



# Magno and parvo stimuli affect illusory directional hearing in normal and dyslexic readers

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## ARTICLE INFO

### Article history:

Received 15 February 2012

Accepted 6 May 2012

Available online 17 May 2012

### Keywords:

Dyslexia

Illusory directional hearing

Magnocellular

Parvocellular

Temporal processing

## ABSTRACT

In an experimental paradigm adapted from Hari (1995), forty observers listened via headphones to 8 binaural clicks: 4 left-ear leading followed by 4 right-ear leading with either 38 or 140 ms interstimulus intervals (ISIs). Concurrently, they viewed either foveal or peripheral visual stimuli designed to activate either the parvocellular or magnocellular pathway. They then reported the perceived location of each click-pair. Our results replicated Hari's finding that observers mistake the perceived location of short ISI click-pairs more often than long. That is, when ISIs were short, the sounds seemed to play across the inside of the head in a phenomenon called illusory directional hearing. However, when click-pairs were accompanied by peripheral visual stimuli that activated the magnocellular pathway, observers were more accurate than when there were no visual stimuli. Conversely, parvocellular-activating foveal visual stimuli produced more illusory hearing than when there were no visual stimuli. These findings suggest that activating the slow sustained parvocellular system may result in a longer processing window. Thirty dyslexic observers who repeated the experimental paradigm had an even longer processing window than control observers indicating that dyslexics may have a magnocellular system deficit.

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## 1. Introduction

The visual system is composed of parallel magnocellular and parvocellular subsystems (Pokorny and Smith, 1997). The fast transient magnocellular system is activated by motion, achromatic low spatial frequencies, and high contrast. The slow sustained parvocellular system responds to color, static images, and high spatial frequencies. Selective parvocellular activation is thought to smear visual stimuli together when they are presented in rapid succession while selective magnocellular activation is thought to shorten the processing window so visual stimuli appear as discrete events (Tove'e, 2008).

A growing body of research suggests that dyslexics have magnocellular abnormalities that impede their ability to process fast transient stimuli (Lovegrove, Garzia, & Nicholson, 1990; Livingstone, Rosen, Drislane, & Galaburda, 1991). During reading, the magnocellular abnormality may prevent inhibition of the parvocellular system so word segments blend together (Boden and Giaschi, 2007). Galaburda, Menard, and Rosen (1994) showed that in dyslexics, but not controls, the left medial geniculate nuclei are 27% smaller than the right, consistent with a left hemisphere-based phonological defect. They suggested that

auditory system neurons are functionally comparable in size to those of the magno-visual system. Dyslexia would then be a pathology of the magnosystem, accounting for both phonological and visual impairment.

The current experiment was designed to explore both visual and auditory pathways simultaneously. If dyslexics cannot process auditory and visual stimuli quickly, individuals with magnocellular visual deficits should show evidence of dyslexia (Stein, 1993; Witton et al., 1998); specifically, an attention deficiency or an inability to engage or disengage transient stimulus sequences rapidly (Hari and Renvall, 2001; Renvall and Hari, 2002). This rate-processing constraint would not depend on modality or language since it also affects nonverbal sounds (Tallal, Stark, & Mellitis, 1985). In some cases, the inability to process language quickly may be subsumed by a more general inability to process any rapidly presented auditory stimuli. We tested for this contingency.

While early work by Vellutino, Pruzek, Steger, and Meshoulam (1973), Vellutino, Smith, Steger, and Kaman (1975a), Vellutino, Steger, Kaman, De Setto (1975b) concluded that a visual disorder is unlikely to cause dyslexia, more recent work has implicated a magnocellular deficit (Lovegrove, Bowling, Badcock, and Blackwood, 1980a, b, 1982, 1986, 1990; Stein, 2001; Stein and Walsh, 1997), although this theory is controversial (Gross-Glenn et al., 1995; Skottun, 2000; Skoyles and Skottun, 2004; Stuart, McNally, & Castles, 2001). For example, Skottun's (2000) extensive literature review on contrast sensitivity noted many studies

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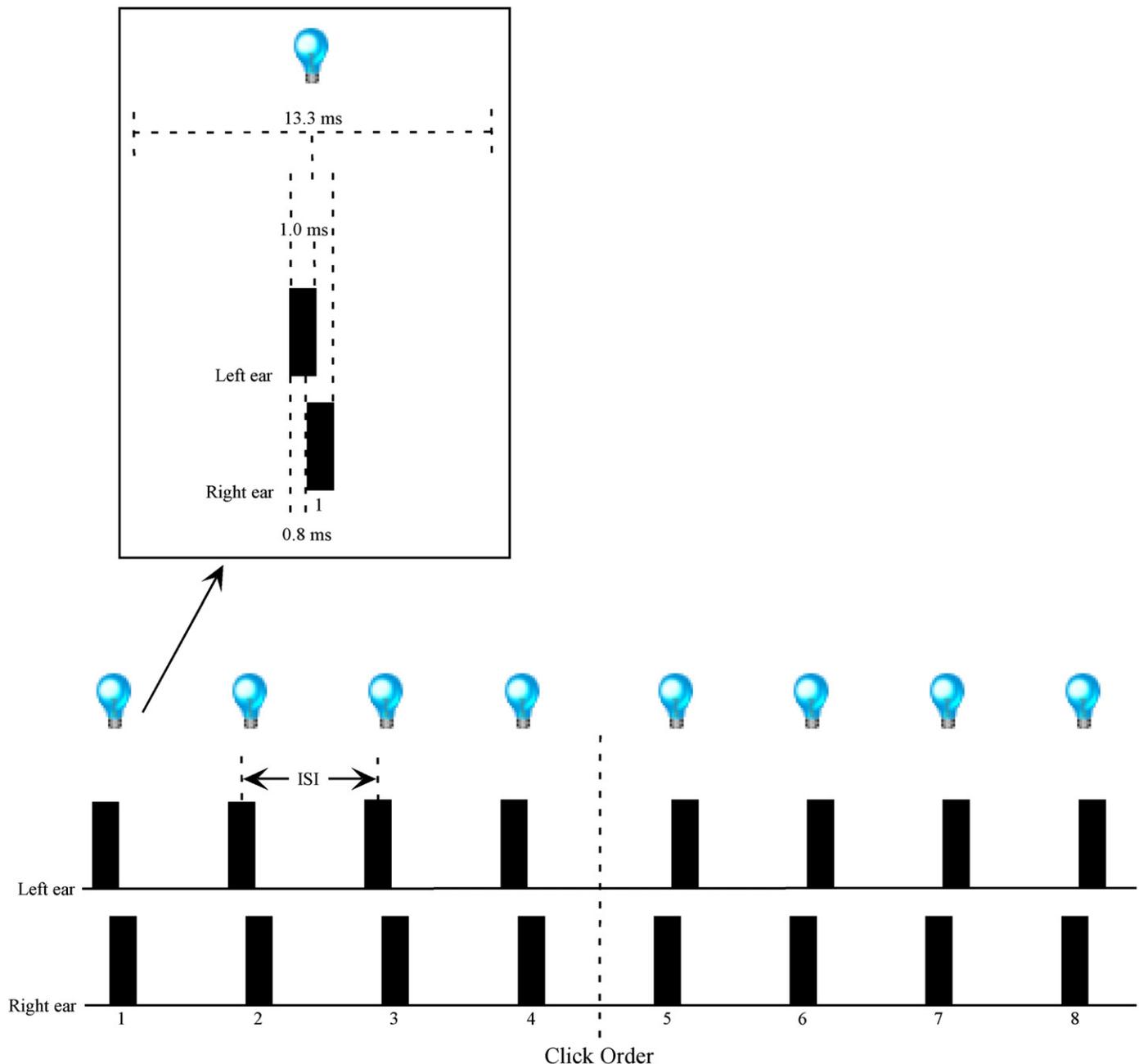
that contradict it. The new consensus concludes that [Breitmeyer's \(1976, 1980\)](#) initial study, which found that dyslexics have a normal parvocellular system and an inadequate magnocellular system, is probably flawed ([Skottun and Parke, 1999](#)), yet [Slaghuis's \(2007\)](#) critique of [Skottun's \(2000\)](#) review pointed out that not differentiating dyslexic subgroups is misleading. We addressed this question using [Boder's \(1970\)](#), [Boder and Jarrico, 1982](#)) classification of dyslexics (see methods).

More recent investigations have shown that dyslexics have difficulty discriminating and processing both simple and complex rapid sound sequences ([Hari and Kiesilä, 1996](#); [Kujala et al., 2000](#); [Renvall and Hari, 2002](#); [Schulte-Körne, Deimel, Bartling, Renschmidt, 1998](#); [Tallal, Miller, Jenkins, & Merzenich, 1997](#)). The present study investigated both visual and auditory

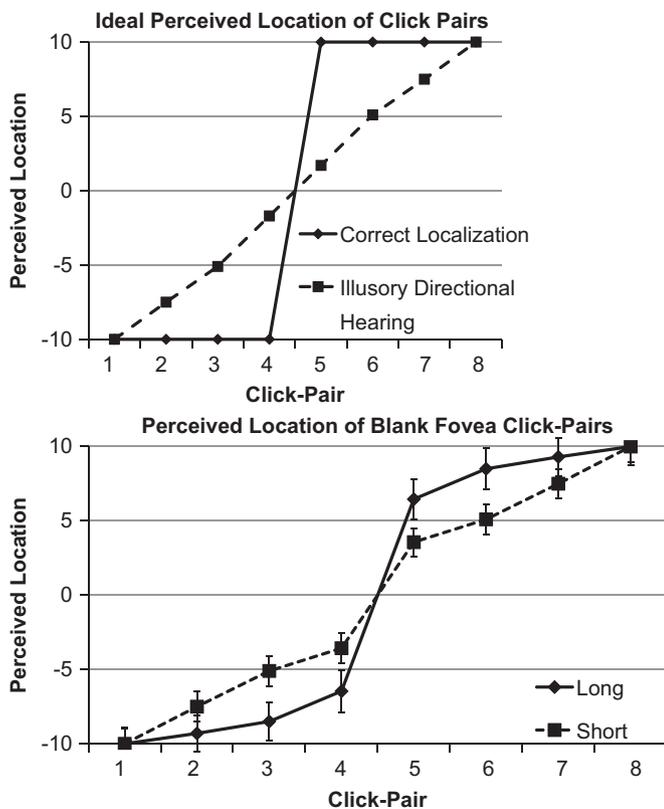
processing using simple stimuli that followed a right-to-left and left-to-right pattern resembling reading.

We ([Mays and Schirillo, 2005](#)) previously showed that light flashes affect auditory processing in a directional hearing illusion ([Hari, 1995](#); [Hari and Kiesilä, 1996](#)). We presented eight binaural click-pairs with an interaural interval of 0.8 ms. Clicks 1–4, presented in isolation, were perceived as coming from the left side (left-ear leading) and clicks 5–8 were perceived as right-ear leading (Fig. 1).

When the interstimulus interval (ISI) between each click-pair was long (140 ms), observers reported that the four left-leading click-pairs originated near the left ear and the four right-leading click-pairs near the right ear (Fig. 2). Completely veridical perceptions would result in a response of  $-10$  for the first 4 click-pair



**Fig. 1. Sequence of eight click-pairs** ([Dellorso and Schirillo, 2010](#)). The first four are left-ear leading, while the second four are right-ear leading. The light bulbs represent the presentation of visual stimuli. (Not to scale).



**Fig. 2.** Blank fovea long and short localizations to a presentation of eight click-pairs. The long responses indicate near veridical localizations (see insert with idealized responses), while short localizations indicate greater illusory directional hearing (see insert).

localizations and +10 for the last four (Fig. 2, insert). In contrast, with short ISIs (38 ms), observers reported illusory directional hearing; that is the sounds appeared to come from inside the head rather than from the left or right ear. Each subsequent click-pair seemed to traverse a roughly equal percentage of space in the head as when the headphone balance of a portable media player is adjusted. Note that a 0.8 ms interaural click-pair offset should not produce a sound that is perceived to be located within the head. Fig. 2 shows the roughly equal steps that characterize short fovea blank localizations between each click-pair, idealized in the insert. To replicate Hari's (1995) original work, Fig. 2 shows only the blank fovea condition for normal observers. While a blank condition refers to only an auditory stimulus, the data are considered foveal because they were collected during the visual fovea trials. Results from the blank visual peripheral condition are comparable.

The simplest way to capture this phenomenon is to measure the difference in localizing the fourth and fifth click-pairs (Fig. 3). A large difference tends toward veridical localization but when the ISIs are short, the eight click-pairs seem to migrate in equal increments through the head. This results in a smaller difference between the fourth and fifth click-pairs. Further, since short ISIs change the perception of the entire train of clicks, later click-pairs must influence the perception of earlier ones. Hari (1995) ascribed this sluggishness to the temporal window of neural integration. Hari and Kiesilä (1996) found that in controls, the illusion disappeared at intervals exceeding 90 ms, while in dyslexics, it persisted up to 500 ms. Thus, dyslexic adults seem to have a deficit in processing rapid sound sequences.

Our experiment's visual stimulus was both dynamic and spatial. It was meant to emulate the side-to-side tracking of

reading and the auditory component reflected the spatial structure of silent reading (e.g., in the head). We hypothesized that presenting visual stimuli designed to activate the parvocellular subsystem would bias the perceived location of the concurrent auditory stimulus (Dellorso and Schirillo, 2010; Mays and Schirillo, 2005; Slutsky and Recanzone, 2001). We predicted that since the parvocellular system integrates information over a longer temporal window than the magnocellular system, it would promote illusory directional hearing. If dyslexics have a deficient magnocellular system, presenting magnocellular-selective stimuli should improve their magnocellular processing.

## 2. Methods

### 2.1. Subject groups

All procedures were approved by the Wake Forest University Institutional Review Board and performed in accordance with the ethical standards in the 1964 Declaration of Helsinki. All observers were native English speakers and none had ADHD. Forty normal-reading control observers (26 women, 14 men, ages 18 to 23) in the undergraduate Psychology research pool and 30 dyslexic readers (12 women, 18 men, ages 18 to 24), paid to participate, from the Teaching and Learning Center, all with self-reported normal hearing and self-reported normal or corrected-to-normal vision, ran in each of six conditions.

Dyslexics were selected if they had an early childhood history of difficulty in learning to read or to spell. All had received tutoring to remediate it. At Wake Forest University, dyslexia is diagnosed by a speech therapist or a psychologist. The inclusion criterion was at least 24 months of school literacy impairment with a normal IQ. They were also tested for significant differences from normal control subjects in phonological processing abilities. All observers took the Boder Test of Reading-Spelling Patterns (Boder, 1970; Boder and Jarrico, 1982), which discriminates three groups—dysphonetic, dyseidetic, and a mixture known as dysphonetic (Boder, 1973). Dysphonetic dyslexia is characterized by phonemic-linguistic (auditory) errors; for example, misspelling known and unknown words based on confusion about their component sounds. On the other hand, dyseidetic dyslexia is characterized by visual perception errors; known and unknown words with clear phonetics are spelled correctly and it is determined by performance in decoding word inventories. Mixed (dysphonetic) dyslexia is characterized by a limited sight vocabulary and bizarre misspellings, reflecting very poor phonetic skills. Borsting et al. (1996) noted that dyseidetic dyslexia, which occurs in 10–30% of the population, may represent the segment that does not have a transient channel disorder. Consequently, we screened for and used only those individuals who had a dysphonetic disorder. This group differed from our normal (controls) that showed no disorder of any type.

### 2.2. Stimuli

Adopting Hari's (1995) experimental procedure, observers listened to a set of 8 click-pairs presented by E-Prime software over circumaural headphones (Koss; Pro/4AA; ~45 dB A at peak measurements) with a randomly assigned ISI (38 or 140 ms). In half of all trials, 4 left-leading click-pairs were followed by 4 right-leading click-pairs. In the other half, the sequence was reversed. We averaged our results over left- and right-leading trials to double our N since the direction of illusory hearing did not differ significantly between which of the ears was leading.

Observers were also presented 13.3 ms visual stimuli on a CRT monitor with a chin rest (19" CTX, model EX95950F, 75 Hz refresh rate) (Figs. 1 and 4). They were randomly assigned to one of three conditions:

- (1) parvocellular-activating stimuli, consisting of an equiluminant red and green, high-contrast (90% Michelson contrast), high-frequency (5.0 cpd) sinusoidal grating in a 5.2° visual angle circular Gaussian envelope that flashed alternately horizontally and vertically in orientation with each succeeding click-pair; observers first underwent heterochromatic flicker photometry to determine their equiluminance point (Borsting, et al., 1996; Cooper, Sun, & Lee, 2012; Elsner, Pokorny & Burns, 1986; McAnany & Alexander, 2006; Pokorny & Smith, 1997; Rudvin, 2005; Stein, 2003);
- (2) Magnocellular-activating stimuli, consisting of a low-contrast (10% Michelson contrast), black-and-white, low-frequency (0.38 cpd) sinusoidal grating in a 5.2° visual angle circular Gaussian envelope that flashed alternately horizontally and vertically in orientation with each succeeding click-pair; and
- (3) a control condition with no visual stimuli, but simply a blank screen.

In one session, all visual stimuli were centered at  $\pm 3^\circ$  visual angle to promote parvocellular activation. In the other session, all visual stimuli were placed at the periphery of the computer monitor (centered at  $\pm 16.5^\circ$  visual angle) to promote magnocellular activation.

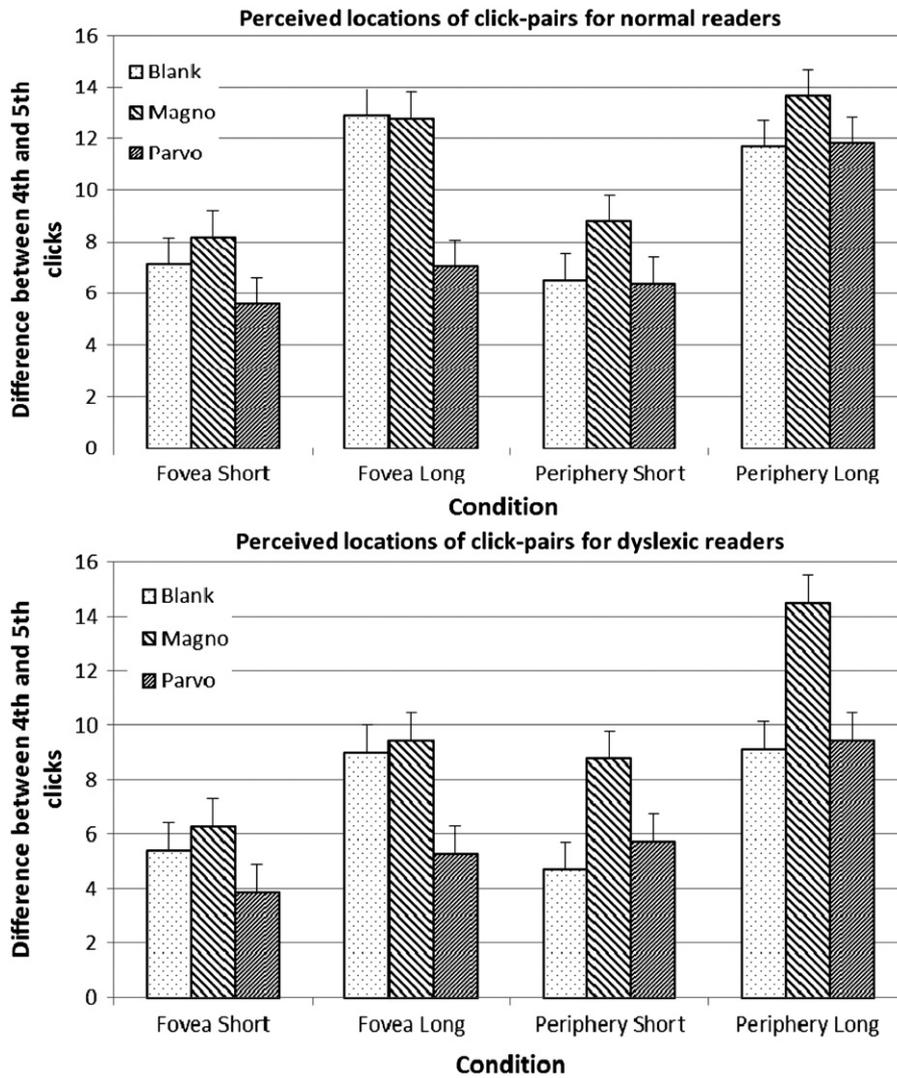


Fig. 3. Perceived locations of click-pairs with visual stimuli. Error bars represent SEM; (top) normal readers, (bottom) dyslexic readers.

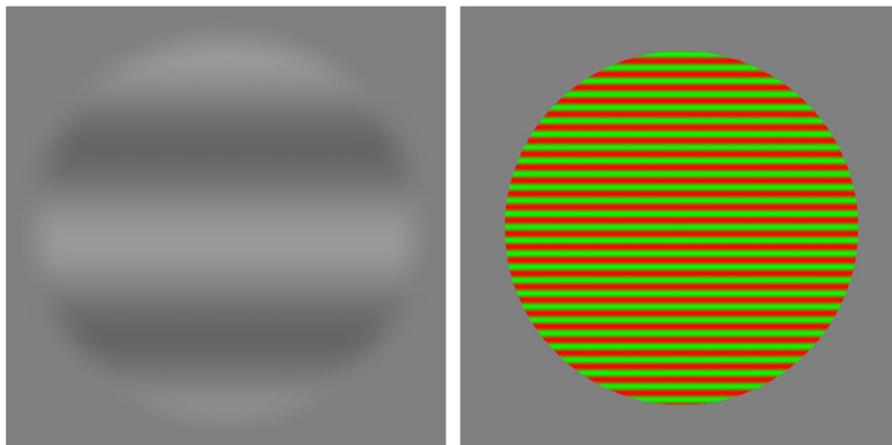


Fig. 4. Horizontally oriented visual stimuli presented on the computer monitor. (left) Magnocellular-activating stimulus; (right) parvocellular-activating stimulus.

2.3. Procedure

After each train of click-pairs, we adopted Dellorso and Schirillo's (2010) modification of Hari's (1995) procedure in which observers move eight analog sliding scales on the computer screen to record the perceived location of each

click-pair inside their heads: -10 represented the leftmost; +10 the rightmost; and 0 the middle. Since this task is computerized, it is undemanding and took less than 20 s/trial. The error bars in Fig. 2 indicate the small variability in making these measures.

Observers completed 72 trials/session which took less than 1 h (2 ISIs—fast [38 ms] & slow [140 ms] X 3 visual conditions (blank, magno, & parvo) X 2 auditory

directions (left- or right-ear leading) X 6 trials/block). Each of the two sessions presented the visual stimuli at one of the two visual angles ( $\pm 3^\circ$  and  $\pm 16.5^\circ$ ).

### 3. Results

As Hari (1995) found, observers tended to respond in two characteristic patterns: (1) nearly correct localization, or (2) with the illusory directional hearing effect (Fig. 2). To quantify the difference between these patterns, we calculated the difference in perceived locations of the 4th and 5th click-pairs, a procedure introduced by Hari and Kiesilä (1996). A large difference indicates near veridical localization since either  $-10$  or  $+10$  for each of 4 clicks was actually presented while a small difference indicates illusory directional hearing since observers localized the 8 clicks in roughly equal steps though their heads. Thus, a steeper function implies more precision and trends away from illusory directional hearing.

Fig. 3a shows mean differences for control observers. Differences are organized by ISI length (short vs. long) and visual stimulus condition (blank, magnocellular-, or parvocellular-activating) and visual stimuli location (foveal or peripheral). A repeated-measures ANOVA identified a statistically significant main effect of ISI length ( $F [1, 39]=12.438, p < 0.001$ ): the difference in the perceived locations of the 4th–5th click-pairs was smaller at the short ISI ( $M=7.1$ ) and larger at the long ISI ( $M=11.7$ ). The interaction between ISI and type of visual stimulus was also statistically significant ( $F [1, 39]=7.627, p < 0.009$ ) due to two effects: (1) a significant difference between the foveal long ISI parvo condition and the foveal long ISI blank condition ( $t=2.916, p=.006$ ) and between the foveal short ISI parvo condition and the foveal short ISI blank condition ( $t=2.243, p=.031$ ); and (2) a significant difference between the peripheral long ISI magno condition and the peripheral long ISI blank condition ( $t=2.613, p=.013$ ) and between the peripheral short ISI magno condition and the peripheral short ISI blank condition ( $t=2.827, p=.007$ ).

Fig. 3b shows these mean differences for dyslexic observers organized by ISI length (short vs. long), visual stimulus condition (blank, magno, or parvo), and visual stimulus location (foveal or peripheral). A repeated-measures ANOVA identified a statistically significant main effect of ISI length ( $F [1, 29]=12.438, p < 0.001$ ): the difference in the perceived locations of the 4th–5th click-pairs was smaller at the short ISI ( $M=5.8$ ) and larger at the long ISI ( $M=6.2$ ).

The interaction between ISI and type of visual stimuli was also statistically significant ( $F [1, 39]=7.627, p < 0.009$ ). It was due to the same two effects observed for the controls: (1) a significant difference between the foveal long parvo condition and the foveal long blank condition ( $t=3.211, p=.003$ ) and between the foveal short parvo condition and the foveal short blank condition ( $t=2.309, p=.028$ ), and (2) a significant difference between the peripheral long magno condition and the peripheral long blank condition ( $t=2.866, p=.007$ ) and between the peripheral short magno condition and the peripheral short blank condition ( $t=2.751, p=.010$ ).

We conducted an omnibus ANOVA between-subjects (normal, dyslexic) by within-subjects interaction for the three visual conditions (blank, magno, parvo), each of the two stimulus locations (fovea, periphery), and two ISIs (long, short). For fovea short  $F (1136)=8.36, p < .0004$  and long  $F (1136)=11.74, p < .0001$ , the difference between the 4th and 5th click in all three visual conditions (blank, magno, parvo) was significantly longer for dyslexics. For periphery short  $F (1, 136)=6.47, p < .01$ , an interaction occurred, and the difference between the 4th and 5th click was significantly longer for dyslexics only in the blank

visual condition (Tukey-Kramer HSD,  $F_{138}=2.91; p < 0.004$ ), not the magno (Tukey-Kramer HSD,  $F_{138}=1.41; p=0.23$ ) nor parvo (Tukey-Kramer HSD,  $F_{138}=1.53; p=0.13$ ) conditions. For periphery long  $F (1, 136)=6.56, p < .01$ , an interaction occurred, and the difference between the 4th and 5th click was significantly longer for dyslexics in both the blank (Tukey-Kramer HSD,  $F_{138}=3.42; p < 0.001$ ) and parvo (Tukey-Kramer HSD,  $F_{138}=3.16; p < 0.002$ ) visual conditions, but not the magno (Tukey-Kramer HSD,  $F_{138}=0.95; p=0.34$ ) condition.

### 4. Discussion

Peripheral parvo stimuli had no effect and magno stimuli increased the 4th–5th difference for nondyslexic controls. For foveal stimuli, they experienced a small but statistically significant effect at the short ISI and a clear decrease in the 4th–5th difference at the long ISI. For dyslexic observers, the most prominent effects were a decrease in the 4th–5th difference at the long ISI compared with the control observers which replicates the findings of Hari and Kiesilä (1996). Interesting, however, that peripheral magno-stimuli caused the dyslexics' deficit to be less problematic.

Our experiment replicated Hari's (1995) findings: at short ISIs, observers tend to report smaller 4th–5th click-pair differences, exhibiting the illusory directional hearing effect in which the sounds seemed to move in equal steps through the head. Long ISI click-pairs with concurrent parvocellular-activating visual stimuli produced more illusory hearing (smaller 4th–5th click-pair difference scores) than the comparable condition without visual stimuli. This effect occurred in the fovea (at  $\pm 3^\circ$  visual angle) where the slow sustained parvocellular system is more likely to be activated which may blur the temporal window that contains successive click-pairs, enhancing illusory directional hearing. These effects were more prevalent in dyslexic observers replicating Hari & Kiesilä results (1996), which indicate that, in dyslexia, the window of temporal integration extends significantly farther backward than in normal subjects.

A sluggish attentional shifting (SAS) hypothesis (Hari and Renvall, 2001; Facoetti et al., 2010; Lallier et al., 2010) might explain our findings. It argues that when dyslexics experience rapid stimulus sequences, their automatic attention system cannot disengage fast enough from one item to move to the next. The SAS theory is supported by studies conducted separately in the auditory (Hari, 1995; Hari and Kiesilä, 1996; Helenius, Uutela, & Hari, 1999) and visual (Hari, Renvall, & Tanskanen, 2001; Hari, Valta, & Uutela, 1999) modalities. Hari and Renvall (2001) claim that SAS may lead to reading acquisition disorders where distorted perception of rapid speech impedes development of correct phonological representations. Our work provides a crucial link between sluggish auditory and visual processes.

Research on amodal temporal deficits in dyslexia (Farmer and Klein, 1995) suggests involvement of the visually transient magnocellular system and its auditory counterpart (Stein and Talcott, 1999; Van Ingelghem et al., 2001; Witton et al., 1998). Our enhanced peripheral magnocellular effect may have helped dyslexic observers shift their attention to a new target, whereas the parvocellular effect worked in the opposite direction. The opposite effects of magno- and parvo-cellular stimuli suggest an attentional deficit originating in the parietal lobe (Hari and Renvall, 2001; Stein and Walsh, 1997; Vidyasagar and Pammer, 2010) since it anatomically separates dyslexics' poor magnocellular functioning from a general dorsal stream malfunction which can also receive parvocellular and koniocellular inputs (Skottun and Skoyles, 2005). Note, however, that our dyslexic sample did not have co-morbid ADHD (Dykman and Ackerman, 1991; Felton,

Wood, Brown, Campbell, & Harter, 1987; Felton and Wood, 1989). Consequently, we feel we measured a perceptual phenomenon and while our findings agree with SAS theory, our experiment did not directly test attentional mechanisms. Although many have suggested that sequential auditory attentional dysfunction result in dysphonetic dyslexia, our methodology and findings can only add that visual processing limitations may also correlate with auditory constraints.

## Acknowledgments

We are indebted to Jeff Muday for programming assistance, Julie Edelson and Lisa Scalzo for editing, and Steven Davis for help with statistical analysis.

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