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TRAITEMENTS SÉQUENTIELS ET PARALLÈLES DANS LA DYSLEXIE LETTRE-PAR-LETTRE: ÉTUDE DE CAS ET SIMULATION CHEZ LE LECTEUR NORMAL

par

Stéphanie Fiset

Département de psychologie,

Faculté des arts et sciences

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ÉTUDE DE CAS ET SIMULATION CHEZ LE LECTEUR NORMAL

présentée par Stéphanie Fiset

a été évaluée par un jury composé des personnes suivantes:

[redacted]
Sylvie Belleville, Ph.D.
président-rapporteur

[redacted]
Martin Arguin, Ph.D.
directeur de recherche

[redacted]
Yves Joanette
membre du jury

[redacted]
Alain Desrochers
examinateur externe

[redacted]
Tony Leroux
représentant du doyen de la FES

RÉSUMÉ

La dyslexie lettre-par-lettre (LPL) est une dyslexie acquise de l'adulte caractérisée principalement par un grand effet de longueur de mot sur les temps de réponse. Une atteinte nuisant au traitement parallèle du mot serait à l'origine de ce trouble de lecture, obligeant les dyslexiques LPL à recourir à une stratégie séquentielle pour identifier le mot.

Dans le premier article de cette thèse, qui a pour objectif de spécifier davantage la nature du déficit limitant le traitement parallèle dans la dyslexie LPL, quatre études ont été effectuées auprès de IH, un dyslexique LPL. Ces expériences ont permis d'observer 1) qu'une augmentation paramétrique du nombre de voisins orthographiques de la cible (i.e. des mots de la même longueur que la cible mais qui en différent par une seule lettre) provoque une réduction linéaire des temps de lecture de ce patient (Exp. 1). 2) La seconde expérience a toutefois montré que l'effet de longueur de mot ne serait pas un artéfact d'une absence du contrôle du voisinage orthographique (VO) (Exp. 2); 3) Les données indiquent également des effets significatifs mais indépendants de longueur de mot et de confusabilité de lettres (i.e. la similarité visuelle des lettres constituant le mot avec les autres lettres de l'alphabet) (Exp. 3). 4) Finalement, l'effet facilitateur du VO est aboli lorsque les lettres composant les mots possèdent une haute confusabilité (Exp. 4). Ces observations suggèrent qu'à l'origine de la dyslexie LPL on retrouverait un trouble dans l'encodage visuel des lettres qui produirait un niveau excessif de bruit dans l'activation des représentations lexicales-orthographiques de la cible lorsque les lettres sont traitées en parallèle. Ceci empêcherait l'identification de la cible et obligerait donc les patients à recourir à un traitement séquentiel des lettres afin d'identifier le mot.

Les second et troisième articles de cette thèse concernent la

simulation de la dyslexie lettre-par-lettre chez des lecteurs neurologiquement sains. En se basant sur l'hypothèse que la dyslexie LPL résulte d'une atteinte de l'encodage de l'information avant l'accès aux représentations orthographiques (hypothèse périphérique), il peut être prédit que l'effet de longueur de mot pourrait être provoqué chez des lecteurs neurologiquement sains si les stimuli sont visuellement dégradés. Les expériences du second article de cette thèse ont permis de conclure que la réduction de contraste de luminance entre les stimuli et le fond sur lequel ils sont présentés n'est pas une dégradation suffisante pour reproduire la dyslexie LPL chez des lecteurs normaux. Dans le troisième article, la simulation de la dyslexie LPL et de l'effet de longueur de mot caractéristique de ce trouble a été tentée en employant une dégradation visuelle des stimuli sensée reproduire les conditions d'encodage parafovéal des stimuli et / ou le traitement initial des stimuli par l'hémisphère cérébral droit par les dyslexiques LPL (en raison de l'hémianopsie homonyme droite souvent observée chez ces patients): les stimuli ont été dégradés en réduisant leur contraste de luminance et en retirant leurs hautes fréquences spatiales. Ces conditions de dégradation ont entraîné 1) des effets de confusabilité de lettres et de longueur de mot et 2) une interaction entre la longueur de mot et la fréquence lexicale. Par contre, 3) l'interaction rapportée plus haut chez IH entre la confusabilité de lettres et le VO n'a pas été reproduite dans les conditions de dégradation employées. Ces résultats suggèrent que ce type de dégradation visuelle ne correspondrait que partiellement au déficit visuel présent dans la dyslexie LPL car elle aurait en effet empêché les sujets d'accéder à de l'information visuelle accessible aux dyslexiques LPL quand ces derniers focalisent leur attention sur les lettres individuelles.

Mots clés: Alexie pure, Dyslexie lettre-par-lettre, Dyslexie, Lecture, Reconnaissance de mots, Simulation chez le lecteur normal, Traitement visuel

ABSTRACT

Letter-by-letter (LBL) dyslexia follows left occipito-temporal lesions in previously adult normal readers. This reading disorder is characterised by a large word length effect on reading latencies, contrarily to what is observed in neurologically intact readers. It seems that these patients present a limitation in the normal parallel processing of words, forcing them to resort to a sequential letter-by-letter strategy to permit word identification.

The goal of the first article of this thesis is to specify the nature of the disorder limiting parallel letter processing in these patients. Four experiments were conducted on patient IH, a LBL dyslexic. Exp. 1 showed that an increase in the number of orthographic neighbours of the target (i.e. words of the same length differing by just one letter; N size) produced a large linear reduction of word naming latencies. Exp. 2. showed that the word length effect is not an artifact of the lack of control of N size and that this effect reflects the strict sequential processing of letters performed by LBL dyslexics. Exp. 3 showed significant but independent effects of word length and of letter confusability (i.e. the visual similarity of the constituent letters of the target word with other letters of the alphabet). Finally, Exp. 4 indicated that the facilitatory effect of N size is abolished when words are made of letters having a high confusability. These results imply that an impairment in the visual encoding of letters, producing an excessive noise level in the activation of lexical-orthographic representations when letters are processed in parallel, is responsible for LBL dyslexia. This impairment prevents word recognition based on parallel letter processing, thereby forcing sequential processing in order to achieve overt identification of the target.

The second and third articles of this thesis focus on the simulation

of LBL dyslexia in neurologically intact readers. Following the hypothesis that this reading disorder is caused by an impairment in visual encoding prior to the access to abstract orthographic representations, it can be predicted that the word length effect characteristic of LBL dyslexia could be elicited in normal readers if stimuli are visually degraded. The experiences of the second article of this thesis lead us to conclude that the luminance contrast reduction between the word and its background on its own fails to reproduce LBL dyslexia.

In the third article, we attempt to simulate, in normal readers, LBL dyslexia and the word length effect characteristic of this disorder by employing a visual degradation of the stimuli reproducing the condition of parafoveal exposure and / or the initial processing of stimuli by the right cerebral hemisphere (because of the right homonymous hemianopia observed frequently in these patients). Stimulus degradation was performed by reducing the contrast of the stimuli against the background and by removing their high spatial frequencies. These conditions of degradation reproduced in normal readers 1) the letter confusability and word length effects present in LBL dyslexics and 2) the interaction between word length and lexical frequency, as in some LBL dyslexics. However, 3) the interaction between letter confusability and N size (i.e. a facilitatory effect only for low-confusability targets) observed in IH was not found. Our results suggest that the type of visual degradation employed here may only partially correspond to the visual deficit present in LBL dyslexia and that this degradation may have prevented the normal readers to access visual information, which is available to LBL dyslexics when they focus their attention sequentially on individual letters.

Key words: Pure alexia, Letter-by-letter dyslexia, Dyslexia, Reading, Word recognition, Simulation in normal readers, Visual processing

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LISTE DES ABRÉVIATIONS

cd/m^2 = candelas par mètre carré

cm = centimeters

Exp. = experience

i.e. = c'est-à-dire

LBL = letter-by-letter

LPL = lettre-par-lettre

M.A.I. = modèle d'activation interactive

ms = millisecond

msec = milliseconde

N = neighbourhood

RT = Reaction time

SD = standard deviation

SMT = Stimulation magnétique transcranienne

VO = voisinage orthographique

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CHAPITRE PREMIER:

INTRODUCTION GÉNÉRALE

Le langage écrit, apparu il y a moins de 5000 ans, est une des plus impressionnante habileté cognitive de l'humanité. En effet, l'analyse de petits traits variés sur une surface quelconque (tissu, papier, pierre…) permet la transmission d'un vaste monde de connaissances à travers le temps et l'espace. Depuis quelques siècles, beaucoup d'êtres humains possède les capacités de décoder ces signes (la lecture) et peut ainsi accéder à un bagage immensément riche sur les plans culturels et scientifiques. L'apparition toute récente de cette habileté sur le plan évolutif rend toutefois son apprentissage long et ardu, probablement car aucun substrat cérébral spécifique n'était génétiquement déterminé à cette faculté cognitive (McCandliss, Cohen et Dehaene, 2003). Dans nos sociétés modernes, où la communication écrite de l'information est primordiale à la pratique de la plupart des professions et des loisirs, une entrave dans le développement de cette fonction cognitive chez l'enfant ou une perte de cette capacité chez l'adulte a des conséquences dévastatrices sur la qualité de vie.

Les dyslexies acquises du lecteur adulte, dont les premières observations scientifiques remontent à plus d'un siècle, ont été séparées en deux groupes : les dyslexies périphériques et les dyslexies centrales. Dans les premières, la lésion neurologique et fonctionnelle serait localisée au niveau du traitement et de l'analyse visuels du mot écrit. Elles comprennent entre autres la dyslexie attentionnelle, la dyslexie de négligence et l'alexie pure. Une lésion des processus plus tardifs et de « haut niveau » impliqués dans le traitement des mots provoquerait les dyslexies centrales. Ce terme regroupe traditionnellement la dyslexie de surface (induite par une sur-utilisation des règles de conversion graphème-phonème, ce qui produit des erreurs de régularisation), la dyslexie phonologique (résultant d'une atteinte des mécanismes de conversion graphème-phonème) et la dyslexie profonde (qui se distingue

de la dyslexie phonologique par de nombreuses erreurs appelées paralexies sémantiques).

Le présent travail s'intéresse plus précisément à une des dyslexies dites « périphériques » : la dyslexie lettre-par-lettre. Celle-ci se produit le plus souvent consécutivement à une atteinte d'origine vasculaire des aires cérébrales postérieures gauches et est caractérisée par la présence d'un important effet de longueur de mot en lecture. Les études rapportées ici s'attardent plus particulièrement 1) à caractériser et à préciser les limites et les capacités de deux processus contribuant à la lecture chez ces dyslexiques, les traitements parallèles et séquentiels des lettres et 2) à reproduire ce trouble de lecture chez des sujets neurologiquement sains.

Plusieurs sections composent cette introduction : en premier lieu, la dyslexie lettre-par-lettre, ainsi que les troubles fonctionnels et les lésions anatomiques qui y sont associés, seront décrits. Par la suite, les hypothèses explicatives de ce trouble de lecture seront présentées. Suivront ensuite une présentation des phénomènes de lecture implicite et des capacités résiduelles de traitement parallèle chez les dyslexiques lettre-par-lettre et une explication du rôle du traitement séquentiel dans la lecture de ces patients. L'impact de variables telles le voisinage orthographique et la confusabilité de lettres sera également présenté. Finalement, le cadre théorique nécessaire à la simulation de ce trouble de lecture sera discuté. Les études composant ce travail et leurs hypothèses sous-jacentes seront présentées au fur et à mesure dans le texte.

I. Description clinique et anatomique de la dyslexie lettre-par-lettre

Le terme « dyslexie lettre-par-lettre » est fréquemment employé pour parler de l' « alexie pure », décrite initialement par Jules Dejerine

(1892), car il décrit qualitativement très bien la lecture résiduelle chez ces patients. Le critère diagnostique de ce type de dyslexie est une lecture très lente et ardue caractérisée par un grand effet de la longueur du mot sur les temps de réponse. On observe en effet une augmentation linéaire du temps requis pour la reconnaissance du mot en fonction du nombre de lettres qu'il comprend (Patterson et Kay, 1982). L'effet de longueur de mot peut varier grandement selon les patients, c'est-à-dire de 500 msec à plusieurs secondes par lettres (Arguin et Bub, 1993; Bowers, Bub et Arguin, 1996b; Farah et Wallace, 1991; Friedman et Lott, 2000; Patterson et Kay, 1982; Reuter-Lorenz et Brunn, 1990; Warrington et Shallice, 1980). Ceci contraste grandement avec la performance de lecteurs neurologiquement sains, chez qui l'effet de longueur de mot est absent ou très faible (environ 6 msec par lettre) (Forster et Chambers, 1973; Frederiksen et Kroll, 1976; Lavidor et Ellis, 2002 ; Weekes, 1997). La présence d'un tel effet de longueur de mot chez les dyslexiques lettre-par-lettre a été interprétée comme reflétant une incapacité à appréhender visuellement les mots de façon parallèle, i.e. à encoder toutes les lettres du mot simultanément. L'information orthographique pourrait par contre être accédée en parallèle lorsque l'entrée est auditive (Miller et Swick, 2003). Ces patients perdent donc l'habileté de lire rapidement et efficacement les mots isolés et les textes, ce qui a un impact dramatique sur leur qualité de vie.

L'alexie pure résulte de lésions occipito-temporales gauches survenant chez un lecteur adulte auparavant normal. Damasio et Damasio (1983) ont identifié trois types de lésions associées à l'alexie pure : l'alexie de type I serait provoquée par une atteinte de l'artère cérébrale postérieure gauche, endommageant la substance blanche de la jonction occipito-temporale, le cortex occipital médial et la partie gauche du splénium et forceps major. Le type II résulterait d'un dommage au cortex calcarin ou aux radiations optiques. Dans le type III, les lésions

seraient similaires à celles décrites dans le type I, mais l'aire atteinte semble plus restreinte. Binder et Mohr (1992) ont observé des lésions communes impliquant le gyrus fusiforme et le lobe temporal ventral gauche chez des dyslexiques lettre-par-lettre. Ces observations sont corroborées par Beversdorf, Ratcliffe, Rhodes et Reeves (1997) qui eux aussi ont relevé chez un dyslexique lettre-par-lettre une atteinte du gyrus fusiforme et de la substance blanche associée.

La dyslexie lettre-par-lettre est le plus souvent associée à une hémianopsie homonyme droite, mais quelques cas font exception (Greenblatt, 1973; Henderson, Friedman, Teng et Weiner, 1985; Leff, Crewes, Plant, Scott, Kennard et Wise, 2001; Montant, Nazir et Poncet, 1998; Verstichel et Cambier, 1997). Selon Damasio et Damasio (1983), les alexies pures de types I et II sont accompagnées d'hémianopsie homonyme droite, alors qu'une préservation totale ou partielle du champ visuel droit caractérise les alexies du type III. Une achromatopsie et d'autres problèmes visuels ont été retrouvés chez certains de ces patients qui ne présentaient pas d'hémianopie homonyme droite.

Aucun autre trouble cognitif n'a été associé de façon systématique à la dyslexie lettre-par-lettre. Mais on retrouve parfois chez ces lecteurs d'autres déficits du langage écrit, tels une dyslexie de surface (Bowers et al., 1996b; Friedman et Hadley, 1992; Patterson et Kay, 1982) ou une dysgraphie de surface (Behrmann et McLeod, 1995; Rapp et Caramazza, 1991). Fréquemment, une anomie légère ou un léger trouble de mémoire verbale sont également observés (Behrmann, Plaut et Nelson, 1998; Bowers, Arguin et Bub, 1996; Hanley et Kay, 1996; Perri, Bartolomeo et Silveri, 1996).

II. Hypothèses explicatives de la dyslexie lettre-par-lettre

Plusieurs hypothèses ont été proposées pour expliquer la dyslexie lettre-par-lettre, mais la cause fonctionnelle de ce trouble de lecture est encore l'objet de débats importants. Les hypothèses peuvent être regroupées en deux types de propositions, périphériques ou centrales, selon la localisation supposée du dommage fonctionnel.

A. Hypothèses périphériques

Plusieurs considèrent qu'un problème survenant avant l'activation des représentations orthographiques du mot expliquerait la dyslexie lettre-par-lettre. Une première observation en ce sens provient de Patterson et Kay (1982). Selon eux, le trouble fonctionnel des dyslexiques lettre-par-lettre consisterait en une limite de la capacité à traiter rapidement et en parallèle des formes visuelles les lettres composant le mot. L'accès à la forme visuelle du mot ne pourrait donc se faire que par le biais d'une lente et séquentielle identification de lettres. D'autres auteurs ont plutôt proposé que les dyslexiques lettre-par-lettre posséderaient des ressources attentionnelles spatiales insuffisantes, ce qui les obligerait à une stratégie séquentielle de gauche à droite (Rapp et Caramazza, 1991). Une hypothèse attentionnelle est également soulevée par Price et Humphreys (1992) qui ont observé chez un patient une difficulté à orienter adéquatement l'attention dans le mot.

Par ailleurs, plusieurs données convergent vers l'hypothèse que le trouble fonctionnel dans la dyslexie lettre-par-lettre serait une atteinte du processus d'identification des lettres qui empêcherait l'activation normale des représentations orthographiques du stimulus (Arguin et Bub, 1993; Arguin et Bub, 1994; Behrmann et Shallice, 1995; Kay et Hanley, 1991;

Reuter-Lorenz et Brunn, 1990). Une atteinte de l'imagerie mentale visuelle spécifique au matériel orthographique a également été observée chez un patient identifiant les mots en nommant isolément chacune des lettres les composant (Bartolomeo, Bachoud-Levi, Chokron et Degos, 2002). Par contre, d'autres observations tendent à indiquer que les dyslexiques lettre-par-lettre présenteraient un déficit perceptif généralisé qui affecterait l'habileté de traiter rapidement des stimuli visuels complexes et non seulement des stimuli orthographiques (Behrmann et al., 1998; Chialant et Caramazza, 1998; Farah et Wallace, 1991; Friedman et Alexander, 1984; Mycroft, Behrmann et Kay, soumis; Perri, Bartolomeo et Silveri, 1996; Sekuler et Behrmann, 1996). Ces études ont en effet démontré des troubles visuo-perceptifs chez des dyslexiques lettre-par-lettre, ce qui a amené certains auteurs à émettre l'hypothèse que ce trouble de lecture serait attribuable à une atteinte visuelle précoce et non-spécifique. Cette hypothèse d'une relation causale reste cependant à démontrer empiriquement.

B. Hypothèses centrales

Certains auteurs, au contraire, croient que le déficit fonctionnel responsable de la dyslexie lettre-par-lettre se trouve à un stade plus tardif dans la lecture de mots. Selon Warrington et Shallice (1980), ce trouble de lecture proviendrait d'un dommage au système orthographique spécifique à la forme, qui empêcherait l'utilisation de représentations lexicales et induirait le patient à employer une stratégie d'épellation. D'autres études indiquent que l'accès aux formes orthographiques des mots serait relativement intact, mais que l'accès aux formes phonologiques du mot serait affecté (Bowers et al., 1996a).

C. Variabilité chez les dyslexiques lettre-par-lettre

Il est important de souligner qu'il existe une grande variabilité dans les performances observées chez les patients présentant une alexie pure, ce qui peut expliquer la variété des explications retrouvées dans la littérature. En effet, alors que certains patients sont presque incapables de lire, d'autres ont recours à une stratégie de lecture séquentielle, lettre-par-lettre, soit en épelant une à une les lettres composant le mot (Bartolomeo et al., 2002), soit en semblant utiliser cette stratégie « mentalement » (Behrmann et al., 1998, Arguin, Bub et Bowers, 1998). Et même parmi ce dernier groupe de lecteurs plus rapides, des différences importantes apparaissent dans la taille de l'effet de longueur de mot (voir Behrmann et al., 1998 pour plus de détails). Ceci a amené certains auteurs à proposer qu'une grande variété de déficits fonctionnels peuvent nuire à la lecture et mener à l'utilisation d'une stratégie séquentielle lettre-par-lettre (Price et Humpheys, 1992). Dans la même veine, Hanley et Kay (1996) ont eux aussi suggéré qu'il existe différents types d'alexie pure en fonction de déficits fonctionnels variés, ce qui les a incité à proposer une division des dyslexiques lettre-par-lettre en deux catégories (type 1 et type 2) selon leurs habiletés de lecture. Finalement, Chialant et Caramazza (1998) ont émis l'hypothèse que différents troubles perceptifs peuvent contribuer à la production de la dyslexie lettre-par-lettre.

III. Lecture implicite et traitements sériel vs parallèle dans la dyslexie lettre-par-lettre

Les explications « périphériques » ou « centrales » de la dyslexie lettre-par-lettre doivent tenir compte d'un phénomène particulier, retrouvé chez plusieurs de ces lecteurs : des capacités résiduelles du traitement parallèle normalement employé en lecture ont été observées

chez les dyslexiques lettre-par-lettre. Il a en effet été démontré, chez quelques patients, des effets de lecture implicite supposant un traitement parallèle des lettres dans le stimulus, c'est-à-dire que ces lecteurs pouvaient démontrer un accès lexical adéquat pour des mots sans pouvoir toutefois les identifier ouvertement (Arguin, Bowers et Bub, 1996; Arguin et al., 1998; Bowers et al., 1996a; Bowers et al., 1996b; Bub et Arguin, 1995; Bub, Black et Howell, 1989; Coslett et Saffran, 1989; Coslett, Saffran, Greenbaum et Schwartz, 1993; Reuter-Lorenz et Brunn, 1990; Shallice et Saffran, 1986). Shallice et Saffran (1986) ont été les premiers à montrer que des décisions lexicales ou sémantiques pouvaient être réalisées par ces lecteurs à un niveau d'exactitude supérieur au hasard et ce, même si la durée d'exposition est trop brève pour permettre l'identification du stimulus par une stratégie lettre-par-lettre. Ces capacités ont été observées également par Coslett et Saffran (1989) et par Bub et Arguin (1995) chez cinq autres dyslexiques lettre-par-lettre. Il a également été démontré que dans ce type de tâche l'effet de longueur de mot est absent, ce qui suggère fortement que la lecture implicite est réalisée par un traitement parallèle des lettres composant la cible.

La plupart des dyslexiques lettre-par-lettre ne présentent pas ces habiletés de lecture implicite, mais certains montrent quand même des indications d'une activation lexicale cachée. Bowers et al. (1996a) ont en effet observé chez IH, un dyslexique lettre-par-lettre incapable de lecture implicite, un effet d'amorçage lorsqu'une amorce orthographiquement identique à la cible mais visuellement différente (e.g. amorce = robe, cible = ROBE) était présentée trop brièvement (100 msec) pour être identifiée par le sujet. On retrouve cet effet lorsque des amorces imprimées en lettres minuscules précèdent des cibles majuscules ainsi que lors de la présentation d'amorces écrites en alternance majuscule - minuscule (par exemple RoBe). Ceci montre qu'une activation lexicale cachée est présente chez ce patient et participe à la lecture explicite des stimuli.

Une autre indication de la présence d'une activation lexicale cachée dans la dyslexie lettre-par-lettre provient de la présence d'un effet de supériorité du mot. En effet, la reconnaissance d'une lettre présentée brièvement puis suivie d'un masque est facilitée quand cette lettre fait partie d'un mot, par opposition à lorsqu'elle est présentée isolément ou à lorsqu'elle fait partie d'une séquence de lettres sans signification (Bub et al., 1989; Reuter-Lorenz et Brunn, 1990). Encore une fois, ce phénomène dénote un traitement parallèle du mot qui se distingue du traitement séquentiel des lettres requis pour l'identification explicite des mots chez les dyslexiques lettre-par-lettre.

Les phénomènes de lecture implicite et d'activation lexicale cachée suggèrent que, conjointement au processus séquentiel lettre-par-lettre que ces lecteurs utilisent pour reconnaître consciemment un mot, ils auraient aussi accès à un processus opérant beaucoup plus rapidement mais qui ne peut produire l'identification explicite du stimulus de façon fiable. Un double processus dans la dyslexie lettre-par-lettre a donc été proposé : 1- une voie résiduelle orthographique abstraite endommagée, ne pouvant plus produire l'identification explicite des mots mais restant partiellement active, et qui serait responsable de l'amorçage abstrait et de la lecture implicite; et 2- une voie compensatoire, spécifique à la forme et opérant sur des lettres individuelles, qui serait nécessaire pour la reconnaissance explicite de mots (Arguin et al., 1998; Friedman et Lott, 2000).

Mais une question importante peut être posée : le traitement parallèle des lettres dans la dyslexie lettre-par-lettre a-t-il un véritable impact sur la performance en reconnaissance de mots de ces dyslexiques? En effet, Coslett et Saffran (1994; Coslett et al., 1993) ont suggéré qu'il existe une opposition fondamentale entre les processus impliqués dans la reconnaissance explicite de mots et la lecture implicite et ont argumenté que cette dernière n'était possible que lorsque les patients ne tentaient pas

d'identifier le mot ouvertement. Toutefois, Howard (1991) a montré, dans une épreuve de lecture de mots, que les réponses correctes les plus rapides (dans la distribution totale des temps de réponse des dyslexiques lettre-par-lettre) résultent du traitement parallèle de l'ensemble des lettres constituant le mot. Le traitement parallèle induisait par contre un haut taux d'erreurs. Les résultats obtenus par Howard nous indiquent donc que le processus d'activation lexicale qui serait responsable de la lecture implicite permettrait en fait aux dyslexiques lettre-par-lettre de reconnaître occasionnellement un mot sans utiliser de traitement séquentiel.

IV. Modèles explicatifs

Les bases anatomiques et fonctionnelles de la lecture implicite, de l'activation lexicale cachée et de la stratégie compensatoire lettre-par-lettre font toujours l'objet de débats. Deux modèles explicatifs opposés ont été proposés : l'hypothèse de l'implication de l'hémisphère droit émise par Coslett et Saffran (Coslett et Saffran, 1994; Saffran et Coslett, 1998) et le modèle intégré de Behrmann et al. (1998).

A. Hypothèse d'une contribution hémisphérique droite

Coslett et Saffran proposent que l'hémisphère droit jouerait un rôle important dans la lecture chez les dyslexiques lettre-par-lettre, contrairement à la latéralisation hémisphérique gauche retrouvée habituellement chez les lecteurs normaux (Beauregard, Chertkow, Bub, Murtha, Dixon et Evans, 1997; Cohen, Martinaud, Lemer, Lehéricy, Samson, Obadia, Slachevsky et Dehaene, 2003; Howard, 1992). Une telle proposition est logique considérant que la plupart de ces patients doivent

initialement encoder les stimuli visuels par le biais de l'hémisphère droit, en raison de l'hémianopsie homonyme droite. Ainsi, pour la majorité des dyslexiques lettre-par-lettre, l'information doit tout d'abord transiter par l'hémisphère droit avant d'être transférée à l'hémisphère gauche par le biais de connections calleuses.

Coslett et ses collègues suggèrent par contre que la contribution de l'hémisphère droit ne se résumerait pas qu'à l'encodage perceptif des stimuli, mais qu'il jouerait également un rôle primordial dans les phénomènes de lecture implicite observés chez plusieurs dyslexiques lettre-par-lettre. Les hypothèses de ces auteurs sont basées 1) sur les études portant sur les lecteurs normaux en faisant usage d'une présentation tachistoscopique latéralisée des stimuli (Bub et Lewine, 1988; Chiarello, Liu, Shears et Kacinik, 2002; Eviatar, Menn et Zaidel, 1990; Iacoboni et Zaidel, 1996; Mohr, Pulvermüller, Zaidel, 1994; Rayman et Zaidel, 1991) et 2) sur les observations menées auprès de sujets callosotomisés et hémisphérectomisés gauches (Baynes, Tramo et Gazzaniga, 1992; Eviatar, Menn et Zaidel, 1990; Patterson, Vargha-Khadem et Polkey, 1989; Reuter-Lorenz et Baynes, 1990) qui montrent certaines capacités de l'hémisphère droit en lecture. Coslett et ses collègues avancent que l'hémisphère droit médierait la lecture implicite observée chez les dyslexiques lettre-par-lettre, alors que l'hémisphère gauche de ces patients serait responsable de la lecture séquentielle. La démonstration la plus impressionnante de ces auteurs a été leur étude de l'impact de la stimulation magnétique transcranienne (SMT) chez un dyslexique lettre-par-lettre (Coslett et Monsul, 1994). Une telle stimulation appliquée à l'hémisphère droit a en effet provoqué une diminution très marquée de la performance de lecture à voix haute de leur sujet, alors qu'une stimulation de l'hémisphère gauche n'a pas eu d'impact sur les taux d'erreurs. Toutefois, une critique peut être apportée à leur manipulation : cette dernière aurait été encore plus convaincante si les

auteurs avaient mesuré la disparition, lors de la stimulation de l'hémisphère droit, de phénomènes de lecture implicite chez leur patient. D'autres résultats viennent par contre ajouter un poids aux observations de Coslett et collègues. Bartolomeo, Bachoud-Levi, Degos et Boller (1998) ont étudié une patiente devenue dyslexique lettre-par-lettre à la suite d'une lésion occipito-temporale gauche et qui a perdu ses capacités résiduelles de lecture lors d'une seconde lésion « en miroir » à l'hémisphère droit. Cette observation supporte l'hypothèse que l'hémisphère droit contribue aux capacités résiduelles observées chez les dyslexiques lettre-par-lettre.

B. Modèle intégré et unifié

En contraste avec l'hypothèse avancée par Coslett et Saffran, Behrmann et al. (1998) ont proposé qu'il n'est pas nécessaire d'invoquer la contribution de mécanismes hémisphériques droits pour expliquer certains phénomènes présents dans la dyslexie lettre-par-lettre, tels la lecture implicite. Ils ont suggéré que ces observations pouvaient être justifiées sur la base d'un unique système intégré de lecture impliquant les hémisphères cérébraux gauche et droit, qui médieraient à la fois la reconnaissance explicite des mots et les phénomènes de lecture implicite. Plus précisément, ces auteurs ont élaboré une théorie « intégrée » de la dyslexie lettre-par-lettre à partir des modèles d'activation interactive (M.A.I.) de reconnaissance de lettres et de mots élaborés par McClelland et Rumelhart (McClelland et Rumelhart, 1981 ; Rumelhart et McClelland, 1982). [Behrmann et ses collègues affirment aussi que leur explication pourrait s'appliquer dans le cadre d'un modèle connexionniste à représentations distribuées (Seidenberg et McClelland, 1989; Plaut et al., 1996)]. Ces modèles connexionnistes ne seront pas décrits ici, pour des raisons de concision.

Behrmann et ses collègues suggèrent, sur la base d'une recension exhaustive de la littérature, que le trouble fondamental dans la dyslexie lettre-par-lettre est un déficit perceptif généralisé qui entraîne une dégradation de l'input visuel. Dans le modèle M.A.I., ce déficit serait localisé au niveau de l'extraction des traits primitifs de lettres ou entre ce niveau et celui de la représentation des lettres. Malgré cette atteinte perceptive généralisée, une activation parallèle des lettres composant les stimuli serait produite, mais elle serait trop faible ou alors partielle, ce qui nuirait à la reconnaissance des mots. Le système doit donc se résoudre à employer une stratégie séquentielle pour augmenter l'activation des lettres individuelles et ainsi parvenir à l'identification des stimuli. Selon ces auteurs, ce traitement séquentiel ne serait pas le reflet d'une stratégie anormale consécutive à une lésion cérébrale, mais il serait plutôt l'emploi d'une stratégie normale d'augmentation du nombre de fixations oculaires quand des difficultés en lecture de texte sont rencontrées (Just et Carpenter, 1987).

Même si l'activation des représentations orthographiques lexicales produite par la faible activation parallèle initiale des lettres est insuffisante pour permettre l'identification explicite des mots, la cible devrait normalement être davantage activée que ses compétiteurs. Pendant le traitement séquentiel, l'activation des lettres individuelles se propagerait dans le système de traitement orthographique visuel et augmenterait ainsi l'activation au niveau des représentations lexicales. Simultanément, l'activation au niveau des représentations lexicales est retransmise par rétroaction aux représentations des lettres, ce qui en facilite la reconnaissance. Les auteurs suggèrent également que les mots de haute fréquence et les mots de haute imageabilité seront plus activés, et qu'ainsi plus de rétroaction des niveaux supérieurs sera transmise au niveau des lettres, en améliorant ainsi l'identification. Le système permettrait donc

des réponses plus rapides et moins sujettes à erreurs pour les mots de haute fréquence et de haute imageabilité.

Le modèle élaboré ici expliquerait ainsi, selon Behrmann et collègues, l'interaction qui est observée parfois chez les dyslexiques lettre-par-lettre entre la longueur du mot et les effets lexicaux et sémantiques [voir la recension effectuée par Behrmann et al. (1998)]: puisque les temps de réponse sont plus élevés pour les mots les plus longs, les représentations lexicales et sémantiques de ces stimuli ont plus de temps pour recevoir l'information provenant des niveaux inférieurs. Par conséquent, cela produit des effets de rétroaction plus importants sur la performance. Les auteurs en concluent donc qu'il est possible et logique d'observer, chez un même dyslexique lettre-par-lettre, un déficit perceptif conjointement à des effets lexicaux et sémantiques en lecture. Leur modèle fournit également une explication de la variabilité observée chez les dyslexiques lettre-par-lettre. En effet, si le déficit perceptif est trop sévère, l'activation initiale sera trop faible pour se rendre aux niveaux supérieurs et pour produire une rétroaction facilitatrice vers les niveaux de traitement responsables de l'identification de lettres. Finalement, par opposition avec la théorie hémisphérique droite de Coslett et collègues présentée plus haut, l'hypothèse de Behrmann et al. n'attribue pas les effets lexicaux et sémantiques à un hémisphère spécifique. Les auteurs suggèrent plutôt que la lecture des dyslexiques lettre-par-lettre et les effets de haut niveau observés chez ces patients sont sous-tendus par le système de lecture prémorbide des deux hémisphères cérébraux, bien qu'il soit affecté par la lésion cérébrale. Behrmann et al. apportent des données provenant de plusieurs dyslexiques lettre-par-lettre à l'appui de leur proposition.

Le modèle de Behrmann et collègues contraste également avec celui proposé par Arguin et al. (1998). En effet, bien que tous deux supposent

l'action simultanée d'un traitement parallèle résiduel et d'un traitement séquentiel lettre-par-lettre, Arguin et al. supposent que ces deux mécanismes sont séparés, alors que Behrmann et al. proposent que ces deux processus opèrent dans un seul système de lecture. Un appui à la proposition de Arguin et collègues vient de l'observation, par Bowers et al. (1996a), d'une dissociation entre l'amorçage de lettres et de mots chez un dyslexique lettre-par-lettre (IH). Chez ce patient, un effet facilitateur de l'amorçage orthographique abstrait était présent pour les mots, mais absent pour les lettres. Ces résultats suggèrent que deux mécanismes séparés de lecture seraient présents chez ce patient. La question de la séparation ou de l'intégration des traitements séquentiels et parallèles n'est donc pas encore résolue.

V. Impact du voisinage orthographique dans la dyslexie lettre-par-lettre

Comme il a été présenté plus haut, il semblerait qu'un traitement parallèle résiduel contribue à l'identification de mots chez les dyslexiques lettre-par-lettre. Des variables reflétant le traitement parallèle des mots pourraient donc influencer la performance chez les dyslexiques lettre-par-lettre. Notamment, on peut donc supposer que ces patients présenteraient un effet de voisinage orthographique, comme les lecteurs normaux.

Les voisins orthographiques d'une cible sont d'autres mots de même longueur qui diffèrent de la cible par une seule lettre (exemple PORT vs. PORC ou MORT). Il est bien connu que les mots possédant plusieurs voisins orthographiques sont lus plus rapidement que les autres mots par les lecteurs neurologiquement sains (Andrews, 1989, 1992 ; Arguin et al., 1998 ; Carreiras, Perea et Grainger, 1997; Sears, Hino et Lupker, 1995). Cet effet facilitateur du voisinage orthographique proviendrait du traitement effectué au niveau de l'encodage

orthographique (Andrews, 1989; 1992; Arguin et al., 1998; Carreiras et al., 1997; Coltheart, Davelaar, Jonasson et Besner, 1977; Pugh, Rexer, Peter et Katz, 1994; Sears et al., 1995). En effet, les mots présentés visuellement n'activeraient pas seulement leurs propres représentations lexicales-orthographiques internes, mais également celles de leurs voisins. En retour, cette activation lexicale produit une rétroaction facilitatrice sur le niveau précédent responsable de l'identification des lettres constituant le stimulus (McClelland et Rumelhart, 1981; Plaut et al., 1996). Ainsi, quand un mot possède plusieurs voisins orthographiques, les représentations de lettres compatibles avec la cible reçoivent plus de rétroaction facilitatrice que lorsqu'un mot possède peu de voisins. Cette rétroaction facilitatrice sur l'identification de lettres serait responsable de l'effet facilitateur du voisinage orthographique sur la lecture explicite de mots.

Pour produire l'effet facilitateur du voisinage orthographique, il semblerait que le traitement parallèle des lettres constituant le mot soit nécessaire i.e. que l'ensemble des lettres doit être transposé en même temps sur les représentations des mots. En effet, il a été démontré que si on empêche le traitement parallèle des lettres, l'effet de voisinage orthographique disparaît ou alors devient inhibiteur. Snodgrass et Minzer (1993) ont présenté des mots « fragmentés » (i.e. dont les lettres étaient partiellement masquées) à des sujets neurologiquement sains et leur ont demandé soit d'identifier le mots en plusieurs tentatives successives (les mots étaient alors présentés de plus en plus «clairement » aux sujets), soit de produire une seule réponse. Dans la première condition, un effet peu substantiel du voisinage orthographique a été observé sur les taux erreurs, alors que dans la seconde tâche un effet inhibiteur important du voisinage orthographique a été remarqué, les sujets commettant significativement plus d'erreurs pour les mots possédant plusieurs voisins orthogra-phiques. De plus, Pugh et al. (1994) ont observé que le voisinage orthographique inhibait la performance au lieu de la faciliter lorsqu'une

lettre permettant de distinguer le mot de ses voisins était retardée de 100 msec par rapport à la présentation des autres lettres. Un argument intéressant provient également d'une étude d'Arguin et Bub (1997) : ils ont étudié une patiente présentant un déficit d'attention visuo-spatiale (et ne pouvant donc traiter certaines positions de lettres) qui présentait un important effet inhibiteur du voisinage orthographique lorsque les lettres qu'elle ne pouvait traiter étaient celles qui distinguaient la cible de ses voisins. L'effet facilitateur du voisinage orthographique semble donc reposer sur le traitement simultané et parallèle des lettres composant le stimulus.

Arguin et al. (1998) ont apporté une contribution supplémentaire à ces observations, en examinant l'effet du voisinage orthographique chez un dyslexique lettre-par-lettre, IH. Ils ont montré que le voisinage orthographique a un effet facilitateur sur la lecture explicite de mots de ce patient, ce qui appuie l'hypothèse d'une contribution du traitement parallèle à la reconnaissance de mots dans ce trouble de lecture. Toutefois, la taille de l'effet facilitateur du voisinage orthographique était beaucoup plus importante que chez les lecteurs normaux. De plus, cet effet n'était pas modulé par la fréquence lexicale des stimuli, comme chez les sujets contrôles : au lieu d'être présent uniquement pour les mots de basse fréquence, comme pour les lecteurs normaux, l'effet de voisinage orthographique chez IH était présent pour tous les mots, peu importe leur fréquence. L'effet facilitateur du nombre de voisins orthographiques a également été répliqué dans la seule autre étude où l'impact de cette variable a été testé (Montant et Behrmann, 2001). Les résultats obtenus par Arguin et al. (1998) révèlent donc que le traitement parallèle chez leur patient n'est pas totalement normal et que ce traitement anormal produirait une activation des représentations lexicales internes trop faible pour rendre possible l'identification explicite des mots. Ce serait pour

cette raison que les dyslexiques lettre-par-lettre auraient recours à un traitement séquentiel des lettres pour l'identification explicite des mots.

VI. Contribution de ce travail à la compréhension des traitements parallèle et séquentiels

Afin de mieux comprendre les causes fonctionnelles de la dyslexie lettre-par-lettre, il semble donc primordial de spécifier davantage quelle est la nature du trouble limitant le traitement parallèle chez ces patients et obligeant le recours à une stratégie séquentielle. La première étude de ce travail tente de contribuer à cet objectif en étudiant, chez un dyslexique lettre-par-lettre, le lien entre le voisinage orthographique et les traitements parallèle et séquentiel observés dans ce trouble de lecture et en observant comment cette variable est modulée par certains paramètres, tels la longueur du mot et la similarité visuelle des lettres.

Les deux premières expériences servent 1) à vérifier que l'effet de longueur de mot observé dans la dyslexie lettre-par-lettre est bien causé par le traitement séquentiel compensatoire et 2) à invalider l'hypothèse que cet effet de longueur ne serait qu'un artéfact du voisinage orthographique, étant donné l'importante corrélation entre la longueur des mots et le nombre de voisins orthographiques (Weekes, 1997) (les mots les plus courts possèdent en effet plus de voisins orthographiques que les mots plus longs).

La troisième expérience de cette étude explore l'impact de la similarité visuelle des lettres sur l'effet de longueur de mot par le biais d'un nouveau paramètre développé dans notre laboratoire, la confusabilité de lettres. La confusabilité de lettres est en fait la similarité de la forme entre une lettre particulière et les autres lettres de l'alphabet.

Les valeurs de confusabilité sont dressées à partir de matrices empiriques de confusion de lettres. Ces matrices ont été élaborées à partir de résultats obtenus chez des sujets neurologiquement sains dans des épreuves où ils doivent identifier des lettres qui sont présentées très brièvement puis masquées (Gilmore, Hersh, Caramazza et Griffin, 1979; Loomis, 1982; Townsend, 1971; Van der Heijden, Malhas et Van den Roovaart, 1984). Ainsi, la valeur de confusabilité pour une lettre précise est la probabilité qu'un lecteur normal produise une erreur en l'identifiant dans cette condition de présentation. La confusabilité de lettres d'un mot est la moyenne de la confusabilité de toutes les lettres qui le composent. Les résultats préliminaires obtenus dans notre laboratoire suggèrent que cette variable augmenterait significativement les temps de réponse des sujets dyslexiques, sans influencer toutefois les performances des sujets neurologiquement sains.

La prédiction sous-jacente à cette expérience est que si un déficit perceptif est présent chez les dyslexiques lettre-par-lettre, les temps de lecture d'un dyslexique lettre-par-lettre seront significativement augmentés lorsque les stimuli sont composés de lettres ayant une haute confusabilité avec d'autres lettres de l'alphabet, par rapport à lorsque les mots sont formés de lettres possédant une basse confusabilité. Il est également attendu que ces résultats contrastent avec les performances des sujets normaux qui ne devraient pas être affectés par cette variable. Un appui à cette hypothèse provient des données de Perri et al. (1996) qui ont observé, chez un dyslexique lettre-par-lettre, des erreurs d'identification de lettres où la réponse erronée partageait plusieurs traits avec la lettre cible (par exemple, leur patient disait « p » au lieu de « q ») et ce, tant en lecture de mots que dans des tâches où les lettres étaient présentées isolément. Patterson et Kay (1982) avaient déjà obtenu des résultats similaires, alors que leurs sujets dyslexiques lettre-par-lettre semblaient sensibles au chevauchement des caractéristiques visuelles des lettres.

Notre expérience vise également à explorer si la similarité visuelle des lettres a un impact sur le traitement séquentiel des lettres, ou si l'effet inhibiteur de cette variable est dû aux capacités limitées du traitement parallèle. Farah et Wallace (1991) ont déjà noté une sensibilité des dyslexiques lettre-par-lettre à la qualité visuelle des stimuli: les temps de réponse des patients étaient accrus lorsqu'un masque composé de lignes était superposé aux mots. De plus, cette variable interagissait avec la longueur de mot, les mots les plus longs étant davantage affectés par la diminution de la qualité visuelle. Il est possible que nos résultats indiquent une telle interaction.

Finalement, l'interaction entre la confusabilité de lettres et le voisinage orthographique est étudiée dans la quatrième expérience. L'hypothèse émise est que ces deux variables devraient interagir : l'effet facilitateur du voisinage orthographique devrait normalement être réduit ou disparaître lorsque la similarité visuelle des lettres composant le mot avec les autres lettres de l'alphabet devient trop importante. Cette prédiction est appuyée par le modèle unifié de Behrmann et collègues, qui propose en effet que les effets lexicaux de haut niveau ne peuvent apparaître lorsque l'activation initiale est trop faible ou imprécise.

VII. Simulation de la dyslexie lettre-par-lettre chez les lecteurs normaux

L'objet du second volet de ce travail est la simulation de la dyslexie lettre-par-lettre chez des lecteurs neurologiquement sains. Il s'avère en effet pertinent, pour mieux comprendre les déficits produisant ce trouble de lecture, de tenter de le simuler chez des lecteurs normaux sur la base des informations déjà recueillies sur l'origine fonctionnelle des déficits. La simulation de divers types de dyslexie a par le passé permis d'accroître nos connaissances sur ces troubles de lecture. Elle est fréquemment

utilisée dans le développement de modèles connexionnistes de la lecture (McCleod, Shallice et Plaut, 2000; Plaut et al., 1996), mais semble être beaucoup moins effectuée auprès de lecteurs neurologiquement sains, ce qui contribue à l'originalité de ce travail.

L'hypothèse d'un trouble périphérique provoquant la dyslexie lettre-par-lettre (présentée plus haut dans cette introduction) représente un excellent point de départ à la simulation de ce trouble de lecture, car elle est appuyée par plusieurs données dans la littérature. Si cette hypothèse est valable, il est permis de croire qu'un certain type de dégradation visuelle du stimulus pourrait provoquer chez les lecteurs normaux un ensemble de performances similaires à celles observées chez les dyslexiques lettre-par-lettre.

A. Dégradation visuelle par la réduction de contraste des stimuli

Nelson, Behrmann et Plaut (communication personnelle), ont testé cette hypothèse [et les prédictions du modèle unifié de la dyslexie lettre-par-lettre élaboré par Behrmann et al. (1998)] en présentant visuellement des mots à différents niveaux de contraste de luminance. Ils ont employé trois niveaux de contraste en variant l'intensité lumineuse du stimulus sur un écran d'ordinateur dont le fond gris avait une intensité constante. Le résultat le plus important de leur expérience a été la production d'un effet de longueur de mot relativement substantiel chez les lecteurs neurologiquement sains lorsque les stimuli étaient présentés dans ces conditions de contraste réduit (effet de longueur de mot de 16,7 msec, 31,4 msec et de 67,8 msec pour les conditions de contraste élevé, moyen et faible respectivement). Les auteurs ont également observé que l'effet de longueur de mot interagissait avec la fréquence lexicale et l'imageabilité des mots, comme Behrmann et al. l'avaient précédemment observé chez

des dyslexiques lettre-par-lettre. Ils en concluent donc que la manipulation visuelle des stimuli provoque une symptomatologie et des performances de lecture caractéristiques de celles retrouvées dans la dyslexie lettre-par-lettre. Leurs données appuieraient donc l'hypothèse d'un trouble périphérique sous-jacent dans ce type de dyslexie.

Leur expérience apparaît à prime abord un apport judicieux à l'hypothèse d'un trouble périphérique à l'origine fonctionnelle de la dyslexie lettre-par-lettre. Toutefois, un examen détaillé des stimuli employés dans leur expérience nous permet de constater que le voisinage orthographique n'a pas été contrôlé. Cette absence de contrôle fait en sorte que les mots les plus courts ont significativement plus de voisins orthographiques que les mots les plus longs, ce qui est caractéristique de la distribution naturelle des voisins orthographiques en fonction de la longueur des stimuli. Ainsi, il apparaît fort probable que l'effet de longueur de mot obtenu par les auteurs soit en fait un artéfact du voisinage orthographique. La démonstration que les résultats de cette étude s'expliquent par un artéfact du voisinage orthographique ébranlerait l'hypothèse que la dyslexie lettre-par-lettre puisse être reproduite par une simple réduction de contraste des stimuli.

La seconde étude rapportée dans ce travail a comme objectif de vérifier si les résultats obtenus par Nelson et collègues sont valides ou encore s'ils sont plutôt un artéfact de l'absence de contrôle du voisinage orthographique. Pour répondre à cet objectif, des stimuli de longueur variée seront présentés à des lecteurs neurologiquement sains en contraste réduit. Certains sujets seront exposés à une liste de stimuli où le voisinage orthographique aura été contrôlé, alors qu'une liste de mots dans laquelle cette variable n'aura fait l'objet d'aucun contrôle sera présentée aux autres sujets. Si l'effet de longueur de mot observé par Nelson et al. auprès de lecteurs normaux est attribuable non pas au nombre de lettres dans le mot

mais plutôt au nombre de voisins orthographiques qu'il possède, un effet de longueur de mot sera présent uniquement lorsque le voisinage orthographique n'aura pas été contrôlé. L'implication principale d'une telle observation serait que la simple réduction de contraste des stimuli est insuffisante pour simuler la dyslexie lettre-par-lettre.

B. Dégradation visuelle par le retrait des hautes fréquences spatiales

Même si un contrôle du voisinage orthographique devait éliminer l'effet de longueur de mot chez des lecteurs normaux en contraste réduit, l'hypothèse qu'une dégradation visuelle des stimuli peut produire la dyslexie lettre-par-lettre ne devrait toutefois pas être rejetée d'emblée. En effet, même si la réduction de contraste de luminance apparaît insuffisante pour simuler ce trouble de lecture, il demeure possible que d'autres conditions de dégradation visuelle, qui augmenteraient davantage la similarité visuelle des stimuli, puissent reproduire les caractéristiques de la dyslexie lettre-par-lettre chez les lecteurs normaux.

La première étude faisant l'objet de cette thèse, menée auprès du dyslexique lettre-par-lettre IH, offre un levier intéressant relativement à l'objectif de simuler la dyslexie lettre-par-lettre chez les sujets neurologiquement sains. En effet, les résultats obtenus démontraient que le sujet dyslexique lettre-par-lettre étudié était très sensible à la confusabilité visuelle des lettres par comparaison avec les lecteurs normaux (Arguin, Fiset et Bub, 2002). En effet, les temps de réponse de ce lecteur étaient significativement plus élevés pour les mots ayant une haute confusabilité de lettres que pour les mots composés de lettres de basse confusabilité. Ce phénomène a depuis été observé chez d'autres dyslexiques lettre-par-lettre (Arguin et Bub, soumis; Fiset, D., Arguin, Fiset, S. et Blais, 2003; Fiset, D., Arguin et McCabe, 2002) et semble donc

solide et fiable. Ces résultats contrastent avec les performances de sujets normaux, qui ne sont absolument pas affectés par la confusabilité de lettres dans des conditions normales de présentation. Par ailleurs, notre première étude révèle que la confusabilité de lettres interagit avec le voisinage orthographique dans la dyslexie lettre-par-lettre. En effet, l'effet facilitateur du voisinage orthographique sur la lecture explicite de mots, qui suggère la contribution d'un traitement parallèle à la reconnaissance de mots dans la dyslexie lettre-par-lettre, est aboli chez ces patients lorsque les mots sont composés de lettres de haute confusabilité. Plus précisément, un effet facilitateur du voisinage orthographique n'est présent que pour les mots ayant une basse confusabilité de lettres. Ces résultats ont également été répliqués chez d'autres dyslexiques lettre-par-lettre (Arguin et Bub, soumis). Notre expérience a également démontré que l'effet inhibiteur de la confusabilité de lettres semble lié au traitement parallèle des lettres.

Cette sensibilité anormale des dyslexiques lettre-par-lettre à la similarité visuelle des stimuli peut plausiblement être expliquée par un problème visuel accompagnant ce trouble de lecture et qui nuirait à la discrimination des lettres visuellement similaires. Deux hypothèses peuvent être élaborées pour expliquer cette sensibilité. En premier lieu, il est possible que l'hémianopsie homonyme droite relevée chez la plupart des dyslexiques lettre-par-lettre soit responsable de cette sensibilité à la confusabilité de lettres, en nuisant à l'encodage fovéal des stimuli visuels. Elle provoquerait ainsi une perte des hautes fréquences spatiales liée à la diminution de l'acuité spatiale observée normalement lorsqu'on s'éloigne de la fovéa (Carrasco et Frieder, 1997). Les hautes fréquences spatiales sont reconnues pour être essentielles dans la discrimination de détails fins et précis : leur perte peut donc induire une difficulté à discriminer entre des lettres visuellement similaires. Pour ces raisons, l'hémianopsie

homonyme droite pourrait contribuer à la symptomatologie des dyslexiques lettre-par-lettre.

La sensibilité de ces lecteurs à la confusabilité de lettres pourrait aussi s'expliquer par le fait qu'ils doivent obligatoirement encoder les mots par le biais de l'hémisphère cérébral droit, ce qui nuirait à l'extraction des fréquences spatiales nécessaires à l'identification de lettres. Cette hypothèse s'appuie sur les données suivantes : l'hémisphère cérébral droit semble plutôt spécialisé dans le traitement des basses fréquences spatiales (Ivry et Robertson, 1998) alors que la reconnaissance de lettres solliciterait l'utilisation de fréquences spatiales moyennes à hautes (Solomon et Pelli, 1994; Majaj, Pelli, Kurshan et Palomares, 2002). Des études pilotes effectuées dans notre laboratoire auprès de sujets neurologiquement sains, dans lesquelles les stimuli étaient présentés à la parafovéa et en contraste réduit, ont offert un appui à l'hypothèse qu'une perte de fréquences spatiales serait un facteur important dans la production de la symptomatologie des dyslexiques lettre-par-lettre, puisqu'un effet de confusabilité important a été observé chez ces lecteurs dans ces conditions de présentation.

La dernière étude de ce présent travail examine donc l'hypothèse que des conditions de présentation capable de produire un effet de confusabilité chez des lecteurs normaux, entraînerait également l'apparition de la symptomatologie associée à la dyslexie lettre-par-lettre. Pour parvenir à cet objectif, des sujets neurologiquement sains ont été exposés à des stimuli visuellement dégradés, i.e. présentés avec un faible contraste de luminance et dont les hautes fréquences spatiales ont été retirées (cette dernière manipulation reproduisant les conditions d'encodage parafovéal des stimuli). Il est attendu que ce type de dégradation visuelle des stimuli devrait provoquer, chez les lecteurs normaux, une symptomatologie similaire à celle observée chez les

dyslexiques lettre-par-lettre. Devraient donc apparaître un effet de confusabilité de lettres, un effet de longueur de mot interagissant probablement avec la fréquence lexicale [tel qu'observé par Behrmann et al. (1998)], ainsi qu'une interaction entre la confusabilité de lettres et le voisinage orthographique (Arguin et al., 2002; Arguin et Bub, soumis).

CHAPITRE DEUXIÈME:

PREMIER ARTICLE

SEQUENTIAL AND PARALLEL LETTER PROCESSING IN LETTER-BY-LETTER DYSLEXIA

Martin Arguin[1], Stéphanie Fiset[1], and Daniel Bub[2]

1- Groupe de Recherche en Neuropsychologie Expérimentale,
Département de psychologie, Université de Montréal,
and
Centre de recherche, Institut Universitaire de Gériatrie de
Montréal, Canada.

2- Department of psychology, University of Victoria, Victoria, Canada.

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ABSTRACT

Four experiments are reported that focus on the issue of sequential vs. parallel letter processing in letter-by-letter (LBL) dyslexia which were conducted on patient IH. Exp. 1 showed a large linear reduction of word naming times with an increase in the number of orthographic neighbours of the target (i.e. words of the same length differing by just one letter; N size). Given the large negative linear correlation existing between word length and N size, this result raises the possibility that the large word length effect diagnostic of LBL dyslexia may be, in fact, an artefact of uncontrolled N size. Exp. 2 falsified this possibility by showing that the word length effect is unaffected by whether N size is controlled for or not. This result also suggested that the facilitatory effect of increased N size in LBL dyslexia is based on the parallel processing of the constituent letters of the target. Further supporting a contribution of parallel letter processing to overt word recognition performance in the disorder, Exp. 3 showed significant but independent effects of word length and letter confusability (i.e. similarity of the constituent letters of the target word with other letters of the alphabet). The letter confusability effect therefore appears to rest on the parallel analysis of the letters in the target word. Finally, Exp. 4 showed that the facilitatory effect of N size is prevented with high letter-confusability targets. These observations suggest that LBL dyslexia rests on an impairment of letter encoding that results in an excessive level of background noise in the activation of lexical-orthographic representations when letters are processed in parallel. This prevents overt identification of the target and forces sequential letter processing in order to achieve this goal.

Key words: Dyslexia, Reading , Visual processing

INTRODUCTION

Letter-by-letter (LBL) dyslexia is an acquired reading disorder caused by left occipital lobe lesions (Damasio & Damasio, 1983; Dejerine, 1892) in previously literate adults. Its diagnostic behavioural features are laborious reading accompanied by a large linear increase in the time required for the overt recognition of a word as a function of the number of letters it comprises (e.g. Patterson & Kay, 1982; Warrington & Shallice, 1980). This latter feature has traditionally been taken as an unambiguous demonstration that overt word recognition in LBL dyslexia proceeds by the slow, sequential identification of individual letters in the stimulus. This feature clearly distinguishes LBL dyslexics from neurologically intact adult readers, who appear to recognize words through the spatially parallel (i.e. simultaneous) processing of all letters in the stimulus. Indeed, in normal readers, the so-called word length effect characterizing LBL dyslexia is either absent or extremely weak (Frederiksen & Kroll, 1976; Forster & Chambers, 1973; Weekes, 1997). Based on this feature, LBL dyslexia should be attributed to some impairment that prevents parallel letter processing either to occur, or at least to access internal lexical representations in a way that would allow the recognition of the stimuli.

Mounting evidence has been gathered in recent years for a residual parallel letter processing capacity in LBL dyslexia. Indeed, several reports have provided indications for a phenomenon labelled implicit reading, whereby the performance of LBL dyslexics suggests an accurate lexical access for words they cannot identify overtly, which is based on the parallel processing of the letters constituting the stimulus (Arguin, Bowers & Bub, 1996; Arguin Bub & Bowers, 1998; Bowers, Bub & Arguin, 1996a; Bowers, Arguin & Bub, 1996b; Bub & Arguin, 1995; Bub, Black & Howell, 1989; Coslett & Saffran, 1989; Coslett, Saffran, Greenbaum & Schwartz, 1993; Reuter-Lorenz & Brunn, 1990; Shallice & Saffran, 1986). Perhaps the

most striking demonstration of implicit reading in LBL dyslexia has been observed in tasks of lexical or semantic decisions, where patients perform well above chance without being able to explicitly identify the stimulus. In these conditions, when assessed, the word length effect is absent, thereby indicating that implicit reading is achieved through the parallel processing of the letters of the target (Bub & Arguin, 1995; Coslett & Saffran, 1989). Related evidence has been provided by Bowers et al. (1996b) and by Arguin et al. (1998) using a priming paradigm where an uppercase target word to be named was preceded by a briefly exposed (i.e. 100 ms) lowercase prime that was then backward masked. Even though the exposure duration of the prime was insufficient for the subject to overtly identify it, substantial reductions of correct response times (RTs) were observed for target words preceded by same-word primes relative to unrelated primes. Moreover, this effect of same-word primes was entirely specific to the prime displayed and it was based on the processing of all of its constituent letters. Thus, no priming effect was observed for targets that differed from the prime by just one letter, no matter what the position of that letter was in the string (Bowers et al., 1996b).

The evidence for implicit reading in LBL dyslexia (i.e. residual lexical access based on parallel letter processing) has been obtained in conditions that did not require overt identification of the stimulus. Thus, the fast semantic or lexical decisions described above or the priming effects observed with brief prime exposure durations were obtained while the patient failed to provide signs of reliable overt recognition of the stimulus. In fact, Coslett and Saffran (1994; Coslett et al., 1993) have argued for a fundamental opposition between the processing strategies involved for overt word recognition and implicit reading, suggesting that the latter would only be possible when the patient is not attempting to identify the stimulus explicitly. These considerations raise questions as to

whether parallel letter processing in LBL dyslexia has any impact on the overt word recognition performance of LBL dyslexics.

The first indication that parallel letter processing may indeed contribute to overt word recognition performance in LBL dyslexia has been provided by Howard (1991; but see Behrmann & Shallice, 1995; for discrepant findings). In a visual word naming task, he showed that the subset of correct reading responses that are the fastest in the response time distribution of LBL dyslexics result from the parallel processing of the constituent letters in the target. He also showed that this parallel process was subject to a significant rate of error. Howard (1991) further proposed that it was when this process failed that patients had to resort to serial letter processing for the overt recognition of a word. This study thus suggests that the lexical activation process assumed to be responsible for implicit reading may in fact allow the patient to recognize a word without serial letter processing on some proportion of trials.

More recent evidence for the notion that parallel letter processing provides a measurable contribution to overt word recognition in LBL dyslexics was reported by Arguin et al. (1998). In their study of LBL dyslexic patient IH, they examined the effect of the number of orthographic neighbours (i.e. N size) of the target word on overt naming latency and accuracy. Orthographic neighbours of a target letter string are words of the same length that differ from it by just one letter (Coltheart, Davelaar, Jonasson & Besner, 1977).

In neurologically intact readers, an increase in the N size of a target word leads to reduced naming latencies and occasionally reduced error rates (Andrews, 1989; 1992; Arguin et al., 1998; Carreiras, Perea & Grainger, 1997; Sears, Hino & Lupker, 1995). This facilitatory effect of increased N size is considered to index a process involved in orthographic

encoding (Andrews, 1989; 1992; Arguin et al., 1998; Carreiras et al., 1997; Coltheart et al., 1977; Pugh et al., 1994; Sears et al., 1995). Specifically, it appears that a visually presented word activates not only its own internal orthographic-lexical representation, but also, albeit to a lesser degree, that of its orthographic neighbours. In reference to the interactive activation model of visual word recognition of McClelland and Rumelhart (1981), this lexical activation is then assumed to provide facilitatory feedback to compatible representations at a preceding processing level responsible for the identification of the constituent letters of the stimulus. With more orthographic neighbours therefore, more intense facilitatory feedback is sent from the activated lexical representations to letter representations compatible with the target. It is this greater degree of facilitatory feedback on the letter identification process with increased N size that is considered responsible for the facilitatory effect of this factor on reading performance. Crucially, the parallel processing of the constituent letters of the target is required for this facilitatory effect of increased N size. Indeed, if parallel letter processing is prevented, the N size effect reverses and becomes inhibitory. Thus, in normal readers, orthographic neighbours inhibit rather than facilitate performance if a letter distinguishing the target from its neighbours is delayed by 100 ms relative to the other letters (Pugh, Rexer, Peter & Katz, 1994). Similarly, Arguin & Bub (1997) have reported a patient with a visuo-spatial attention deficit who showed a severe inhibitory effect of the number of orthographic neighbours that differed from the target by the letter positions she had difficulty attending.

Arguin et al. (1998) have shown that N size has a facilitatory effect on the word naming performance of their LBL dyslexic patient (IH), thereby supporting the hypothesis of a contribution of parallel letter processing to overt word recognition. This parallel letter processing did not appear normal however, since the facilitatory N size effect in IH did not vary as a function of the lexical frequency of the target, in contrast to

what is found in neurologically intact readers. The latter show substantial facilitation from increased N size with low frequency target words but a markedly reduced or absent N size effect with high frequency words (Andrews, 1989; 1992; Arguin et al., 1998; Sears et al., 1995). IH's results therefore suggest that, although parallel letter processing may contribute to overt word recognition in LBL dyslexia, this processing is anomalous in some unknown manner, thus leading to an activation of internal lexical representations that is too weak or imprecise to support overt word recognition. Arguin et al. (1998) proposed that it would be because of this anomaly that LBL dyslexics must resort to sequential letter analysis to explicitly recognize visually presented words. In this respect, a specification of the impairment affecting parallel letter processing in LBL dyslexia appears fundamental for a proper understanding of the functional causes of the disorder and for any prospect of developing adequate rehabilitation methods (e.g. Arguin & Bub, 1994; Behrmann & McLeod, 1995). The present paper is a contribution toward this goal.

The experiments reported in this article were conducted on LBL dyslexic patient IH. They investigate the relation of the N size effect in LBL dyslexia to the issue of serial vs. parallel letter processing and use the interactive effects of N size with other stimulus parameters as an index of the impairment that prevents parallel letter encoding from reliably supporting overt word recognition. Based on the fact that the number of letters in English words is highly correlated with N size, Exps. 1 and 2 investigate whether the word length effect in the naming task, which is diagnostic sign of the disorder, does indeed reflect serial letter processing or whether this observation is an artefact of the uncontrolled effects of N size. Exps. 3 and 4 investigate the role of visual similarity among letters in the modulation of the reading performance in LBL dyslexia and its interactive effects with N size and word length.

CASE REPORT

The subject who took part in all the experiments described in this article, IH, has also been described in other papers (Arguin et al., 1998; Bowers et al., 1996a; Bowers et al., 1996b). We will thus only briefly summarize his clinical and neurological features. IH is a right-handed English speaking male with 15 years of formal education who suffered from a subarachnoid haemorrhage in 1983. He was 59 years old at the time of testing. No CT or MRI scan are available but the neurological report indicates that the haematoma was located in the left temporo-occipital area. Performance on the WAIS intelligence scale indicated an IQ in the low normal range (global score of 90), with no asymmetry between the verbal (score of 89) and performance scales (score of 92). IH's main behavioural symptoms were a complete right-homonymous hemianopia, anomia, surface agraphia, and reading problems. His reading latencies averaged 1200-1250 ms for four-letter words and increased linearly by about 500 ms for each additional letter in the word. This linear effect of word length on naming latency classifies IH as a letter-by-letter dyslexic. IH also presents symptoms of surface dyslexia; his reading performance is affected by the regularity of spelling-to-sound correspondences as well as by lexical frequency (see Arguin et al., 1998 for additional details). The patient thus suffers from a combination of LBL dyslexia and surface dyslexia, a disorder identified by Patterson and Kay (1982) as Type II LBL reading, and by Friedman and Hadley (1992) as letter-by-letter surface dyslexia.

A number of subtests of the Psycholinguisitc Assessment Language Processing in Aphasia (PALPA; Kay, Lesser & Coltheart, 1992) were administered to IH. These showed normal auditory phonological processing, with 67/72 correct on same-different matching with auditory minimal nonword pairs, and 71/72 correct with minimal word pairs.

Auditory lexical processing also appeared intact, with IH accepting all the auditory words presented as being lexical items. Semantic processing was relatively spared, with a performance of 38/40 correct in matching spoken words to visual pictures. IH's poor performance (6/60 correct) on the Boston naming task (Kaplan, Goodglass & Weintraub, 1983) however, revealed a severe anomia. This deficit was also markedly apparent in the patient's sponteneous speech, which comprised very many hesitations and circumlocutions, and a frequent incapacity in finding any term appropriate to formulate even relatively common statements. The studies reported in this paper have been approved by the Ethics committee of the Institut Universitaire de Gériatrie de Montréal, where this work has been conducted.

EXPERIMENT 1

A strong linear inverse relationship exists between N size and word length in English. Specifically, the longer the word, the smaller the number of its orthographic neighbours. For words ranging from three to six letters, this negative correlation is $r = -0.98$; with an average of 9.25, 6.01, 1.68, and 0.61 orthographic neighbours for three-, four-, five-, and six-letter words, respectively (see also Weekes, 1997). As indicated above, increased N size has a very large facilitatory effect on word naming performance in LBL dyslexia. For instance, Arguin et al. (1998) reported a reduction of correct RTs of 222 ms as N size increased from 0-3 to 11+ neighbours. This effect is about one order of magnitude greater than that observed in normal controls (e.g. Andrews, 1989; 1992; Arguin et al., 1998; Sears et al., 1995). In addition to Arguin et al. (1998), a large facilitatory N size effect has also been obtained by Arguin and Bub (1996) and by Montant & Behrmann (2001) in other LBL dyslexics. Given the magnitude of the N size effect and the fact that the value of this parameter decreases dramatically as word

length increases, one legitimate question that may be raised is whether the word length effect in LBL dyslexia actually reflects the sequential processing of the individual letters comprising the word or whether it is an artefact of N size.

On its own, a linear increase of correct RTs as a function of word length, no matter how large, remains ambiguous as to whether letter processing is serial or parallel (see Snodgrass & Townsend, 1980; Townsend, 1990; for relevant discussions). For instance, one could imagine that letter processing still occurs in parallel in LBL dyslexia, but that this parallel processing capacity has become limited in a way that makes it overly sensitive to the effect of orthographic neighbourhood size. If this were so, RTs would increase linearly with increased word length, not because of serial letter processing, but instead because longer words have fewer orthographic neighbours. At present this alternative account of the word length effect cannot be excluded given that, to our knowledge, tests examining this effect in LBL dyslexia have never controlled for the likely mismatches in N size across words of different lengths.

The tests performed in Exps. 1 and 2 examine whether the word length effect characteristic of LBL dyslexia is an artefact of N size or whether it truly reflects the sequential processing of individual letters in the word. The results of these experiments will also be relevant to the issue of whether the occurrence of a facilitatory effect of increased N size in LBL dyslexia implies that parallel letter processing provides a significant contribution to overt word recognition. If the word length effect in LBL dyslexia is indeed an artefact of the negative linear correlation of this factor with N size, one prediction that may be made is that correct RTs in the word naming task will decrease linearly with increasing N size for words that are of a constant length. Although the observation of this result would not falsify the standard interpretation of

the large word length effect characterizing LBL dyslexia, it would certainly question its validity.

In Exp. 1, LBL dyslexic patient IH was asked to read aloud individually presented words of a constant length that varied according to four distinct levels of N size. Items from different levels of the N size factor were precisely matched on other fundamental stimulus parameters that would be likely to affect performance if left uncontrolled.

Methods

Stimuli. Stimuli were 160 four-letter English words divided equally into four conditions defined according to orthographic neighbourhood size: 0-1; 4-5; 8-9; or 12-13 neighbours. Across conditions, words were matched on lexical frequency (Kucera & Francis, 1967) [$F(3,156) = 1.44$; *ns*], and single-letter [$F(3,156) = 2.51$; *ns*] and bigram frequencies [$F(3,156) = 1.32$; *ns*] (Mayzner & Tresselt, 1965).

Procedure. Each trial began with a 1500 ms fixation point, displayed at the centre of a computer screen. This was followed by the target printed in lower-case, presented to the left of fixation (because of the patient's right hemianopia) and which remained visible until response. The task was to name the target as rapidly as possible while avoiding errors.

All stimuli appeared in black over a white background and were printed in Geneva 24-point bold font. The Psychlab software (Bub, D. & Gum, T., 1998. Psychlab Software, University of Victoria, Canada) was used to control the presentation of those stimuli. Responses were registered by a voice-key (engineered in our laboratory) connected to the computer controlling the experiment (a MacIntosh LC-575 connected to a 13 inches screen). After each response, the

experimenter wrote the subject's response via the computer keyboard and then triggered the next trial by a key press.

Throughout the experiment, a total of four trials (2.5 %) were lost due to the failure of the subject's response to trigger the microphone. These trials were not considered in the data analyses.

Results

Average correct response times (RTs) and error rates are shown in Figs. 1 and 2, respectively. The correlation between RTs and error rates was of +0.97 ($p < .05$), which indicates no speed-accuracy trade-off. Two data points (1.53% of correct trials) were removed from the RT analysis because response latencies were more than three standard deviations away from the mean of their condition.

Insert Figs. 1 and 2 near here.

An ANOVA performed on correct RTs showed a main effect of N size [$F(3,125) = 4.56$; $p < 0.005$], with RTs decreasing linearly as orthographic neighbourhood size increased. A linear regression analysis of RTs as a function of N size indicated that each additional orthographic neighbour reduced response times by 37 ms and that this effect was linear ($r^2 = 0.98$).

A Chi-square analysis applied on error rates showed that the effect of orthographic neighbourhood size on error rates was significant [$c^2(3) = 9.21$; $p < .05$]. Orthogonal comparisons revealed that IH was more accurate with words having 8-9

neighbours [$c^2(1) = 5.37$, $p < .025$] and 12-13 neighbours [$c^2(1) = 5.37$, $p < .025$] when compared to words having fewer neighbours. A linear regression analysis of error rates as a function of N size indicated that, for each additional orthographic neighbour, error rates declined linearly by 2.03% ($r^2 = 0.90$).

Discussion

The results of Exp. 1 show a substantial linear decrease of correct RTs and of error rates as the N size of the target words is gradually increased. The facilitatory effect of increased N size replicates previous observations from IH that were reported by Arguin et al. (1998). For every additional orthographic neighbour the target word has, RTs decrease by an amount of 37 ms and error rates decrease by about 2%. The negative relation demonstrated by IH between his correct RTs and N size is congruent with the hypothesis presented above that the word length effect in LBL dyslexia might not actually reflect sequential letter processing, but rather that it is an artefact of the correlation existing between word length and N size.

It may be noted however that, given the present results, the word length effect predicted for IH by the N size artefact hypothesis is 112 ms/letter[1] for word lengths ranging between three and six letters. This is well below the relatively stable word length effect of about 500 ms/letter shown by IH. Even if this observation does not falsify the hypothesis that the word length effect in LBL dyslexia is entirely an artefact of uncontrolled N size, it certainly weakens its credibility. Nevertheless, the present results do suggest that N size may contribute significantly to the magnitude of the word length effect in the disorder. This will be examined in Exp. 2.

EXPERIMENT 2

If the word length effect in LBL dyslexia is an artefact of N size, either partially or completely, then it should be reduced or abolished if words of different lengths are accurately matched in terms of their numbers of orthographic neighbours. In Exp. 2, IH was required to overtly identify individually presented words comprised of a variable number of letters. In one condition, words of different lengths were matched on several important stimulus parameters, but not on N size. Consequently, in this uncontrolled list, items of increasing length had markedly decreasing numbers of orthographic neighbours. In another condition, words of different lengths were accurately matched again on several important variables, this time including N size. The key test performed in Exp. 2 was to determine whether the word length effect on the correct RTs of IH varied as a function of whether N size was controlled or not[2].

Method

Stimuli. Two sets of 120 stimuli were used, including an equal number (n = 40 per level) of four-, five-, and six-letter words in each. In one set, words of different lengths were matched according to their number of orthographic neighbours. Thus, words of different lengths all had an average N size of 1.5 [$F(2, 117) < 1$]. In the other set of items, no control of neighbourhood density was performed. Consequently, there was a significant variation of N size across words of different lengths [$F(2,117) = 46.38$, $p < .001$], with average N sizes of 4.85, 2.18, and 0.48 for four-, five-, and six-letter words respectively. Otherwise, in both stimulus sets, words of different lengths were matched according to lexical frequency, single-letter frequency, bigram frequency, and letter confusability (all tests for these matches produced [$F(2,117) < 1$]; letter

confusability is an index of the shape similarity between a particular target letter and the remaining letters of the alphabet, see Exp. 3 for a more detailed description).

Procedure. The procedure was exactly the same as in Exp. 1, except that the stimuli were printed in capital letters[3]. Throughout the experiment, a total of three trials (1.25 %) were lost due to the failure of the subject's response to trigger the microphone. These trials were not considered in the data analyses.

Results

Average correct RTs and error rates are shown in Figs. 3 and 4, respectively. The correlation between RTs and error rates was of +0.65 (*ns*) which indicates no speed-accuracy trade-off. One data point (0.56 % of trials) was removed from the RT analysis because the response latency was more than three standard deviations away from the mean of its condition.

Insert Figs. 3 and 4 near here.

A two-way ANOVA performed on correct RTs, with neighbourhood size control (N size control vs. no N size control) and word length as factors showed main effects of length [$F(2,185) = 46.88$; $p < .001$] and of neighbourhood size control [$F(1,185) = 3.98$; $p < .05$]. These indicate that correct RTs increased as word length increased and that RTs were longer with than without N size control. The interaction between word length and N size control was not significant [$F(2,185) = 1.50$; *ns*].

Chi-square analyses of error rates as a function of word length showed a significant effect of length when N size was controlled for [$c^2(2) = 10.14$; $p < 0.01$] but no effect when it was not controlled [$c^2(2) = 0.13$; *ns.*].

Discussion

The results of Exp. 2 indicate that the large and linear word length effect that has traditionally been taken as the diagnostic sign for LBL dyslexia is not an artefact of the correlation of this factor with N size. Thus, whether or not N size was controlled for in the examination of the word length effect on IH's RTs, the magnitude of this effect remained unchanged. One significant difference as a function of whether N size was controlled for or not is a uniform increase of correct RTs, independent of word length, in the former condition compared with the latter. This result simply reflects the fact that average N size was smaller when words of different lengths were matched for N size than when they were not. The observation therefore, is congruent with the large facilitatory effect of increased N size in LBL dyslexia previously found in Exp. 1 as well as by Arguin et al. (1998), Arguin & Bub (1996), and Montant & Behrmann (2001). Another difference in the results as a function of whether or not N size was controlled pertains to error rates, which increased significantly with word length in the controlled list but not in the uncontrolled list. Although no obvious interpretation may be proposed for this latter observation, it should be underlined that it is the opposite of what should be predicted if the word length effect in IH was an artefact of N size.

Apart from being relevant for an interpretation of the word length effect in LBL dyslexia, the results of Exp. 2 also have implications for our understanding of the N size effect in the disorder. In the first examination of this factor in an LBL dyslexic, Arguin et al. (1998) argued that a

facilitatory effect of increased N size must reflect a residual capacity for the parallel processing of the constituent letters of the stimulus. The main arguments involved in this reasoning, which are listed in the Introduction of the present paper, are indirect and concern other classes of readers than LBL dyslexics. The present results provide a direct indication that the effect of orthographic neighbourhood size may be independent from the sequential letter processing that mediates the word length effect. This therefore supports the position that the facilitatory N size effect in LBL dyslexia reflects a contribution of parallel letter processing to overt word recognition performance in the disorder. The possible reason why this residual parallel letter processing would fail to consistently support overt word recognition in the disorder (i.e. why serial letter processing is required) is the issue addressed in the following experiments.

EXPERIMENT 3

A wide variety of markedly different hypotheses have been proposed in the literature on LBL dyslexia to account for the disorder (see e.g. Arguin et al., 1998; Behrmann & Shallice, 1995; Bowers et al., 1996a, for reviews). Current evidence points to a low-level deficit prior to lexical access as the most likely possibility (Arguin et al., 1998; Arguin & Bub, 1993; Behrmann & Shallice, 1995). It remains unclear however, exactly what this deficit might be. Some authors have argued that the letter encoding deficit in LBL dyslexia rests on a general impairment of the visuo-perceptual function that would affect the encoding of the shapes of letters and of other non-linguistic visual materials as well (Behrmann, Nelson & Sekuler, 1998a; Behrmann, Plaut & Nelson, 1998b; Farah & Wallace, 1991; Friedman & Alexander, 1984; Kinsbourne & Warrington, 1962; Levine & Calvanio, 1978; Rapp & Caramazza, 1991). However,

except for the single-case study of Farah and Wallace (1991), the more or less subtle visual impairments demonstrated have never been shown directly to actually impact on reading performance in such a way that it could account for the disorder. Furthermore, the generality of the hypothesis is not entirely clear since LBL dyslexia may exist without concomitant evidence for a general visuo-perceptual impairment, despite reasonable efforts on the part of experimenters to reveal such a problem (e.g. Arguin & Bub, 1993). Even then however, evidence may be found that visual similarity among letters plays a key role in determining reading performance (Arguin & Bub, 1993). This kind of observation points to a possible deficit in either the encoding of letter shapes or in the function mapping letter shape information to subsequent processing stages.

In the context of previous unpublished tests of the impact of visual similarity among letters, our laboratory has developed a new stimulus parameter labelled "letter confusability". Letter confusability is defined as the shape similarity between a particular target letter and the remaining letters of the alphabet, with confusability values determined from empirical letter confusion matrices obtained in previous studies conducted in neurologically intact observers (Gilmore, Hersh, Caramazza & Griffin, 1979; Loomis, 1982; Townsend, 1971; Van Der Heijden, Malhas & Van Den Roovaart, 1984). Specifically, the confusability value for a particular letter consists in the error probability in the identification performance of normal readers for that letter when it is displayed very briefly and then masked. For target words, letter confusability is the average of the confusability of their constituent letters. In pilot testing, increased letter confusability had no impact on the reading performance of neurologically intact observers (see also Exp. 4). Their correct word naming latency was of 485 ms for low letter-confusability target words (i.e. with confusability values of 0.42 or lower) and of 490 ms for high letter-confusability words (i.e. with confusability values of 0.53 or higher) [$F(1,9) = 1.03$; ns]. Error

rates in the corresponding conditions were of 0.8% and of 1.1% [$F(1,9) < 1$]. By contrast, the average overt word recognition latencies of four LBL readers for four-letter words were increased by 225 ms (increase of 3.5% in error rates) by the same increase in letter confusability.

Exp. 3 examined the effect of letter confusability in patient IH in relation to the sequential letter encoding process that characterizes the disorder. Specifically, the word length effect was tested in the naming task with items of either low or high letter confusability. Otherwise, items from different conditions were matched on several other stimulus parameters that may impact on performance. This test allows an examination of the effect of letter confusability (i.e. visual similarity among letters) on the processing of each individual letter in the sequential analysis required by LBL dyslexics for overt word recognition. If such a relation exists, the word length effect should vary as a function of the confusability of the letters constituting the words. In particular, the magnitude of the word length effect should increase with high compared to low letter-confusability target words.

An alternative result is possible however, in light of our previous observations on the N size effect, which suggest a residual capacity for parallel letter processing in LBL dyslexics that contributes to their overt word recognition performance. This alternative is that letter confusability significantly affects overt word recognition performance in the disorder, but that this effect remains invariant across words of different lengths. The occurrence of such a result would further support the contribution of parallel letter processing in LBL dyslexia. It would also point to a possible cause as to why this parallel letter analysis cannot support overt word recognition on its own in the disorder and hence, why sequential letter encoding is required for this performance.

Method

Stimuli. Targets were 300 words divided equally into six conditions according to their length (4, 5 or 6 letters) and their letter confusability (low: confusability of 0.41 or below; high: confusability of 0.54 or higher). There were thus 50 words per condition. Across levels of the letter confusability factor, words of the same length were matched pairwise on lexical frequency, N size, single-letter and bigram frequencies [all F's (1,98) < 1].

Procedure. The procedure was the same as in Exp. 2, except that the duration of the fixation point was 750 ms instead of 1500 ms. There was also a 250 ms blank interval between the fixation point and the onset of the target.

Throughout the experiment, a total of six trials (2%) were lost due to the failure of the subject's response to trigger the microphone. These trials were not considered in the data analyses.

Results

Average correct RTs and error rates in IH are shown in Figs. 5 and 6, respectively. The correlation between RTs and error rates was +0.84 ($p <$ 0.05) which indicates no speed-accuracy trade-off. Correct RTs that were more than three standard-deviations away from the mean for their condition were discarded. Only one data point (0.45 % of correct trials) was removed from the analysis on this criterion.

Insert Figs. 5 and 6 near here.

A two-way ANOVA conducted on correct RTs with word length and letter confusability as factors showed main effects of word length [$F(2,216) = 103.92$; $p < 0.01$] and of letter confusability [$F(1,216) = 3.83$; $p = 0.05$]. The interaction between these factors was not significant [$F(2,216) < 1$]. The main effects indicate increasing RTs with longer words and shorter RTs for low than high letter-confusability words.

Chi-square analyses of error rates as a function of word length indicated a significant effect of length for low confusability words [$c^2(2) = 6.29$; $p < 0.05$] while this effect failed to reach significance with high letter confusability words [$c^2(2) = 4.72$; *ns*].

Discussion

The results of Exp. 3 show that whereas letter confusability had an impact on IH's overt word recognition performance, it failed to significantly modulate the magnitude of his word length effect. The main implication of this observation is that, while the visual similarity among letters does affect overt word recognition performance in IH, it appears to have no impact on the time required to identify individual letters in the sequential letter processing which is the hallmark of LBL dyslexia. In turn, this suggests that letter confusability mainly affects a form of processing independent of sequential letter analysis, whereby the constituent letters of the target word would be processed in parallel. In this respect, Exp. 3 provides converging evidence for one main conclusion of Exp. 2, namely that parallel letter processing appears to contribute to overt word recognition performance in LBL dyslexia, even though it seems incapable of supporting this performance on its own; i.e. without eventually resorting to sequential letter analysis.

An additional implication of the results of Exp. 3 is that they identify visual similarity among letters as a probable determinant of LBL dyslexia. Thus, the apparently abnormal susceptibility to letter confusability observed in IH (a formal test of the letter confusability effect in normal readers is provided in Exp. 4) points to an impairment affecting the discrimination between visually similar letters. In particular, it suggests that parallel letter processing in the patient cannot resolve completely and in a reliable manner the difference between visually similar letters. This problem is exacerbated with high letter-confusability target words. It is proposed that this difficulty may explain why parallel processing fails to support overt word identification in the disorder, as well as the obligation of LBL dyslexics to revert to a compensatory strategy of sequential letter identification for that purpose.

More specifically, instead of being able to positively identify each letter in the word examined, parallel letter analysis in LBL dyslexia may leave a significant degree of uncertainty about the identity of these letters. Transmitted to the level of lexical-orthographic representations, this uncertainty about letter identities should cause several candidate words to be significantly activated because of their compatibility with the degraded letter input. In terms of the decision mechanism involved in the absolute identification of the target word (i.e. "*x*, and not any other word, is the target"), this widespread activity among lexical representations translates into a high level of background noise against which the activation of the best (i.e. most highly activated) lexical candidate is assessed (e.g. Arguin & Bub, 1995; Luce, 1959; 1977). This background noise at the level of lexical orthographic representations may be excessive and thus prevent the reliable identification of the target, except perhaps in a minority of trials leading to particularly fast overt identification responses, as shown by Howard (1991). Apart from these exceptional trials however, LBL dyslexics would be forced to revert to sequential letter analysis for overt

word recognition. One possible function of sequential processing would be to allow LBL dyslexics to focus their processing resources on single letters rather than spread them across the entire word, and thus, more effectively resolve the difference between the letters that are shown and visually similar alternatives.

Exp. 4 will provide a test for the hypothesis that visual similarity among letters is a fundamental determinant of the incapacity of parallel processing of reliably supporting overt word recognition in LBL dyslexia. It will also provide a formal test of the letter confusability effect in neurologically intact readers, which is required to establish that the effect of this factor documented in LBL dyslexia is indeed abnormal.

EXPERIMENT 4

Since LBL dyslexics may show a facilitatory N size effect on their overt word recognition performance, it must be concluded that they are capable of performing a degree of discrimination between the target word and its orthographic neighbours. Indeed, were this otherwise, the lexical-orthographic activation produced by the target would be lost in the background noise produced by the equally high activation of its orthographic neighbours. In such circumstances, there would be no reason to suppose that a larger N size should have a facilitatory effect on performance. In fact, we might assume just the opposite since a greater degree of background noise should occur with high than low N size targets because of the larger number of activated lexical-orthographic representations (see Introduction for a relevant discussion of this issue).

Until now, the investigation of the N size effect in LBL dyslexia has only been conducted in the context of experiments that ignored any possible role of the visual similarity among the constituent letters of the target and other letters of the alphabet. The negative impact of increased letter confusability documented in Exp. 3 and the interpretation proposed above for this effect suggest that words with different levels of letter confusability may differ in their potential of leading to a facilitatory effect of N size. In particular, since high letter confusability is assumed to magnify the degree of background noise in the activation of lexical-orthographic representations resulting from parallel letter processing, it should also negatively affect the capacity of an LBL dyslexic to discriminate between the target and its orthographic neighbours. Since this latter capacity is obviously essential for a facilitatory N size effect, it may be predicted that high letter confusability will either reduce or prevent a facilitatory effect of increased N size. With low letter-confusability target words however, it should be possible to replicate the facilitatory N size effect previously found in IH (Arguin et al., 1998) and in other LBL dyslexics (Arguin & Bub, 1996; Montant & Behrmann, 2001).

Exp. 4 will assess this prediction in IH by using a word naming task where the N size and letter confusability of the target words are manipulated factorially. Distinct groups of young and age-matched neurologically intact readers will also be examined in Exp. 4 in order to determine whether their performance is affected by letter confusability and whether this factor interacts with N size. Words belonging to different conditions will be matched on other important properties to control their potential impact on the results.

Method

Subjects. Subjects were IH, a group of 12 age-matched neurologically intact controls with a mean age of 55 years (range: 51-66) and a mean schooling duration of 16 years (range: 13-20), and another group of 10 neurologically intact university students aged between 20 and 26 years. All were right-handers and had normal or corrected vision.

Stimuli. Targets were 200 four-letter words varying orthogonally on their numbers of orthographic neighbours (N size; low: 0-4 neighbours; high: 9 or more neighbours) and on their letter confusability (low: confusability below 0.45; high: confusability of 0.53 or higher). There were 50 items in each condition. Across conditions, words were matched on lexical frequency and on single-letter and bigram frequencies [all F's $(1,196) < 1$].

Procedure. For IH, the procedure was identical to that of Exp. 2. From his complete set of data, a total of seven trials (3.5%) were lost due to the failure of the subject's response to trigger the microphone. These trials were not considered in the data analyses.

The age-matched control subjects were tested on the same list and using the same procedure as for IH. Throughout their data set, a total of 27 trials (1.1%) were lost because the subject's oral response failed to trigger the microphone.

For the young normal controls, the observations for the relevant words were extracted from a large database comprising their reading performance on a total of 1285 four-letter English words. These databases were obtained for each subject in a sequence of 10 blocks of 120-130 trials within which items were distributed in a random order, with blocks also

administered in a random order across subjects. Each trial began with fixation point of a 500 ms duration, followed by a 250 ms blank interval, and then by the uppercase target word centred on the location of ocular fixation. Subjects were required to name the target as rapidly as possible while avoiding errors. Across all trials on the relevant items, no trial was lost due to failure of the subject's response to trigger the voice-key.

Results

Figs. 7 and 8 show the correct RTs of the age-matched normal readers in each condition. Correct RTs that were more than three standard deviations away from the mean for their condition (1.1% of trials) were rejected as outliers. The correlation between correct RTs and error rates was of +0.54 (*n.s.*), thus showing no speed-accuracy trade-off. The ANOVA applied on the correct RT's observed in these age-matched with factors of letter confusability and N size showed a highly significant effect of N size [$F(1,11) = 28.1$; $p < 0.001$], no effect of letter confusability [$F(1,11) < 1$], and no interaction between those two factors [$F(1,11) = 1.8$; *n.s.*]. The significant neighbourhood size effect indicates shorter RT's with targets that have many orthographic neighbours than with targets that have few. The analysis applied on error rates showed no main effect of either N size [$F(1,11) < 1$] or of letter confusability [$F(1,11) < 1$], but a significant cross-over interaction of N size x confusability [$F(1,11) = 4.7$; $p = 0.05$]. However, simple effect analyses of this interaction revealed no significant effect of orthographic neighbourhood size for either low [$F(1,11) = 1.3$; *n.s.*] or high letter confusability targets [$F(1,11) = 2.1$; *n.s.*].

Insert Figs. 7 and 8 near here.

Figs. 9 and 10 show the correct RTs obtained in the young neurologically intact readers in each condition. Correct RTs that were more than three standard deviations away from the mean for their condition (0.6% of trials) were rejected as outliers. The correlation between correct RTs and error rates was of +0.82 (*n.s.*), thus showing no speed-accuracy trade-off. The analysis of correct RTs in the young normal readers showed a significant effect of N size [$F(1,9) = 57.8$; $p < 0.001$] but no effect of letter confusability [$F(1,9) = 2.1$; *n.s.*] and no interaction [$F(1,9) < 1$]. The N size effect indicated shorter RT's for target words that have a large number of orthographic neighbours than for words that have few. The outcome of the data analyses on error rates largely paralleled that for RTs. Thus, the results showed a marginally significant facilitatory effect of increased N size [$F(1,9) = 4.8$; $p = 0.05$] but no effect of letter confusability [$F(1,9) = 1.7$; *n.s.*] and no interaction between N size and letter confusability [$F(1,9) < 1$]. Again the N size effect indicates improved performance with the increased neighbourhood size of the target.

Insert Figs. 9 and 10 near here.

Average correct RTs and error rates for IH are presented in Figs. 11 and 12, respectively. The correlation between RTs and error rates was +0.98 ($p = 0.02$) which indicates no speed-accuracy trade-off. Correct RTs that were more than three standard deviations away from the mean for their condition were discarded. A total of two data points (1.36 % of correct trials) were removed from the analysis on this criterion. A two-way ANOVA conducted on correct RTs with orthographic neighbourhood size and letter confusability as factors showed a main effect of neighbourhood size [$F(1,141) = 14.50$; $p < 0.001$], but no main effect of letter confusability [$F(1,141) = 2.90$; *ns*]. However, the interaction of N size

and letter-confusability was significant [$F(1,141) = 8.18$; $p < 0.01$]. Simple effects analyses indicated that increased N size had a facilitatory effect with low letter-confusability words [$F(1,141) = 22.90$, $p < 0.001$]. In contrast, N size had no effect with high letter-confusability targets [$F(1,141) < 1$]. A chi-square analysis showed there was no significant difference in error rates across conditions [$c^2(1) = 1.70$; *ns*].

Insert Figs. 11 and 12 near here.

Discussion

The present results indicate that the word naming performance of neurologically intact normal readers (in age-matched subjects and younger university students) is resistant to the effect of letter confusability. Thus, this factor had no impact on their performance and it did not prevent the facilitatory N size effect. In contrast, the results of LBL dyslexic IH show that the facilitatory effect of increased N size on his overt word recognition performance is prevented with high letter-confusability target words.

This result is accounted for by a degradation of the letter identity information that is passed by parallel processing to the lexical-orthographic representation system with high letter confusability. This degradation prevents the effective discrimination between the target and its neighbours (required for increased N size to facilitate overt word recognition performance) and thus blurs the contrast between their activations. However, this discrimination between the target and its orthographic neighbours appears possible with low letter-confusability

target words since a facilitatory effect of increased N size is observed with these items.

This residual discrimination capacity for low letter-confusability words observed in IH cannot be conceived as normal by any means, however. Indeed, even with low letter-confusability words, the patient remains incapable of overtly recognizing items based on parallel letter processing in a consistent manner (cf. Exp. 3), in contrast to neurologically intact readers (Forster & Chambers, 1973; Frederiksen & Kroll, 1976; Weekes, 1997). Furthermore, the magnitude of the N size effect with low letter-confusability is many times greater in IH (277 ms) than in either group of normal controls (age-matched: 8 ms; young: 18 ms). Again, this points to a severe anomaly of orthographic encoding in IH.

The account that is proposed therefore is that parallel letter processing in IH provides an impoverished input to the lexical-orthographic system with respect to the discrimination between visually similar letters and that an increase in letter confusability only worsens the problem. Because of this impoverished input, parallel letter processing cannot support overt word recognition and the patient is forced to examine the constituent letters of the target in sequence. With low letter-confusability targets, it is assumed that the activation contrast between the lexical representations of the target and its neighbours, although not sufficient to permit definite identification of the target, is large enough to allow a facilitatory effect of increased N size. With high letter confusability however, it is proposed that the patient is no longer capable of reliably discriminating the target from its neighbours. In other words, it is hypothesized that the activation contrast between the target and its neighbours is either null or too weak to support the neighbourhood size effect.

GENERAL DISCUSSION

The research reported in the present paper has focussed on the issues of sequential and parallel letter processing in the acquired reading disorder of LBL dyslexia. Exp. 1 demonstrated that a parametric increase in the number of orthographic neighbours of the target causes a continuous, linear decrease of word naming times in LBL patient IH. The results of Exp. 2 however, showed that the magnitude of the word length effect in IH is not modified significantly by whether words of different lengths are matched or not on N size. These findings suggest that the word length effect in LBL dyslexia is not an artefact of N size, but rather that it truly reflects sequential letter processing. In turn, this also suggests that the facilitatory effect of increased N size in the disorder rests on parallel letter processing. The results of Exp. 3 demonstrated a cost of increased letter confusability on overt word identification performance in IH. They also suggest that the patient's rate of sequential letter identification is not affected by letter confusability, and thus that the latter effect only impacts on parallel letter processing. Congruently, Exp. 4 showed that the facilitatory effect of increased N size in the word naming performance of LBL patient IH is abolished if the words used are made of high-confusability letters. In sharp contrast, no effect of letter confusability was observed in the performance of neurologically intact readers, who only showed a facilitatory effect of increased N size in Exp. 4. Taken together, the effects of N size and of letter confusability as well as the form of the interactive effect of these factors in IH provide key indications regarding the nature of his functional impairment and of the mechanisms involved in his residual reading capacity. These are discussed below.

Parallel letter processing in LBL dyslexia

The notion that a residual capacity for parallel letter processing exists and that it may contribute to overt word recognition performance in LBL dyslexia is not widespread among investigators of the disorder. In fact, the only authors to argue for such a possibility thus far are Howard (1991) and Arguin et al. (1998). A residual contribution of parallel letter processing to overt word recognition in LBL dyslexics may be more widespread than previously thought, however. Indeed, in a separate investigation of three other LBL dyslexics Arguin & Bub (1996) have found a facilitatory effect of increased N size on word naming performance in all of them (see also Montant & Behrmann, 2001). From the theoretical and empirical arguments discussed in this paper, the occurrence of such a facilitatory N size effect suggests that parallel letter processing contributes to overt word recognition performance in these patients as well. Clearly however, parallel letter processing is incapable of supporting overt word recognition on its own in the disorder, hence the necessity of sequential letter identification. A likely reason for this incapacity is indicated by the letter confusability effect and the interaction of this factor with orthographic neighbourhood size that were demonstrated in Exps. 3 and 4.

The results of Exp. 3 demonstrate a cost of high letter-confusability that does not vary significantly across different word lengths. This shows that orthographic encoding is constrained by visual similarity, which is congruent with previous observations pointing to an early impairment of letter encoding in LBL dyslexia (see Behrmann & Shallice, 1995, and Arguin et al., 1998; for reviews). In Exp. 4, the interaction of letter confusability x N size indicates that the facilitatory effect of increased orthographic neighbourhood size, although present for low letter-confusability words, is prevented by high letter confusability. We argue

that this observation provides a fundamental insight with respect to the processes involved in visual word recognition in IH.

The occurrence of a facilitatory effect of increased N size implies that the activation of lexical-orthographic representations resulting from parallel letter processing can discriminate between the target and its orthographic neighbours. This discrimination is achieved by IH with low letter-confusability targets since his performance does benefit from increased N size in this condition. However, this discrimination between the target and its neighbours appears impossible with high letter-confusability items. In terms of lexical activations, this suggests that the background noise created by a high level of activation of the orthographic neighbours of the target has become too severe (compared to low letter-confusability targets) to permit the resolution of the target that is required for increased N size to facilitate reading performance. Given that this elimination of the N size effect occurs through a manipulation at the letter-level of the stimulus (i.e. letter confusability), it is proposed that the mechanisms involved in parallel letter processing in IH are impaired, such that they fail to provide a definite discrimination between the constituent letters of the target and other, visually similar letters.

With low letter-confusability targets, the difference in the activation of the lexical representation of the target and those of its orthographic neighbours that results from parallel letter processing seems larger (i.e. reduced background noise relative to target activation) than with high letter-confusability words, thus allowing the N size effect to occur. Even then however, the activation contrast between the target and other words appears insufficient to support overt identification performance. Thus, even with low letter confusability targets, overt word recognition requires serial letter processing (c.f. large word length effect in Exp. 3 with low letter-confusability words). It is argued that this situation is caused by the

fact that a substantial degree of background noise still occurs in the activation of lexical-orthographic representations with low letter-confusability words, which prevents the reliable absolute identification of the target (i.e. that the item presented is *x* and not any other word; e.g. Arguin & Bub, 1995; Luce, 1959; 1977) based on parallel letter processing;

It may be speculated that the residual parallel processing capacity of LBL dyslexics suggested by the present findings is also responsible for the implicit reading phenomena previously described in a number of patients (see Introduction). Indeed, it appears that the key difference between tasks of absolute identification (as in the word naming task) and classification (as in the lexical or semantic decision tasks) concerns the decision criteria that are applied over the relevant set of internal representations (here, lexical-orthographic representations). Thus, Arguin and Bub (1995; see also Luce, 1959; 1977) have shown, on the basis of both empirical evidence and computational simulations, that absolute identification decisions rest on a signal-to-noise ratio criterion. Specifically, this type of decision appears to require the ratio of the activation of the target representation over that of all other representations within the relevant representation domain to reach a particular level before an identification response can be made. If this ratio is too low because of an excessive level of background noise produced by the activation of alternative representations, no absolute identification response may be emitted. In contrast, Arguin and Bub (1995) proposed that classification decisions only require a sufficient overall level of activation to be reached within the relevant representation system, and that the degree of background noise existing in that system is irrelevant. This more lenient decision criterion therefore, would allow correct classification responses even though the background noise produced by the activation of words other than the target would be so high as to prevent its absolute identification. Given these assumptions about decision criteria, the

observation of implicit reading in LBL dyslexia is entirely congruent with the hypothesis discussed above of 'noisy' lexical-orthographic activation resulting from a residual but impaired parallel letter analysis.

The neural basis of implicit reading in LBL dyslexia is currently an unresolved issue (see e.g. Behrmann et al., 1998; Saffran & Coslett, 1998). The above proposal that the residual parallel letter processing capacity of LBL dyslexics that contributes to overt word recognition is the functional basis for implicit reading relates to this issue. Saffran and Coslett (1998; see also Coslett & Saffran, 1994) have argued that particular right-hemisphere mechanisms, which are otherwise not involved in overt word recognition, would be responsible for implicit reading in LBL dyslexics. Clearly, most LBL dyslexics must encode visual stimuli via their right hemisphere because of a right hemianopia caused by the left occipital damage. Saffran and Coslett have presented arguments suggesting that the right hemispheric contribution may extend well beyond perceptual encoding and that it may entirely support implicit reading. Possibly the most direct support for an extended contribution of the right hemisphere in LBL dyslexia comes from the study of Coslett and Monsul (1994). They have shown that transcranial magnetic stimulation (TMS) applied to the right temporo-parietal area of an LBL dyslexic had a dramatic impact on his reading accuracy (71% correct without TMS; 21% correct with TMS). TMS had no impact when applied to the homologous area of the left hemisphere. In contrast, Behrmann et al. (1998) have argued that there is no need to invoke special right hemispheric mechanisms to explain phenomena such as implicit reading. They proposed that these observations can be well accounted for on the basis of a single integrated reading system involving the left and right hemispheres, which would subserve both implicit and overt word recognition. By identifying the mechanisms involved in implicit reading with the residual parallel letter processing capacity modulating overt word recognition performance in

LBL dyslexia, the hypothesis proposed above sides with the view proposed by Behrmann et al. It should be emphasised however, that this position does not rule out the possibility of an advanced right hemispheric contribution to the residual parallel letter processing capacity of LBL dyslexics. This remains an open issue that will require direct empirical testing to be resolved.

Sequential letter processing in LBL dyslexia

Since residual parallel letter processing fails to consistently support overt word recognition in LBL dyslexia, it may appear unsurprising that sequential letter identification is required for this type of performance. At a deeper level of analysis however, a fundamental issue concerns the reasons why the impasse reached by parallel letter processing with respect to word identification can be solved by patients reverting to sequential letter identification.

The independent effects of word length and of letter confusability observed in Exp. 3 suggest that sequential letter analysis in LBL dyslexic patient IH is relatively impervious to the negative impact of visual similarity among letters, which only appears to significantly affect parallel processing. This implies that whereas parallel processing may provide ambiguous information to lexical-orthographic representations about the constituent letters of the target word, sequential letter processing appears to provide clearer and more decisive information in this regard.

The difference between sequential and parallel letter processing may be characterized in terms of the way selective visual attention is allocated to target words. Specifically, parallel processing is associated with attention being spread over the entire surface area of the word. In

contrast, sequential processing is associated with the narrowing down of the focus of attention on individual letters, which are scanned one after the other. One assumed function of focussed attention is to increase the signal-to-noise ratio in the processing of the stimulus (Bashinski et Bacharach, 1980; Hawkins, Hillyard, Luck, Mouloua, Downing & Woodward, 1990; Henderson, 1991; Hummel & Stankiewicz, 1998). Consequently, the focussed attention to individual letters that is involved in sequential letter processing may be conceived as improving the capacity of the letter identification system to resolve the difference between visually similar letters and therefore, of passing a more definite signal to the lexical-orthographic representation system about the identities of the letters constituting the target than parallel processing. This, of course, would be fundamental in LBL dyslexia since parallel letter analysis on its own is incapable of supporting overt word recognition due to unresolved uncertainty at the letter level. It is conceivable that focussed attention at the letter level may also contribute to reading performance in neurologically intact observers when stimulation conditions, for instance, are particularly unfavourable (see e.g. Behrmann et al., 1998b; Plaut 1999; for relevant discussions). Similarly to LBL dyslexia, sequential letter processing in these circumstances would serve to improve the low signal-to-noise ratio at the lexical-orthographic level, that is itself caused by uncertainty regarding the identity of the constituent letters of the target word.

CONCLUSIONS

The above findings suggest a view of LBL dyslexia whereby parallel letter processing still occurs and may contribute to overt word recognition performance. However, lexical activation resulting from this

parallel letter analysis suffers from a level of background noise that is too high to permit the absolute identification of the target. Based on the letter confusability effect, it is argued that the background noise at the lexical level is a consequence of a problem preventing the letter identification stage in fully resolving the differences between visually similar letters. This implies, congruently with a number of previous observations, that the disorder of LBL dyslexia may be assigned to an impaired letter encoding stage (Arguin & Bub, 1993; Behrmann & Shallice, 1995; Kay & Hanley, 1991; Reuter-Lorenz & Brunn, 1990). It is proposed that these difficulties associated with parallel letter processing render sequential letter analysis mandatory for overt word recognition. Further, it is proposed that this sequential processing involves focussed attention at the level of individual letters, which serves to reduce noise at the letter identification stage. Although the present study was conducted in a single LBL dyslexic, observations congruent with the present view of the disorder have been obtained from distinct series of experiments in three other cases (patients DM, JL, and JT), which will be the object of a separate report.

FOOTNOTES

1. Prediction based on the observation of a linear reduction of correct RTs of 37 ms per additionnal neighbour and on the variation of the average number of orthographic neighbours across word lengths (see above).

2. A possible alternative experimental strategy would have been to construct a factorial design of Word length x N size. This was attempted but it proved impossible to obtain a sufficient number of words per condition due to the very high correlation existing between word length and N size and to the limited number of words that are available in the English vocabulary.

3. Letter confusion matrices are only available for uppercase letters and not for lowercase letters. Consequently, all experiments in which letter confusability is a factor (Exps. 3 and 4) or a control variable (Exp. 2) were conducted with stimuli printed in uppercase letters.

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FIGURE CAPTIONS

Fig. 1. Average correct response times (ms) in IH as a function of orthographic neighbourhood size (N size; Exp. 1).

Fig. 2. Error rates (%) in IH as a function of orthographic neighbourhood size (N size; Exp. 1).

Fig. 3. Average correct response times (ms) in IH as a function of word length and N size control (Exp. 2).

Fig. 4. Error rates (%) in IH as a function of word length and N size control (Exp. 2).

Fig. 5. Average correct response times (ms) in IH as a function of word length and letter confusability (Exp. 3).

Fig. 6. Error rates (%) in IH as a function of word length and letter confusability (Exp. 3).

Fig. 7. Average correct response times (ms) in neurologically intact readers age-matched to IH as a function of letter confusability and N size (Exp. 4).

Fig. 8. Error rates (%) in neurologically intact readers age-matched to IH as a function of letter confusability and N size (Exp. 4).

Fig. 9. Average correct response times (ms) in young neurologically intact readers as a function of letter confusability and N size (Exp. 4).

Fig. 10. Error rates (%) in young neurologically intact readers as a function of letter confusability and N size (Exp. 4).

Fig. 11. Average correct response times (ms) in IH as a function of letter confusability and N size (Exp. 4).

Fig. 12. Error rates (%) in IH as a function of letter confusability and N size (Exp. 4).

FIGURES

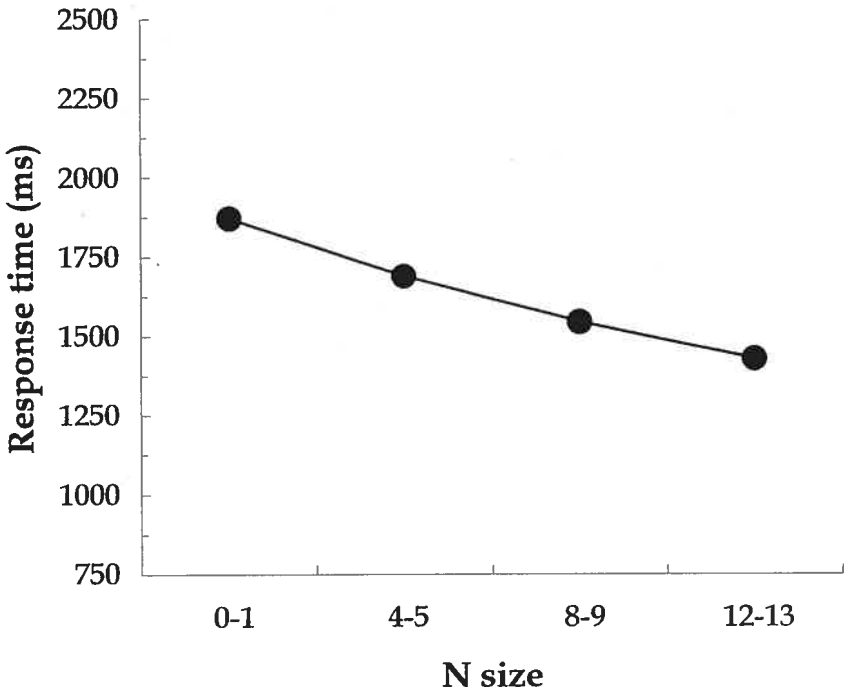

Fig. 1 — Arguin, Fiset & Bub

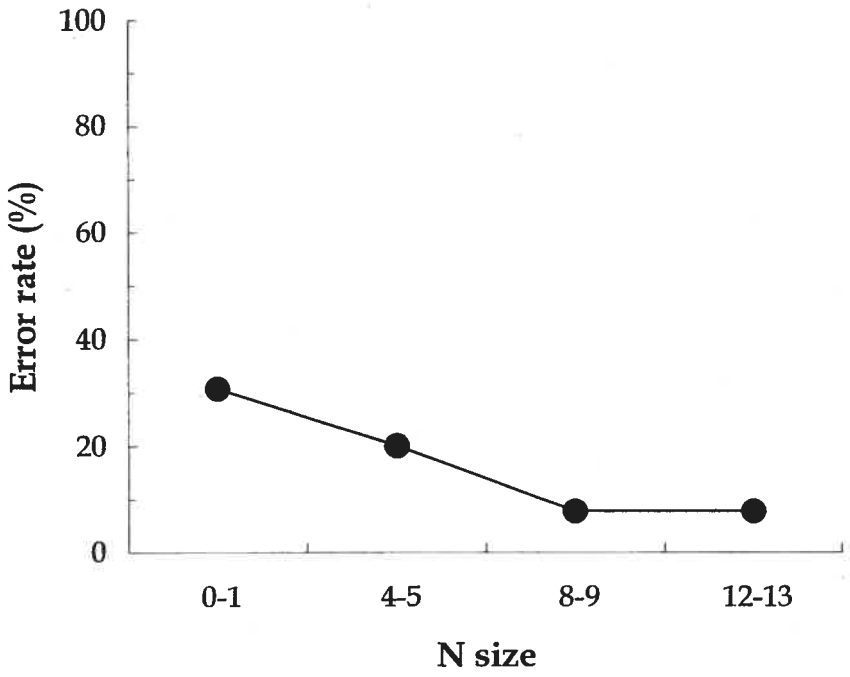

Fig. 2 — Arguin, Fiset & Bub

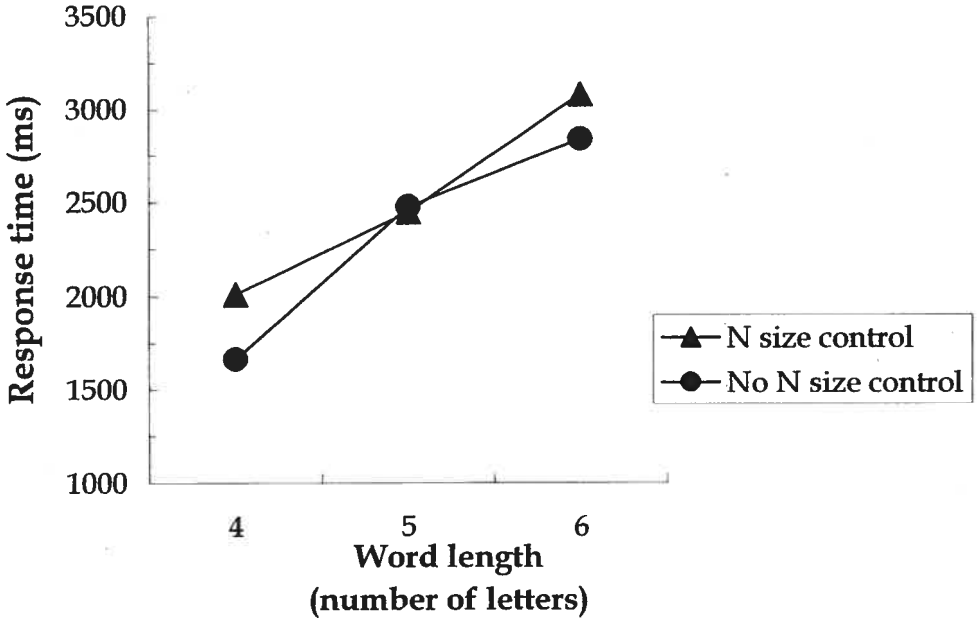

Fig. 3 — Arguin, Fiset & Bub

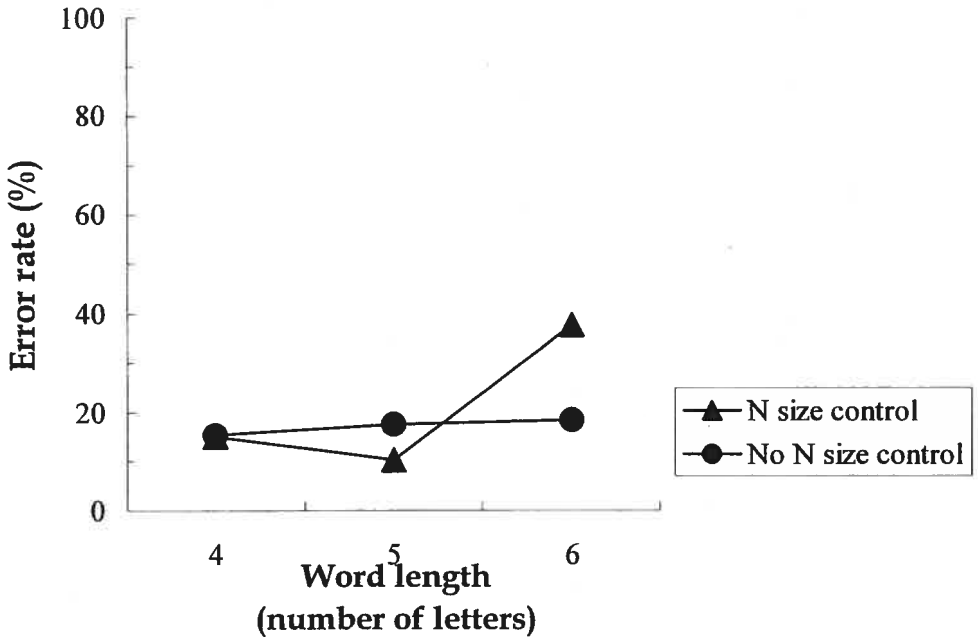

Fig. 4 — Arguin, Fiset & Bub

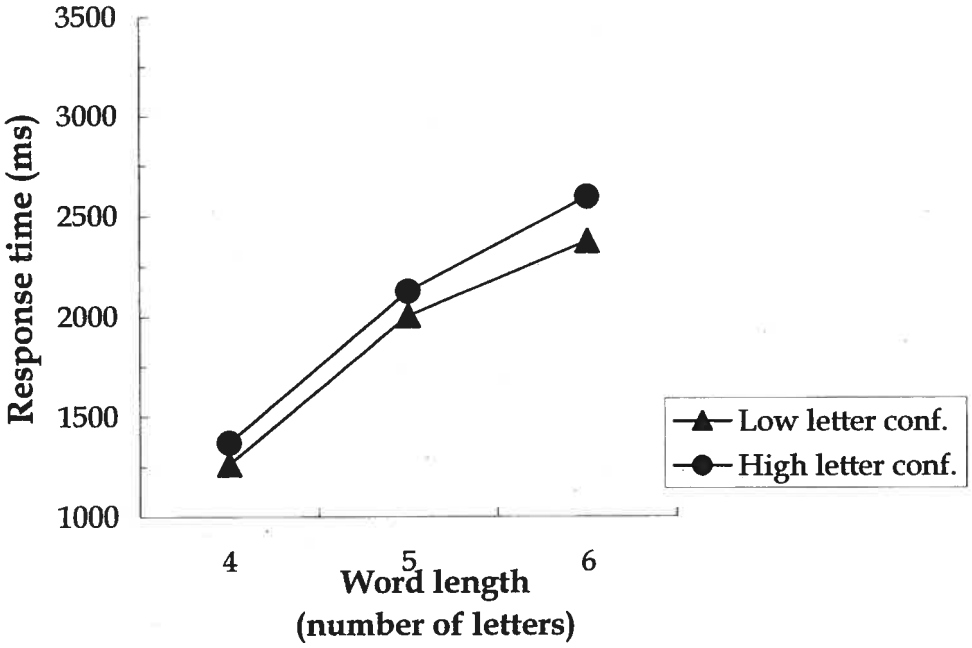

Fig. 5 — Arguin, Fiset & Bub

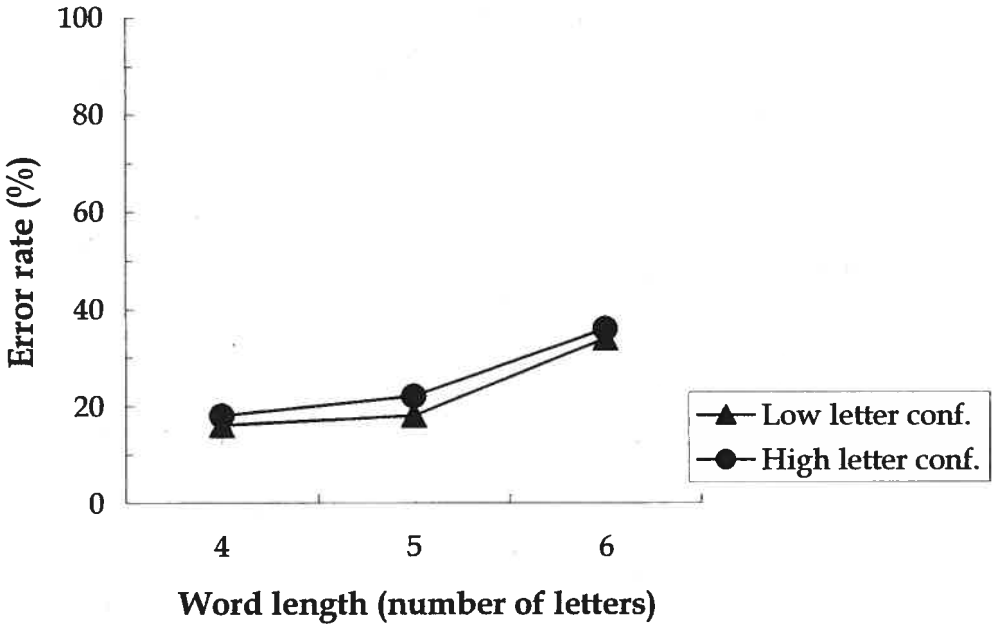

Fig. 6 — Arguin, Fiset & Bub

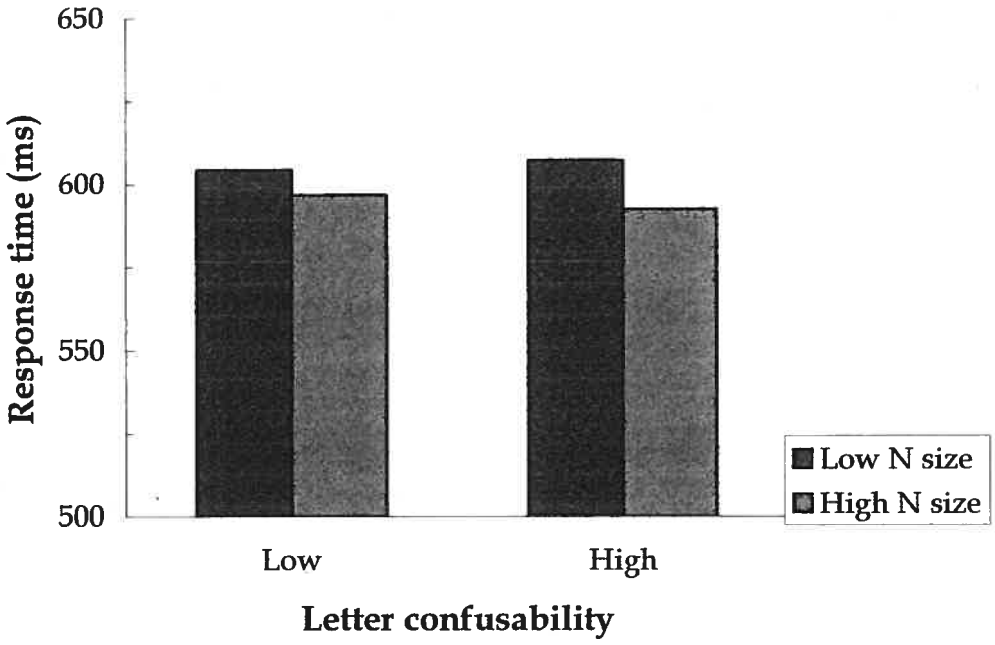

Fig. 7 — Arguin, Fiset & Bub

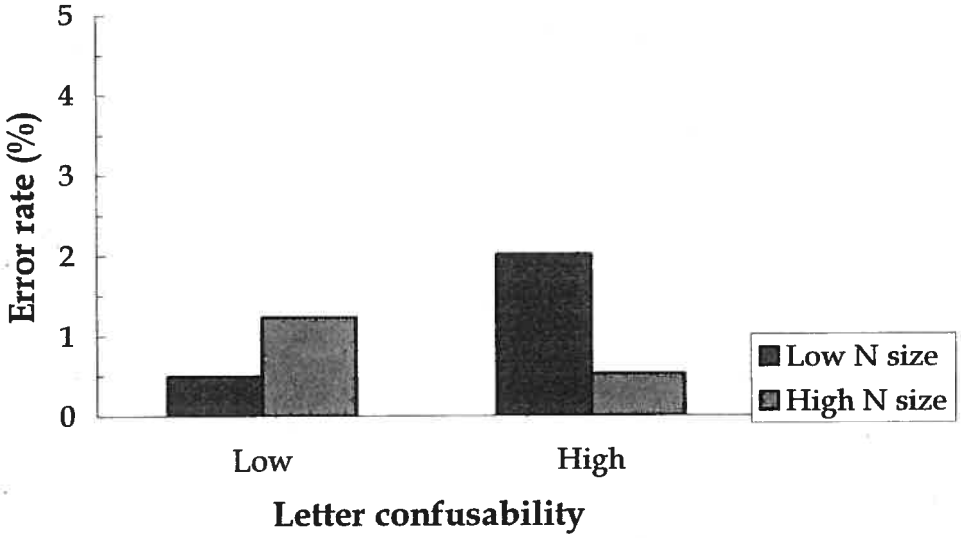

Fig. 8 — Arguin, Fiset & Bub

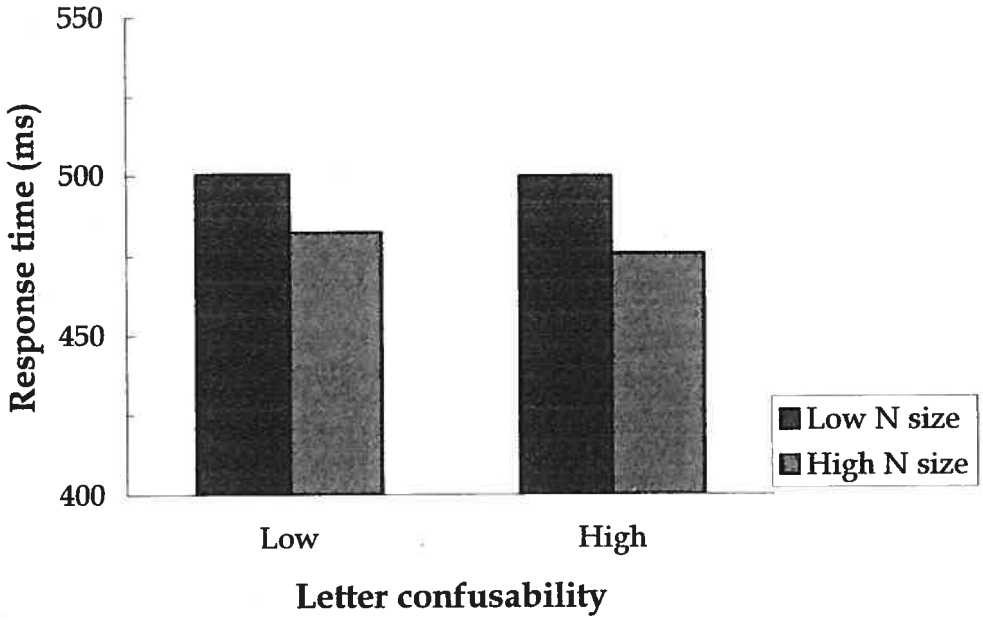

Fig. 9 — Arguin, Fiset & Bub

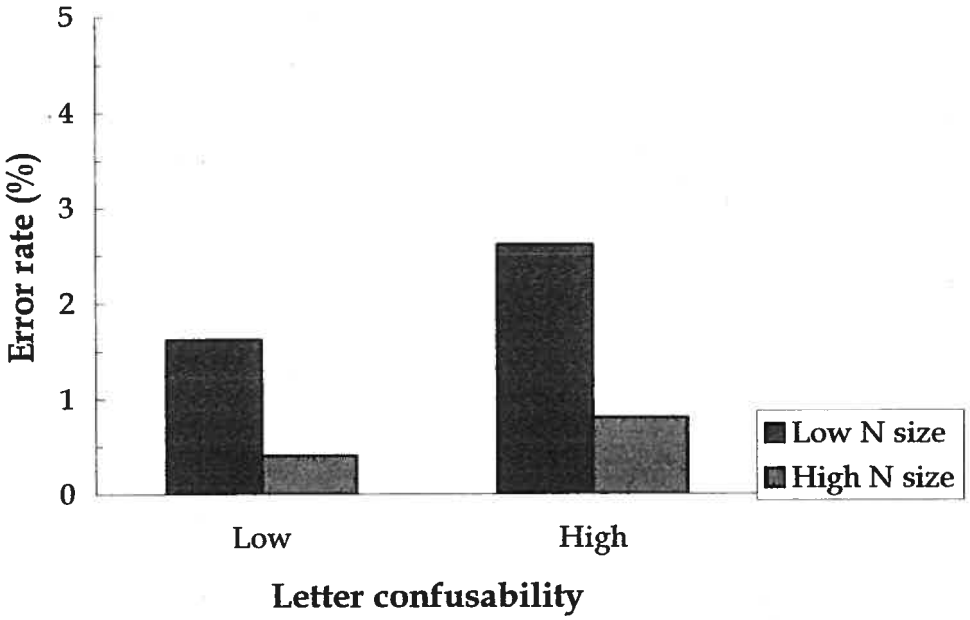

Fig. 10 — Arguin, Fiset & Bub

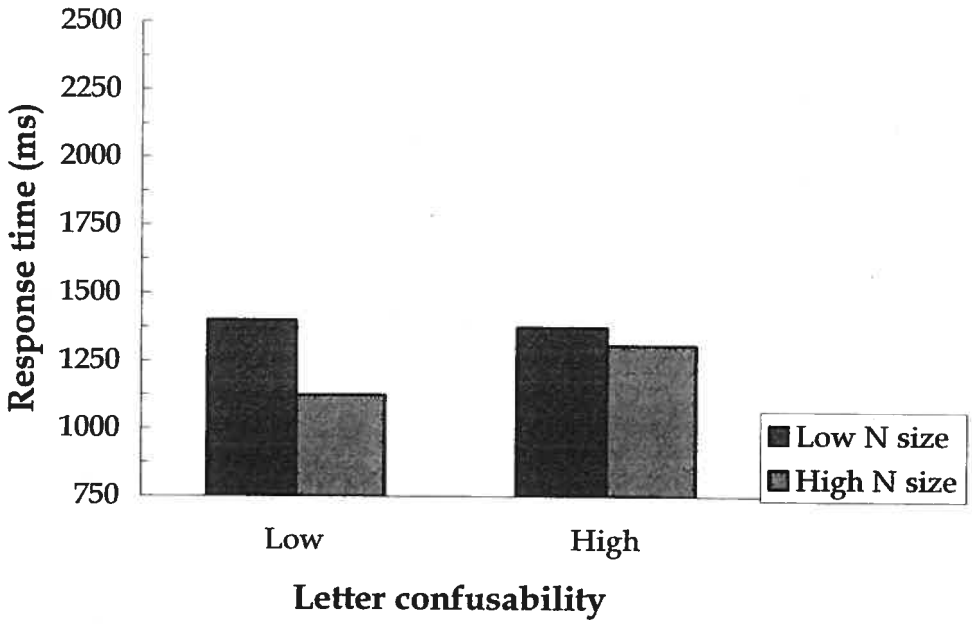

Fig. 11 — Arguin, Fiset & Bub

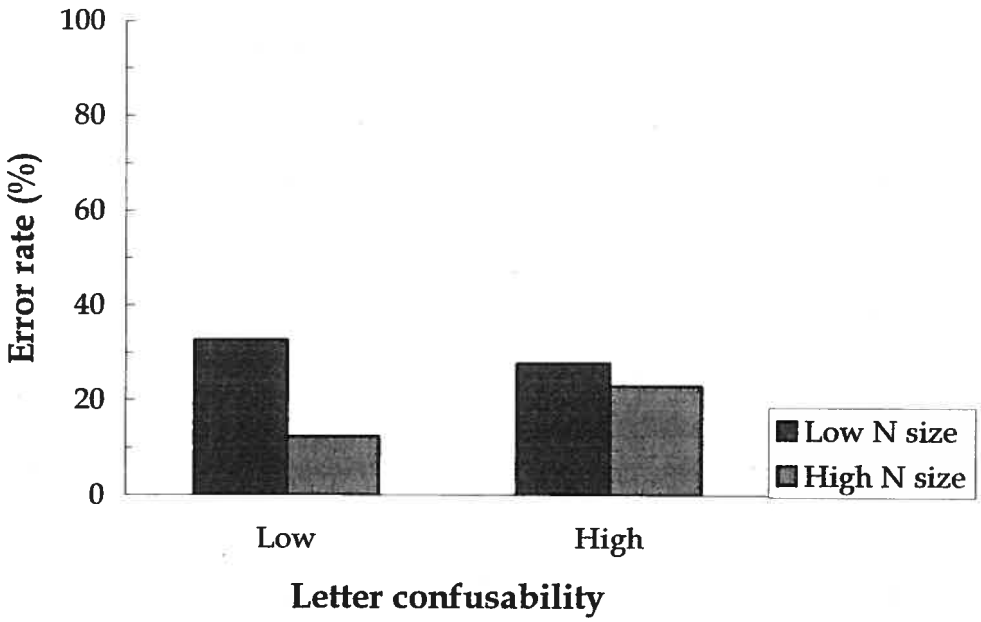

Fig. 12 — Arguin, Fiset & Bub

CHAPITRE TROISIÈME :

DEUXIÈME ARTICLE

SEEKING A NORMAL MODEL OF LETTER-BY-LETTER DYSLEXIA: REDUCED CONTRAST AND N SIZE JOINTLY ACT ON THE WORD LENGTH EFFECT

Stéphanie Fiset, Martin Arguin & Daniel Fiset

Groupe de Recherche en Neuropsychologie et Cognition,
Département de psychologie, Université de Montréal

and

Centre de recherche de l'Institut Universitaire de Gériatrie de Montréal

ABSTRACT

One account for the abnormally large word length effect in letter-by-letter (LBL) dyslexia is that visual encoding is impaired at a processing stage prior to accessing abstract orthographic representations. Following this hypothesis, the word length effect characteristic of LBL dyslexia should be reproducible in neurologically intact readers by visually degrading the stimulus such as by reducing the luminance contrast between the word and its background. In support of this prediction, Nelson, Behrmann & Plaut (personal communication) succeeded in obtaining a significant word length effect in normal readers with low-contrast stimuli. Our experiment attempted to replicate these findings while controlling for orthographic neighbourhood size. With this control exerted, normal readers did not show a significant word length effect with low-contrast stimuli whereas they did when neighbourhood size was not controlled for. This contrasts with LBL dyslexics, who show a word length effect that is unaffected by orthographic neighbourhood size control. It is concluded that luminance contrast reduction on its own fails to reproduce the visual input degradation that seems to affect LBL dyslexics.

Key words: Letter-by-letter dyslexia, Dyslexia, Reading, Simulation in normal readers, Neighbourhood size, Visual processing

INTRODUCTION

Letter-by-letter (LBL) dyslexia is an acquired reading disorder that follows left occipito-temporal lesions (Black & Behrmann, 1994; Damasio & Damasio, 1983; Dejerine, 1892) in previously fluent adult readers. It is usually accompanied by right hemianopia. These dyslexics require an abnormally long time to read aloud single words, therefore resulting in the loss of the ability to read text quickly and efficiently. Some patients, but not all, have to name the individual letters out loud before being able to put the whole word together. The essential diagnostic feature of LBL dyslexia is the word length effect, i.e. a large linear increase in the time required for the overt recognition of a word as a function of the number of letters it comprises (e.g. Patterson & Kay, 1982). Thus, the time required to read a word aloud increases from 500 ms to several seconds (depending on the patient) for each additional letter in the stimulus (Arguin & Bub, 1993; Bowers, Bub & Arguin, 1996b; Farah & Wallace, 1991; Patterson & Kay, 1982; Reuter-Lorenz & Brunn, 1990; Warrington & Shallice, 1980).

The locus of the functional damage responsible for LBL reading and its associated features is still being debated. Various accounts have been proposed to explain this disorder. To summarise briefly, the different explanations can be grouped into the peripheral and the central hypotheses. The peripheral explanation assumes that the locus of damage is located prior to the activation of the orthographic representation of words. It has been argued that this damage may relate to generalised poor visual encoding (Farah & Wallace, 1991; Friedman & Alexander, 1984; Kinsbourne & Warrington, 1962; Levine & Calviano, 1978; Rapp & Caramazza, 1991), a deficit in abstract letter identification, or some other impairment of the encoding stage that prevents the normal activation of the orthographic representations of the stimulus (Arguin & Bub, 1993;

Arguin & Bub, 1994; Arguin, Fiset & Bub, 2002; Behrmann, Plaut & Nelson, 1998; Behrmann & Shallice, 1995; Kay & Hanley, 1991; Montant & Behrmann, 2001; Reuter-Lorenz & Brunn, 1990). The alternative account of LBL reading, the central hypothesis, suggests that the deficit occurs at a late stage in word reading, which may be a damaged orthographic word-form system (Warrington & Shallice, 1980), or an impaired access to phonological word-forms following a relatively intact access to orthographic word-forms (Bowers, Arguin & Bub, 1996a).

Normal model of LBL dyslexia

If the peripheral hypothesis is the correct explanation of LBL dyslexia, then some type of visual degradation of the stimulus should be found which provokes a pattern of performance in neurologically intact readers that is similar to that characterising LBL dyslexia. Nelson et al. (personal communication) tested this hypothesis by presenting their stimuli at different levels of luminance contrast. They used three different levels of contrast that were produced by varying the luminance intensity of the stimulus (high contrast: intensity at 40% of the maximum of their monitor; medium contrast: intensity at 47.7% of maximum; and low contrast: intensity at 48.2% of maximum) which was presented over a background of 50% intensity. The background and stimuli were all gray, so the colour contrast was null. The main observation of Nelson et al. is that a relatively substantial word length effect can be found in normal readers with contrast-reduced stimuli. Thus, the word length effect was of 16.7 ms/letter, 31.4 ms/letter and 67.8 ms/letter for the high, medium, and low contrast conditions respectively. This suggests that symptoms similar to those associated with LBL dyslexia can be elicited by manipulating the quality of the visual input. In turn, this is congruent with the peripheral type of account of LBL dyslexia outlined above.

However, there appears to be a significant drawback in the Nelson et al. experiment that prevents a definitive conclusion. Thus, the authors failed to control for orthographic neighbourhood size in the construction of their word lists and it is possible that this may be involved in the word length effect they observed in their normal readers. Orthographic neighbours of a target word are other words of the same length that differ from it by just one letter (e.g. horse / house). On every occasion that it was tested, a major effect of orthographic neighbourhood size (N size) has been found in LBL dyslexics in overt word reading (Arguin & Bub, 1996; Arguin, Bub, & Bowers, 1998; Arguin et al., 2002; Arguin & Bub, under review; Montant & Behrmann, 2001): words having a high number of orthographic neighbours are read faster than words having few neighbours. This facilitatory effect is in the same direction as in normal readers, but only much larger (by about one order of magnitude; Andrews, 1989, 1992; Arguin et al., 1998; Carreiras, Perea & Grainger, 1997; Sears, Hino & Lupker, 1995). For a discussion of the larger amplitude of the N size effect in LBL readers, see Arguin et al. (2002).

Arguin et al. (2002) noted that a strong linear inverse relationship exists between word length and N size in English (see also Weekes, 1997); longer words having fewer neighbours than shorter words. As underlined above, Nelson et al. did not match words of different lengths according to N size. Congruently, an examination of their stimulus set reveals that the 3-letter, 5-letter and 7-letter words they used had an average of 7.63, 2.27 and 0.08 neighbours, respectively. It thus appears possible that the word length effect obtained in their experiment was an artefact of N size, with shorter words being read faster because they have more orthographic neighbours than longer words.

The present experiment will assess this alternative account of the results of Nelson et al. This test is relevant for the issue of whether stimulus degradation can actually produce symptoms of LBL dyslexia in normal readers, as well as for the peripheral account of the disorder. In our study, normal readers were presented low luminance-contrast words of varying lengths. In one stimulus set, words of different lengths were matched individually on N size, as well as on other important variables. In the other set, no control of N size was performed: thus, items of increasing lengths naturally had markedly decreasing numbers of orthographic neighbours. If the alternative account of the Nelson et al. results outlined above is correct, then a word length effect should be observed only in the condition where N size has not been controlled for.

METHODS

Subjects:

Twenty normal readers took part in the experiment. They were aged between 18 and 29 years (mean = 23 years old). All were right-handed university students, had no history of learning disabilities, and had normal or corrected vision.

Stimuli:

Two sets of 120 stimuli were used, including an equal number (n = 30) of four-, five-, six- and seven-letter French words (taken from the French BRULEX data base: Content, Mousty et Radeau, 1990). None of the words contained diacritic marks (é, è, ê, etc.), because subjects could not see them clearly when luminance contrast was reduced. In French, diacritic marks dictate word pronunciation and keeping words with accents in the present context could have led to an inflation in

error rates. In one set, words of different lengths were matched item-wise according to their number of orthographic neighbours (N size). Consequently, there was no significant variation in N size across words of different lengths (average N sizes of 1.8, 1.4, 1.1, 1.0 for four-, five-, six-, and seven-letter words respectively, $F(3, 116) = 1.0$, *n.s*). N size values were obtained from a software designed in our laboratory. In the other set of items, no control of N size was performed. Consequently, there was a significant difference in N size across words of different lengths (average N size of 6.47, 2.93, 1.03, 0.33 for four-, five-, six-, and seven-letter words respectively $F(3, 166) = 35.4$, $p < .001$.

Stimuli were also matched across lengths and neighbourhood size control (N size control or no N size control) according to lexical frequency (BRULEX: Content et al., 1990), letter confusability (see Arguin et al. 2002), and bigram frequency (obtained from a software designed in our laboratory). For lexical frequency and letter confusability, a two-way ANOVA with neighbourhood size control and word length as factors indicated the absence of main effects or of an interaction (all $F's < 1.5$, *n.s*). However, we were unable to perfectly match the N size control and no N size control word sets on bigram frequency $F(1,238) = 16.7$, $p < .001$, the list without N size control having a higher bigram frequency than the list with N size control). However, since bigram frequency does not seem to have an effect on word naming (Andrews, 1992), it appeared unlikely that this variable may have affected response latencies in this experiment. Moreover, a comparison of bigram frequency as a function of both N size control and word length showed only a main effect of N size control $F(1,232) = 16.4$, $p < .001$, without any effect of length $F(3,232) < 1$ and no interaction $F(3,232) < 1$. This means, that any effect of word length or difference in the word length effect as a function of N size control on the reading performance observed in the present experiment cannot be attributed to a bigram frequency artefact.

Procedure:

Half the subjects (10) were shown the set of words where no control of N size was performed, whereas for the other subjects the set with N size controlled for was presented. In each set, words of different lengths were randomly mixed in a single block of 120 trials. Each trial began with a 750 ms fixation point, displayed at the centre of the computer screen. This was immediately followed by the target printed in uppercase and displayed at the centre of the screen. The subject's task was to name the target as quickly as possible while avoiding errors. Subjects were seated 55 cm away from the computer screen. Stimuli were shown on a Viewsonic E-771 screen, linked to a Power PC 7100/80. The Psychlab software (Bub, D. & Gum, T., 1998. Psychlab Software, University of Victoria, Canada) was used to control the presentation of stimuli. All stimuli appeared in reduced contrast (luminance of 4.51 cd/m^2 over a background of 4.61 cd/m^2, as measured with a photometer). Stimuli were printed in Geneva 24 point bold font. Responses were registered by a voice-key (engineered in our laboratory) connected to the computer controlling the experiment. After each response, the experimenter entered the subject's response via the computer keyboard and then triggered the next trial. A total of 41 (1,7%) trials were lost due to the failure of the subjects' response to trigger the voice-key. These trials were not considered in the data analysis.

RESULTS

Average correct RTs and error rates are shown in Figs. 1 and 2 respectively. The correlation between RTs and error rates was of -.58, which suggests a speed-accuracy trade-off. The detailed data analysis presented below however, falsifies this possibility.

Insert Figs. 1 and 2

RTs analysis by subjects:

Seventy-nine data points (3,19% of all trials) were removed on an individual basis from the RT analysis because the response latencies were more than 2.5 SDs away from the mean of their condition. A two-way ANOVA performed on correct RTs, with neighbourhood size control (N size control vs. no N size control) and word length as factors showed a main effect of word length $F(3,54) = 3.7$, $p < .05$, but no effect of N size control $F(1,18) = 1.1$, *n.s.* The results also indicated an interaction between word length and N size control $F(3,54) = 3.1$, $p < .05$. Further analysis showed that the word length effect was significant with the word set on which no control of neighbourhood size was exerted $F(3,27) = 6.0$, $p < .01$, but not when N size was controlled for $F(3,27) = 1.3$, *n.s.* This is congruent with the outcome of the regression analyses performed on correct RTs as a function of word length, which showed a substantial slope of 31.9 ms/letter without N size control ($R^2 = 0.55$) and a weak slope of 0.8 ms/letter with N size control ($R^2 = 0.50$).

RTs analysis by item:

One data point (0.4% of all trials) was removed on an individual basis from the RT analysis because the response latency was more than 2.5 SDs away from the mean of its condition. An ANOVA conducted on the mean RTs for each item, with neighbourhood size control and word length as factors, indicated a main effect of N size control $F(1,231) = 7.8$, $p < .01$, but no word length effect $F(3,231) < 1$. The interaction term was marginally significant $F(3, 231) = 2.2$, $p = .09$. Simple effect analyses revealed that the word length effect was significant when no N size control was performed $F(3,116) = 2.8$, $p < .05$, but that it was not significant when N size was controlled for $F(3, 115) < 1$. This is congruent with the analysis of RTs by subjects.

Error rates analysis:

A two-way ANOVA conducted on error rates, showed no effect of word length nor of neighbourhood size control $F(3,54) < 1$ and $F(1,18) = 1.1$, *n.s.*, respectively. The interaction of length x neighbourhood size control was not significant either $F(3,54) = 1.7$, *n.s.*

Covariance analysis:

As noted above, there seemed to be a speed-accuracy trade-off in the data (c.f. negative correlation of RTs with error rates). A closer look suggests that the negative correlation may have originated from the high error rate with 4-letter words in the condition where no control of neighbourhood size was performed (see Fig. 2). To examine this issue further, a covariance analysis was performed on RTs by subjects, with error rate as the covariate. The results indicated a main effect of word length $F(3,51) = 5.9$, $p = .001$, but not of N size control $F(1,17) = 1.2$, *n.s.* Moreover, the interaction between N size control and word length was significant $F(3,51) = 5.6$, $p < .01$. Simple effects of this interaction showed that the word length effect was significant only when N size was not controlled for (no N size control: $F(3,24) = 6.6$, p .01; N size control; $F(3,24) = 2.4$, *n.s.*).

DISCUSSION

The results from the present experiment indicate that normal readers show a word length effect with low contrast stimuli when no control of N size is performed, but not when N size is controlled for. Since a significant word length effect only occurs when words of different

lengths are not matched on N size, it can be concluded that this length effect is an artefact of N size. Indeed, shorter words (in French and English) have more neighbours than longer words (Arguin et al. 2002) and N size has a facilitatory effect on word reading (Andrews, 1989, 1992; Arguin et al., 1998; Carreiras et al. 1997; Sears et al., 1995). Consequently, shorter words are responded to faster than longer words.

Our findings are incongruent with the claim of Nelson et al. (personal communication) that contrast reduction is a stimulus manipulation that is sufficient to produce a normal model of LBL dyslexia. Indeed, the word length effect that may be found with this type of manipulation does not resist N size control. In contrast to normal readers however, the word length effect is entirely unaffected by whether N size is controlled for in LBL dyslexics (Arguin et al. 2002). These observations indicate that it is critical to match words of different lengths on N size in tests intended to produce a normal model of LBL dyslexia. They also indicate that the sole reduction of luminance contrast is insufficient to provoke symptoms that are truly characteristic of LBL dyslexia in normal readers, even though a marked reduction of luminance is known to increase their response latencies (Legge, Rubin & Luebker, 1987).

This failure to produce a word length effect that resists N size control in the present experiment is unlikely to be a consequence of an insufficient contrast reduction. Thus, we note that the error rate was relatively high in the present experiment, and slightly higher than what is usually found in LBL dyslexics. Moreover, pilot studies in our laboratory have shown that if luminance contrast is reduced to a greater degree that in the present experiment, normal readers exhibit major difficulties in correctly identifying the words and error rates are dramatically high.

It should be underlined that the present conclusions do not imply that the peripheral hypothesis of LBL dyslexia should be rejected, nor that

any visual degradation of stimuli should fail to produce a normal model of LBL dyslexia. Rather, we propose that simple contrast reduction has an impact on the reading performance of neurologically intact individuals that is qualitatively distinct from the functional deficit responsible for LBL dyslexia. In this respect it should be noted that LBL reading is almost always associated with right homonymous hemianopia. Thus, LBL dyslexics have to encode the stimuli parafoveally. Visual spatial acuity falls off quite rapidly as we move away from the fovea, thereby resulting in a loss of high spatial frequencies, which are fundamental for the discrimination of fine details. This loss of high spatial frequencies is most likely to be involved in the typical LBL dyslexia symptomatology and it may possibly explain why letter encoding is in part disrupted in LBL reading.

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FIGURE CAPTIONS

Fig. 1. Average correct response times as a function of word length and N size control.

Fig. 2. Error rates (%) as a function of word length and N size control.

FIGURES

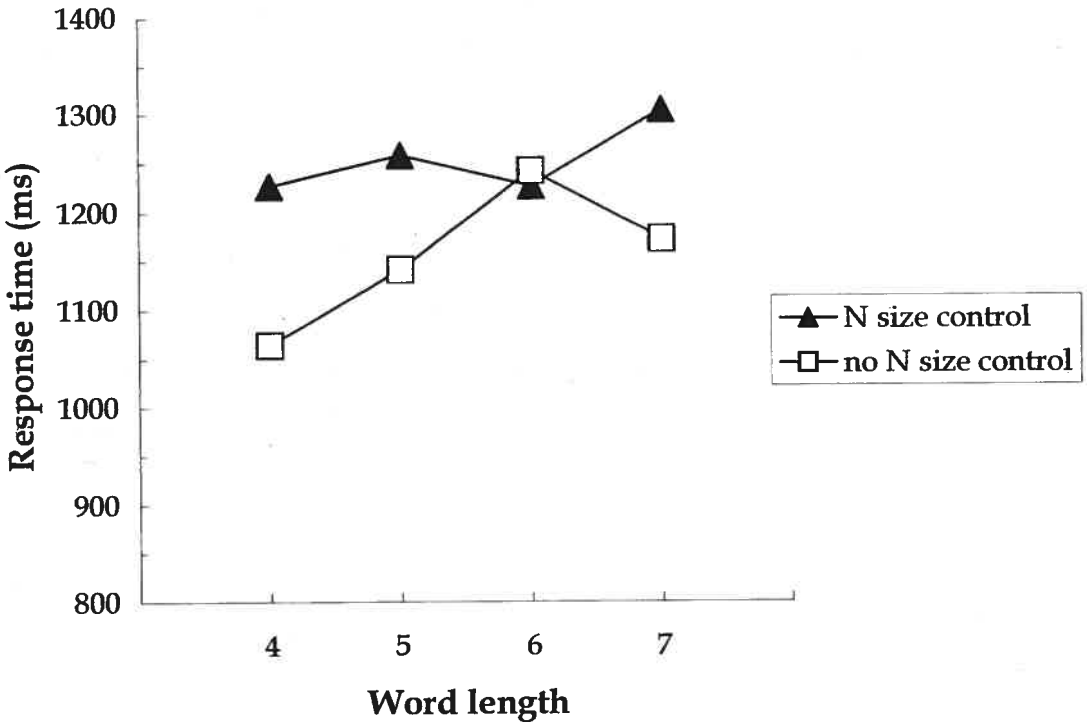

Fig. 1 — Fiset, Arguin & Fiset

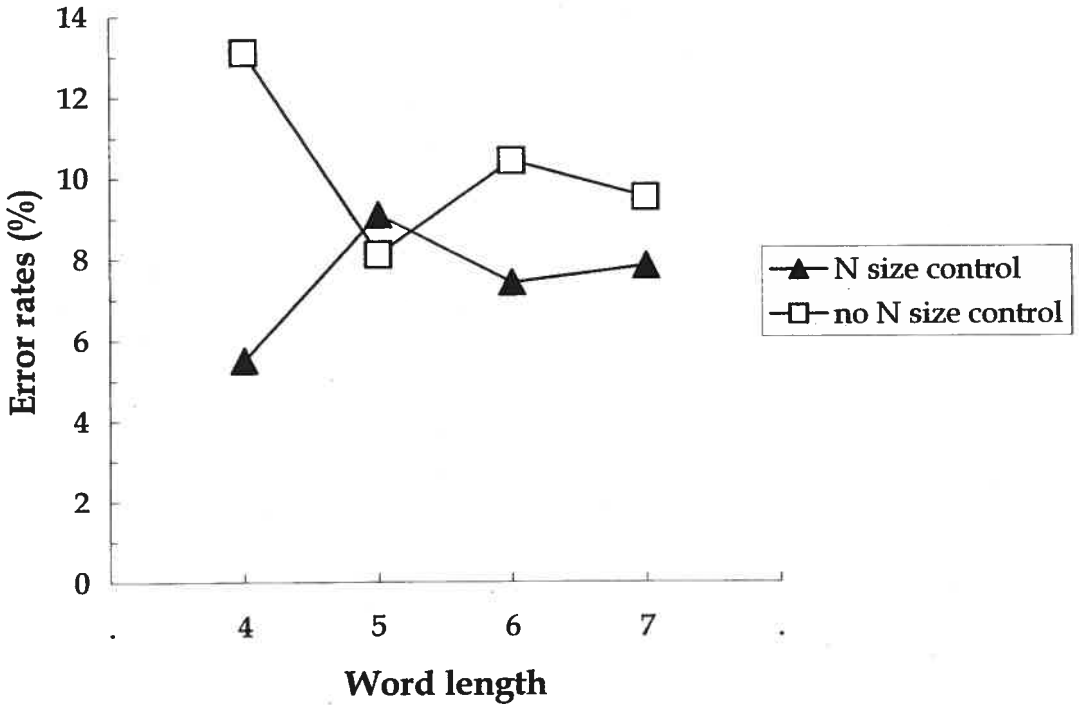

Fig. 2 — Fiset, Arguin & Fiset

CHAPITRE QUATRIÈME :

TROISIÈME ARTICLE

AN ATTEMPT TO SIMULATE LETTER-BY-LETTER DYSLEXIA IN NORMAL READERS

Stéphanie Fiset, Martin Arguin & Daniel Fiset

Groupe de Recherche en Neuropsychologie et Cognition,
Département de psychologie, Université de Montréal

and

Centre de recherche de l'Institut Universitaire de Gériatrie de Montréal

ABSTRACT

According to the peripheral hypothesis of letter-by-letter (LBL) dyslexia, the reading disorder is provoked by a functional impairment prior to the activation of the orthographic representation of words, which thus prevents their normal access. From this, it was hypothesised that a visual degradation of the stimuli reproducing the condition of parafoveal exposure or the initial processing of stimuli by the right cerebral hemisphere could induce the reading pattern characteristic of LBL dyslexia in neurologically intact readers. The present investigation assessed this hypothesis. Stimulus degradation was performed by reducing the contrast of the stimuli against the background and by removing their high spatial frequencies. In Exp. 1, we demonstrate that this visual manipulation can produce, in normal readers, the letter confusability effect characteristic of LBL dyslexia. Exp. 2 indicates that a word length effect appears under these same simulation conditions, and that this effect interacts with lexical frequency, as in LBL dyslexia. However, Exp. 3 failed to replicate the interaction between letter confusability and orthographic neighbourhood size (N size, i.e. a facilitatory N size effect for targets of low confusability only) which has been previously found in LBL dyslexics. Our results suggest that the type of visual degradation employed here may only partially correspond to the visual deficit present in LBL dyslexia and that this degradation may have prevented the normal readers to access visual information, which is available to LBL dyslexics when they focus their attention sequentially on individual letters.

Key words: Simulation in normal readers, Visual processing, Pure alexia, Letter-by-letter dyslexia, Dyslexia, Reading, Word recognition, Simulation in normal readers, Visual processing

INTRODUCTION

Letter-by-letter (LBL) dyslexia is an acquired reading disorder that is caused by a left occipito-temporal damage in previously literate adults (Black & Behrmann, 1994; Damasio & Damasio, 1983; Déjerine, 1892). It is usually associated with a right homonymous hemianopia. Apart from hemianopia, no other cognitive impairment of any kind has been systematically associated with LBL reading. The main behavioural feature of LBL dyslexia is very slow reading characterised by a large word length effect i.e. a linear increase in the time required for the overt recognition of a word as a function of the number of letter it comprises (e.g. Patterson & Kay, 1982). Depending on the patient, the time needed to read a word aloud can increase from 500 ms to several seconds for each additional letter in the stimulus (Arguin & Bub, 1993; Bowers, Bub & Arguin, 1996b; Farah & Wallace, 1991; Friedman & Lott, 2000; Patterson & Kay, 1982; Reuter-Lorenz & Brunn, 1990; Warrington & Shallice, 1980). Thus, these patients often lose the ability to read words and text quickly and efficiently. To many, the presence of a word length effect suggests that LBL readers decode words as a sequence of isolated letters, without any access to the spatially parallel process (i.e. simultaneous encoding of all the letters in a word) of normal readers. Indeed, a very weak or absent word length effect is found in neurologically intact readers (Forster & Chambers, 1973; Frederiksen & Kroll, 1976; Weekes, 1997).

A wide variety of accounts has been formulated over the years to explain LBL reading, but the functional cause of this dyslexia is still being debated. These different explanations can be grouped according to the locus of the functional damage that is hypothesised into the peripheral and the central views. In the peripheral hypothesis, the locus of damage is considered to be located prior to the activation of the orthographic representations of words. This damage may relate to a generalised poor

perceptual encoding (Farah & Wallace, 1991; Friedman & Alexander, 1984; Kinsbourne & Warrington, 1962; Levine & Calviano, 1978; Rapp & Caramazza, 1991), a deficit in abstract letter identification, or some other impairment of the encoding stage that prevents the normal activation of the orthographic representation of the stimulus (Arguin & al., 1993, Arguin & Bub, 1994; Arguin, Fiset & Bub, 2002; Behrmann, Plaut & Nelson, 1998; Behrmann & Shallice, 1995; Kay & Hanley, 1991; Montant & Behrmann, 2001; Reuter-Lorenz & Brunn, 1990). According to the alternative account of LBL dyslexia, the central hypothesis, the deficit occurs at a later stage in word reading: it can be provoked by a damaged orthographic word-form system (Warrington & Shallice, 1980), or by an impaired access to phonological word-forms following a relatively intact access to orthographic word-forms (Bowers, Arguin & Bub, 1996a).

Normal model of LBL dyslexia

One attempt to reproduce LBL reading in neurologically intact readers based on the peripheral view was carried out by Nelson, Behrmann and Plaut (personal communication). The authors hypothesised that if the peripheral view is the proper explanation of LBL reading, the visual degradation of stimuli should provoke, in normal readers, reading patterns similar to those that are characteristic of LBL dyslexia, such as a word length effect. In their experiment, they presented stimuli at three different levels of contrast reduction on a computer screen. The main result of their experiment is that a relatively substantial word length effect can be found in normal readers when the luminance contrast of the stimuli is reduced. This observation suggests that symptoms similar to those associated with LBL dyslexia can be elicited by manipulating the visual quality of the input.

Fiset, S., Arguin & Fiset , D. (in preparation) intended to replicate those results while controlling for orthographic neighbourhood size (N size: number of words differing from a target word by just one letter), a control that had not been performed by Nelson et al. The particular importance of N size control originates from the following combination of facts (see Arguin et al., 2002): a) word length shows a strong negative correlation with N size; b) an abnormally strong facilitatory effect of N size is found in LBL dyslexia; c) the word length effect in LBL dyslexia is entirely resistant to N size control. The results of Fiset et al. showed a significant word length effect in normal readers with contrast-reduced stimuli when no control of N size was exerted in order to match words of different lengths on this parameter. However, the word length effect was absent when N size was controlled, i.e. when the same number of orthographic neighbours was maintained across words of different lengths. Accordingly, Fiset et al. concluded that the word length effect previously obtained by Nelson et al. was an artefact of N size, shorter words being read faster because they have more orthographic neighbours than longer words (Arguin et al., 2002). Consequently, it appears that the sole reduction of luminance contrast is insufficient to provoke symptoms that are truly characteristic of LBL dyslexia in normal readers. Thus, even though this manipulation has a negative impact on the performance of normal readers (reading times are increased in low contrast conditions), it seems qualitatively distinct from the one responsible for LBL dyslexia. Thus, if the peripheral account outlined above is correct, then another type of degradation of the stimuli must be applied in order to replicate symptoms of LBL dyslexia in neurologically intact readers. The purpose of this article is to examine this issue.

Letter confusability

Studies conducted in our laboratory have underlined the influence of another variable which strongly affects the reading latencies of LBL patients: letter confusability (Arguin et al., 2002; unpublished studies). Letter confusability is defined as the shape similarity between a particular letter and the remaining letters of the alphabet. The confusability values are determined from empirical letter confusion matrices that were obtained in studies of neurologically intact observers (Gilmore, Hersh, Caramazza & Griffin, 1979; Loomis, 1982; Townsend, 1971; Van der Heijden, Malhas & Van den Roovaart, 1984). Specifically, the confusability value for a particular letter is the probability that a normal reader will make an error identifying it, when the target is presented very briefly and then masked. The letter confusability of a word is the average of the confusability of its constituent letters.

In normal viewing conditions, letter confusability has an inhibitory impact on the reading latencies of LBL readers: words with a high letter confusability content are read more slowly than low confusability words (Arguin et al., 2002, Arguin & Bub, under review; Fiset, D., Arguin, Fiset, S., & Blais, 2003; Fiset, D., Arguin & McCabe, 2002). By contrast, neurologically intact readers show no letter confusability effect with high-contrast stimuli presented in a regular format (Arguin et al., 2002). The fact that a letter confusability effect is only present in LBL dyslexics reveals that these patients are abnormally sensitive to the visual similarity among letters. Moreover, it has been shown previously that the facilitatory effect of increased N size in word naming, which suggests that a parallel letter processing contributes to overt word recognition in LBL dyslexia, is prevented in LBL dyslexics if the words used are made of high confusability letters (Arguin et al., 2002; Arguin & Bub, under review). This suggests that the discrimination between the target and its

orthographic neighbours, which is required to produce the facilitatory N size effect, is only possible with low letter confusability items.

A plausible explanation of the abnormal sensitivity of LBL dyslexics to visual similarity is that a visual problem accompanies the disorder, which may be prejudicial to the discrimination of visually similar letters. One hypothesis is that this sensitivity of LBL dyslexics to letter confusability may be a function of their frequent right homonymous hemianopia. Indeed, hemianopia typically interferes with the foveal encoding of visual stimuli. It is known that spatial acuity diminishes quite rapidly as we move away from the fovea, thus resulting in a loss of high spatial frequencies (Carrasco & Frieder, 1997). These are essential for the discrimination of fine details, and their loss could result in a difficulty in discriminating among visually similar letters. Thus, the homonymous hemianopia often accompanying LBL dyslexia could be directly involved in the production of LBL symptomatology.

Another possible explanation of the strong sensitivity of LBL dyslexics to visual similarity is linked to the fact that these readers process words initially through the right cerebral hemisphere. It is known that the right hemisphere is specialised in the processing of low spatial frequencies (Ivry & Robertson, 1998). Some authors have also shown that letter recognition in normal readers is more effective at a spatial frequency of 3 cycles / letter (Solomon & Pelli, 1994; Majaj, Pelli, Kurshan & Palomares, 2002). Since the visual information transits via the right hemisphere before it is transferred (through the corpus callosum) to the left hemisphere, it may thus be more difficult for LBL dyslexics to extract the mid spatial frequencies that seem necessary in normal reading.

Pilot studies conducted in our laboratory have added support for the hypothesis that a loss of high spatial frequencies may be important in determining the LBL dyslexia symptomatology. Indeed, an inhibitory letter confusability effect was found when normal readers were required to identify parafoveally presented words that had a 10% luminance contrast (dark grey printed over lighter grey, 32 cd/m^2) $F(1, 9) = 14.03$, $p < .01$. In contrast, letter confusability had no significant impact on their reading latencies when low-contrast stimuli were shown at the fovea even when stimulus contrast was reduced down to 3% $F(1, 9) = 1.03$, *ns*. These results support the idea that stimuli deprived of high spatial frequencies, in conjunction with a reduced luminance contrast can provoke, in neurologically intact readers, the letter confusability effect observed in LBL dyslexics. This manipulation of the visual appearance of the stimuli thus appeared promising for the development of a normal model of LBL dyslexia.

In the present article, we attempt to reproduce, in neurologically intact readers, the symptoms characteristic of LBL dyslexia through the visual degradation of the stimuli. In the three experiments reported here, words were presented in low luminance contrast, and high spatial frequencies were removed by low pass filtering. This manipulation was performed in replacement of the parafoveal presentation of the stimuli used in the pilot studies mentioned above for reasons of feasibility and simplicity. Also, as noted above, low pass filtering may provide a way to simulate the right hemisphere's limited ability in processing high spatial frequencies .

In Exp. 1, we validated the hypothesis that a reduced luminance contrast in combination with the removal of high spatial frequency can indeed induce a letter confusability effect in neurologically intact readers. In Exp. 2, we reproduced, in normal readers, the word length effect that is

diagnostic of LBL dyslexia. Finally, Exp. 3 was designed to verify if the visual degradation employed could provoke the interaction between letter confusability and neighbourhood size observed in LBL dyslexics by Arguin et al. (2002) and Arguin & Bub (under review), which is characterised by the fact that the facilitatory effect of N size is prevented with high letter-confusability words.

EXPERIMENT 1

Exp. 1 was designed to determine if contrast reduction combined with low pass filtering of the stimuli could indeed produce, in normal readers, the letter confusability effect which is a key feature of LBL dyslexia. Thus, in Exp. 1, two types of presentation of the stimuli were employed: some subjects had to read aloud words of high or low letter confusability presented normally, whereas others had to identify words that were visually degraded. In this latter condition, the stimuli were presented in low luminance contrast and were low pass filtered to remove their high spatial frequency content. Based on the pilot study described above, we stipulate that this manipulation will provoke an increase in reading latencies for high confusability items compared to low letter confusability stimuli, whereas no confusability effect should be obtained with undegraded stimuli (i.e. normal format).

Method

Subjects:

Twenty-four normal readers took part in the experiment. They were aged between 19 and 34 years (mean = 23.3 years old). All were right-handed and had a normal or corrected vision and none had a history of learning disabilities. Twenty-two out of twenty-four were university students (Bachelor or Master's degrees). The others had 18 years of formal education (Master's degrees).

Materials and stimuli:

The stimuli were printed in Geneva 24 point bold font. They were either presented normally (black letters on a gray background) or either presented in reduced contrast (luminance of 4.51 cd/m^2 against a background of a luminance of 5.09 cd/m^2) and were low pass filtered (a 2.4 pixels gaussian blur) using the Adobe Photoshop program. For the normal format (i.e. high contrast) displays, stimuli were shown using a Dell Dimension computer connected to a 17 inches monitor. The E-Prime software (produced by Psychology Software Tools), was used to control the presentation of the stimuli. For the degraded stimulus condition, stimuli were shown on a Viewsonic E-771 screen, linked to a Power PC 7100/80. In both conditions, subjects were seated 55 cm away from the computer screen. The visual angle sustended by each letter was approximately 0.73° wide, and 0.88° high.

A total of 120 five-letter words were used, with an equal number of words having a low or a high letter confusability, which is calculated as the sum of the confusability of all letters in a word divided by the number of letters (low: average confusability of 0.450 or below; high: average

confusability of 0.495 or higher). Words of different letter confusabilities were matched according to lexical frequency (we used the log frequency) given in the French BRULEX data base (Content, Mousty & Radeau, 1990), their number of orthographic neighbours, and their bigram frequency (data obtained by a software designed in our laboratory). None of these variables differed significantly, all *F*'s *(1,118) < 1.* None of the words contained diacritic marks (é, è, ê, etc.), because subjects could not see them clearly when the luminance contrast was reduced. In French, diacritic marks dictate word pronunciation and keeping words with accents in the present context could have led to an inflation of error rates.

Procedure:

The subjects were separated into two groups: for half the subjects, the stimuli were presented normally (this group will be referred to as the "normal presentation" group). For the other half, the stimuli were visually degraded (the "degraded presentation" group). Each group of subjects saw the same list of words. Subjects in the "degraded presentation" group were given 10 practice trials.

Words were presented in two blocks of 60 items each, each block comprising an equal number of low and high confusability words. The order of presentation of each block was counterbalanced across subjects. Each trial began with a 1000 ms fixation point that was displayed at the centre of the computer screen. This was immediately followed by the target printed in uppercase (because letter confusability matrices are exclusively based on uppercase letters) displayed at the centre of the screen. The task was to name the target as quickly as possible while avoiding errors. Responses were registered by a voice-key (engineered in our laboratory) connected to the computer controlling the experiment.

After each response, the experimenter entered the answer given by the subject via the computer keyboard and then triggered the next trial.

Results

The data analyses were performed on twenty-one subjects only, because one subject (in the "degraded presentation" group) showed a dramatically high response latency (his mean RT was of 4750 ms, which is 2.85 SD away from the mean of the other subjects in his group– 2270 ms). For the "normal presentation" group, 27 (1.88%) trials were lost overall due to the failure of subjects responses to trigger the voice-key, whereas, for the "degraded presentation" group, a total of 19 (1.44%) trials were lost for this reason. These trials were not considered in the data analyses. Average correct RTs and error rates are shown in Figs. 1 and 2, respectively.

Insert Figs. 1 and 2 near here.

RTs analysis:

For the "normal presentation" and "degraded presentation" groups respectively, twenty-seven data points (1.91 % of all trials) and forty data points (3.07% of all trials) were removed on an individual basis from the RT analysis because the response latency was more than 2.5 SDs away from the mean of their condition.

An ANOVA conducted on RTs, with presentation type (normal vs. degraded) and letter confusability (low vs. high) as factors showed a main effect of presentation type $F(1, 21) = 188.87$, $p < .001$ and of letter confusability $F(1, 21) = 13.93$, $p < .01$, with shorter RTs for stimuli presented normally (vs. visually degraded), and for low confusability words compared to high confusability items. Moreover, these factors interacted significantly $F(1, 21) = 13.96$, $p < .01$.

Simple effects analysis were performed to determine the confusability effect for each presentation type. For the "normal presentation" group, no confusability effect was observed $t(11)=0.07$, *n.s.*, whereas for the "degraded presentation" group, a large confusability effect was present $t(10)= 3.57$, $p < .01$, with shorter RTs for low compared to high confusability words.

Error rates analysis:

An ANOVA conducted on error rates, with presentation type and letter confusability as factors showed a main effect of presentation type $F(1, 21) = 49.99$, $p < .001$ and of letter confusability $F(1, 21) = 6.97$, $p < .05$, with less errors for stimuli presented normally (vs. visually degraded), and for low confusability words compared to high confusability items. Moreover, these factors interacted significantly $F(1, 21) = 14.16$, $p < .01$.

Simple effects analysis indicated that error rates were not affected by letter confusability when stimuli were presented normally $t(11)=1.32$, *n.s.*, contrarily to when they were visually degraded $t(10)= 3.57$, $p < .01$, where error rates were significantly lower for words having a low confusability.

Discussion

The present results clearly indicate that the overt word naming performance of normal readers is negatively affected by letter confusability with visually degraded stimuli, but not when stimuli are presented normally. Indeed, when words were visually degraded, high confusability words were read 375 ms slower than low confusability words, and subjects made significantly more errors in reading high confusability stimuli. These results are in great contrast with the performance of neurologically intact readers under normal reading conditions, where no letter confusability effect is observed on either RTs or error rates.

The present results with degraded stimuli are congruent with those obtained in LBL dyslexics, who show a substantial letter confusability effect with unaltered [i.e. normal format stimuli (Arguin et al., 2002; Arguin & Bub, under review; Fiset, D. et al., 2003; Fiset, D. et al., 2002)].

It may be noted that the error rate with visually degraded stimuli is relatively high (about 27% and 18% in the high and low letter confusability conditions, respectively). These error rates are somewhat higher than what is usually found in the LBL dyslexics studied in our laboratory. However, pilot studies have indicated that if the luminance contrast is slightly increased or if the low pass filtering is less severe, normal readers do not exhibit any particular difficulty in identifying the words and their reading latencies are practically unaffected by stimulus degradation.

The present experiment confirms that the visual degradation employed (the combination of luminance contrast reduction and the filtering out of high spatial frequencies) is capable of producing, in normal readers, the letter confusability effect that is otherwise found only in LBL

dyslexics. These stimulus manipulations thus seem promising with respect to our attempt of producing a normal model of LBL dyslexia. In Exps. 2 and 3, we attempt to reproduce other effects that appear typical of LBL dyslexia, namely the word length effect, and the interactive effects of N size and letter confusability.

EXPERIMENT 2

The main diagnostic feature of LBL reading is the word length effect. The magnitude of this effect varies between 500 ms to several seconds for each additional letter in the stimulus, depending on the patient. This contrasts with the performance of neurologically intact readers, where a very weak or absent word length effect is usually found (Forster & Chambers, 1973; Frederiksen et al., 1976; Weekes, 1997). The absence of a word length effect is an indication that normal readers recognise words through a spatially parallel (i.e. simultaneous) processing of all the letters in the stimulus.

It has been argued previously (Arguin et al., 2002) that a likely cause of LBL dyslexia is a problem affecting the discrimination among visually similar letters when attention is spread across the whole word. This deficit impedes the identification of the stimuli through a spatially parallel mode of letter processing. It would be for this reason that LBL readers have to employ a compensatory serial strategy to identify the words. It would be by focusing their attention at the letter level, directly involved in the compensatory serial letter processing characterising this disorder, that they would be able to effectively discriminate the constituent letters of the words. By this theory, LBL reading should occur in normal readers if stimulus conditions causing a large letter confusability effect can be found.

Experiment 1 has shown that a letter confusability effect can be produced in normal readers when stimuli are shown in reduced contrast and when their high spatial frequencies are removed. It is therefore predicted that a word length effect should also be evident under these viewing conditions because subjects will need to focus their attention at each letter individually (or on a small group of letters) to identify the target words. In the next experiment, we will test this hypothesis by presenting to normal subjects, words of varying lengths displayed in the same degraded conditions as in Exp. 1.

Exp. 2 will also assess the influence of another variable, lexical frequency, as well as its interaction with word length. Lexical frequency is known to influence overt word naming performance in normal readers, high frequency words being read faster than low frequency words. It has been shown that this variable interacts with word length in its effect on reading latencies. Indeed, Weekes (1997) has reported that lexical frequency interacted with length in the word naming task in normal readers with normal exposure conditions i.e. whereas no length effect was apparent on the naming latency of high frequency words, there was a small effect for low frequency words and a greater one for non-words. The results of pilot studies conducted in our laboratory as well as those from the study of Nelson et al. (personal communication) cited above, are congruent with this observation. Most importantly, LBL dyslexics have also demonstrated interactive effects of frequency and length, with a greater length effect on naming latencies for low than high frequency words (see Behrmann et al., 1998, for a complete review). The interaction between word length and lexical frequency has been interpreted by Behrmann et al. within the context of their unitary interactive account of LBL dyslexia: since LBL dyslexics are thought to present only a deficit in prelexical processing in an otherwise normal reading system, the interaction between word length and lexical frequency reflects the impact

of intact top-down processes. Thus, because long words take longer to process, there is additional time, relative to short words, for these top-down effects to interactively affect reading performances. However, some studies have shown that the lexical frequency effect does not always vary as a function of word length in LBL dyslexics (Bowers et al., 1996a; Arguin, Bub & Bowers, 1998).

Finally, pilot studies conducted in our laboratory have shown that it may be advantageous to add pseudowords to the list of items to avoid the application of guessing strategies. Indeed, in some pilot experiments using degraded stimuli, participants showed very high error rates when trying to identify five- to seven-letter words. It appeared that one cause for these high error rates was that subjects frequently attempted to guess the word instead of actually identifying it. Pseudowords were therefore included in the stimulus lists used in Exps. 2 and 3 in order to force subjects to properly identify the stimuli displayed. The instructions given to the participants also emphasised the importance of correctly identifying the stimuli.

Method

Subjects:

Sixteen normal readers, aged between 19 and 34 (mean = 22,81 years old) took part in this experiment. They were selected according to the same criteria as in Exp. 1. Eleven of the participants were university students. The others had 16 years of education or more. None had participated in Exp. 1.

Materials and stimuli:

Four hundred and eighty stimuli, including 240 words and 240 pseudowords, were used in this experiment. There was an equal number of five-, six- and seven-letter words and pseudowords.

Words: Half the words had a high lexical frequency (BRULEX database: over 2145 per 100 millions) and half had a low lexical frequency (under 154 per 100 millions). Words of different lengths and lexical frequencies were matched according to their letter confusability, orthographic neighbourhood size, and bigram frequency. Conditions did not differ significantly on these variables, F's (2, 234) and (1, 234) < 1.

Pseudowords: Pseudowords were created by changing one or two letters in a word. The same matching procedure across lengths was applied for pseudowords. Pseudoword conditions did not differ significantly on letter confusability, neighbourhood size , and bigram frequency, all F's (2, 237) < 1.

Words vs. pseudowords: Words and pseudowords differed on their letter confusability $F(1, 474) = 5.01$, $p < .05$, pseudowords having a slightly higher average letter confusability than words (mean letter confusability was 0.469 and 0.461, respectively). In addition, pseudowords had a higher bigram frequency than words $F(1, 474) = 10.58$, $p < .05$, (2.74 and 2.68 for pseudowords and words, respectively). These differences were very weak however, and they were considered not to be problematic since pseudowords were used mainly in order to force subjects to actually read the words rather than to attempt to guess them. Moreover, since bigram frequency is known not to impact overt word naming performances (Andrews, 1992), it appeared unlikely that this variable would affect the results in the present experiment.

The complete list of stimuli (words and pseudowords) was divided in 6 blocks of 80 items, each comprising an equal number of words and pseudowords, of high and low frequency words, and of five-, six- and seven-letter stimuli. Stimuli were randomised within each list and their order of presentation varied across subjects. The order of presentation of the blocks was counterbalanced across subjects. The exposure conditions of the stimuli were the same as in Exp.1.

Procedure: The course of each trial was the same as in Exp. 1. Subjects were given 10 practice trials before the task.

Results

The data analyses were performed on only 15 subjects: one of the subject was eliminated because he showed a dramatically high error rate (48.3%) compared to the other participants (average of 34.4%). A total of 112 (1.56%) trials overall were lost due to the failure of the subject's responses to trigger the voice-key. These trials were not considered in the data analyses. Average correct RTs and error rates are shown in Figs. 3 and 4 respectively. The correlation between RTs and error rates was of +.92, which indicated no speed-accuracy trade-off.

Insert Figs. 3 and 4 near here.

RTs analysis:

One hundred and sixty five data points (2.18% of all trials) were removed on an individual basis from the RTs analysis because the

response latency was more than 2.5 SDs away from the mean of their condition. A two-way ANOVA performed on correct RTs, with length (five-, six- and seven-letter words) and stimulus class (three levels: high frequency words, low frequency words and pseudowords) as factors showed a main effect of length $F(2,28) = 18.27$, $p < .001$, shorter words being read faster than longer words, and of stimulus class $F(2,28) = 27.94$, $p < .001$, with shorter RTs for high frequency words than for low frequency words, which in turn were read faster than pseudowords. The results also indicated a significant interaction between length and stimulus class $F(4,56) = 10.42$, $p < .001$.

Simple effects analysis were performed on stimuli to determine the length effect for each stimulus class. The length effect turned out to be significant for each type of stimulus: high frequency words $F(2,28) = 5.71$, $p < .01$; low frequency words $F(2,28) = 9.38$, $p < .001$; pseudowords $F(2,28) = 17.04$, $p < .001$. However, pairwise contrasts on the effect of word length across stimulus classes revealed significant differences. Thus, a two-way ANOVA, with length and lexical frequency as factors, revealed a marginally significant interaction of length x frequency $F(2,28) = 3.30$, $p = .052$, indicating that the length effect was greater for low frequency words (slope = 186.3/letter, $r^2 = 0.99$ for low frequency words; slope = 68.5 ms/letter, $r^2 = 0.71$ for high frequency words). An additional ANOVA was performed to contrast the length effect across low frequency words and pseudowords. This analysis indicated a highly significant interaction of length x stimulus $F(1, 28) = 10.59$, $p < .001$: the length effect was substantially larger for pseudowords than for low frequency words (slope = 456.1, $r^2 = 0.99$ for pseudowords).

Error rates analysis:

A two-way ANOVA conducted on error rates, showed no length

effect $F(2,28) = .16$, *n.s.*, but the main effect of stimulus class was significant $F(2,28) = 218.86$, $p < .001$. The length x class interaction was also significant $F(4,56) = 9.01$, $p < .001$. Simple effects analyses showed a significant $F(2,28) = 5.07$, $p < .05$, but reversed word length effect for high frequency words (see Fig. 4). For low frequency words, the word length effect was also significant $F(2,28) = 3.59$, $p < .05$, but error rates were higher for six-letter words than for five- and seven-letter words (see Fig. 4). Finally, pseudowords showed a significant length effect $F(2,28) = 9.39$, $p < .001$, which had a regular form, i.e. monotonically increasing error rates with increasing length.

Pairwise contrasts of the length effect across low and high frequency words revealed a significant difference $F(2,28) = 5.02$, $p < .05$ which is attributed to the differing patterns of length effects described above. A similar outcome was observed in contrasting low frequency words and pseudowords on the length effect, $F(2,28) = 17.85$, $p < .001$.

Discussion

The main result of Exp. 2 is that visually degrading the stimuli (by presenting them in low luminance contrast and removing their high spatial frequencies) triggers, in neurologically intact readers, the word length effect characteristic of LBL dyslexia. Indeed, the analysis performed on correct RTs showed a main effect of stimulus length: items with fewer letters were read faster than those with many. Moreover, the error rates for pseudowords were significantly lower for short items than for long ones. The average length effect obtained on reading latencies for words is of 127.4 ms for each additional letter, which is considerably greater than that found in neurologically intact readers under normal reading conditions (Forster et Chambers, 1973; Frederiksen et al., 1976; Weekes, 1997), which ranges from 6 to 63 ms for each additional letter.

The results of Exp. 2 also revealed the presence of a lexical class effect: in general, overt word naming was facilitated (in terms of both response latencies and error rates) for high frequency compared to low frequency words, and for low frequency words compared to pseudowords. As reported earlier, these results are congruent with what is usually found in normal readers as well as in LBL dyslexics. One can argue that the frequency effect obtained here is statistically weak ($p = .052$). But this may be related to the use of pseudowords in our experiment: Indeed, Peereman & Content (1995) have found that the frequency effect can be reduced significantly when words and pseudowords are presented to normal readers in the same blocks of trials, as was done here.

In addition, the RTs analysis also indicated an interaction between stimulus length and lexical class: the length effect was more pronounced for pseudowords than for low frequency words, and for low frequency words than for high frequency words. This interaction between lexical frequency and word length has been reported previously by Behrmann et al., 1998, in five LBL readers. Nelson et al. (personal communication) also found such an interaction between lexical frequency and word length when stimuli were presented to neurologically intact readers under low luminance contrast conditions. As mentioned earlier, these findings are interpreted by Behrmann and colleagues as reflecting the impact of intact top-down processes on the reading system of LBL dyslexics, which is generally considered to be normal, apart from a deficit in prelexical processing.

In Exps. 1 and 2, we have shown that visually degrading words (by removing their high spatial frequencies and reducing their contrast) produces in normal readers some of the effects characteristic of LBL dyslexia, i.e. the word length and the letter confusability effects. The

presence of these effects in neurologically intact readers under such viewing conditions constitutes a strong support for the hypothesis of a low level deficit in LBL dyslexia, since these effects are not usually found in normal subjects under normal viewing conditions.

Exp. 3 will provide another test of the hypothesis that a visual deficit is involved in LBL dyslexia by studying the interaction of letter confusability and N size in normal readers under the same stimulus degradation as that used in the first two experiments. Indeed, it has been shown that those two variables interact systematically in LBL readers (Arguin et al., 2002; Arguin et al., in preparation), whereas they do not in neurologically intact readers under normal viewing conditions (Arguin et al., 2002).

EXPERIMENT 3

Words with many orthographic neighbours are read faster than words with a small neighbourhood size. This is true both in normal readers (Andrews, 1989, 1992; Arguin et al., 1998; Carreiras, Perea & Grainger, 1997, Sears, Hino & Lupker, 1995) and in LBL dyslexics (Arguin & Bub, 1996; Arguin et al., 1998; Arguin et al., 2002; Fiset, S. & Arguin, 1998; Arguin & Bub, under review; Montant & Behrmann, 2001). This facilitatory effect of increased N size is considered to index a process involved in orthographic encoding (Andrews, 1989; 1992; Arguin et al., 1998; Carreiras et al., 1997; Coltheart, Davelaar, Jonasson & Besner, 1977; Pugh, Rexer, Peter & Katz, 1994; Sears et al., 1995). In particular, it appears that visual words activate not only their own internal orthographic-lexical representations, but also those of their orthographic neighbours. In turn, this lexical activation is thought to provide a

facilitatory feedback to an earlier level of processing responsible for the identification of the letters constituting the stimulus (McClelland & Rumelhart, 1981; Plaut, Seidenberg, McClelland & Patterson, 1996). Therefore, when a word possesses many orthographic neighbours, the letter representations compatible with the target receive more facilitatory feedback from the activated lexical representations than when a word has few neighbours. This facilitatory feedback on the letter identification process is assumed to be responsible for the facilitatory effect of N size on word reading.

Since the cause for the facilitatory N size effect involves the letter identification stage of processing, we can hypothesise that letter confusability may influence the occurrence of the N size effect. Indeed, if high letter confusability amplifies noise in the activation of lexical-orthographic representations, it may also negatively affect the reading system's capacity to discriminate between the target and its orthographic neighbours by blurring the contrast between the different representations activated. This in turn should prevent or reduce the facilitatory effect of increased N size (see Arguin et al., 2002 for a more detailed explanation).

Arguin et al., 2002, have already tested the modulation of N size by letter confusability in letter-by-letter dyslexic IH and in neurologically intact readers (university students and age-matched subjects to IH) under normal viewing conditions. The word naming performance of normal readers was resistant to the effect of letter confusability i.e. the facilitatory effect of N size was the same for low and high confusability words. By contrast, the results obtained in LBL dyslexic IH showed that the facilitatory effect of increased N size occurred with low confusability words but was prevented with high letter-confusability targets. This suggests that the discrimination between the target and its orthographic neighbours, which is required to produce the facilitatory N size effect, is

only possible with low letter confusability items. More recently, these findings have been replicated in three other cases of LBL dyslexia (Arguin & Bub, under review).

Exp. 3 will further address this issue by examining whether N size and letter confusability interact in normal readers with words that have been visually degraded as in Exps. 1 and 2. Since an important letter confusability effect was found under such conditions in Exp. 1, we expect an interaction between N size and letter confusability similar to the one previously observed in LBL dyslexics.

In Exp. 3, neurologically intact readers were presented visually degraded (low contrast with high spatial frequencies removed) words and pseudowords of varying confusability and N size. As in Exp. 2, pseudowords were used to force the subjects to identify all the letters constituting the stimuli instead of attempting a guessing strategy. It is predicted that subjects will show a letter confusability effect which will interact with N size; specifically, the facilitatory N size effect should be present with low confusability items but it should be altered or eliminated with high confusability targets.

Methods

Subjects:

Fifteen normal readers took part in this experiment. They were aged between 19 and 27 years old (mean = 22.6 years). All of them, except two, were university students. The others had 16 and 17 years of formal education. The inclusion criteria were the same as in Exps. 1 and 2. None had participated in Exps. 1 and 2.

Stimuli:

Words: The targets were 168 words varying orthogonally on their letter confusability (low confusability: below 0.45; high confusability: 0.52 or higher) and their number of orthographic neighbours (low N size: 0 neighbour; high N size: 4-8 neighbours). Also, we used stimuli of different lengths (four-, five-, six- and seven-letter items), since not enough words of the same length were available in French to construct the task while controlling for other relevant lexical variables. Words were matched across conditions according to their length, lexical frequency (Content et al., 1990), and bigram frequency. Conditions did not differ significantly on these variables $F(3, 164) < 1$, except for bigram frequency $F(3, 164) = 14.04$, $p < .001$, which was higher for high N size words. Bigram frequency shows a large positive correlation with neighbourhood size and thus a perfect match on this variable across low and high neighbourhood size is extremely difficult to achieve. As indicated previously, bigram frequency on its own does not significantly affect word reading performance (Andrews, 1992).

Pseudowords: Pseudowords were created by changing one or two letters in a word. There were 168 pseudowords of four to seven letters in length, which varied orthogonally on their number of orthographic neighbours (low N size: 0 neighbour; high N size: 3-9 neighbours) and on their letter confusability (low: 0.47 and lower; high: confusability of 0.48 or higher).

Across corresponding conditions, words and pseudowords were matched on word length $F(1, 328) < 1$, letter confusability $F(1, 328) < 1$, N size $F(1, 328) < 1$, and bigram frequency $F(1, 328) < 1$.

The stimuli were divided into four blocks comprising 84 stimuli each: half of them were words, half were pseudowords. Each block

contained equal numbers of words and pseudowords of low or high letter confusability, and of low or high N size. The order of presentation of the blocks was counterbalanced across subjects.

Procedure: The course of trials was the same as in Exps. 1 and 2.

Results

A total of 73 (1.45%) trials were lost because responses failed to trigger the voice-key. These trials were not considered in the data analysis. Average correct RTs and error rates are shown in Figs. 5 and 6 respectively. The correlation between RTs and error rates was of +.86, which indicates the absence of a speed-accuracy trade-off.

Insert Figs. 5 and 6 near here.

RTs analysis:

Ninety-seven data points (1.95% of all trials) were removed on an individual basis from the RTs analysis because the response latency was more than 2.5 SDs away from the mean of their condition. A three-way ANOVA performed on correct RTs, with lexicality (words vs. pseudowords), letter confusability (low vs. high) and neighbourhood size (low vs. high) as factors showed main effects of lexicality $F(1,14) = 25.51$, $p < .001$, of letter confusability $F(1,14) = 18.29$, $p < .001$, and of neighbourhood size $F(1,14) = 21.35$, $p < .001$, with shorter RTs for words, for low confusability stimuli, and for targets having a large

neighbourhood size. The results also indicated that the interaction between lexicality and neighbourhood size was significant $F(1,14) = 21.42$, $p < .001$ (with a larger N size effect for pseudowords), as well as the interaction between lexicality, letter confusability and neighbourhood size $F(1,14) = 4.76$, $p < .05$. The overall experimental design was broken down to allow the analysis of the interaction of letter confusability with N size for words and pseudowords separately.

Words: A two-way ANOVA performed on words only, with letter confusability and neighbourhood size as factors, revealed a main effect of letter confusability $F(1,14) = 17.22$, $p < .001$, and of N size $F(1,14) = 5.82$, $p < .05$, with shorter RTs for low confusability targets and for words having a high neighbourhood size. The interaction term was marginally significant $F(1,14) = 3.66$, $p = .08$, and tended to show a facilitatory effect of N size with high confusability words $F(1,14) = 7.86$, $p < .05$, but no effect with low confusability words $F(1,14) = 1.55$, *n.s.*

Pseudowords: For pseudowords, the analysis revealed main effects of letter confusability $F(1,14) = 9.38$, $p < .01$, and of N size $F(1,14) = 24.40$, $p < .001$, with faster RTs for low confusability items and for targets having a large neighbourhood size. These variables did not interact significantly $F(1,14) = 2.75$, *n.s.*

Error rates analysis:

A three-way ANOVA conducted on error rates, showed main effects of lexicality $F(1, 14) = 95.63$, $p < .001$ and letter confusability $F(1, 14) = 71.89$, $p < .001$, but no N size effect $F(1, 14) = .04$, *n.s.* The triple interaction term was not significant $F(1, 14) = .15$, *n.s.*, but lexicality and letter confusability interacted significantly $F(1, 14) = 10.34$, $p < .01$, as well

as lexicality and N size $F(1, 14) = 8.76$, $p = .01$. Also, the interaction of confusability by N size was marginally significant $F(1, 14) = 4.34$, $p = .056$.

Words: An ANOVA conducted on error rates for words only indicated simple effects of letter confusability $F(1, 14) = 92.38$, $p < .001$ and N size $F(1, 14) = 5.57$, $p < .05$, as well as an interaction between these two variables $F(1, 14) = 5.80$, $p < .05$. A significant N size effect was found with high (decreased error rates with low N size words) , but not with low confusability words.

Pseudowords: For pseudowords, a letter confusability effect was present: error rates were significantly higher with high confusability pseudowords than with low confusability pseudowords $F(1, 14) = 25.13$, $p < .001$. Error rates did not differ according to N size $F(1, 14) = 2.71$, *n.s.*, and letter confusability and N size did not interact $F(1, 14) = 1.56$, *n.s.*

Discussion

The results of Exp. 3 revealed the presence of a letter confusability effect, with shorter RTs and reduced error rates for low confusability items compared to high confusability targets. This replicates the results of normal readers in Exp. 1, and it is congruent with the performance of LBL dyslexics (Arguin et al., 2002; Arguin & Bub, under review). Moreover, the RTs analysis showed that increased N size had a facilitatory effect on word and pseudoword naming: targets with a large neighbourhood size were read faster than those with few neighbours. This latter finding replicates the performance of normal readers with normally printed stimuli (Andrews, 1989, 1992; Arguin et al., 1998; Carreiras et al. 1997, Sears et al., 1995) as well as that of LBL dyslexics (Arguin et al., 1996; Arguin et al., 1998; Arguin et al. 2002; Arguin & Bub, under review; Montant & Shallice, 2001).

In contrast to our predictions however, the RTs did not show the expected interaction of letter confusability and N size, i.e. a reduction or elimination of the N size effect with high confusability items. Indeed, letter confusability failed to modulate the N size effect for pseudowords, whereas the interaction trend was in a direction opposite to that predicted for words. These observations depart from those obtained in LBL readers, who showed a facilitatory N size effect only with words of low letter confusability.

Partial analysis:

A detailed examination of individual subject means revealed an important variation in the response times among subjects, some of them being considerably slower than others (range of overall RTs means across subjects: 1609 ms to 5680 ms, words and pseudowords combined). It is interesting to note that there is a relatively substantial correlation of +.56 between mean RTs for words and the degree by which the advantage for high N size items (relative to low N size) is reduced with high (relative to low) letter confusability items (Fig. 7). This reduction generally tends to be substantial (up to 837 ms) in the slower subjects but null or even markedly reversed for the faster subjects (i.e. amplified advantage of high N size words with high confusability). For pseudowords, the correlation between RTs and the reduction of the N size effect is not as strong as for words (+.38) (Fig. 8). However, the interaction between N size and letter confusability for pseudowords has never been directly tested in LBL dyslexics and we may only speculate as to whether or not it would be in the same direction as for words. Given this, data from pseudowords does not appear as determinant as that obtained with words.

Insert Figs. 7 and 8 near here.

Congruently with the correlation reported above, if the subjects are divided into two groups according to their mean RTs for words (more vs. less than 2200 ms), we observe that the slowest readers tend to show a similar interaction to that documented in LBL dyslexia (N size effect for low and for high letter confusability words of 354 ms and 73 ms, respectively), whereas the faster subjects tend to present an interaction in the opposite direction i.e. an increased facilitatory N size effect for high letter confusability words (N size effect for low and for high letter confusability words of 25 ms and 324 ms, respectively). It thus appears that the degree to which the subjects are affected by the visual degradation may profoundly transform the way N size and letter confusability jointly affect reading performances. This observation will be addressed further in the general discussion.

GENERAL DISCUSSION

The research reported in the present paper has focused on an attempt to simulate LBL dyslexia in normal readers by visually degrading stimuli. According to the peripheral hypothesis of LBL dyslexia, this reading disorder is provoked by a functional impairment prior to the activation of the orthographic representation of words, thereby preventing normal lexical access. It was thus hypothesised that visually degrading the stimuli by reducing their contrast against the background and by removing their high spatial frequencies could induce the reading pattern characteristic of LBL dyslexia in neurologically intact readers. Indeed, this

latter stimulus alteration is thought to replicate the impact of the right hemianopia that usually accompanies LBL dyslexia: either the loss of high spatial frequencies associated with parafoveal encoding of the stimulus, or the poor processing of high spatial frequencies that appears to characterise the right hemisphere.

Exp. 1 demonstrated that contrast-reduced low passed stimuli provoke in normal readers the letter confusability effect that has been previously observed in LBL dyslexics. These results appeared very promising with respect to our attempt of producing a normal model of LBL dyslexia, given the assumption that the word length effect reflects a difficulty in discriminating among visually similar letters. Exp. 2 supported this assumption, by showing that the stimulus degradation used causes, in normal readers a substantial word length effect, which is the hallmark of LBL dyslexia. Another interesting aspect of the results of Exp. 2 was the production of a lexical frequency effect, which is congruent with that usually found in LBL dyslexics. Moreover, the effects of lexical frequency and word length interacted, i.e. the length effect was more pronounced for low frequency than for high frequency words. An interaction of the same form has been previously reported in five LBL dyslexics (Behrmann et al., 1998). The production of well-controlled effects of word length and of letter confusability in normal readers under degraded stimulus conditions is a novel finding that closely approximates crucial features of the reading performance of LBL dyslexics. These results therefore add weight to the hypothesis of a deficit in LBL dyslexia that occurs early in word processing, prior to the activation of lexical orthographic representations.

Exp. 3 was designed to replicate the interaction between letter confusability and neighbourhood size that was observed by Arguin et al. (2002) and Arguin & Bub (under review) in LBL dyslexics, i.e. a facilitatory

N size effect with low confusability items and the elimination or reduction of this effect with high confusability items. The mean results of the subject group tested in Exp. 3 failed to show the expected pattern: although significant effects of letter confusability and N size were observed, these variables did not interact significantly. A detailed examination of individual data however, revealed major individual differences in reading ability with our degraded stimuli, which seems to have affected the way in which N size and letter confusability jointly affected performances. Thus, we found that the slowest readers tend to show the same interaction pattern between N size and letter confusability (i.e. a facilitatory N size effect for low confusability words only) as LBL readers, while the faster readers tend to present an interaction between those two variables in the opposite direction. An account of this observation will be elaborated hereafter.

Does low pass filtering reproduce the visual disorder of LBL dyslexics?

It may be noted that the word length effect obtained in Exp. 2 was about 200 ms for each additional letter in the stimulus. This word length effect is substantially weaker than what is found in the vast majority of LBL dyslexics. One possible explanation for this result could be that the task was not difficult enough to force normal readers to resort to a strict letter-by-letter strategy such as the one employed by LBL dyslexics. However, this account is incompatible with the fact that the error rates obtained by our subjects were rather higher (ranging from 15% to 38% for words depending on lexical frequency and N size) than the ones usually observed in LBL readers. Thus, the relatively weak length effect observed here seems not to be a function of low task difficulty.

Another explanation for the small amplitude of the word length effect in normal readers may be directly linked to the type of visual degradation employed in the present experiments. An important aspect of the stimulus degradation used here, low pass filtering the stimuli, attempted to replicate one cause of difficulty for LBL dyslexics, i.e. the loss of high spatial frequencies due to parafoveal viewing or to the initial processing of stimuli by the right hemisphere. However, this type of degradation may differ in significant ways from the visual disorder affecting word recognition in LBL dyslexia.

Indeed, LBL dyslexics appear to compensate their difficulties in visual word processing by resorting to sequential letter processing, involving foveation and the narrowing down of attention at the level of individual letters instead of attempting to encompass the whole word (see Arguin et al., 2002 for more detailed explanation). It has been shown that attention can improve visual performance by enhancing spatial acuity (Yeshurun & Carrasco, 1998). Thus, the focusing of attention sequentially on individual letters may enhance the ability of LBL dyslexics to extract midrange spatial frequencies, which appear more optimal for letter identification and less susceptible to visual confusions than low spatial frequencies (Solomon & Pelli, 1994).

With the type of stimulus degradation used here however, normal readers could not benefit from such compensation: They could not use focused attention or sequential foveation on individual letters to improve the encoding of mid-to-high spatial frequencies since these frequencies were absent from the stimuli. Thus, the incentive of using a strictly serial strategy was weaker than in LBL dyslexia. This incapacity of subjects to fully compensate for stimulus degradation may thus be responsible for the weaker slopes and higher error rate observed her than those typical of LBL dyslexics.

These factors may also explain why, in Exp. 3, the interaction between N size and letter confusability tended to be in the opposite direction of what is usually found in LBL readers. It has been argued elsewhere (Arguin et al., 1998; 2002; Arguin & Bub, under review) that the facilitatory N size effect observed in LBL dyslexics originates from a parallel analysis of the letters within the word. These studies have shown that this parallel letter processing is prevented with high letter confusability. Indeed, high confusability impairs the letter identification process because of the presence of an abnormal degree of noise between the constituent letters of the stimuli and the other letters of the alphabet. The parallel analysis of letters thus fails to reliably support overt word recognition in LBL dyslexics, who must then rely on a sequential letter encoding to identify words. The facilitatory effect of N size therefore disappears under these conditions of visual uncertainty. We have proposed earlier that the visual degradation employed in the present experiments may discourage the normal readers from resorting to a sequential strategy similar to that of the LBL dyslexics, since they can not extract the higher spatial frequencies by narrowing down their attention on individual letters. In this context, the subjects still have to rely on a parallel process to achieve word identification. Therefore, high level lexical effects, such as the facilitatory effect of N size still largely influence word naming. This interpretation applies to the majority of the subjects in our study, mainly the fastest readers. The other readers, who show an interaction between N size and letter confusability similar to the one observed in LBL dyslexia, seem more sensitive to the visual degradation employed (c.f. their longer RTs). For them, the difficulty in discriminating between letters in high confusability targets may have reached such a level that increased N size failed to remain facilitatory. Our results therefore suggest that the same level of visual degradation can cause different levels of internal noise in different subjects and, by extension, a variability in task difficulty. Relatedly, the degree to which the subjects are affected by the

visual degradation may modify the way N size and letter confusability together affect reading performances.

The impact of right hemianopia

The goal of our study was to simulate LBL dyslexia in neurologically intact readers, based on the peripheral account of this disorder. This was done by visually degrading the stimuli in order to prevent normal encoding of letters prior to the activation of the orthographic representation of words. The type of visual degradation employed (reduction of contrast and low pass filtering) was thought to possibly replicate the effect of the right hemianopia that generally accompanies LBL dyslexia, i.e. a loss of high spatial frequencies associated with the parafoveal encoding of the stimuli and/or the bias of the right hemisphere for low spatial frequencies. Pilot studies conducted in our laboratory added support to the hypotheses that a loss of high spatial frequencies could induce, in normal subjects, reading patterns similar to these observed in LBL dyslexics (such as the letter confusability effect). However, some criticisms may be formulated against these assumptions.

Indeed, it can be argued that even though LBL dyslexia is frequently accompanied by right homonymous hemianopia, not all LBL dyslexics are hemianopic (Henderson, Friedman, Teng & Weiner, 1985; Leff, Crewes, Plant, Scott, Kennard & Wise, 2001; Montant, Nazir & Poncet, 1998; Verstichel & Cambier, 1997). However, a closer examination indicates that most of these LBL dyslexics without hemianopia manifest visual difficulties in their right visual hemifield (achromatopsia, higher threshold for detecting a flashed light dot, etc.). It could thus be argued that such disorders signal a difficulty of the left hemisphere in properly encoding the written inputs which may precipitate a right hemisphere involvement even when there is no hemianopia.

There is another argument against the idea that a right hemianopia is responsible for the difficulty in letter identification present in this reading disorder. Leff et al. (2001) examined the word reading performance of four types of subjects: an LBL dyslexic with right hemianopia, patients with hemianopic alexia (a reading disorder associated with right hemianopia), patients who suffered from right hemianopia but who were not alexic, and normal controls. Results indicated that patients with hemianopic alexia or hemianopia did not show the word length effect characteristic of LBL dyslexics (even though a larger length effect than that observed in normal controls was present). It is interesting to note that for the two groups of patients, the visual encoding conditions were identical. In particular, visual information had to transit through the right hemisphere before being transferred to the left hemisphere via the splenium of the corpus callosum. The observations by Leff et al. argue against the view that LBL dyslexic symptoms arise exclusively because of the righ-hemisphere's visual encoding properties. Rather, they indicate that LBL dyslexia requires damage to a specific left hemispheric structure/process separate from that involved in maintaining a functional right visual hemifield and which is unavailable in the right hemisphere.

CONCLUSION

The present report adds weight to the hypothesis of a deficit in LBL dyslexia that occurs early in word processing, prior to the activation of lexical orthographic representations. Indeed, effects of letter confusability and of word length (up to 200 ms / letter), and an interaction between word length and lexical frequency have been provoked in normal readers with visually degraded stimuli. However, the interaction between N size

and letter confusability found in LBL dyslexics has been reproduced only in a small subset of readers. From these observations, it has been argued that the visual degradation employed (reduction of contrast and low pass filtering) may differ in important ways from that present in LBL dyslexia. In particular, it appears most likely that patients still have access to the higher spatial frequencies, which were absent from the stimuli used in the present experiments. Another major issue is the apparently large variability in the sensitivity of normal readers to the visual degradation, which appears to have triggered variable patterns of results in Exp. 3 examining the interactive effects of N size and letter confusability. Further studies will be necessary in order to achieve a complete simulation of LBL dyslexia in neurologically intact readers.

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FIGURE CAPTIONS

Fig. 1 –Average correct response times (ms) as a function of visual presentation (normal vs. degraded) and letter confusability (Exp. 1).

Fig. 2 – Error rates (%) as a function of visual presentation (normal vs. degraded) and letter confusability (Exp. 1).

Fig. 3 – Average correct response times (ms) as a function of word length and lexical frequency (Exp. 2).

Fig. 4 – Error rates (%) as a function of word length and lexical frequency (Exp. 2).

Fig. 5 – Average correct response times (ms) as a function of letter confusability (Conf.) and orthographic neighbourhood size (N size) (Exp. 3).

Fig. 6 – Error rates (%) as a function of letter confusability (Conf.) and orthographic neighbourhood size (N size) (Exp. 3).

Fig. 7 – Reduction (in ms) of the N size effect by high letter confusability as a function of individual means reading latency for words (Exp. 3).

Fig. 8 – Reduction (in ms) of the N size effect by high letter confusability as a function of individual means reading latency for pseudowords (Exp. 3)

FIGURES

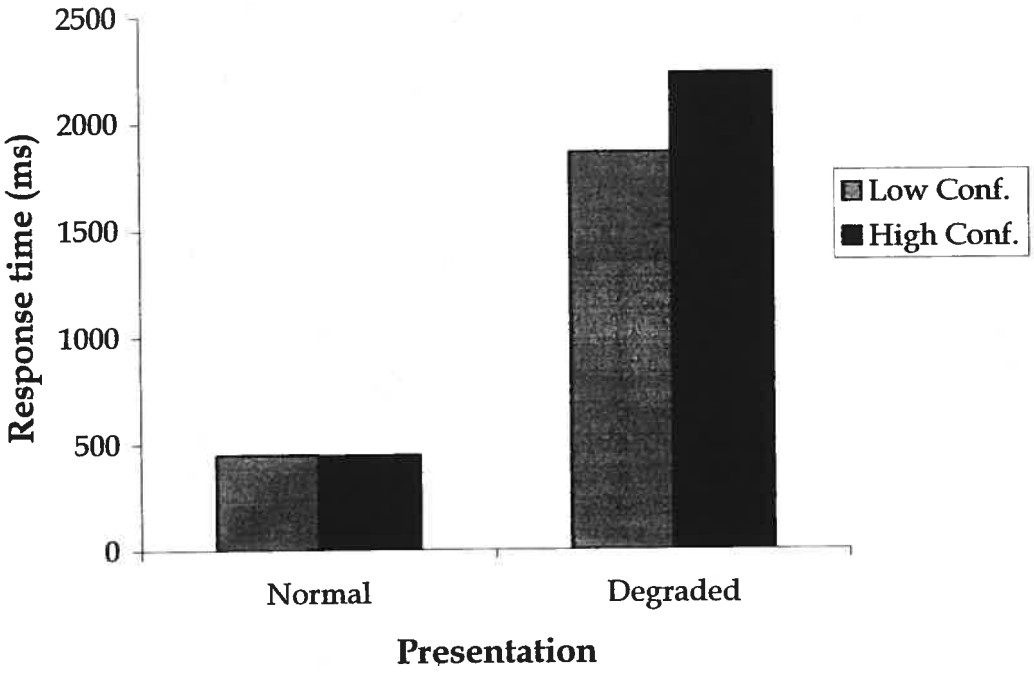
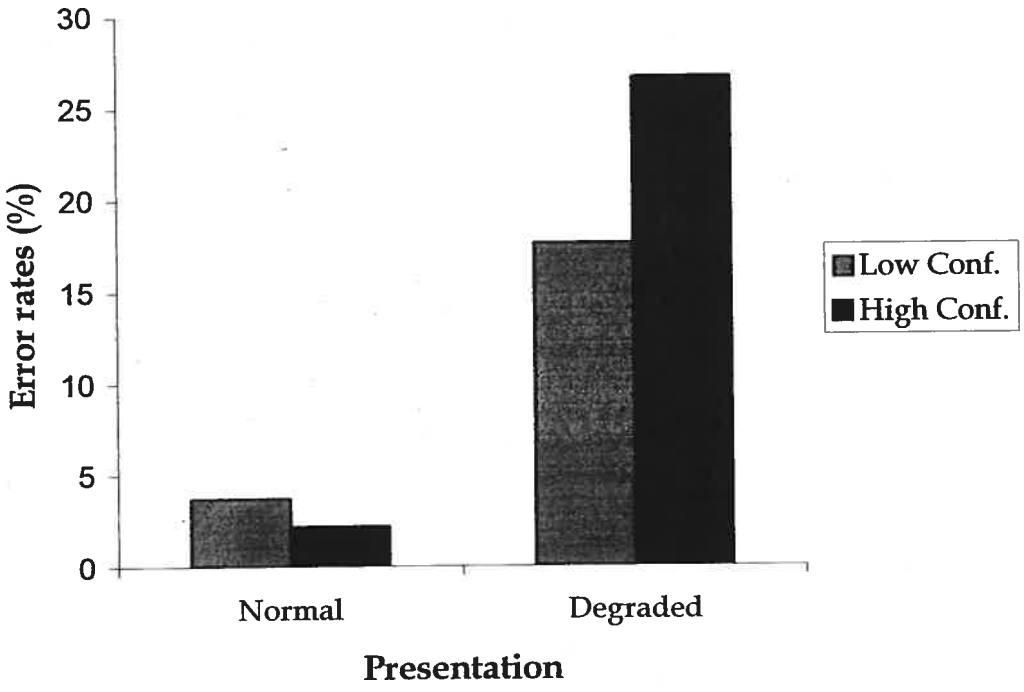


Fig. 1 — Fiset, Arguin & Fiset



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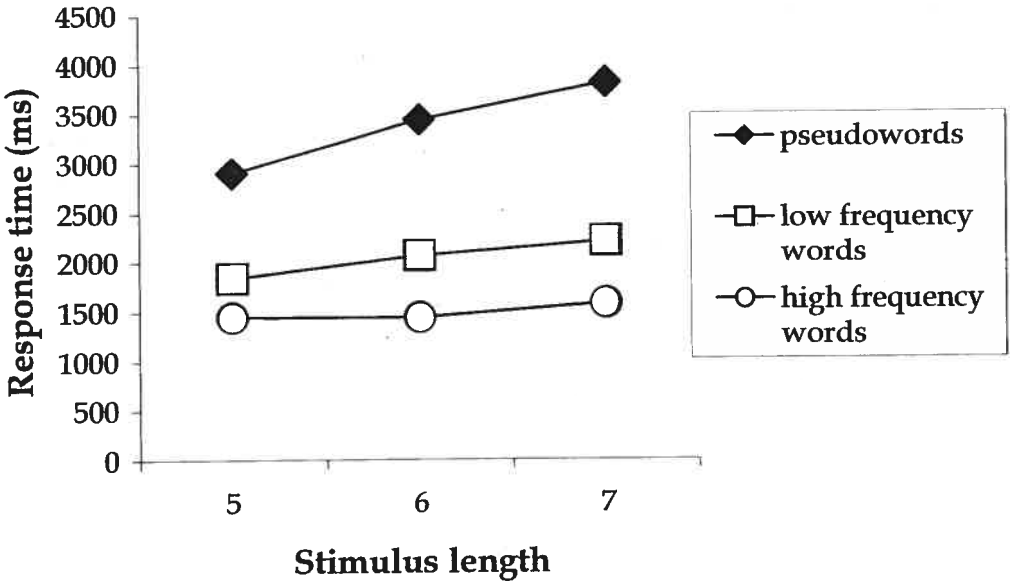

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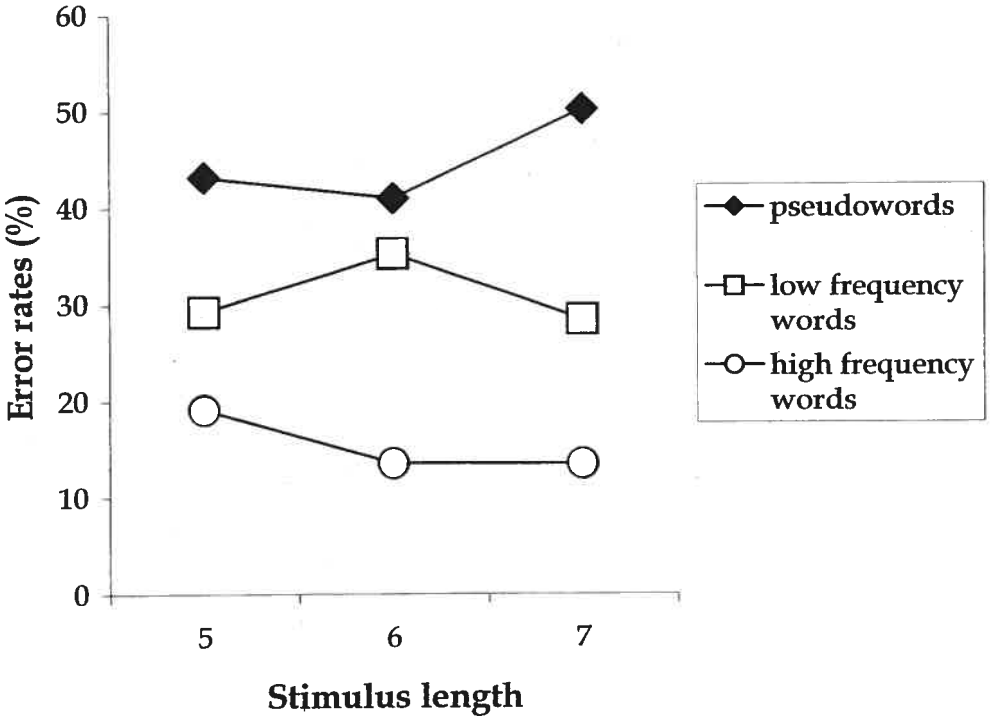

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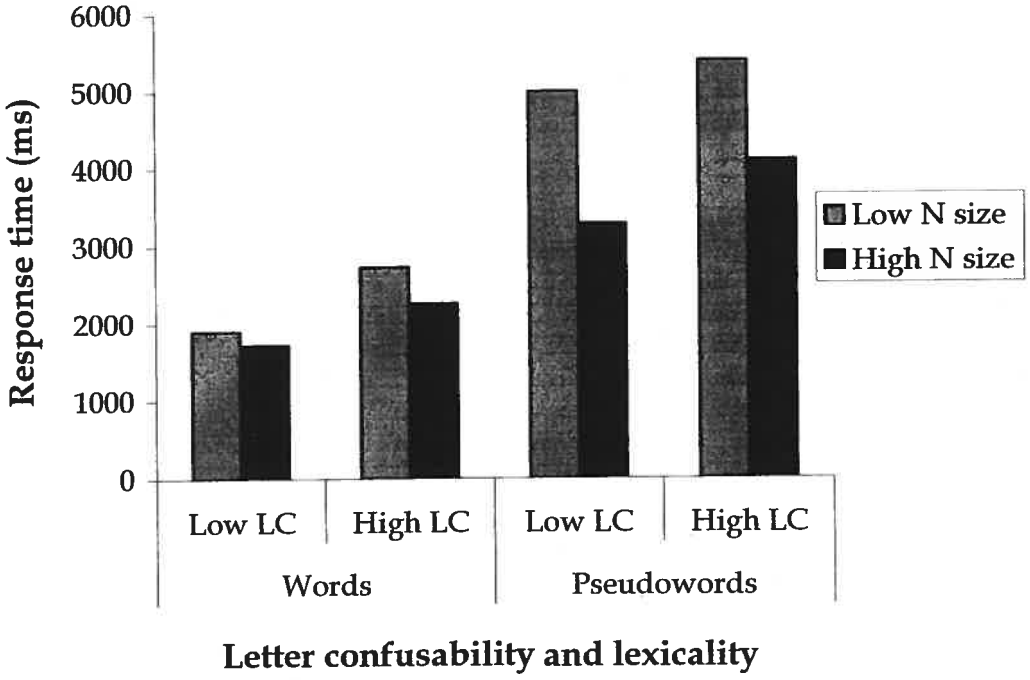

Fig. 5 — Fiset, Arguin & Fiset

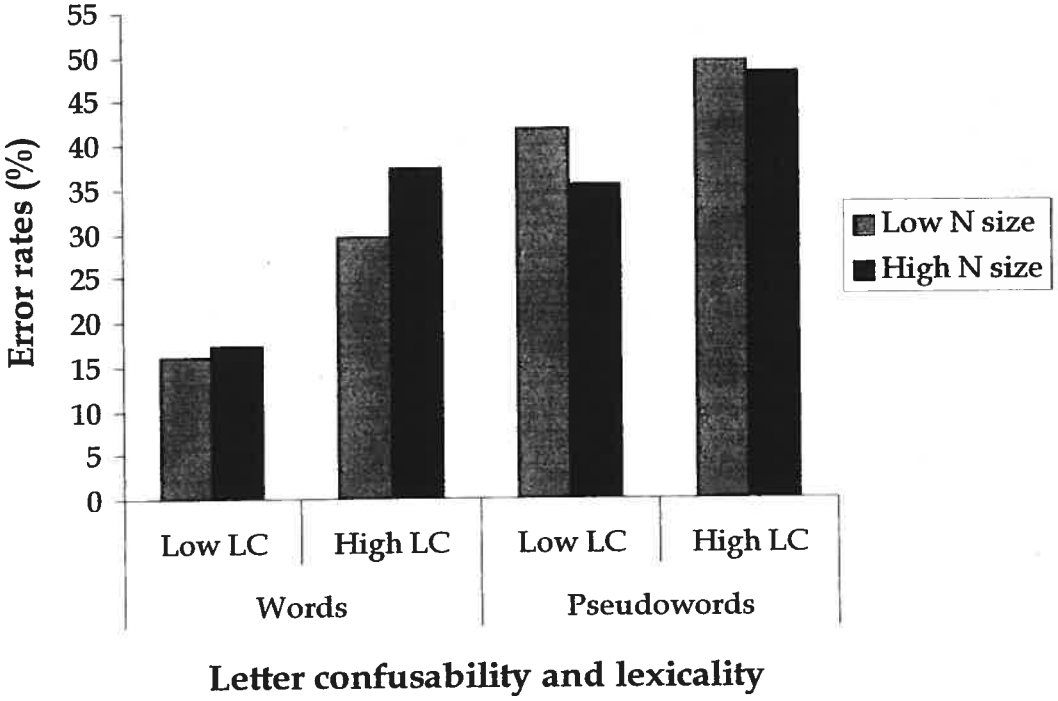

Fig. 6 — Fiset, Arguin & Fiset

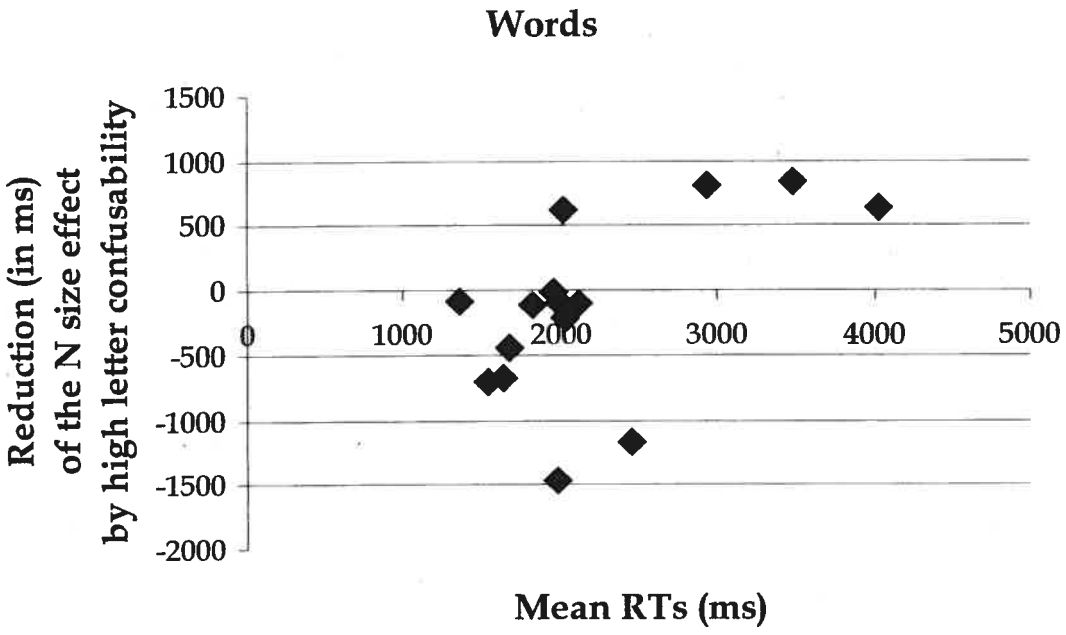

Fig. 7 — Fiset, Arguin & Fiset

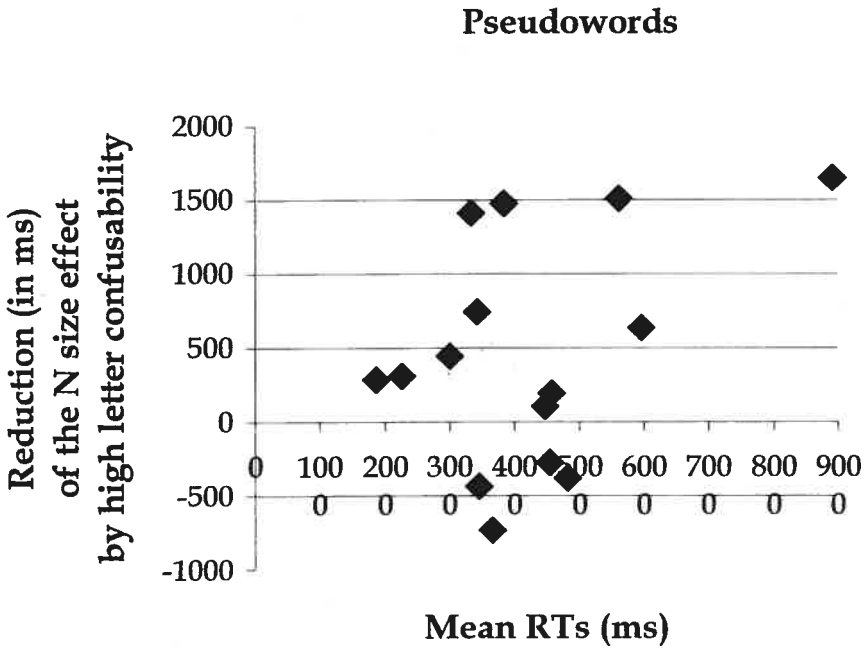

Fig. 8 — Fiset, Arguin & Fiset

CHAPITRE CINQUIÈME:

DISCUSSION GÉNÉRALE

L'objectif principal de ce travail de recherche consistait à mieux préciser les limites du traitement parallèle des lettres chez les dyslexiques lettre-par-lettre et à tenter d'expliquer leur recours à une stratégie séquentielle pour identifier les stimuli. Ces observations ont permis, dans un second volet, de tenter une simulation de ce trouble de lecture chez des sujets neurologiquement sains.

Dans un but de clarté, le présent travail sera divisé en deux sections :

1) Dans un premier temps, les principaux résultats des études rapportées précédemment dans cette thèse seront résumés, en débutant par l'étude portant sur la dyslexie lettre-par-lettre, puis en enchaînant avec le volet portant sur la simulation de ce trouble de lecture chez des sujets normaux. La contribution théorique de nos résultats à la dyslexie lettre-par-lettre sera discutée.

2) Ensuite, certaines hypothèses visant une explication fonctionnelle de la dyslexie lettre-par-lettre seront révisées à la lumière de nos résultats.

I. Résumé des principaux résultats

A. Étude des traitements parallèles et séquentiels dans la dyslexie lettre-par-lettre

La première étude, effectuée auprès de IH, un dyslexique lettre-par-lettre, avait pour objectif de spécifier les troubles affectant le traitement parallèle des lettres et qui amèneraient ces dyslexiques à privilégier un traitement sériel des lettres.

En premier lieu, l'impact du voisinage orthographique sur l'identification de mots dans la dyslexie lettre-par-lettre a été étudié. En effet, Arguin, Bub et Bowers (1998) ont observé la présence d'un effet facilitateur du nombre de voisins orthographiques chez le dyslexique lettre-par-lettre IH. Cet effet était interprété comme étant lié à une faible activation des représentations lexicales orthographiques produite par un traitement parallèle, mais déficient, des lettres dans ce trouble de lecture.

La première expérience a démontré que, pour une longueur de mot constante, une augmentation paramétrique du nombre de voisins orthographiques provoque, chez le dyslexique lettre-par-lettre IH, une décroissance linéaire des temps de réaction. C'est-à-dire que plus le mot possède de voisins orthographiques, plus les temps de réponse à cette cible sont réduits. Ces résultats permettaient de supposer que l'effet de longueur de mot observé chez les dyslexiques lettre-par-lettre pouvait être un artéfact de la corrélation naturelle existant entre la longueur des mots et le voisinage orthographique (se référer à l'introduction pour plus de détails), au lieu de refléter le strict traitement séquentiel des lettres par ces dyslexiques.

La seconde expérience a clairement permis d'infirmer cette hypothèse, en démontrant que l'effet de longueur de mot demeurait présent chez IH malgré un contrôle strict du voisinage orthographique. Cette démonstration venait également appuyer l'hypothèse que l'effet facilitateur du voisinage orthographique reflète la capacité résiduelle, dans ce trouble de lecture, du traitement en parallèle de l'ensemble des lettres constituant le stimulus. En effet, de nombreuses données suggèrent qu'un effet facilitateur du voisinage orthographique dépendrait d'un traitement parallèle des lettres (Arguin et al., 1998, Pugh, Rexer, Peter et Katz, 1994; et revoir l'explication dans l'introduction de ce travail). Toutefois, ce traitement parallèle semble incapable d'assumer seul l'identification des

cibles; un recours à un traitement séquentiel est donc nécessaire. Par ailleurs, les résultats obtenus nous indiquent également que puisque les dyslexiques lettre-par-lettre montrent une réduction de leurs temps de réaction avec les mots possédant un grand nombre de voisins orthographiques, ils peuvent donc, dans une certaine mesure, discriminer entre la cible et ses voisins.

La troisième expérience avait pour objectif de tester l'impact sur l'identification de mots d'une nouvelle variable développée dans notre laboratoire, la confusabilité de lettres. Des études pilotes avaient permis de soupçonner un effet inhibiteur de cette variable, les temps de réaction des dyslexiques lettre-par-lettre pour les mots composés de lettres de haute confusabilité étant plus élevés que pour les mots dont les lettres sont de basse confusabilité. Par contre, cette variable n'influençait pas les temps de réaction des sujets neurologiquement sains. Les hypothèses suivantes ont été posées : si une relation existe entre le traitement séquentiel et la confusabilité des lettres, l'effet de longueur de mot devrait varier en fonction du degré de confusabilité des lettres composant le mot (i.e. que l'effet de longueur de mot serait plus marqué pour les mots ayant une haute confusabilité). Alternativement, il est possible que la confusabilité des lettres affecte l'identification de mots dans la dyslexie lettre-par-lettre, mais sans modifier l'effet de longueur de mot, ce qui supposerait que cette variable soit en lien avec un traitement parallèle dans ce type de dyslexie. Les résultats obtenus appuient cette seconde hypothèse : un important effet de confusabilité a effectivement été observé chez IH (ses temps de réponse étant plus lents pour les mots possédant une haute confusabilité), mais cette variable n'a pas réussi à moduler l'important effet de longueur de mot observé chez ce lecteur, ce qui confirme son impact sur le traitement parallèle des lettres.

Dans la dernière expérience de cette étude, l'interaction entre le voisinage orthographique et la confusabilité de lettres a été examinée. Puisque les dyslexiques lettre-par-lettre démontrent un effet facilitateur du voisinage orthographique, il est suggéré que la discrimination entre la cible et ses voisins est toujours préservée. Mais qu'advient-il lorsque la similarité visuelle entre les lettres est importante? L'impact négatif de la confusabilité de lettres, rapporté plus haut, suggère que cette variable aurait le potentiel de moduler l'effet de voisinage orthographique. Notre dernière expérience le confirme. En effet, l'effet facilitateur du voisinage orthographique chez IH est aboli lorsque les lettres composant les mots possèdent une haute confusabilité. Cet effet facilitateur est toutefois préservé lorsque les mots possèdent des lettres de basse confusabilité. Ces résultats contrastent fortement avec les performances de lecteurs normaux qui 1) ne sont pas incommodés par la confusabilité des lettres et 2) qui présentent un effet facilitateur du voisinage orthographique, tel qu'observé dans la littérature (Andrews, 1989, 1992; Arguin et al., 1998; Carreiras, Perea et Grainger, 1997; Sears, Hino et Lupker, 1995), mais qui n'est pas modulé par la confusabilité de lettres. Globalement, les résultats de ces expériences contribuent grandement à clarifier la nature des limites du traitement parallèle des lettres chez les dyslexiques lettre-par-lettre et apportent de nouvelles informations sur l'aide apportée par le recours à la stratégie compensatoire séquentielle. Ces éléments seront abordés ci-dessous.

a) Le traitement parallèle dans la dyslexie lettre-par-lettre

L'observation d'un effet facilitateur du voisinage orthographique dans nos expériences a été interprétée comme indicateur d'un traitement parallèle dans la dyslexie lettre-par-lettre (se référer à l'introduction). Ce traitement ne peut toutefois supporter à lui seul l'identification des mots, et c'est pourquoi les dyslexiques lettre-par-lettre doivent utiliser un

processus compensatoire basé sur l'identification séquentielle des lettres composant les stimuli.

Une explication possible de cette incapacité des dyslexiques lettre-par-lettre à identifier les mots en parallèle comme chez le lecteur normal, est révélée en premier lieu par l'effet inhabituel de confusabilité de lettre observé chez notre patient. Le coût important (augmentation des temps de réaction) provoqué par la haute confusabilité des lettres indique la présence, dans ce trouble de lecture, d'un déficit de bas niveau affectant l'identification de lettres (qu'il s'agisse de la discrimination entre des lettres similaires ou la transposition de leurs représentations perceptives en représentations identitaires). Ces observations sont en accord avec celles de plusieurs auteurs qui suggèrent la présence d'un déficit dans l'encodage initial des lettres avant l'activation des représentations orthographiques (Arguin et Bub, 1993, Arguin et Bub, 1994; Arguin, Fiset et Bub, 2002; Behrmann, Plaut et Nelson, 1998; Behrmann et Shallice, 1995; Farah et Wallace, 1991; Friedman et Alexander, 1984; Kay et Hanley, 1991; Kinsbourne et Warrington, 1962; Levine et Calviano, 1978; Montant et Behrmann, 2001; Patterson et Kay, 1982; Rapp et Caramazza, 1991).

Étant donné qu'il a été démontré qu'un nombre élevé de voisins orthographiques facilite l'identification des mots chez notre dyslexique lettre-par-lettre, cela implique que le traitement parallèle permet encore la discrimination entre une cible et ses voisins orthographiques. Toutefois, comme la dernière expérience de notre étude le démontre, cette discrimination n'est possible que lorsque les mots sont composés de lettres de basse confusabilité. En effet, lorsque les cibles possèdent une haute confusabilité, l'effet de voisinage orthographique est aboli. Ces résultats peuvent s'expliquer par le phénomène suivant : la haute confusabilité des lettres dans un mot provoque une forte ambiguïté sur l'identité des lettres qui le composent. Cette incertitude est communiquée aux représentations

lexicales de ce mot et de ses voisins orthographiques. L'activation des voisins orthographiques relativement à la cible devient donc trop élevée, ce qui entraîne une disparition de l'effet facilitateur du voisinage orthographique.

Lorsque les mots sont composés de lettres de basse confusabilité, la différence entre l'activation des représentations lexicales de la cible et celles de ses voisins orthographiques résultant du traitement parallèle est plus importante que lorsque les mots possèdent une haute confusabilité. L'effet facilitateur du voisinage orthographique peut donc se manifester. Toutefois, la présence d'un effet de longueur de mot nous indique que, même dans ces conditions, le traitement parallèle demeure inefficace et insuffisant pour parvenir à l'identification des mots et que les dyslexiques lettre-par-lettre doivent encore recourir à une stratégie séquentielle. Ce recours à une stratégie compensatoire s'explique par la persistance, dans le traitement parallèle, d'un niveau élevé de bruit dans l'activation des représentations lexicales-orthographiques qui empêche l'identification fiable et absolue de la cible présentée et ce, même si ce bruit est moins élevé que pour les mots de haute confusabilité.

Il est pertinent d'ajouter que le résultat le plus intéressant de cette étude sur un plan théorique, i.e. l'interaction entre le nombre de voisins orthographiques et la confusabilité des lettres, a été répliqué par Arguin et Bub (soumis) chez trois dyslexiques lettre-par-lettre anglophones et par Fiset, D., Arguin et McCabe (soumis), chez un dyslexique francophone. En effet, ces dyslexiques lettre-par-lettre ont individuellement montré un effet facilitateur du voisinage orthographique, mais uniquement lorsque les cibles étaient composées de lettres de basse confusabilité. Cette observation ajoute grandement au poids de notre démonstration chez IH, qui n'apparaît donc plus fortuite ou spécifique à ce dyslexique lettre-par-lettre.

b) Lecture implicite dans la dyslexie lettre-par-lettre

Ce présent travail a également offert un apport théorique original à la problématique du phénomène de lecture implicite observé chez plusieurs dyslexiques lettre-par-lettre : il a été suggéré que les capacités résiduelles du traitement parallèle chez les dyslexiques pouvaient expliquer les habiletés de lecture implicites relevées chez certains patients. Cette hypothèse se base sur les données suivantes : Arguin et Bub (1995) avaient proposé qu'il existe une différence importante entre les tâches d'identification (comme la lecture de mots) et les tâches de classification (telles la décision lexicale et la classification sémantique) et que cette différence réside dans le critère de décision que les sujets appliquent sur les représentations internes activées. Ces auteurs avaient suggéré que l'identification absolue d'un mot dépend d'un certain critère dans le rapport signal / bruit : pour parvenir à lire une cible, l'activation de sa représentation lexicale doit être supérieure à toutes les autres représentations activées (dans le domaine de représentation pertinent). Si ce rapport est trop bas, en raison d'un bruit excessif dans le système, l'identification de la cible devient ardue et compromise. Par opposition, les épreuves de décision lexicale et de classification sémantiques ne requièrent pas l'identification absolue du stimulus, et leur réussite ne demande qu'un niveau suffisant d'activation dans le domaine de représentation pertinent. Le niveau de bruit devient alors peu contraignant. Ainsi, la notion de lecture implicite dans la dyslexie lettre-par-lettre est entièrement congruente avec l'idée d'activations lexicales imprécises résultant d'un traitement parallèle des lettres encore présent mais lésé.

Il est pertinent de rapporter que certaines conditions peuvent toutefois nuire au phénomène de lecture implicite de mots dans la dyslexie lettre-par-lettre. En effet, Fiset, D., Arguin, Saumier, Humphreys et

Riddoch (en préparation) ont étudié l'impact de la confusabilité de lettres sur les effets de lecture implicite qui avaient déjà été observés chez IH (amorçage associatif, amorçage orthographique, effet de supériorité du mot et capacités de décision lexicale à des durées de présentation trop brèves pour permettre l'identification des cibles). Ils ont remarqué que ces effets sont abolis lorsque les cibles sont composées majoritairement de lettres de haute confusabilité. Il est possible qu'une haute confusabilité de lettres entraîne une augmentation tellement importante du bruit de fond dans le système que le signal d'activation des représentations pertinentes n'est plus assez fort pour permettre une réalisation efficace de la tâche. Avec des mots dont les lettres ont une basse confusabilité, l'input serait suffisamment précis pour permettre l'utilisation des capacités résiduelles du traitement parallèle. Rappelons que les expériences examinant la lecture implicite chez les dyslexiques lettre-par-lettre n'avaient jusqu'à présent jamais considéré l'impact de la confusabilité des lettres (ou une autre variable concernant la similarité visuelle des lettres) et qu'il est possible que ce manque de contrôle au niveau des stimuli utilisés ait pu nuire à la capacité de produire ou moduler ces phénomènes chez divers dyslexiques lettre-par-lettre, ce qui expliquerait le fait que la lecture implicite n'a été observée que chez un nombre relativement faible de patients.

c) Rôle du traitement séquentiel dans la dyslexie lettre-par-lettre

L'étude effectuée auprès de IH nous renseigne également un peu plus sur le traitement compensatoire séquentiel employé par les dyslexiques lettre-par-lettre pour identifier de façon absolue le stimulus. La troisième expérience révèle que la confusabilité de lettres n'a pas réussi à moduler l'important effet de longueur de mot caractéristique de la dyslexie lettre-par-lettre. Ceci indique que les difficultés perceptives

occasionnées par des mots composés de lettres de haute confusabilité n'affecteraient pas le traitement séquentiel chez ce patient. En effet, si la source de cette difficulté visuelle était un processus opérant sur les lettres individuelles, alors les mots les plus longs auraient dû produire de plus grands effets de confusabilité que les mots les plus courts.

Par ailleurs, cette étude nous éclaire sur une fonction importante du traitement séquentiel dans la dyslexie lettre-par-lettre. Cette stratégie compensatoire, qui n'est pas influencée par la similarité entre les lettres, différerait du traitement parallèle quant à la manière dont les ressources d'attention visuelle sélective sont allouées à la cible. En effet, le traitement séquentiel serait associé avec le rétrécissement du faisceau attentionnel sur les lettres individuelles traitées les unes après les autres. Par opposition, dans le traitement parallèle, l'attention serait distribuée sur la surface entière du mot. Certains des rôles de l'attention sélective seraient d'augmenter le rapport signal / bruit dans le traitement des stimuli (Hawkins, Hillyard, Luck, Mouloua, Downing et Woodward, 1990); Hummel et Stankiewicz, 1998) ou encore d'augmenter la résolution spatiale des représentations par une amplification du signal (Yeshurun et Carrasco, 1998; 2000). L'attention agirait même très tôt dans le traitement visuel et pourrait modifier les réponses d'aires visuelles primaires (Yeshurun et Carrasco, 1998; Talgar, Pelli et Carrasco, 2003). Des données un peu plus détaillées sur la contribution de l'attention dans le traitement visuel des lettres seront présentées plus loin dans le volet simulation de la dyslexie lettre-par-lettre.

La focalisation de l'attention sur les lettres individuelles, telle qu'accomplie lors du traitement séquentiel des lettres, permettrait donc d'améliorer la capacité du système d'identification des lettres à différencier les lettres visuellement similaires entre elles. Par conséquent, un signal plus défini concernant l'identité des lettres constituant le mot est

envoyé au système de représentations lexicales-orthographiques, comparé au signal émis par le traitement parallèle résiduel.

L'apport original du présent travail consiste donc 1) à préciser l'origine des difficultés du traitement parallèle à identifier de façon absolue les lettres composant les cibles, en raison de sa sensibilité à la confusion visuelle entre les lettres; et 2) à expliquer le rôle compensatoire de la stratégie séquentielle lettre-par-lettre, qui permet, par la focalisation attentionnelle sur les lettres individuelles, de réduire cette confusion visuelle.

B. La simulation de la dyslexie lettre-par-lettre chez des lecteurs normaux

La seconde partie de ce travail, regroupant les deuxième et troisième articles, avait pour objectif de simuler la dyslexie lettre-par-lettre chez des sujets normaux. En effet, il avait été proposé, à partir de l'hypothèse d'une atteinte périphérique dans la dyslexie lettre-par-lettre, que ce trouble de lecture pouvait être reproduit chez des sujets normaux en dégradant visuellement les stimuli. Dans un premier temps, nous avons examiné la proposition de Nelson, Behrmann et Plaut (communication personnelle) de simuler la dyslexie lettre-par-lettre en réduisant le contraste d'intensité lumineuse entre le stimulus et le fond. Par la suite, étant donné l'échec de cette proposition, une autre forme de dégradation visuelle sensée provoquer chez les lecteurs normaux l'effet de confusabilité a été étudiée.

a) Impact de la réduction de contraste

La première étude concernant la simulation de la dyslexie lettre-par-lettre chez des lecteurs normaux avait pour objectif de répliquer les travaux de Nelson et al. dans des conditions mieux contrôlées. Ces auteurs ont obtenu, chez des lecteurs neurologiquement sains, des effets de longueur de mot, de fréquence lexicale et d'imageabilité avec des stimuli présentés avec un contraste réduit. Les auteurs en concluaient que plusieurs effets lexicaux, sémantiques et implicites observés chez les dyslexiques lettre-par-lettre pouvaient être obtenus chez les lecteurs normaux quand le traitement perceptif des stimuli était affecté ou limité. Toutefois, notre étude a apporté un bémol à la proposition de ces auteurs. En effet, dans notre expérience (où les stimuli étaient également présentés en contraste réduit), l'effet de longueur de mot n'était présent que lorsque les mots de différentes longueurs n'avaient pas été appariés entre eux quant au voisinage orthographique et donc que les mots les plus courts possédaient significativement plus de voisins que les mots les plus longs (consulter l'introduction pour plus d'explications). Lorsqu'un contrôle strict du voisinage orthographique était effectué, i.e. lorsque que le nombre de voisins orthographiques ne différait pas à travers les différentes longueurs de mots, l'effet de longueur de mot disparaissait. Il a donc été conclu que l'effet de longueur de mot obtenu par Nelson et al. en présentation en contraste réduit est en fait un artéfact du nombre de voisins orthographiques, variable que les auteurs n'avaient pas contrôlée.

Cette expérience a donc permis de mettre en lumière deux aspects importants. En premier lieu, la réduction de contraste n'apparaît pas suffisante pour produire, chez le lecteur neurologiquement sain, l'effet de longueur de mot caractéristique de la dyslexie lettre-par-lettre. Il est toutefois important de souligner que nos résultats ne signifient pas que l'hypothèse périphérique de ce trouble de lecture doit être rejetée, ni

même que l'emploi d'autres types de dégradation visuelle échouerait dans la simulation de la dyslexie lettre-par-lettre. Il est plutôt suggéré que, bien que la simple réduction de contraste ait un impact sur la lecture chez les sujets normaux en augmentant les temps de réponse (Legge, Rubin et Luebker, 1987), elle produit un effet qualitativement différent de celui observé chez les dyslexiques lettre-par-lettre.

Par ailleurs, ce travail fournit également une contribution méthodologique à l'étude de la dyslexie lettre-par-lettre. En effet, cette étude permet de confirmer l'importance du contrôle du voisinage orthographique dans les études portant sur la dyslexie lettre-par-lettre ou sa simulation chez des normaux lecteurs, étant donné l'influence de cette variable sur le traitement parallèle des lettres dans ce trouble de lecture (Arguin et al., 1998; Arguin et al., 2002; Fiset, S. et Arguin, 1998). Un tel contrôle ne semble pas encore très répandu dans la littérature portant sur la dyslexie lettre-par-lettre, bien que d'autres auteurs aient souligné que cette variable pouvait moduler la production de l'effet de longueur de mots (Lavidor et Ellis, 2002).

b) Impact du retrait des hautes fréquences spatiales

L'observation que les difficultés dans la dyslexie lettre-par-lettre proviendraient de la confusion visuelle entre les lettres produite par une atteinte du traitement parallèle des lettres a permis d'élaborer l'hypothèse suivante : une dégradation visuelle des stimuli provoquant un effet de confusabilité des lettres chez des lecteurs neurologiquement sains pourrait entraîner l'apparition de symptômes caractéristiques de la dyslexie lettre-par-lettre (tel l'effet de longueur de mots). Notre troisième étude visait à vérifier cette hypothèse. Dans ces expériences, la dégradation visuelle employée consistait en une réduction de contraste et en un retrait des

hautes fréquences spatiales, manipulations devant reproduire les conditions d'une exposition parafovéale et /ou le traitement initial par l'hémisphère droit provoqué par l'hémianopsie homonyme droite présente chez la grande majorité des dyslexiques lettre-par-lettre.

Dans la première expérience, des mots de cinq lettres possédant une haute ou basse confusabilité de lettres ont été présentés dans ces conditions de dégradation à des sujets neurologiquement sains. Cette étude révèle sans ambiguïté que ce type de dégradation visuelle provoque un important effet de confusabilité chez les lecteurs normaux, alors que cet effet est totalement absent dans des conditions normales de présentation. À la lumière de ces résultats, la manipulation visuelle employée apparaissait donc très prometteuse dans l'objectif de simuler la dyslexie lettre-par-lettre chez des lecteurs normaux.

La seconde expérience a ajouté du poids à cette observation et à l'hypothèse, dans la dyslexie lettre-par-lettre, d'un déficit survenant tôt dans le traitement des mots, avant l'activation des représentations lexicales orthographiques. En effet, cette expérience a démontré que la dégradation visuelle provoquait un effet de longueur de mot chez nos sujets et que la taille de cet effet (200 msec par lettre additionnelle dans les stimuli), était beaucoup plus grande que ce qui a été observé chez des lecteurs normaux en condition normale de présentation (Weekes, 1997). Cet effet de longueur de mot a été observé dans toutes les classes lexicales utilisées, i.e. pour les mots de basse et de haute fréquence, ainsi que pour les pseudomots. Par contre, ces deux variables interagissaient : on notait ainsi un effet de longueur de mot plus prononcé pour les pseudomots que pour les mots de basse fréquence, et plus accentué pour les mots de basse fréquence par comparaison avec les mots de haute fréquence. Une telle interaction entre la fréquence lexicale et la longueur des stimuli a déjà été observée par Behrmann et al. (1998) chez cinq dyslexiques lettre-par-lettre.

La production chez des sujets neurologiquement sains dans des conditions de dégradation visuelle d'effets de confusabilité et de longueur de mots similaires à ceux observés dans la dyslexie lettre-par-lettre, est un apport original important du présent travail.

La troisième expérience de cette étude avait pour objectif de reproduire chez les lecteurs normaux l'interaction entre le voisinage orthographique et la confusabilité de lettres observée précédemment par Arguin et al. (2002), Arguin et Bub (soumis) et Fiset, D. et al. (soumis) auprès d'autres dyslexiques lettre-par-lettre. En accord avec les résultats obtenus dans ces études, l'hypothèse sous-jacente était que l'effet de voisinage orthographique devrait être présent pour les mots de basse confusabilité et aboli pour les mots de haute confusabilité. Les résultats obtenus chez nos sujets ne correspondaient toutefois pas aux observations précédentes. En effet, bien que les sujets présentaient des effets de confusabilité et de voisinage orthographique, comme les dyslexiques lettre-par-lettre, ces variables n'interagissaient pas de la même façon que chez nos patients. Ainsi, contrairement à ce qui était attendu, la dégradation visuelle employée semblait entraîner une accentuation de l'effet facilitateur du voisinage orthographique pour les mots de haute confusabilité. Ce résultat étonnant sera discuté plus loin.

c) Le retrait des hautes fréquences spatiales reflète-t-il le déficit perceptif caractéristique de la dyslexie lettre-par-lettre?

Une première critique pouvant être adressée à nos résultats concerne la taille de l'effet de longueur de mot observé chez nos lecteurs en présentation visuelle dégradée. En effet, dans la seconde expérience de cette étude, l'effet de longueur de mot moyen était d'environ 200 msec par lettre additionnelle dans le stimulus, taille largement plus réduite que ce

qui est observé chez la majorité des dyslexiques lettre-par-lettre. Une explication possible pourrait être que notre tâche (i.e. le niveau de dégradation visuelle employée) n'était pas assez ardue pour obliger nos sujets à recourir à une stratégie séquentielle lettre-par-lettre. Les taux d'erreurs obtenus par nos sujets dans ces expériences (variant de 15 à 38% pour les mots, selon la fréquence lexicale et le nombre de voisins orthographiques), sont largement supérieurs à ceux observés chez les dyslexiques lettre-par-lettre, ce qui nous amène à rejeter cette hypothèse. La difficulté de la tâche ne semble donc pas expliquer la faiblesse relative de l'effet de longueur de mots obtenu dans notre étude.

Une autre explication possible de l'obtention d'un faible effet de longueur de mot relativement à celui observé chez les dyslexiques lettre-par-lettre résiderait dans le fait que le type de dégradation visuelle employé ne provoquerait pas, chez nos sujets, le trouble visuel affectant la reconnaissance de lettres chez les dyslexiques lettre-par-lettre. La manipulation employée, qui consistait à extraire les hautes fréquences spatiales des stimuli et à les présenter en contraste réduit, avait pour objectif de reproduire la perte des hautes fréquences spatiales provoquée par un encodage parafovéal des stimuli et/ou le traitement initial des stimuli par l'hémisphère droit, en lien avec l'hémianopsie homonyme droite observée fréquemment chez les dyslexiques lettre-par-lettre. Ceci devait induire chez les sujets neurologiquement sains un effet de confusabilité de lettres similaire à celui observé dans la dyslexie lettre-par-lettre. L'extraction des hautes fréquences spatiales pourrait par contre avoir privé les stimuli d'éléments nécessaires à la lecture. Les données appuyant cette hypothèse sont issues des expériences psychophysiques menées chez le sujet normal. Elles seront abordées brièvement ici.

La première étude de ce travail suggérait que les dyslexiques lettre-par-lettre adoptent une stratégie séquentielle compensatoire pour réduire

la confusion visuelle entre les lettres similaires résultant d'un traitement parallèle déficitaire, en focalisant leur attention sur les lettres individuelles. Des études psychophysiques permettent d'ajouter un poids à cette proposition : en effet, Yeshurun et Carrasco (1998; 2000) ont montré que l'attention peut améliorer la performance visuelle en augmentant la résolution spatiale. Il est donc possible que le fait de diriger l'attention sur les lettres individuelles permette aux dyslexiques d'améliorer leur habileté à extraire les fréquences spatiales moyennes / hautes [environ 3 cycles/lettre selon Solomon et Pelli (1994)], qui seraient plus optimales que les basses fréquences spatiales dans la reconnaissance de lettres.

Dans nos expériences, avec la dégradation visuelle employée, les lecteurs normaux ne pouvaient bénéficier de cette stratégie compensatoire séquentielle. En effet, ils ne pouvaient utiliser une focalisation attentionnelle ou une fovéation séquentielle sur les lettres individuelles pour améliorer l'encodage des fréquences spatiales moyennes à hautes, car ces fréquences avaient été retirées complètement des stimuli. Par conséquent, le recours à une stratégie séquentielle lettre-par-lettre s'avérait peu efficace. Cette incapacité à compenser totalement pour la dégradation des stimuli pourrait également être la cause du faible effet de longueur de mot relativement aux dyslexiques lettre-par-lettre et des taux d'erreurs plus élevés que ce qu'on observe habituellement dans ce trouble de lecture : en effet, l'absence des fréquences spatiales nécessaires à l'identification des lettres individuelles rend cette tâche ardue même après une focalisation de l'attention, ce qui entraînerait des erreurs d'identification.

d) Variabilité individuelle : différences dans la sensibilité des sujets à la dégradation visuelle?

Dans la troisième expérience de l'article portant sur la simulation de la dyslexie lettre-par-lettre (dans lesquelles les hautes fréquences spatiales ont été extraites), une analyse plus détaillée des moyennes individuelles des sujets a permis de constater une importante variabilité dans les temps de réponse, certains sujets étant beaucoup plus lents que d'autres. Il a également été observé qu'une corrélation substantielle existait entre les temps de réaction moyens pour les mots et la réduction de l'amplitude de l'effet facilitateur d'un haut voisinage orthographique en condition de haute (vs. basse) confusabilité. Cette réduction est substantielle pour la plupart des sujets les plus lents mais elle est renversée pour les sujets les plus rapides (i.e. l'effet facilitateur du voisinage orthographique est amplifié pour les mots composés de lettres de hautes confusabilité). Pour les pseudomots, cette corrélation entre les temps de réaction et la réduction de la taille de l'effet facilitateur du voisinage orthographique n'est pas aussi forte. Il convient de préciser toutefois que l'interaction entre le voisinage orthographique et les pseudomots n'a jamais été directement testée chez les dyslexiques lettre-par-lettre et qu'une telle interaction entre ces variables, ainsi que sa direction, ne peuvent que faire l'objet de spéculations.

Une division à posteriori des sujets en deux groupes selon leurs temps de réponse en lecture de mots uniquement (lents vs. rapides) confirme la tendance observée pour les mots. Les sujets les plus lents tendent à présenter une interaction similaire à celle documentée dans la dyslexie lettre-par-lettre, alors que chez les sujets les plus rapides, l'effet facilitateur du voisinage orthographique tend à être amplifié avec les mots dont les lettres sont de haute confusabilité. Il semble donc que l'ampleur avec laquelle les sujets sont affectés par la dégradation visuelle employée

peut transformer l'interaction entre le voisinage orthographique et la confusabilité de lettres, ainsi que leur impact sur la lecture des stimuli.

Une explication de cette variabilité entre les sujets quant à l'interaction du voisinage orthographique avec la confusabilité des lettres est fournie par l'hypothèse élaborée plus haut, quant à l'incapacité des sujets à améliorer l'encodage des fréquences spatiales optimales dans la lecture en focalisant leur attention sur les lettres individuelles.

Tel qu'expliqué précédemment dans cette discussion (section « traitement parallèle dans la dyslexie lettre-par-lettre »), lorsqu'un mot possède plusieurs voisins orthographiques et que les lettres composant ce mot sont de haute confusabilité, l'incertitude visuelle est très élevée et l'effet facilitateur du voisinage orthographique est aboli (Arguin et al., 2002 ; Arguin et Bub, soumis). Ceci est dû au niveau de bruit élevé causant une forte compétition entre les signaux provenant des voisins de la cible et les signaux émis par les lettres composant cette cible. La dégradation visuelle utilisée dans les expériences rapportées ici peut amener les lecteurs normaux à rejeter l'utilisation d'une stratégie séquentielle similaire à celle employée par les dyslexiques lettre-par-lettre, car ils ne peuvent extraire les fréquences spatiales optimales en focalisant leur attention sur les lettres individuelles comme le font les sujets dyslexiques. Les sujets ne peuvent donc compter que sur le traitement parallèle des lettres pour identifier les mots présentés. En conséquence, des effets lexicaux de haut niveau, comme les effets facilitateurs du voisinage orthographique, peuvent encore influencer la lecture de la cible. Cette interprétation permet d'expliquer les résultats de la majorité des sujets ayant participé à la troisième expérience de la dernière étude, en particulier les lecteurs les plus rapides. Les autres sujets, qui présentent une interaction entre le voisinage orthographique et la confusabilité de lettres similaire à celle observée dans la dyslexie lettre-par-lettre, semblent

plus sensibles à la dégradation visuelle employée. Pour eux, la difficulté dans la discrimination entre les lettres pour les cibles possédant des lettres de haute confusabilité peut avoir atteint un tel niveau qu'une augmentation du nombre de voisins orthographiques cesse d'être facilitatrice. Les résultats obtenus dans cette expérience suggèrent donc qu'un même niveau de dégradation visuelle induit, chez les sujets, différents niveaux de bruit interne et, par conséquent, une variabilité dans la difficulté de la tâche.

Il aurait été intéressant de pouvoir corréler les résultats des sujets de la troisième expérience, qui mesurait l'interaction entre le voisinage orthographique et la confusabilité de lettres, avec les données obtenues lors de la deuxième expérience, dans laquelle l'effet de longueur de mot et sa modulation par la fréquence lexicale était étudiés. Cet examen aurait permis de vérifier si les sujets présentant une interaction entre le voisinage orthographique et la confusabilité de lettres similaire aux dyslexiques lettre-par-lettre montraient également des effets de longueur de mot et de fréquence lexicale plus accentués que les autres sujets. Malheureusement, cette observation n'a pu être possible car les mêmes sujets ne pouvaient participer à plus d'une expérience. Cette précaution avait été employée pour éviter que les sujets exposés trop fréquemment à nos stimuli ne développent une expertise particulière qui aurait pu moduler les effets observés.

La simulation de la dyslexie lettre-par-lettre chez le lecteur normal par le biais de la dégradation visuelle des stimuli est une avenue prometteuse. Cette manipulation devrait par contre être modifiée afin de produire de façon plus réaliste le trouble perceptif sous-tendant cette dyslexie. Il serait intéressant par exemple d'examiner l'impact du retrait des basses fréquences spatiales sur la lecture de sujets normaux : dans ces conditions, une prédiction plausible est que ces lecteurs se montreraient

insensibles à la confusabilité de lettres (i.e. que la confusabilité des lettres n'influencerait pas les temps de lecture), mais qu'ils présenteraient un important effet de longueur de mot, car ils devraient avoir recours à une focalisation attentionnelle sur les lettres individuelles afin d'extraire l'information visuelle pertinente à l'identification des lettres. Une manipulation alternative serait de n'extraire que les fréquences spatiales optimales à l'identification de lettres vers 3 cycles / lettre: les performances des sujets normaux devraient alors refléter celles observées dans la dyslexie lettre-par-lettre, c'est-à-dire la présence à la fois d'importants effets de confusabilité et de longueur de mot. Dans ces conditions, il deviendrait également très intéressant d'étudier, chez les lecteurs normaux, les phénomènes de lecture implicite observé chez les patients. Notre travail ouvre donc la voie à plusieurs pistes de recherches sur la dyslexie lettre-par-lettre, qui sont également prometteuses dans l'optique d'une remédiation de ce trouble de lecture : en effet, une possibilité à envisager est que la modulation des fréquences spatiales dans les stimuli visuels puisse aider à l'identification de lettres chez ces dyslexiques.

II. Apports aux modèles explicatifs de la dyslexie lettre-par-lettre

Depuis Jules Dejerine (1892), de nombreuses hypothèses ont été proposées pour tenter d'expliquer la symptomatologie de l'alexie pure ou de la dyslexie lettre-par-lettre. Dans l'introduction de ce travail, quelques théories explicatives de la dyslexie lettre-par-lettre ont été présentées. La plupart de ces hypothèses suggèrent que ce trouble de lecture est causé par un déficit affectant soit la perception des formes des lettres, soit la transposition de cette information (sur la forme des lettres) en représentations abstraites des identités de lettres (Arguin et Bub, 1993; Arguin et al, 2002; Behrmann et al., 1998; Behrmann et Shallice, 1995;

Farah et Wallace, 1991; Kay et Hanley, 1991; Rapp et Caramazza; 1991; Reuter-Lorenz et Brunn, 1990). D'autres auteurs (Coslett et Saffran, 1989; Coslett, Saffran, Greenbaum et Schwartz, 1993; Saffran et Coslett, 1998) ont proposé que l'hémisphère droit contribuait grandement à certains phénomènes de lecture observés dans ce type de dyslexie, notamment la lecture implicite. Les résultats provenant de nos études sur IH, dyslexique lettre-par-lettre, et sur la simulation de ce trouble de lecture chez des sujets neurologiquement sains permettent d'apporter une contribution aux théories de la dyslexie lettre-par-lettre.

A) L'encodage initial par l'hémisphère droit joue-t-il un rôle dans la symptomatologie de la dyslexie lettre-par-lettre?

Plusieurs auteurs ont émis l'hypothèse d'une contribution de l'hémisphère droit au profil de lecture observé dans la dyslexie lettre-par-lettre (Cohen, Martinaud, Lemer, Lehéricy, Samson, Obadia et al., 2003; Coslett et Saffran, 1989; Coslett et al., 1993; Saffran et Coslett, 1998). Une telle proposition est logique considérant que la plupart de ces patients doivent encoder les stimuli visuels parafovéalement et par le biais initial de l'hémisphère droit, en raison de la présence de lésions occipitales gauches provoquant une hémianopie homonyme droite. Ainsi, pour la majorité des dyslexiques lettre-par-lettre, l'information doit en premier lieu transiter par l'hémisphère droit, avant d'être transférée à l'hémisphère gauche par le biais de connections calleuses. Cette hypothèse expliquait pourquoi, dans nos expériences portant sur la simulation de la dyslexie lettre-par-lettre, la dégradation visuelle employée consistait à retirer les hautes fréquences spatiales, car cette manipulation reproduisait les conditions d'encodage parafovéal et le traitement préférentiel des basses fréquences spatiales par l'hémisphère droit (Ivry et Robertson, 1998). Des études pilotes effectuées dans notre laboratoire confirmaient la pertinence

d'effectuer une telle dégradation visuelle, car elle favorisait l'apparition d'un effet de confusabilité chez les lecteurs normaux.

Toutefois, certaines critiques peuvent être formulées contre le postulat sous-jacent à notre manipulation visuelle. En premier lieu, bien que la dyslexie lettre-par-lettre soit fréquemment accompagnée d'une hémianopsie homonyme droite, tous les dyslexiques lettre-par-lettre ne sont pas hémianopsiques (Henderson, Friedman, Teng et Weiner, 1985; Leff, Crewes, Plant, Scott, Kennard et Wise, 2001; Montant, Nazir et Poncet, 1998; Verstichel et Cambier, 1997). Cette constatation va donc à l'encontre de l'argument d'une implication hémisphérique droite dans la symptomatologie des dyslexiques lettre-par-lettre. Toutefois, il semblerait que les patients ne présentant pas d'hémianopsie droite démontreraient quand même des difficultés dans leur champ visuel droit (telles que l'achromatopsie, un seuil plus élevé pour détecter des points lumineux, etc.). Ces troubles perceptifs pourraient donc entraîner une difficulté de l'hémisphère gauche dans l'encodage adéquat des mots présentés visuellement. Il est donc possible que l'hémisphère droit doive obligatoirement être sollicité dans le traitement visuel de ces stimuli, même en l'absence d'hémianopsie.

Il existe par contre un autre argument réfutant l'idée que l'hémianopsie droite serait responsable des difficultés d'identification de lettres dans la dyslexie lettre-par-lettre. Des auteurs (Leff et al., 2001) ont testé les performances en lecture de mots isolés d'alexiques hémianopsiques et de patients présentant une hémianopsie droite (sans dyslexie) et les ont contrastées avec celles d'un dyslexique lettre-par-lettre. L'alexie hémianopsique est une autre forme d'alexie périphérique survenant chez des patients atteints d'hémianopsie droite. Cette alexie est caractérisée par des difficultés importantes en lecture de texte, en raison de la présence de mouvements oculaires anormaux (Leff et al., 2001).

Les résultats de cette étude indiquent que les alexiques hémianopsiques et les hémianopsiques droits ne présentent pas, selon les auteurs, d'effet de longueur de mot caractéristique des dyslexiques lettre-par-lettre (même si un effet de longueur de mots plus important que les sujets contrôles était observé) [voir également Cohen et al. (2003)].

Pour les trois groupes de patients testés dans leur étude, les conditions initiales d'encodage sont les mêmes : l'information doit nécessairement être traitée par l'hémisphère droit avant d'être transférée à l'hémisphère gauche par les connections calleuses. La constatation de l'absence d'un effet de longueur de mot similaire aux dyslexiques lettre-par-lettre chez les patients hémianopsiques et alexiques hémianopsiques est un donc un argument allant contre l'hypothèse avancée ici. En effet, nous avons suggéré la possibilité que l'encodage parafovéal des mots et / ou le traitement préférentiel des basses fréquences spatiales par l'hémisphère droit entraînait la perte des fréquences spatiales optimales à la lecture (fréquence moyennes à hautes), ce qui pouvait rendre compte des problèmes de lecture rencontrés par nos patients. Or, le fait que les patients présentant les mêmes conditions d'encodage initial que les dyslexiques lettre-par-lettre ne démontrent pas d'effet de longueur de mot similaire aux dyslexiques lettre-par-lettre diminue la plausibilité de cette hypothèse.

Une autre hypothèse est que des lésions dans l'hémisphère gauche additionnelles à celles strictement nécessaires pour causer une hémianopsie droite sont en cause en ce qui a trait aux problèmes affectant l'identification des lettres. En effet, nous pouvons supposer qu'une ou plusieurs régions de l'hémisphère gauche seraient impliquées dans le traitement des fréquences spatiales optimales à la lecture. Les données anatomiques récentes semblent converger vers l'implication du gyrus fusiforme dans la dyslexie lettre-par-lettre, plusieurs des patients ayant en

commun une lésion touchant cette région (Beversdorf, Ratcliffe, Rhodes et Reeves, 1997 ; Binder et Mohr, 1992). De plus, les études par imagerie fonctionnelle démontrent qu'une région du gyrus fusiforme gauche est activée par des mots ou des stimuli visuels similaires à des mots (tels des suites de consonnes chez des lecteurs normaux (Cohen et al., 2003; Dehaene, Le Clec'H, Poline, Le Bihan et Cohen, 2002 ; McCandliss, Cohen et Deheane, 2003 ; Polk et Farah, 2002). Ceci a amené plusieurs auteurs à conclure que s'y logeait entre autres le lexique orthographique (Beauregard, Chewrtkow, Bub, Murtha, Dixon et Evans, 1997) ou l'aire visuelle spécifique à la forme des mots (visual word-form area) qui répondrait à des stimuli ressemblant à des mots (comme des suites de consonnes par exemple) (Cohen et al., 2003).

B) Modèle intégré de la dyslexie lettre-par-lettre

Les observations recueillies dans le présent travail, concernant les traitements parallèle et séquentiel chez les dyslexiques lettre-par-lettre, l'effet de confusabilité et l'implication de fréquences spatiales optimales à l'identification des lettres, nous permettent d'apporter quelques précisions sur les modèles de la dyslexie lettre-par-lettre élaborés par Behrmann et al. (1998), Saffran et Coslett (1998) et Cohen et al. (2003).

En raison de l'hémianopsie homonyme droite ou de troubles perceptifs dans le champ visuel droit, les stimuli visuels « verbaux » sont initialement traités par l'hémisphère droit. Puisque cet hémisphère ne serait pas spécialisé dans le traitement des fréquences spatiales optimales à l'identification des lettres (Ivry et Robertson, 1998), il est possible que cela entraîne la production d'un effet de longueur de mot plus élevé si les stimuli sont traités initialement par les deux hémisphères cérébraux ou par l'hémisphère gauche. Plusieurs auteurs ont observé un tel effet en

décision lexicale lorsque les stimuli étaient présentés de façon latéralisée à l'hémisphère droit de sujets neurologiquement sains (Lavidor et Ellis, 2002). Par ailleurs, les sujets hémianopsiques étudiés par Leff et al. (2001) présentaient un très léger effet de longueur de mot en lecture à voix haute (pente de 4 à 16 msec / lettre), et qui était globalement supérieur à celui observé chez leurs sujets contrôles (pente de -6 à 10 msec / lettre). Donc, un léger effet de longueur de mot pourrait relever de la limite de l'hémisphère droit à traiter les fréquences spatiales particulièrement importantes pour l'identification des lettres.

Par contre, il convient de rappeler que l'effet de longueur de mot dans l'hémisphère droit de sujets normaux ou hémianopsiques est beaucoup plus faible que dans la dyslexie lettre-par-lettre. Un autre déficit chez les patients présentant ce trouble apparaît donc nécessaire pour produire l'important effet de longueur de mot qui caractérise la dyslexie lettre-par-lettre.

Après le transfert interhémisphérique de l'hémisphère droit au gauche, l'information devrait normalement être dirigée vers une portion du gyrus fusiforme gauche (surnommée par plusieurs auteurs comme étant l'aire visuelle spécifique à la forme du mot) comme les études par imagerie chez le lecteur normal le démontrent (Beauregard et al., 1997; Cohen, Dehaene, Naccache, Lehéricy, Dehaene-Lambert, Hénaff, et al., 2000; Cohen et al., 2003; Dehaene et al., 2002; Fiez et Peterson, 1998; Polk, Stallcup, Aguirre, Alsop, D'Esposito, Detre et Farah, 2002). À la lumière de nos observations, cette structure pourrait avoir comme rôle d'extraire les fréquences spatiales optimales à l'identification des lettres, étant donné son implication dans le traitement de mots, mais aussi de stimuli visuellement similaires à des mots, comme des suites de consonnes (Cohen et al., 2003). Toutefois, cette région étant lésée chez les dyslexiques lettre-par-lettre, le mot ne peut être traité efficacement.

Les dyslexiques lettre-par-lettre ont alors recours à une stratégie compensatoire lettre-par-lettre afin d'identifier les lettres individuelles composant le mot. Par le biais de cette stratégie, l'attention est focalisée sur les lettres individuelles (Arguin et al., 2002), ce qui permettrait d'extraire les fréquences spatiales optimales à la reconnaissance des lettres. Ce processus attentionnel pourrait s'effectuer dans l'hémisphère droit, étant donné la prédominance de cet hémisphère dans le contrôle de l'attention spatiale (Gitelman, Nobre, Parrish, LaBar, Kim, Meyer et al., 1999). Alternativement, certains auteurs attribuent la capacité d'identification des lettres à une section du gyrus fusiforme droit équivalente à l'aire spécifique à la forme du mot de l'hémisphère gauche. Cette aire prendrait la relève chez les dyslexiques lettre-par-lettre et assumerait la fonction d'identifier séquentiellement les lettres individuelles (Cohen et al., 2003).

Lorsque la confusabilité entre les lettres est suffisamment réduite par le biais de la focalisation attentionnelle, l'information serait acheminée au gyrus angulaire gauche, qui aurait un rôle majeur dans la lecture (Dejerine, 1892). En effet, cette structure serait une interface transmodale entre les formes auditive et visuelle des mots et les connaissances lexico-sémantiques (Mesulam, 1998). Par contre, étant donné la lenteur relative du processus séquentiel d'identification des lettres, des effets lexicaux de haut niveau (tels la fréquence lexicale, les effets d'imageabilité, de voisinage orthographique etc.) auraient l'occasion de se manifester et pourraient contribuer à l'identification des lettres par le biais de processus de rétroaction (top-down processes) (Behrmann et al., 1998). Notons toutefois que lorsque la confusabilité des lettres composant le mot est trop élevée, certains effets de haut niveau ne peuvent se produire: en effet, une disparition de l'effet facilitateur du voisinage orthographique est observée lorsque les mots sont composés de lettres ayant une haute confusabilité (Arguin et al., 2002). Dans des conditions de basse confusabilité de lettres,

les phénomènes de lecture implicite observés chez les dyslexiques lettre-par-lettre pourraient également s'expliquer par l'accès rapide d'une information visuelle imprécise à des stades de représentations phonologiques ou sémantiques (Arguin et al., 2002). Enfin, les effets lexicaux de haut niveau de même que de lecture implicite pourraient être sous-tendus soit par l'hémisphère droit (voir Saffran et Coslett, 1998), soit par les deux hémisphères cérébraux (voir Behrmann et al. 1998).

Le modèle proposé ici permet de réconcilier les hypothèses quant à la contribution d'un traitement hémisphérique droit d'une part et la théorie unifiée de Behrmann d'autre part. Il concorde également avec les arguments suggérant la présence d'un déficit perceptif dans la dyslexie lettre-par-lettre. Toutefois, le débat sur la localisation cérébrale (i.e. hémisphère droit ou gauche ou les deux) des effets lexicaux de haut niveau observés dans la dyslexie lettre-par-lettre est loin d'être résolu et mérite de plus amples investigations. Pour résoudre ces divergences, il conviendrait peut-être d'obtenir des données anatomo-cliniques plus précises sur les patients qui présentent ce trouble de lecture, car des lésions différentes pourraient entraîner une variabilité dans la symptomatologie des patients. Des études ont déjà suggéré la présence d'une variabilité importante dans ce trouble de lecture, tant sur le plan anatomique (Damasio et Damasio, 1983) que fonctionnel (Hanley et Kay, 1996; Price et Humphreys, 1992). Nous ne croyons pas toutefois que différents modèles soient nécessaires à la compréhension de la dyslexie lettre-par-lettre, mais plutôt que des lésions anatomiques ou fonctionnelles survenant à divers stades de traitement de l'information donneraient lieu à des manifestations plus ou moins importantes de la symptomatologie (taille de l'effet de longueur de mot, présence ou absence de phénomènes de lecture implicite, taille des effets de haut niveau, etc.) de la dyslexie lettre-par-lettre.

CHAPITRE SIXIÈME:

CONCLUSION

Les observations recueillies dans le premier volet de cette thèse auprès d'un dyslexique lettre-par-lettre, IH, ont montré qu'un traitement parallèle, quoique déficient, demeure présent dans ce trouble de lecture et que ce traitement participe à l'identification explicite des mots. Toutefois, l'effet facilitateur du voisinage orthographique observé chez ce patient, qui repose sur le traitement parallèle des lettres, est aboli lorsque les mots sont composés de lettres possédant une haute confusabilité. La grande sensibilité de ce dyslexique à la confusabilité des lettres indique que l'activation lexicale résultant du traitement parallèle résiduel des lettres n'est pas optimale en raison de la présence d'un bruit élevé produit par les mots qui sont visuellement similaires à la cible, nuisant ainsi à son identification absolue. Pour cette raison, les dyslexiques lettre-par-lettre doivent recourir à une stratégie séquentielle lettre-par-lettre qui, en permettant l'allocation des ressources attentionnelles sur les lettres individuelles, réduit le problème lié à la similarité visuelle entre les lettres et permet ainsi la reconnaissance du mot. Ces observations mènent à croire, comme d'autres auteurs l'ont proposé, que ce trouble de lecture serait induit par un trouble affectant le traitement des mots avant l'activation de leurs représentations lexicales.

Dans le second volet de cette thèse, cette hypothèse dite "périphérique" de la dyslexie lettre-par-lettre a servi de base à une simulation de ce trouble de lecture chez des sujets neurologiquement sains. Les études menées ici ont indiqué que des conditions de dégradation visuelle des stimuli (réduction du contraste de luminance et retrait des hautes fréquences spatiales) peuvent produire un effet de confusabilité de lettres chez ces lecteurs, ainsi qu'une interaction entre la longueur de mot et la fréquence lexicale. Par contre, l'interaction entre le voisinage orthographique et la confusabilité de lettres observée chez le dyslexique lettre-par-lettre IH, n'apparaît pas chez la majorité des sujets normaux dans ces conditions de dégradation. Ces résultats suggèrent que

la dégradation visuelle utilisée ne reproduit pas entièrement les difficultés d'encodage des lettres présentes chez les dyslexiques lettre-par-lettre, principalement car ces derniers ont accès aux hautes fréquences spatiales qui étaient absentes des stimuli employés dans nos expériences. Nos études, à considérer comme exploratoires étant donné la rareté des travaux portant sur la simulation de la dyslexie lettre-par-lettre chez les lecteurs normaux, suggèrent toutefois des avenues intéressantes pour la reproduction de ce trouble de lecture.

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