

Covert orienting to exogenous and endogenous cues in children with spina bifida

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Abstract

Children with spina bifida meningomyelocele and hydrocephalus (SBM) have congenital dysmorphology of the midbrain and thinning of the posterior cortex, brain regions associated with the control of covert orienting. We studied cued covert orienting in 92 children with SBM, and 40 age-matched typically developing controls. Cues were of three types: exogenous (luminance change in a peripheral box either valid or invalid for upcoming target location), endogenous arrow (a central arrow either valid or invalid for upcoming target location), or endogenous word (a central word either valid or invalid for upcoming target location). Compared to controls, children with SBM showed slowed covert orienting to both exogenous and endogenous cues and a higher cost of attentional disengagement (e.g., a greater cue-validity effect) for exogenous although not for endogenous cues. Covert orienting deficits were associated with midbrain dysmorphology in the form of beaking of the tectum, and with right posterior brain volume loss.

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In our visual environment, we orient to information that is either salient or interesting. Orienting may involve overt movements of the head, eyes, or body, or covert shifts of attention whereby the head, eyes, or body remain stationary. Covert orienting, which changes attentional priorities, may be either automatic, as when we orient to salient information, or effortful, as with voluntary shifts of attention

to something of mental interest (Jonides, 1980; Posner & Raichle, 1994).

In covert orienting paradigms, the task is to detect a cued target appearing with a specified probability in either of two locations. Facilitated detection of targets after informative (valid) cues represent the benefit associated with having oriented to the cue; slowed detection of targets after misleading (invalid) cues represents the orienting costs. For all types of cue and for both valid and invalid trials, the motor response is the same. No eye movements occur while central fixation

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is being maintained, so the enhancement of detection when the cue is valid is attributed to an internal shift of covert attention towards the cue. In studies of covert orienting, the variables that have been explored in both adults and children include cue type, whether exogenous or endogenous; cue-validity and the costs and benefits of cuing; stimulus onset asynchrony (SOA), the time interval between cue and target; plane, whether horizontal or vertical; and performance variability.

A cue is an event appearing at the future location of the target, or an event that appears elsewhere but which indicates where the target will appear. Salient information is exogenously cued by sensory changes such as a peripheral flash or a bright box. Interesting information is endogenously cued by cognitive signals, such as an arrow or word indicating a target location of interest, that prompt a voluntary shift of attention to the upcoming target. Exogenous and endogenous orienting are separable in normal adults (Posner, Cohen, & Rafal, 1982), such that attention is summoned automatically by exogenous cues, but can also be deployed endogenously by cognitive cues.

On standard covert orienting tasks, both adults and school-aged children show an exogenous cue-validity effect; that is, they respond more quickly to a peripheral target if a visual marker has correctly cued the location of the upcoming target than if the wrong location has been cued (Akhtar & Enns, 1989; Brodeur, Trick, & Enns, 1997; Enns & Brodeur, 1989; Jonides, 1980). Children and adults differ in the magnitude of the cue-validity effect. Compared to older children and adults, younger children show a larger exogenous cue-validity effect (e.g., Akhtar & Enns, 1989), suggesting they are less able to shift attention from an incorrectly cued location to the target position. For endogenous cues, younger children may show a larger cue-validity effect than adults (Brodeur et al., 1997). Adult-like performance has been described in 8–10 year olds, albeit with less consistency than adults (Goldberg, Maurer, & Lewis, 2001), suggesting that even when accurate, children may be more variable than adults. There is some uncertainty about just which measures of covert orienting improve with age: all (e.g., Schul, Townsend, & Stiles, 2003), or only disengagement measures (e.g., Wainwright & Bryson, 2002).

The interval from cue to target (SOA) affects the size of the validity effect in adults. Children and adults differ in the time they require to use predictive information. Adults show a cue-validity effect when sufficient time is available (Klein, 1994). Although they demonstrate reliable cuing benefits at longer SOAs (Wainwright & Bryson, 2002), children may require more time than adults to use predictive cue information (Brodeur & Boden, 2000). Validity effects are typically smaller in the vertical than in the horizontal plane (e.g., Rafal, Posner, Friedman, Inhoff, & Bernstein, 1988).

The neuroanatomical substrates underlying covert and overt orienting overlap substantially (Corbetta & Shulman, 2002), although they are not identical (Posner, 1980; Posner et al., 1982; Posner & Peterson, 1990). Studies of the brain

bases of covert orienting have identified a distributed neural system (Mesulam, 1981, 2002), including the midbrain, the pulvinar, and the posterior parietal cortex. The type of cue and the elementary operations of covert orienting may engage different neural components.

The midbrain, which includes the superior colliculus, is involved in both visual attention and visual perception. The condition of progressive supranuclear palsy is associated with degeneration of midbrain structures, and produces vertical gaze disorders as well as deficits in spatial orienting that are not directly caused by disorders of eye movements (Rafal & Grimm, 1981). Midbrain lesions produce a delay in covert orienting, especially in the vertical plane, suggesting that retinotectal pathways are important for reflex orienting to exogenous signals.

Higher-level attentional control engages a cortical attention system that includes the prefrontal, anterior cingulate, and parietal cortices. This system has to do with suppression of irrelevant information, resolution of conflicts, and reprogramming of information. For example, unattended stimuli are suppressed in the prefrontal (Everling, Tinsley, Gaffan, & Duncan, 2002), and parietal cortex (Gottlieb, Kusunoki, & Goldberg, 1998), and parietal lesions impair the ability to filter irrelevant information (Friedman-Hill, Robertson, Desimone, & Ungerleider, 2003).

Endogenous cues appear to engage the posterior parietal system and thalamus (Posner et al., 1982; Posner, Walker, Friedrich, & Rafal, 1984). Patients with parietal lesions (although not those with progressive supranuclear palsy) have longer reaction times to invalid cues in the visual field contralateral to the lesion (Posner et al., 1984), suggesting a deficiency of disengagement. Parietal lesions appear to disrupt not so much the ability to *move* attention as the ability to *disengage* attention. Right parietal lesions, especially, produce problems with endogenously cued orienting.

Covert orienting has been studied in normal adults, adults with brain lesions, and typically developing children. The techniques for studying covert orienting are applicable to the investigation of attention in congenital brain disorders such as spina bifida.

Spina bifida meningocele (SBM), the most severe form of spina bifida, is the most common severely disabling birth defect in North America, occurring in about 0.5 per 1000 births. It affects brain as well as spine. Children with SBM have characteristic congenital malformations of the cerebellum and hindbrain (the Chiari II malformation), midbrain, and corpus callosum. The Chiari II malformation leads to hydrocephalus, which involves enlarged cerebral ventricles and produces a range of primary and secondary effects on the brain (del Bigio, 1993; Fletcher, Dennis, & Northrup, 2000). The effects on the brain reflect both primary, specific malformations and the secondary effects of hydrocephalus.

The characteristic pattern of brain anomalies in SBM (reviewed in Dennis et al., 1981, 2004; Fletcher et al., 1996, 2000; Hannay, 2000) make it a condition of considerable interest to the study of attention in general, and to covert

orienting, in particular. SBM involves a congenital abnormality of the midbrain, tectal beaking, which is a mechanical consequence of brain development in a small posterior fossa. The effects of hydrocephalus in SBM affect posterior cortical structures more than anterior cortical structures, proceeding on a back to front Laplacian progression that reflects the extent of ventricular dilation. The effects are maximized in midline and posterior temporal-parietal structures, producing variable degrees of thinning in the parietal cortex and selective reductions in posterior cortical volume.

To date, attention skills in children with SBM have not been fully studied at the behavioral level. Nor have brain–behavior relations been explored in relation to the neural substrates of covert attention.

Some earlier behavioral studies of SBM noted problems in attention (e.g., Tew, Laurence, & Richards, 1980), while others did not (e.g., Lollar, 1990), and still others noted problems on selected attention tasks (e.g., Willoughby & Hoffman, 1979). Differences in psychometric attention outcomes may have reflected factors in the children (e.g., different types of spina bifida, shunt history), as well as differences in attention task demands.

Children with SBM often show beaking of the tectum, as well as paralysis of upward gaze during shunt malfunctions. It is not known whether these congenital disorders of the midbrain are associated with problems in exogenously cued attention shifting, particularly in the vertical plane, such as occur in adult conditions like progressive supranuclear palsy associated with midbrain degeneration.

Children with SBM show significant volume reduction in posterior brain systems that include the parietal lobe, with their posterior cortex being thinner than their anterior cortex (Dennis et al., 1981; Fletcher et al., 1996). Volume reductions in the posterior cortex are associated with poorer perceptual and perceptual-motor function in children with SBM (Dennis et al., 1981; Fletcher et al., 1996). The relation with attention is not fully understood. In a study of covert orienting to an exogenous cue, a group of children with hydrocephalus (including children with SBM and children with hydrocephalus from other etiologies) showed problems with disengagement, suggesting that they had problems with posterior rather than anterior attention function (Brewer, Fletcher, Hiscock, & Davidson, 2001).

It is not known whether posterior attention deficits in children with hydrocephalus are related to volume reductions in the posterior cortex. Nor is it known whether disengagement deficits occur similarly with exogenous as well as endogenous cues, the latter of which are thought to require more anterior, top-down neural control. It is also not known whether the volume of the parietal lobe and anterior brain is related to disengagement from endogenous cues.

A study of the neurobiology of covert orienting in children with SBM would provide information, not only about attention skills in this population, but also about the bases of covert orienting in the developing brain. The present study investigated the neuroanatomical basis of covert orienting

in SBM by using paradigms with established validity. We studied covert orienting in children with SBM and typically developing age peers with respect to cue type, cue-validity, SOA, and plane; compared covert orienting in children with SBM and MRI-identified anomalies of the midbrain in the form of congenital tectal beaking, children with SBM and no MRI-identified anomalies of the midbrain, and typically developing controls; and related covert orienting to MRI-identified volumetric measurements of anterior and posterior cortex, brain regions that have been implicated in covert orienting in the mature brain.

2. Methods

2.1. Participants

Participants were 132 children and adolescents between 8 and 19 years of age recruited from clinics from two sites, The Hospital for Sick Children in Toronto ($N = 74$) and the University of Texas-Houston Medical School in Houston ($N = 58$). The study was approved by the ethics boards at each site and was conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. Prior to being included in the study, participants and their parents gave informed assent and consent. One group ($N = 92$) had been diagnosed with SBM at birth, and had been treated with a shunt shortly thereafter. Twenty-four of those children had no shunt revision, 32 had 1 revision, 24 had 2–4 revisions, 11 had 5–9 revisions, and 1 child had 14 shunt revisions. The other group comprised typically developing, age-matched controls ($N = 40$). All participants had IQ scores within two standard deviations of the population mean of 100; that is, they had IQ scores ≥ 70 on either or both the Verbal Reasoning and/or Abstract/Visual Reasoning subtests of the Stanford Binet Test of Intelligence-Revised (Thorndike, Hagen, & Sattler, 1986). Individuals were excluded from participation if they had neurological disorders unrelated to SBM, severe psychiatric disorder, uncontrolled seizure disorder, uncorrected sensory disorder, or inability to control the upper limbs.

Table 1 provides information on IQ and socio-demographic characteristics. Children with SBM had lower average IQ scores than controls, $t(130) = 8.74$, $p < 0.001$, although each group IQ was within the normal range (i.e., within \pm standard deviation of the population mean). Differences in age, gender, ethnicity, and socioeconomic status (Table 1) were not statistically significant ($p > 0.05$).

2.2. Orienting procedures

2.2.1. Uncued orienting

The uncued orienting task was administered using an IBM-compatible personal computer, was coded in Delphi, and scored in SAS. A chin rest with a forehead bar was used to ensure a distance of 32.5 cm between the computer monitor and participant's eye level. Participants were required to

Table 1
Demographic and IQ information for control and spina bifida participants

	Group	
	Control (N=40)	Spina bifida (N=92)
Age (years; mean \pm standard deviation)	14.53 \pm 2.58	15.31 \pm 2.92
Gender		
Male	20	47
Female	20	45
Ethnicity		
Caucasian	29	64
Hispanic	2	19
Asian	5	3
African American	2	3
Other	2	3
Socioeconomic status	46.38 \pm 11.66	41.46 \pm 13.56
Stanford-Binet composite IQ	108.4 \pm 9.68	89.1 \pm 12.42

Notes: Socioeconomic status was determined by the Hollingshead (1975) four-factor scale.

maintain fixation on a central black cross on a white background that remained on the computer screen throughout the task. They were instructed to click the computer mouse as soon as a black circle appeared in the periphery, and reaction time for each trial was recorded. Participants completed 120 trials, consisting of 30 targets that appeared randomly in each of four quadrants: upper right (0–90°), upper left (91–180°), lower left (181–270°), and lower right (271–360°). In order that children be unable to predict the appearance of the target, inter-trial interval ranged from 500 to 1500 ms and varied randomly across trials. Trials with reaction times less than 200 ms were considered anticipatory responses and excluded from analysis. Median and standard deviation of reaction times in each quadrant were calculated for each participant.

Participants were given the following instructions. “In this game, you are going to put your chin in this chin rest and your forehead against this bar. You will keep your eyes focused on a black cross in the center of the screen. While you are looking at the black cross, some circles are going to appear in different parts of the screen one at a time. I want you to click the left mouse button as soon as you see one of the circles appear on the screen. Once the experiment starts, the mouse pointer will disappear and you will not need to move the mouse to any of the dots. Just click the button as soon as you see a circle anywhere on the screen. Make sure you keep your eyes focused on the black cross in the center of the screen. Remember, press the left mouse button as fast as you can once you see a circle appear.”

2.2.2. Cued orienting

The cued orienting task was based on Rafal et al. (1988). It was administered using an IBM-compatible personal computer, coded in MEL, and scored in SAS. A chin rest with a forehead bar was used to ensure a distance of 47.5 cm between the computer monitor and participant’s eye level.

While maintaining central fixation on a black cross, participants were required to press a button when a target appeared in one of four peripheral boxes on the computer screen. The four boxes were located to the right, left, above, and below the central fixation point. The outside edges of the boxes subtended a visual angle of 10°, with each box and the center cue subtending 1°.

On each trial, a cue was presented at one of two stimulus onset asynchronies (SOA, either 200 ms or 1000 ms prior to the target).

Cues were of three types:

1. *Exogenous cue*: This was a luminance change in one of the peripheral boxes.
2. *Endogenous arrow cue*: This was an arrow that replaced the cross at the central fixation point.
3. *Endogenous word cue*: This was a word (LEFT, RIGHT, UP, DOWN) that replaced the cross at the central fixation point.

The cues accurately indicated the location of the target on approximately 50% of the trials for the exogenous cues, and approximately 80% of the trials for the endogenous cues.

The primary cue comparison is between exogenous and endogenous cues, the rationale being that children with SBM have midbrain and posterior cortex compromise, both brain regions that are important for exogenous orienting. The rationale for including two types of endogenous cues was that, because children with SBM have better verbal than visual perception skills, an endogenous word might be a better cue than an endogenous spatial locator such as an arrow.

Children had five practice trials prior to each block, and then completed two blocks of at least 80 trials each, for each cue type. There were two blocks of trials of 80 trials for each cue type, making six blocks in all. Block order was randomly determined. There was a fixed inter-trial interval of 1500 ms. Any trial with a reaction time less than 150 ms or greater than 2000 ms was considered spoiled and was excluded from analyses, and was presented again after trial 80 in each block.

Participants were given the following instructions: “In this computer game, a star will appear in one of those boxes. Your job is to make the star go away as quickly as you can by pressing the blue button on this box. There are three clues as to which box the star will appear in: a bright box, an arrow, or a word. Let’s take a look at the different clues. The bright box is not such a good clue, but the arrow and word are pretty good clues about where the star is going to be. Put your chin in this chin rest and rest your forehead against the black bar. There is one more important thing. Always keep looking at the center of the screen where the cross is. Try not to move your eyes, no matter what happens. You will be able to see the star in all the boxes without moving your eyes. Let’s try some for practice. Keep your finger on the blue button.”

Examiners monitored eye movements throughout the session, and participants were reminded to maintain fixation throughout each task. Only one participant was excluded from analyses because of the inability to maintain fixation.

Recent studies of covert orienting in typically developing children using a variation of the Posner paradigm have not used invasive eye movement monitoring (Rueda et al., 2004), and, in studies of covert orienting with young people, eye movements that slow performance seldom exceed 4% (Rafal et al., 1988).

2.3. Brain imaging procedures

Structural MRI scans were obtained from 94 participants (69 SBM, 25 controls). Two pediatric neuroradiologists coded the state of the midbrain. Tectal beaking was coded as either present or absent. In our sample, 56 children with SBM had tectal beaking, and 13 did not. Fig. 1 shows a normal tectum and two examples of tectal beaking from the group with SBM. Fifty-two children of those who completed the attention tasks (34 with SBM, 18 controls) had structural magnetic resonance (MR) brain scans that were artefact-free and suitable for quantitative segmentation.

2.3.1. Image acquisition

Three sets of images were acquired, including a T1-weighted coronal series for assessment of white and gray matter and a T2-weighted coronal series for assessment of CSF. To co-register and position normalize the scans, external fiducial markers were placed on the nasion, and external meatus. An initial series (spin echo T1-weighted sagittal localizer, FOV 24, TR 500, TE 14, 256×192 matrix, 3 mm skip 0.3, two repetitions) was used for anatomical landmark identification. One whole-brain coronal series consisted of a fast spin echo Proton density and heavily T2-weighted images (FOV 20, TR 4000, TE1 15, TE2 112, 256×192 matrix, with two repetitions). This series was obtained in contiguous 1.5 mm slices across the whole brain. Another whole-brain coronal series consisted of a 3D-spoiled grass (3D SPGR) gradient echo contiguous 1.5 mm coronal series (TR 21, TE4, Flip angle 35° , 124 locations, 256×192 matrix, one repetition).

2.3.2. Image preprocessing

Prior to tissue segmentation, each slice series was stored in a single volume file and the pixel grayscale limits were

expanded by increasing the gain within the 0–255 (byte data) range. Each sequence volume was then reformatted so that voxel dimensions were isotropic. The T1- and T2-weighted reformatted volumes were aligned with each other through the use of the fiducial markers. Rigid-body translation and rotation routines programmed in IDL software were used for the realignment procedure itself, which was manually and visually checked at each step. Each volume was placed within a 256 cubic voxel bounding box with the fiducial marker cross point placed at the center of the volume. The two reformatted and aligned volumes were filtered using a non-linear anisotropic diffusion filter, which increased the overall signal-to-noise ratio of each volume an average of 100% (Gerig, Kubler, Kikinis, & Jolesz, 1992). This automated non-linear filter served to sharpen areas of high intensity gradient (boundaries) and to smooth regions of low-intensity gradient within the tissue borders.

2.3.3. Automatic segmentation

The method used a fully automated fuzzy cluster analysis (Pao, 1989) that obtained whole brain and regional brain tissue and CSF volumes (Brandt et al., 1992, 1994, 1996). The T1-weighted scan volume, which provides superior white-gray contrast compared to the T2-weighted scan, was used to obtain white and gray matter tissue volumes. The T2-weighted scan was fuzzy clustered separately from the T1-weighted scan to extract CSF volumes, and this was used to adjust the white and gray matter volume measures obtained from the T1-weighted volume. Solution images were derived from the final computed fuzzy cluster membership values for each voxel, which could then be viewed graphically on screen and compared with the actual scan images.

For the *quantitative* analyses, separate tissue volumes (white matter, gray matter, CSF) were obtained for various cortical regions including the whole brain, each hemisphere and three regions based on a division of the corpus callosum into a precallosal region, pericallosal (including the corpus callosum) and retrocallosal region (Filipek et al., 1992). In this categorization, the pericallosal region subtended the coronal brain volume extending from the most anterior to the most posterior aspect of the corpus callosum. The pre-



Fig. 1. Tectal beaking in mid-sagittal structural MRI slices. Control participant with normal tectum (left), SBM participant with normal tectum (middle), and SBM participant with beaked tectum (right). Arrows point to midbrain/tectal area. In our sample, 56 children with SBM had tectal beaking, and 13 did not.

callosal region extended fully frontally from the pericallosal region and the retrocallosal region extended fully posterior from the pericallosal region. For the present study, the regions of interest are: total retrocallosal region gray matter; total retrocallosal region white matter; and total retrocallosal region cerebrospinal fluid; retrocallosal region left gray matter; retrocallosal region left white matter; retrocallosal region left cerebrospinal fluid; retrocallosal region right gray matter; retrocallosal region right white matter; and retrocallosal region right cerebrospinal fluid. The percent retrocallosal volumes were calculated as the absolute total retrocallosal volumes divided by the absolute whole retrocallosal volumes.

The method used for subdividing the brain was developed for morphometric cortical parcellation studies of the normal brain, and has subsequently been applied in many studies involving morphometric analysis of MRI in a variety of conditions, including dyslexia, autism, and various acquired disorders (Filipek et al., 1992). The anatomical subdivision has been validated in various behavior–brain studies (Fletcher et al., 1996). Although attempting more refined subdivisions would be of interest, it has not been our experience that finer subdivisions are reliably achieved in SBM because of the variable appearance of different anatomical landmarks. This subdivision has established reliability in children with hydrocephalus (Brandt et al., 1996).

3. Results

3.1. Uncued orienting

Reaction times for each trial were recorded and median reaction time by quadrant for each child was calculated, and then averaged for each group. Between-group comparisons of reaction times were made using group \times quadrant repeated measures ANOVA. In addition, we calculated the average standard deviation by quadrant for each group, and variability between groups was compared using group \times quadrant repeated measures ANOVA. Post hoc analyses of significant effects were made using Bonferroni corrected comparisons.

Children with SBM detected targets as rapidly as controls in all four quadrants ($p > 0.2$; Table 2). Targets were detected most rapidly in the upper left quadrant for both groups of children (main effect of quadrant, $F(3, 118) = 15.23$, $p < 0.001$). Comparisons of variability in reaction times revealed no differences between groups ($p > 0.4$), and variability in reaction times was lowest in the upper left quadrant and greatest in the lower left quadrant, $F(3, 118) = 4.32$, $p < 0.01$. Children with SBM as a group do not show neglect in any one quad-

rant of space, and they do not differ from controls in uncued orienting.

3.2. Cued orienting

There were two measures of interest in the cued orienting task, response speed and disengagement cost.

3.2.1. Response speed

Median reaction time for each child was calculated by cue type (exogenous, endogenous arrow cue, endogenous word cue), plane (horizontal, vertical), SOA (200 ms or 1000 ms), and trial validity (valid, invalid), and then averaged for each group. These data were analyzed statistically. Separate comparisons were made for each cue type and each SOA, using group \times cue-validity \times plane repeated measures ANOVA, covarying for age. Post hoc analyses of significant effects were made using Bonferroni corrected comparisons.

3.2.2. Disengagement cost

The cue-validity effect may be described as the cost of misdirecting attention on invalid trials. We defined a disengagement cost measure as the difference between median reaction time on valid and invalid trials, and calculated this measure for each child by cue type and cue-target interval. This measure is a relative score accounting for individual differences in reaction time. Disengagement cost data were presented graphically, but not analyzed statistically because these data are isomorphic with the cue-validity effect.

3.2.3. Structure–function comparisons

Structure–function comparisons were theoretically motivated by relations described in the adult brain damage literature. We were particularly interested to test relations between the midbrain and exogenous cues, and between right posterior brain volumes and endogenous cues.

To examine the relationship between tectal dysmorphology and orienting attention to the exogenous cue, median reaction time data were analyzed using group (control; SBM-no tectal beaking; SBM-tectal beaking) \times cue-validity \times plane repeated measures ANOVA, covarying for age. In addition, partial correlations between median reaction time and right posterior cortical volume were made for the SBM group, covarying for age.

To examine the relationship between brain dysmorphology and orienting attention to the endogenous cues, partial correlations (between median reaction time and anterior brain volume, and between median reaction time and right posterior

Table 2
Average reaction time by quadrant, uncued orienting attention task [mean (standard deviation) of median reaction time data]

Group	Upper right	Upper left	Lower right	Lower left
Control	437.03 (107.5)	412.4 (99.2)	423.06 (106.1)	431.12 (119.4)
Spina bifida	473.61 (129.5)	443.66 (118.0)	453.42 (126.6)	463.68 (132.0)

cortical volume) were made for the SBM group, covarying for age, for each of the endogenous cue types.

3.3. Cued orienting, exogenous cue

The reaction time data are shown in Fig. 2 (short SOA) and Fig. 3 (long SOA). Children with SBM were slower than typically developing children at both SOAs (main effect of group, short SOA: $F(1, 118) = 23.40, p < 0.001$; long SOA: $F(1, 118) = 23.21, p < 0.001$). At the short SOA, all participants were slower at responding to invalidly cued targets than to validly cued targets (main effect of cue-validity, $F(1,$

$118) = 27.56, p < 0.001$), and this effect was mediated by age (validity \times age interaction: $F(1, 118) = 8.78, p < 0.005$). In addition, although reaction times to targets in the vertical plane were longer than those in the horizontal plane for all participants at the short SOA (main effect of plane, $F(1, 118) = 4.02, p < 0.05$), there was a significant group \times cue-validity \times plane interaction ($F(1, 118) = 8.31, p < 0.01$). Whereas typically developing children showed comparable cue-validity effects in both planes, children with SBM showed a greater cue-validity effect in the vertical than in the horizontal plane (Bonferroni corrected post hoc comparisons, $p < 0.05$). At the long SOA, it is notable that there was no cue-validity effect ($p > 0.1$).

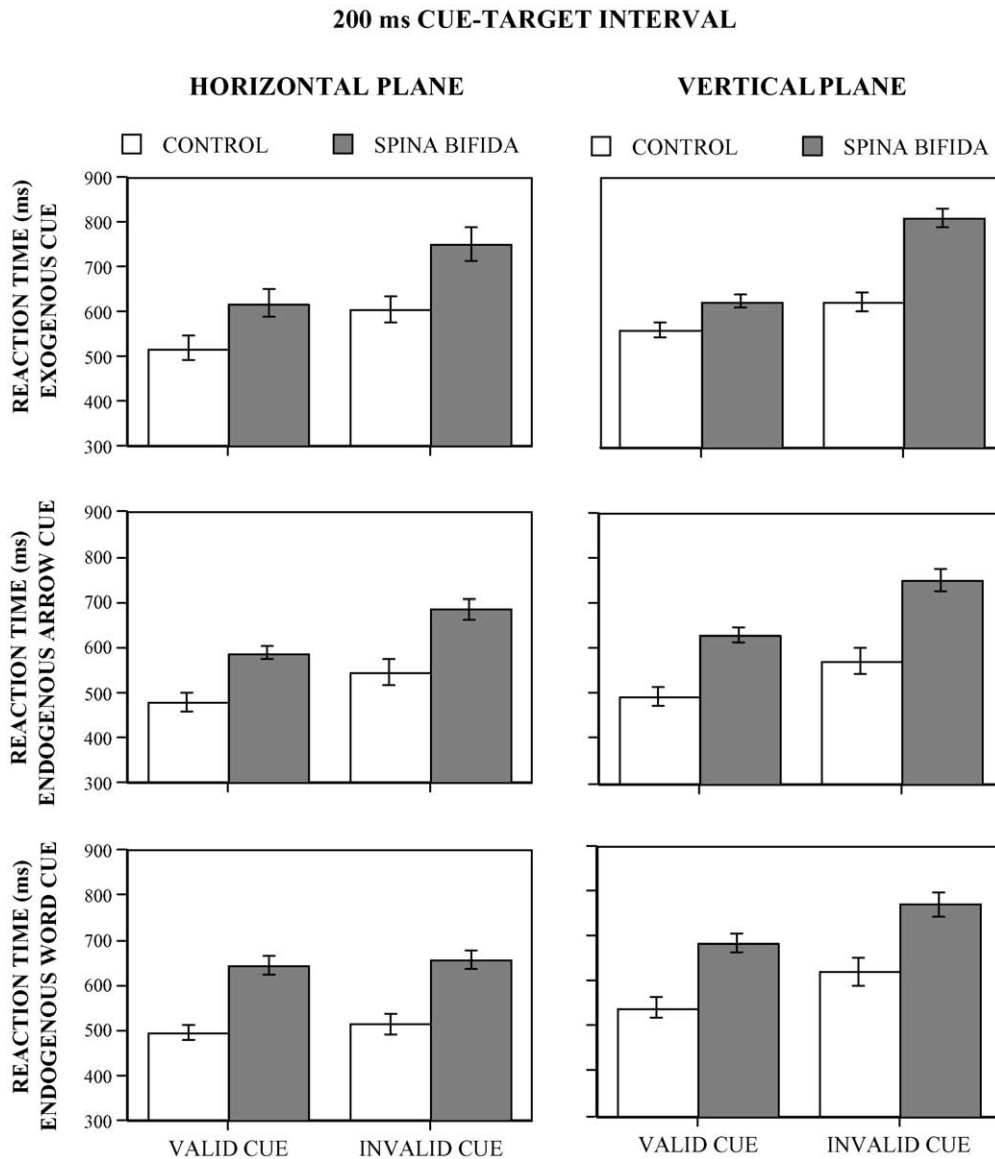


Fig. 2. Reaction time data by cue type, cue-validity, and plane, for the short SOA. Top: Significant effects of group, plane, cue-validity, and group \times plane \times cue-validity. Children with SBM show larger cue-validity effects in the vertical plane than the horizontal plane; controls show comparable cue-validity effects in both planes. Middle: Significant effects of group, cue-validity, and group \times plane. Children with SBM are slower at detecting targets in the vertical plane than the horizontal plane; reaction times of controls are comparable in both planes. Bottom: Significant effects of group and plane. No cue-validity effect (for statistics, see Section 3).

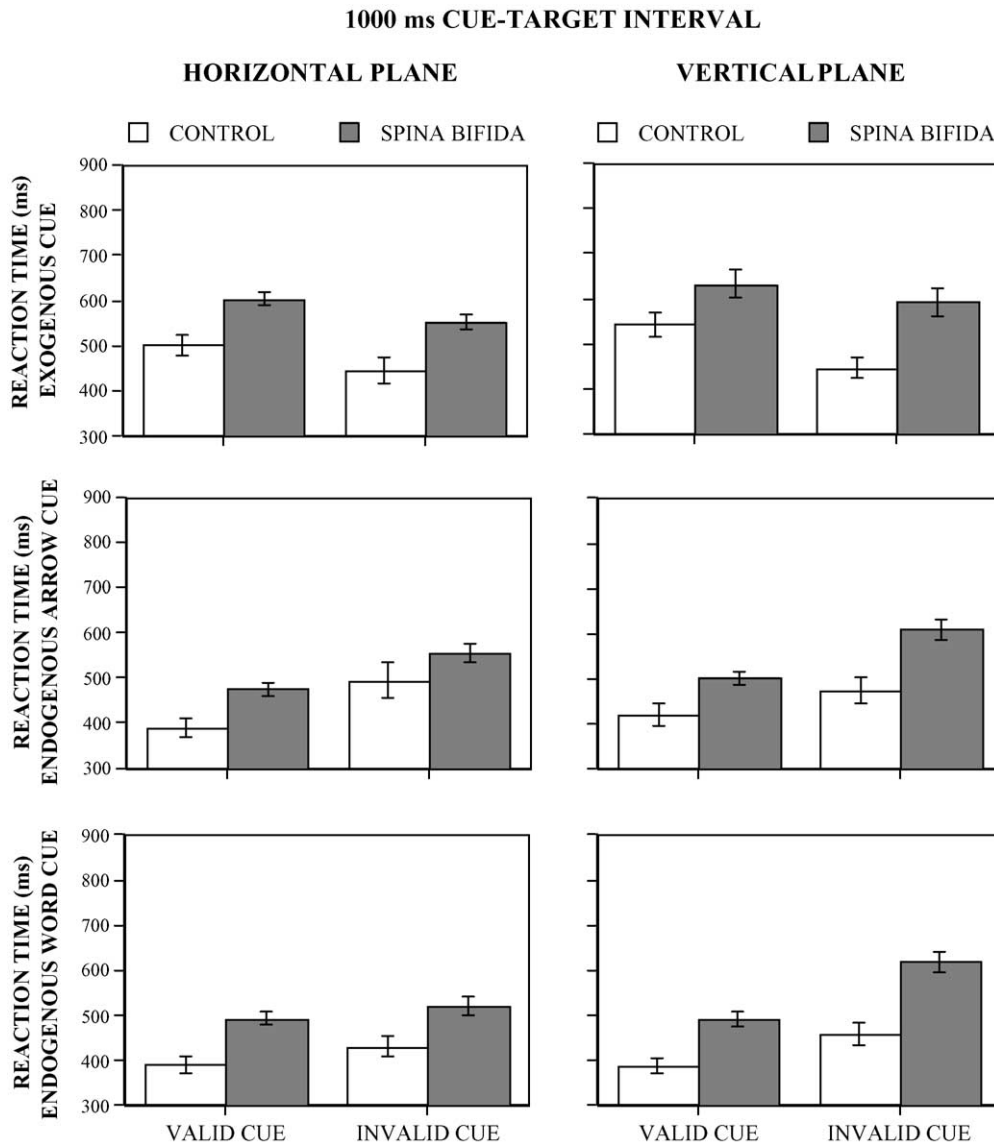


Fig. 3. Reaction time data by cue type, cue-validity, and plane, for the long SOA. Top: Significant effect of group. No cue-validity effect. Middle: Significant effects of group and cue-validity. Bottom: Significant effects of group, cue-validity, and group \times plane. Children with SBM are slower at detecting targets in the vertical plane than the horizontal plane; reaction times of controls are comparable in both planes (for statistics, see Section 3).

3.3.1. Structure–function relationships: tectal beaking

As with the results described above, we found main effects of group and plane at both SOAs. Overall, children with tectal beaking had slower reaction times (Table 3; main effect of group, short SOA: $F(2, 90) = 10.96, p < 0.001$; long SOA: $F(2, 90) = 13.74, p < 0.001$), being slower at detecting targets than either typically developing children or children with SBM and no tectal beaking (Bonferroni corrected pairwise comparisons, $p < 0.05$). Reaction times to targets in the vertical plane were longer than those in the horizontal plane for all participants (main effect of plane, short SOA: $F(1, 90) = 4.29, p < 0.05$; long SOA: $F(1, 90) = 4.99, p < 0.03$).

At the short SOA, all participants were slower at responding to invalidly cued targets than to validly cued targets (main

effect of cue-validity, $F(1, 90) = 20.11, p < 0.001$), and this effect was mediated by age (validity \times age interaction: $F(1, 90) = 6.96, p < 0.01$). There was also a significant group \times cue-validity interaction at the short SOA ($F(2, 90) = 3.37, p < 0.04$) such that children with tectal beaking showed a larger cue-validity effect than either controls or children with SBM with no tectal beaking ($p < 0.05$).

At the long SOA, in contrast, we found a group \times cue-validity interaction ($F(2, 90) = 4.03, p < 0.02$). At the long SOA, controls and children with SBM and no tectal beaking were faster at detecting invalidly cued targets than validly cued targets whereas children with tectal beaking did not show this inhibition of return (IOR) effect (further analyses of IOR are in Dennis et al., in press).

Table 3
Average reaction time by SOA and cue-validity for the exogenous cue [mean (standard deviation) of median reaction time data]

Plane	Group	Cue type	200 ms SOA	1000 ms SOA
Horizontal	Control	Valid	527.64 (134.20)	496.22 (138.34)
		Invalid	609.26 (143.96)	422.46 (110.35)
	Spina bifida-no tectal beaking	Valid	521.96 (123.37)	542.27 (138.73)
		Invalid	645.08 (167.10)	439.81 (120.84)
	Spina bifida-tectal beaking	Valid	646.86 (153.78)	617.08 (140.25)
		Invalid	776.82 (192.45)	583.85 (151.25)
Vertical	Control	Valid	549.02 (129.47)	538.82 (156.20)
		Invalid	625.34 (160.70)	435.86 (117.52)
	Spina bifida-no tectal beaking	Valid	540.35 (104.20)	573.19 (138.26)
		Invalid	676.31 (158.09)	490.69 (159.23)
	Spina bifida-tectal beaking	Valid	648.66 (143.93)	651.80 (140.66)
		Invalid	830.25 (196.17)	624.82 (189.41)

3.3.2. Structure–function relationships: right posterior cortical volume

In children with SBM, retrocallosal white matter volume on the right was negatively correlated with median reaction time to invalidly cued targets in the vertical plane at the long SOA ($r = -0.40$, $p = 0.02$).

3.4. Cued orienting, endogenous arrow cue

Children with SBM were slower than controls at both SOAs (Figs. 2 and 3; main effect of group, short SOA: $F(1, 118) = 27.77$, $p < 0.001$; long SOA $F(1, 118) = 12.92$, $p < 0.001$). All participants were slower at detecting invalidly cued targets than validly cued targets (main effect of cue-validity, short SOA: $F(1, 118) = 22.61$, $p < 0.001$; long SOA: $F(1, 118) = 18.02$, $p < 0.001$), and this effect was mediated by age (cue-validity \times age interaction, short SOA: $F(1, 118) = 7.31$, $p < 0.01$; long SOA: $F(1, 118) = 5.95$, $p < 0.02$). At the short SOA, children with SBM were slower than controls at detecting targets in the vertical plane than in the horizontal plane (group \times plane interaction, $F(1, 118) = 4.36$, $p < 0.05$), but there was no effect of plane for controls.

3.4.1. Structure–function relationships: posterior and anterior brain volumes

In children with SBM, reaction time to horizontal validly cued targets, presented within the long SOA, was negatively correlated with right retrocallosal white matter volume ($r = -0.49$, $p = 0.003$) and with precallosal white matter volume ($r = -0.44$, $p = 0.01$).

3.5. Cued orienting, endogenous word cue

Children with SBM were slower than controls at detecting targets (main effect of group, short SOA: $F(1, 118) = 24.97$, $p < 0.001$; long SOA: $F(1, 118) = 22.54$, $p < 0.001$). At the short SOA, all participants were slower at responding to cues in the vertical plane than the horizontal plane (Figs. 2 and 3;

main effect of plane: $F(1, 118) = 6.47$, $p < 0.02$). At the long SOA, in contrast, only children with SBM showed a slower reaction time in the vertical plane compared to the horizontal plane (group \times plane interaction, $F(1, 118) = 3.93$, $p < 0.05$). There was no cue-validity effect for the word cue at the short SOA.

3.5.1. Structure–function relationships: posterior and anterior brain volumes

In children with SBM, reaction time to targets in the horizontal plane presented within the long cue-target interval was negatively correlated with right retrocallosal white matter (valid cued target: $r = -0.37$, $p = 0.03$; invalidly cued target: $r = -0.57$, $p = 0.001$), and positively correlated with right retrocallosal gray matter (invalidly cued targets only; $r = 0.38$, $p = 0.03$). Reaction time to invalidly cued targets presented in the horizontal plane within the long SOA was negatively correlated with precallosal white matter ($r = -0.46$, $p = 0.006$) and positively correlated with precallosal grey matter ($r = 0.36$, $p = 0.04$).

3.5.2. Disengagement cost

The disengagement cost data are shown in Fig. 4, which depicts the group differences in the cue-validity effect for the three cue types. Fig. 4 summarizes and confirms the reaction time analyses. For exogenous but not endogenous cues, and relative to typically developing peers, children with SBM showed a larger disengagement cost in the vertical plane for targets cued at the short interval.

4. Discussion

Children with SBM have impairments in covert orienting that concern attention disengagement. The deficits are not global, however, because these children performed as well as controls on a task of uncued orienting as well as on a number of cued orienting measures.

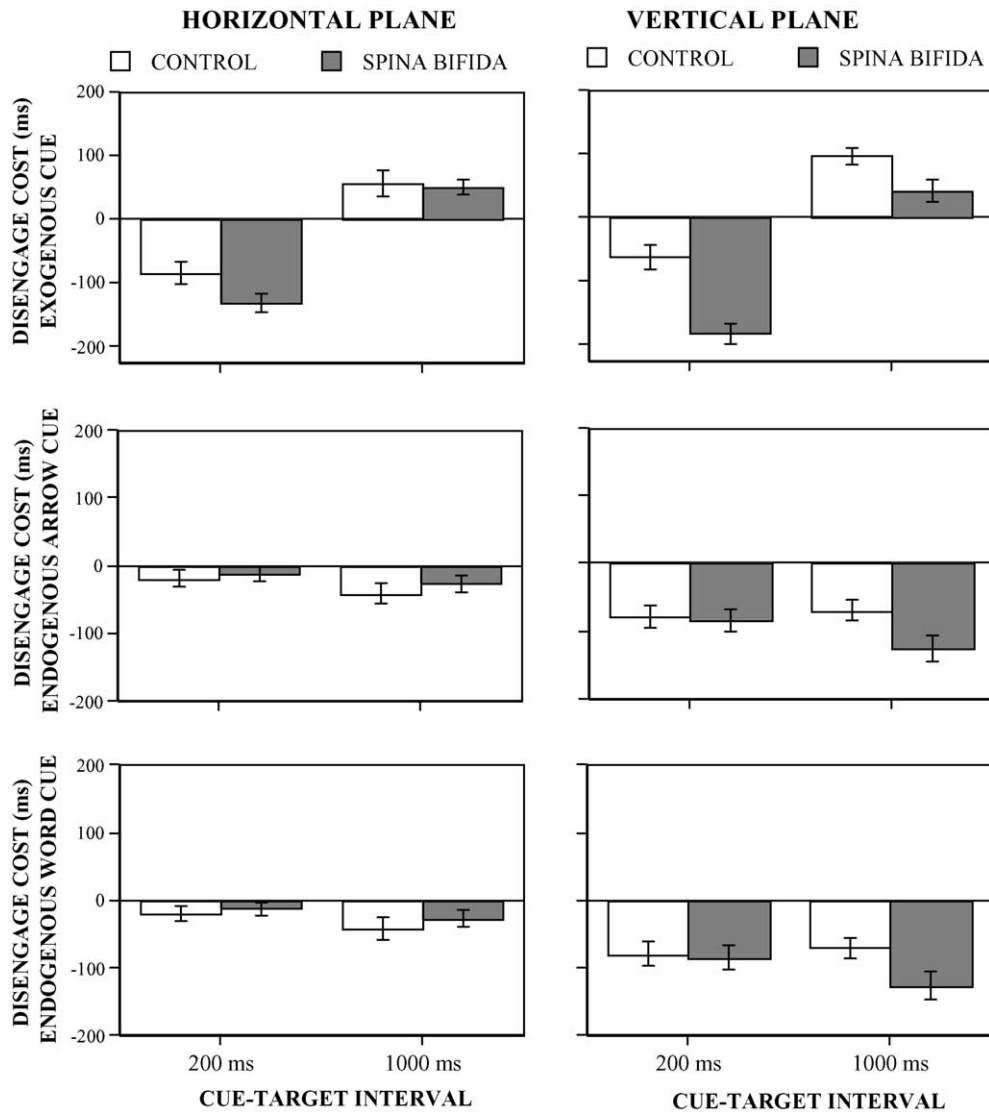


Fig. 4. Disengagement cost data by cue type and stimulus onset asynchrony (SOA).

Both children with SBM and controls showed the typical covert orienting task effects. Like typically developing children (Akhtar & Enns, 1989; Brodeur et al., 1997; Enns & Brodeur, 1989; Jonides, 1980), they showed a cue-validity effect (they responded more rapidly to valid than to invalid cues for both horizontal and vertical planes and for each cue type) that varied with age (e.g., Akhtar & Enns, 1989), as well as exhibiting greater disengagement costs at shorter SOAs.

Nevertheless, the children with SBM differed from controls. They responded more slowly in both planes; they were slower responding to invalid cues in the vertical plane; and, like younger children, they were more disadvantaged by a short SOA. In this respect, children with SBM performed like younger children, who require longer SOAs for successful covert orienting.

Children with SBM differed from controls primarily in the vertical plane, where they had longer reaction times and

greater disengagement costs, especially for invalid cues. Clinically, these children commonly show a Parinaud syndrome during shunt blocks. Although they were not symptomatic during testing, it appears that the vertical plane is highly vulnerable to attention deficits in this population (see also Dennis et al., 2003).

Group differences in the cue-validity effect occurred for the exogenous cue, but for neither of the endogenous cues, demonstrating a cost for attentional disengagement from salient but not interesting information. These data confirm impaired disengagement in children with hydrocephalus (Brewer et al., 2001) in a large, independent sample of children with hydrocephalus and SBM, adding the new information that the disengagement deficit is specific to exogenous cues.

For children with SBM, directional symbols and closed-class prepositions that represent orientation in egocentric

space produced similar covert orienting. The interpretation of this result is not entirely clear; how language maps on to space is not fully understood, even in normal populations (Hayward & Tarr, 1995).

The broad dissociation between exogenous and endogenous cues occurred at a neural as well as a behavioral level. The data bear on the developmental role of the midbrain superior colliculus in exogenous orienting and the role of the cortical attention system in endogenous orienting.

The superior colliculus is important for saccade generation and for bringing visual and motor information together in a common reference frame. Specifically, retinotectal pathways are important for reflex orienting to exogenous signals, and for linking covert attention to the oculomotor systems of overt orienting (Rafal et al., 1988). Children with SBM and tectal beaking were slower orienting to exogenous cues, as well as being slower at other types of visual attention involving overt movement of the effectors (Dennis et al., 2003), which suggests that the midbrain, which includes the superior colliculus, is important for both overt and covert orienting in children. Recent computational models of the superior colliculus propose that its function is to bring visual and motor information together in a common reference frame (Optican, 1995). Covert orienting deficits are evident in both congenital midbrain dysmorphology and adult midbrain degeneration, and likely contribute to deficits in integrating vision and movement that have been described in each of these conditions (e.g., Dennis, Fletcher, Rogers, Hetherington, & Francis, 2002; Rafal et al., 1988).

As with the cortical attention system in the mature brain, the developing brain shows relations among regional brain volume measures and components of attention. The parietal lobes are thought to be involved in attentional disengagement (Posner et al., 1984). In both the mature and the immature brain, right posterior brain regions appear to be important for successful disengagement from attentionally salient information, and, perhaps more broadly, for creating saliency maps (Treue, 2003). Anterior brain regions were correlated with reaction time to valid targets, although not to invalid targets, or to invalid, verbally cued information.

Correlations between brain volumes and attention function existed for white matter rather than gray matter. This underscores the fact that significant white matter disturbances are common in SBM as well as in many other neurodevelopmental disorders, and that white matter volume is predictive of neurocognitive function in children with SBM (Fletcher et al., 2000).

The study of covert orienting in children with SBM, who have brain dysmorphologies in the midbrain and posterior cortex, enhances understanding of the SBM condition. More generally, it provides converging evidence for the importance of critical components of the distributed neural system of attention orienting—specifically, the midbrain and the right parietal lobe—that have been identified in the mature brain.

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