

Paying attention to reading errors in acquired dyslexia

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The important role of selective attention in normal reading is illustrated by a relatively rare form of acquired dyslexia known as 'attentional dyslexia'. Theories of the functional deficit underlying this disorder differ as to the locus of the attentional dysfunction. A recent study by Mayall and Humphreys has contributed to this debate by studying letter migration errors in an attentional dyslexic.

FM, a 68-year-old businessman, first noticed something strange about his reading when he blew his nose and had the experience of 'yellow letters spilling out over his head like beer out of a jug'. When he returned to what he was reading, he found 'the letters all over the place on the page'. Four weeks later he underwent an operation to remove a large tumour in his left parietal cortex. Although his symptoms improved following the operation, he continued to suffer a serious impairment in his ability to read. Perhaps the most curious aspect of his reading disorder was his frequent inability to identify the constituent letters of words that he read correctly. This characteristic brought him to the attention of the cognitive neuropsychologists Tim Shallice and Elizabeth Warrington. After studying FM's reading in more detail, as well as that of another patient (PT) with similar symptoms and lesion, Shallice and Warrington concluded that the reading disorder exhibited by these two patients was the result of a deficit of selective attention [1]. As a result, this type of acquired dyslexia has come to be known as 'attentional dyslexia'. Although it was a long time before any further cases of attentional dyslexia were described, several independent cases have now been reported [2–7].

Shallice and Warrington found that the ability of FM and PT to read isolated letters was more or less unimpaired. When a string of letters was presented, however, identification performance declined considerably. This 'flanker interference' effect was also observed for words flanked by other words. However, flankers of a different category (e.g. digits

flanking letters) did not interfere with identification. These observations led Shallice and Warrington to suggest that the perceptual classification processes in FM and PT were unimpaired, but that the transmission of information along a limited-capacity channel from perceptual to semantic processes was disrupted. When there are multiple items to transmit, an attentional control mechanism is required to select one item at a time; if this mechanism is damaged, attentional dyslexia results. This post-perceptual account can explain the flanker-category effect by assuming that channels are category-specific (e.g. there are separate channels for transmitting letters, words and digits).

Letter migration errors

When FM and PT attempted to read words flanked by other words, their reading errors were often blends of the correct word and the flanking words; for example, the word pair *win fed* was reported as *fin fed* [1]. Other attentional dyslexics have also exhibited errors of this sort, which are called 'letter migration' errors [5,8,9]. Such errors are also occasionally observed in normal readers, although usually only when words are presented very briefly and followed by a pattern-mask [8–12]. Migration errors in normal readers are interesting for a variety of reasons, including their implications for the coding of letter position (C.J. Davis and Bowers, unpublished). Letter migrations in attentional dyslexia pose a problem, however, for the post-perceptual account, as there is no reason why the correctly perceived word *win* should suffer interference from the letters of *fed* during transmission to the semantic system.

An alternative account of attentional dyslexia attributes letter migrations to the failure of a selection mechanism that normally attenuates the output of letter-level analyses outside a selected attentional 'window' in the visual field [13,14]. The inability to narrow this window to a single word enables letters in other words to participate in the

activation of word form units, which sometimes results in the perception of illusory words [10,11,13,14]. This account can potentially explain letter migration errors in both normal readers and dyslexics. In the case of normal readers, very brief exposures of multiple words might not provide sufficient time for an initially broad (low resolution) attentional window to be focussed to a narrow (high resolution) window [13,14]. In attentional dyslexics, the difficulty in focussing the attentional window appropriately is due to brain damage rather than temporal constraints. The additional assumption that recovery of location information requires focal attention [12,14] explains the tendency to report flanking items by mistake, and explains why words can be reported better than their constituent letters. When the flankers belong to a different category, however, location information is not critical (for example, a target letter surrounded by digits can be reported even if its exact location is uncertain).

According to one account of attentional dyslexia, then, the locus of the attentional deficit is relatively early (prior to word identification) [13,14], whereas another account posits a relatively late locus (post-perceptual) [1]. Both accounts can explain flanker interference effects, including the flanker category effect, but only the early-locus account can explain letter migration phenomena. For this reason, Mayall and Humphreys' recent study of letter migrations in an attentional dyslexic is highly pertinent to understanding the functional deficit underlying this disorder.

New evidence

Patient FL suffered carbon monoxide poisoning at the age of 58 years. This initially resulted in severe memory problems, low-level agnosia (which had improved by the time of testing), and difficulties in word recognition. An MRI scan showed bilateral occipito-parietal damage. FL showed typical signs of attentional dyslexia, including better identification of words than their constituent letters and impaired

identification of letters flanked by other letters.

In order to test FL's tendency to make letter migrations, Mayall and Humphreys generated a set of 80 three-letter words that could be paired such that exchanging the initial letters produced other English words (e.g. *led-bit*, *bed-lit*). They then presented these word pairs to FL (in lower case, with a single space separating the words), and asked him to read aloud both words. Over half of FL's errors (24/43) were responses that included a letter from the other word. As a baseline control, all of the 80 words were presented individually. Under these conditions, FL's performance was significantly better, although still impaired: he read 54/80 words correctly. Of the 26 errors, only 7 corresponded to what might be called 'pseudomigration' responses – responses that would have been classified as migration errors in the paired word condition. This is the first experiment to establish conclusively that an attentional dyslexic exhibits genuine letter migration errors. A tendency to report an illusory word like *bed* more often in response to *led bit* than in response to the isolated word *led* indicates that FL is making genuine letter migrations rather than straightforward visual errors.

Mayall and Humphreys also examined how migration errors were affected by manipulations of the display conditions. They found that increasing the spacing between the two words from a single space to five spaces reduced the frequency of letter migration responses from 56% to 39%. The incidence of migration errors was also reduced to 39% when the two words were presented in different typecases (i.e. one lower case, one upper case). In both of these manipulations, the reduced letter migration rate did not differ significantly from the rate of 'pseudomigration' responses in the single-word baseline condition.

As a further test of FL's ability to focus his attention, Mayall and Humphreys included some conditions in which FL was required to read only one of two words presented. When the target word was specified by spatial position (e.g. 'read the lefthand word'), the rate of migration responses decreased relative to the condition in which both words had to be read, although the decrease was not statistically significant. The decrease was more pronounced when the target word was specified by typecase (e.g. 'read the

uppercase word'), and the frequency of migrations in this condition did not differ significantly from the frequency of pseudomigrations in the baseline condition. A final interesting condition concerned FL's ability to read one word when he was required to name just the initial letter of the other word in the display. FL's reading was better in this condition than in the condition in which he had to read both words aloud, and was also better than in a condition in which he had to read a word and a single isolated letter, even though the responses were identical in these two conditions.

Do letter migrations in attentional dyslexics and normal readers have the same causes?

Although not specifically noted by Mayall and Humphreys, perhaps the strongest evidence that FL's letter migration errors have a different locus from that in normal readers was that he exhibited letter migrations despite the fact that the word pairs were selected so as to have no letters in common (e.g. *led-bit*). By contrast, normal readers make letter migrations only between words that share letters; for example, the first word of *cape cone* might be misreported as '*cope*' or '*cané*', but these migration responses do not occur for the pair *cape song* [9–11]. This 'surround-similarity' effect supports a lexical locus of letter migrations, whereby the attempt to recognize two words simultaneously results in the perception of illusory words (like '*cané*') that overlap with both of the presented words [10, 11, 14]. The absence of this similarity effect in FL points to an earlier locus, and suggests that different mechanisms underlie letter migrations in attentional dyslexics and normals.

What do letter migrations reveal about the functional deficit in attentional dyslexia?

The functional deficit in FL is inconsistent with a post-perceptual locus of attentional dyslexia [1]. According to that account, the spacing and typecase manipulations in Mayall and Humphreys's experiment should not have affected word identification. The finding that FL's word identification was disrupted by the presence of single-letter distractors is also inconsistent with the failure of transmission along category-specific channels. The notion of an excessively broad attentional window [13] provides a better account of FL's reading deficit. The fact that FL's migrations do not

depend on the similarity of the words within this window suggests a relatively early locus for these errors. In particular, an excessively wide attentional window could result in the loss of location information [14, 15], allowing letters to drift between words [12]. Within this framework, the effect of the physical properties of stimuli on the frequency of letter migrations might indicate that FL can exploit physical differences between words to focus his attentional window [5, 15]. Alternatively, these physical differences might make interactions between the words less likely; for example, it would be easier to reject the unusual percepts that result from mismatched combinations (e.g. *bed*).

In conclusion, the phenomenon of letter migrations offers insights into the functional deficit underlying attentional dyslexia, as well as the role of selective attention in normal reading. Although the errors made by attentional dyslexics and normal readers share some features, it seems likely that different causal mechanisms underlie the patterns of letter migration in these two populations. The evidence reported by Mayall and Humphreys reinforces this conclusion. However, their patient differs in certain respects from previously reported attentional dyslexics (for example, in not showing a flanker-category effect). Further research is required in order to determine whether there are different subtypes of attentional dyslexia, as well as the relationship between this type of reading disorder and other forms of dyslexia in which there is loss of information regarding letter location [16].

Acknowledgement

We thank Jeff Bowers for helpful comments on the manuscript.

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Decision-making deficits in drug addiction

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Core aspects of addictive behaviour can be explained in terms of abnormal decision-making. Using recording of autonomic function during performance of two gambling tasks, Bechara *et al.* have recently identified three distinct neuropsychological subtypes in individuals with substance dependence. These subtypes may reflect dissociable patterns of disruption in limbic brain circuitry.

Characterization of the cognitive mechanisms involved in chronic substance abuse has clear implications for pharmacological and rehabilitative treatment strategies, and also impacts upon our understanding of cognitive and motivational processing in the healthy brain. The relationship of decision-making to addiction has received particular attention recently. Decision-making involves, first, the assessment of reward and punishment associations to the available response options, and second, the selection of the option calculated to be optimal. In chronic substance abuse, decision-making appears to be altered in terms of the trade-off between short-term reward and the long-term negative consequences of drug abuse on health, employment and family life [1].

Neuropsychology of decision-making

Two recent studies by Bechara and colleagues [2,3] have investigated the decision-making profile in substance abuse, using a neuropsychological measure known as the Gambling Task (see Fig. 1). In this task, subjects are

presented with four decks of cards and must make a long series of decisions, picking from any deck on each go without knowing that there are 'safe' and 'risky' decks. Over 100 choices, healthy subjects typically develop a preference for the 'safe' decks (C and D) over the 'risky' decks (A and B). This learning has a physiological correlate in the development of an 'anticipatory' skin conductance response (SCR) prior to selection from the risky decks [4], which is assumed to reflect some awareness that the decision could result in high punishment.

Patients with brain damage in the ventromedial region of prefrontal cortex (PFC) perform poorly on the Gambling Task [4]. They continue to select from the risky decks even after accruing considerable financial debt, and fail to develop anticipatory SCRs to the risky choices, despite showing normal post-choice autonomic responses to reward and punishment. Their decision-making deficit has been labelled 'myopia for the future' – these patients are unable to use ongoing feedback to guide future responses, and therefore evaluate each decision in terms only of the immediate reward available. This pattern is consistent with the real-life cognitive difficulties seen in patients with lesions in this area. However, ventromedial PFC is unlikely to function in isolation: in particular, the ventral striatum and amygdala are known to be part of an extended neural network involved in motivational processing and goal-driven behaviour [5]. Amygdala damage also impairs performance on the

Gambling Task, but is associated with a distinct autonomic profile, with blunted responses to task punishment and to aversive noise [6].

Decision-making heterogeneity in substance-dependent individuals

Grant *et al.* have previously tested a group of multiple-drug users recruited from the community on the Gambling Task, and reported increased selection from the risky decks relative to controls [7]. Bechara *et al.* [2] extended these findings in a larger and more clinically severe group by administering the task with physiological monitoring to a group of 46 individuals, who were undergoing drug rehabilitation at an inpatient centre and who met DSM-IV criteria for alcohol or substance dependence. The group consisted of approximately similar numbers of alcohol-, cocaine-, and amphetamine-preferring poly-drug users. Performance was compared against a healthy control group and 10 patients with ventromedial PFC damage. Gambling performance in the substance-dependent group fell midway between the control and ventromedial groups, and differing significantly from both. This pattern might plausibly reflect a learning deficit, which causes subjects to persist with the risky decks for longer at the start of the task, before eventually acquiring the successful strategy – this would be compatible with the mild deficit reported previously in mania [8], for example. By contrast, the profile in the substance-dependent group resembled more closely a bimodal distribution: 63% of