The neurocognition of recovery patterns in bilingual aphasics

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Abstract

We have yet to explain the variety of recovery patterns in bilingual aphasics despite the practical and theoretical importance of doing so. I consider the reasons for this state of affairs and identify what is needed in order to achieve a causal understanding. Theoretically, I distinguish the issue of the representation of a linguistic system from its control and explore the neuroanatomical bases of representation and control. Methodologically, I argue for the importance of neuroimaging studies (PET, fMRI) to complement psycholinguistic and neuropsychological data and propose a number of psycholinguistic and neuroimaging studies aimed at clarifying the causal basis of recovery patterns. A final section briefly considers the implications for rehabilitation.

Introduction

There are two related reasons for seeking to understand the nature of recovery patterns in bilingual aphasics. First, we need a principled basis for specifying rehabilitation programs. As a result of migration patterns and intermarriage, the number of bilingual and polyglot speakers will increase and with it, as a result of stroke, closed head injury or neurodegenerative disease, the incidence of bilingual aphasia. In the case of the United States, for example, Paradis (2001) estimates, on the basis of census data, that there will be well over 45,000 new cases per annum. Second, the patterns of recovery in bilingual aphasics challenge accounts of the representation and control of language in the brain. This challenge will drive the theoretical and empirical innovations necessary to achieve a principled basis for rehabilitation.

A key requirement is a causal account of recovery patterns that can provide predictors of recovery and guidance for rehabilitation. At present we lack such a causal account and cannot predict clinical outcomes. The following quote indicates the scale of the task: ".. no correlation has been found between the pattern of recovery and neurological, etiological, experiential or linguistic parameters: not site, size or origin of lesion, type or severity of aphasia, type of bilingualism, language structure type or factors related to acquisition or habitual use"(Paradis, 1995, p. 211). This chapter outlines a blue print for tackling the problem. The first section identifies the basic patterns of recovery and discusses why it is that we lack a causal account. A key factor is the need for a neurocognitive approach to the representation and control of language in the brain informed by neuroimaging studies of patients. The second section describes such an approach and the third section applies it to some key patterns of recovery. Before concluding, a final section briefly considers the implications for rehabilitation.

The nature of the problem

Patterns of loss and recovery

Different patterns of recovery can be identified in terms of the relative impairment of two or more languages (L1, L2 ..) and the course of recovery. I describe these patterns and then consider their incidence and practical impact.

Paradis (1977) identified six basic recovery patterns. Languages can be affected equally, differentially or selectively. *Parallel* recovery occurs when both languages are impaired and restored at the same rate; *differential* recovery occurs when languages recover differentially relative to their pre-morbid

levels and *selective* recovery occurs when at least one language is not recovered at all. In *blended* recovery, patients mix their languages inappropriately.

There are different trajectories to a particular "end state". Two or more languages may eventually recover but the second language may only begin to recover when the first has (fully) recovered. This is termed *successive* recovery. A special case of selective recovery, *antagonistic* recovery, occurs when as one language recovers a second language becomes impaired.

Two further patterns, *alternating antagonism* and *selective aphasia* may be considered variants of antagonistic and selective recovery, respectively (Paradis, 2001). In alternating antagonism, patients can access only one of their language in spontaneous speech for alternating periods of time (Nilipour & Ashayeri, 1989; Paradis, Goldblum & Abidi, 1982). In the case of selective aphasia (Paradis & Goldblum, 1989), in contrast to selective recovery, there are aphasic problems in one language with no obvious deficits in the other.

These basic patterns do not exhaust the set of possibilities. A language may be recovered in an antagonistic fashion and a third never recovered at all. Or, in the case of alternating antagonism, there may be a temporary inability to translate into the language that the patient can use spontaneously (patient A.D., Paradis et al., 1982). There are also other rare, but important, cases involving a selective deficit such as the loss of the ability to avoid switching between languages (patient S.J. , Fabbro, Skrap, & Agliotti, 2000; see Ansaldo & Joanette, 2002 for a review and interpretation of reported cases of pathological language switching and language mixing).

Incidence and impact
The true incidence of the basic patterns of recovery is unknown. Fabbro (1999, see earlier, Paradis, 1977), estimated, on the basis of published cases, that the typical pattern of recovery is one in which both languages recover in parallel (40% of cases). Better recovery of the mother tongue (L1) occurs in 32% of the case and better recovery of the second language (L2) in 28% of the cases. In a review of cases reported between 1990 and 2000, that does include reports of unselected cases, Paradis (2001) found that the majority exhibited parallel recovery (81/132: 61%), 24 (18%) showed differential recovery, 12 (9%) a blended recovery pattern, 9 (7%) selective recovery and 6 (5%) successive recovery, but he rightly cautions against inferring population values from these figures.

The communicative impact of these different patterns of recovery varies. Parallel recovery allows an individual eventually to achieve pre-morbid levels of communication with family, peers and the wider public. In contrast, other patterns such as antagonistic or selective recovery, can create severe communication problems. A person may be unable to communicate linguistically with their immediate family and friends or be unable to work.

Why don't we have a causal account of recovery patterns?

Paradis (2001) argues that we have the theoretical tools to account for the patterns but do not understand what determines a particular type of recovery or, in the case of non-parallel recovery, what determines the language that is preferentially recovered. The argument in this section is that we lack two key ingredients to answer these questions: critical data and explicit neurocognitive accounts that specify the relevant causal parameters. This argument is pursued by considering three aspects of the problem: the nature of language, individual differences in recovery processes, and the lesion deficit methodology.

Abstract characterisation of language
Languages differ and a myriad of language combinations is possible. We need to consider languages at a suitable level of abstractness if we are to find unity amongst the diversity of outcomes. At an abstract level, there are four linguistic means for communicating experience (see, for example, Tomasello, 1995) – individual symbols (lexical items); markers on symbols (grammatical morphology), ordering patterns of symbols (word order) and prosodic variations of speech (e.g., stress, intonation, timing). Languages differ in the weight they attach to these different means. In some languages, word order is basically free and information on "who did what to whom" is conveyed by word endings, or by prosody in tone languages. By contrast, in English, such information is conveyed by word order and this is relatively rigid. These different

linguistic means require different processes. A given lesion can therefore give rise to different outcomes in different languages because one process is relatively more important in one language rather than another and so there can be more opportunities for errors of a certain type to reveal themselves in one language rather than another (e.g., Paradis, 2001). Damage to a device implementing that process will exert a greater effect in one language rather than another and so underlie differential recovery in one instance and selective recovery in another.

Individual differences in recovery
Recent years have seen a number of developments that have improved the quality and validity of data. Standardised instruments for assessing language performance in different languages (e.g., the Bilingual Aphasia test, see Paradis, 2001) are vital to establishing valid data sets. Further, records of unselected cases of bilingual aphasia (see Paradis, 2001, p. 71) help overcome any bias in published case reports towards the unusual. These developments are welcome but they do not go far enough. Individuals differ in their ability to recover from damage.

A lesion at a given site and extent may yield different effects (e.g., parallel recovery versus differential recovery) because of more effective repair process in one individual compared to the other. A number of factors are known to affect the likelihood of recovery from a focal lesion. These factors include: age, premorbid IQ /education level and the integrity of the frontal lobes (see, for example, Robertson & Murre,1999). It follows that neuropsychological assessments that focus only on language tasks may fail to detect dimensions critical to recovery.

Lesion deficit approach
The patterns of recovery provide evidence of potentially dissociable cognitive systems underlying different languages (e.g., Gollan & Kroll, 2001; Paradis, 2001). Consider, for instance, cases of the preferential recovery of one language over another. In some cases the mother tongue is recovered better than a language acquired second. In other cases, the converse obtains. Case reports also indicate that the devices involved in translation from one language to another are cognitively distinct from those mediating picture naming or spontaneous speech production. A neurocognitive account must take the further step of identifying the neuroanatomical bases of these devices and systems. Until recently the primary means for establishing the neuroanatomical representation of such devices was the lesion deficit approach.

This approach is important because it indicates the cortical regions necessary for performance of a linguistic task (e.g., speaking in L1) but it cannot establish whether or not a given deficit reflects damage to a specialised device at the site of the lesion or to a distributed network with connections that pass through the lesion site. More seriously from the point of view of correlating lesion site, and extent, to recovery patterns, it cannot establish whether or not there is residual capacity in the damaged tissue. It also leaves open other possible mechanisms of recovery (e.g., the use of a duplicate but previously inhibited mechanism) or cognitive changes in the way a given task is performed. This lesion deficit approach needs to be complemented by one involving neuroimaging (see Price & Friston, 1999; Green & Price, 2001).

In the normal brain, positron emission tomography (PET) and functional magnetic imaging (fMRI) can identify the complete set of regions associated with one task relative to another, and critically, how one region interacts with another. However, such methods (see below for a description) have inherent limitations too. One limitation is pertinent here: these methods (along with other physiological measures such as single- and multi-unit electrophysiology or EEG) tell us about the activation or engagement of a system in the performance of a task but not about its necessity (e.g., Sarter, Bernston, & Cacioppo, 1996; Brown & Hagoort, 2000). A combination of neuropsychological assessment, data on lesion site and neuroimaging data (together with other techniques such as transcranial magnetic stimulation) can help identify regions that are both necessary and sufficient for task performance. Critical to such an endeavour is a view of how the processes mediating language use map onto the neuroanatomical substrate. The next section addresses this question.

A neurocognitive approach: representation and control

A neurocognitive approach must characterise the bilingual system at a cognitive level, state how the devices at this level map onto the neuroanatomical networks, and show how damage at the neuroanatomical level

can give rise to the observed behaviour (see Morton & Frith, 1995, for an insightful account of causal modelling). It must also state how damaged networks and circuits recover function.

The first part of this section considers the cognitive devices comprising the bilingual system. We will presuppose a distinction between thought and language (Clark, 1996; Johnson-Laird, 1983) but grant that in thinking for speaking (Slobin, 1996) bilinguals formulate their messages in terms of the concepts of the language (Black & Chiat, 2000; Green, 1998a; Levelt, Roelofs, & Meyer, 1999). The second part specifies how these devices and their properties may be implemented in the neural system.

Cognitive level description

The devices used to perform different tasks refer to actual neurocomputational machines and so an adequate cognitive description needs to include parameters relevant to the working of real devices. The focus in what follows is on the types of device but we need to keep in mind that each device is not only dedicated to processing information of a certain type, it is both capacity-constrained (that is, it processes inputs of a certain type at a limited rate) and resource-constrained. It will fail to operate, for example, without the metabolic means to do so. Although the concepts of capacity and resource are distinct and, as we will see, can be identified with distinct neural properties, there is a relation between the two concepts. As resources decline, the "functional capacity" of the system will decrease though the precise nature of the decline is an open question.

We distinguish between a device representing the meanings of words, their syntactic properties and the word forms (the bilingual lexico-semantic system) and devices involved in controlling the outputs from that system. This contrast leads us to expect that certain patterns of recovery may arise from problems in controlling the bilingual lexico-semantic system rather than from damage to it. Damage to different components of the control system may yield different outcomes. Alternatively, the same broad clinical outcome may arise for different reasons (e.g., damage to the lexico-semantic system or to components of the control system).

In order to appreciate the problem of control, consider the task of naming a picture in L1. To perform this task, individuals must avoid performing other tasks such as free associating to the picture or assessing its aesthetic qualities. Following Green (1998b) we will say that individuals must activate a particular task schema that co-ordinates relevant devices (see also Monsell, 1996). In the case of picture naming, the schema pairs a picture name in L1 (say) with the output of a picture recognition device that has activated a set of lexical concepts. The schema for producing a name in L1 may be in competition with the other schemas, in particular with one to name the picture in L2. Top-down control is achieved in the normal case by a higher level, or executive, system that boosts the relative activation of the target schema (Shallice, 1988). On this account, language control is part of a system for the control of action in general (e.g., Green,1986; 1998b; Meuter & Allport, 1999; Paradis, 1981) though such a claim does not preclude circuits specialised for the control of linguistic actions (e.g., Paradis, 2001). Problems in production can arise because of a difficulty in ensuring that the intended schema is dominant.

For bilingual speakers to name the picture in the intended language, the lexical representations of words also need to be coded or tagged for language in some way (Albert & Obler, 1978) to allow their selection by the task schemas (Green, 1998b; see also de Bot & Schreuder, 1993; Dijkstra & van Heuven, 1998, for various views on such tagging). Comparable tags (or units coding for language) are part of the monolingual speaker's repertoire too – allowing the selection of vocabulary suited for different registers. Disconnection, or noisy transmission, between schemas and tags or between the representation of lexical concepts and tags provide ways in which a selective pattern of recovery could arise.

How is selection actually achieved? Given a requirement to speak in only one language, selection could be achieved at a late stage by filtering or by inhibiting "lemmas" or lexical nodes that lack the requisite tag (Green, 1998b). Individuals must, of course, select appropriate words in other tasks. Neuroimaging studies indicate that such selection involves inhibitory processes both in the standard Stroop colour-word task (Peterson et al., 1999) and, more significantly, in a picture-word interference task (De Zubicarary, McMahon, Eastburn & Wilson, 2002). In the case of selecting between words in different languages, selection may be biased against the non-target language by selectively deactivating entire language systems or parts of such systems (Grosjean, 1998; 2001; Paradis, 1981, 2001; de Bot & Schreuder, 1993 –see also Rodriguez-Fornells, Rotte, Heinze, Nösselt, & Münte, 2002 for evidence of selection in visual word recognition) or by inhibiting such systems or their components (Green, 1986;

Dijkstra & van Heuven, 1998). In pathological cases such a mechanism explains the temporary loss, or permanent inaccessibility, of a language – it is not destroyed but inhibited - a conjecture first proposed by Pitres (1895, see Paradis, 2001).

There is debate on the extent, or the conditions under which, naming in one language rather than another involves selection amongst lexical candidates from both languages or from just one language (e.g., Costa, Miozzo, & Caramazza, 1999; see also Costa in this volume). A detailed analysis of the relevant normal data is outside the scope of this chapter but current evidence suggests that words in L1 invariably compete for selection when individuals are naming pictures in L2 (Hermans, Bongaerts, de Bot, & Schreuder, 1999). L2 names for pictures also compete for selection when individuals are naming in L1 in circumstances in which individuals are required also to name in L2 within the same block of trials (Kroll & Peck, 1998, cited in Gollan & Kroll, 2001). Unfortunately, there appear to be no experimental studies examining competition in grammatical encoding.

Competitive costs can be reduced by differentiating lexical concepts in the two languages. MacWhinney (1997, p. 120) comments that individuals can limit competition by directly linking the L2 term to its concept rather than by linking it to L1 lexical item (as in the lexical route in the Revised Hierarchical Model of bilingual memory representations, see Kroll & de Groot, 1997). The extent of such differentiation will presumably depend on the type of concept (e.g., is it language-specific or not). It may also depend on proficiency and usage.

Fluency within a language may be viewed as an outcome of tightening within-language links over between-language links. Over time the two systems achieve a quasi-independent status equivalent to the subset hypothesis (Paradis, 1981; 2001). Usage may also be important. Where, for instance, individuals are required to translate between languages, selection may rely less on concept differentiation and more on selection via language tags. Computational modelling of systems evolving under different circumstances would be helpful as a means to explore these conjectures.

Neuroanatomical description and the convergence hypothesis

An important question concerns the extent to which a second-language is processed differently from the first. In the normal brain, language functions in monolingual, right-handed individuals, are typically represented in a distributed left hemisphere network (Loring, Meador, Lee et al., 1990; Springer, Binder, Hammeke et al., 1999). In principle, different languages might be represented in a different neuroanatomical substrate (e.g., in homologous areas of the right-hemisphere). However, Rapport, Tan, & Whitaker (1983) in a study of right-handed polyglot aphasics prior to surgery found no evidence of the disruption of picture naming following intracarotid injection of sodium amytal into the right-hemisphere. In contrast, naming was massively disrupted following injection into the left-hemisphere. Further, in a study of 88 reported cases of right-handed bilingual aphasics, Fabbro (1999, p. 210-211) found that only 8% had a lesion to the right-hemisphere. Taking into account reporting biases, he concluded that the incidence of aphasia in bilinguals with right hemisphere lesions is not in fact higher than that shown by monolingual aphasics. In sum, current data indicate that although languages form distinct subsets (see Paradis, 1981; 2001) they are represented in a common substrate.

I will suppose, more specifically, the *convergence hypothesis* (see Green, submitted, for more details). According to this hypothesis, as proficiency in L2 increases, the representation of L2 and its processing profile (i.e., ERP and neuroimaging data) converge with those of native speakers of that language. That is, any qualitative differences between native speakers of a language, and L2 speakers of that language, disappear as proficiency increases (for rather different views see, for example, Paradis, 1994 and Ullman, 2001). Current ERP and neuroimaging data are consistent with this hypothesis (e.g., Abutalebi, Cappa, & Perani, 2001; Osterhout & McLaughlin, 2000; Weber-Fox & Neville, cited in Ullman, 2001 but see also Hahne & Friederici, 2001- see also Vaid & Hull, 2002 for a critical review of the neuroimaging data and Kroll & Dussias, in press, for an assessment of the ERP and psycholinguistic data on syntactic processing in L2). Notice the convergence hypothesis is a claim about neural representation and processing profiles and not a claim about whether or not an L2 speaker of a language can simulate or pass off as a native speaker of that language. Unfortunately, there is a dearth of longitudinal on-line psycholinguistic or functional imaging studies specifically on L2 grammatical processing and encoding so the robustness of this hypothesis is open to question. For the present, the convergence hypothesis allows us to simplify the

problem of mapping devices to neuroanatomical structures. We suppose an identity, at least at the broad anatomical level, with the representations of monolingual speakers and differentiation at the microanatomical level (Paradis, 1977, 2001).

In the following paragraphs I propose a series of identifications (Green & Price, 2001). First, I consider how the bilingual lexico-semantic system, and its control system, map onto neural structures. Conceivably, a given device maps onto a specific neural mechanism in a restricted neuroanatomical area. But this appears not to be the case with language. Second, I consider the neural identifications of capacity and resource.

The bilingual lexico-semantic system
Regions sustaining word production can be divided into those involved in articulation - these include the premotor cortex, the supplementary motor area (SMA) and the cerebellum - and those involved in retrieving phonology. Phonological retrieval (Price, 2000) involves the left anterior insula and the left frontal operculum (part of Broca's area). In the case of reading, the bilateral supramarginal gyri are implicated in the mapping of orthography to phonology (see below for discussion of the claim that they are the site of a language "switch").

Both neuropsychological and neuroimaging studies indicate that there is a degree of specialisation within monolingual speakers for syntactic and semantic processes. Breedin and Saffran (1999) reported a patient, D.M., who was good at detecting grammatical violations despite a pervasive loss of semantic knowledge. Event-related potential (ERP) data from normal individuals indicate that there are distinct mechanisms mediating at least post-lexical syntactic and semantic processes (Hagoort, Brown, & Osterhout, 2000). For instance, N400 (found 400 ms after an event) is sensitive to violations of semantic expectancy whereas P600 (found 600 ms after an event) is sensitive to syntactic violations.

ERP data cannot provide direct evidence of the neural sources of such effects. However, functional imaging studies on grammatical processing and encoding in native speakers (Hagoort et al., 2000) indicate a common syntactic component subserved by the left frontal area (a dorsal part of Broca's area and adjacent parts of the middle frontal gyrus). Finally, research on semantic representation of words identifies regions in the temporo-parietal region - the left extrasylvian temporal cortex and the left anterior inferior frontal cortex. A possible area associated with the integration of syntax and semantics lies in the anterior temporal pole (e.g., Dronkers, Redfern & Knight, 2000; Noppeney & Price, submitted).

Neurocognitive level of control
The devices for controlling language tasks are likely to be implemented by control circuits involving both frontal attentional and subcortical mechanisms (see, for example, Price, Green, & von Studnitz,1999). For instance, language task schemas may be mediated in part by subcortical neural mechanisms (e.g., in the basal ganglia, see also Crosson, Novack, & Trenery, 1988) with the level of activation modulated both by external input and by frontal systems including the anterior cingulate (but cf.Carter, MacDonald, Botvinick et al., 2000). If production in one language rather than another requires suppression of the schema for producing utterances in the non-selected language, it follows that there must be an executive input when individuals are required to switch languages on a designated cue (see Jackson, Swainson, Cunnington, & Jackson, 2001, for ERP evidence in a numeral naming task, and Hernandez., Dapretto, Mazziotta, & Bookheimer, 2001, for fMRI evidence in a picture naming task). It follows that damage to frontal structures should impair the ability to either maintain a given language or to avoid switching between languages.

Fabbro, Skrap, and Agliotti (2000) report the case of S.J. (a Friulian-Italian speaker) with a lesion to the left prefrontal cortex and part of anterior cingulate. S.J. showed normal comprehension in both Italian and Friulian and intact clausal processing in both languages. However, S.J. was unable to avoid switching into Friulian even when addressing an Italian speaker whom S.J. knew spoke no Friulian. Likewise when required to speak Friulian only, S.J. would switch into Italian. Switching can only be considered problematic when it arises inappropriately as in the case of S.J. (see Grosjean, 2001). We infer that this selective deficit in preventing a language switch, or in maintaining a monolingual output, was a consequence of a lesion in the anterior cingulate which precluded one language schema maintaining dominance over the other, though this outcome may also partly reflect an inability to maintain the communicative goal of speaking in the target language.

The need to suppress an alternate schema should also arise in the case of translation. Presentation of word in L1, say, will also trigger naming. Translation in this sense is analogous to a Stroop task in which

an habitual response must be suppressed. In order to translate from L1 to L2, say, individuals must inhibit an L1 production schema and activate the schema for L2. This schema level process can then modulate output from the lexico-semantic system. Functional imaging studies of performance in Stroop-like tasks all show increased activation in the anterior cingulate which may serve to modulate task schemas. Price et al. (1999; see also Klein et al.,1995) confirmed such an increase for translation.

If subcortical mechanisms in the basal ganglia are also implicated in selecting the relevant action then translation should also increase activation in these regions. The study by Price et al. (1999) confirmed increases in the relevant areas (the bilateral putamen and the head of caudate). Increases were observed too in the areas associated with articulation (SMA, a ventral region of the left anterior insula and the cerebellum), consistent with the notion that during translation responses associated with the input orthography must be inhibited.

Left subcortical lesions also lead to outcomes compatible with the present proposal. E.M. (Agliotti, Beltramello, Girardi, & Fabbro, 1996) suffered damage to the caudate nucleus and the putamen and had difficulty maintaining her native Venetan but would constantly switch back into Italian – a language learned only at school and rarely spoken. Damage to the basal ganglia could have limited her ability to activate the production schema in L1 in competition with that for L2. Such a difficulty would also lead to problems in naming even in the absence of lexical deficits in L1. Lesions located at the head of caudate nucleus in the left hemisphere can also elicit pathological language mixing in which no one language dominates (Abutalebi, Miozzo, & Cappa, 2000). Such cases are consistent with the idea that lexical representations are accessed under the control of frontal-basal ganglia circuits (see also Abutalebi et al., 2001).

Lesions in other areas can result in bilinguals displaying good comprehension in both languages but with an ability to speak in just one of them. Pötzl (1925) supposed that the left parietal area played a central role in language switching and that damage to it prevented switching from one language to another. However, there are patients with lesions in that area who show no such difficulties (see Paradis, 2001, p. 81). The precise role of the parietal regions in language switching still needs to be determined. Price et al. (1999) found increased activation during language switching not only in a region of Broca's area (BA 44) but also in the bilateral supramarginal gyri. The former region has been associated with phonemic segmentation and the latter with mapping orthography to phonology (see above) but since a given region may subserve a number of functions it is preferable to claim that both regions are activated in phonological processing tasks. Jackson et al. (2001) report a sustained increase in the size of an ERP component (the late positive complex) over the parietal region in a numeral naming task when individuals had to switch from naming in one language to naming in another. One interpretation of these two sets of data, consistent with the present proposal, is that the parietal region is involved in implementing a change in stimulus-response mapping driven by the task schema.

Capacity and resource

We can identify the capacity of a cognitive device with the average number of functionally intact neural units in the relevant neural mechanism (see Shallice, 1988, p. 233: 3). The capacity of the device for retrieving the phonology of words, for instance, may relate to the number of functioning cells in specific regions such as the posterior inferior frontal cortex (Broca's area). Alternatively, a better index of capacity may be the interconnectivity of neural units. By contrast, we can identify the resources with the metabolites, neurotransmitters or neuromodulators needed to operate the neural mechanism. These two identifications lead to the expectation that restricting the number of neural units (e.g., via a stroke) reduces capacity and so may impair performance. Likewise, reducing resource (e.g., through the loss of the cells producing a resource) to a given neural mechanism in the absence of any change in its capacity may impair performance. One line of support for this claim comes from unmedicated patients with Parkinson's disease. They have reduced dopamine levels in the prefrontal cortex (due to damage of the cells in the substantia nigra) and show deficits in working memory (e.g., Levin, Labre, & Weiner, 1989). Conversely, healthy adults given a dopamine agonist, show working memory improvements (e.g., Muller, von Cramon, & Pollmann, 1998).

Exploring recovery patterns

The key aim of this section is to consider how neuroimaging studies can be used to examine the causal basis of different patterns of recovery. I first review different possible mechanisms of recovery. I argue for the importance of Hebbian learning as a primary mechanism for the restitution of function and point to how different mechanisms of recovery might be distinguished in terms of their activation patterns. PET and fMRI allow us to assess such patterns. I describe these methods briefly and discuss some of the methodological prerequisites for studying patients using these methods. The final part outlines possible studies of patients with different patterns of recovery with a view to determining their causal basis.

Mechanisms of recovery

Recovery may be achieved via different mechanisms (e.g., Code, 2001; Papathanasiou & Whurr, 2000; Rickard, 2000) yielding recovery based on normal cognitive processes or not. Individuals might compensate for loss of function by developing a new strategy and so deploy different cognitive processes involving different neural regions. In other cases, there may be restitution of function within the same, or neighbouring, neural networks using learning processes identical to those that led to the formation of the network. Following Robertson and Murre (1999) we suppose that recovery from brain damage involves a process of Hebbian learning (Hebb, 1949).

In Hebbian learning two neurons or neuronal groups or circuits can reconnect if they are activated at the same time. Spontaneous recovery can arise in the case of well-connected networks with small lesions by random activation of one of the groups. Activation spreads through the network and any currently activated groups become reconnected. At the other extreme neural self-repair is impossible if circuits are too disconnected or lack neurones and only compensation is possible. At some intermediate point restitution is possible given suitable input. Intuitively, a less well connected network may be more sensitive to the precise nature of the inputs, and simulations discussed in Robertson and Murre (1999, pp.553-557) show that where an intermediate number of connections is lost, restitution does depend on the partially disconnected network receiving targeted (patterned) stimulation that allows appropriate reconnection (see Harley, 1996, and Plaut, 1996, for existence proofs of recovery of function in other types of networks).

Hebbian learning may be important in allowing function to be restored in areas surrounding the lesion site. Restitution of function may also be achieved by creating patterns of connectivity in neighbouring neural networks (neural plasticity). Neuroimaging can differentiate these alternatives, given the tasks are performed normally, by determining whether or not the regions activated are identical to those in normal bilingual controls. Where different regions are involved, neuroimaging will reveal activity in different areas for patients relative to normal controls.

What further factors (specific or general) may affect recovery of function? Damage to an area can sometimes suppress activity in a relatively remote undamaged area ("diaschisis") thereby temporarily impairing performance in tasks in which the functionality of that area is required. Restitution of function occurs when diaschisis is reversed and yields normal activity in that region. Behaviourally, recovery due to the reversal of diaschisis is also likely to occur earlier than recovery due to compensation.

Damaged circuits may also fail to recover function because of suppression from undamaged circuits that compete to control output. In this case, new lesions that reduce the activation of intact networks can lead to enhanced functioning in the network that was damaged initially by allowing Hebbian learning to take place (see Kapur, 1996, for a review of "paradoxical facilitation").

More generally, recovery may also reflect attentional factors. Deficits in attentional control (specifically, sustained attention) are strong predictors of recovery from brain damage. Attentional control may be important not simply because it is a factor in providing suitable input to the damaged areas but also because of its connection to the arousal system. Neurotransmitters (e.g., noradrenaline) associated with the arousal system are also strongly implicated in cortical plasticity. Hence the importance of wider cognitive, and pharmacological, assessments of bilingual aphasics in the study of recovery patterns.

Neuroimaging

Haemodynamic methods (PET and fMRI) rely on a close coupling between changes in the activation of a population of neurons and change in blood supply. A haemodynamic effect arises only when there is a change in the overall metabolic demand in a neuronal population. PET and fMRI track different signals. PET measures the decay of a short-lived isotope, which accumulates in a neural region in proportion to the

amount of blood flowing through that region. The most typical fMRI method indexes metabolic demand and hence relative neural activity by assessing the ratio of deoxyhaemoglobin to oxyhaemoglobin in the blood. Each method has advantages and disadvantages. Minimally, PET studies examine changes in patterns of activation by contrasting conditions that differ in the cognitive operation of interest. Trials of a certain type have to be blocked and the number of observations is restricted because PET involves the administration of ionising radiation. There are no such constraints in the case of fMRI. However, PET has the advantage that it is more or less equally sensitive to activity in all brain regions whereas fMRI signals are not. The magnetic signal is susceptible to factors other than blood oxygenation levels making it difficult to record from certain regions (e.g., the orbitofrontal region).

PET and fMRI offer important advantages for the study of recovery patterns. In order to explore recovery, we need to be able to chart changes. At a minimum, performance needs to be assessed after the acute phase and at some later time. Since both methods track changes in the whole brain, neural activity can be measured in the absence of overt manual or vocal response (Price & Friston, 2002). In consequence, processing can be assessed even when there is no ability to speak a language or even, apparently, to understand it. There may also be normal effects in one region but abnormal effects in another. So, for example, in listening to a story in a non-recovered language, activity in the auditory regions may be normal, but there may be abnormal effects in regions associated with semantics. At a later point in recovery, both regions may show normal response. Neuroimaging also allows us to consider how areas work together. In the normal case there is a functional integration of different areas. If control is normal, then the anterior-cingulate will modulate activity in the basal-ganglia normally. This circuit will provide a normal modulatory influence on the systems mediating word production. In contrast, diaschisis, for example, will yield abnormal patterns of activation. Büchel, Friston and Frith (2000) describe methods for examining effective connectivity ("the influence one neuronal system exerts on another", p.339) using structural equation modelling of the patterns of activation in different regions of interest.

Methodological cautions

Functional imaging studies of patients require that the performance level of the patient and normal controls is matched (Price & Friston, 1999). If the patient cannot perform the task, for instance, the corresponding neuronal responses will not be elicited. Further, if the patient performs the task but does so in a different way to normal bilinguals, the neuronal abnormality will covary with the cognitive abnormality and it is not possible to distinguish the cause of the neuronal abnormality (see Green & Price, 2001 for elaboration within the bilingual context). Differences in the way a task is performed may be detectable in the patterns of reaction time or error to stimuli of different types or in individuals' verbal protocols of what they are doing (Rickard, 2000).

Differences in activation pattern may also reflect differences in the relative difficulty of the task for the patient compared to a matched control even when the overt performance level is closely matched. One check here is to examine changes in activation patterns with variations in task difficulty. If restitution of function is occurring within normally activated regions, then, as relative difficulty decreases for the patient, there will be convergence with the patterns shown by normal controls. In contrast, if patients and controls show different stable patterns over variations in task difficulty then it is reasonable to infer that restitution is an outcome of neural plasticity (Rickard, 2000, p. 308).

Neuroimaging bilingual aphasics

Problems of control seem to offer a ready account of certain recovery patterns. The previously mentioned case of S.J. (Fabbro et al., 2000) offers a clear instance. Lexical representations are intact but there is a problem in ensuring that one language schema continues to dominate another. We attribute this difficulty to the lesion in the area of the anterior cingulate. Drugs (e.g., dopamine agonists) that modulate activity in the anterior cingulate, and so alter the resources available to it, may improve the ability of patients like S.J. to speak just one of their languages. It follows that the effective connectivity of regions associated with language control and regions associated with word production should once again be normal when the individual is switching between languages.

Alternating antagonism also seems suited to a pure control explanation (Green, 1986). A.D. (Paradis et al., 1982) was a French-Arabic speaker with a lesion in the temporo-parietal region of the left

hemisphere who presented with a specific form of alternating antagonism during the course of recovery. On one day she was able to speak French spontaneously but not Arabic. On the following day she was able to speak Arabic spontaneously but not French. However, on the day, for instance, when she was unable to speak Arabic spontaneously but could speak French spontaneously, she was able to translate into Arabic, suggesting that the lexical representations of that language were available for production. By contrast, on the same day that she could speak French spontaneously, she could not translate into it. This pattern of performance suggests that part of her problem lay in selecting between competing language task schemas of a given type (e.g., translating into L1 vs. translating into L2) once one had become dominant or perhaps in linking a non-dominant schema to the relevant lexical concepts. An exploration of the control problem might begin by considering the patients' ability to handle conflict tasks. For instance, with standard Stroop stimuli, the patient must suppress the normal reading response in order to name the hue in which a colour word is printed. Compared to normal bilingual controls, patients with alternating antagonism might show an abnormal pattern of correlated activity in the anterior cingulate when required to process such stimuli. As in the case of S.J. described earlier, if resource constraints underlie the problem, impaired performance should improve with the administration of a dopamine agonist.

These patterns of recovery are rare and so it is important to examine whether or not other more common patterns reflect problems with the control mechanism. In the next section we consider how neuroimaging studies may contribute to a better understanding of four such recovery patterns.

Parallel recovery

If control processes are intact then the regions associated with control and those associated with word production should modulate normally in both languages (i.e., there should be the normal pattern of effective connectivity) during word production and conflict tasks. Recovery will then primarily reflect restitution in the lexico-semantic system. Hebbian mechanisms provide more complete recovery where the network connections are better preserved and so the extent of peri-lesional activation is likely to be critical to the recovery of function (see Warburton, Price, Swinburn, & Wise, 1999, for evidence of peri-lesional activation in monolingual aphasic patients). If this is so, we can explore the link between the amount of preserved functional capacity in the peri-lesional tissue and different recovery patterns. Peri-lesional activity might initially be greater for both L1 and L2 in patients showing parallel recovery of both their languages compared to patients showing either selective or antagonistic recovery

Parallel recovery does not entail that both languages are recovered in the same manner. If there is restitution of function in one case but compensation in the other, then only the former language will show activation patterns during task performance (e.g., picture naming) indistinguishable from those of normal bilingual controls.

Differential, selective and antagonistic patterns of recovery

Viewed dynamically, a differential pattern may arise because use of one language rather than the other during the initial phase of recovery leads to a greater restitution of its network via Hebbian learning. Selective recovery, in contrast, may arise because progressive use of just one language consolidates its network and progressively isolates it from the other.

An alternative possibility is that both patterns reflect problems of control. For instance, there could be damage to the mechanism that selects the intended language, or a disconnection ,or disruption, of the link connecting the representation of the meanings of words and the units coding for language (or alternatively, between the units coding for language and the schema). Selective recovery, as opposed to differential recovery, reflects a greater difficulty in selecting one language over another. A third possibility is that the lexico-semantic system of one language is marginally more impaired than that of the other language and this impairment leads to a problem in controlling that language. This lack of control blocks its recovery via Hebbian mechanisms and isolates it from the other language. One way of advancing research in this area is to determine the nature of the representations accessible under these patterns of recovery.

Consider a selective pattern of recovery. Under a strong control hypothesis there is access to meaning but an inability to select lexical concepts in the non-recovered language. If there is access to the meaning of words but an inability to select lexical concepts in the non-recovered language, then individuals should still show semantic interference. Consider a Spanish-English bilingual with selective recovery of

Spanish performing the following task. Individuals are required to press one key provided an arrow points in one direction and another key if the arrow points in the opposite direction. Pairing the arrow with an incongruent direction word in English (e.g., ← RIGHT) as contrasted with a row of XXXXXs should slow reaction time reflecting an increase in response conflict. Such a conflict might also be detectable in specific brain regions such as the anterior cingulate. In the recovered, or better recovered language, where semantic access is preserved, as assessed by this on-line task, neuronal activity in bilingual aphasics should pattern in the same way as bilingual controls. In contrast, where there is evidence of semantic access but an inability to select the lexical concept for production, the regions associated with language control will activate abnormally - an example of "differential" diaschisis (cf. Price, Warburton, Moore et al., 2001).

If there is access to meaning is there also access to word form? Consider the task of determining whether or not a predesignated phoneme (e.g., "t") is present in the Spanish name of a pictured object such as a table, "mesa" in Spanish (Colomé, 2001; Hermans, 2000). Normal bilinguals are slower to reject the target phoneme if it is present in the translation equivalent compared to control trials where it is not. If segments of the word form of the non-recovered language are activated then patients should show this phoneme interference effect too. Further, there will be increased activation in the anterior cingulate on incongruent trials compared to control trials. If, on the other hand, there is no access to the word form, then bilingual aphasics will react similarly to monolingual controls.

Antagonistic recovery may be construed as a special case of selective recovery.
Why should the recovery of one language be impaired when a second language improves (e.g., Paradis & Goldblum, 1989). A control explanation of the antagonistic pattern of recovery might run as follows. Initial language use is probabilistically determined and does not directly reflect the rate at which recovery will occur for the two languages. A small difference in the rates of recovery, reflecting perhaps different degrees of damage to the lexico-semantic system, will be sufficient to induce different end states in the course of language use. An initially less dominant language schema becomes more and more dominant (via Hebbian learning), inhibiting use of the other language schemas and increasing the connectivity within the lexico-semantic system for the selected (and initially less well recovered) language (see above). Such an account presumes that after an initial phase in which competition between languages is weak, inhibiting the schemas for the better recovered language becomes more difficult.

As in the case of selective recovery, we can assess what information is available for the less recovered language. The arrow task described earlier offers a partial test. At an early stage of recovery, the patient would be tested in the most recovered language and we would look for evidence for response conflict from the less recovered language. At a later stage, the patient would be tested in the other language and we would look for the reverse effects. Comparable studies are possible for examining access to word form.

Implications for rehabilitation

Understanding recovery patterns has the practical goal of developing a principled basis for rehabilitation. The argument is that understanding the causal basis permits a targeted intervention. Rehabilitation in aphasia is a complex topic (see, for example, Code, 2000) and so my illustrations here are not intended as clinical prescriptions. Consider a pure control problem, as in the case of S.J. (above), a pharmacological intervention might be appropriate. But to the extent inappropriate switching stems from a failure to maintain the communicative goal (e.g., speak in L1), an appropriate intervention might seek to increase the person's capacity to sustain attention (see Robertson & Murre, 1999 for relevant techniques) and so maintain the communicative goal.

As indicated, different patterns of recovery may reflect the consequences of random stimulation to a damaged network. Such stimulation, at least for small lesions, can lead to an adaptive outcome (e.g., parallel recovery of both languages) but it can also lead to maladaptive outcomes for larger lesions: a given circuit may become connected to a formerly distinct circuit. One relevant factor here may be the extent to which individuals are aware of their speech output (Robertson & Murre, 1999). Shuren et al. (1995) report the case of a jargon aphasic who was unaware of his errors in the normal course of events but recognised error when listening to a tape recording of his own speech. This suggests that part of his problem was attentional -when the attentional load (involved in planning and in producing speech) was reduced he was able to recognise problems. Recognition of error is important because in its absence circuits involved in flawed production may become connected. In the case of bilingual aphasics, pathological mixing, may arise

out of an initial problem of control becoming entrenched. A possible intervention might then be to record, and to play back, the mixed speech and to create conditions, with short utterances at first, where the same language is maintained. Training individuals to overcome interference (e.g., in the standard Stroop task) may also be helpful.

One possible cause of selective recovery is a difficulty in binding lexical concepts to a language tag. Associating various kinds of language-specific contextual cues (e.g., music or scenes) to lexical items may help re-create the units coding for language and allow these to be linked to set an initial set of lexical concepts.

It is reasonable to expect that treatments that work for monolingual aphasics may be helpful for bilingual/polyglot aphasics (Juncos-Rabadán, Pereiro, & Rodríguez, 2002) but there is one possibility potentially available for the bilingual aphasic and used quite spontaneously. Bilingual aphasics with parallel recovery frequently self-cue and produce the correct word in the non-target language in order to retrieve the intended word (e.g., Juncos-Rabadán et al., 2002; Roberts & Le Dorze, 1998). One important area for future research will be to explore the benefits of implicit techniques such as priming. Can priming in a language that is accessible affect access to representations in a language previously inaccessible? What are the conditions for such an effect to occur?

Conclusions

Paradis (2001) asked: What determines the particular type of recovery? In the case of non-parallel recovery what selects a particular language for preferential recovery over another? Our answer is a call to action. We need to gain a more complete neurocognitive picture of patients in order to construct an adequate causal theory. With this aim in mind, this chapter has emphasized the usefulness of the contrast between representation and control.

In terms of the studies required, neuroimaging research on normal bilinguals, guided by adequate theory, is a critical prerequisite. Given our goal, we must develop tests that are maximally general - they should be readily convertible to different pairs of languages and be capable of being carried out at different stages in the recovery process.

Conjectures on the causal basis of recovery patterns will also be usefully complemented by simulation studies examining the conditions under which different patterns of recovery may arise. Such studies provide existence proofs and need to be constrained by data from neuroimaging studies to ensure their neurological plausibility.

Finally, the project to understand the patterns of recovery requires suitable databases. Reports of unselected cases of bilingual aphasics are rare. Ideally, we need to create researchable databases in which relevant data (lesion site, language background, performance on standardised tests, cognitive test performance, and functional imaging data) can be used to test and explore different models. In the short term, the most tractable way forward is through intensive studies of single cases combined with psycholinguistic and neuroimaging studies of normal bilinguals.

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