Impairment in Shifting Attention in Autistic and Cerebellar Patients

Eric Courchesne, Jeanne Townsend, Natacha A. Akshoomoff, Osamu Saitoh, Rachel Yeung-Courchesne, Alan J. Lincoln, Hector E. James, Richard H. Haas, Laura Schreibman, and Lily Lau

MRI and autopsy evidence of early maldevelopment of cerebellar vermis and hemispheres in autism raise the question of how cerebellar maldevelopment contributes to the cognitive and social deficits characteristic of autism. Compared with normal controls, autistic patients and patients with acquired cerebellar lesions were similarly impaired in a task requiring rapid and accurate shifts of attention between auditory and visual stimuli. Neurophysiologic and behavioral evidence rules out motor dysfunction as the cause of this deficit. These findings are consistent with the proposal that in autism cerebellar maldevelopment may contribute to an inability to execute rapid attention shifts, which in turn undermines social and cognitive development, and also with the proposal that the human cerebellum is involved in the coordination of rapid attention shifts in a fashion analogous to its role in the coordination of movement.

The concept that the cerebellum might influence higher mental operations in humans is not a new concept but one that began to emerge more than 70 years ago. At that time, Beyerman (1917) observed that “cerebello-ataxic mental deficiency” (p. 651) occurred in some individuals who did not have extracerebellar pathology. More than 40 years ago, Snider (1950, p. 219) anticipated recent theory and the present research study by declaring that one should adopt broader concepts of cerebellar functions [which] must encompass cerebellar influences on the sensory and motor centers of the cerebrum as well as related influences on the diencephalic, mesencephalic, and medullary centers. The cerebellum stands out as “the great modulator of neurologic function,” and new horizons of cerebellar action are introduced into neurology and psychiatry [italics added].

Although the narrower concept that the cerebellum is primarily linked to motor control and motor learning continues to dominate contemporary views on the role of the cerebellum, the concept that the cerebellum might influence higher mental operations in humans is not a new concept but one that began to emerge more than 70 years ago. At that time, Beyerman (1917) observed that “cerebello-ataxic mental deficiency” (p. 651) occurred in some individuals who did not have extracerebellar pathology. More than 40 years ago, Snider (1950, p. 219) anticipated recent theory and the present research study by declaring that one should adopt broader concepts of cerebellar functions [which] must encompass cerebellar influences on the sensory and motor centers of the cerebrum as well as related influences on the diencephalic, mesencephalic, and medullary centers. The cerebellum stands out as “the great modulator of neurologic function,” and new horizons of cerebellar action are introduced into neurology and psychiatry [italics added].

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Like the Hamilton et al. (1983) and Botze et al. (1985) conclusions, these conclusions were initially derived from observations of patients, but in this case, autistic patients. In the very first description of an autistic patient, it was noted that arousal, attention, and sensory responsiveness were severely impaired (Kanner, 1943), and in the first neurobiologic theory of autism, disturbances in the functioning of the reticular activating system (RAS) were hypothesized to underlie the characteristic social and cognitive deficits in this disorder (Rimland, 1964). The first evidence of neuropathology in autism was Purkinje neuron loss throughout the cerebellum of a single patient (Williams, Hauser, Purpura, DeLong, & Swisher, 1980); Purkinje neurons are key to cerebellar function. No other pathology was observed in that patient. The question, then, was whether this cerebellar finding in a single autopsy case was anomalous and of no significance in explaining the attention, arousal, sensory, and social symptomatology in autism.

The cerebellum has been shown in animals to have physioanatomical connections with the RAS and attentional systems (Itoh & Mizuno, 1979; Kitano, Ishida, Ishikawa, & Murayama, 1976; Moruzzi & Magoun, 1949; Nieuwenhuys, Voogd, & van Huijzen, 1988; Sasaki, Jinnai, Gemba, Hashimoto, & Mizuno, 1979; Sasaki, Matsuda, Kawaguchi, & Mizuno, 1972; Schnallmann & Pandya, 1989; Snider, 1950; Steriade & Stoupel, 1960; Vilensky & Van Hoesen, 1981), and to have a modulatory effect on sensory responsiveness (Crispino & Bullock, 1984). The cerebellum also has physioanatomical connections with brainstem, thalamic, and parietal systems implicated in shifting attention in studies of humans with focal lesions (Posner & Petersen, 1990; Posner, Walker, Freidrich, & Rafal, 1984; Rafal & Posner, 1987; Rafal, Posner, Freidman, Inhoff, & Bernstein, 1988). Therefore, substantial Purkinje neuron loss throughout the cerebellum could very well be of great significance in explaining attention, arousal, and sensory dysfunction in autism.

We hypothesized, therefore, (a) that the cerebellum might play a role in the coordination of attention in a fashion analogous to the role it plays in motor control and (b) that in autism, cerebellum maldevelopment is a consistent feature that renders the child unable to adjust his or her mental focus of attention to follow the rapidly changing verbal, gestural, postural, tactile, and facial cues that signal changes in a stream of social information (Courchesne, 1985, 1987, 1989a, 1989b, 1991; Courchesne et al., 1988; Courchesne, Chisum, & Townsend, in press; Courchesne, Townsend, et al., 1994). Such cues signal the normal child to move his or her "spotlight of attention" from one source of information (e.g., auditory) to another (e.g., visual). This process involves disengaging attention from one source and then moving and reengaging it on another (i.e., inhibition of one source and enhancement of another; Posner et al., 1984; Posner & Petersen, 1990). To selectively adjust the focus of attention, the nervous system must quickly and accurately alter the pattern of neural responsiveness to sensory signals—from an enhanced neural response to certain stimuli (e.g., vocalizations) to an enhanced response to other stimuli (e.g., gestures) and from inhibited neural response to some stimuli to inhibited response to others.

Our earlier prediction that cerebellar pathology would be a consistent feature of infantile autism has now been verified by 16 quantitative magnetic resonance (MR) and autopsy reports from nine independent research groups involving 240 autistic cases (see Table 1). Two distinctly different forms of cerebellar pathology have been found (Courchesne, Saitoh, et al., 1994; Courchesne, Townsend, & Saitoh, 1994). One form is hypopla-

### Table 1

**Quantitative Magnetic Resonance (MR) and Autopsy Studies of the Cerebellum in Infantile Autism**

<table>
<thead>
<tr>
<th>Studies finding abnormalities</th>
<th>Researchers</th>
<th>Study</th>
<th>Studies finding no abnormalities</th>
<th>Researchers</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bauman &amp; Kemper (1985)</td>
<td>Autopsy</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bauman &amp; Kemper (1986)</td>
<td>Autopsy</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ritvo et al., 1986</td>
<td>Autopsy</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gaffney, Tsai, Kuperman &amp; Minchin (1987)</td>
<td>MR</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bauman &amp; Kemper (1990)</td>
<td>Autopsy</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ciesielski, Allen, et al. (1990)</td>
<td>MR</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Arin, Bauman, &amp; Kemper (1991)</td>
<td>Autopsy</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bauman (1991)</td>
<td>Autopsy</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Piven et al. (1992)</td>
<td>MR*</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Kleiman, Neff, &amp; Rosman (1992)</td>
<td>MR*</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Courchesne, Saitoh, et al. (1994)</td>
<td>MR</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Saitoh, Courchesne, Egaas, Lincoln, &amp; Schreibman (in press)</td>
<td>MR</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hashimoto et al. (in press)</td>
<td>MR</td>
<td></td>
<td>MR</td>
<td></td>
</tr>
</tbody>
</table>

*Evidence based on reanalyses of study data by Courchesne, Townsend, & Saitoh (1994).
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The midsagittal area of vermian lobules VI–VII from each of 91 normal subjects (a) and 78 autistic patients (b) from separate MR studies of the vermis (Courchesne, Yeung-Courchesne, Press, Hesselink, & Jernigan, 1988; Courchesne, Saitoh, et al., 1994; Kleiman, Neff, & Rosman, 1992; Piven et al., 1992; Raz, Torres, Spencer, White, & Acker, 1992). Graphs show the degree to which the size distributions of vermian lobules VI–VII approximate a unimodal Gaussian curve of normal distribution. A perfectly normal unimodal distribution of data forms a straight line when plotted against expected values on a Gaussian curve. Data from autistic patients (b) show that the distribution of lobules VI–VII size deviates significantly (p < .001) from a normal distribution. Instead, the distribution is bimodal. Specifically, there are two separate clusters of autistic patients: one with smaller than normal lobules VI–VII (88% of all patients) and the second with enlarged lobules VI–VII (12% of all patients). Data in (a) from normal subjects (Courchesne et al., 1988; Courchesne, Saitoh, et al., 1994; Raz et al., 1992) closely approximated a straight line for vermian lobules VI–VII. The line from these normal data is graphed for comparison against the autism data (b). The arrows point to individual autistic and normal subjects who were randomly selected to participate in the present study of shifting attention. Courchesne 1 = Courchesne et al., 1988; Courchesne 2 = Courchesne, Saitoh, et al., 1994.
as quantified by MR techniques (Saitoh, Courchesne, Egaas, Lincoln, & Schreibman, in press).

To test our theory that the cerebellum contributes to the dynamic, moment-to-moment control of the mental focus of attention, in a preliminary study (Courchesne, Akshoomoff, & Ciesielski, 1990) we examined the ability of 10 adult autistic patients to alternate attentional focus between auditory and visual stimulation. The task was a distillation of the social world, in which cues presented at unpredictable time intervals direct patients to voluntarily initiate shifts of attention (see Figure 2). We found that these autistic patients were unable to accurately alternate attentional focus if required to do so rapidly (within < 2.5 s) but were accurate when given much more time (2.5 to 30 s) to shift their attention between auditory and visual stimulation. They had no difficulty maintaining a fixed focus of attention on one modality for many seconds. To verify that this shifting attention deficit was due to cerebellar pathology, we tested patients with acquired cerebellar lesions and other patients with acquired cerebral lesions in similar shift and focus attention tasks (Akshoomoff & Courchesne, 1992, 1994). The cerebellar patients were significantly impaired in their ability to make rapid and accurate voluntary shifts of attention. Whereas cerebellar patients required several seconds to successfully shift attention, normal control subjects in our study required less than 1 s. This is consistent with previous literature showing that normal adults may require as little as 300 ms to shift attention (Sperling & Reeves, 1980; Weichselgartner & Sperling, 1987).

In the present study, a new group of autistic patients performed the identical shift attention task given to our patients with acquired cerebellar or cerebral lesions, and their performance was statistically compared with the published data on these two groups of lesioned patients as well as with chronologic- and mental-age-matched normal volunteer controls. Because disengaging of attention has been proposed to be impaired by parietal damage (Posner et al., 1984; Posner & Petersen, 1990) and some autistic patients have been reported to have parietal damage (Courchesne, Press, & Yeung-Courchesne, 1993; Egaas, Courchesne, & Saitoh, 1994), we included among the autistic patients in the present experiment those with and those without MR evidence of volume loss in posterior cerebral regions. In addition, we recorded event-related brain potentials (ERPs) from several autistic subjects in order to assay attention-related brain responses to stimuli that immediately followed cues to shift attention. We now report the results from these tests.

Method

Study Groups

Patients With Autism

Subjects were 8 patients (mean age = 13.9 years) diagnosed with infantile autism as defined by the revised third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R; American Psychiatric Association, 1987; see Table 2). All patients also passed the criteria for infantile autism defined by the Autism Diagnostic Interview (LeCouteur, et al., 1989) and the Autism Diagnostic Observation Schedule (Lord et al., 1989). In these patients, autism was not complicated by severe mental retardation, cerebral palsy, epilepsy, or other neurologic disease. DNA analyses demonstrated that all 8 were negative for fragile-X. Table 3 lists additional patient characteristics.

In addition to these 8, another autistic patient (Wechsler Adult Intelligence Scale—Revised [WAIS-R] Verbal IQ = 74; Performance

Figure 2. Schematic drawing of the shift attention (A) and focus attention (B) tasks. Visual stimuli were red and green (g) flashes; auditory stimuli were 2-kHz (hi) and 1-kHz (lo) tone pips. HIT = correctly detected target; MISS = a failure to respond to a target; FA = an erroneous response to a rare stimulus that was in a modality to be ignored; IGN = a rare stimulus that was correctly ignored. In the example of the shift experiment, the subject pressed a button (arrow) to the first rare visual target stimulus (RED). This served as a cue to shift attention to the auditory stimuli, ignore (IGN) the rare visual stimuli, and respond to the next auditory target (HI). The auditory target in turn served as a cue to shift attention back to the visual stimuli.
IQ = 92) was tested in the shift and focus attention tasks described further on. He was tested on six occasions, each separated by 1-2-week intervals. Because he was an adult (26 years of age), his data are presented separately and were not included in the statistical analyses with the 8 younger autistic patients.

**Measures of the cerebellum.** All 8 had participated in an MR study (Courchesne, Saitoh, et al., 1994) which demonstrated that there are two distinctly different types of vermal pathology in autism: hypoplasia of cerebellar vermal lobules VI–VII, which occurs in about 89% of autistic cases, and hyperplasia of vermal lobules VI–VII, which occurs in about 11% of autistic cases. Their MR data were also used in a meta-analysis of recent findings from other research laboratories which demonstrated comparable findings of vermal pathology across four research laboratories involving 78 total autistic patients (see Figure 1; Courchesne, Saitoh, et al., 1994). The 8 autistic subjects were selected strictly on the basis of age and availability for participation in the present study. Seven of the 8 had vermal hypoplasia and 1 had vermal hyperplasia (see values indicated by arrows in Figure 1B). Vermian lobules VI–VII in the 8 patients differed significantly from normal subjects are a representative sample of the larger population of autistic patients studied by several different laboratories. In the present study, these vermis measures serve as a convenient and reliable index of cerebellar abnormality in these autistic patients, but it should be reiterated here that autopsy and MR studies show that the cerebellar hemispheres as well as the vermis are abnormal in autism (Gaffney, Tsai, et al., 1987; Murakami et al., 1989).

**Measures of posterior corpus callosum.** A new MR study has reported qualitative evidence of parietal volume loss in 43% of autistic patients (Courchesne, Press, & Yeung-Courchesne, 1993). Following up on this qualitative finding, another new quantitative and statistical MR study found the posterior segments of the corpus callosum, which include axons from parietal cortex, to be significantly reduced in cross-sectional size in 41 autistic patients compared with 41 age- and gender-matched normal healthy volunteer controls (Egaas et al., 1994); frontal segments were nonsignificantly different between autistic and normal groups.

In the present study, this quantitative measure of the posterior segment of the corpus callosum was used as an objective MR index of posterior cerebral abnormality. For 4 of the 8 autistic patients in the present study, the cross-sectional area of the posterior segment of the corpus callosum was 2.6 standard deviations below the normal mean reported by Egaas et al. (1994); also, each of these 4 autistic patients had areas that were smaller than even the autistic mean reported by Egaas et al. For the other 4 of the 8 autistic patients in the present study, the cross-sectional area of the posterior segment of the corpus callosum was 0.1 standard deviation below the normal mean reported by Egaas et al. (1994).

**Normal Controls**

Eighteen normal volunteer controls were tested (see Tables 2 and 3); they had no history of substance abuse, special education, major medical illness, psychiatric illness, or developmental disorder. MR scans of the cerebellar vermis were obtained from 7 of the normal controls. Figure 1A (see values indicated by arrows) shows that these 7 normal subjects are a representative sample of the larger population of normal volunteer subjects studied by several different laboratories.

### Table 2

**Matching Characteristics of Normal and Autistic Subjects**

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Age (years M ± SD)</th>
<th>WISC–R Block design (M ± SD)</th>
<th>WISC–R Object assembly (M ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Adolescent 6M, 2F</td>
<td>13.8 ± 0.8</td>
<td>11.0 ± 1.7</td>
<td>10.5 ± 2.6</td>
</tr>
<tr>
<td>Child 7M, 3F</td>
<td>8.6 ± 1.7</td>
<td>10.5 ± 2.8</td>
<td>12.0 ± 3.2</td>
</tr>
<tr>
<td>Autistic adolescent 6M, 2F</td>
<td>13.9 ± 1.6</td>
<td>11.8 ± 3.9</td>
<td>12.1 ± 3.1</td>
</tr>
</tbody>
</table>

**Note.** The Wechsler Intelligence Scale for Children—Revised (WISC–R) was administered to children and adolescents. Average Wechsler subtest scores for the normal population = 10.0 ± 3.0. M = male; F = female.

### Table 3

**Additional Characteristics of Subjects**

<table>
<thead>
<tr>
<th>Subject group</th>
<th>PPVT–R (M ± SD)</th>
<th>Verbal IQ (M ± SD)</th>
<th>Performance IQ (M ± SD)</th>
<th>Clinical seizure activity</th>
<th>Medication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Adolescent</td>
<td>118 ± 11.5</td>
<td>114 ± 10.5</td>
<td>107 ± 8.6</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Child</td>
<td>111 ± 12.1</td>
<td>108 ± 6.6</td>
<td>111 ± 12.2</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Autistic</td>
<td>54 ± 13.4</td>
<td>59 ± 13.7</td>
<td>89 ± 10.4</td>
<td>7/8: negative</td>
<td>7/8: none;</td>
</tr>
<tr>
<td>Cerebellar lesions</td>
<td>97 ± 14.8</td>
<td>97 ± 15.5</td>
<td>96 ± 23.2</td>
<td>—</td>
<td>1/8: Trilafon and Cogentin</td>
</tr>
</tbody>
</table>

**Note.** The Peabody Picture Vocabulary Test—Revised (PPVT–R) is a test of receptive language ability. Average PPVT–R and Wechsler Intelligence Scale for Children—Revised (WISC–R) IQ scores in the normal population = 100 ± 15.0. Note that the large discrepancy between Verbal and Performance IQ scores in this sample of autistic subjects was principally due to two very low Verbal subtest scores (Vocabulary and Comprehension: 2.8 and 1.4, respectively) and to two relatively normal Performance subtest scores (Block Design and Object Assembly: 11.8 and 12.1, respectively). Such an extreme discrepancy has been suggested to be typical of patients with infantile autism (Bartak, Rutter, & Cox, 1975; Lincoln, Allen, & Fiascentini, 1994; Lincoln, Courchesne, Kalman, Elmasian, & Allen, 1988; Ohta, 1987) and stands in contrast to the normally expected pattern of comparable scores across all subtests (e.g., in the normal adolescents, these four subtest scores were 11.6, 11.9, 11.0, and 10.5, respectively). Empty cells indicate that medical history was negative for abnormality or use of medication at time of testing.
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**Mental-age-matched normal controls.** Ten normal children (mean age = 8.6 years) were matched with the 8 autistic patients on mental age, which was based on the Wechsler Intelligence Scale for Children—Revised (WISC-R) Verbal IQ; this IQ index reflects areas of verbal and cognitive weaknesses typical of autism (see Table 2; Bartak, Rutter, & Cox, 1975; Lincoln, Allen, & Piacentini, 1994; Lincoln, Courchesne, Kilman, Elmasian, & Allen, 1988; Ohta, 1987). Specifically, the mean mental age of this normal control group was 9.3 years (chronologic age × VIQ/100), and that of the autistic adolescents was 8.2 years. The data from these 10 controls are from Akshoomoff and Courchesne (1992).

**Chronologic-age-matched normal controls.** The 8 older normal controls (mean age = 13.8 years) were matched with 8 autistic patients on chronological age, gender, and performance on selected subtests of the WISC-R that reflect nonverbal cognitive strengths in autism (see Table 2; Bartak et al., 1975; Lincoln et al., 1988, 1994; Ohta, 1987).

**Comparison Group: Patients With Acquired Cerebellar Lesions**

The data from these patients are from Akshoomoff and Courchesne (1992). Neocerebellar damage was substantial in 5 of these 6 patients. Five of the 6 patients were children who had undergone surgical resection of cerebellar astrocytomas, and, as in Akshoomoff and Courchesne (1992), they were age- and IQ-matched to the 10 normal children described above. The mean age at the time of diagnosis was 5.2 years (SD = 1.2), and the mean age at the time of testing was 8.6 years (SD = 1.8). Neurological symptoms associated with cerebellar damage had improved since the time of surgery but persisted in all patients (e.g., dysmetria and gait difficulty). Two received focal irradiation of the posterior fossa, but 3 did not; none of the patients received chemotherapy. Three patients had unilateral neocerebellar hemisphere lesions (including the ventral dentate nucleus in 1 case), and 1 patient had damaged superior cerebellar peduncles bilaterally, which thus eliminated the principal output pathways of the neocerebellum. The 5th patient had a small focal unilateral lesion and no indication of extensive damage to neocerebellar structures or input and output pathways. The 6th patient was a 21-year-old man with a normal developmental history who exhibited signs of an idiopathic cerebellar degenerative disorder at 16 years of age (Akshoomoff, Courchesne, Press, & Iraqui, 1992). He had extensive parenchymal volume loss in the cerebellum but relatively little cerebral cortical atrophy. Prior to his disability, this patient was an honors student in high school.

**Protocol for Neurobehavioral Testing**

Each subject participated in two experimental tasks—focus attention and shift attention—in which visual and auditory stimuli were randomly intermixed and rapidly presented. In each attention task, subjects were required to detect and press a button in response to specified target stimuli (see Figure 2). Responses were continuously digitized, and computer algorithms were used to score behavioral performance. Both the baseline focus task and the shift task required subjects to make equally rapid detections and responses to successive targets, but compared with the baseline task, the shift task additionally required subjects to shift attention between sensory modalities to make such successive correct detections.

We took several steps to ensure that test performance was not affected by head, hand, and eye position and movements. Each subject always had the button in hand and placed the thumb of the preferred hand on the top of the button; to respond, the subject merely had to squeeze down. Sounds were presented over headphones, so each subject did not have to move head or eyes to optimize sound detection.

Each subject was trained to maintain eye fixation on the video monitor, where visual stimuli were presented. Experimenters continuously monitored compliance during testing. In addition, compliance was electrophysiologically monitored by placing silver-silver chloride electrodes on one outer canthus and above and below one eye in 4 autistic patients and 2 cerebellar patients. These electrophysiologic recordings showed that the patients followed our instructions well and kept their eyes fixated on the location where the visual stimuli were presented.

Visual stimuli were red and green squares subtending 1.2° of visual angle and presented on a video monitor; auditory stimuli were 1- and 2-kHz binaural tones presented over headphones. All visual and auditory stimuli were 50 ms in duration, and interstimulus intervals varied between 450 and 1,450 ms. Twenty-five percent of all stimuli were target stimuli to which subjects were required to respond by pressing a button. Prior to beginning each experimental task, each subject was required to achieve at least a 75% overall correct response rate in practice trials; and once the experimental task was under way, performance data were included only if the overall correct response rate in any set sequence of 80 stimuli was at least 50%. A correctly detected target was scored as a hit if the response occurred between 200 and 1,400 ms after stimulus onset, and failure to respond to a target during this time window was a miss. An erroneous response to a nontarget stimulus was scored as a false alarm.

The focus attention task tested the subject's ability to continuously maintain a focus of attention and to detect rare target stimuli in one sensory modality (e.g., visual) while ignoring all stimuli in the other modality (e.g., auditory; see Figure 2B). This served as a control, or baseline, on the shift attention task. The focus attention task consisted of two conditions: a visual focus attention condition and an auditory focus attention condition. A minimum of five sets of 80 stimuli each were presented in each condition. To assess subjects' ability to rapidly make two detections and responses in a row while continuously maintaining a focus of attention, we analyzed the hit and false alarm data in the focus attention task at five intervals of elapsed time immediately following the onset of the last correctly detected target: 0.4–2.5 s, 2.5–4.5 s, 4.5–6.5 s, 6.5–10.5 s, and 10.5–30.0 s.

In the shift attention task (see Figure 2A), subjects were presented a minimum of 10 stimulus sequences identical to those used in the focus attention task. The shift task, however, was different: Subjects were required to alternate attention between visual and auditory stimuli as signaled by the appearance of the rare target stimuli (see Figure 2A). Correct detection of a target (hit) in the attended modality served to signal subjects to disengage their attention to stimuli in the current modality and to move and reengage their focus of attention as rapidly as possible to stimuli in the other modality in order to detect the very next target appearing in that modality (see Figure 2A). A failure to fully disengage and inhibit attention to stimuli in the same modality following a signal to do so would register as a false alarm (i.e., when a subject pressed the button to a target in the unattended modality after a signal to shift attention away from that modality; see Figure 2A). A failure to rapidly move and reengage attention in the opposite modality would register as a miss and would result in a reduced percentage of correct target detections in the time period immediately following a signal to shift attention (see Figure 2A). Therefore, to assess the time course of disengaging, moving, and reengaging attention, we analyzed hit and false alarm data in the shift attention task at the same five intervals of elapsed time immediately following the onset of each correctly detected target, as we did for the focus attention task.

**Post hoc focus attention experiment.** Analyses of these focus and shift experiments indicated significant group differences only in the shift experiment, not in the focus experiment (see Results section). Therefore we conducted a post hoc experiment that further challenged the capacity of the autistic patients and normal children to perform under focused attention conditions. We accomplished this by increasing the difficulty of the stimulus discrimination during the auditory
focus attention task. (With our software, fine adjustments were possible with auditory frequency, but not color.) The question was whether a significant increase in attentional and perceptual demands in the focus attention task would reveal group differences analogous to those seen in the shift task.

Whereas the easy focus attention task described previously prompted very nearly no false alarm responses to nontarget stimuli in the attended modality, in the difficult discrimination attention experiment described next, subjects produced some false alarm responses to such stimuli. Therefore, we calculated the percentage of hits using a correction described by Swets (1986a, 1986b)—namely, corrected percentage of hits (H') equals percentage of hits minus percentage of false alarms.

Five of the normal children and 7 of the autistic patients were available for this retesting. Using a tone separation of 30 Hz between target (1020 Hz) and nontarget (1000 Hz) sounds, we tested each normal child across five sets of 80 stimuli under the auditory focus attention condition. If a normal subject's H'c performance level exceeded 70%, the tone separation was reduced to 20 Hz, and if performance continued to be better than 70%, then the tone separation was reduced to 15 Hz. Data collected from the focus attention sets with the smallest tone separation that produced 70% or better H'c were used in statistical comparisons with autistic patients. This 70% performance criterion was met at the 30-Hz separation for 1 normal subject, at 20 Hz for 3 normal subjects, and at 15 Hz for 1 normal subject.

Autistic patients were tested in the same way. The 70% H'c performance criterion was met at the 30-Hz separation for 3 autistic patients and at the 15-Hz separation for 4 others. One of these autistic patients far exceeded the performance of all normal and other autistic patients by achieving 98% H'c at the 15-Hz level; the best any normal subject did at this separation was 73%.

In sum, thresholds established during the calibration phase were based on overall accuracy (H'c). For the test phase, the dependent variable of interest was the difference between groups in accuracy during the shortest time interval, given comparable overall performance. It is important to note that between the two groups overall accuracy and the range of pitch differences were comparable.

Event-related brain potentials. ERPs were recorded during the experiments for 3 of the autistic patients, 2 of the cerebellar patients, and the normal control subjects with the same method used in a previous study (Ciesielski, Courchesne, & Elmasian, 1990). The ERP effects from 1 cerebellar patient and 1 normal child were from Akshoomoff and Courchesne (1992).

Statistical Analysis

There were no significant modality by group effects for any of the comparisons presented next, so all performance data values presented were collapsed across modalities. Autistic patients were statistically compared (a) with adolescent normal subjects who matched them on gender, chronological age, and measures of nonverbal IQ and (b) with normal children who matched them on measures of verbal IQ (in areas of relative cognitive weakness in autism; see Tables 2 and 3). Akshoomoff and Courchesne (1992) previously reported that the cerebellar patients had significantly fewer correct target detections in the 0.4–2.5-s time zone in the shift attention experiment than did their chronological- and mental-age-matched normal subjects.

Results

Mental-Age-Matched Comparisons

Comparison with normal but much younger, mental-age-matched children provided a strong test of whether autistic patients have significant deficits in shifting attention, particularly because target detection performance among the normal children, autistic patients, and cerebellar patients was below ceiling on the shift attention task (see Figure 3).

Autistic and cerebellar patients performed similarly to the normal children in the focus attention task but were significantly impaired in their ability to accurately and rapidly respond to targets in the shift attention task; repeated measures analysis of variance using a Huynh-Feldt correction factor showed a significant Group × Experiment × Time interaction, $F(8, 84) = 2.29, p < .029$.

Focus Attention Task

Percentage of hits, percentage of false alarms, and reaction times (RT). In the focus attention task, accuracy (as indexed by percentage of hits and percentage of false alarms) and speed (as indexed by RT) in detecting targets as a function of elapsed time since the last correct target detection were not significantly different across normal children, autistic patients, and cerebellar patients: For percentage of hits, percentage of false alarms, and reaction times, the Group × Time comparison yielded $F(8, 84) \leq 0.99, p \geq .45$ (see Figure 4 and Table 4).

![Figure 3](image-url)  
*Figure 3.* Mean percentage of hits (top) and mean percentage of false alarms (bottom) for normal children, autistic patients, and cerebellar patients in the shift attention task. All data were analyzed at five intervals of time (in seconds) elapsed since the onset of every immediately preceding cue that had been correctly detected; data values were collapsed across visual and auditory modalities. With less than 2.5 s between visual and auditory cues in the shift attention task, autistic and cerebellar patients were significantly impaired at target detection relative to normal subjects.
which attentional and perceptual demands were increased values were collapsed across visual and auditory modalities. In the Figure 4.

Mean percentage of hits (top) and mean percentage of false over the first, easier focus attention task, the performances of targets in the same modality.

baseline focus tasks when autistic and cerebellar patients did not have immediately preceding target that had been correctly detected; data five intervals of time (in seconds) elapsed since the onset of every patients in the baseline focus attention task. All data were analyzed at normal subjects and autistic patients were not significantly different from each other: For H’c and reaction times, each Group x Time comparison yielded $F(3, 30) = 0.98, p > .41$. H’c was 75.8 ± 5% (with 3.4% false alarms) for normals and 77.4 ± 7% (with 4.6% false alarms) for autistic patients; RT was 673 ± 76 ms for normals and 632 ± 81 ms for autistic patients. In the shortest time interval (0.4–2.5 s), H’c was 64.3% for normals and 72.7% for autistic patients (see Figure 5); RT was 707 ms for normals and 628 ms for autistic patients.

**Shift Attention Task**

**Percentage of hits.** By contrast, in the shift attention task, accuracy (percentage of hits) in target detection as a function of elapsed time since the last correctly detected target was significantly different across groups: Group x Time, $F(8, 84) = 2.88, p < .007$. When 2.5 s or less had elapsed since the last correct target detection, autistic and cerebellar patients correctly detected only 58.9% and 59.2%, respectively, of the targets, compared with 78% correct detection by normal children (Bonferroni corrected pairwise t tests of autistic vs. normal children and of cerebellar patients vs. normal children were significant at $p < .05$; see Figures 3 and 6). When more time (2.5–30.0 s) had elapsed, however, autistic (86%) and cerebellar patients (90%) were much more accurate and did not perform significantly differently from the normal children (91%) or from their own performance levels in the focus attention task (Figure 4).

Figure 7 shows that for a single autistic subject who was retested on six separate occasions, this impairment in the ability to accurately and rapidly shift attention was persistent and profound despite repeated training and experience in these attention tasks. In comparison, his target detection accuracy in the focus task was excellent regardless of elapsed time since the last correct target detection (see Figure 8).

**Reaction times.** Reaction times for all subject groups as a function of time interval and attention task are presented in Table 4. In the shift attention task, all subject groups had faster reaction times overall than did the age-matched control subjects, $F(1, 21) = 4.74, p < .05$. In the focus

**Post hoc focus attention experiment.** In this experiment in which attentional and perceptual demands were increased over the first, easier focus attention task, the performances of normal subjects and autistic patients were not significantly

### Table 4

**Mean Reaction Times to Targets in the Shift and Focus Attention Tasks**

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Time since last cue (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.4–2.5 (M ± SD)</td>
</tr>
<tr>
<td><strong>Shift task</strong></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>494 ± 37</td>
</tr>
<tr>
<td>Adolescent</td>
<td>598 ± 116</td>
</tr>
<tr>
<td>Child</td>
<td>622 ± 81</td>
</tr>
<tr>
<td>Autistic</td>
<td>740 ± 190</td>
</tr>
<tr>
<td>Cerebellar lesions</td>
<td>438 ± 41</td>
</tr>
<tr>
<td><strong>Focus task</strong></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>560 ± 104</td>
</tr>
<tr>
<td>Adolescent</td>
<td>514 ± 103</td>
</tr>
<tr>
<td>Child</td>
<td>632 ± 143</td>
</tr>
<tr>
<td>Autistic</td>
<td></td>
</tr>
<tr>
<td>Cerebellar lesions</td>
<td></td>
</tr>
</tbody>
</table>
Figure 5. Results from the post hoc experiment, a focus attention task with more difficult discrimination. Mean percentage of hits (bottom left), mean percentage of false alarms (bottom right), and \( H'c \) (corrected percentage of hits; top) are shown for normal children and autistic patients as a function of time between correct target detections.

attention task there was a tendency for reaction times in all groups to be longer with shorter intervals between targets, \( F(4, 84) = 2.07, p < .092 \), but there was no significant difference in reaction times between cerebellar lesion patients and age-matched control children, \( F(1, 21) = 1.82, p < .20 \). Autistic patients had shorter reaction times than either the cerebellar lesion patients or the normal controls, but because the autistic patients were also chronologically older, these statistical comparisons were not performed. To further examine these findings, we performed a post hoc analysis on reaction time data from the shortest time interval (0.4–2.5 s) and the next longest one (2.5–4.5 s) in the focus and shift tasks. The results are graphed in Figure 9. Compared with normal control subjects, autistic and cerebellar lesion patients showed a significantly greater difference in reaction times at the two time intervals in the two tasks, \( F(1, 21) = 4.71, p = .042 \). In the focus attention task, all three groups were only slightly slower (by 2% to 13%) in correctly responding to targets in the shortest time interval relative to the longer one. However, in the shift attention task, autistic and cerebellar patients were substantially slower (by 30% and 31%) when only 2.5 s or less had elapsed compared with when there was more time to shift attention.

Percentage of false alarms. For each group, the false alarm rate was higher in the shift task than in the focus task, \( F(1, 21) = 19.96, p < .0003 \). In the shift task, the percentages of false alarms for autistic, cerebellar, and normal children were 15.6%, 4.3%, and 8.7%, respectively; and in the focus task, they were 1.5%, 0.5%, and 0.4%, respectively. There were no significant effects of elapsed time since the last correctly detected target, \( F(4, 84) = 1.14, p > .34 \), and no interaction of subject group, time, and experimental task, \( F(8, 84) = 0.91, p > .50 \).
ATTENTION IN AUTISTIC AND CEREBELLAR PATIENTS

Figure 7. Shift attention task data for a single autistic patient who was tested on 6 separate days (dates of testing are given in inset). There was a persistent and profound impairment in his ability to accurately and rapidly shift attention despite repeated training and experience. Data values were collapsed across visual and auditory modalities. The numbers in parentheses indicate the number of sessions in which this patient achieved a 100% hit rate in a specified time interval. Slower in shift tasks by 26%; the autistic subgroup without a normal posterior corpus callosum was slower by 34%.

In addition, there was no systematic difference in false alarm rates for these patients as a function of parietal abnormality. Among the 4 autistic patients with MR evidence of parietal abnormality, 2 had elevated levels of false alarms (25% and

Parietal Abnormal Versus Normal Comparisons

With short intervals between targets, the 4 autistic patients with MR evidence of parietal abnormality, indexed by reduced posterior corpus callosum, performed the shift attention task with slightly, but nonsignificantly, better speed and accuracy than the 4 autistic patients without MR evidence of parietal abnormality: t(6) = 0.58, p > .59 and t(6) = 0.64, p > .54. When only 2.5 s or less had elapsed since the last correct target detection, the former subgroup correctly detected only 62.1% (RT = 604 ms) of the targets compared with 55.9% (RT = 639 ms) correct detection by the latter subgroup. The autistic subgroup with a reduced posterior corpus callosum also showed slightly less difference in response time at short intervals in shift tasks compared with focus tasks, responding

Figure 8. Focus attention task data for a single autistic patient who was tested on 6 separate days (dates of testing are given in inset). In the focus attention task, in contrast to the shift attention task, his target detection accuracy was excellent regardless of time elapsed since the last correct target detection. Data values were collapsed across visual and auditory modalities. The numbers in parentheses indicate the number of sessions in which this patient achieved a 100% hit rate in a specified time interval.
In the shift attention task, autistic and cerebellar patients were substantially slower to detect targets when only 2.5 s or less had elapsed compared with when there was more time to shift attention. The index of the speed of shifting and detecting targets is shown as the percentage difference score, the poorer the ability to rapidly shift and detect targets; on this score, autistic and cerebellar patients were as good as normal controls. The single exception was that autistic patients had significantly more false alarms in the shift task than their chronologic-age-matched normal controls: 15.6% vs. 1.8%, F(1, 14) = 6.57, p < .023.

Chronologic-Age-Matched Comparisons

With one exception, all significant and nonsignificant findings from comparisons between autistic patients and normal children were also obtained from comparisons between autistic patients and their chronologic-age-matched normal controls. The single exception was that autistic patients had significantly more false alarms in the shift task than their chronologic-age-matched controls: 15.6% vs. 1.8%, F(1, 14) = 6.57, p < .023.

P3b Responses

To verify that autistic and cerebellar patients had not mentally shifted their attention when they missed targets, we recorded the P3b component of the ERP for several subjects. Like control subjects, the autistic and cerebellar patients exhibited a P3b response to correctly detected targets but not to missed stimuli that occurred 2.5 s or less following a cue (see Figure 10; also see Figure 5 of Akshoomoff & Courchesne, 1992).

Discussion

Our results indicate that like patients with acquired cerebellar damage, patients with autism are severely impaired in their ability to coordinate accurate and rapid voluntary shifts of attention between sensory modalities. Within 2.5 s or less following a cue to attention to a different sensory modality, both autistic and cerebellar patients frequently failed to detect target information in the new focus (see Figures 3, 6, and 7), and had slower reaction times to those that they did correctly detect (see Figure 9). This shift deficit was persistent and profound despite repeated training and experience in these tasks in 1 autistic patient who was able to retested on six separate occasions (see Figures 7 and 8). In the Akshoomoff and Courchesne (1992) study, 3 patients with cerebral cortical lesions did not show this extreme impairment when performing the identical shift task. When given more time (more than 2.5 s), autistic and cerebellar patients were able to shift attention and detect targets nearly as accurately as normal controls. Therefore, in these two patient groups, it appears that neural pathology did not prevent the execution of shifts of attention, but rather seems to have created suboptimal performance of such shifts.

The inability to accurately and rapidly shift attention in the autistic and cerebellar patients was not due to motor control problems. First, as noted above, these patients were not significantly impaired in responding to targets that occurred 2.5 s or less after a correctly detected target as long as there was not a requirement to shift attention.

Second, all of the reaction times for the autistic and cerebellar patients were within the 200–1,400-ms time window allotted for responses to count, which eliminates the possibility that responses were so slow they were not counted. Also, relative to this broad time window allowed for responding to targets, there were relatively small absolute differences in RTs between the autistic patients and their chronologic- and mental-age-matched normal controls as well as between the cerebellar patients and their normal controls (see Table 4).

Third, the presence of the P3b can be a sign of covert attention, namely the recognition of rare target information independent of overt motor action (Courchesne, Hillyard, & Courchesne, 1977), and it is absent when a target stimulus is ignored or missed (Ciesielski, Courchesne, & Elmasian, 1990; Squires, Hillyard, & Lindsay, 1973). In the present study and in a study by Akshoomoff and Courchesne (1992), autistic and cerebellar patients, like normal controls, exhibited a P3b response to correctly detected targets but not to missed stimuli that occurred 2.5 s or less following a cue. These findings
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Figure 10. Event-related potentials elicited during the shift attention task by correctly detected (A), correctly ignored (B), and missed visual rare (C) stimuli are shown for the autistic patient (solid line) whose data appear in Figures 7 and 8 and for the average of age-matched normal controls (dotted line). Potentials were recorded from the Pz scalp electrode located above parietal regions. Visual stimuli were red and green (g) flashes; auditory stimuli were 2-kHz (hi) and 1-kHz (lo) tone pips. HIT = correctly detected target; MISS = a failure to respond to a target; FA = an erroneous response to a rare stimulus that was in a modality to be ignored; IGN = a rare stimulus that was correctly ignored. In the example of the shift experiment, the subject pressed a button (arrow) to the first rare visual target stimulus (RED). This served as a cue to shift attention to the auditory stimuli, ignore (IGN) the rare visual stimuli, and respond to the next auditory target (HI). The auditory target in turn served as a cue to shift attention back to the visual stimuli.

suggest that when these patients missed targets that rapidly followed a cue, they were not covertly attending and thus had not fully shifted their attention to the new focus. As with the preceding observations, this one also reflects a mental rather than a motor output error.

Possible Neural Bases for Shift Deficits in Autism

Contemporary models for the neural basis of shifting the mental focus of selective attention do not include the cerebellum but do include numerous other structures such as parietal cortex, superior colliculus, pulvinar, and frontal cortex (e.g., Posner & Petersen, 1990).

In autism, parietal lobe abnormalities have been reported to occur in 43% of cases (Courchesne, Press, & Yeung-Courchesne, 1993). In nonautistic patients, parietal damage has been suggested to result in deficits in disengaging visuospatial attention (Posner et al., 1984; Posner & Petersen, 1990). In the present study there were two subgroups of autistic patients: those with significant reductions in the posterior segment of the corpus callosum and those without. The former subgroup performed slightly and nonsignificantly better (faster and more accurately) than the latter subgroup. The present study, therefore, does not provide evidence pointing to a clear parietal (or posterior cortex) contribution to the shift attention deficits seen in the autistic patients.

The most likely explanation for an absence of parietal effects on shift performance in the present study is that parietal damage may primarily affect visuospatial attention operations, which are not invoked by the present between-modality shift attention task. Another possibility is that parietal circuitry involved in shifting attention operations may not hinge on interhemisphere communication via the posterior segment of the corpus callosum. Still another possibility is that our corpus callosum index of posterior cortical abnormality may not have been sensitive enough to characterize the full extent of posterior cortical anatomic abnormality in our subjects.

In autism, neuroanatomic abnormalities have not been found in any other structure previously implicated in models of shifting attention. Cerebellar pathology has, however, been found in 16 quantitative MR and autopsy reports from nine independent research groups involving 240 autistic cases (see Table 1 and Figure 1) and therefore appears to be a consistent feature of autism as earlier proposed (Courchesne et al., 1988). Because cerebellar abnormalities are common to autistic and cerebellar patient groups, the present results lead to the suggestion that the cerebellar pathology in autistic patients
may contribute to their impairment in shifting attention. This may happen via cerebellar connections with brainstem, thalamic, and cortical systems that have been implicated in various attentional operations, including moving and engaging attention (Rafal & Posner, 1987; Rafal et al., 1988; Posner & Petersen, 1990).

**Role of the Cerebellum**

Since the time of Galen, the cerebellum has traditionally been linked by medical science to motor control and motor learning (Ghez, 1991; Gilman, Bloedel, & Lechtenberg, 1981; Glickstein & Yeo, 1990). However, by the turn of the last century, the question of cerebellar involvement in mental functioning had been raised (Beyerman, 1917). Then, in 1949, a short comment marked an important new branch in understanding about the role of the cerebellum. While reporting the discovery of the reticular activating system (RAS), Moruzzi and Magoun (1949) stated, “Exploration of the overlying cerebellum has revealed excitable points in its fastigial nuclei, the responses possibly being mediated by connections of the (cerebellar) roof nuclei with the brain stem reticular formation” (p. 458).

Shortly thereafter, “sensory attention” deficits were reported in cats with lesions to vermian lobules VI–VII (Chambers & Sprague, 1955)—the same lobules first reported by MR techniques to be abnormal in autism (Courchesne et al., 1988). Furthermore, data from Steriade and Stoupel (1960) suggested that the cerebellum may control levels of excitability in the RAS. Much evidence has now accumulated that the cerebellum has physiopharmacological connections with many systems thought to be associated with attention, arousal, and sensory responsiveness (Itoh & Mizuno, 1979; Kitano et al., 1976; Moruzzi & Magoun, 1949; Nieuwenhuys et al., 1988; Sasaki et al., 1972, 1979; Schmahmann & Pandya, 1989; Snider, 1950; Steriade & Stoupel, 1960; Vilensky & Van Hoesen, 1981).

More recently, Crispino and Bullock (1984) found that stimulation of vermian lobules VI–VII can modulate brainstem, thalamic, and cortical neural responses to auditory and visual sensory input in nonbehaving rats. For example, the neural response of the rat superior colliculus to auditory and visual stimuli is enhanced when the stimulus is preceded at an optimum interval by stimulation of vermian lobules VI–VII (Crispino & Bullock, 1984); similar effects have been reported for neural responsiveness in the hippocampus following stimulation of vermian lobules VI–VII in cats (Newman & Reza, 1979). Moreover, when background luminance is sufficient to obscure the rat colliculus response to a brief flash, the colliculus response to the brief flash will emerge above background noise levels if, once again, it is preceded at optimum intervals by stimulation of vermian lobules VI–VII (Crispino & Bullock, 1984).

It seems possible, therefore, that within the domain of sensory processing, the cerebellum anticipates upcoming events and the context in which they will occur and then performs operations that optimize the neural signal-to-noise conditions in whichever systems (e.g., brainstem, thalamic, cerebral, and hippocampal) will be involved in processing such events. Selective attention may involve an analogous anticipatory enhancement of signal (the desired, to-be-attended information) relative to noise (the information to be ignored). If so, perhaps vermian lobules VI–VII as well as other cerebellar regions perform analogous anticipatory or preparatory optimizing operations on neural activity in structures known to mediate attention and arousal.

Such a function would be especially important during conditions that require rapid, unpredictable, and frequent shifts in the focus of selective attention. In our paradigm, the cues to shift attention signaled the need to rapidly prepare to detect and respond to a different target-event-background context. Under such demanding conditions, our cerebellar and autistic patients made many more errors than normal. However, an important additional observation was that given more time, their performance approached normal levels. Perhaps, then, cerebellar pathology does not eliminate attentional operations but instead results in an inability to rapidly implement a new optimal pattern of selective neural responsiveness. (It is possible that suboptimal attentional functioning may very well be sufficient to satisfactorily accomplish some tasks and thus could go unnoticed.)

A parsimonious suggestion is that the cerebellum provides an analogous function for all of the systems with which it is interconnected, including the motor, attention, arousal, sensory, memory, limbic, hypothalamic, serotonergic, dopaminergic, and noradrenergic systems (for reviews, see Courchesne, 1989b, 1991). That is, without the aid of the cerebellum, each system continues to perform its prescribed specific function, but suboptimally. In his treatise on signs of human cerebellar dysfunction, Gordon Holmes (1939) quoted a cerebellar patient as saying, “The movements of my left [unaffected] arm are done subconsciously, but I have to think out each movement of the right [affected] arm. I come to a dead stop in turning and have to think before I start again” (p. 22). So, cerebellar pathology does not eliminate voluntary motor action but instead makes motor action slow, inaccurate, and effortful; the patient may often have to consciously “think” through each step in preparation for action and during the execution of each action. In a parallel fashion, the evidence cited above suggests that cerebellar pathology does not eliminate voluntary shifts of attention but instead makes such shifts slow and inaccurate. Similarly, cerebellar pathology apparently does not prevent relatively good perceptual judgments of time intervals between any two stimuli but instead makes such judgments more variable. For instance, whereas normal subjects and cerebellar-lesioned patients differ from each other by as little as 1% in correctly tapping out a 550-ms time interval between two tones, cerebellar patients show a significant increase relative to normals in the standard deviations associated with such performance or perceptual judgments (e.g., standard deviations for perceptual judgments between two tones were ± 45.7 ms for cerebellar patients vs. ± 26.1 ms for normals; Ivy & Keele, 1989). Likewise, cerebellar pathology apparently does not eliminate the classically conditioned nictitating membrane response or its engram but instead produces variability in response onset latency and amplitude (e.g., see Figure 16.13 from Welsh & Harvey, 1992). So, it appears that in motor, attentional, perceptual, and associa-
tion learning domains, cerebellar damage does not eliminate function but does increase suboptimal variability in response thresholds, times, amplitudes, and effort.

As reviewed by Welsh and Harvey (1992), rooted in the work of Rolando (1809–1823) and Flourens (1824–1842) is the concept that the cerebellum ensures “the optimal performance of the voluntary motor act” (p. 332). It may do so by adjusting the “excitability thresholds of motor nuclei” (p. 332) so that they respond in a manner proportional to motivational significance, associative strength, and intensity of eliciting stimuli (Welsh & Harvey, 1992). A new variant on this theme is the proposal that by monitoring sensory input, the cerebellum guides movement so as to optimize the quality of sensory information obtained during exploration (Bower & Kassel, 1990).

We suggest that these concepts be extended and propose that the cerebellum optimally shifts excitability thresholds in neurons likely to be used in any sensorimotor or mental action. That is, the cerebellum adjusts responsiveness in whatever neural array or network is anticipated to be needed to attain a prescribed goal (the goal perhaps being prescribed by cerebral cortical or other subcortical systems). Optimal preparation of neural networks needed to achieve such goals may require the cerebellum to implement a succession of precisely timed and selected changes in the pattern or level of neural activity in diverse networks. The patterns chosen and the time implemented hinge on the goals, current context, and anticipated intervening events. As the actual sequence of events occurs, the cerebellum updates and adjusts patterns of responsiveness. Thus, the cerebellum is continuously providing the optimal neural conditions necessary for handling anticipated events. The cerebellum is no more an “attentional” structure or a “timing module” than it is a motor one. Rather, it is a master computational system for providing the optimal context for the smooth interdigitated, coordinated neural action of whatever systems are needed from moment to moment to achieve a specified goal within the context of continuously fluctuating internal and external contexts.

**Possible Role of Cerebellar Dysfunction in Social and Cognitive Development**

In normal development, social knowledge and many higher cognitive, affective, and communicative functions spring from early infant–mother interactions (Bakeman & Adamson, 1984; Bruner, 1975; Trevarthen & Hubley, 1978; Tronick, 1982; Werner & Kaplan, 1963). Tronick (1982) wrote that “successful regulation of joint interchanges . . . results in normal [cognitive, affective, and social] development” (p. 1), and “The crucial element is that the infant and mother . . . share the same directional tendencies or focus of attention during the interaction” (italics added; p. 4). Because during any normal joint interchange the locus of information (objects, actions, sounds, expressions, internal experience, etc.) frequently, rapidly, and often unpredictably changes, shifting attention is of fundamental importance. By 12 to 15 months of age, infants normally achieve the skill of coordinating their attention between a social partner and objects of mutual interest, a major developmental milestone in social communication (Bakeman & Adamson, 1984; Bruner, 1975; Trevarthen & Hubley, 1978; Tronick, 1982; Werner & Kaplan, 1963). To achieve this major milestone, an infant needs to do more than simply focus his or her attention on a single, captivating aspect of an object or person. He or she must follow the rapid and unpredictable ebb and flow of human social activity, such as words, gestures, touching, postures, facial expressions, and actions on objects. These activities provide signals that direct the infant to shift his or her attention in order to follow the varying sources of social, emotional, and situational information. By being able to smoothly, selectively, and rapidly shift his or her attention with these signals, the infant is able to combine, as a single entity in memory, the various and separate elements of a social situation.

Tronick (1982) postulated that “failure to succeed in the regulatory process . . . derails [the] infant . . . from the normal developmental track” (p. 1). Presumably, brain damage that causes early and extreme developmental failure of this regulatory system could produce severe developmental abnormality.

Infantile autism is a prime example of just such a failure of normal infant–mother joint attentional interactions. On the basis of the evidence that autism involves prenatal or early postnatal cerebellar damage and that this cerebellar damage might impair the capacity to control the direction of attention, it is reasonable to suggest that these deficits—present throughout infancy—could contribute to the deviation in social, affective, and cognitive development that characterizes patients with autism. In the autistic infant, such attentional dysfunction may interfere with the ability to continuously follow the rapidly changing events that compose reciprocal social interactions. Much would be missed, and the fragments caught would lack “context or temporal continuity” (Courchesne, 1987, p. 314). Thus, the autistic infant’s “knowledge of the [social] world would be made up of disconnected fragments of [gestural, facial, vocal, and emotional] information” (Courchesne, 1987, p. 314). In addition to failing to normally apprehend information, the cerebellar-damaged autistic infant might also be severely impaired in the ability to express in a timely fashion his or her own affective and cognitive reactions to the information he or she does experience.1 According to Tronick’s (1982) model, such difficulties should seriously hinder the child’s ability to engage in joint social interchanges, which in turn should lead to deficiencies in social knowledge and communication, including imitation, turn-taking, symbolic play, and the ability to exchange experiences and emotions with others about topics of mutual interest. Each of these deficiencies is, in fact, highly characteristic of infantile autism (Curcio, 1978; Dawson & Lewy, 1989; Kanner, 1943; Landry & Loveland, 1988; Loveland & Landry, 1986; Mundy, Sigman, & Kasari, 1990; Mundy, Sigman, Ungerer, & Sherman, 1986; Sigman, Ungerer, Mundy, & Sherman, 1987; Wetherby & Prutting, 1984).

Thus, it is possible to construct a hypothetical outline of one

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1 Brain damage sustained after normal development of affective and communicative reactions and knowledge would not necessarily lead to their regression or loss, for example, just as hippocampal damage sustained after normal language development does not lead to loss of already acquired language knowledge.
path of maldevelopment in autism from Purkinje neuron loss, to deficits in the timely and accurate control of the direction of attention and the expression of intention, to deficits in infant–mother joint social and affective interactions, to deficits in more complex verbal and nonverbal communication abilities (Courchesne, 1985, 1987, 1989a, 1989b; Courchesne et al., 1988; Courchesne, Townsend, et al., 1994; Courchesne, Chisum, & Townsend, in press).

This suggestion does not preclude the possibility that additional neurocognitive abnormalities contribute as well to these social, affective, and cognitive deficits in autism. For example, as mentioned earlier, a new site of neuroanatomical abnormality has been tentatively identified in a subset (43%) of autistic patients—the parietal lobes (Courchesne, Press, & Yeung-Courchesne, et al., 1993). This neural abnormality has now been linked to a striking ability to concentrate attention in one, narrow spatial location at the expense of awareness of sensory events at other locations (Townsend & Courchesne, 1994).

Such an exaggerated narrowing of attention, we speculate, may result in a form of functional sensory neglect of events outside such a narrowed focus of conscious attention. Along with the cerebellar findings, this parietal–behavioral finding may also help explain why some autistic patients seem so often to miss social signals. Uta Frith (1989) wrote, "Minimal stimuli, such as a slightly raised eyebrow . . . may have profound communicative effects" (p. 141). Such important social cues may be entirely missed by autistic patients with parietal abnormalities. As another example, some have proposed a role for the hippocampus and the amygdala in the difficulty autistic patients have in associating social stimuli with emotional states. However, recent studies indicate that such social–emotional associations can be established without the hippocampus and the amygdala (Tranel & Damasio, 1993). Moreover, in a recent study Maurer (1988) reported that a patient with bilateral dysgenesis of the hippocampal formation and the amygdala did not have autistic social, language, cognitive, emotional, or behavioral features.

In addition to helping explain the principal social communication deficits in autism, the impairment in the control of shifts of attention may also be at the root of a wide variety of commonly observed cognitive abnormalities in autism, including stimulus overselectivity, uneven memory, insistence on sameness, perseveration, repetitive and ritualistic behaviors, narrowed interests, formation of peculiar associations, and poor performance on executive function tasks requiring shifting of mental sets (Kanner, 1943; Lovaas, Koegel, & Schreibman, 1979; Lovaas, Schreibman, Koegel, & Rehm, 1971; Schreibman & Lovaas, 1973). For example, stimulus overselectivity or "overselective attention" (long suggested to contribute to social deficits in autism) is the tendency of the autistic child to respond to only one stimulus element of a complex array of stimuli composing an object, place, or episode (Lovaas et al., 1971, 1979; Schreibman & Lovaas, 1973). The full exploration and apprehension of such complex stimulus arrays requires the ability to voluntarily shift attention from element to element smoothly, precisely, and effortlessly. The present data suggest that autistic patients lack this normal ability. The failure to attentively and fully explore in a timely fashion nonsocial as well as social stimulus arrays would result in incomplete memory for events, more so for transient and variable events (e.g., social, language, or acoustic) and less so for invariant or predictably repeatable events (e.g., calendars or flushing toilets). What would be retained would not necessarily be stimulus elements that had causal relationships with each other or spatial or temporal contiguity. For the autistic patient, the normal coherence of elements is lost, as would the predictive correlations between elements. Instead, disparate fragments would often be registered. They would compose a rather impoverished picture of the actual events and the context in which they occurred; imprecise, misleading, and possibly bizarre associations and predictions about relationships between successive fragments could ensue. This condition, we suggest, could provoke disjointed, disorganized, and unexpected behavioral and emotional reactions by the autistic person during interactions with the nonsocial environment and with social partners. Furthermore, this condition could promote a preference for repeatable, predictable, or invariant objects, events, and activities over novelty, exploration, and social partners and social settings. That is, the preference for the former situations would be expressed as an insistence on sameness, repetitive behavior, and narrowed interests, and the latter situations would be avoided as confusing and distressing.

Finally, neurobiological data show that early damage in one system can alter neural organization in other systems with which it is interconnected (Courchesne, Chisum, & Townsend, in press). One wonders, therefore, whether the early cerebellar damage in autism might not contribute to abnormal organization and functioning within the attention, arousal, memory, limbic, sensory, hypothalamic, serotonergic, dopaminergic, noradrenergic, and motor systems with which it is interconnected (Bava, Manzoni, & Urbano, 1966; Chambers & Sprague, 1955; Crispino & Bullock, 1984; Haines & Dietrichs, 1987; Ito, 1984; Itoh & Mizuno, 1979; Kitano et al., 1976; Moruzzi & Magoun, 1949; Newman & Reza, 1979; Nieuwenhuys et al., 1988; Saint-Cyr & Woodward, 1980a, 1980b; Sasaki et al., 1972, 1979; Schmahmann & Pandya, 1989; Snider, 1950, 1967; Steriade & Stoupel, 1960; Vilensky & Van Hoesen, 1981; Watson, 1978). In fact, many parallels exist between these systems and systems often posited to be abnormal in autism (Courchesne, 1987, 1991; Courchesne et al., 1988).

**Concluding Comments**

For nearly a century, it has been recognized that when attention is deficient, cognition suffers: William James (1890) stated that "an object once attended will remain in memory, whilst one inattentively allowed to pass will leave no traces behind" (p. 427). Autism is a disorder in which attention is markedly deficient. In contrast to the agility with which a normal child can control shifts of attention, the very first published description of an autistic child stated that the child displayed "an abstraction of mind which made him perfectly oblivious to everything about him . . . and [that] to get his attention almost requires one to break down a mental barrier between his inner consciousness and the outside world" (Kanner, 1943, p. 218). Surprisingly, this mental barrier apparently involves damage to cerebellar structures that may
play a role in the coordination of voluntary shifts of attention. This and a host of animal and human behavioral and physio-anatomical data (see previous paragraph) strongly support the concepts first introduced by Snider (1950, 1967) that the cerebellum is involved in a broad array of nonmotor functions, including higher mental functions, and that damage to it can contribute to psychiatric disorder.

References


ATTENTION IN AUTISTIC AND CEREBELLAR PATIENTS


