Impaired Velocity Perception in Patients with Lesions of the Cerebellum

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Abstract

■ In three psychophysical experiments, cerebellar patients were impaired in making perceptual judgments of the velocity of moving stimuli. Performance was normal when the judgment concerned the position of the stimuli (Experiment 1). The dissociation between the velocity and position tasks suggests the cerebellar group was selectively impaired in velocity perception. EOG data were obtained in Experiments 2 and 3 to assess whether the deficit was oculomotor in origin. Perceptual errors were not correlated with the occurrence of intrusive eye movements. These results provide a novel demonstration of the role of the cerebellum in perceptual functions that require precise timing. ■

INTRODUCTION

In previous research, we have found that the integrity of the cerebellum is critical for the operation of an internal timing process (Ivry & Keele, 1989; Ivry, Keele, & Diener, 1988). Patients with cerebellar lesions are impaired at producing periodic movements of approximately 2 Hz and at judging the duration of comparable auditory intervals. The latter deficit on a purely perceptual task was most surprising given that the cerebellum has historically been associated with motoric computations. Expectation of a perceptual deficit in cerebellar patients required a reconceptualization of one aspect of cerebellar function in terms of time-based computations. We hypothesize that, within a certain temporal range, the cerebellar timing mechanism is invoked whenever a temporal computation is required (Keele & Ivry, 1991).

To further test this hypothesis, we investigated other possible perceptual functions that may require timing or a time-based computation. A straightforward extension is to substitute visual stimuli for the auditory stimuli. In preliminary experiments, cerebellar patients were impaired in judging the duration of visual stimuli (Ivry, 1989). In the current studies, we test the patients on a new task that would appear to require accurate timing, velocity perception. Further motivation was provided by evidence demonstrating the role of the cerebellum in the control of both smooth pursuit and saccadic eye movements (Aschoff & Cohen, 1971; Lisberger & Fuchs, 1978; Ritchie, 1976; Ron & Robinson, 1973; Westheimer & Blair, 1974). Since one important function of eye movements is to track a moving object, it is incumbent that the eye movement system have access to the representation of the object velocity. This ability may utilize the timing capabilities of the cerebellum.

Cells in the cerebellum respond to visual stimuli (Buchtel, Rubia, & Strata, 1973; Ito, 1984; Larsell, 1953; Snider & Stowell, 1944; Suzuki & Keller, 1988). Moving visual stimuli are at least as effective an input as stationary objects (Donaldson & Hawthorne, 1979; Waespe & Henn, 1985). The functional significance of this response characteristic, however, remains unclear. Motion information can be useful for many separable computational processes (Nakayama, 1985) including object identification and localization, as well as metrical information concerning speed and direction. Given that cells in many regions of the central nervous system respond to visual stimuli (e.g., Huerta & Harting, 1984; Maunsell & Van Essen, 1983), it is unlikely that all of these computations are performed by each region. More likely, there is a division of labor. Thus, a functional description of a neural system may be facilitated by a computational theory of the processing goals for that system. The timing hypothesis of cerebellar function (Ivry & Keele, 1989; Keele & Ivry, 1991) suggests that information about the

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accurate on the velocity task than the control subjects. The two groups did not differ on the position task.

A second method of analyzing the data is further supportive of this dissociation. The two scores for each subject can be compared to establish which task proved more difficult for that subject. Twelve of the 16 control subjects performed better on the velocity task in comparison to the position task.[1] In comparison, only four of the 18 cerebellar patients were better on the velocity task with one subject producing identical scores on both tasks. These values are significantly different than expected by chance ($X^2 = 7.58, p<.01$).

In summary, the cerebellar patients were found to be impaired in making perceptual judgments comparing the velocity of two moving stimuli. No differences were found on the position task. The dissociation in performance between the two tasks demonstrates that the patients' deficit cannot easily be attributed to generalized difficulties in psychophysical testing. The patients were specifically impaired in the velocity task, the test that we assume involves a temporal computation. We believe this finding, coupled with our earlier results concerning time perception (Ivry & Keele, 1989), provides a second demonstration that the functions of the cerebellum may extend beyond strictly "motoric" tasks. The timing hypothesis postulates that the integrity of the cerebellum will be critical for perceptual or motor tasks that require precise timing. The results in the current experiment support our hypothesis that velocity perception would involve the cerebellar timing mechanism.

EXPERIMENT 2

An oculomotor-based explanation could account for the perceptual deficit observed in Experiment 1. Suppose the patients are unable to maintain fixation, generating pursuit or saccadic tracking movements. This might render the stimulus less salient. Alternatively, perceptual judgments concerning the velocity of a stimulus may be arrived at indirectly by reference to a movement elicited by that stimulus. For example, a test stimulus might be judged faster than a standard if the pursuit eye movement elicited by the test stimulus is faster than that elicited by the standard. If the eye movements are impaired and serve as the basis for perception, then the perceptual deficit could be secondary to the motor problem. Another variation of an oculomotor explanation rests on the assumption that velocity perception is based on a retinal slip signal. If the eye movement gain is near unity, slip will be minimal and target velocity can be inferred from eye movement velocity. However, if the gain is aberrant, there will be a greater mismatch between stimulus velocity and eye velocity. This mismatch might lead to an increase in erroneous perceptual judgments.

Eye movements were not monitored in Experiment 1 and the position task precluded the use of a fixation point. Experiment 2 was designed to test whether the velocity perception deficit can be attributed to unintended eye movements with cerebellar patients. To do this, subjects' eye movements were monitored during the presentation of the stimuli. This allowed us to assess two aspects of the movement hypothesis. First, were the subjects able to maintain fixation? If not, was the intrusion of eye movements associated with erroneous percepts?

In addition, we sought to explore whether the velocity perception deficit would be obtained over a different range of test velocities. McKee (1981; McKee & Welch, 1985) has reported that, although the Weber fraction for velocity perception (Velsd/Base Vel) is constant over a wide range of velocities, there is an increase in the discrimination threshold for velocities below 2°/sec. Thus, in Experiment 2, the tested velocities ranged from 2 to 5°/sec.

Results and Discussion

Velocity Acuity

The mean standard deviation scores for the three groups are presented in Figure 2. The estimated variability for all three groups is quite small.[2] The mean for the control group is approximately 7% of the mean velocity for the stimulus set and the percentage is only slightly larger for the cerebellar group. These values are in agreement with previous estimates of normal velocity perception (McKee, 1981; McKee & Welch, 1985). Nonetheless, the variability estimates for the cerebellar patients are significantly higher than for the age-matched control subjects [$t(16) = 2.78$; $p<.01$]. This result extends the findings of Experiment 1: the velocity perception deficit

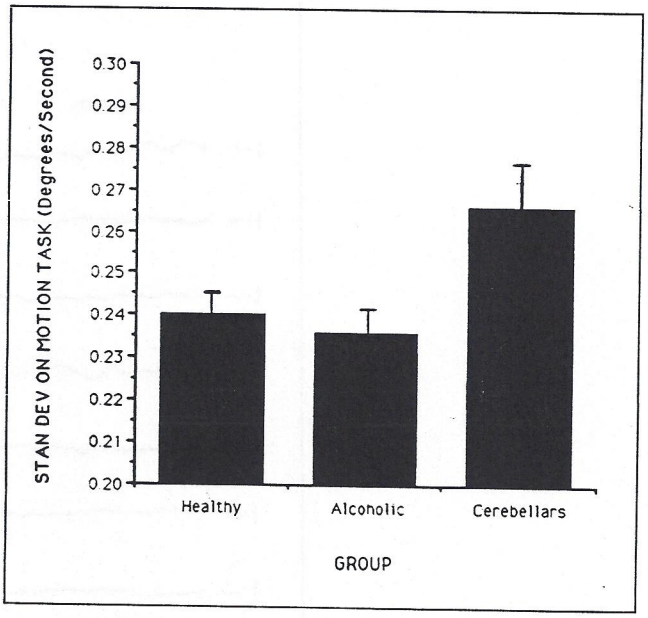

Figure 2. Mean standard deviation estimates for each group in Experiment 2. The bars indicate the standard errors of each mean.

surement noise, a quantitative estimate of the additional eye movement can be made for the patient data. This technique indicates that, on average, the patients produced approximately 0.61° greater eye movement per trial.

However, the EOG data tended to be bimodal: on some trials, the subjects were able to maintain fixation whereas on other trials they were not. This can be seen in Figure 3. Many of the traces show no consistent deviation from baseline. On other trials, especially the last three for DEH, pursuit eye movements are evident. Of primary interest is whether there is a relationship between velocity perception and the occurrence of eye movements. Specifically, were perceptual errors more likely to occur on trials in which the subject made an eye movement? For the second quantitative analysis, separate mean EOG difference scores were calculated for correct and incorrect trials. The EOG difference scores are essentially identical for correct and incorrect trials for all three groups. The critical statistical test is superfluous since the cerebellar patients showed slightly less EOG activity on trials in which their responses were incorrect. This result argues against a motor-based explanation for the velocity deficit. Moreover, the data for the alcoholic patients further demonstrate a lack of correspondence between the amount of EOG activity and performance on the velocity task. The EOG difference scores for each of the three alcoholic patients were higher than the mean for the cerebellar patients. Nonetheless, the alcoholic patients showed normal performance on the perception task.

Third, modified correlation coefficients were calculated for each subject between EOG activity and perceptual performance. The EOG difference score for each trial was correlated with the type of response for that trial by entering a 1 for every correct response and a 2 for every incorrect response. If errors were associated with eye movements, then these correlations should be positive. Contrary to this prediction, the correlations for all of the patients and control subjects were nonsignificant, ranging from $-.10$ to $+.12$.

One other oculomotor-based account of the perceptual deficit should be considered. As stated earlier, the crucial variable is not whether eye movements occurred, but whether the velocity of the eye movement matched the velocity of the stimulus. A mismatch between these two variables would lead to greater retinal slip and this might underlie the perceptual deficit. This possibility, however, is weakened by the preceding analyses. More errors would be expected on trials with intrusive eye movements since these are the only times when a mismatch could occur. As discussed above, there was no apparent relationship between perceptual performance and the magnitude of EOG activity. Taken together, the EOG analyses indicate that a motoric explanation is unlikely to account for the deficit in velocity perception observed in cerebellar patients.[3]

EXPERIMENT 3

Experiment 3 provides a third test of velocity perception in cerebellar patients. A different technique for generating the apparent motion displays was employed which allowed for finer step differences in the stimulus set. Eye movements were again monitored and a new range of stimulus velocities was tested.

Results and Discussion

Velocity Acuity

The mean standard deviation scores for the three groups are presented in Figure 4. The results are in agreement with those obtained in the previous two experiments. The cerebellar patients demonstrate a deficit in velocity perception in comparison to the control subjects [$t(13) = 2.86, p<.01$]. As in Experiment 2, the alcoholic patients performed within the range of normal scores.

The individual data demonstrate that the difference is quite consistent across the patients. Whereas the five best scores were obtained with the control subjects, the patients produced the four poorest scores. The scores for the three focal patients, F.E.N., K.A.L., and P.R.I. were .347, .344, and .352, respectively. Thus, the deficit was at least as pronounced in the patients with focal lesions as it was in patients with cerebellar atrophy. Indeed, the only patient who performed better than the mean for the control subjects was the patient with olivopontocerebellar atrophy (OPCA).

Analysis of Eye Movements

The EOG analysis indicates that the perceptual deficit cannot be parsimoniously attributed to a problem in the control of eye movements. There was no difference between the control and cerebellar groups in terms of the

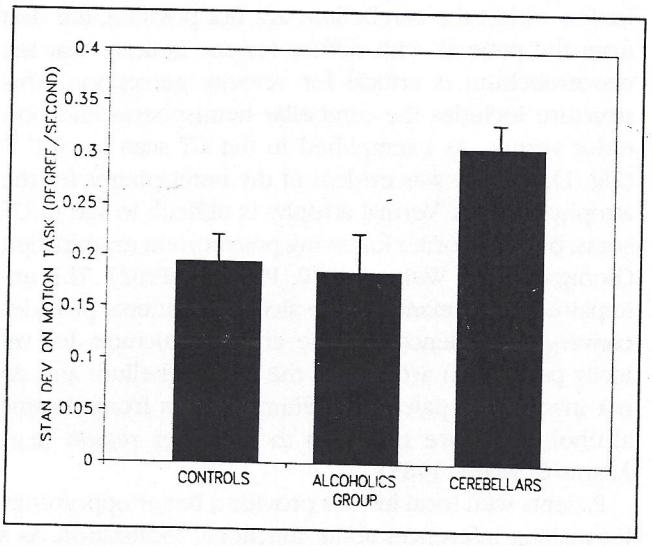

Figure 4. Mean standard deviation estimates for each group in Experiment 3. The bars indicate the standard errors of each mean.

Neuroanatomical data indicate that the cerebellum receives visual input that is not cortically mediated. There is extensive subcortical distribution of information carried by retinocollicular fibers (see Huerta & Harting, 1984 for an extensive review) including projections to the pons (Hashikawa & Kawamura, 1977; Kawamura & Brodal, 1973) and inferior olive (Saint-Cyr & Courville, 1982). The primary cerebellar afferent systems originate in these regions.[4] It is also clear that the visual areas of the cortex project to the cerebellum via the pons (Brodal, 1972a,b; Glickstein, May, & Mercier, 1985; Legg, Mercier, & Glickstein, 1989). However, pontine projections from striate, extrastriate, and inferotemporal cortex are minimal in primates (Glickstein et al., 1985), making it unlikely that the cerebellar input for velocity information is mediated by area MT.

Timing and the Cerebellum

Coupled with our earlier findings (Ivry & Keele, 1989), the current studies provide converging evidence implicating the cerebellum in perceptual functions requiring precise timing. The mechanisms by which these computations are performed remain unknown. We (Ivry & Keele, 1989; Keele & Ivry, 1991) have proposed that the cerebellum is involved in a real-time computational process that can compute intervals between successive stimulus events (e.g., time perception), subcomponents of response sequences (e.g., repetitive tapping), or intervals between stimuli and responses (e.g., classical conditioning). Alternatively, the cerebellum may not be involved in computing the intervals, but rather in encoding the onset and offset of stimulus and response events. A loss of temporal resolution could underlie both the auditory deficit observed in Ivry and Keele (1989) and the current visual deficit. It remains for further study to explicate the role of the cerebellum in these or other component operations of timing.

METHOD

Subjects

Cerebellar Patients

Twenty patients with a cerebellar disorder were tested. The diagnosis, based on clinical examination and radiographic material (CT or MRI), was attributed to atrophy (n = 14), tumor (n = 3), stroke (n = 2), or an autoimmune reaction (n = 1). Two of the atrophy patients were brother and sister indicating a familial component to their disease. For the other atrophy patients, the disease appeared to be spontaneous. Patients were excluded who showed signs of other neurological disorders or for whom the radiographic data showed atrophy of the cerebral cortex or enlarged ventricles.

The lesions were unilateral for four of the patients due to either tumor (n = 3) or stroke (n = 1). The focus of the lesion was on the right side for three of the patients (see Fig. 5) and on the left side for the other patient (see Ivry et al., 1988, Case 2). Clinical symptoms were mild to moderate for patients K.A.L., P.R.I., and C.O.N. and severe for F.E.N., P.R.I. and F.E.N. presented symptoms consistent with involvement of the cerebellar hemispheres such as dysmetria and intentional tremor in pointing. F.E.N. and K.A.L. also had disturbances in balance and gait.

The other 16 patients had diffuse, bilateral lesions of the cerebellum. For all of the atrophy patients, the lesions and symptoms were consistent with atrophy primarily affecting the hemispheres and posterior vermis (Dichgans & Diener, 1984; Konigsmark & Weiner, 1970; Plaitakis, 1982). The CT scan for one of the atrophy patients, C.U.T., is included in Figure 5. This patient presented severe intentional tremor in finger-to-nose testing and gross hypermetria in pointing. He also had some disturbances in postural control. The deficits were generally milder in the other patients with a diagnosis of cerebellar atrophy. However, the diagnosis for five of the atrophy patients was OPCA based on clinical presentation of extracerebellar symptoms and/or neuroimaging evidence of pontine atrophy. In many cases, the distinction between cerebellar atrophy and OPCA is a matter of degree, with the latter representing a more advance stage of the atrophic process (Klockgether, Schroth, Diener, & Dichgans, 1990). The autoimmune patient presented an array of moderate cerebellar signs including dysmetria, gait problems, and dysarthria. The remaining stroke patient had suffered an ischemic attack that led to mild bilateral symptoms.

The twenty patients ranged in age from 21 to 76. Eighteen of these patients participated in Experiment 1 (mean = 55.7 years, SD = 15.5), six in Experiment 2 (mean = 66.3 years, SD = 5.8), and seven in Experiment 3 (mean = 61.2 years, SD = 10.4).

Control Subjects

A total of 27 control subjects were recruited. They reported no history of neurological disease. These subjects were all between the ages of 51 and 77. Sixteen of these subjects participated in Experiment 1 (mean = 68.0 years, SD = 6.9), 12 in Experiment 2 (mean = 66.3 years, SD = 5.8), and 8 in Experiment 3 (mean = 66.6 years, SD = 7.7). Note that the mean ages for the control subjects were older than the patient group in all three experiments. This should provide a conservative bias if age is a factor in performance on the experimental tasks.

Cerebellar Controls

A second control group was recruited for Experiments 2 and 3. These groups consisted of patients with cere-

for 1 sec after which the second stimulus was presented. The duration of Stimulus 2 was also randomly determined. One second after the offset of Stimulus 2, the words "Faster or Slower" (velocity task) or "Higher or Lower" (position task) appeared on the screen. The subjects were instructed that, in the velocity task, they were to judge if Stimulus 2 was faster or slower than Stimulus 1 and in the position task, whether Stimulus 2 was higher or lower than Stimulus 1. The subjects responded by pressing one of two keys on a response board. An intertrial interval of 1 sec preceded each trial.

Velocity Task. The speed of the stimulus was manipulated by varying the time between the successive 6′ of arc displacements of the stimulus. There were seven stimulus steps, created by using different stimulus-onset asynchronies (SOA). These ranged from 83 to 183 msec in steps of 16.67 msec. The fastest speed simulated was 1.20°/sec when the SOA = 83 msec. The slowest speed of 0.54°/sec was produced when the SOA = 183 msec. The middle SOA of 133 msec (speed = 0.75°/sec) was presented as the standard stimulus on every trial. The comparison stimulus was set to one of the seven SOA values. By randomly varying the number of dots in the line, density over time was unconfounded with velocity. Furthermore, by randomly varying the duration of the stimuli, subjects could not use a strategy such as to count the number of dots that appeared during each stimulus duration. No subjects ever reported attempting to utilize such a strategy.

The standard stimulus was presented first on half of the trials and second on the other half. There were 70 trials per block, 10 for each of the seven values for the comparison stimulus. The subjects were tested on two blocks of the velocity task.

Position Task. As in the velocity task, each trial in the position task consisted of two moving lines of dots. The speed of both stimuli was held constant at 0.75°/sec. The manipulated variable was the vertical position of the two stimuli. On each trial, the vertical position of the standard stimulus was first selected. This stimulus was randomly placed within ±1.36° of the vertical center of the monitor. Seven stimulus steps were employed by shifting the position of the comparison stimulus to one of seven relative locations. These ranged from 3 pixels above or below the position of the standard stimulus in steps of 1 pixel. At 100 cm, each pixel corresponded to a visual angle of 3.4′ of arc. Thus the largest shift was 10.2′ of arc and the two smaller shifts were 6.8′ and 3.4′ of arc. For the middle stimulus level, the comparison stimulus was at the same location as the standard.

All other aspects of the position task were identical to the velocity task.

Control of Task Factors. Subjects were tested individually in a dark room, seated 100 cm from the monitor. The subjects were instructed to keep their eyes fixated at the center of the screen. A fixation point was not provided since this would have provided a clear reference mark for the position task. While the edges of the monitor were made visible by the illumination of the stimuli, they would only be capable of providing a weak reference since they were continuous without any specific markings. Each subject completed four blocks of trials, two of each task. Half of the subjects performed the tasks in the order velocity, position, position, and velocity. For the other half, the order was position, velocity, velocity, and position. There was a 30-min break between the second and third blocks. The first block of each task was preceded by a practice block of 28 trials, four of each stimulus value.

The test values for Experiment 1 were selected after pilot testing with college students. The goal of the pilot testing was to determine stimulus parameters that would produce approximately equal performance on the two tasks when measured in normalized standard deviation scores, or step units. This allows comparison across tasks despite the difference in units. The mean standard deviation scores for the pilot subjects were 1.55 and 1.62 steps for the velocity and position tasks, respectively.

Experiment 2

The stimulus set was composed of six different velocities ranging from 2.00 to 5.00°/sec. The set was created by combining three displacement distances and two stimulus-onset asynchronies. Displacements of 10′, 15′, and 20′ of arc were used (2, 3, and 4 pixels). The two SOAs were 67 and 83 msec. Thus the fastest stimulus of 5°/sec is created by moving each dot 20′ of arc every 67 msec. Given the larger displacements in Experiment 2, it was necessary to increase the spacing between neighboring dots so that false correspondences would not occur. The density of the line was reduced so that each stimulus contained between 9 and 12 dots.

Only a single stimulus was presented on each trial, the exposure duration ranging from 600 to 700 msec. The subject judged whether the velocity of this stimulus was slower or faster than the velocity of an implicit standard. Two techniques were used to help the subjects establish a criterion of this standard. First, 20 practice trials with only the two endpoint stimuli were presented prior to each test block. Second, the first 24 trials of a test block were not scored, but included to allow the subjects to establish a criterion. The 24 trials included four presentations of each stimulus velocity. This modified method of constant stimuli has been found to produce robust variability estimates in psychophysical testing (see Kling & Engen, 1971) including experiments on velocity perception (McKee & Nakayama, 1984).

Subjects completed two practice blocks of 20 trials and two test blocks of 120 trials. Sixteen trials for each of the

from the intermediate and deep layers. However, some tectopontine fibers originate in the optic stratum and there are extensive intralaminar connections within the superior colliculus (Hashikawa & Kawamura, 1977).

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