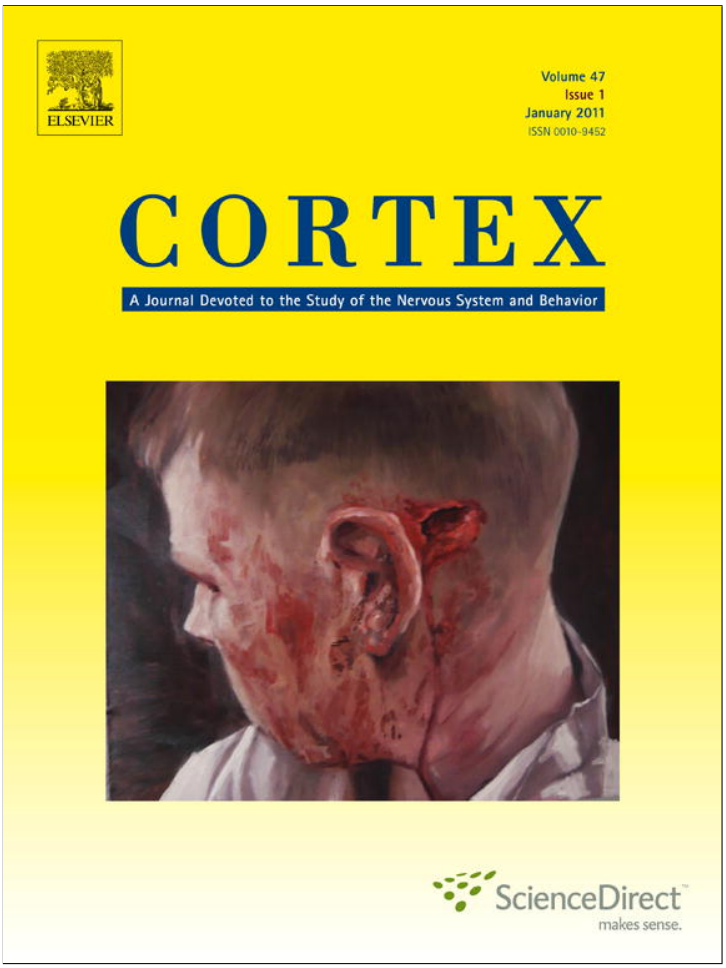

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Viewpoint

Line bisection in homonymous visual field defects – Recent findings and future directions

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ABSTRACT

Homonymous visual field defects are a frequent consequence of brain damage occurring in some 20–40% of stroke patients. They are often accompanied by a peculiar spatial bias termed "Hemianopic Line Bisection Error" (HLBE). Although known for more than 100 years the explanations for the HLBE put forward remain controversial. One explanation holds that the HLBE is a direct consequence of the field defect itself and reflects a compensatory shift of attention towards the scotoma. Another, contradicting position states that although the HLBE is frequently found in any type of homonymous visual field defect – not only hemianopia – it is not simply a direct consequence of the field defect itself, although it does contribute to it. According to this position, the HLBE arises from additional damage to extrastriate cortex, thus causing the spatial bias towards the blind field. In the present article we summarize the main arguments of both theoretical positions and argue that although both accounts are valid, they are incomplete and several important issues remain unresolved. These include the potential contribution of eccentric fixation to the HLBE, the question of multimodal impairments, the role of (visuo)-motor processes, the relation between the HLBE and visual field recovery, and the exact clinical significance of the HLBE. Thus, far from concluding the research on the line-bisection error in hemianopia, the recent series of publications on this topic serve as a welcome reminder of how much more research is needed.

1. Introduction

Homonymous visual field defects are a frequent consequence of brain damage (Zhang et al., 2006), occurring in some 20–40% of stroke patients, with a rising incidence in older age (Gilhotra et al., 2002). These visual field defects are caused by unilateral lesions to the postchiasmatic visual pathways, and are often accompanied by lesions to extrastriate cortex. Homonymous hemianopia is the most frequent type of field defect, followed by quadranopia and paracentral scotomas. These patients often suffer from three problems associated with their field defect: (a) hemianopic alexia: a laborious,

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slowed reading of words due to the parafoveal field loss in the absence of alexia for letters or words (Schuett et al., 2008); (b) a visual exploration or scanning deficit, consisting of numerous, hypometric (too small) and spatially disorganized saccades in the blind, less so in the intact field (Pambakian et al., 2000); and (c) the less well-known hemianopic line bisection error (HLBE), which can be characterized as a spatial bias towards the blind field when bisecting horizontal lines (Barton and Black, 1998; Doricchi et al., 2005; Hausmann et al., 2003). This bias goes far beyond the small left-ward bias in healthy subjects termed pseudo-neglect (Jewell and McCourt, 2000). A similar contralesional bias is observed in hemianopia when indicating the subjective visual straight ahead direction (Ferber and Karnath, 1999).

The HLBE is both a common and well-established aspect of homonymous visual deficits, which was first described more than 100 years ago by the German physician (Axenfeld, 1894). But despite its long history this error has been studied much less and as a consequence we know much less about this symptom than about either the exploration deficit, the hemianopic dyslexia or the much-studied ipsilesional line-bisection error typically found in patients suffering from unilateral neglect. It can be speculated that this lack of interest in the HLBE reflects the assumption that it is a somewhat trivial direct or indirect consequence of the visual field defect itself and is therefore neither of great theoretical nor clinical interest. This assumption was most explicitly stated by Barton and Black (1998) who argued that the HLBE may be a direct consequence of the fact that hemianopic patients are forced to view the line in just one hemifield. This may lead to an asymmetrical distortion of the spatial representation of the line and consequently produce the well-known bias in patients' bisection performance. In this case we would expect that the same bias is also found in healthy subjects when they are forced to view the line in only one hemifield and this prediction was in fact confirmed by Nielsen et al. (1999) who asked subjects to fixate either the left or right end of the line during the line-bisection task. Barton and Black (1998) also considered a more indirect link between HLBE and visual field defects. They argued that the visual field loss in one hemifield might lead to a strategic shift of attention into the contralesional hemispace, thereby producing the line-bisection bias. This explanation is supported by eye-movement recordings during line-bisection in hemianopic patients, which show that the fixation pattern is shifted towards the hemianopic side (Barton et al., 1998; Ishiai et al., 1989) and echoes a similar hypothesis proposed by Williams and Gassel (1962).

However, Zihl, Schuett and their colleagues reject the idea that the HLBE is a direct or indirect consequence of the visual field deficit. Instead they suggest that the HLBE should be regarded as an independent spatial deficit caused by additional damage to extrastriate brain structures. The starting point for their studies is a series of straightforward predictions. If the HLBE was a direct consequence of the visual field deficit, we should expect to see a significant correlation between the severity of the visual loss and the size of the HLBE, furthermore it could be predicted that if a comparable visual loss was simulated in healthy subject, the same HLBE should be observed. With regards to Barton and Black's second account, namely that the HLBE is caused indirectly by a compensatory shift of attention into the affected hemispace, Zihl and colleagues predict that spontaneous or therapy-induced recovery should in this case lead to an increased HLBE. They also predict that in the case of simulated hemianopias a correlation between the shift of the fixation pattern to the blind hemispace and the size of the HLBE should be observed. However, in a recent series of publications, which included two large-scale studies on patients with visual field deficits (Zihl et al., 2009; Schuett et al., 2011, this issue) and a study on simulated hemianopia (Schuett et al., 2009), they showed that all of these predictions are wrong.

It is thus tempting to conclude that the long-drawn out debate on the nature and origin of the hemianopic line-bisection error has finally been settled and that we can now re-focus our attention on the seemingly more relevant topics of the hemianopic exploration and reading deficits. However, in this article, we will argue that this would be premature. Firstly, because the issue of the origin of the HLBE is far from settled. Secondly, because both accounts are incomplete and thirdly, because the question of its origin is not the only unresolved issue associated with the HLBE.

2. The origin of the HLBE: an ongoing debate

Not everybody is convinced by the evidence put forward by Zihl, Schuett and colleagues. Mitra et al. (2010) argue that an attentional shift towards the blind side in combination with the detrimental effects of having to bisect the line in just one hemifield still provides the most convincing account for both the HLBE found in patients and that found in healthy observers with simulated visual field defects. Firstly, they dismiss the lack of a correlation between visual field deficit severity and the magnitude of the line-bisection-error as a null-result that is to be expected, given that the range for this bias is quite narrow (0–3%). It could be added that there may also be good reasons why the degree of central sparing, the measure which was used by Zihl and colleagues to quantify the severity of visual field deficits, is not related to bisection bias. One could argue that only sparing which is extensive enough to allow patients to see at least one half of the line within the affected hemifield could potentially lead to a normal bisection-strategy, whereby the midpoint of the line is used as the fixation point, and thus lead to a reduction of the bisection error. Since the sparing in the vast majority of examined patients was too small for that, the lack of a correlation between sparing and line-bisection bias may be expected. Secondly, Mitra and colleagues re-examined the effect of simulated visual field deficits on line-bisection performance and contrary to Schuett et al. (2009) found a line-bisection bias which both in terms of direction and magnitude corresponded to the bisection bias found in hemianopic patients. They conclude that since a bisection bias can be observed in healthy subjects without brain damage, the presence of a visual field deficit is sufficient and no extrastriate brain damage is required to produce the bisection error, thus directly contradicting the account offered by Zihl et al. (2009). However, the debate is far from over. Mitra et al. (2010) found the bisection bias only when the endpoints of the lines were marked. This is in contrast to real hemianopia

where a significant bias can be found without endpoint markings (e.g., Kerkhoff, 1993; Kerkhoff and Bucher, 2008; Zihl et al., 2009). Whether we find Mitra's or Zihl's account of the HLBE more convincing will depend on how we interpret the fact that endpoint markings are critical to produce a bisection bias in simulated hemianopia but not in real hemianopia. In this respect Mitra et al. (2010) argue that it is critical to distinguish between acute and chronic hemianopia. The contralateral bisection bias is typically found in chronic hemianopia, while an ipsilesional bias reflecting unawareness of the blind field has been reported in acute hemianopia (Machner et al., 2009). Simulated hemianopia is more like acute hemianopia in this respect and also leads to ipsilesional bias (Schuett et al., 2011, this issue) unless endpoint markers are used to make observers aware of their visual deficit and thus aware of the true extent of the contralesional half of the line (Mitra et al., 2010). However, a less sympathetic researcher could argue that the fact, that HLBE in simulated hemianopia is only found when endpoint markings are used, shows that it is a different phenomenon and therefore bears no relevance to the line-bisection error found in true hemianopia. The two groups (Zihl, Schuett and colleagues and Barton, Mitra and colleagues) do, however, agree on at least one thing, namely that long-term adaptation is not necessary to produce the hemianopic line-bisection bias.

3. More questions

We argued above that it still remains to be established whether the HLBE is a consequence of the visual loss or a spatial deficit caused by additional extrastriatal brain damage. In this context, it is important to recognize that both accounts regardless of their validity are in fact incomplete. For example in the case of the visual-loss hypothesis (Barton, Mitra and colleagues) it remains unexplained why a shift of attention towards the blind field should lead to an expansion of subjectively perceived space. For the second explanatory factor, namely the need to represent the line in just one hemifield, an explanation, for why this could lead to an overestimation of the contralesional half of the horizontal line, is at least offered. It is argued that in this case the contralesional half of the line will be closer to the fovea and given the well-know cortical magnification of foveal and parafoveal aspects of the retina, this could explain why subjectively the contralesional half of the line, which also happens to be closer to the fovea, might appear bigger. While this account is certainly physiologically plausible, it nevertheless remains speculative and alternative explanations should not be ruled out. For example we also know that normal subjects will during the bisection task typically fixate the horizontal line at its midpoint (see for example, Schuett et al., 2009). This means that in principle the oculomotor and thus extra-retinal signal from the gaze-direction could be used to guide the bisection response. However, if such a strategy is used and if the subject is then forced by instruction (Nielsen et al., 1999) or by the presence of a visual field deficit (Mitra et al., 2010) to choose a fixation point, which is shifted away from the line's true midpoint, a corresponding bias in the bisection response could be expected. In this context it is of interest to note that chronic eccentric fixation has been reported repeatedly for patients with hemianopia (Fuchs, 1922; Teuber et al., 1960; Trauzettel-Klosinski, 1997). Given the possible influence of oculomotor signals on bisection performance, it is possible, that this tendency for eccentric fixation might also be related to the observed contralesional line-bisection bias.

We argued above that the visual-loss account is incomplete, the situation is not much better for the spatial-deficit hypothesis (see Zihl et al., 2009). This hypothesis specifies only that the HLBE is independent of the visual loss, caused by additional extrastriate brain damage and somehow spatial in nature but it remains unclear which specific extrastriate brain structure(s) is/are critical in this respect. Zihl et al. reported that the lesions of patients with and without HLBE overlapped considerably but patients with HLBE showed additional damage to basal occipital–temporal areas. However, the outcome of this analysis has to be treated with caution since the two relevant groups of patients, those with ($n = 21$) and without HLBE ($n = 6$), were highly unequal in number. Moreover, the implicated brain region is fairly extensive and comprises a number of separate cortical areas. It thus remains unclear which of those areas, if any, is actually causally relevant for the HLBE. Moreover, it might be questioned whether every different type of visual field defect (hemianopia, quadranopia, paracentral scotoma, etc.) would require a *different* extrastriate area to be damaged in order to produce the line-bisection error. Furthermore, such extrastriate cortex lesions might also produce an underextension of the contralesional line segment instead of the overextension as often found in the HLBE. This leads to a further question regarding the HLBE: which mechanism(s) determine(s) over-versus under-extension of the contralesional line segment if a specific extrastriate damage is responsible for the HLBE?

If little is known about the identity of the critical extrastriate brain structure, even less is known about the nature of the spatial deficit. With few exceptions (see below) only the ability to indicate the straight ahead or to bisect a horizontal line were assessed. However, Kerkhoff (1993) showed that patients with altitudinal field defects show a vertical shift in a line-bisection task and Doricchi et al. (2003) found a striking, retinotopic dependency of visual distance judgments in a patient who combined a lower and upper quadrantanopia with mild left-sided visual neglect. These findings suggest that the spatial distortions found in patients with visual field deficits may go well beyond the classically reported horizontal line-bisection deficit and furthermore that the nature of these spatial distortions may depend on the nature of the field defects.

In this context, the preliminary findings of two patients with lower homonymous quadranopia may be interesting (Fig. 1). Patient A, a 58-year-old man, had suffered from a right middle cerebral artery occlusion 5 months prior to study and showed left lower quadranopia with a visual field sparing of 32° in the blind quadrant. He showed normal performance in a reading task (no neglect dyslexia), number cancellation and figure copy, thus did not show any sign of visual neglect. In horizontal line bisection of a 20 cm long line he erred 12 mm to the left side, which is a pathological deviation indicating the typical HLBE (normal cutoff: ±5 mm in this task). Patient B, a 54-year-old woman, had right lower quadranopia due to

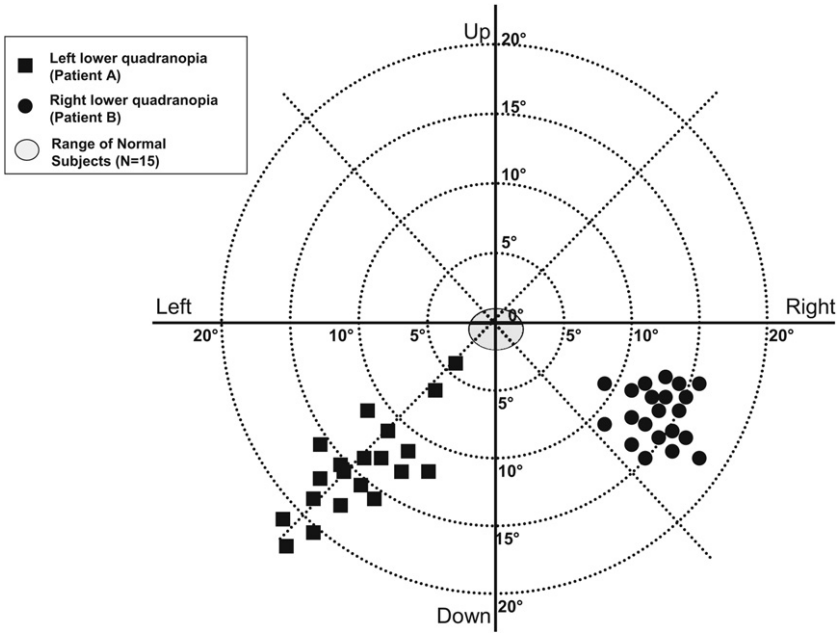

Fig. 1 – Results of the subjective visual straight ahead orientation tested in two patients (patient A and B, see text for details), and 15 healthy control subjects with intact visual fields. Twenty trials are displayed graphically in both patients (see legend) and clearly show an *oblique* shift of subjective visual straight ahead orientation towards the blind quadrant in both patients. Normal control subjects performed this task with small deviations of ±2° for the vertical and horizontal dimension (see shaded area).

a left parieto-temporal haemorrhage 3 months prior to study. Field sparing was 4° in her right lower quadrants. She performed like patient A, i.e., normal in reading (no neglect dyslexia), number cancellation and figure copy, hence did not show any sign of visual neglect either. Both patients were required to indicate verbally their subjective visual straight ahead direction in the horizontal and vertical dimension with a small red dot while looking into a dark perimeter sphere (Tübingen Perimeter) in a completely dark room. Both patients performed 20 trials in this task. They clearly showed an *oblique*, contralesional shift in their subjective visual straight ahead orientation, which was directed toward their blind quadrants. Normal control subjects performed this task with small errors of ±2° around the veridical centre (see Fig. 1). These preliminary findings (Kuhn and Kerkhoff, unpublished findings) have been extended in a larger sample (Kuhn et al., 2010) and corroborate similar findings in patients with quadrantic visual field defects reported by (Doricchi et al., 2005, Experiment 2). They also extend the previous findings reported by Ferber and Karnath (1999) who showed a *horizontal*, contralesional bias in subjective visual straight ahead orientation in hemianopic patients, which was opposite to that of hemineglect patients with intact visual fields. If replicated in a larger sample, these preliminary findings indicate another spatial distortion in patients with homonymous quadranopia, beyond the HLBE described by Schuett et al. (2011, this issue).

But even more fundamentally, it remains to be established whether the spatial deficit in hemianopia is multimodal or purely visual. While Lewald et al. (2009) concluded that the subjective straight ahead in hemianopic patients was only impaired for the visual but not the auditory domain, in contrast to these findings, Kerkhoff et al. (unpublished results) found similar though not completely identical visual *and* auditory shifts towards the blind field in hemianopia which might indicate a potential multimodal deficit akin but opposite to that found in patients with spatial neglect.

Another unsettled question is whether the HLBE impacts on the postural system which might be expected if we adopt the view of the "action–perception-cycle". In a largely ignored study Rondot et al. (1992) showed that hemianopic patients (without neglect) have a contralesional shift of their centre of gravity when standing vertical with their eyes open on a posturo-graphic platform. Interestingly, eliminating visual input (closing their eyes) reduced this contralesional bias. Moreover, patients with quadranopia (hence smaller field defects) showed a smaller contralesional bias as patients with hemianopia. Unfortunately, Rondot et al. (1992) did not report whether their patients showed the HLBE, and whether this was correlated with the bias in posture. Rondot et al.'s data however clearly suggest that the contralesional "spatial" deficit in hemianopia might in fact go beyond a purely visual deficit.

But even if we accept that the spatial deficit in hemianopia is purely visual this still allows for a substantial range of different types of visuospatial deficits. Such deficits can either be allocentric (i.e., based on an coordinate system anchored in the visual scene) or egocentric (i.e., based on a coordinate system anchored in some part of the observer's body) and within the class of egocentric coordinates systems we can again distinguish between numerous reference systems,

e.g., eye-centred, head-centred, or hand-centred systems (Committeri et al., 2004). It is assumed that these different forms of visuospatial representations are processed in different parts of the visual brain and linked to different aspects of behaviour, namely allocentric representations for perceptual judgments and egocentric representations for visually-guided action (Milner and Goodale, 2006; Schenk and MCintosh, 2010). Evidence for the claim that specific spatial maps are relevant only to specific aspects of behaviour comes from studies of patients with unilateral neglect, who may show a significant ipsilesional bias in a line-bisection task (perceptual task) but not in an obstacle-avoidance task (action task) (Mcintosh et al., 2004). Convergent and complementary patterns of neuropsychological dissociations were reported for apperceptive agnosia (Rice et al., 2006) and optic ataxia (Schindler et al., 2004). In the case of hemianopia we already know that the spatial bias affects the line-bisection performance, but will it also affect the performance in a visually-guided action task? This question is not just of theoretical but also of potential clinical relevance. Clearly a spatial distortion which leaves the patient's motor behaviour unaffected would be of little clinical relevance.

4. What is the clinical relevance of the HLBE?

Little is known about the clinical impact of the HLBE. What can be inferred from a 3° contralesional shift in horizontal line bisection on a sheet of paper obtained while the patient is sitting comfortably at a table? Does this spatial error occur also in daily life, with more dynamic situations such as walking on a floor, striding through a doorway, parking a car into a parking slot or throwing a ball towards a target? Is the HLBE under these circumstances the same? Moreover, it might be rewarding in future studies to evaluate whether the HLBE may predict the degree of partial visual field recovery that can be achieved with a training that is aimed at visual field restitution (Kasten and Sabel, 1995; Kasten et al., 1998, 2000), and if so, which mechanisms are involved in this process. Theoretically, a large contralesional HLBE by 3–4° might guide the patient's attention further towards the blind field, which eventually might lead to an increase of visual stimulation in (amblyopic) transition zones close to the field border, which in turn might promote partial field recovery in these regions. Furthermore, we should know more about how the HLBE interacts with other spatial–perceptual disorders such as impaired visual depth (Danta et al., 1978) and horizontal distance perception (Cramon and Kerkhoff, 1993) in patients with visual field defects? The only thing we do know is that the HLBE is not improved by standard treatment for visual field deficits (Zihl et al., 2009). Therefore, if the HLBE turns out to lead to disability, a specific treatment will be required.

5. Conclusions

In this article we reviewed a series of recent studies which examined the origin of the contralesional line-bisection bias found in patients with visual field deficits. We argued, that despite these recent findings the origin of this deficit is still open to debate, that the accounts, which are currently offered, are incomplete and, that apart from the theoretical interpretation of the line-bisection error, its clinical relevance remains unclear. Thus, far from concluding the research on the line-bisection error in hemianopia, the recent series of publications on this topic serve as a welcome reminder of how much more research is needed.

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