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Rehabilitation of acquired alexia

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Introduction

The terminology of reading disorders can be a little confusing; the terms “alexia” and “dyslexia” are often used to label reading impairment. We use “alexia” throughout this article to refer to a reading disorder acquired after the ability to read has been successfully established. This is to differentiate these acquired disorders from the common but completely separate developmental disorder of “dyslexia” where the process of learning how to read normally is not fully established during development. We do not discuss developmental dyslexia here.

Acquired alexia has been traditionally divided into two types, “peripheral” and “central” depending on whether other language functions (such as auditory comprehension, speaking and writing) are spared or not. In the following sections we will review the behavioural profiles of each type of alexia and then the evidence for their rehabilitation. Both types are most commonly caused by stroke but almost any form of focal, multifocal or diffuse brain injury can cause a reading problem to develop. Generally the pathology is on the left side of the brain, especially for central alexias, usually the dominant hemisphere for language. When arterial territories are relevant, such as in embolic stroke, damage to brain structures supplied by the posterior cerebral artery is the most common cause of peripheral alexia, with damage to the territory of the middle cerebral artery more likely to cause central alexia. Neglect alexia is an exception to this general rule as it is usually caused by lesions affecting the right parietal lobe.

Peripheral alexia

Hemianopic alexia

This is the commonest type of acquired, peripheral reading disorder (Schuett *et al.* , 2008), and the one with most evidence supporting its rehabilitation. The conventional view is that this disorder is caused by visual field loss subsequent to damage to primary visual cortex. A characteristic visual field plot for a patient with hemianopic alexia is shown in figure 1E. If visual field loss encroaches close enough to the point

of fixation, the reader is robbed of important visual information required for the planning and guidance of text reading saccades (Leff et al. , 2000, McDonald et al. , 2006, Rayner and McConkie, 1976, Zihl, 1995). A typical patient will complain of slow text reading but may be able to read single words normally, or near-normally. They may be slower to read longer words that do not fit into their preserved visual field (Pflugshaupt et al. , 2009). The side and extent of the visual field deficit critically affects reading performance. Readers of English and other left-to-right written texts are much more affected by right-sided homonymous field defects than by left-sided deficits (Zihl, 1995) and vice-versa for texts written in the opposite direction e.g. Hebrew and Farsi (Pollatsek *et al.* , 1981). The severity of reading impairment is also inversely related to the number of degrees of visual sparing to the right of the visual fixation point; patients with only one degree of spared vision are twice as slow as those with three degrees of sparing and four times as slow as those with five degrees of sparing. This latter group read at almost normal rates (Pflugshaupt et al. , 2009, Zihl, 1995).

Patients with hemianopic alexia display abnormal patterns of eye-movements when reading (a typical text reading scanpath is shown in figure 1C). Visual field deficit has been shown to cause functional abnormalities in brain regions distant from the causative lesion, such as the parietal and frontal cortices which are involved in the generation and maintenance of reading scanpaths (Leff et al. , 2001). A Positron Emission Tomography study demonstrated that patients with hemianopic alexia do not show normal activation of their right frontal eye field while reading word arrays, despite making many more reading saccades than normal subjects. Controls or patients with a hemianopia that didn't cause hemianopic alexia had normal activity in this region while reading word arrays (Leff et al. , 2000). It has been argued that the severity of the disorder may be worse, and persist for longer, if there is concomitant damage to connections between visual cortex and brain regions that control eye-movements and visuo-spatial attention (Schuett, 2009, Schuett et al. , 2008)[a]

Given that hemianopic alexia is characterized by poorly executed reading saccades secondary to a visual field impairment, there are two obvious targets for rehabilitation: the visual field itself and the oculomotor system. There is some evidence that visual function can be restored to some extent after damaged by retro-chiasmal lesions. Given that the severity of hemianopic alexia is tightly linked to the extent of visual field damage, it follows that an excellent target for therapy would be the restoration of visual fields. Unfortunately, current evidence suggests that visual field therapy leads to only partial restitution of damaged visual fields, with the quality of vision in improved regions not being high enough to support normal reading. Reading requires high acuity (it is no accident that the commonest test of acuity uses letters: the Snellen chart) so anything other than full restitution of vision in the damaged field will result in no useful gains in reading behaviour: for a review of these therapies see (Schofield and Leff, 2009). For this reason, retraining eye-movements seems a more promising approach. There have been several excellent studies on this and the good news is that these programmes seem to work. Although they do not return reading speeds to normal, the effect size is an impressive 20-80% improvement on baseline text reading speeds. Therapies have produced worthwhile improvements over training periods in the region of 7-20 hours (Schuett, 2009),[a] this is a relatively short programme compared with, visual therapies aimed at improving blindsight (Huxlin et al. , 2009, Sahraie et al. , 2006).

The original therapy studies dating back to 1992 used moving text to rehabilitate text reading eye-movements. The text scrolls horizontally from right-to-left at a user specified speed and the reader practices reading aloud (Kerkhoff et al. , 1992, Zihl, 1995) under the auspices of a therapist. This type of "Times-square" text presentation induces an automatic form of eye-movement called small-field optokinetic nystagmus. "Times-square" presentation of text will speed up normal people's reading (at least on the animated text) (Kang and Muter, 1989). "Times-square" presentation also increases the reading speed of hemianopic patients, presumably via the same mechanisms; however when the patients return to static (normal) text the increased reading-speed persists. The reason for this is unclear, although the saccadic component of the automatically induced eye-movements is always in the direction of the defective saccades seen when normal text is read (left-to-right). A recent study employed this technique using distance therapy (the patients were sent video tapes with the rehabilitation materials recorded on them) in a group of severely alexic patients. The study was the first controlled trial for this type of therapy, including a 'sham' therapy period (half the patients practiced spot-the-difference puzzles in one therapy block rather than "Times-square" reading). The previously reported beneficial effects of this technique were confirmed (Spitzyna et al. , 2007). A more recent study has shown that other forms of oculomotor rehabilitation that require neither moving text, nor words, also have a beneficial effect (Schuett et al. , 2008). However, not all oculomotor therapies have equal effects; another study has demonstrated that practice with visual search tasks like the 'spot-the-difference' task used in the Spitzyna et al. study do not "carry over" and improve reading saccades (Schuett et al. , 2009)[b]. This suggests that a successful rehabilitation therapy needs to induce the same small amplitude saccades required for reading along a line of text, rather than the larger amplitude saccades required for visual search tasks.

It has been argued that oculomotor rehabilitation is best done with a therapist present to control the use of materials and help guide the patient with hemianopic alexia through therapy (Schuett, 2009)[a]. However, given the common occurrence of the disorder, the dispersion of patients with the syndrome and the lack of co-ordinated services for patients with hemianopia (at least in the UK) this is not always possible. Given the simple nature of the therapeutic material, it seems reasonable to use the worldwide web to deliver this therapy. We have recently set up a site where patients with hemianopic alexia can test their visual fields, reading speeds and gain access to the rehabilitation materials (moving text), for free: http://www.readright.ucl.ac.uk/

Pure and Global alexia

Pure alexia was the first of the peripheral alexias to be described (Dejerine, 1892) and is considered to be the archetypal form (Bub, 2003, Coslett, 2000, McCarthy and Warrington, 1990). Patients can generally recognize (and name) individual letters but have problems efficiently reading single words (Behrmann et al. , 1998, Behrmann et al. , 2001). Other language functions are generally considered to be normal or near normal. Despite being the focus of many studies, the exact deficit is still not agreed upon at either a psychological or an anatomical level, although it is clear that the dominant (left) fusiform gyrus has a key role to play (Binder and Mohr, 1992, Gaillard et al. , 2006). Evidence is emerging that the syndrome may not be as "pure" as was once thought; patients can usually be shown to have difficulties in processing numbers or non-linguistic stimuli (Behrmann et al. , 1998, Starrfelt et al. , 2009).

Patients with pure alexia can read but experience difficulty as word length increases, the so-called 'word-length effect'. This effect is taken as evidence that when these patients read, they: (1) dissemble words into their constituent letters, (2) identify these letters, and then (3) reassemble them in a laborious sequential manner - i.e. 'reading' the word via a process of reverse spelling. This process has been called letter-by-letter reading and the term has become synonymous with the condition of pure alexia, with pure alexic patients often labelled 'letter-by-letter' readers. We think this is a mistake, as it conflates a reading strategy with the underlying disorder, and the term should be reserved only for those patients with pure alexia who can definitely be shown to be using this strategy to aid reading. Many patients reported in the literature read too quickly to be employing a letter-by-letter strategy (Leff et al. , 2001). There is also good evidence that patients with pure alexia do have problems with letter identity, especially ones that are visually similar (e.g. i, l, j) (Fiset et al. , 2005, Fiset et al. , 2006). At the more severe end of the spectrum, where patients have a word length effect of several seconds for each letter added, (Leff et al. , 2001) pure alexia blends into global alexia. Patients with the global form either cannot recognize letters at all or make many errors naming them, despite often being still able to write normally or near normally, although these patients are more likely to have other language impairments and thus may straddle the peripheral/central division of alexia. These patients tend to have more extensive lesions including the fusiform gyrus and, usually, the deep white matter of the occipital lobe (Binder and Mohr, 1992). Global alexia is the most severe form of peripheral alexia; no-one has yet reported a patient who has been successfully rehabilitated.

There have been many attempts to rehabilitate patients with pure alexia but as the condition is considerably rarer than hemianopic alexia, patients with it tend to be studied individually or in small cohorts; there have been no randomized treatment studies. Perhaps the simplest method employed is to use mass-practice with stimuli designed to 'stimulate' the damaged system, in this case asking patients to read aloud a corpus of words many times. Recent case-reports show that this therapy may have a modest and probably genuine effect: patients' word-length effect improved and there was no speed/accuracy trade off (Ablinger and Domahs, 2009, Beeson et al. , 2005).

Another approach used to rehabilitate pure alexia is the use of cross-modal therapy such as kinaesthetic or motor cross-cuing therapy whereby patients practice tracing out the letters of the word they are trying to read (Lott et al. , 1994). There have been several, broadly positive, case-reports of this treatment with the most recent one using a small pool of words (Sage et al. , 2005). This approach may be best suited for patients at the slower end of the spectrum.

Neglect alexia

The commonest pattern of errors seen in neglect alexia is prefixes either being missed or substituted (e.g. /bone/ read as "one" or /bat/ read as "cat"). Patients with neglect alexia usually have a right parietal lesion and signs of a more general visuo-spatial neglect syndrome although the two can dissociate (Costello and Warrington, 1987). A cohort study of two neglect populations showed that when patients with neglect and neglect alexia were compared against those with neglect only the lesion tended to spread more ventrally within the non-dominant temporal lobe (Lee et al. , 2009). Therapeutic use of prism glasses to induce visuo-spatial adaptation may well help. A study of prism therapy in eight patients with neglect alexia demonstrated that after

therapy patients' initial fixations moved towards the (neglected) left side of the word and correct reading responses improved by a third (from 45% to 60%) (Angeli et al. , 2004). The authors did not report how long this effect lasted for after rehabilitation ended; however, a review of prism adaptation for generalized visuo-spatial neglect suggests that the effects may last for hours to several weeks (Redding and Wallace, 2006), meaning subjects may have to 'top-up' their rehabilitation therapy once in a while.

Attentional alexia

Patients with attentional alexia complain of letter crowding or migration, sometimes blending elements of two words into one. The lesion usually affects the left parietal lobe (Warrington et al. , 1993). Patients perform better when word stimuli are presented in isolation rather than flanked by other words and letters. Using a large magnifying glass may help as this should reduce the effects of flanking interference from nearby words; however, no trials of this or indeed any other therapy for left parietal syndromes have been published.

Central alexia

These patients have a general language disorder; that is, they have alexia and aphasia. Dejerine's term for this pattern of acquired alexia, still in use today particularly among neurologists, is alexia with agraphia (he referred to pure alexia as alexia without agraphia) (Dejerine, 1892). In the acute and post-acute phase, these patients and their therapists are often primarily concerned with deficits of auditory speech perception or speech output, with the concurrent alexia and its treatment taking a back seat. This is perfectly understandable as for most people, speaking is socially more important than reading. If alexia persists into the chronic phase, however, patients often complain about their alexia as much as any other part of their language disorder. Unfortunately the evidence base for how to treat central alexia is rather weak. The different types of central alexia are classified according to the dominant error pattern when reading words aloud (the subtypes are discussed in the following section). In our experience these classifications often overlap; pure cases where the patient will only make one type of error and this type alone are rare. Also, some error patterns do not have their "own" syndrome despite being common, e.g. semantic errors, errors of perseveration.

Phonological alexia

Patients with phonological alexia read familiar words (both regular and irregular) by virtue of preserved lexical-semantic representations, the so-called 'direct' route to reading. However, because they rely heavily on prior exposure to extant word forms they often fail to read novel words and legal or pronounceable non-words (e.g. /mune/) (Tree, 2008). They are said to suffer from phonological alexia because they cannot use the 'indirect' or spelling-to-sound (phonological) route for reading. When reading real words, factors such as concreteness (e.g. nouns are more concrete than verbs) and frequency (high frequency: increased previous exposure) affect performance. Treatment can be directed at the sublexical process (grapheme to phoneme correspondences), the lexical process (whole word reading), or both. Sublexial therapies usually target components of a 'key word' to instantiate the grapheme-phoneme correspondence and then example words are used to reinforce this association. One approach has been to pair difficult words such as function words

and verbs with homophones (e.g.: ‘be; with ‘bee’; (Friedman et al. , 1993)) or with words that share an initial phoneme (‘much’ with ‘mud’; (Lott et al. , 2008)). This approach has met with some success. In contrast, lexical reading treatments usually involve practicing spoken production of written stimuli either at a single word or supra-word level. In a recent study with one patient with phonological alexia, a technique called "multiple oral rereading" was used. This improved reading of both known and novel texts, with the reading speed for the latter increasing from 34 to 44 wpm (Cherney, 2004). Interestingly, comprehension of text also showed some improvement after therapy.

Deep alexia

Deep alexia has all the features of phonological dyslexia, but, in addition, patients make numerous semantic errors (e.g.: reading /cat/ as “dog”) (Crutch and Warrington, 2007) and have particular problems with function words, even very common ones such as /and/ or /of/ (Jones, 1985). These effects are striking: a patient may read /off/ as “on” and be unable to read the word /of/ at all. Some authors consider deep alexia to be a more severe version of phonological alexia and similar treatment approaches are often used for both disorders, although most studies focus on the deep alexia aspect. In one recent study a patient was trained using a bigraph-syllable pairing method (e.g.: pa - /pae/), and demonstrated an increase in reading performance. The improvement was especially marked when reading abstract words, perhaps because the training enabled the patient to employ phonemic cues when little semantic information is available (Kim and Beaudoin-Parsons, 2007). Pairing words with pictures to strengthen semantic access also appears to improve reading, a recent study showed an improvement of 80% on trained versus untrained items in a patient with deep alexia (Ska et al. , 2003).

Another approach is to try repetitive multimodal stimulation (Cherney, 2004) or to pair multiple outputs or inputs (cross-modal therapy). In one example, the patient repeatedly read sentences of personally-relevant scripts and, at the same time, practised writing a subset of the words from the same scripts (Orjada and Beeson, 2005). This had quite an impressive effect, the patient improved from a baseline reading speed of 23 wpm to 60 wpm and there was some generalization on untreated material. In a separate study two patients were treated using a similar approach, concentrating on a set of 40 words. Both patients made significant gains on the treated material but showed little generalization to untrained items (Beeson et al. , 2005).

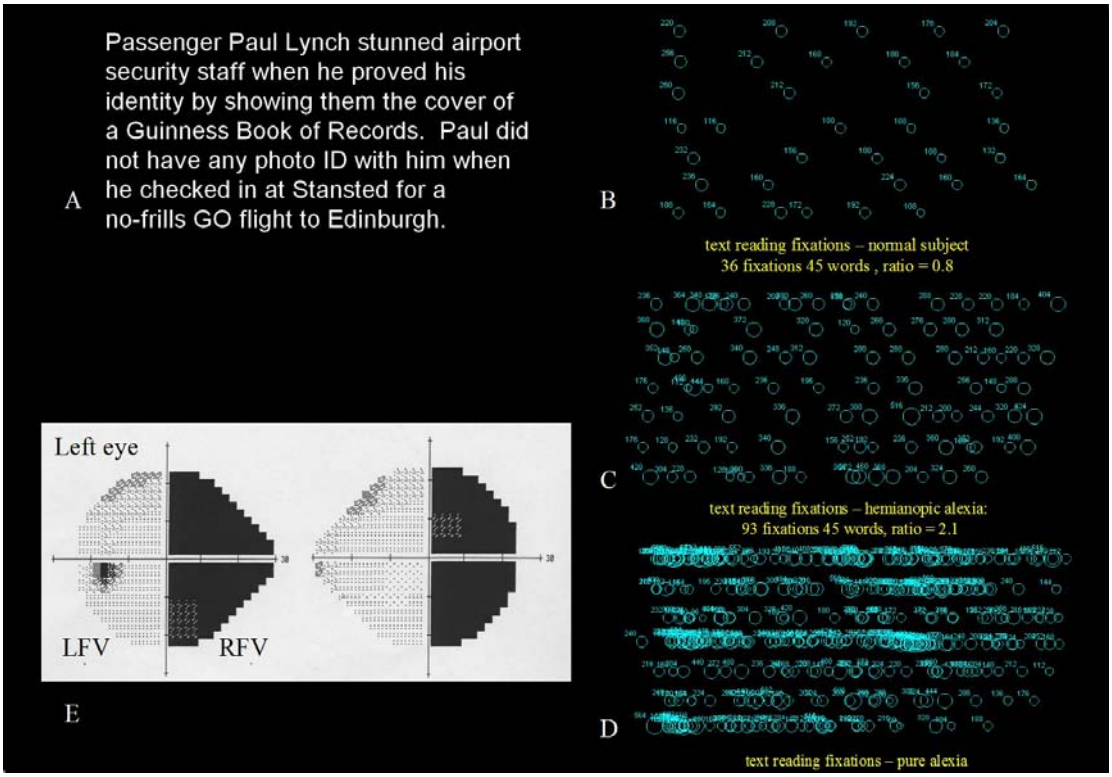
Surface alexia

Surface alexia is the converse of phonological alexia. Here the phonological route is preserved (allowing patients to read non-words), but the direct route is impaired. This leads to problems reading irregular words e.g. colonel pronounced “col-on-el”. Treatments typically retrain orthographic knowledge of specific words, by pairing the specific written word with relevant semantic information (a picture or definition of word) to enhance the use of the lexical-semantic form of reading (Behrmann and Byng, 1992, Byng and Coltheart, 1988). Words of particular relevance to the patient are usually selected because generalization to untrained items is unlikely.

Summary

Acquired reading impairments are common. The evidence base for treatment is strongest for the most 'peripheral' of the alexic syndromes, hemianopic alexia. A variety of well-designed studies have shown the efficacy of mass practice therapies aimed at improving reading saccades into the blind field. For the other syndromes, including the central alexias, the evidence base is weaker; primarily because the current literature is dominated by small group studies. Current best practice involves therapists identifying each patient's pattern of impairment and devising behavioural strategies to improve reading performance based on the extant literature.

Figure



Subjects read the sample of text (A) silently while eye-movements were measured. Reading fixations are shown for a normal subject (B), a patient with hemianopic alexia (C) and a patient with pure alexia (D). The blue circles show where the fixations occurred in two-dimensional space (the normal subject makes five fixations for the first line and four for the second), and how long they lasted for (in milliseconds, relative size of the circle). The patient with hemianopic alexia makes more fixations than the normal subject; these are of a longer duration. The patient with pure alexia makes many more fixations than the patient with hemianopic alexia, having to make more fixations than there are letters in a word with many refixations. Again, the average time for each fixation is increased compared to both other subjects. The visual fields of a (different) patient with hemianopic alexia are shown (E). The subject maintains fixation at the crosshairs while individual spots of light are projected eccentrically. Missed locations are shown in black. LFV = left visual field, RVF = right visual field

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