).

**Semantics**

Figure 1 is an over-simplified model of the reading system because it says nothing about reading **com-**
prehension, and this is because it lacks a component representing word meanings—a semantic system. That system is sketched in the figure, but represented in grey because it has not been implemented in the model. If one implemented a semantic system in this model, one would have to decide how this system should be interfaced with the other components. Some preliminary work on this question in the context of the DRC model has been reported by Watters & Patel (1998a, 1998b). They developed a system of semantic representations for a small set of words using the hierarchical semantic representations provided by the lexical database Wordnet (Miller, Beckwith, Fellbaum, Gross, & Miller, 1990) and they interfaced this system with the DRC model of Figure 1 via a system of nodes which they referred to as a word sense system, but which I will refer to as a lemma system. This produces the architecture shown in Figure 2.

In models of speech production such as that of Levelt, Roelofs, & Meyer (1999; see also Roelofs (2000) for the computational version of this model, known as WEAVER+), production of spoken words during spontaneous speech or picture naming involves communicating from a semantic system to a phonological lexicon. Intervening between these two levels of representation is a system of lemmas. It is at this level that syntactic information about a word is held (for example, grammatical gender, the mass noun vs. count noun distinction, and—a Japanese language example—information about classifiers in this language). Adding a system of lemmas to the DRC model, as is sketched in Figure 2, might allow it effectively to be merged with the WEAVER++ model to produce a more general model which can simulate all the effects in visual word recognition and reading aloud that the DRC model can simulate and all the effects in speech production that the WEAVER++ model can simulate.

So far I have only discussed the effects of brain damage on reading aloud. But in other patients it is semantics that is affected i.e. the ability to understand language. There are a number of ways in which the Figure 2 model could be impaired in order to generate a comprehension impairment. Damage to the lemma system or its communication with semantics would produce impaired comprehension of both spoken and written words even though the semantic system itself is still intact. Or there could be damage to the semantic system itself.

Consider the following conversation with the patient AC (Coltheart, Inglis, Cupples, Michie, Bates, & Budd, 1998):

MC: "How many legs does an oyster have?"
AC: "A few"
MC: "I see. What about an ant?"
AC: "Some."
MC: "A caterpillar?"
AC: "No legs"
MC: "What about a snake?"
AC: "None"
MC: "And a seagull?"
AC: "Four legs"

Why could not AC do this task? Coltheart et al. (1998) investigated a number of possibilities, as follows:

(a) AC has lost semantic knowledge in the semantic category of animals: No; because he was still at chance when asked to classify inanimate ob-

![Figure 2. A schematic elaboration of the DRC model to include a lemma system as an interface to semantics.](image-url)
jects as possessing legs or not;
(b) AC has lost semantic knowledge about the property "possesses legs": No; because he was still at chance when asked to classify animals as possessing tails or not;
(c) AC has lost semantic knowledge about the parts that objects possess: No; because he was still at chance when asked to classify objects by their overall shape (round versus long) or by their colour (typically coloured or not);
(d) AC has lost all semantic knowledge about properties of objects: No; because he was not impaired at classifying objects according to nonperceptual properties (dangerous versus harmless; an Australian animal versus a non-Australian animal; edible versus inedible; land creature versus sea creature);
(e) AC has lost semantic knowledge about the perceptual properties of objects: No; because he was not impaired at classifying objects according to nonvisual perceptual properties (typically makes a noise versus does not make a noise; possesses an odour versus does not possess an odour).

In sum, then, AC has intact knowledge of nonperceptual semantic properties of animate and inanimate objects, and intact knowledge of perceptual semantic properties of such objects, as long as these are not visual properties. What he is lost is, specifically, all knowledge of the visual properties of such objects. These results suggest that the semantic system is not a single body of knowledge, but instead is a set of separate subsystems of knowledge, one of which is knowledge of the visual properties of objects: it is that separate subsystem which AC has lost. This led Coltheart et al. (1998) to propose a conception of the overall semantic system as a set of perceptual knowledge subsystems, one for each sensory modality, plus a nonperceptual (i.e. conceptual) knowledge subsystem.

This study of AC indicated one way in which semantic impairments can be category-specific: sometimes they are restricted to a particular semantic category (e.g. animate objects) and sometimes they are restricted to a particular modality of input (e.g. pictures versus printed words). Thus cognitive neuropsychology has led us to an especially rich and complex conception of the organization of the semantic system; for further discussion of this extremely large topic, see e.g. the book on this subject by Forde & Humphreys (2002).

So far I have discussed the contribution of theories from experimental psychology to neuropsychology in just two domains of cognition, reading and semantics. The same kinds of contribution have also occurred in other domains, such as music processing (Peretz & Coltheart, 2003), attention, object recognition, face recognition, calculation, spelling, skilled action and many others; for reviews of such work, see Rapp (2001).

As well as contributing to our knowledge of cognition in specific cognitive domains, however, cognitive neuropsychological work has contributed in a rather general way to our overall conceptions of cognition—our general conceptions concerning, for example, the modularity of mind and the nature of mental representations.

Modularity and Local Representation

Two fundamental and interrelated questions here are:
(a) How modular is cognition?
(b) Are all mental representation distributed, or do at least some cognitive systems use local representations?

I will discuss these issues in relation to the concept of “mental lexicon”, so I need to say first what I mean by this concept.

Lexicon: a body of local representations representing stimulus forms in some particular domain

Phonological lexicon: contains the phonological forms of all the words whose phonology you know, one entry per word.

Orthographic lexicon: contains the orthographic forms of all the words whose orthography you know, one entry per word.

Pictorial (visual-object) lexicon: contains the visual
forms (structural descriptions?) of all the objects whose appearance you know, one entry per object.

Does the human mind contain such components? The idea that there are lexicons is frequently rejected in current connectionist models of cognition such as the connectionist models of reading proposed by Seidenberg & McClelland (1989) and Plaut, McClelland, Seidenberg, & Patterson (1996) or the connectionist model of speech recognition proposed by Gaskell & Marslen-Wilson (1997). Such rejections arise because of connectionism's reliance on distributed rather than local representations, and because connectionists are intrinsically unsympathetic to modularity.

A task frequently used in experimental psychology is lexical decision: judging whether or not a printed (or spoken) stimulus is a word or a nonword. There's a comparable task with pictures, known as object decision: pictures of real objects or of non-objects (composed of components from real objects, with these components put together in plausible ways) are shown and the subject has to decide whether the visual stimulus is a real object or not.

How do people carry out such tasks? If mental lexicons with local representations exist, the answer is easy: just look the stimulus up in the relevant lexicon. If it is there, say YES; otherwise say NO. But if there are no mental lexicons, explaining how people are able to perform these tasks so quickly and so accurately is not simple. The connectionist answer here has typically been: people interrogate their semantic system. If the stimulus has activated the semantic system, then it must be a real word or object, so respond YES; if semantic activation is absent, or if it is weaker than some criterion, respond NO (see Plaut, 1997, for this approach to visual lexical decision and Gaskell & Marslen-Wilson (1997) for this approach to auditory lexical decision). Such an answer makes a very clear prediction: people with impaired semantic systems cannot be normal at visual lexical decision, auditory lexical decision, or object decision.

There have been numerous falsifications of this prediction in the cognitive-neuropsychological literature:

**Visual lexical decision**

(a) DC (Lambon Ralph, Ellis, & Franklin, 1995) was severely impaired at comprehending printed words but in the normal range at visual lexical decision.

(b) JO (Lambon Ralph, Sage, & Ellis, 1996; Lambon Ralph, Ellis, & Sage, 1998) was also severely impaired at comprehending printed words but in the normal range at visual lexical decision.

(c) EM (Blazely & Coltheart, submitted) was also severely impaired at comprehending printed words but in the normal range at visual lexical decision.

**Auditory lexical decision**

(a) KW (Hall & Riddoch, 1997) was severely impaired at understanding spoken words but in the normal range at auditory lexical decision.

(b) Dr O (Franklin, Howard, & Patterson, 1994) was impaired at comprehending spoken words when they were abstract, but in the normal range on an auditory lexical decision task containing many abstract words.

**Object decision**

(a) SB (Sheridan & Humphreys, 1993) showed a selective semantic impairment for animals and foodstuffs but was in the normal range on object decision tasks even though the task stimuli included many representations of animals and foodstuffs.

(b) JB (Humphreys & Riddoch, 1987) had a general semantic impairment but performed in the normal range on an object decision task.

It is therefore not correct to claim that lexical decision and object decision tasks are performed by consulting the semantic system. That leaves our ability to perform these tasks unexplained by theorists who argue that mental lexicons of local representations do not exist; and this supports the claim that the word and object processing system is highly modular (being composed of separate lexicons) and uses local rather than distributed representations, since this claim predicts that there will be patients who are semantically impaired but normal on lexical decision and object decision.
Cognitive Neuropsychiatry

In the early days of its renaissance, cognitive neuropsychology mainly focussed on studying disorders of basic cognitive processes such as language or memory or perception, and examples of this kind of work are discussed above. In recent years, however, the attention of some cognitive neuropsychologists has turned to disorders of higher-level mental processes such as reasoning or belief formation. Abnormalities of such higher-order cognitive processes are often regarded as psychiatric conditions; hence the application of cognitive neuropsychology to such conditions is known as cognitive neuropsychiatry. Its ultimate aim, since it is a branch of cognitive neuropsychology, is to develop models of these higher-order cognitive processes on the basis of data from people with disorders of these processes, and to seek to use such models to interpret such disorders.

So far, most work in cognitive neuropsychiatry has been on delusional beliefs. A number of different kinds of delusional belief have been recognized for a long time. They include:

- I am dead (the Cotard delusion).
- I am constantly being followed around by a group of people I know, but I can not recognize them because they are always disguised (the Fregoli delusion).
- My spouse has been replaced by an impostor (the Capgras delusion).
- Other people are inserting thoughts into my mind (thought insertion; usually associated with schizophrenia).
- Other people can control the movements of my body (alien control; usually associated with schizophrenia).
- This arm [the speaker's left arm] is not mine, it's yours.
- When I look into the mirror, the person I see is not me, but some stranger who looks just like me (mirrored-self misidentification).

Although conditions like these have typically been explained in psychiatric terms, it has become clear recently that many, and even possibly all, are neuropsychological in origin. For example, patients with Capgras delusion fail to show the normal autonomic arousal response to pictures of familiar faces (Ellis, Young, Quayle, & de Pauw, 1997); it seems highly plausible that this is implicated in the belief that one's spouse (the sight of whose face ought to activate one's autonomic nervous system maximally) has been replaced by a complete stranger (since a stranger's face would be much less arousing). This neuropsychological disconnection between the face recognition system and the autonomic nervous system seems a necessary component of Capgras delusion, but it is not sufficient to cause the delusion, since the disconnection is also seen in people who are not deluded (Tranel, Damasio, & Damasio, 1995). My colleagues and I (see e.g. Langdon & Coltheart, 2001; Davies & Coltheart, 2001; Davies, Coltheart, Langdon, & Breen, 2002) have therefore proposed a two-factor theory of delusion. The two factors which we regard as jointly necessary for the development of a delusional belief are:

(a) the presence of some neuropsychological abnormality responsible for the initial occurrence of the belief (e.g. the autonomic disconnection in Capgras delusion) and;

(b) damage to a right cerebral hemisphere system for belief evaluation is responsible for failure to reject the belief; if this system were intact, the initial belief would be evaluated and then rejected (because, for example, the belief is bizarre or implausible, or because everyone is telling the deluded person that the belief is a false one).

This two-factor account appears to offer plausible explanations of many of the forms of delusion listed above, though far more work is needed if the account is to be adequately evaluated.

Future Directions in Cognitive Neuropsychology

- Developmental cognitive neuropsychology. There is an important distinction between acquired and developmental disorders of cognition. When a person had acquired some cognitive ability to a normal level but then suffered brain damage which reduced or eliminated this ability, this repre-
sents an acquired disorder of cognition. When a person has never attained a normal level of performance of some cognitive ability, this represents a developmental disorder of cognition. Developmental cognitive neuropsychology is the application of experimental cognitive psychology to the understanding of developmental disorders of cognition such as developmental dyslexia or specific language impairment. Its aim is to learn more about the ways in which children normally acquire particular cognitive abilities by studying children who are having specific difficulties in acquiring any such cognitive ability.

- The relationship between cognitive psychology and cognitive neuroimaging of the brain. In the past decade, a popular (and very expensive) method of studying cognition has been to measure neural activity within the brain (using PET, fMRI, MEG etc.) as a person is performing a cognitive task. What aims does such work have?

One obvious aim is localization of cognitive modules in the brain. However, you cannot locate cognitive modules in the brain using imaging unless you have a theory from experimental psychology about what those modules actually are. So you cannot use imaging to discover what the modules are. That means that cognitive neuroimaging is dependant upon experimental psychology.

Are there other possible aims of cognitive neuroimaging? For example, could data from cognitive neuroimaging be used to develop new theories about cognition, or to decide between existing theories? I don't think there has been any cognitive neuroimaging work which has achieved this particular aim; I am even doubtful about whether this will ever be achieved in future cognitive neuroimaging work.

Concluding Remarks

- Cognitive models from experimental psychology can provide explanations of acquired or developmental disorders of cognition, methods for assessing such disorders, and information about where treatment should be directed.
- Data from people with such disorders provides ways of testing these models.
- This approach works even for high level cognitive disorders such as delusions or hallucinations.
- Results obtained with this approach strongly suggest that cognitive systems involve a very high degree of modularity.
- Results obtained with this approach also strongly suggest that cognitive representations are in many cases local rather than distributed.

References


