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Chronic motion perception deficits from midline cerebellar lesions in human

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Abstract

A selective motion perception deficit is seen in patients with acute midline cerebellar lesions. Patients with more lateralized acute cerebellar damage do not demonstrate such a deficit (Nawrot M, Rizzo M. Vis Res 1995;35:723–731). However, as these patients were tested only between 10 and 14 days post-ictus, the stability of this perceptual deficit into the chronic phase remained undetermined. The current study extends the previous findings by showing that the motion perception deficit caused by mid-line cerebellar lesions remains permanent at least 2 years into the chronic phase. The extent and longevity of this deficit resembles that of the well known motion-blind patient LM who has a large cerebellar lesion in addition to her extensive cortical damage. Again, we propose that the mid-line cerebellar damage may produce a severe motion perception deficit by disrupting the visual-motor integration mechanisms involved in perceptual stabilization, even though cortical motion processing mechanisms are unaffected.

Keywords: Motion perception; Cerebellum; Brain lesion

1. Introduction

There is growing appreciation for the role of the cerebellum in normal cognitive and perceptual function [2,3]. Recently, a functional MRI study [4] suggested that motor control is not the cerebellum's primary function. Instead, Gao and colleagues found that the cerebellum is most strongly activated during the acquisition and discrimination of sensory information suggesting that high cerebellar activity during motor or cognitive activities is due to the requirement of processing sensory information. Therefore, while the cerebellar role in motor control is well known [5], it appears that the cerebellum is involved in a wider range of neural processing including sensory acquisition and visual function.

Previously we have shown that acute midline cerebellar damage can produce a selective motion perception deficit in human [1]. This deficit closely mimicks the functional deficit seen with acute MT damage in monkey ([6]; see also Ref. [7]). This is an interesting result due to the fact that the motion blind patient, LM, has a large cerebellar lesion affecting some midline structures [8]. We were intrigued by the possibility that this cerebellar lesion might have some role in LM's extraordinary reports of her motion perception problems [9].

Of course, it is unlikely that the cerebellum is the primary motion processing site in the brain. Previously we proposed that cerebellar involvement in motion processing might be related to the brain mechanism of image stabilization necessary due to body, eye, or head movement. That is, as the body moves it creates concomitant eye movement and also movement of the visual scene across the retina. The visual system must determine whether this image movement was due to body movement or movement in the environment. With neurons selective to the direction of image motion [10,11]) and neurons conveying motor and vestibular information [12], the cerebellum appears well suited to provide the means for the visual system to prevent the misinterpretation of motion information in conditions of self movement. Disruption within this processing network may produce a motion perception deficit.

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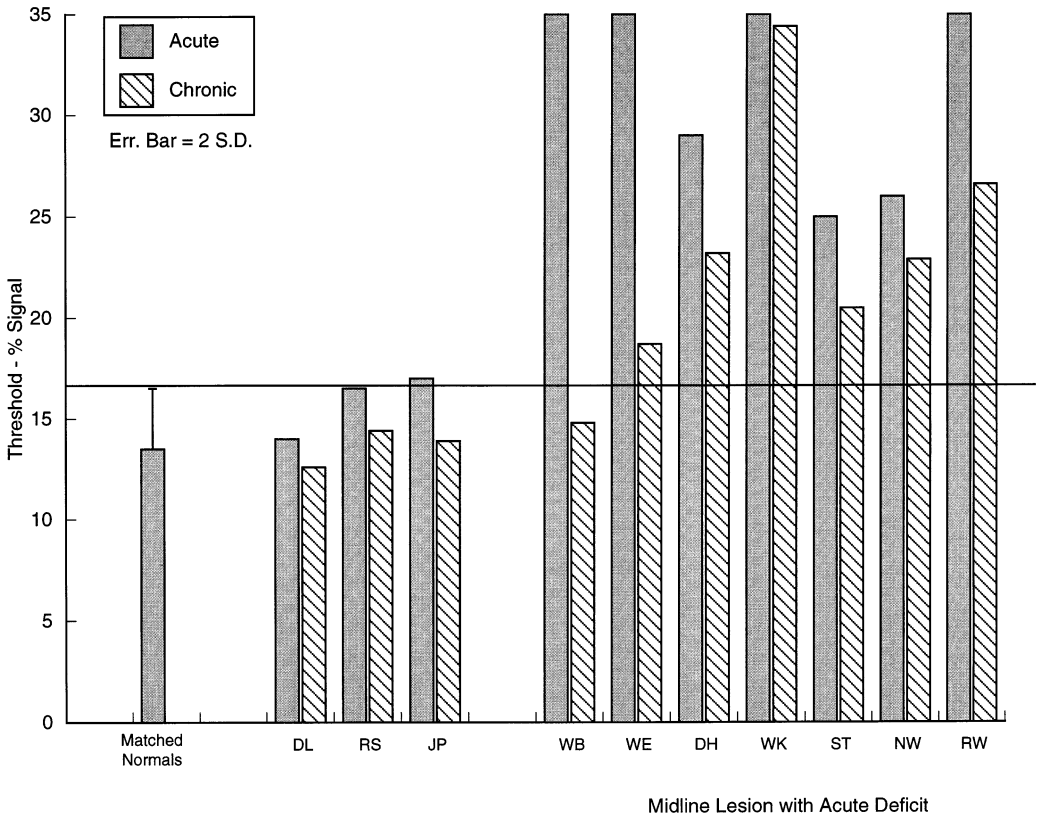

Fig. 1. Shown are the thresholds for the 11°/s motion direction discrimination task. On the right are acute and chronic thresholds for a group of patients who had both mid-line cerebellar lesions and acute motion perception deficits. Filled bars show acute motion thresholds and hatched bars show chronic thresholds. Shown for comparison are: (1) thresholds from patients with lateral (DL and RS) or mid-line (JP) lesions who did not demonstrate an acute phase deficit, and (2) thresholds from a group of age matched controls ($n = 10$, mean age = 62 years). The error bars denote 2 S.D. ($P < 0.023$).

With this background, it is important to establish the duration and possible recovery of motion perception following cerebellar lesions. With MT lesions, small or incomplete lesions produce deficits that improve quickly after the initial damage [6]. However, Pasternak and Merigan [7] show that the persistence of these deficits is related to the size and extent of the lesion. As 'recovery is unlikely for specific functions controlled by localized brain areas if all of that area is removed [13]', a persistent deficit is evidence that this region is involved in a specialized function that can not be taken over by neighboring undamaged neurons.

To determine whether or not the deficit seen with acute lesions is still evident in a much later chronic testing, we had patients participating in the original acute study return a couple years after their initial stroke. Patient performance was measured with the same array of motion and static tests. When possible, we also made eye movement recordings from the patients.

2. Methods

2.1. Stimuli

The method was similar to those detailed in Nawrot and Rizzo [1]. Briefly, computer generated random-dot cinematogram (RDC) stimuli were used to assess motion perception. RDCs comprised 150 randomly positioned small (2×2 min arc) black dots presented on a white computer screen. Each of the dots moved about within an invisible 4° aperture. When a dot's movement took it outside of this aperture, a wrap-around procedure reintroduced the dot to the opposite side of the aperture where the dot continued the original movement.

Dot movement was created by assigning small positional displacements to each dot every 15 ms frame. While the size of these displacements was constant for each frame (10 or 6 min in different conditions), dots were randomly assigned a new direction of displace-

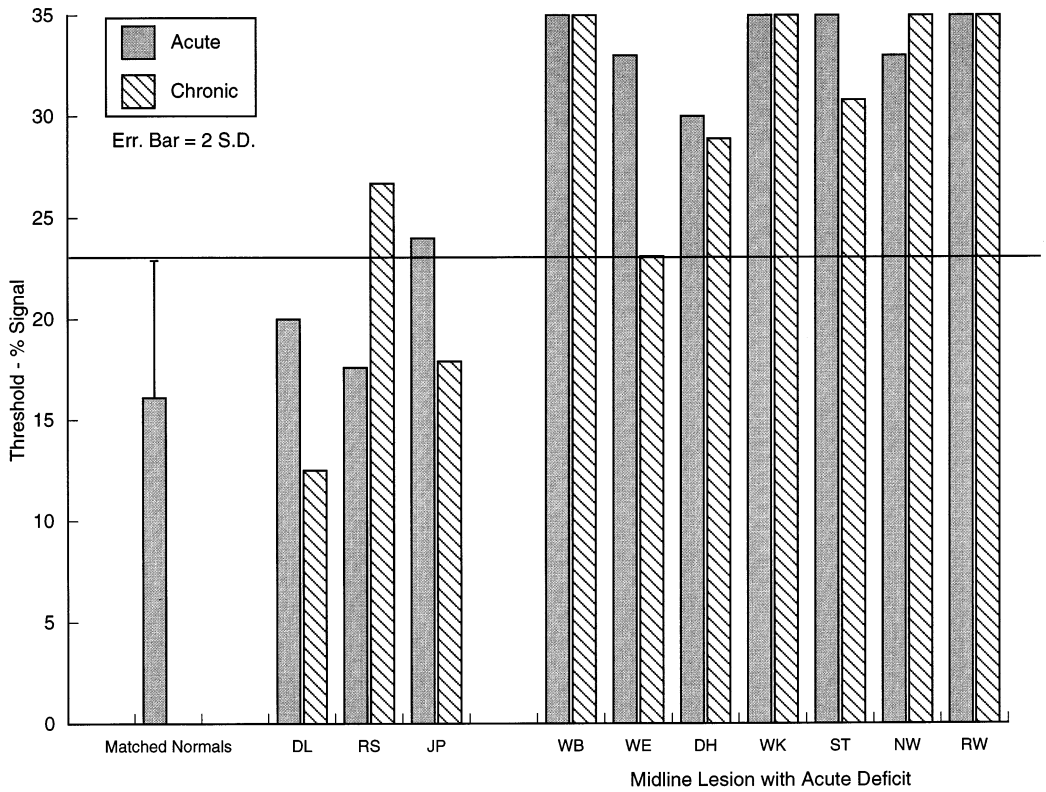

Fig. 2. Shown are the thresholds for the 3.3°/s motion direction discrimination task.

ment from one of two distributions. The signal distribution contained displacements in one particular direction, either upwards, downwards, leftwards, or rightwards, which varied between trials. The noise distribution contained a flat distribution of displacement directions encompassing 360°. The proportion of dots assigned displacements from either of these two distributions varied between trials using the method of constant stimuli. When the proportion of dots assigned signal displacements was large (e.g. 50% signal), it was easy to distinguish the particular direction of the signal displacements and dot movement. When the signal proportion was low (e.g. 10% signal) it was difficult to determine the direction of signal dot movement due to the high proportion of noise dot movement, which has a zero net flow for each frame in the stimulus presentation. Integrity of the motion processing neural architecture appears to influence the proportion of signal required to make these directional judgements in RDCs [6,14]. Therefore, patients exhibiting a high threshold for detecting the direction of signal dot movement are said to have a motion perception deficit. Like the study of patients with acute cerebellar lesions, motion perception was assessed with RDC stimuli ranging from 5 to 35% signal.

A static control screening task was designed to rule out simple visual, decision, or response problems that might have led these observers to problems in performing the motion perception task outlined above. Since many aspects of the motion and control task were the same, general problems influencing performance in the motion perception task can be excluded. To rule out problems with a brief presentation of stochastic information, the static control task used 15 randomly positioned, small (24×10 min arc) arrows. In each trial all of the arrows were oriented in the same cardinal direction. Observers made the same four AFC directional response used in the motion perception task. This ruled out decision and response problems as a cause of deficits in the motion perception task. Therefore, normal performance on this control task indicates that observers would not have difficulty with the motion perception task because of some more general visual, decision, or response deficiencies.

Observers viewed the stimuli from 57 cm while seated in a quiet, dimly lit testing room. Observers were required to fixate a small cross in the center of the computer monitor and initiated trials at their own pace only when they were confident of their fixation. To preclude shifts in fixation while the RDC was on the screen, each stimulus presentation lasted a brief 200 ms. Additionally, RDCs were presented unpredictably either in the center of the screen or 5° eccentric in one of the four quadrants. Equal numbers of trials were presented in each of the five different locations. Observers made either verbal or gestural responses which were entered into the computer by the experimenter.

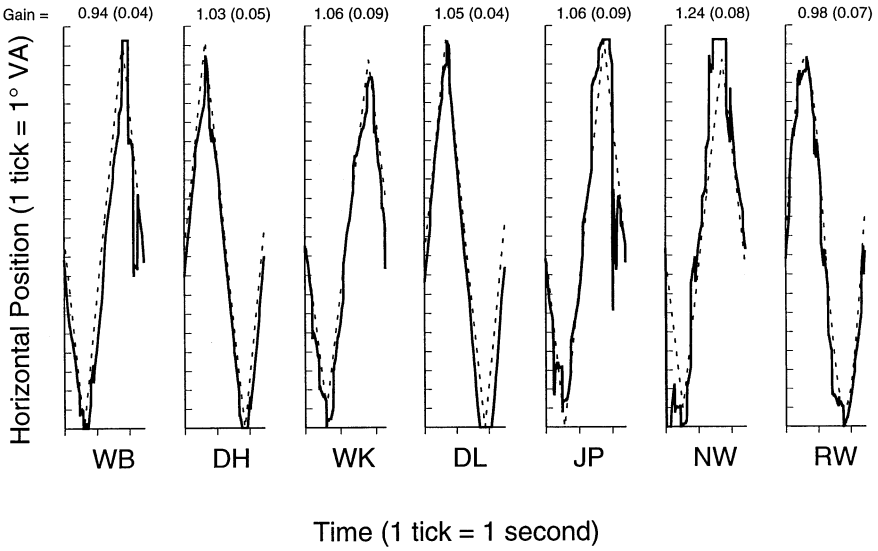

Fig. 3. Shown are representative left and right cycles of smooth pursuit eye-movement recordings for a set of patients with chronic cerebellar lesions. Horizontal eye position is shown on the vertical axis with each tick representing 1° of visual angle. Time is shown along the horizontal axis with each tick representing 1 s. Pursuit target path is shown with the bold dotted line. Patient eye position is shown with the thin solid line. Above each tracing is each patient's average (and S.D.) pursuit gain for a predictably moving target.

2.2. Observers

Ten of the 16 patients previously tested with acute lesions returned for testing between 9 and 39 months after their initial cerebellar stroke (mean = 25 months, S.D. = 12 months). None of these patients was known to have suffered additional strokes or new complications related to their previous stroke. Of these ten patients, two had lateralized lesions while eight had mid-line lesions. During the acute phase of stroke, the two patients with lateralized lesions and one patient with a midline lesion performed normally on these motion perception tasks. The remaining seven patients with midline lesions showed elevated thresholds on these tasks in the period shortly after their stroke [1].

We deliberately avoided testing these patients in the interim to prevent the possibility of improvement due solely to repetition of the task (although we have no evidence that this would occur). However, if this were the case, improvement due to practice effects would be indistinguishable from improvement due to recovery from the damage due to stroke. Additionally, it is important to note that the standard psychophysical procedures used here reduce the possibility of improvement due to behavioral compensation of any sort. Improvement would have to be due to recovered visual function.

Of course, vast improvements in general function were evident in these patients. For instance, all of these patients were brought into the laboratory by wheelchair for acute testing whereas all patients walked into the laboratory for chronic testing.

2.3. Eye movements

Eye movement deficits are a possible concern when patients have suffered cerebellar lesions [15,16]. Nystagmus or a failure to maintain stable fixation could influence performance in a patient with otherwise normal motion perception. To address this possibility we used a limbus tracking eye movement system (Skalar Iris; Delft, Netherlands) to assess the fixation and smooth pursuit ability of patients with cerebellar lesions. The fixation test required patients to simply fixate a small dot on a computer screen. The smooth pursuit condition had patients follow a moving dot as it moved back-and-forth across the computer monitor with either a triangular or sinusoid motion. After a brief calibration procedure, eye position was recorded at 200 Hz and recorded to disk for subsequent analysis.

3. Results

The threshold values, the proportion of signal dots needed to accurately determine (63% correct) the direction of signal dot movement, were calculated from the psychometric function by probit analysis for each observer, for each of the five spatial locations, for each of the two speeds. Again, since performance in all locations was very similar and we have no hypothesis for differences between locations, the performance across the five locations was averaged to yield a single quantitative threshold value.

Figs. 1 and 2 show both the acute and chronic motion thresholds of patients and the average threshold

(error bar = 2 S.D.) from a group of age matched normal controls ($n = 10$, normal mean age = 62 years, S.D. = 11 years, patient mean age = 59 years, S.D. = 11 years). At both the 11 and the 3.3 d/s speeds, the midline lesion and acute deficit patient group show higher thresholds than the two comparison groups. Patients with midline cerebellar lesions continue to perform poorly on a motion direction discrimination task well into the chronic phase of their stroke. Although most patients showed some improvement, their performance is much worse than both a matched normal control group and the group of patients showing no acute deficit from either lateral (DL and RS) or midline (JP) cerebellar lesions.

In contrast, performance on the static control task was perfect (100% correct) for all cerebellar patients. In the acute phase, the patient group generated an average of 94% correct. Problems performing the general psychophysical task with brief exposure to noisy stimuli do not account for these ongoing deficits.

To quantitatively address a possible eye-movement explanation for these deficits, we had the opportunity to record both smooth pursuit eye-movements and fixation in seven of these chronic patients. Even though smooth pursuit eye-movements were not part of the motion discrimination paradigm, Fig. 3 shows that these seven patients were capable of normal smooth pursuit with small fast moving targets ($> 15°$/s for triangle wave target). Shown above each tracing is the average pursuit gain (eye velocity/target velocity) for each patient calculated from eight excursions of the predictably moving target. Smooth pursuit gain in these patients very good compared to both normals and patients with visual tracking deficits resulting from cortical lesions (see Table 2 in Ref. [17]). Fig. 4 shows an unfiltered, 2 s record of each patient fixating a small spot, similar to the fixation spot in the motion detection paradigm. The recordings show that these patients could maintain stable fixation for the 200 ms required in the motion paradigm. These records indicate that abnormal smooth pursuit or abnormal fixation are not the underlying cause of the motion perception deficit. Instead, as we proposed earlier [1], the motion perception deficit might be a factor in the gain control problems occasionally seen when cerebellar patients pursue moving targets with their eyes.

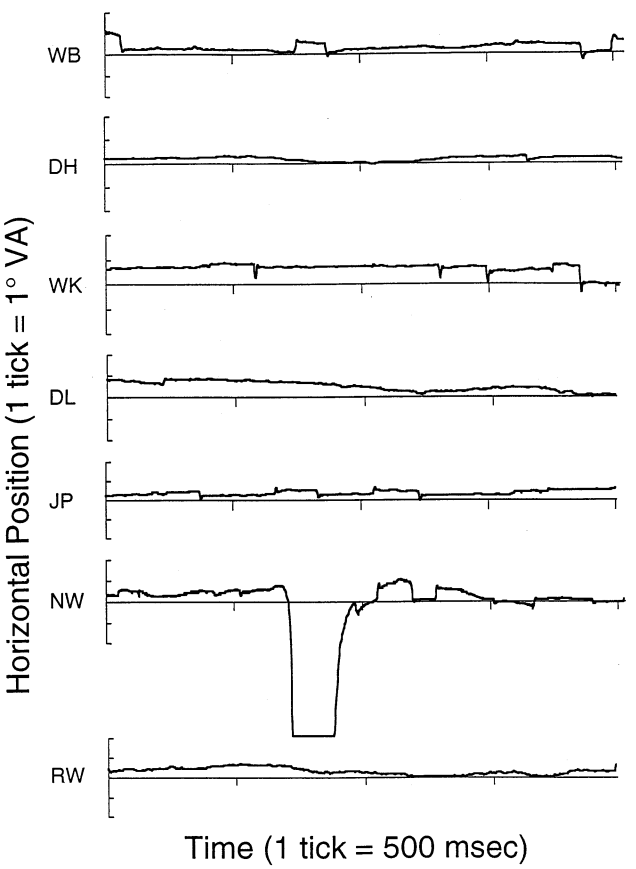

Fig. 4. Shown are representative fixation recordings for a set of patients with chronic cerebellar lesions. Like the previous figure, horizontal eye position is shown on the vertical axis with each tick representing 1° of visual angle. Time is shown along the horizontal axis with each tick representing 500 ms.

4. Discussion

Patients with acute mid-line cerebellar damage demonstrate a deficit in a motion discrimination task. The current study shows that these patients continue to have elevated motion perception thresholds well into the chronic epoch. While there is improvement, the limits of normal function are usually not attained even after two years of recovery. The stability of this deficit underscores the significance of the cerebellum in normal motion perception.

Of course, the precise role of the cerebellum in normal motion perception has yet to be established. Certainly, cortical regions are of primary significance for motion perception. However, clues to the cerebellar role may come from several areas: First, from neurophysiology we know that neurons in the cerebellar vermis can show selectivity for a particular direction and velocity of visual motion [10,12,18] and for binocular disparity [19]. Second, from neuroanatomy we know the cerebellum has broad thalamo-cortical connections that may subserve some visual function [20,21]. Third, from eye movement and vestibular research we know that the cerebellum is required for generating appropriate eye movements as the head is moved [22]. Therefore, the cerebellum appears well suited for the integration of information from many sources including visual motion and depth, vestibular, and eye movement information.

We previously proposed that the cerebellum might have a role in perceptual stabilization as head, body, or

eye movement cause movement of the visual scene across the retina [1]. Another related possibility that we are currently investigating is a cerebellar role in the perception of motion parallax. The perception of motion parallax requires the integration of relative visual movement, vestibular, and eye movement information. The cerebellum may have a role in the subcortical integration of these various sources of information before it is passed on to higher cortical areas. This latter possibility makes an even closer link between known cerebellar function and the evidence that the cerebellum has a role in normal motion perception. In addition, it provides an interesting paradigm to further study the motion perception deficit in patients with midline cerebellar lesions.

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