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REVIEW

Neglect dyslexia: a review of the neuropsychological literature

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Abstract Neglect dyslexia (ND) is reviewed, based on published single-patient and group studies. ND is frequently associated with right hemispheric damage and unilateral spatial neglect (USN), and typically involves the left side of the letter string. Left-brain-damaged patients showing ND, ipsilateral (left) or contralateral (right) to the side of the left-sided hemispheric lesion, have also been reported, as well as a few patients with bilateral damage, with more frequently left than right ND. As USN, ND is temporarily ameliorated by lateralized stimulations (vestibular caloric, visual prism adaptation). ND may occur independent of USN, suggesting the damage to specific visuospatial representational/attentional systems, supporting reading. ND errors comprise omission, substitution, and, less frequently, addition of letters on one side of the stimulus, resulting in words or nonwords, also with reference to the stimulus' linguistic features. Patients with ND may show preserved lexical–morphological effects and implicit processing, up to the semantic level, of the misread string. This preserved processing is a feature of ND, shared with the USN syndrome. The mechanisms modulating error type and lexical–morphological effects are partly independent of each other. Different levels of representation of the letter string may be affected, giving rise to egocentric, stimulus-centred, and word-centred patterns of impairment. The anatomical correlates of ND include the temporo-parieto-occipital regions.

Keywords Neglect dyslexia · Unilateral spatial neglect · Right hemisphere · Visuospatial attention

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Neglect dyslexia and the neglect syndrome

Historical introduction

Patients with unilateral spatial neglect (USN), when they read passages of prose, sentences, or individual words may commit errors in the side of the stimulus (i.e., the text line, the word or letter string) contralateral to the side of the lesion (contralesional). Errors include omissions (e.g., *education* → *tion*) and substitutions (e.g., *wine* → m*ine*) (Kinsbourne and Warrington 1962b). The deficit, termed "neglect dyslexia" (ND), is a component of the USN syndrome (Vallar 1998).

ND had long been known. At the end of the XIX century, Arnold Pick (1898) described a patient (case #2), with a left hemianopia, who omitted the first (left-sided) word of each line. A postmortem examination showed encephalomalacia in the left temporal lobe and in the right thalamus, with the latter lesions being responsible for the reading deficit. Another patient (case #2), with a right frontal meningioma, shows "…a tendency to ignore objects exposed on the left side….While reading the patient preferred to hold the paper to the right or she would read on the right side of the paper." The patient, confronted with left-sided and right-sided stimuli presented simultaneously, "invariably looked at the right

object first, only later to the left" (Silberpfennig 1941). These disturbances gave an impression of hemianopia, but Silberpfennig concluded that the deficit was attentional rather than sensory. This patient is one of the few reported cases with a lesion confined to the frontal lobe. These early observations show a close association of ND with other manifestations of USN, but probably not with left hemianopia. Other early studies report reading disorders associated with left USN after right-brain damage (Brain 1941; Paterson and Zangwill 1944).

ND, USN, and hemianopia

The features of ND were clearly defined by Kinsbourne and Warrington (1962b), who, in six right-brain-damaged patients, confirmed the association of a reading disorder (characterized by left-sided substitutions, and much less frequently, additions), with left USN, but not with left visual half-field deficits. These patients are typically unaware of the ND (Kinsbourne and Warrington 1962b). Anosognosia for ND is a component of the more general anosognosia for USN, but may occur in the absence of anosognosia for hemiplegia (Berti et al. 1996).

Hemianopia is closely associated with dyslexia (Poppelreuter 1917/1990). Warrington and Zangwill (1957) described a patient (TS) with a left occipito-parietal meningioma who makes errors on the end (right side) of the word. TS does not show USN, is aware of his difficulty in reading, and reports being "unable to see" the end of words (loc. cit., p. 209). Warrington and Zangwill (1957) trace back this deficit to an uncompensated right hemianopia, with defective oculomotor adaptation (see also Shallice and Rosazza 2006, for a discussion in terms of right ND). Most patients with hemianopia show reading deficits, with omissions of letters, syllables, and short words, and meaningful, but incorrect, completions of partially seen words (guessing errors), and preserved letter-by-letter reading and spelling. The planning and guiding of reading eye movements are impaired (see Warrington and Zangwill 1957). At variance from ND, "hemianopic alexia" is related to the side of the visual field loss, rather than to spatial reference frames (see Costello and Warrington 1987, for a patient showing a dissociation between the sides of homonymous hemianopia and of ND). The term "hemianopic alexia" is used to denote the reading disturbance brought about by a left hemispheric lesion and a right-sided visual half-field deficit (Leff et al. 2006; Spitzyna et al. 2007), while Schuett et al.'s (2008) "hemianopic dyslexia" concerns the reading deficit associated with both left- and right-sided homonymous visual field losses. Patients with right hemianopia show greater reading impairments than patients with left-sided visual deficits (at least in left-to-right reading systems) (Leff and Behrmann 2008; Schuett 2009).

ND may occur independent of USN. Costello and Warrington (1987) report a right-handed patient with a left hemispheric lesion extending in the right hemisphere, who shows right homonymous hemianopia, right visual USN (in line bisection, and copy drawing), and left ND. Cubelli et al. (1991) report a right-handed left-brain-damaged patient with right visual USN, right-sided homonymous hemianopia, and left ND; in this patient, the occurrence of a reading deficit ipsilateral to the side of the lesion is accounted for in terms of "ipsilateral neglect" (Kwon and Heilman 1991). In a study including 39 right-brain-damaged patients, Bisiach et al. (1990) found two patients with severe spatial USN without ND and six patients with hemianopia with no ND. Nineteen patients show left ND; all of them have a visual field defect, but only nine exhibit left visual USN. In 120 right-brain-damaged patients, McGlinchey-Berroth et al. (1996) found by a factorial analysis that word reading is independent of left attentional processing and line bisection, although no individual patient with dissociable left ND was observed. In ten right-brain-damaged patients with left USN, Behrmann et al. (2002) found no relationship between USN and left ND. Reading accuracy is however related to the fixation of the words, and, importantly, hemianopic patients show no dyslexia in this task. In 64 right-handed patients with focal epilepsy (Schwartz et al. 1997), right-sided amobarbital injection brings about word deletions and substitutions from the left side of the line of the sentence, and within-word left ND errors (Ellis et al. 1987).

Most experimental studies are concerned with reading words and meaningless pronounceable letter strings (non-words). However, tasks requiring reading sentences and text may be more sensitive diagnostic tools (Schwartz et al. 1997) and are currently used for the clinical assessment of USN (Caplan 1987; Pizzamiglio et al. 1989; Towle and Lincoln 1991).

ND as a component deficit of USN

The conclusion that ND, though often cooccurring with USN, is likely to be a specific and independent impairment makes the disorder interesting in two respects: (a) the deficit may be investigated as a disorder of spatial representations and attention, as other aspects of USN; (b) the focus may be on the relationships with language.

In the context of USN, ND may be conceived as a higher-order impairment either of discrete spatial representations supporting perceptual awareness (Bisiach et al. 1990; Bisiach and Vallar 2000) or of spatial attention (Cubelli et al. 1991). Currently, the contraposition between attentional vs. representational accounts of USN is not a main matter of debate (Benke et al. 2004). First, it is a widely accepted view that many manifestations of USN may be interpreted both within an attentional and a

representational theoretical frame, suggesting a substantial overlap of these two theoretical constructs (Bisiach and Vallar 2000). Secondly, current multicomponent accounts of the manifold manifestations of USN allow for multiple (possibly interacting) pathological mechanisms, including, for instance, defective directed attention, and corruption, or loss, within the representational medium. Nevertheless, the representational interpretation—which qualifies USN as a spatially localized deficit of conscious perception ("the representational scotoma", see Bisiach et al. 1983, p. 36)—may readily account for both unconscious processing in the neglected side, and the finding that USN patients may exhibit not only "defective" but also "productive" pathological behaviours in the neglected side of space (Vallar 1998; Berti 2004; Vallar and Mancini 2010).

ND and the other "peripheral" dyslexias

Seen from the vantage point of the dyslexias, ND is classified as a "peripheral" dyslexia, namely, a disorder affecting the initial stages of reading. These early stages involve visual feature analysis of the letter string's individual components, which activate the appropriate letter representations in a system of abstract letter units. The process of letter identification precedes reading proper and is common to both "lexical" and "non-lexical" reading aloud. Thus, "peripheral" dyslexias are distinct from "central" dyslexias, which are characterized by damage to one or more of the components specific to the reading routines. A standard dual-route model of the reading processes is shown in Fig. 1.

ND should be distinguished from three other main types of "peripheral" reading disorders:

(a) "Visual" dyslexia (Marshall and Newcombe 1973) is characterized by visual errors (e.g., "colonel" → "colour", "pod" → "pad", errors of letter order, see Shallice and Rosazza 2006), not systematically occurring on one side of the letter string.

(b) The classical "cécité verbale" ("word blindness") (Dejerine 1892; Hanley and Kay 2003, for an historical review), "pure alexia", or "alexia without agraphia", "letter by letter reading", "global alexia", "agnosic alexia", (Cohen et al. 2003; Shallice and Rosazza 2006; Leff and Behrmann 2008), "spelling dyslexia" (Kinsbourne and Warrington 1962a) include a variety of reading difficulties, with the patients' impairment ranging from the inability to identify single letters ("global alexia"), to preserved letter identification and the development of letter-by-letter reading strategies ("pure alexia"), which are not used by "visual dyslexic" patients (Shallice and Rosazza 2006). These varieties of peripheral dyslexia are interpreted in terms of damage to a "visual word form system" (Marshall and Newcombe 1973; Patterson and Shewell 1987; Shallice and Rosazza 2006). A discussion of the neural correlates of this system (Visual Word Form Area: VWFA) may be found in Cohen et al. (2003) and Price and Devlin (2003).

(c) "Attentional" dyslexia is a rare reading disorder that shows up when more than one stimulus of the same type is present in the visual field: such patients are able to read single letters, but not a row of letters, a single word but not a row of words; text reading is also very impaired (Shallice and Warrington 1977; Warrington et al. 1993).

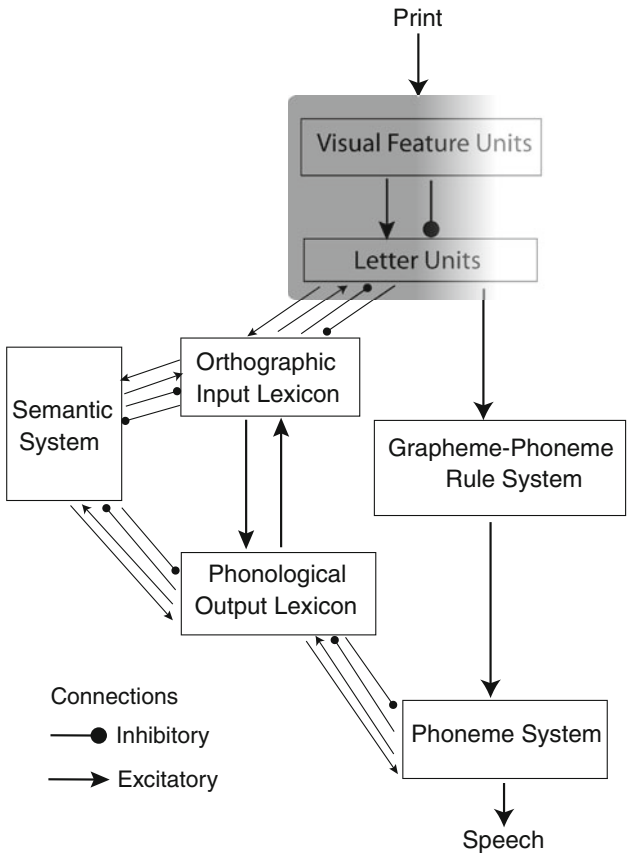

Fig. 1 The dual-route cascaded (*DRC*) model of visual word recognition and reading aloud (adapted from Coltheart et al. 2001). The grey area covering the first two stages of processing of written material denotes the locus where ND deficits occur. The early stages of processing of written letter strings are further specified in Fig. 2

A review of published patients with neglect dyslexia

Left and right ND and USN in single-patient and group studies

The present review was based on a search on the PubMed and PsychInfo databases. Single case studies are summarized in Tables 1 and 2.

Table 1 Single case reports of ND

Patient	Age/Sex	Hand	Hemispheric lesion site	VFD	ND	Error pattern of ND				Implicit	Visual USN
						Error type	WLE	LE	L-ME		
Left brain damage											
JM (Katz and Sevush 1989)	64/M	R	L-V (CT: T, O, BG, post ic)	RuQ Rle	L[a]	S	–	–	–(WF, R)	/	R
LS (Katz and Sevush 1989)	60/M	R	L-V (CT: T, P)	Re	L[a]	S	–	+	+(R)	/	/
TB (Patterson and Wilson 1990)	70/M	R	L-V (CT: PCA, Th, BG)	RH	L[a]	S	/	+	+(LC)	/	–
NG (Caramazza and Hillis 1990b; Hillis and Caramazza 1995a)	76/F	L*	L-V (CT: BG, Pwm)	–	R	S, O	+	–	+ (WF, GC)	/	R
HH (Hillis and Caramazza 1990)	57/M	R	L-V (CT: T, O, Th, post ic)	RH	R	S, O	+	–	+ (WF, R)	/	[R]
RYT (Warrington 1991)	40/M	R	L-V (CT: wm)	–	R	S, O	+	/	+(COM) –(WF, I)	/	–
AR (Cubelli et al. 1991)	33/M	R	L-V (CT: P, O)	RH	L[a]	S	–	/	/	/	R
YM (Cohen and Dehaene 1991)	58/M	R	L-N (CT: T, P, sple CC)	RuQ	L[a,c]	S	–	/	/	/	–
#1 (Binder et al. 1992)	40/F	R	L-V (MRI: O med, L sple)	RH	L[a]	S, O	/	+	/	/	–
#2 (Binder et al. 1992)	28/M	R/L	L-V (MRI: O med, L sple)	RH	L[a]	S	/	+	/	/	–
#3 (Binder et al. 1992)	30/F	R	L-V (CT: L sple)	–	L[a]	S	/	/	/	/	–
HB (Hillis and Caramazza 1995b)	65/M	R	L-V (CT: F, P, BG)	/	R	S, O, A	/	+	+(WF) –(R, GC)	/	R
Right-Brain damage											
VB (Ellis et al. 1987; Ellis et al. 1993)	81/F	L	R-V (no brain imaging)	LH	L	S	–	–	–(WF, I, R, GC)	/	L
JB (Riddoch et al. 1990)	50/M	/	R-V (CT: T, P)	LuQ	L#	S, O, A	+	+	+(WF, ON) –(I)	/	L
MO (Riddoch et al. 1990)	41/M	/	R-V (CT: T, P)	LH	L	O, S, A	/	+	+(ON)	/	–
HR (Behrmann et al. 1990)	61/M	R	R-V (CT: MCA)	LH	L	S, O, A	–	+	+ (COM)	/	L
AH (Behrmann et al. 1990)	70/M	R	R-V (CT: T, P, BG)	LH	L	S, O, A	+	+	– (COM)	/	L
RB (Hillis and Caramazza 1990)	64/M	R	R-V (CT: neg MCA)	Le	L	S, O	+	+	+ (WF)	/	L
SP (Young et al. 1991)	53/F	R	R-V (CT: MCA)	LH	L	S, O	/	/	/	/	L
RW (Hillis and Caramazza 1991)	57/F	R	R-V (CT: P, O)	Le	L	S, O, A	–	+	+(WF) –(C, R, GC)	/	L
WC (Siéroff 1991)	62/M	R	R-V (CT: P, O)	LH	L	/	–	+	/	/	L
GG (Nichelli et al. 1993)	25/M	R	R-T (CT: T, O)	LuQ	L	O	+	+	–(WF, C, I)	/	L
SH (Tegnér and Levander 1993)	84/F	R	R-V (CT: PCA, T, O)	LH	L	S	–[b]	/	–(WF)	/	L
SA (Tegnér and Levander 1993)	68/M	R	R-V (CT: MCA, T, P)	LH	L	S	–[b]	/	–(WF)	/	L
MD (Berti et al. 1994)	24/M	L	R-T (CT: T, ic, P atr)	–	L	O, S, A	/	/	/	+(ST)	L
Cub[d] (Cubelli et al. 1994)	62/M	R-L	R-V (CT: F, P, BG)	LH	L	S, O	+	–	– (WF, C)	/	L
BPN (Hillis and Caramazza 1995a)	67/M	R	R-V (CT: F, BG, T, P)	Le	L	S, O	/	/	/	/	L
JOD (Hillis and Caramazza 1995a)	73/M	R	R-V (CT: ACA, F, P)	Le	L	S, O	/	/	/	/	L
ML (Hillis and Caramazza 1995b)	59/F	L	R-V (CT: F, P, BG)	[–]	L	S, O, A	–	+	+(WF) –(C,GC)	/	L
ES (Vallar et al. 1996)	82/F	R	R-V (MRI: F, T, P, O)	LH	L	O	+	+	–(WF)	+(SA)	L
EB (Arguin and Bub 1997)	89/F	R	R-V (CT neg)	LH	L	O, S, A	/	+	+(ON)	/	L
XX (Cantoni and Piccirilli 1997)	33/F	R	R-T (MRI: ic, wm, Th)	/	L	S, A	–	+	–(WF, C, GC)	+(LD)	L
CS (Hillis et al. 1998)	66/M	R	R-V (CT: MCA)	Le	L	S, O, A	/	/	/	/	L
AS (Hillis et al. 1998)	69/M	R	R-V (CT: MCA)	Le	L	O, S	/	/	/	/	L
AWR (Hillis et al. 1998)	61/M	R	R-V (CT: MCA)	LH	L	S, O, A	/	/	/	/	L
CC (Làdavas 1998)	80/F	R	R-V (CT: F, P wm)	–	L	O	/	+	/	+(SR)	L
WV (Shillcock et al. 1998)	64/M	/	R-V (MRI: T, P)	LlQ	L	S, O, A	/	/	+(P)	/	L
MR (Subbiah and Caramazza 2000)	73/F	R	R-V (MRI: PCA, T, Th)	/	L	S, O	+	/	–(WF, R, C)	/	L
SVE (Miceli and Capasso 2001)	70/M	L*	R-V (MRI: F, P, T, ins, BG, ic, Th)	Le	L	S	–	+	–(WF, C, GC)	/	[L]
FC (di Pellegrino et al. 2001–2002)	63/M	R	R-V (CT: F, T, P)	L	L	O, S	–	+	/	/	L

Table 1 continued

Patient	Age/Sex	Hand	Hemispheric lesion site	VFD	ND	Error pattern of ND				Implicit	Visual USN
						Error type	WLE	LE	L-ME		
Bilateral damage											
JOH (Costello and Warrington 1987)	71/M	R	Bil-T (CT: L P, O, sple CC- > R)	RH	L	S, A	−	/	+(COM)	/	R
Piazza (Beschin et al. 2000)	67/M	R	Bil-V (CT, MRI: L-P, O; R:Th)	−	R	O, S	+	/	/	/	R external L imaginal
RR (Haywood and Coltheart 2001)	75/F	/	Bil-V (CT: L-T, P)	BilH	L	S	−	+	+(LC) −(WF)	/	−
MC (Savazzi 2003)	59/F	R	Bil-V (CT: R-F, T, P) [L-T, P]	−	L	O, S, A	+	/	/	/	L
RCG (Arduino et al. 2005)	72/M	R	Bil-V (MRI: T, P, atr wm)	RuQ	L	S, O	−	+	/	/	−
Developmental deficit											
NT (Friedmann and Nachman-Katz 2004)	9/M	R	Dev	+	L	O, S, A	−	−	−(GC)	−(LD)	−

M/F male/female, / not reported or assessed, +/− present/absent, R/L right/left, Bil bilateral, [] mild deficit/small lesion

USN unilateral spatial neglect, *ND* neglect dyslexia

Hand handedness, *(corrected), R/L ambidextrous

CT/MRI computerized tomography/magnetic resonance imaging, *V/N/T* aetiology (vascular, neoplastic, traumatic), *Dev* developmental deficit, *ACA/MCA/PCA* anterior/middle/posterior cerebral artery vascular territory, *F/P/T/O* frontal/parietal/temporal/occipital lobe, *ins* insula Reyli, *BG* basal ganglia, *Th* thalamus, *ic* internal capsule, wm: white matter, *CC* corpus callosum, *sple* splenium of the CC, *atr* atrophy, *post* posterior, *med* medial, *neg* negative

VFD visual field deficit, *H/Q* homonymous hemianopia/quadrantanopia, *u/l* upper/lower quadrant, *e* extinction to double simultaneous stimulation

ND error types, *S/O/A* substitutions/omissions/additions; the left-to-right order of error types denotes their frequency in the patient's error corpus

WLE word length effect, with ND being more severe with longer letter strings

LE lexicality effect, *L-ME* lexical and morpholexical effects (*WF* written word frequency effect, *R* ortho-phonological regularity, *GC* grammatical class, *C* concreteness, *I* imageability, *ON* orthographic neighbourhood, *COM* compounds, *P* palindromes, *LC* lexical constraints)

Implicit processing tasks: *LD* lexical decision, *SA* semantic association, *SR* semantic reading, *ST* Stroop

ND only with limited time exposure of the stimulus

[a] ND ipsilateral to the side of the lesion (ipsilesional)

[b] Trend

[c] ND involving numbers, with no ND for letter strings

[d] The beginning letters of the first author's name (Haywood and Coltheart 2000)

Table 2 Published single-patient reports of ND by side of the lesion or aetiology and side of ND

Side of ND	Side of the hemispheric lesion			Developmental patients	Total
	Left	Right	Bilateral		
Left	8[a]	28	4	1	41
Right	4	0	1	0	5
Total	12	28	5	1	46

[a] Ipsilesional

The majority of the 45 reported brain-damaged patients shown in Table 1 have right hemispheric lesions (28 out of 45, 62%), 12 patients have left hemispheric lesions (27%), and five a bilateral damage (11%). ND is contralesional (left-sided in right-brain-damaged patients, and right-sided in left-brain-damaged patients) in 32 out of the 40 patients with unilateral damage (80%), and ipsilesional in eight patients (20%), all with left-brain damage. Considering the side of ND independent of the side of the lesion, and including the one developmental person, the deficit is left-sided in 41 out of 46 patients (89%) and right-sided in five patients (11%). In some bilateral patients with left ND, lesions are more extensive in the right hemisphere (Costello and Warrington 1987; Beschin et al. 2000).

Four out of the 12 left-brain-damaged patients (33%) show right visual USN, and all but one (patient MO: Riddoch et al. 1990) 28 right-brain-damaged patients exhibit left USN. Also, group studies show that ND and USN are often associated, involving the left side of space, and resulting from a right hemisphere lesion (Brunn and Farah 1991, eight USN patients; Chatterjee 1995, five USN patients; Takeda and Sugishita 1995, seven USN-ND patients; Làdavas et al. 1997a, 23 USN-ND patients; Arduino et al. 2002, six USN-ND patients; Rusconi et al. 2004, 12 USN-ND patients, one showing only ND; Savazzi et al. 2004, 31 patients, with 17 showing USN, and ND in the experimental task; Cubelli and Beschin 2005, six USN-ND patients; Stenneken et al. 2008, 16 USN-ND patients). In the study by Lee et al. (2009), USN was found in 80 out of 138 (58%) right-brain-damaged patients, while ND was present in 31 (22.5%) patients (of the 80 USN patients with USN, the frequency of ND was 37.5%, since one of the 31 ND patients did not show USN). Although visual left USN and

left ND are often associated, right-brain-damaged patients with left visual USN may not show left ND, which, in turn, may occur with no evidence of USN (Bisiach et al. 1990; Rusconi et al. 2004, one patient with ND without USN; Lee et al. 2009).

A few patients show dissociations between ND and USN with respect to the affected side of space (the above-mentioned patients reported by Costello and Warrington 1987; Cubelli et al. 1991; one brain-damaged patient in the series of Lee et al. 2009, who shows left ND, and left USN in copying, but right USN in line bisection). One patient with a bilateral lesion exhibits right ND and visual USN, but left USN for imaginal objects (Beschin et al. 2000). Finally, one 9-year-old child shows a left developmental ND, with neither associated USN nor detectable brain damage (Friedmann and Nachman-Katz 2004). The dissociation between USN and ND is supported by the finding of a complete recovery of ND, with a persistence of visual USN (patient AB, Cantoni and Piccirilli 1997, Table 1).

Lexical effects in reading aloud

ND offers the opportunity to investigate how visuospatial attention and lexical access interact, by assessing the effects of lexical and morpholexical variables on the reading performance of ND patients. Several explanations have been proposed as to how stored lexical knowledge may serve to compensate for the attentional deficit (Caramazza and Hillis 1990a; Patterson and Wilson 1990; Brunn and Farah 1991; Siéroff 1991). One such account simply assumes that ND patients are more likely to infer, or guess, the neglected portion of a word than of a nonword, due to the orthographic constraints of written language (Patterson and Wilson 1990). In the multistage model of word recognition proposed by Caramazza and Hillis (1990a), lexical effects may arise at each of three reference frames [retino-, stimulus-, and object- (word, graphemic)-centred], where impairments may occur. The accurate encoding of the letter identities of the non-neglected portion of a word may be sufficient to address its internal lexical representations adequately, allowing correct identification and reading. Several patients with ND have been described within this theoretical framework (see Haywood and Coltheart 2000, for a review).

Another interpretation of lexical effects in ND, inspired by McClelland and Rumelhart's (1981) interactive activation model of word recognition, rests on the assumption that the activation of lexical representations results in a facilitatory feedback to lower-order units responsible for letter encoding, thus leading to an improved identification of letters, provided they are part of a word. A number of investigators have interpreted the lexical effects observed in ND patients along these lines (see Sieroff et al. 1988; Behrmann et al. 1990; Brunn and Farah 1991).

How stored lexical knowledge may interact with the attentional deficit may be assessed by the *lexicality effect*, whereby words are read better than nonwords (see, e.g., Pagliuca et al. 2008). A number of single-patient studies focus on this issue, showing that most ND patients exhibit the effect (see Table 1). Furthermore, when lexical and morphological psycholinguistic variables are manipulated, for some patients their effects in reading are comparable to those found in neurologically unimpaired participants: high-frequency words are read better than low-frequency words (*word frequency effect*), and nouns better than verbs and adjectives (*grammatical class effect*). Moreover, accuracy is influenced by the number of orthographic word neighbours (*orthographic neighbourhood or N-size effect*, also referred to as *lexical constraints effect*), concrete words are read better than abstract ones (*word concreteness effect*), and regularly spelled words better than irregular words (*word regularity effect*). The presence of morphological constituents in the stimulus (i.e., roots, suffixes, and word constituents in compounds) guides and constraints the patients' reading performance. Palindromes (i.e., words that remain the same when written straight and reversed, e.g., *noon*) are read better than non-symmetrical words (Shillcock et al. 1998).

Fewer studies have considered ND for numbers. Kinsbourne and Warrington (1962b) report that their right-brain-damaged patients show left ND for both letter and number strings. The prevailing error patterns are different, however, substitutions for words and omissions for numbers, suggesting that discrete functional components may be involved. One left-brain-damaged patient (YM: Cohen and Dehaene 1991) exhibits an error pattern, akin to 'positional' dyslexia (Katz and Sevush 1989), in reading the left-most digit of any number, showing an ipsilesional deficit. Other patients show left ND for both words and numbers (patient VB, Ellis et al. 1987; patient TB, Patterson and Wilson 1990; patient RR, Haywood and Coltheart 2001; patient RCG, Arduino et al. 2002).

Also, most of the group studies focus on the sensitivity to the lexical status of the target. Similar to the evidence from single-patient investigations, a number of group studies have shown that ND patients produce less errors in reading words than nonwords (Brunn and Farah 1991; Chatterjee 1995; Arduino et al. 2002; Rusconi et al. 2004; Cubelli and Beschin 2005; Stenneken et al. 2008), and are sensitive to lexical variables, showing the word frequency effect (Arduino et al. 2002; Stenneken et al. 2008) and an effect of word stress location. As for stress, the limited available evidence shows that the presence of an accent mark [e.g., *papa* (*pope*), *papà* (*dad*)] may improve word reading in right-brain-damaged patients with left ND, so

acting as a cue for lexical access (no effect was found for nonwords), although the accent mark effect is not present in all patients (Cubelli and Beschin 2005). In another study in right-brain-damaged patients with left ND, the location of stress in the target word is preserved in the paralexic response (Rusconi et al. 2004).

A few group studies do not report the presence of significant lexical effects, such as word compounding (Takeda and Sugishita 1995). In the multiple-single-patient study by Arduino et al. (2002), only two out of the six left ND right-brain-damaged patients do not show lexical effects, and their performance is overall unaffected by the morphological structure of the stimulus.

Error type

ND patients produce different types of errors including *substitutions* (Kinsbourne and Warrington 1962b; Ellis et al. 1987; Tegnér and Levander 1993), *omissions* (Takeda and Sugishita 1995; Làdavas et al. 1997b; Lee et al. 2009) and, less frequently, *additions* (see different patterns of error type in left ND in Arduino et al. 2002). A widely used measure for evaluating error type was introduced by Ellis et al. (1987): left ND errors consist of "errors in which target and error words are identical to the right of an identifiable neglect point in each word, but share no letters in common to the left of the neglect point" (loc. cit. p. 445). Examples of ND errors (from the error corpora of Ellis et al. 1987; Arduino et al. 2002) are as follows: (a) letter *substitutions* [*boat* → c*oat*, *albero* (the Italian word for *tree*) → po*bero* (a nonword resulting from the substitution of the two initial letters of the target word)]; (b) letter *omissions* [*fate* → *ate, famiglia* (*family*) → *miglia* (*miles*, a word resulting from the omission of the two initial letters of the target word)]; letter *additions* [*owl* → b*owl, luna* (*moon*) → mo*luna* (a nonword resulting from the addition of two letters to the left of the target word)]. Responses that do not meet these criteria may be classified as non-ND errors. According to the lexical status of the response, a distinction may be drawn between real-word and nonword ND errors (Ellis et al. 1987). A similar, though more lenient, criterion for substitutions is that the target and the response (i.e., the paralexic error) may share some letters occupying the left portion of the string (Hillis and Caramazza 1990). A substitution error [namely, a "backward completion error" according to Hillis and Caramazza's (1990) terminology] is a response satisfying the following criteria: (a) it is identical to the target by at least two letters from the right end, (b) it includes at least one unshared letter on the left, (c) it does not contain two or more letters in the same relative order left of the shared portion. One example is *ration* → cre*ation*. Omission errors, termed by Caramazza and Hillis (1990a, b) "neglect errors" are responses identical to the target on the right, but omitting all letters to the left of the shared portion, such as *ration* → *ion*, as previously proposed by Ellis et al. (1987).

The relative proportion of the error types varies across patients. As illustrated in Table 1, in some of the reported single-patient studies, substitution errors predominate, in others there is a prevalence of omissions. Similarly, group studies have shown that patients vary according to the error type. In some patients, omissions of the initial left-sided letters or characters predominate (Takeda and Sugishita 1995; Làdavas et al. 1997b; Savazzi et al. 2004; Cubelli and Beschin 2005), while in others substitutions are the most frequent errors (Kinsbourne and Warrington 1962b; Tegnér and Levander 1993). In line with these findings, one multiple single-case study (Arduino et al. 2002) shows that different patients may commit more substitutions (e.g., 91%, patient PP), more omissions (e.g., 88%, patient AA), and a comparable number of these neglect error types (e.g., 44% substitutions vs. 56% omissions, patient CI); addition errors are infrequent (less than 1%).

Finally, ND error rate may be higher with longer letter strings, as the contralesional bias in line bisection increases with line length (see Anderson 1999, for a computational model; e.g., see Vallar et al. 2000), or no effect of length may be present (see Table 1). One study in seven Japanese right-brain-damaged patients with left USN and ND (Takeda and Sugishita 1995) shows that the length effect is greater when patients read from left to right than when they read from right to left (in Japanese, horizontally written words can be written from left to right, or from right to left, maintaining the meaning of the word without mirror-reversed characters).

A related, but independent (Tegnér and Levander 1993), parameter concerns the relationship between the length (in letters) of the stimulus and that of the paralexic response. These two lengths may be related (e.g., Ellis et al. 1987; Miceli and Capasso 2001), with a substitution error type, suggesting that the position of the misread letters is encoded by the patient. These effects may be modulated by lexical factors (Tegnér and Levander 1993).

Error type, lexical effects, and the severity of ND

The relationships between these factors are a controversial issue. For example, patient VB (Ellis et al. 1987) produced the same percentage of errors in reading words and nonwords and her more frequent errors were substitutions. Conversely, patient EB's reading performance was affected by the lexical properties of the stimulus (e.g., neighbourhood size), but her most common errors were omissions (Arguin and Bub 1997). Patients HR and AH made more substitution errors, and their performance was affected by lexical factors (Behrmann et al. 1990). Patients JB and MO

showed lexical effects on reading, but the former produced more substitutions, the latter more omissions (Riddoch et al. 1990). Different combinations between error type and lexical effects in reading are possible, suggesting that the pattern of error type and lexical effects in ND reflect discrete impaired and preserved mechanisms. One study has shown some association between substitution errors and the presence of lexical effects in reading on the one hand, and between omissions and the absence of lexical effects on the other hand (Arduino et al. 2002). A prevalence of omissions may reveal a more severe attention disorder, whereas a prevalence of substitutions may indicate a less severe deficit, with the presence of the letter being encoded (Ellis et al. 1987; Behrmann et al. 1990; Arduino et al. 2002).

In a study in Hebrew right-brain-damaged patients, who read from right to left, and show left ND for the (left) end of words, the reading performance is affected by letter form: words with final-form letter (that can be located only at the end of the word) bring about less addition errors than letters that can be located in all positions of the letter string. These findings suggest a modulation of the reading performance by orthographic information present in the neglected side (Friedmann and Gvion 2005).

In the theoretical frame of "early" (unattended information is discarded prior to stimulus identification) vs. "late" (unattended information undergoes lexical-semantic processing before stimulus identification) selection views of spatial attention (review in Umiltà 2001), the presence vs. absence of lexical effects in ND may be traced back to functional deficits at early ("non lexical ND", in which no lexical effects are present) versus late ("lexical ND", in which the reading performance is affected by the lexical status of the stimulus) processing stages. This account implies that these two ND patterns reflect discrete functional deficits, in accord with multicomponential accounts of USN (Vallar 1998). There are, however, models that include both early and late selection mechanisms, where peripheral and central factors need not to be mutually exclusive (Van der Heijden et al. 1984; Pashler and Badgio 1985; Navon 1989). One such computational model is MORSEL (Mozer and Behrmann 1990; Behrmann et al. 1991), which integrates a word recognition system [similar to McClelland and Rumelhart's (1981) Interactive Activation Model] with an attentional mechanism, with high-level knowledge interacting with perceptual processing. In MORSEL, the visual input brings about a pattern of activity on a "retina", which feeds a hierarchy of processing levels in the Letter and Word Recognition System (BLIRNET). The output of the BLIRNET is processed by a Pull Out Network, supported by lexical-semantic units, which selects, through letter cluster units, sets of clusters representing single items. The model includes an attentional mechanism (AM), which controls the flow of information through the BLIRNET, and constructs a spotlight centred on different regions of the "retina". The AM receives inputs from a variety of sources, collectively termed "Higher Levels of Cognition". ND results from a damage of the bottom-up connections from the input feature map of BLIRNET to the AM. This, in turn, affects the modulatory effects of the AM on later processing stages of the BLIRNET. The damage is graded monotonically, most severe at the left extreme of the retina and least severe at the right, simulating left ND.

Following MORSEL, ND may be regarded as resulting from a single functional impairment, which manifests itself with different degrees of severity. In this way, a less severe attentional disorder may be expected to produce a prevalence of substitutions and lexical effects in reading, whereas a more severe attentional disorder would cause a prevalence of omissions and no lexical effects in reading aloud (see the related account by Ellis et al. 1987, who interpret substitution errors and the preservation of the stimulus length in the paralexic response in terms of spared positional encoding). However, as discussed previously (see also the following section on "implicit" processing in ND), the omission versus substitution error patterns found in ND patients appear to be overall unrelated to the preservation versus absence of lexical effects. The double dissociation between ND and USN indicates in any case that the concept of "severity", namely, the "graded damage to the bottom up Attentional Mechanism inputs" (Mozer and Behrmann 1990, p. 118) is to be ascribed to the reading domain only (Arduino et al. 2002).

Dissociation between "explicit" and "implicit" reading processing

The account of USN as a disorder of spatial awareness (Bisiach et al. 1979; Berti 2004) implies that, also in the domain of reading, evidence for unconscious processing (see Berti 2002, for review) may be gathered from the performance of USN patients. In both single-patient and group studies, the performance of ND patients has been investigated, contrasting the "explicit" task of reading aloud the stimulus with a variety of "implicit" paradigms, which probe the patients' knowledge about the visually presented letter string and do not involve reading.

These implicit tasks include lexical (words vs. nonwords, see Cantoni and Piccirilli 1997; Làdavas et al. 1997b; Arduino et al. 2003; Stenneken et al. 2008) and semantic decision [living vs. non-living decision, see Làdavas et al. (1997a, b)], semantic reading (i.e., making semantic decisions and reading the letter strings immediately afterwards: Làdavas et al. 1997a), and association (i.e., production of a word associated to the target: Vallar et al. 1996), and a Stroop task (Berti et al. 1994). A consistent result is that ND patients show a better performance when

their processing of the words they misread is assessed by an implicit task. These findings provide converging evidence (see also "Lexical effects in reading aloud" and "Error type") that lexical-semantic processes are largely preserved in ND patients. The ND patients' reading errors are unrelated to their performance in the tasks mentioned earlier, which do not involve reading aloud (Arduino et al. 2002, 2003), in line with the view that ND, as USN, may be primarily qualified as deficits of perceptual awareness (Vallar and Daini 2006; Vallar and Mancini 2010).

The suggestion has been made that spatial attention may play a role at some stage of prelexical computation, necessary for reading, whereas a more central attentional resource is involved in semantic access (Duncan 1987; Yantis and Johnston 1990; Carr 1992; McCann et al. 1992). This view is in accordance with the hypothesis that in right-brain-damaged patients with left ND, the dissociation between reading and semantic tasks (e.g., lexical decision, semantic association) arises as a consequence of the differential involvement of spatial coordinate frames, possibly relying on differences in the output demands of the two tasks. Reading aloud includes, in addition to the contribution from the lexical system, some activation of the non-lexical phonological route. The two routes are supposed to be activated in parallel in both word and nonword reading, with the relatively stronger contribution of each being dependent on several factors, among which the degree of transparency of a given orthography (Zevin and Balota 2000; Coltheart et al. 2001). The non-lexical phonological route may be more dependent on the preservation of a representation of the letter string, arranged—in orthographies such as Italian (e.g., Arduino et al. 2002), English (e.g., Ellis et al. 1987), and Korean (Lee et al. 2009)—in a spatial, left-to-right fashion, in orthographies such as Hebrew from right to left (Friedmann and Nachman-Katz 2004), and in Japanese in both directions (Takeda and Sugishita 1995). Implicit tasks, by contrast, may be solely based on the orthographic whole word-form representation of the stimulus, with no need for the sequential visuospatial and phonological processing required for reading aloud. In sum, these differences in the processes involved in the two types of tasks may account for the dissociation between the defective reading aloud vs. the less impaired or largely preserved performance in tasks such as lexical or semantic decision.

The relationships between ND and USN

The effects of physiological stimulations and of rehabilitation

A variety of lateralized or directionally specific physiological stimulations may temporarily ameliorate (or worsen) a number of manifestations of USN (Vallar et al. 1997; Rossetti and Rode 2002; Kerkhoff 2003). These effects involve also left ND in right-brain-damaged patients, as shown by caloric vestibular stimulation (Silberpfennig 1941; Rubens 1985), and prism adaptation (Farnè et al. 2002; Angeli et al. 2004), suggesting that the impairment of left ND concerns spatial representations functionally similar to those affected in USN.

A number of rehabilitation studies, using behavioural training to enhance the exploration of the left-hand side of space (Pizzamiglio et al. 1992, 2006), and prism adaptation (Frassinetti et al. 2002; Fortis et al. 2010) have shown that the improvement of the USN syndrome in right-brain-damaged patients includes left ND.

The reference frames of ND and of USN

The different manifestations of the USN syndrome may occur in two main spatial reference frames (see a general review concerning spatial reference frames in Lacquaniti 1997; Vallar 2003): (a) *egocentric*, where the neglected side is defined with reference to the patients' body, with the investigated divide being most frequently the mid-sagittal plane of patient's trunk; (b) *allocentric* or *object-based*, with the relevant divide being the centre of the object, independent of its position with reference to the patient's body (Walker 1995; Halligan et al. 2003; Vallar 2003). Patients with USN may show egocentric, allocentric and mixed patterns of impairment (Heilman and Valenstein 1979; Calvanio et al. 1987; Ota et al. 2001; Marsh and Hillis 2008). ND, in the light of a canonical orientation for reading of the letter string that constitutes a word or a pronounceable nonword (left to right in languages such as English, French, German, Italian, Korean; right to left in Hebrew) offers a unique opportunity to investigate patterns of impairment related to the damage to the different frames in which a letter string is encoded.

ND may be due to the damage of three different levels of representation of the letter string (Hillis and Caramazza 1995a; Haywood and Coltheart 2000). These discrete levels of representation, and patterns of impairment of ND, develop early work by Marr (1982), who distinguishes, after early processing of the visual image (*primal sketch*), a viewer-centred coordinate frame (*2½ D sketch*), and an object-centred coordinate frame (*3D model representation*).

ND in a viewer-centred, egocentric, spatial reference frame, such as the mid-sagittal axis of the body, the head, or the fixation point

This pattern, defined, with reference to the visual system, as *retinocentric* (Hillis and Caramazza 1995a; Haywood and Coltheart 2000), is exemplified by patients such as JOD

(Hillis and Caramazza 1995a), CS, AS and AWR (Hillis et al. 1998), JB (Riddoch et al. 1990), HR and AH (Behrmann et al. 1990). The main feature of this pattern is that errors occur in the contralesional side (the left in all reported patients) of the letter string, with reference to a viewer-centred reference frame. In reading words presented in columns on a page, error rate decreases from left to right positions with respect to the body, and less errors are made in reading words presented in the right than in the left half-field (patient JOD: Hillis and Caramazza 1995a; patients CS, AS, AWR: Hillis et al. 1998). No ND errors are reported in vertical reading (HR and AH: Behrmann et al. 1990; JOD: Hillis and Caramazza 1995a; patients CS, AS, AWR: Hillis et al. 1998). ND occurs on the left physical side of 180° anticlockwise-rotated words, which in this condition includes the final letters of the word (patient JB, see Riddoch et al. 1990). Patient MO (Riddoch et al. 1990) is classified by Haywood and Coltheart (2000) as showing a retinocentric left ND, but the lack of data from vertical and 180° anticlockwise-rotated words prevents definite conclusions. Hillis et al. (1998) attempt at disambiguating a trunk- vs. head- vs. visual field-referred ND, misaligning the position of the trunk, of the head, and of the stimulus. The finding that the percentage of left-sided neglect errors made by patient AWR does not vary suggests a viewer-based defective reference frame (within AWR's right preserved visual half-field), head- and trunk-based frames being relatively uninvolved.

In a viewer-based frame, increasing the spacing between letters (WORD vs. W O R D) is expected to worsen ND (see Hillis et al. 1998; see also Haywood and Coltheart 2000, who assign spacing effects only to stimulus-centred deficits), as the extension of the stimulus is increased (a similar effect occurs in line bisection, see, for example Vallar et al. 2000, and references therein), with the patients' attention being further pathologically directed rightwards. Such spacing effects have been found in patients JOD (Hillis and Caramazza 1995b), CS, AS, and AWR (Hillis et al. 1998), and AH (Behrmann et al. 1990).

ND in an allocentric, object-based, or stimulus-centred reference frame

The defining feature of this pattern of ND is that the patients' performance on the neglected side of the letter string is relatively unaffected by its spatial position with respect to the body, as well as by tasks in which a digit printed just to the left of the to-be-read word is to be reported first (3*WORD*). Under these conditions, the letter string lies in the right-hand side of the egocentric reference frame or to the right of fixation in the 3*WORD* paradigm. The finding that these manipulations do not affect left ND supports the view that a stimulus-centred frame is affected (see also Kinsbourne and Warrington 1962b, for early evidence; patient VB: Ellis et al. 1987; patient SP: Young et al. 1991; patient Cub: Cubelli et al. 1994, who shows a paradoxically better reading performance when words are presented in the left-hand side of space; patient BNP: Hillis and Caramazza 1995a). In line bisection, cueing by requiring patients to report digits or letters placed on the left side of the line is either ineffective (Heilman and Valenstein 1979) or, as found by more investigators, able to diminish the rightward bias (Riddoch and Humphreys 1983; Nichelli et al. 1989; Mennemeier et al. 1997).

The stimulus-centred deficit shares with the viewer-centred deficit a preserved vertical reading (patient VB, text: Ellis et al. 1987; patient BNP, individual words: Hillis and Caramazza 1995a) and the finding that spacing worsens ND (patient RW: Hillis and Caramazza 1991; patient WC: Siéroff 1991; patient BNP: Hillis and Caramazza 1995a).

Deficits confined to the first letter of the word (patients JM and LS: Katz and Sevush 1989; patient TB: Patterson and Wilson 1990; Binder et al. 1992, three patients whose errors are confined to the first letter in 90% of the trials) may be considered as concerning a stimulus-centred representation (Haywood and Coltheart 2000). However, the finding that the deficit for the first letter is present also with vertical presentation (patient JM, Katz and Sevush 1989; no reliable data for patient TB, see Patterson and Wilson 1990) suggests that this "positional" (Katz and Sevush 1989) reading deficit may be unrelated to USN (see Haywood and Coltheart 2000, for further discussion). In line with this conclusion, these patients show no evidence of USN in the side (left) of ND.

The suggestion has been made that the affected stimulus-centred representation is shared by both words and objects (Subbiah and Caramazza 2000), but see Haywood and Coltheart (2000) for a different view. Patients may show left ND (with a positional deficit for the first letter), but no USN for left-sided shapes (patient TB, see Table 1, Patterson and Wilson 1990), and left facial USN, but no left ND (right-brain-damaged patient KL, see Young et al. 1990). These findings suggest independent representations.

Mixed viewer/stimulus-centred patterns of impairment

In some patients, the deficit may be traced back to a combination of impairments. One defining feature of a stimulus-centred deficit is its independence of the position of the stimulus with respect to viewer-centred frames. By contrast, viewer-centred deficits are influenced by the position of the stimulus with reference to the affected frame (body, head, retina). For instance, right-brain-damaged patient JOD (Hillis and Caramazza 1995a) shows a viewer-centred deficit, being more accurate with words presented for 200 ms in the right half-field (44%) than in the left

half-field (8%). However, performance in the right half-field is far from flawless. The residual right-sided deficit may reflect an associated stimulus-centred impairment. Conversely, patients CS and AWR (Hillis et al. 1998) make fewer reading errors in the right half-field (CS: left 33%, right 7%; AWR; left 60%, right 3%), suggesting that the stimulus-centred component is marginal.

Similarly, in the 3*WORD* task patient GG (Nichelli et al. 1993) still makes ND errors, indicating a stimulus-centred impairment. Error rate (neglect errors 56%) is however lower than in the standard WORD condition (90%), suggesting an associated deficit at a viewer-centred level (Haywood and Coltheart 2000). The original interpretation was in terms of an only partially preserved ability to direct attention towards the neglected side (Nichelli et al. 1993), rather than of reference frames.

Patient SP (Young et al. 1991), previously discussed as suffering from a stimulus-centred deficit, reading sets of 15 words in different positions, makes whole word omissions only in the left-hand side of the display (indicating a deficit of a body-centred frame), but ND errors are comparable in the five left-to-right columns (indicating a stimulus-centred deficit). Young et al. (1991) distinguish defective leftward attention towards the whole page (i.e., in an egocentric coordinate frame), responsible for whole word omissions, and towards the individual word, responsible for misreadings, namely, neglect errors (see a similar pattern in patient RW: Hillis and Caramazza 1991; Haywood and Coltheart 2000, for a discussion). Patient VB (Ellis et al. 1987) exhibits a similar performance reading a passage of text.

Patient WC (Siéroff 1991) may suffer from a viewer-centred or stimulus-centred deficit, or both: reading in the left half-field could not be assessed (due to the presence of left hemianopia), and different positions of the letter string in the right half-field were not investigated, in order to rule out possible position effects in a viewer-centred coordinate frame, within the unaffected field (Haywood and Coltheart 2000).

A word-centred or graphemic reference frame

This pattern involves the canonical (for the purpose of reading) representation of the letter string. ND concerns the neglected side (namely, letters), independent not only of the position of the stimulus with respect to the body but also of the arrangement of the letter string (standard, vertical, mirror-reversed). At variance from the previous patterns, spacing of the string does not affect ND (Hillis and Caramazza 1995a; Hillis et al. 1998; Haywood and Coltheart 2000). This pattern is illustrated by left-brain-damaged patients NG (Caramazza and Hillis 1990a), HH (Hillis and Caramazza 1990), HB (Hillis and Caramazza 1995b), and, possibly, RYT (Warrington 1991; see Miceli and Capasso 2001), for whom the relevant available data are however confined to vertical reading (see Haywood and Coltheart 2000, who do not consider this patient as suffering from a graphemic deficit). Right-brain-damaged patients include ML (Hillis and Caramazza 1995b) and SVE (Miceli and Capasso 2001). The deficit of right-brain-damaged patient RB (Hillis and Caramazza 1990) is classified as graphemic by Haywood and Coltheart (2000), but not by Miceli and Capasso (2001): in this patient, neither vertical reading nor spelling was assessed (Hillis and Caramazza 1990).

A graphemic deficit may be hypothesized in patients showing contralesional errors in spelling, both forward and backwards (Baxter and Warrington 1983, right-brain-damaged patient ORF, with mild ND and no other evidence of visual USN, who made spelling errors in the beginning, leftward part of words; Barbut and Gazzaniga 1987, right-brain-damaged patient JL, with a similar deficit). Contralesional spelling errors were found also in patients NG (Caramazza and Hillis 1990b; Hillis and Caramazza 1995a), HB, and ML (Hillis and Caramazza 1995b).

The graphemic error pattern involves one side of the canonical letter string (Caramazza and Hillis 1990b; Miceli and Capasso 2001), and in this respect it may be considered a manifestation of USN, and spatially based. This pattern should be distinguished from the positional impairments, involving exclusively, or mainly, the first letter (see "ND after left-brain damage").

Interestingly, these patients, particularly when the damage affects the right hemisphere, are left-handed: ORF (Baxter and Warrington 1983), JL (Barbut and Gazzaniga 1987), ML (Hillis and Caramazza 1995b), SVE (Miceli and Capasso 2001), left-brain-damaged patient NG (Caramazza and Hillis 1990b; Hillis and Caramazza 1990, 1995a).

Finally, the three patterns of ND errors discussed earlier may be not mutually exclusive, but they may be elicited by different conditions of presentation of the stimulus: canonical, rotated, mirror-reversed, seen through a mirror (Savazzi 2003).

Anatomical correlates of neglect dyslexia

Early studies suggest a main role of temporo-parietal damage (Kinsbourne and Warrington 1962b). Some of the more recent group studies are broadly consistent with these conclusions. A gross localization by the affected lobes and deep structures indicates that lesions involving the temporo-parietal-occipital regions are present in more that 50% of the patients (Takeda and Sugishita 1995; Làdavas et al. 1997a, b; Arduino et al. 2002; Behrmann et al. 2002; Rusconi et al. 2004; Stenneken et al. 2008), while lesions confined to the frontal lobe are much infrequent. This pattern is clear in the

studies by Takeda and Sugishita (1995), with six patients showing temporo-parieto-occipital lesions and one a fronto-temporo-parieto-occipital damage, and by Stenneken et al. (2008), with 13 out of 16 patients showing lesions involving the temporo-parieto-occipital regions. In line with these conclusions, a perusal of Table 1 shows no patient with damage confined to the frontal lobe (but see the early report by Silberpfennig 1941).

More precise evidence comes from a series of 156 Korean right-brain-damaged patients (Lee et al. 2009). USN patients have lesions involving the superior and middle temporal gyri, the inferior parietal lobule, and the posterior insular cortex, while ND patients have additional lesions in the lingual and fusiform gyri. These findings are in line with the behavioural dissociations discussed earlier, suggesting that ND is a specific component of the USN syndrome, brought about by posterior damage. This lesion localization is also in line with the higher percentage of left visual half-field deficits in ND USN patients, when compared with USN patients without ND, and, importantly, may overlap with the possible anatomical site of the VWFA in the left hemisphere (Cohen et al. 2003), further corroborating the specificity of ND, in the context of the USN syndrome. The preservation of the implicit effects in the processing of letter strings by patients with left ND (see 'Dissociation between "explicit" and "implicit" reading processing') may be based on mechanisms involving such regions, suggesting a neurofunctional dissociation between the representation of the VWFA in the left hemisphere, and the spatial attentional system damaged by the right hemispheric lesion in patients with ND.

A neurofunctional framework for neglect dyslexia

The data summarized in Tables 1 and 2 indicate, first of all, a definite lateral asymmetry of ND, which, in 41 out of 46 published reports (89%) involves the left-hand side of the letter string. The reading deficit is contralateral to the side of the lesion in all 28 right-brain-damaged patients, and is left-sided in four out of the five patients with bilateral lesions, as well as in the developmental patient.

Left ND after right-brain damage

Considering that all group studies concerning ND involve right-brain-damaged patients with left-sided ND, the number of patients with a putative right-sided ND is proportionally even lower, being confined to the 12 patients summarized in Tables 1 and 2. The conclusion may be therefore drawn that left ND is a component deficit of the syndrome of USN, with its primary pathological determinant being a defective conscious representation of (or faulty orientation of spatial attention towards) the contralesional side of the stimulus. In line with this view, ND patients show evidence of unconscious processing of the neglected letter string (see Table 1), as repeatedly found for other manifestations of the USN syndrome (Berti 2002). Furthermore, the lateralized sensory stimulations which modulate a number of manifestations of the USN syndrome affect left ND in a similar fashion (Rubens 1985; Farnè et al. 2002; Angeli et al. 2004; Fortis et al. 2010). The left-sided lateralization of ND is likely to reflect the general asymmetry of spatial representation and attention. The right hemisphere possesses a bilateral representation of space, while the left hemisphere is mainly concerned with the right-hand side of it (Bisiach and Vallar 2000; Mesulam 2002). This asymmetrical representation would result in the more frequent occurrence of a left-sided deficit, as it is indeed the case. Linguistic deficits are not the primary determinant of left ND, while linguistic factors modulate the patients' reading performance (see Table 1). In Japanese, horizontally written words can be written from left to right, or from right to left, maintaining the meaning of the word without mirror-reversed characters, but the reported patients have right-sided lesions and a left-sided ND (Takeda and Sugishita 1995). In Hebrew, words are read from right to left, but, again, ND concerns the left side of the word in right-brain-damaged patients (Friedmann and Gvion 2005).

In most right-brain-damaged patients with left ND, the impairment appears to involve the left-hand side of the physical stimulus, in egocentric and object-based frames, or both. A "word-centred" deficit, with left-sided errors, independent of the physical presentation of the letter string (canonical, 180°-rotated clockwise, mirror-reversed), was found in two right-brain-damaged patients: ML (Hillis and Caramazza 1995b) and SVE (Miceli and Capasso 2001). The fact that both patients are left-handed—as right-brain-damaged patients ORF (Baxter and Warrington 1983) and JL (Barbut and Gazzaniga 1987), who show contralesional errors in spelling—on the one hand supports the view that this pattern of impairment involves language-specific representations, on the other hand prompts a cautionary note as to the possibility of non-typical patterns of neurofunctional organization of the reading processes and of their impairments.

ND after left-brain damage

The evidence is limited to 12 single-patient studies, with no group studies being available.

In eight patients, ND is ipsilateral to the side of the hemispheric lesion, concerning the left-hand side of the letter string. In three patients, the deficit involves the initial (mainly the first, see Katz and Sevush 1989; Binder et al. 1992) or only the first (Patterson and Wilson 1990) letter of the string. This initial-letter deficit extends to vertical

presentation in patient JM reported by Katz and Sevush (1989), suggesting a graphemic deficit. Conversely, patient #1 of Binder et al. (1992) makes no initial-letter errors in the vertical condition, indicating a viewer-centred impairment that extends to the initial numeral of multidigit strings. These deficits have been interpreted as "positional" (Katz and Sevush 1989) or "position-specific" (Patterson and Wilson 1990), rather than a manifestation of USN, with which they share only the phenomenon of the laterality of the deficit.

Binder et al. (1992), who use the term "left hemiparalexia", discuss their findings in terms of a retinotopic disconnection, due to damage to the splenium of the corpus callosum. The deficit is "positional" involving the first letter of the string. One of their patients (case #3) is also unable to read words presented in the left visual half-field, showing a deficit broadly similar to the "left hemialexia" exhibited by patients with posterior callosal (splenial) damage. Patients with left hemialexia, however, typically read normally with time-unlimited presentation of the stimuli, showing a deficit only with brief exposure in the left visual half-field, which prevents ocular exploration (Siéroff and Lavidor 2007).

A standard neurofunctional model of word reading (Dejerine 1892; Cohen et al. 2003) postulates that visual information is initially processed by the occipito-temporal areas contralateral to the stimulated hemifield, being subsequently transferred to the VWFA, localized in the left inferior occipito-temporal region, and specifically devoted to the processing of letter strings (Cohen et al. 2003; but see Price and Devlin 2003). For stimuli displayed in the left visual field, this transfer proceeds from the right to the left hemisphere, through the posterior portion of the corpus callosum (Cohen et al. 2000). Accordingly, damage to this region brings about a reading impairment for letter strings briefly presented in the left visual half-field/right hemisphere.

The left reading deficit of the patients reported by Binder et al. (1992, "left hemiparalexia") differs from "left hemialexia" in two important respects: first, it is present under conditions of free vision, with unlimited exposure time, and second, it is confined to the first letter of the string. In sum, "left hemiparalexia" may reflect a "positional" functional impairment, rather than a callosal disconnection (as suggested by Binder et al. 1992). Accordingly, these three patients are listed in Table 1. However, it should be noted that for the "positional" account, no neurological interpretation is available.

The main difference between these left-sided (but ipsilesional) deficits and left ND after right-brain damage consists in the fact that the deficit of the latter involves the impaired processing of left-sided letters, with a right-to-left gradient of severity, that may differ across patients (Mozer and Behrmann 1990). The ipsilesional "positional" deficit, by contrast, concerns one slot in the letter string, namely, the first one. Patient #1 of Binder et al. (1992) reports the presence of the initial letter (showing therefore no USN for it) but is unable to read it aloud, indicating a position-specific deficit. A similar argument may be applied to patient YM (Cohen and Dehaene 1991), whose deficit is confined to the leftmost digit of any number. None of these patients shows ipsilesional USN (Kwon and Heilman 1991; Kim et al. 1999), which is contralesional (right-sided) in patient JM (Katz and Sevush 1989). The pattern of the left-sided reading disorder of left-brain-damaged patient AR (Cubelli et al. 1991) is similar to that of left ND after right-sided brain damage: an interpretation in terms of ipsilateral USN (Kwon and Heilman 1991) might then be considered.

However, the observation that all reported patients with ipsilesional deficits have left-sided lesions suggests a linguistic impairment of the encoding of letter position in the string (see Patterson and Wilson 1990, for one such model), rather than a deficit of mechanisms of spatial attention, such as MORSEL (Mozer and Behrmann 1990). Broadly in line with this conclusion, patient AR (Cubelli et al. 1991) shows a left ND, but a right visual USN.

Patients NG, a corrected left-hander (Caramazza and Hillis 1990b; Hillis and Caramazza 1995a), HB (Hillis and Caramazza 1995b), and HH (Hillis and Caramazza 1990) show a word-centred right ND. One patient, RYT (Warrington 1991), shows a right ND deficit broadly similar to left ND, which may possibly reflect a damaged word-centred representation (but see Haywood and Coltheart 2000; Miceli and Capasso 2001).

In sum, the pattern of left-brain-damaged patients includes the following: (a) ipsilesional deficits, which may be positional, with respect to the letter string, and linguistic in nature, rather than related to USN; (b) right word-centred ND.

ND after bilateral brain damage

Four out of five patients with bilateral lesions exhibit left ND, further supporting the main role of right hemispheric damage in bringing about the disorder: JOH (Costello and Warrington 1987), RR (Haywood and Coltheart 2001), MC (Savazzi 2003), and RCG (Arduino et al. 2005). Patients RCG (Arduino et al. 2005) and RR (Haywood and Coltheart 2001) show a stimulus-centred deficit. Only in patient Piazza (Beschin et al. 2000) ND occurs in the right-hand side of letter strings.

Conclusion

Figure 2 illustrates a neurofunctional simplistic model which includes the three processing stages of a letter string proposed by Hillis and Caramazza (1995a), and subsequently confirmed by the review of Haywood and Coltheart (2000). The hemispheric asymmetry of ND,

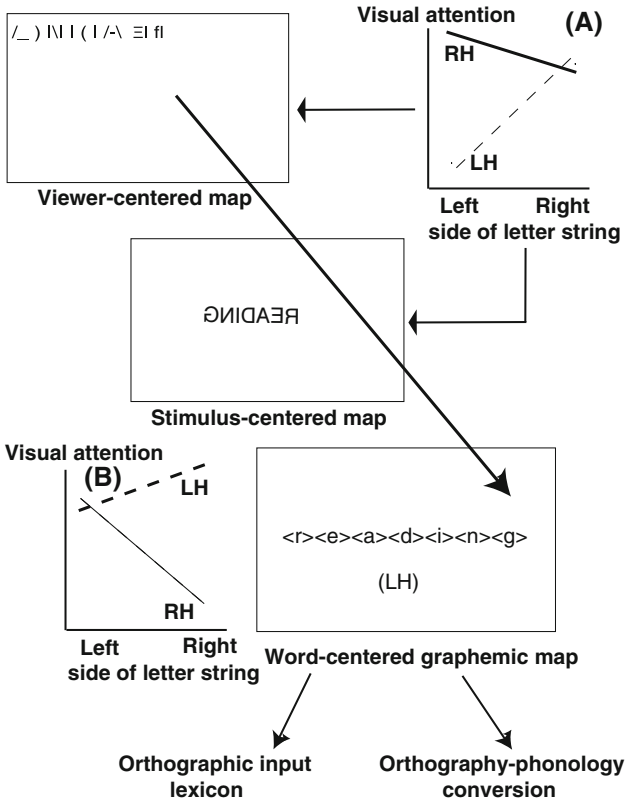

Fig. 2 A model of ND including three levels of coordinate frames of a letter string (viewer-, stimulus-, and word-centred) (Hillis and Caramazza 1995a) and two hemispheric patterns of the lateral distribution of spatial attention/representation. LH/RH: left/right hemisphere

which is more frequent after right-brain damage and involves the left-hand side of the letter string, is explained as the asymmetry of USN in general (see pattern A of the hemispheric distribution of spatial attention in Fig. 2), with the right hemisphere possessing a bilateral representation of extra-personal space (with a milder contra-ipsilateral gradient), and the left hemisphere being concerned mainly with the contralateral right-hand side of space (Bisiach and Vallar 2000; Mesulam 2002). The model draws a distinction between representations of the letter string (processed in the visual areas, see Cohen et al. 2003) and visuospatial attention, supporting perceptual awareness of it. The main neural correlates of this attentional system include the premotor frontal and the posterior–inferior parietal regions, the temporo–parietal junction, and their connections (Vallar 2001; Committeri et al. 2007; Doricchi et al. 2008; Verdon et al. 2010), with a main role, for the reading processes, of the posterior areas. This account applies to viewer-centred and stimulus-centred types of ND, which are brought about by right hemispheric lesions, and, as USN in general, involve the left-hand side of the letter string. Dissociations between these two forms of ND may be accounted for hypothesizing specific attentional systems for these two coordinate frames. These conclusions are in line with the wide evidence suggesting a multicomponent structure of spatial attention and its impairments (Vallar 1998; Vallar and Maravita 2009; Vallar and Mancini 2010).

In the case of word-centred ND, the graphemic map is likely to be mainly lateralized to the left hemisphere, at least in patients with a typical cerebral functional organization (see also Hillis and Caramazza 1989). Let us assume that the organization of the attentional system devoted to the word-centred representation is reversed with respect to spatial cognition in general, as discussed earlier, with the left hemisphere being concerned with both sides of the graphemic space, as shown in pattern B) of Fig. 2. Were this the case, word-centred USN would be caused by left-brain damage and involve the right-hand side of the letter string, since the right hemispheric spatial attention resources devoted to the ipsilateral right side of the string are much limited. Illustrative patients are NG (Caramazza and Hillis 1990a), HH (Hillis and Caramazza 1990), and HB (Hillis and Caramazza 1995b).

Conversely, in right-brain-damaged patients, who exhibit non-typical patterns of hemispheric lateralization, as for left-hander or corrected left-hander patients, the neural correlates of the graphemic map may be lateralized to the right hemisphere, with the standard pattern of hemispheric organization of spatial attention [(A) in Fig. 2], namely, a bilateral, panoramic distribution for the right hemisphere and a prevailingly contralateral (right-sided) orientation for the left hemisphere. Accordingly, in these patients, the graphemic deficit would be left-sided, as USN in general. Illustrative left-hander right-brain-damaged patients are ML (Hillis and Caramazza 1995b) and SVE (Miceli and Capasso 2001). The relative rarity of the disorder may reflect the frequent concomitant presence of damage to left hemisphere-based components of the reading system, including the VWFA and beyond. These additional deficits may mask a selective ND disorder in most patients.

Finally, the few reported left-brain-damaged patients with an ipsilesional deficit may be accounted for in terms of a positional, graphemic-related, impairment. An interpretation in terms of ipsilesional USN seems unlikely, since the deficit of these patients is confined to the positional (first-letter) reading impairment.

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