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Motion Perception Deficits from Midline Cerebellar Lesions in Human

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Although visual motion processing is commonly thought to be mediated solely by visual cortical areas, this human lesion study suggests that the cerebellum also has a role. We found motion direction discrimination deficits in a group of patients with acute midline cerebellar lesions. Unlike normals and patients with hemispheric cerebellar lesions, these patients with midline lesions were unable to discern a global motion vector in a local stochastic motion display. This resembles the perceptual defect reported following cortical area MT lesions in primates. This motion perception deficit may result from damage to a cerebellar mechanism involved in perceptual stabilization. Disruption of this comparator mechanism is sufficient to produce a severe motion perception deficit even though cortical visual processing mechanisms are still intact.

Motion perception Cerebellum Brain lesions Cortical circuits Subcortical circuits

INTRODUCTION

The cerebellum's role in mediating fine motor control and visuomotor coordination is well known (Ito, 1984; Leigh & Zee, 1991). Cerebellar lesions are commonly associated with defective reaching under visual guidance, and with defective ocular-motor control, including the inability to track moving targets with smooth pursuit eye movements (Bogousslavsky & Meienberg, 1987). A disorder of temporal coordination appears central to these deficits (Ivry & Keele, 1989). However, cerebellar lesions also impair motor planning, visual perception and even higher cognitive function (Botez, Gravel, Atting & Vézina, 1985; Schmahmann, 1991; Akshoomoff, Courchesne, Press & Iragui, 1992). Among these higher, non-motor functions, normal visual motion perception appears to be impaired by some types of cerebellar lesions.

Recently, Ivry and Diener (1991) found that patients with cerebellar disease, most with diffuse cerebellar atrophy, showed increased performance variability in a phi movement, velocity comparison task. Patient performance was more variable than that of normal observers when judging the relative speed of a small row of dots flashed horizontally across a computer screen on successive presentations. The authors postulated that this deficit is related to the cerebellum's role in timing functions (Ivry & Keele, 1989). Their hypothesis was that the perception of phi motion relies on a neural calculation of change in retinal position over a time interval generated by the cerebellum. A compromised timing mechanism accounts for the more variable velocity discriminations made by patients with cerebellar lesions.

However, motion perception does not require this high-level internal timing mechanism. Psychologists since Exner (1875) have known that observers perceive motion even though they are unable to resolve, either spatially or temporally, changes in the stimulus elements. This means that motion perception is not the product of the perception of space and the perception of time. Rather, motion perception is a true "sensation" or a "fundamental visual dimension" (Graham, 1965; Nakayama, 1985). High-level computations of time intervals between stimulus events are not required. Instead, the favored explanation for motion perception is a low-level spatio-temporal autocorrelation mechanism (Reichardt, 1961; Watson & Ahumada, 1985; Van Santen & Sperling, 1984). Cavanaugh and Mather (1989) even propose that this type of mechanism underlies the perception of all motion phenomena, including phi movement (Wertheimer, 1912), cognitive apparent motion (Anstis, 1980), and high-level, long-range apparent motion (Braddick, 1980). Therefore, while Ivry and Diener's (1991) high-level timing-theory explanation may account for their results, an alternative low-level explanation remains a strong possibility. Perhaps, the cerebellum has a more direct role in the perception of visual movement.

Indeed, it is important to note that the properties of some cerebellar neurons makes them well suited for this

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possible role in visual motion perception. In monkey single-cell recordings, visual response properties of some cerebellar neurons, especially in posterior vermis, resemble those of neurons in monkey extrastriate visual cortex, in particular those in MT and MST that show direction, velocity, and disparity selectivity (Maunsell & Van Essen, 1983a, b; Albright, 1984; Motter, Steinmetz, Duffy & Mountcastle, 1987; Roy & Wurtz, 1990). Like these cortical motion processing neurons, posterior vermis neurons have large receptive fields, respond to moving visual targets, show direction and velocity selectivity (Buchtel, Rubia & Strata, 1973; Suzuki, Noda & Kase, 1981; Suzuki & Keller, 1988), and even show binocular disparity selectivity (Donaldson & Hawthorne, 1979). Strong similarities are also found between MT/MST and the dorsolateral pontine nucleus (DLPN) (Suzuki, May, Keller & Yee, 1990), which is a major source of projections to the cerebellum. Like MT and DLPN neurons, posterior vermis neurons seem ideally suited to monitor visual movement. However, these cerebellar neurons are usually ascribed the duty of monitoring retinal slip velocities related to eye movements with no role in actual motion perception.

To investigate the cerebellar role in motion perception further, we tested whether patients with acute cerebellar lesions would show poor discrimination for direction of movement in random-dot motion stimuli. Similar random-dot stimuli were used by Newsome and Paré (1988) to show motion perception deficits in monkeys following acute area MT lesions. Our current understanding of motion processing (Sekuler, Anstis, Braddick, Brandt, Movshon & Orban, 1990) suggests that random-dot stimuli are better suited to address the cerebellar role in motion perception than are the long-ISI apparent motion displays used by Ivry and Diener (1991). Random-dot cinematograms are ideal for isolating neural mechanisms selective to visual motion, excluding the contribution of non-motion visual mechanisms that might infer movement from change in position over time [e.g. those that might infer the movement of hands on a clock (see Anstis, 1980)]. Such a mechanism cannot be excluded, and is even implied, in the Ivry and Diener study.

METHODS

Stimuli

Motion perception was tested with computer generated random-dot cinematograms (RDC). Cinematograms comprised 150 randomly placed, small (2×2 min arc) black dots that moved within a 4 deg square region. Dot movements were generating by applying small positional displacements to each dot's position on subsequent cinematogram frames. The direction of each dot displacement was randomly selected from either a "signal" or "noise" distribution. The signal distribution contained displacements in a single given direction, either up, down, left or right. The noise distribution contained a uniform distribution of displacements in all directions (see Fig. 1).

The ratio of dots drawing movements from the signal or noise distributions was varied in a method of constant stimuli. Varying the percent signal, the main independent variable, gave different levels of difficulty to the task. The movement of a single dot could not be used to generate a correct response because dots could vary between a signal and noise assignment on every RDC frame. Both signal and noise displacements were of the same magnitude, giving all the dots in the display the same constant speed: either 11 or 3.3 deg/sec. The two speeds were produced by displacing each dot, both signal and noise, by 10 min arc every 15 msec or by 6 min arc every 30 msec respectively. This method of generating signal and noise movements produces a display wherein all dots appear to move the same speed. In contrast, some motion displays generate noise by giving "noise dots" a new, randomly determined position between RDC frames. These random position noise displays appear to have dots moving at many different speeds. For this reason the "noise" portion of this stimulus is less effective in masking the direction of signal movement due to transparency (Bravo & Watamaniuk, 1992). Finally, these displays have a troublesome definition of "signal-to-noise ratio" due to the ostensibly random correspondences that would be made between both signal and noise dots on consecutive RDC frames. The current display avoids these problems by having a fixed dot displacement, a low dot density (0.04%) and a low probability of a correspondence mismatch on each frame [0.56% with the 11 deg/sec stimulus (Williams & Sekuler, 1984)].

As mentioned earlier, the percent signal varied within a method of constant stimuli. Two ranges of signal were used: First, in a "warm-up and learning phase", stimuli ranging from 20% to 80% signal were used to determine

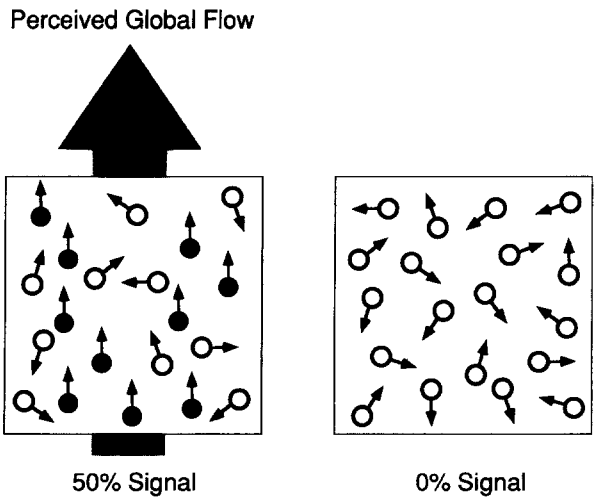

FIGURE 1. Shown is a schematic of the psychophysical stimuli used to test motion perception. Signal dots are depicted as solid, noise dots are depicted as open. On the left is a schematic of 50% signal. This stimulus should normally produce a percept of global upward flow. On the right is a schematic of 0% signal. This stimulus should not produce a consistent percept of coherent motion in any particular direction.

that each observer could perform the task. Next, the experimental phase consisted of stimuli ranging from 5% to 35% signal. This fixed range, chosen from pilot studies in normals and brain damaged patients, spans normal motion threshold values at the low end and statistically abnormal threshold values at the high end. Every observer completed 80 trials in the warm-up phase and 240 trials in the experimental phase for each of the two speeds tested.

Observers were also presented with a simple static control version of this test in central fixation to both familiarize them with the apparatus and ensure that patients could perform a simple direction discrimination with brief presentations. The static stimulus comprised 15 small (24 min arc trunk, 10 min arc arms), randomly positioned arrows, all pointing in one same cardinal direction. The task was to indicate the direction that the arrows were pointing.

Stimuli were generated on an Apple Macintosh computer and presented in black and white on an Apple monochrome monitor. Observers viewed the display from 57 cm while seated in a dimly lit testing room. Observers fixated a small cross at the screen center and initiated trials at their own pace. To preclude shifts in fixation, on each trial the stimulus presentation lasted just 200 msec. Observers were asked to fixate the cross for the duration of the trial. Cinematograms were presented unpredictably either in the center of the screen, or 5 deg eccentric in one of the four quadrants. Equal numbers of trials were presented in each of the five locations. Responses were verbal or gestural and were recorded on computer by an experimenter.

Observers

Sixteen patients (ranging from 42 to 75 yr, mean age 62 yr) with acute cerebellar lesions, verified by magnetic resonance imaging ($n = 15$) or by computed axial tomography ($n = 1$), were tested with this motion direction discrimination paradigm. Patients with degenerative cerebellar syndromes were excluded. The cerebellar degenerations comprise a variegated group of syndromes with differing pathology and clinical signs as well as pathological lesions that are well described outside the cerebellum, and are even known to affect the cerebral cortex (deJong, Bolhuis & Barth, 1991; Rewcastle, 1991; Bromberg, Junck, Gebarsky, Melian & Gilman, 1990). Table 1 presents a summary of patient information. Thirteen lesions (6 left, 7 right) were caused by infarct; four lesions (3 left, 1 right) were caused by hemorrhages. Twelve lesions affected cerebellar midline regions, while four lesions affected the cerebellar hemispheres alone. Four representative MRI scans are shown in Fig. 2. The top two scans show hemispheric cerebellar lesions that do not encroach on midline regions. Contrast these with the lower two scans that show cerebellar lesions affecting the cerebellar midline region, in addition to the hemisphere.

Patients were tested 10–14 days after the ictus. Neurological evaluation showed no evidence of intellectual dysfunction. Neuro-ophthalmological testing showed dysmetric saccades in some patients as might be expected with cerebellar lesions (Leigh & Zee, 1991; Kase, Norrving, Levine, Babikian, Chodosh, Wolf & Welch, 1993), but no fixation, nystagmus, or perceptual problems to interfere with the psychophysical procedure at the time of testing. Patients had visual acuity of 20/40 or better. All patients were cooperative and fully able to perform the psychophysical task.

TABLE 1. Summary of the patients with cerebellar lesions

Patient	Age	Gender	Acuity	Hemisphere	Lesion type
Lateral cerebellar lesions					
BB	70	M	20/20	L	Infarct
DL	56	M	20/20	L	Infarct
RM	75	F	20/25	L	Hemmorhage
RS	66	M	20/20	L	Infarct
$N = 4$	$\mu = 67$				
Midline cerebellar lesions					
WB	70	M	20/25	L/R	Infarct/ hemmorhage
PC	70	F	20/25	L	Infarct
EE	55	F	20/30	L	Infarct
WE	57	M	20/30	L	Hemmorhage
JG	64	M	20/40	R	Infarct
DH	42	F	20/20	R	Infarct
WK	63	F	20/25	L	Hemmorhage
VL	67	M	20/40	R	Infarct
JP	73	F	20/20	R	Infarct
ST	54	F	20/30	R	Infarct
NW	42	M	20/20	R	Infarct
RW	69	M	20/30	R	Infarct
$N = 12$	$\mu = 61$				
Total					
$N = 16$	$\mu = 62$				

In addition, we tested normal volunteer control subjects who had no history of eye or CNS disease. To assess the possible effect of age, these normal observers were divided into three age groups: 20–40 yr ($n = 16$, mean of 27 yr), 40–60 yr ($n = 6$, mean of 43 yr), and 60 and over ($n = 13$, mean of 72 yr).

Eye movements

While this is not a study of eye movements, we did have the opportunity to record fixation and tracking of visual targets in several subjects; JG, NW, VL, and DH. Eye movement recordings used a head mounted infrared Eye Position Meter 1500 with a resolution of 2 min arc (Skalar Medical, Delft, The Netherlands). Eye positions were digitized at 400 Hz. Patients viewed a small (26 min arc) suprathreshold light target displayed on a video monitor 57 cm away. A Macintosh computer controlled both target position and the eye movement digitization.

Two types of eye-movement targets were presented. To measure fixation, a stationary target was presented in the center of the monitor. To measure smooth pursuit, a moving target with a 20 deg horizontal excursion moved across the monitor at approx. 0.4 Hz. The smooth pursuit target followed a sinusoidal or triangle waveform. Eye movements were recorded at 400 Hz.

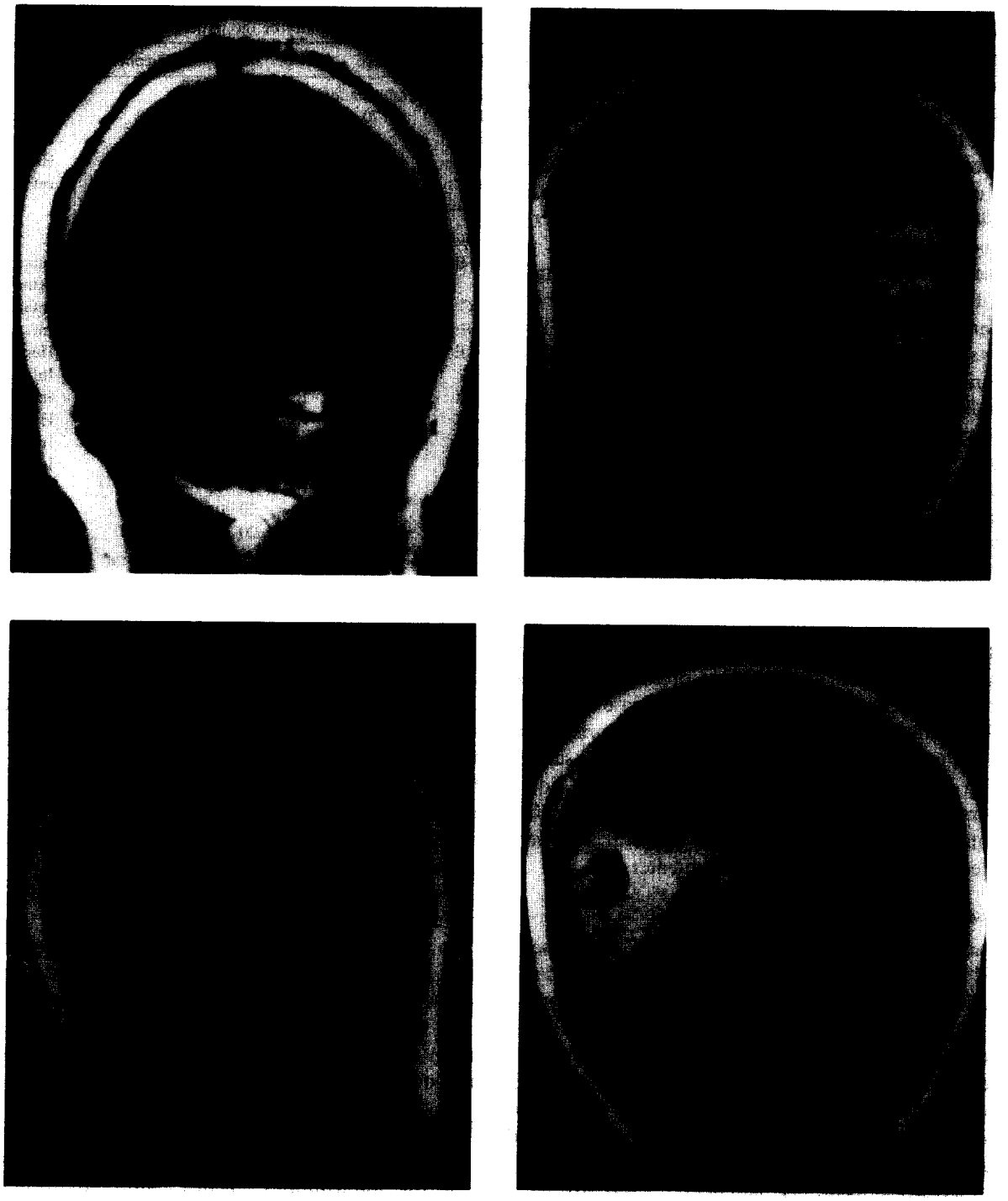

FIGURE 2. These representative coronal magnetic resonance images show cerebellar lesions in four different patients. By convention, the left side of the brain is shown on the right. Lesion locations are indicated by arrows. Shown are: (A) the left hemisphere lesion of patient BB; (B) the left hemisphere lesion of patient DL; (C) the small midline lesion of patient WE; and (D) the large left hemisphere and midline lesion of patient PC. The small arrowheads denote the lesion limit.

RESULTS

Threshold values, corresponding to the signal proportion needed to perform at 63% correct, were calculated from the psychometric function for each observer, at each speed, and at each of the five target locations (Woodworth & Schlosberg, 1954). Because there were no consistent differences in performance between the different test regions in either the cerebellar or normal groups, performance in the five regions was averaged and a single descriptive threshold value was determined. These threshold values are shown in Fig. 3.

As unilateral brain lesions often produce unilateral deficits, it was reasonable to expect unilateral deficits for these patients. However, their bilateral deficits might be explained by neuroradiological evidence that their lesions encroached upon cerebellar midline structures, damaging the vermis. The cerebellar vermis in the monkey contains visually responsive neurons with large receptive fields that span the vertical meridian, some extending out 20 deg to either side (Suzuki *et al.*, 1981).

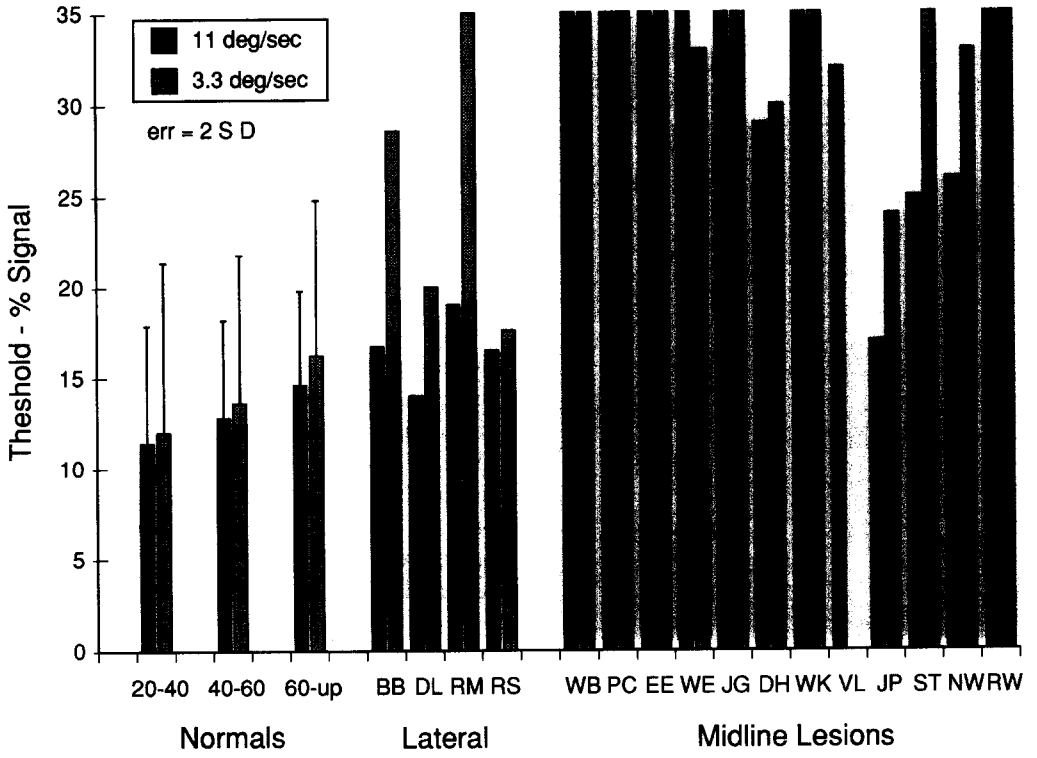

FIGURE 3. The performance of 16 cerebellar patients on a test of motion direction discrimination is shown with normal controls in different three age groups. The normal controls are shown on the left. The error bars denote 2 SDs corresponding to 96% of the normal distribution for the normal observers at that speed. Cerebellar patients, with lateralized and midline lesions, are shown on the right. Solid bars show motion thresholds for the 11 deg/sec condition, and hatched bars for the 3.3 deg/sec condition. When the calculated threshold value for a patient was above the range of our independent variable, the threshold value was truncated to 35%. This procedure under-estimates the patient's deficit but avoids large errors in estimating the threshold value beyond the range of the independent variable used for testing. One patient, VL, was not tested with the 3.3 deg/sec stimulus speed due to the occurrence of a second stroke.

Damage to such cells in the human could result in bilateral motion processing deficits.

A 3×2 repeated measures analysis of variance compared the performance of midline cerebellar and lateral cerebellar patient groups with the 60 and older patient group for the two stimulus speeds. There was a strong effect for lesion location [$F(2,26) = 77.1$, $P < 0.001$]. From the group means, it is clear that performance of the midline patients was much worse than that of the normal and the lateral patient group. There was also an effect of speed [$F(1,26) = 11.9$, $P = 0.002$] and a small indication of an interaction between lesion location and stimulus speed [$F(2,26) = 4.2$, $P = 0.026$]. This means that as a group all subjects performed worse at the 3.3 deg/sec speed than at the 11 deg/sec speed, and that this effect was more pronounced for the lateral lesion group. However, the dominant effect was for lesion location.

All patients performed well in the static control condition. The cerebellar patients had an average of 94% correct in the static control task. Short presentation durations are not a problem for cerebellar patients. Additionally, this fine performance on the control condition also indicates that the direction discrimination task was not a problem for these patients. From their performance on the control task and on the warm-up phase of the experiment we know that all the cerebellar patients understood the instructions and could perform the direction discrimination task.

Eye-movement recordings

Figure 4 shows the results from a representative patient with a midline cerebellar lesion, JG, who performed poorly on the motion perception test (see Fig. 3). JG has a midline cerebellar lesion encompassing posterior midline regions and posterior, inferior right hemisphere regions. However, JG's eye movements were quite normal. JG's eye position closely matched the target position and he was able to follow the target using smooth pursuit without stops, lags, or saccadic intrusions. JG also had stable fixation, with an average deviation of only 16 min arc over a period of 12 sec. For comparison, a subject from the 60 and older normal control group had a similar fixation recording and performed normally on the motion perception tests. Abnormal eye-movements or defective fixation were not the cause of the motion perception deficit in these cerebellar patients.

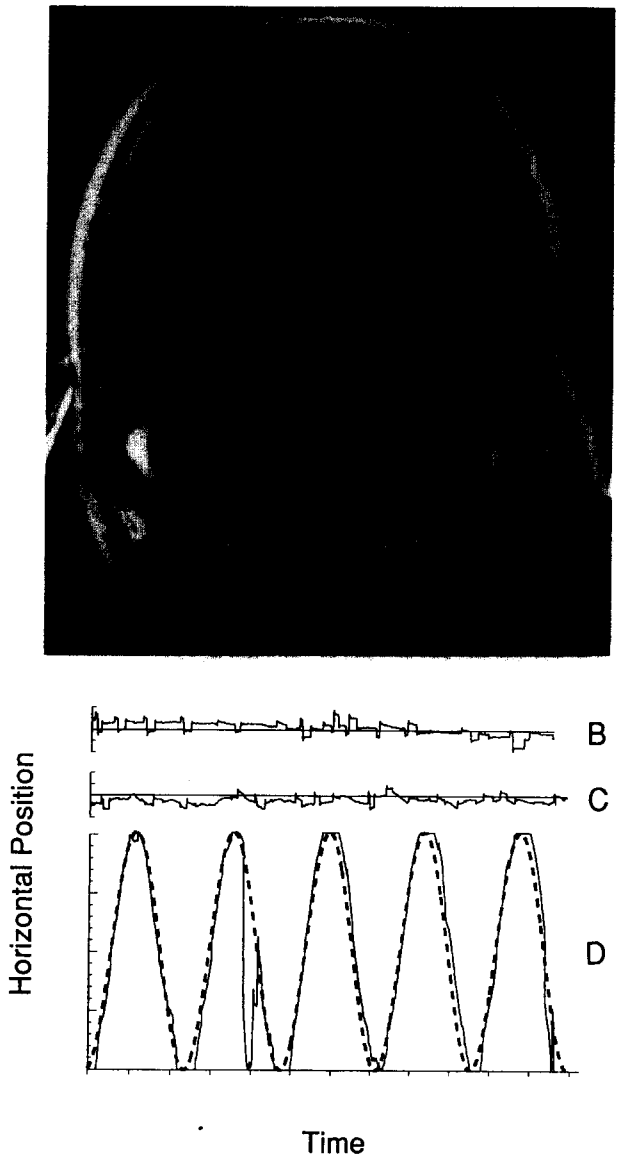

FIGURE 4. (A) This coronal magnetic resonance image shows the location of patient JG's cerebellar lesion. Similar to the representative midline lesions shown in Fig. 2, this lesion also affects posterior midline structures and part of the right cerebellar hemisphere. Shown below are eye-movement recordings from this patient. Horizontal eye position is shown on the vertical axis with each tick representing 1 deg of visual angle. Time is shown along the x-axis, with each tick representing 1 sec. (B) This tracing shows the fixation of a normal control from the 60 and older group. (C) This is a record of this patient's fixation over a period of 12 sec. The average deviation of eye position was 16 min of visual angle, smaller than the fixation target size. (D) The bottom tracing shows this patient's normal smooth pursuit of a moving target (target path shown with the bold dotted line, eye position by the thin solid line). The excursion in eye position in the second cycle is a blink artifact.

DISCUSSION

Patients with acute midline cerebellar lesions show elevated thresholds in a motion direction discrimination task. Nine of the 12 cerebellar patients with midline lesions had significantly higher thresholds than normal observers at both speeds (>2 SDs); the slopes of these patients' psychometric functions were more shallow than those of normal observers. These patients were no longer able to indicate the direction of global motion in these displays until the proportion of signal to noise was very high.

However, five patients BB, DL, RS, JP, and RM (in 11 deg/sec condition) performed very similar to normals. Correlation of the psychophysical results with the locus of cerebellar damage suggests that motion direction discrimination performance was related to lesion location. Four of the five patients performing normally has lesions of the lateral cerebellar hemisphere. The patients showing deficits in the motion task had cerebellar lesions affecting cerebellar midline structures, including the vermis, and the connections to and from those structures. Of course, we found evidence of variability in the effect of these midline lesions in one patient. Patient JP had a large midline lesion (very similar in location and size to that of patient JG shown in Fig. 4) yet performed normally in these motion perception tests. Some variability is expected when correlating lesion location with a functional deficit.

Comment on fixation and eye movements

The performance deficits in these patients were not the result of defective fixation and eye movements. First, all patients demonstrated stable fixation prior to testing. A second factor is the design of our motion testing procedure: patients triggered trials themselves only when confident of fixation, the subsequent stimulus presentation duration was brief (200 msec), and the fixation point was visible on the screen for the duration of the stimulus presentation. Together, these factors make eye movements during the actual cinematogram presentation highly unlikely. Third, it is significant that patients *could* perform the task at higher signal proportions; only at lower signal proportions did patients begin to have difficulty. If defective fixation caused poor performance, patients would have performed poorly at all signal proportions. Similarly, a *post hoc* analysis shows that errors were similar in normals and patients: in both groups close to one-third of the errors were in the direction opposite actual signal movement. This pattern of errors suggests that the proportion of stimulus signal was too low for detection and essentially random errors resulted. Finally, we have documented normal fixation and normal smooth-pursuit eye movements in a sub-set of these patients who show motion perception deficits. Therefore, the perception deficit seen here is not the simple and direct result of an ocular motor problem.

A cerebellar role in visual motion perception

This finding suggests that the cerebellum has an important role in the normal perception of movement. However, the cerebellum is certainly not of singular importance. The cerebellum is likely one component of a motion perception processing network. This network may include visual areas in occipital cortex, temporal visual cortex, posterior parietal cortex, the frontal eye fields, and the superior colliculus. Connections to the cerebellum pass through the DLPN, which also participates in the visual guidance of movement (Spatz &

Tigges, 1973; Schmahmann & Pandya, 1991; Glickstein, Cohen, Dixon, Gobson, Hollins, LaBossier & Robinson, 1980; Glickstein, May & Mercier 1985; Leichnetz, 1989; Fries, 1990; Suzuki *et al.*, 1990; Mustari, Fuchs & Wallman, 1988). Completing the circuit, the cortical feedback pathway originates with cerebellar efferents to the thalamus, which in turn sends reafferent projections up to association and motor cortex (Asanuma, Thach & Jones, 1983; Yeterian & Pandya, 1989; Shinoda, Kakei, Futami & Wannier, 1993). The similarities between vermis and MT neuron response properties may reflect the central role of this cortico-ponto-cerebellar pathway, the detection and processing of broad areas of movement across the retina. Interconnections between cortex and the cerebellum would integrate those areas performing similar processing tasks. Clearly, these results indicate that damage to this circuit at the level of the cerebellum is sufficient to produce a motion processing deficit in humans. This was previously thought to be the result of damage to cortical areas alone.

Additional support for the idea that this circuit, as a whole, may play an important role in motion perception comes from the "motion blind" patient LM of Zihl, Von Cramon and Mai (1993). LM has extraordinary complaints regarding the appearance of movement in her environment and shows profound motion perception deficits (Hess, Baker & Zihl, 1989; Baker, Hess & Zihl, 1991). LM's deficit is associated with bilateral temporo-parieto-occipital lesions that presumably damaged the human homolog of the monkey's area MT in both hemispheres. When tested with the type of stimuli used in the current experiment, LM had a motion detection threshold of about 40% (Rizzo, Nawrot & Zihl, 1993). This performance is similar to that of patients with midline cerebellar lesions. Interestingly, LM is now also noted to have a large right cerebellar lesion extending into midline regions (Zihl, Von Cramon, Mai & Schmid, 1991). Given the findings in the current study, it is important to consider whether this cerebellar lesion may also play a role in her deficit. Indeed, the findings in the current study indicate that damage to the cerebellum alone, in the absence of a cortical lesion, such as in a human area MT homolog, is sufficient to cause a motion perception deficit.

Given the traditional view of the cerebellum as solely a motor coordination center, it is not immediately obvious why a cerebellar lesion would produce the perceptual deficit described here. We propose that cerebellar involvement in motion perception may relate to the perceptual stabilization of the visual image, a perceptual concomitant of the mechanism for ocular motor stabilization.

The visual panorama is in constant movement across the retinal array due to eye, head, or body movement. A neural mechanism is needed to distinguish whether object movement across the retina is the result of actual object movement in the environment, or the result of self movement. The perceptual difference between active and passive movement of the eyes is often explained by a corollary discharge hypothesis (Teuber, 1960) that postulates a comparator mechanism to integrate information on visual motion with eye, head or body movement. A corollary discharge hypothesis was advanced by Yasui and Young (1975) to explain the stabilization of images on the retina during smooth pursuit. However, corollary discharge and a comparator mechanism can also be invoked to explain the stabilization of perceptual experience.

The cerebellum, with its multiple sensory and motor connections, seems ideally suited for a role in this perceptual stabilization. A cerebellar lesion may disrupt perceptual stabilization, or generate internal noise in this stabilization process, resulting in a deficit for the discrimination of visual movement. Moreover, the abnormal smooth pursuit eye movements observed in some cerebellar patients may have a perceptual component just as they do in primates with motion perception deficits caused by acute lesions of area MT (Newsome, Wurtz, Dürsteler & Mikami, 1985) or humans with unilateral cortical lesions (Thurston, Leigh, Crawford, Thompson & Kennard, 1988; Morrow & Sharpe, 1993). Defective smooth pursuit eye movements in some patients with cerebellar lesions might also be caused by an abnormality of the visual movement signal needed to monitor pursuit accuracy.

This study shows that cerebellar lesions affecting midline regions, including the vermis, produce a deficit in a motion direction discrimination task. This motion perception deficit may be related to the cerebellum's role in the perceptual stabilization of visual movement produced by eye, head, or body movement. Cortical mechanisms are still of primary importance for the conscious appreciation of motion, but the cerebellum is an important component in a broader visual motion processing network. This network is subserved by the cortico-ponto-cerebello-thalamo pathway, with multiple connections between several brain regions. Integrity of this circuit appears critical for normal motion perception. We have evidence for motion perception deficits resulting from both posterior parieto-temporal lesions (Zihl *et al.*, 1991; Nawrot, Rizzo & Damasio, 1993) and from midline cerebellar lesions. However, lesions affecting other brain regions or connections between brain regions may also disrupt this circuit and produce similar motion perception deficits. Further lesion studies in both humans and primates may identify other cortical and subcortical areas with similar importance in motion perception.

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