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NEUROBEHAVIORAL GRAND ROUNDS—INTRODUCTION

Temporal processing deficits in letter-by-letter reading by Ingles and Eskes

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Based on his seminal contributions in 1891 and 1892, Dejerine is widely credited for initiating the investigation of acquired dyslexia. In the first contribution (1891) he described a patient who developed an inability to read and write after suffering an infarction of the left hemisphere. Dejerine designated the disorder "Alexia with Agraphia"; he attributed it to a loss of the "optical images" of words that were presumed to be supported by the angular gyrus. In 1892, Dejerine described a patient who was able to write but could not read what he (and others) had written (Dejerine, 1892). This disorder, variously known as Alexia without Agraphia, Pure Alexia or Agnosic Alexia, has been reported with regularity since the original account. Although subsequent reports have noted aspects of the disorder not emphasized in Dejerine's index case—pure alexia may be associated with surface (Patterson & Kay, 1982) or deep (Buxbaum & Coslett, 1996) dyslexia—the essential features of the disorder were well documented by Dejerine and the syndrome remains quite consistent with respect to its core phenomenology. Similarly, the pathologic substrate of the disorder has proven to be remarkably constant: the vast majority of patients with pure alexia have lesions involving the occipital lobe of the dominant hemisphere and either the forceps major or, less commonly, the splenium of the corpus callosum. The latter component of the lesion interrupts the white matter tracts connecting the visual association cortices of the right and left hemispheres.

Dejerine attributed the disorder to a disconnection of the visual input into the right hemisphere from the stored information regarding word forms mediated by the angular gyrus. Thus, on his account, the occipital lobe (or optic radiation) lesion prevents visual information from reaching the left hemisphere while the lesion of the white matter tracts prevents visual information arriving in the right hemisphere from reaching the left hemisphere language areas. Many subsequent investigators endorsed Dejerine's original account (e.g, Damasio & Damasio, 1983; Greenblatt, 1973); Geschwind (1965) considered the disorder to represent a prototypical "disconnection syndrome."

Investigations in recent years have focused on the nature of the processing impairment in pure alexia. Investigators have attributed the disorder to an impairment of stored information regarding words (e.g., Shallice & Warrington, 1980; Warrington & Langdon, 1994), difficulty accessing phonological codes (e.g, Arguin et al., 1998) or a deficit in the parallel processing of letter information (Patterson & Kay, 1982). Other investigators have argued that pure alexia is attributable, at least in part, to impaired visual processing (Kinsbourne & Warrington, 1962; Farah & Wallace, 1991; see Behrmann et al, 1998). For example, Buxbaum and Coslett (1996) reported investigations of a pure alexic subject who exhibited a restriction in the "spotlight" of attention as well as a deficit in maintaining an abstract representation of the right side of letter arrays. More recently, Fiset et al. (2005) reported data from 7 subjects with pure alexia and letter-by-letter reading whose reading was significantly influenced by the extent to which the letters of the words were visually similar. All of their subjects exhibited the classic word length effect—that is, the time taken to read a word aloud was directly related to word length. They found that when words were equated for the degree to which the letters were visually confusable, the word length effect was markedly reduced or eliminated. Fiset et al. (2005) suggested that pure alexia resulted from an "abnormally low signal-to-noise ratio for letter identification when visual attentional resources are spread over the entire surface of the target word, as is necessary with parallel processing."

In the present issue of *JINS*, Ingles and Eskes report additional data relevant to the hypothesis that pure alexia is attributable to a pre-lexical or "visual processing" deficit. Following on work of Kinsbourne and Warrington (1962) and Behrmann and Shallice (1995), they administered an RSVP paradigm in which 2 letters were presented in a sequence of digits to a pure alexic, GM, and 6 brain lesion controls without visual or reading deficits. Stimuli were presented at a rate of 6/second. One critical manipulation

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was the interval between the two letters (T1 and T2); on different trials, the interval varied between 1 (that is, the two letters were presented in succession) and 10 (that is, 9 digits separated the 2 letters). Subjects were asked to report both letters. Only those trials on which the first letter was named correctly were scored. Brain lesion controls exhibited the normal pattern of performance in that they performed relatively well with successive letters but exhibited a substantial decrement on trials with one or two intervening digits. This extensively studied period during which identification of successively presented stimuli is impaired is known as the "attentional blink" (Raymond et al, 1992).

GM differed from brain lesion control subjects in several respects. First, in the context of the RSVP task he exhibited a mild impairment in letter recognition. Second, his "attentional blink" was abnormally protracted relative to controls. Finally, he performed quite poorly with no intervening trials. That is, on trials with letters presented sequentially, he reported both letters on only approximately 5% of trials as compared to approximately 68% of trials for brain lesion controls. Thus, this elegant investigation confirms and extends previous demonstrations that visual processing deficits may play an important role in the pathogenesis of pure alexia. As confirmed by Ingles and Eskes, pure alexia may be associated not only with an impairment in the parallel but also serial processing of letters and, at least in some instances, other types of visual stimuli.

Although the results of this and previous studies provide strong evidence that pure alexia may be associated with visual processing deficits, there is one aspect of the syndrome that remains puzzling. Under appropriate conditions, many patients with pure alexia perform well above chance on lexical decision and semantic categorization tasks with briefly presented stimuli that they are unable to report (Landis et al., 1980; Shallice & Saffran, 1986; Coslett & Saffran, 1989). For example, Coslett et al. (1993) reported a patient with pure alexia who, when asked to perform semantic categorization tasks ("Is it an animal name?), performed well above chance with brief exposure but was unable to name the words; in contrast, when asked to name the words he typically reported only one or two letters and performed at chance on the categorization task. Based on these and other data, Saffran and Coslett (1998) suggested that the "implicit" reading is supported by the right hemisphere, whereas letter-by-letter reading that supports explicit word identification is a product of the left hemisphere.

If visual processing deficits preclude the parallel processing of letters for the purposes of word identification, how can patients with pure alexia perform well on semantic judgment tasks with briefly presented stimuli? Although a full discussion of this issue is beyond the scope of this introduction, it is clear that accounts of pure alexia that attribute the disorder to a deficit in visual processing—to which Ingles and Eskes have made a significant contribution—must accommodate data demonstrating preserved lexical access. Similarly, the claim that pure alexia reflects a disturbance in lexical or semantic processing must accommodate the evidence demonstrating that pure alexia is often associated with pre-lexical deficits.

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