Grey scales uncover similar attentional effects in homonymous hemianopia and visual hemi-neglect

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Abstract

Multi-component models of visual hemi-neglect have postulated that visual hemi-neglect is characterised by various attentional deficits. A grey scales task has been developed to quantify the early, automatic, (perhaps obligatory) ipsilesional orienting of visual attention, frequently assumed as the first of these attentional deficits. Explanations for this attentional imbalance are up until now mainly formulated in terms of right hemisphere activation. This lateral attentional bias has also been demonstrated in controls, in whom it is expressed as a leftward perceptual asymmetry. We reproduced previous literature findings on a grey scales task, considering controls and neglect patients. Three patients with neglect showed an extreme ipsilesional lateral bias. This bias did not change during or after cognitive rehabilitation. Additionally, we presented this grey scale task to 32 patients with left- and right-sided homonymous hemianopia (HP). HP is the loss of sight in one visual hemi-field. The HH patients had no clinical signs of impaired lateralised attention. Results revealed that HH patients showed a similar ipsilesional bias, albeit to a lesser degree than in neglect. Left-sided HH patients presented a quantitatively similar, but qualitatively opposite bias than the right-sided HH patients. We suggest that sensory effects can be an alternative source of attentional imbalance, which can interact with the previously proposed (right) hemispheric effects. This suggests that the perceptual asymmetry in the grey scales task is not necessarily an indicator of impaired right hemisphere attention. It rather suggests a pattern of functional cerebral asymmetry, which can also be caused by asymmetric sensory input. © 2002 Elsevier Science Ltd. All rights reserved.

Keywords: Attentional imbalance; Perceptual bias; Hemispheric specialisation; Laterality

1. Introduction

Several authors (e.g. [11,16,23]) suggested that the clinical syndrome of unilateral visual spatial neglect (UN) can be described/explained as a series of successive attentional events beginning with (1) an early, automatic, chronic, perhaps obligatory, orienting of attention toward the ipsilesional half space, followed by (2) a deficit in disengaging attention from that side in order to reorient it toward the contralateral half space. In addition to these two deficits, (3) a generalised (i.e. directionally non-specific) reduction in attentional-information processing capacity is assumed. The first component underlies an anomalous lateral preference. The second component gives rise to the clinical signs of UN (e.g. left-sided omissions on cancellation tasks).

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[23]. Karnath [16] proposed that this second component (reorienting) recovers faster than the other two, and this has been confirmed by several authors (e.g. [23,28]). Mattingley et al. [23] concluded that the apparent recovery of UN constituted of the restitution in reorienting of attention, but that the early ipsilesional orienting remained. They further postulated this (residual) attentional bias to be characteristic of right hemisphere dysfunction, and posed that it could be predictive of persistent neglect-type behaviours.

This attentional bias has been demonstrated in right hemisphere patients, not only using RT paradigms (e.g. [3]), but also under more naturalistic free viewing conditions. It has been demonstrated using several indexes and tasks. Gainotti et al. [11] operationally defined it as a “position preference”, namely as the tendency to identify first (and consistently) those parts of a composite diagram lying on the right or on the left of its centre. As a result of the early, automatic orienting of attention, UN patients frequently start scanning.
on the right side of a given composite stimulus (i.e. show a rightward bias). A further frequently used index expressing this lateral orienting bias is an asymmetry index (AI) derived from mainly paradigms using chimeric stimuli. For example, Mattingley et al. [23,24] concluded that this lateral preference is expressed (in UN) by a tendency to choose or prefer the right side of a composite image (rightward bias). In a face-matching task by Mattingley et al. [23], subjects were required to indicate which of two asymmetrical composites (one composed of the two left halves of an original face, the other composed of the two right halves) more closely resembled the (inherently asymmetrical) original. Patients with UN tended to judge the faces composed of the two right halves as more similar to the original one than the face composed of two left halves (rightward bias). In another chimeric faces task, presented by the same authors, patients were required to judge which face of a given pair appeared “happier”. The faces were composed of two half-faces of the same person, one half smiling, the other in a neutral expression. In one pair, the smiling face was on the left, in the other on the right. Again, UN patients tended to judge the face with the “right-smile” as happier. This rightward bias was also demonstrated using grey scales [24]. In this task, the patient was required to compare two vertically aligned rectangular bars and indicate which one appears overall darker. The bars consisted of scales of semi-continuous shades of grey, ranging from white on one end to black on the other. Both bars were identical, but mirror-reversed. Patients with UN tended to choose the bar which was black on the right side as the darker one.

Lateral biases have also been demonstrated in healthy subjects using identical or comparable paradigms (e.g. [21,23,24,27]). Contrary to patients with UN, healthy subjects exhibit a significant leftward bias. Since this bias is displayed by healthy subjects, and hence is considered to be “normal”, it is often termed as a “perceptual asymmetry” instead of a “bias” (which suggests deviation from normality).

This left perceptual asymmetry in healthy subjects has been demonstrated using face-stimuli (judgements of emotions, similarity, and femininity) [21,23,24], using grey scales (e.g. [24]), and using stimuli asking for comparisons of dot numerosity and roundness (e.g. [21]), and size (e.g. [27]). The leftward bias occurs in all these tasks in more or less comparable intensities. Despite of the similar levels of perceptual asymmetry, only low to modest intercorrelations are observed. Nicholls et al. [27] suggest that these tasks do not index one single common factor, but tap a set of attentional processes, some of which are overlapping, and others which are task-specific. The community is suggested to consist in the common right hemisphere involvement.

Summarising the explanations provided in the literature, in healthy subjects the lateral bias is explained as the result of more right hemisphere activation due to the visuo-spatial nature of the stimuli [21–23,28]. It is argued that the differential activation of the right hemisphere generates a bias of attention to the left hemispace, creating an attentional imbalance. In UN patients, the lateral bias results from disturbed right hemisphere function. It is suggested [19,20,23,24] that each hemisphere controls a contralaterally directed attentional vector. Damage to one hemisphere results in dysfunction of the associated vector and gives rise to an ipsilesional bias. In all accounts, the perceptual asymmetry is explained in terms of functional cerebral asymmetry and more specifically in terms of differential attentional right hemisphere activation. One other alternative account was proposed by Nicholls et al. [27]. They suggested the possibility that the asymmetry may be related to effects of directional scanning. In support of this proposal, they report a study by Sakhuja et al. [29] who found that readers of Hindi (left-to-right) showed the expected leftward bias, whereas readers of Urdu (right-to-left) showed the opposite bias. Nicholls and colleagues argue that the preferred directional scanning habit may lead to an over-representation of one side (i.e. ipsi-directional) of the stimulus and hence can influence the nature of the perceptual asymmetry. This conceptualisation, namely as a lateralised over-representation, also can be interpreted as an attentional account. It suggests an alternative nature or cause of attentional imbalance.

In our opinion, further alternative causes of the attentional imbalance cannot be ruled out on the basis of previous experiments. Mattingley et al. [23] demonstrated that patients who no longer showing classical signs of UN, continued to show the ipsilesional attentional bias. The authors interpreted the persisting ipsilesional attentional bias in terms of a higher-order attentional right hemisphere dysfunction. However, five of the 13 patients also had visual field defects (VFDs), i.e. either homonymous hemianopia (HH) or quadranopia. Hence, the observed residual (group-) effects in terms of the bias) could be attributable, not to a higher-order right hemisphere attentional problem, but alternatively to effects of the (lower-order) left-sided VFDs.

It is well recognised that visuo-spatial perception can be impaired in “pure” hemianopic patients (i.e. in patients with HH and without UN) [39]. Hemianopic patients have been reported to show impaired visuo-spatial exploration, especially in the hemianopic hemi-field [40]. Also a deviated subjective midline or subjective straight-ahead in visuo-spatial judgements has frequently been reported (e.g. [28,18]). Karnath and Feser [17] discuss reports which show that misperception of horizontal space (hemimicropsia) exists in (some) pure hemianopic patients. It is thus apparent that a homonymous VFD can give rise to lateralised visual impairments. Hence, it is not inconceivable that HH, which results inherently in a chronic differential lateralised visual input, also gives rise to an imbalance in processing efficiency of the visual space. We thus suggest that an attentional imbalance is not necessarily the result of a higher-order attentional right hemisphere dysfunction, but also can arise by the presence of a lower-order VFD.

It is hence our aim to investigate what is or can be the cause of the attentional imbalance resulting in the observed lateral biases. As argued, hemispheric specialisation for
visuo-spatial processing, hemispheric specificity with respect to directional attentional vectors and reading habits or scanning direction have been suggested as underlying mechanisms. We investigate if homonymous VFDs (i.e. HH), resulting in asymmetric visual input, can also be added to the list of mechanisms or factors producing attentional imbalance. If so, it should do so both in left-sided and right-sided HH, but in opposing directions (i.e. both contralaterally to the side of the VFD). If this is confirmed, previous explanations of the attentional imbalance stressing exclusively higher-order right hemisphere involvement may have to be revised.

2. Method

2.1. Participants

2.1.1. Controls

Sixty-three control subjects participated in this study (25 females, 38 males). All participants were naive as to the aims and expected outcomes of the study and reported to be right-handed. They all had normal or corrected-to-normal visual acuity. Their mean age was 47 years, ranging from 17 to 86 years.

2.1.2. Patients

Prior to testing, we administered a screening battery to exclude dementia [5,9], aphasia [7] and apraxia [6]. No impairments were found. All patients performed within the normal limits on the form discrimination screening test [36] confirming perceptual functions to be adequate for form discrimination. The nature and extent of the VFD was determined using the Humphrey Field Analyzer, which is a clinically widely used automated perimeter. We used the Full Field 246, age corrected, 3-zone strategy, screening program.

In order to identify patients with severe UN, we constructed a battery of clinical UN tests, namely, four clinical cancellation tasks, and a line bisection task. For Albert’s line cancellation test the cut-off score is two omissions [13,35]. For the Mesulam structured shape cancellation this was three omissions [38], for the Bells test four omissions [12,35], and three omissions on the search for Os. This last unstructured cancellation task is not publically available, but very frequently used for diagnostic purposes in The Netherlands. Also the line bisection task was scored as a function of omitted lines (cut-off = 2) [30,31,34].

For each task, we additionally imposed more stringent criteria. This was done in order to make a distinction between a general inattention deficit resulting in a general scanning deficit, and hemi-inattention resulting in a lateralised scanning deficit. We therefore imposed an additional “lateralisation-requirement”, namely that for a “UN-score” (as opposed to a “general attention deficit-score”) the difference between left-sided and right-sided omissions should also be equal to or exceed the cut-off score. For example, if the cut-off score for a particular test is three omissions, a UN-score is obtained only if also the number of omissions on either side exceeds the other side by at least three. Two left-sided omissions and one right-sided omission hence would not result in a UN-score, although it is indicative of a general attention and scanning deficit.

We decided that using this battery and cut-off criteria, a patient is considered to suffer severe UN if at least three (of maximally five) UN-scores are obtained and if these scores are identical in laterality (i.e. reach the lateralisation-requirements of the respective tests due to omissions on the same side).

2.1.3. UN patients

Three patients were classified as UN patients using our criteria. They were all males and suffered a right-sided stroke, resulting in UN and left-sided HH. One patient underwent extensive clinical rehabilitation in a clinical setting before participating, but the UN persisted. The other two patients were referred by their ophthalmologists because of “peculiar visual behaviour”. Their mean time since lesion was 16 months. Their visual acuity and contrast sensitivity were within normal limits. Their mean age was 64 years. Additional clinical information is provided in Table 1. On average they omitted 13 items (S.D. = 9) on the Albert’s line cancellation test, 23 items (S.D. = 22) on the Mesulam structured shape cancellation, 17 items (S.D. = 9) on the Bells test, 17 items (S.D. = 14) on the Search for Os, and three lines (S.D. = 3) on the line bisection task.

2.1.4. HH patients

Thirty-two patients with HH participated in this study. Their mean age was 51 years. The mean time since lesion was 55 months (S.D. = 80). Sixteen patients had left-sided HH (16 males, 2 females). Sixteen patients had right-sided HH (11 males, 5 females). All patients had normal or corrected-to-normal visual acuity and normal contrast sensitivity. For additional clinical data, see Table 1. None of these patients fulfilled the aforementioned UN criteria. Neither of them had ever been treated for or diagnosed with UN. They omitted no items on the Albert’s line cancellation test and on the line bisection task, on average three items (S.D. = 9) on the Mesulam structured shape cancellation, three items (S.D. = 4) on the bells test, and one item (S.D. = 3) on the search for Os.

2.2. Stimuli

We used grey scales as described in Mattingley et al. [24]. Our version contains 26 items. An item consists of an A4 landscape orientation white sheet of paper with two vertically aligned rectangular grey scales of equal lengths. A grey scale is a rectangular bar with a thin black border (see Fig. 1). Its dimensions are 20 mm in height and 20–260 mm in width with 20 mm increments. This rectangular is filled-in by a semi-continuous scale of different grey shades varying...
Table 1
Clinical data for the brain-damaged subjects

<table>
<thead>
<tr>
<th>S. no.</th>
<th>Age/gender</th>
<th>TSL*</th>
<th>Type of HH and macular sparing</th>
<th>Location* and cause* of lesion</th>
<th>Other remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50/M</td>
<td>34</td>
<td>C–I, no</td>
<td>T-O-P IC, CVA</td>
<td>Extensive clinical rehabilitation</td>
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<tr>
<td>2</td>
<td>74/M</td>
<td>7</td>
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<td>O dS, CVA</td>
<td>Left leg hemiparesic</td>
</tr>
<tr>
<td>3</td>
<td>70/M</td>
<td>7</td>
<td>C–C, no</td>
<td>O-P, CVA</td>
<td>Left hemiparesic</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Right-sided brain damage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left-sided HH group</td>
<td>Right-sided brain damage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>76/M</td>
<td>13</td>
<td>I–I, yes</td>
<td>T-O-P, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>2</td>
<td>69/M</td>
<td>12</td>
<td>I–C, yes</td>
<td>O-P, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>3</td>
<td>53/M</td>
<td>12</td>
<td>I–I, yes</td>
<td>O-P, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>4</td>
<td>56/M</td>
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<td>O, CVA</td>
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</tr>
<tr>
<td>5</td>
<td>49/M</td>
<td>18</td>
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<td>O-T. tumour</td>
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<tr>
<td>6</td>
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<td>O, CVA</td>
<td>Left hemiplegia</td>
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<tr>
<td>7</td>
<td>36/F</td>
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<td>Left hemiplegia</td>
</tr>
<tr>
<td>8</td>
<td>56/F</td>
<td>157</td>
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<td>O-T, CVA</td>
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<tr>
<td>9</td>
<td>73/M</td>
<td>6</td>
<td>I–L, yes</td>
<td>O-P, CVA</td>
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<tr>
<td>10</td>
<td>31/M</td>
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</tr>
<tr>
<td>11</td>
<td>70/M</td>
<td>13</td>
<td>I–C, no</td>
<td>O, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>12</td>
<td>34/M</td>
<td>64</td>
<td>I–C, yes</td>
<td>O-P-F, CHI</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>13</td>
<td>54/M</td>
<td>24</td>
<td>I–C, yes</td>
<td>T-O-P Th, CVA</td>
<td>Left hemiplegia, agnosia</td>
</tr>
<tr>
<td>14</td>
<td>53/M</td>
<td>11</td>
<td>C–C, no</td>
<td>O, CVA</td>
<td>Letter-by-letter reading</td>
</tr>
<tr>
<td>15</td>
<td>37/M</td>
<td>12</td>
<td>I–C, yes</td>
<td>O, CVA</td>
<td>Letter-by-letter reading</td>
</tr>
<tr>
<td>16</td>
<td>67/M</td>
<td>47</td>
<td>C–C, no</td>
<td>O-T, CVA</td>
<td>Left hemiplegia</td>
</tr>
</tbody>
</table>

Right-sided HH group

<table>
<thead>
<tr>
<th>S. no.</th>
<th>Age/gender</th>
<th>TSL*</th>
<th>Type of HH and macular sparing</th>
<th>Location* and cause* of lesion</th>
<th>Other remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>390</td>
<td>C–C, yes</td>
<td>O, tumour</td>
<td></td>
</tr>
<tr>
<td>2</td>
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<td>37</td>
<td>C–C, yes</td>
<td>O, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>3</td>
<td>50/M</td>
<td>28</td>
<td>I–I, no</td>
<td>T-O-P, CHI</td>
<td>Word finding difficulties</td>
</tr>
<tr>
<td>4</td>
<td>39/F</td>
<td>142</td>
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<td>O, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>5</td>
<td>66/F</td>
<td>123</td>
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<td>O, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>6</td>
<td>18/M</td>
<td>225</td>
<td>I–I, yes</td>
<td>O-P, hydrocephalus</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>7</td>
<td>43/F</td>
<td>60</td>
<td>C–C, yes</td>
<td>O-T, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>8</td>
<td>52/M</td>
<td>6</td>
<td>I–C, yes</td>
<td>Nl, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>9</td>
<td>64/F</td>
<td>10</td>
<td>I–C, yes</td>
<td>O-T, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>10</td>
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<td>32</td>
<td>I–C, yes</td>
<td>Nl, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>11</td>
<td>48/F</td>
<td>11</td>
<td>I–C, yes</td>
<td>O-P, CVA</td>
<td>Left hemiplegia</td>
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<tr>
<td>12</td>
<td>53/M</td>
<td>22</td>
<td>C–C, yes</td>
<td>O-T, CVA</td>
<td>Left hemiplegia</td>
</tr>
<tr>
<td>13</td>
<td>56/M</td>
<td>14</td>
<td>I–I, yes</td>
<td>O-T-Th, CVA</td>
<td>Left hemiplegia, blindsight</td>
</tr>
<tr>
<td>14</td>
<td>68/M</td>
<td>25</td>
<td>I–I, no</td>
<td>T-O-P, CVA</td>
<td>Word finding difficulties</td>
</tr>
<tr>
<td>15</td>
<td>24/M</td>
<td>63</td>
<td>C–C, no</td>
<td>Nl, CHI</td>
<td>Word finding difficulties</td>
</tr>
<tr>
<td>16</td>
<td>57/M</td>
<td>3</td>
<td>C–C, no</td>
<td>O, CVA</td>
<td>Word finding difficulties</td>
</tr>
</tbody>
</table>

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* Time since lesion in months.
* Complete (C) vs. incomplete (I)—congruent (C) vs. incongruent (I) homonymous hemianopia.
* O: occipital, T: temporal, P: parietal, F: frontal, Th: thalamus, IC: internal capsule, oC: optic chiasm, dS: diffuse subcortical damage, Nd: no abnormalities detected on CT, and Na: no CT available.
* CVA: cerebrovascular accident and CHI: closed head injury.
* Patient refused to give permission for scan inspection. Localization is based on clinically motivated assumption and verbal description.

Fig. 1. Example of an item in the grey scales task. Upper and lower bar are identical but mirror-reversed.

between black and white at the extremes. This filling-in is achieved by defining 33 strips of different grey shades. The width of these band is adjusted according to the length of the rectangular. Grey scales are thus presented in pairs (and vertically aligned) so that one grey scale is identical to, but the mirror reverse of, the other. Each item is presented once with top/bottom position counterbalanced, resulting in 26 items.

2.3. Procedure

A booklet containing the 26 items is placed and remains in front of the subject at reading distance. The subject is
asked to judge which of the two grey scales appears overall darker. The choice is indicated by saying “top” or “bottom” after which the page is turned and the next item is presented. The subject is encouraged to make a judgement based upon spontaneous and immediate apprehension rather than on prolonged and detailed inspection but is told that there is no time limit and hence can view freely. Most patients responded fluently and confidently. Many controls, on the other hand, felt they were making arbitrary choices.

In addition to this standard procedure, on a second occasion, we asked the UN patients to touch the left side of each bar, prior to judging, to ascertain the perception of the full length of the bars.

2.4. Scoring

Scoring is achieved as in Mattingley et al. [24]. For each stimulus, a response is defined as left-bias or right-bias, respectively, if the subject chose the grey scale with the black side on the left and right side, respectively, as the darker one. The asymmetry index (AI) was calculated as the number of items with a rightward bias, minus the number of items with a leftward bias, divided by the total number of items. This AI varies between −1 and +1, representing an extreme leftward and rightward bias, respectively. An AI of zero indicates no bias.

3. Results

We firstly checked whether we were able to replicate previous findings with control subjects. The mean AI was −0.3370 (S.D. = 0.4304) which is significantly different from zero (t(62) = −6.215, P < 0.0005). This confirms a significant leftward bias in control subjects. Secondly, we confirmed the extreme lateral bias displayed by our three UN patients. All AIs were equal to one (mean = 1, S.D. = 0), also on the second occasion, when both left ends of the bars had to be touched.

We then performed a one-way ANOVA, with both left-sided and right-sided HH groups and control subjects as a between-subjects (group) factor. This revealed a significant group effect (F(2, 92) = 40.757, P < 0.0005). The mean AI for left-sided hemianopic patients was 0.6317 (S.D. = 0.3725) and for right-sided hemianopic patients −0.5417 (S.D. = 0.3967). Post-hoc comparisons with Bonferroni correction revealed the HH groups to differ from each other (t(30) = 8.6, P < 0.0005) and the left-sided HH patients to differ from the control group (t(77) = 8.2, P < 0.0005). There was no significant difference between the right-sided hemianopic patients and control subjects (t(77) = −1.7, ns). The patients with UN were not included in the ANOVA analysis because of the low number of patients and the absence of variation in their AIs. To test whether the AIs by the left-sided HH patients significantly deviated from the AIs by the UN patients, we performed a one sample T-test on the data by the left-sided hemianopic patients with the AI from the UN patients (i.e. 1) as test value. This analysis revealed a significant difference (t(15) = −3.956, P < 0.001). With the same type of analysis but with the absolute value of the AI by the right-sided HH patients as the test value, we confirmed that the strength of the AI by both HH groups did not differ from each other (t(15) = 0.966, ns).

In the control group, we found no effects of educational level, nor of age. However, in the pooled HH-group, the effect of age was marginally significant as indicated by a Pearsons correlation of age with the absolute value of the AI (r(32) = 0.338, P < 0.059). Further, time since lesion proved to correlate significantly with the absolute value of the AI (r(32) = −0.456, P < 0.05). Time since lesion and age did not correlate in this sample (r(32) = −0.283, ns). None of the measures of the clinical UN battery correlated significantly with the absolute value of the AI.

We further had the opportunity to test 15 HH (seven left-sided and eight right-sided) patients on two different occasions (1 week interval, same standard procedure). The AIs on both occasions correlated significantly (r(15) = 0.968, P < 0.0005), and a paired T-test comparison showed no significant difference (t(14) = −1.662, ns) between the means.

4. Discussion

We replicated previous findings confirming (left) perceptual asymmetries under free viewing conditions in control subjects. Our AI (−0.337) clearly is in line with the AI reported by Mattingley et al. [24] using similar grey scales (−0.323). It is also well within the range of other AIs, using different types of chimeric stimuli ranging from −0.208 to −0.450 [21,23,24,27]. In controls, we found no effect of age, nor of educational level, suggesting the lateral bias to be a fairly robust phenomenon.

We secondly observed an extreme right-sided bias (AI = 1) in patients with UN. At first hand, our AIs might appear to be more extreme than those reported by Mattingley et al. [24] (AI = 0.849 for the grey scales). However, the authors report that four (of the 12 right-sided brain damaged) patients did not have UN. Removing those four patients from their results would increase their observed AI, since three of the four lowest scores on the grey scales are by a non-UN patient. Not including these non-UN patients would result in all AIs (except one) to be above 0.9.

One of our patients with UN participated in a cognitive rehabilitation program based on the principles mentioned in Prizzamiglio et al. [28] and was relatively successfully trained [32]. His AI, after rehabilitation, remained at its extreme. This confirms claims made by Mattingley et al. [23] that the AI represents a strong ipsilesional attentional bias which is insensitive to rehabilitation. We further confirmed the persistence of the lateral bias by, additionally and on a second occasion, asking our left-sided hemianopic UN patients...
the attentional field. Damage to one hemisphere results in an ipsilesional bias. As such the attentional field is characterised by a gradient which allocates “more weight” or processing efficiency to the ipsilesional side. A unilateral lesion would also release the opposing hemisphere from inhibition, and thereby further inducing a pathological ipsilesional bias. A second critical element in Kinsbourne’s vectorial model is that the strength of the attentional vectors controlled by either hemisphere can be modulated by the activation of that hemisphere.

Hence, Kinsbourne’s vectorial model in combination with the assumed hemispheric specialisation for visuo-spatial events, accommodates the rightward bias in UN and the leftward bias in controls (attentional/hemispheric account). By this view, the perceptual asymmetries reflect patterns of differential functional cerebral activity and specifically stress that right hemisphere activity is a key concept. This right hemisphere predominance is considered to be exclusively based on its own internal properties, i.e. its directional attentional nature or its specialisation for visuo-spatial stimuli or tasks. We however argue that, in addition to this hemispheric influence, also differential sensory input can be of influence. Several indications are provided by our results.

Firstly, we found differential performances within the right hemisphere brain damage group. Namely, all our UN patients presented extreme rightward biases, while the patients with left-sided HH were significantly less extreme, though clearly in the same direction and significantly different from no bias and from controls. The difference in performance, within the right hemisphere damage group, suggests that mere right hemisphere involvement (as suggested by previous accounts) cannot be the sole explanation for the observed rightward bias. However, since we did not have access to detailed neurological information, we cannot rule out the possibility that the size of the right hemisphere lesion can account for the observed difference. A second confounding factor in our data is the marked difference in time since lesion between both right hemisphere brain damage groups. This difference could thus also, at least partly, account for the differential performance within this group. Hence, our data show differential performance in the right hemisphere brain damage group, suggesting other factors to be at hand than mere right hemisphere involvement. But alternatively, size of, and time since the right hemisphere lesion cannot be ruled out as valid determinants.

However, secondly, we showed that right- and left-sided HH patients present a qualitatively similar, but qualitatively opposite pattern of results. Both HH groups are virtually identical, but suffer a mirror-reversed visual dysfunction and present an identical but also reversed lateral bias. The side of the attentional imbalance is clearly linked to side of the HH. We hypothesise, conceptually in line with the previously mentioned “reading habit” assertion, that the VFDs lead to an over-representation of the ipsilesional hemi-space. It is commonly assumed that visual attention has two aspects, namely exogenous (stimulus-induced) and endogenous
Attentional deficit in UN, or a VFD as in HH. Previous literature suggested by Kinsbourne's model) or whether it resulted as a consequence of another dysfunction (e.g. a contralesional hemisphere can be the result of asymmetric sensory input, caused by the HH.

We hence argue that the sensory effects can be another source of attentional imbalance, which can interact with the hemispheric effects. In controls, the (normal) leftward bias is due to right hemisphere specialisation for visuo-spatial events. This bias seems enhanced by the sensory effect of a right-sided VFD. This enhancement did however not reach statistical significance in our sample. Damage to the right hemisphere removes the (hemispheric) leftward bias, and induces a rightward bias. Right hemisphere brain damage can disrupt typical visuo-attentional and directional processes, thought to be typical in UN. But a similar rightward bias can also be elicited by left-sided VFDs, for the same (i.e. sensory) reason as with right-sided VFDs.

In our study, we cannot dissociate the sensory and attentional/hemispheric components, but it was shown by Mattingley et al. [24] that UN patients without VFDs, all showed an extreme AI on the grey scales task. In our patients with UN (and left-sided HH), both sensory and hemispheric components are combined, leading in all cases to extreme and persistent rightward biases. In our left-sided VFD patients, only the sensory component is present (with possibly a minor hemispheric component). The bias is qualitatively similar to the UN patients, but less extreme. Warranted, as already argued, by the possible size and time since right hemisphere lesion effects, this suggests the sensory component to be less dominant.

Firstly, this would strengthen the claim that UN is more severe when it occurs simultaneously with HH (e.g. [1,37]) since this condition entails both sensory and attentional/hemispheric components. Secondly, this would underpin our claim that one symptom of UN behaviour (namely the lateral bias) can also be displayed by non-UN patients, namely also by HH patients. This suggests, at least at the behavioural level, a continuum in disability, giving rise to the notion “subclinical neglect”. This term would indicate subtle indications of UN (behaviour), without objective clinical signs or evidence of UN.

In previous literature, it was not clear whether the attentional imbalance was considered to be the cause of UN (as suggested by Kinsbourne’s model) or whether it resulted as a consequence of another dysfunction (e.g.: a contralesional attentional deficit in UN, or a VFD as in HH). Previous literature had shown that, in pure UN (UN without VFDs), an ipsilesional bias could be demonstrated (e.g. [23,24]), suggesting an attentional/hemispheric component. We found that HH also gives rise to a qualitatively similar bias, suggesting a sensory component. We therefore conclude that the attentional imbalance can be multiply influenced and is hence a consequence rather than cause. This has the further implication that an attentional imbalance is not necessarily and unequivocally to be associated with UN.

We feel that the grey scales task has strong clinical potential. Firstly, as was suggested in previous literature, the AI can be considered a sensitive measure of attentional imbalance, with UN as its extreme. Secondly, the AI can give the clinician a clear indication of the possible presence and side of a homonymous VFD. Namely, in our brain damaged patient group with homonymous VFDs, we observed a sensitivity and specificity of 0.94 and 0.88 in predicting the side of the HH, given the direction of the AI. Thirdly, contrary to most cancellation tasks or other tasks clinically used to diagnose differential lateral performance, almost any patient can perform the grey scales task, because it has no identification component. We hence successfully applied this test to a patient with complete object-agnosia, while all cancellation tasks appeared unachievable. Finally, although not extensively investigated, we feel that the AI can also have some practical significance. In a larger study investigating practical fitness to drive in patients with HH (to be published), we found evidence that the AI was significantly related to visual performance during driving ($r(25) = -0.510, P < 0.005$), while AIs from other tasks were not or significantly less strongly related. This suggests the grey scales task to have some practical significance to at least this type of activity of daily living.

In conclusion, we do not refute that perceptual biases reflect a pattern of functional cerebral asymmetry. But the imbalance cannot be uniquely related to specialisation of the right hemisphere for visuo-spatial attentional function, since left- and right-sided hemianopic patients, with right- and left-sided brain damage, respectively, show similar but inverse lateral biases. Asymmetric activation of one hemisphere can be the result of asymmetric sensory input, caused by the HH.

To further understand the nature and cause of the different components which can give rise to the attentional imbalance, future research could concentrate on patients with left- and right-sided brain damage, without clinical signs of UN and without VFDs. This could elucidate the possible differential hemispheric involvement. Further, other types of homonymous VFDs could also contribute to the insight into the involvement of the sensory influences. In bilateral superior and inferior quadrantanopia (i.e. missing a lower and upper hemifield, respectively) and with the grey scales items 90° rotated, the attentional imbalance should result in a quantitatively similar upper and lower bias, respectively. We also envisage experiments where different types of homonymous VFDs can be simulated on (non-brain damaged) controls using sophisticated eye-movement equipment.
these kinds of paradigms, the observed asymmetries (if any) are unconfounded with respect to VFDs and brain damage. Finally, for clinical and practical use, the relationship with performance during activities of everyday life should be further investigated and confirmed.

References