Roles of Proprioceptive Input in the Programming of Arm Trajectories

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It has been known for more than 100 years that loss or impairment of sensation in our limbs may produce severe disorders of movement and that sensory input plays a critical role in controlling movement. Indeed, the skin, muscles, and joints of our limbs are richly innervated by a variety of sensory receptors that convey proprioceptive information to all levels of the nervous system. What role this input plays in movement control has been a question of recurring interest but remains incompletely understood. In 1895, Mott and Sherrington demonstrated that surgical deafferentation of a monkey's limb produces severe disorders of movement and an unwillingness to use the limb in purposeful action. They therefore concluded that movement initiation requires the support of afferent information and proposed that coordinated movement results from the concatenation of reflex responses. Subsequently, however, it was established that deafferentation does not abolish the capacity to make purposeful movements and that motor performance may be substantially preserved when deafferentation is bilateral or when specific training procedures are used (Munk 1909; Knapp et al. 1963; Taub and Berman 1963; Polit and Bizzi 1979). This indicated that sensory input from the limbs is required neither to initiate movement nor to perform complex motor acts, as originally believed by Sherrington. Rather, the central nervous system makes use of motor programs to direct movements (Keele 1968). Nevertheless, the movements of deafferented monkeys have been repeatedly described as clumsy, inaccurate, and poorly coordinated (Munk 1909; Bossom 1974). Thus, although not necessary for the production of movement, sensory information, particularly from muscle receptors, clearly plays an important role in its control.

To examine the role of proprioceptive input in the control of limb movement, several investigators (Forget and Lamarre 1982, 1983, 1987; Rothwell et al. 1982; Sanes et al. 1985) have examined control of single-joint arm movements in patients with large-fiber sensory neuropathies. In this rare condition, there is degeneration of large afferent fibers, notably those conveying proprioceptive information, with little or no effect on motor fibers. These investigations have shown that such patients have major deficits in the feedback control of movement, i.e., in the ability to correct errors based on information from the moving limb. Thus, patients are neither able to maintain the limb in a fixed position nor to maintain their force at a constant level without visually monitoring the position of the limb or the force applied. They also cannot compensate for unexpected changes in loads encountered during the course of limb movements (Rothwell et al. 1982; Sanes et al. 1985). Errors are reduced, however, when patients are able to watch the limb during movement, so that visual feedback is apparently able to compensate, at least in part, for the loss of proprioceptive information. Lack of cutaneous feedback also readily explains the loss of dexterity in fine movements, such as buttoning clothes or grasping small objects, in which tactile cues are used to guide movement.

In contrast, the mechanisms involved in programming the trajectories of movements before they begin, a process referred to as feedforward control, have appeared largely normal in the deafferented patients of these studies. However, several considerations suggest that, in addition to deficits in feedback control, impairments of feedforward control might also contribute to the motor impairments that occur in these patients. First, Rothwell and colleagues (1982) reported that the learning of new and complex tasks, such as driving an unfamiliar automobile, was impaired in their deafferented patient. Second, the increased variability and lack of precision in the patterning of muscle contraction in isotonic (Forget and Lamarre 1983, 1987) and isometric tasks (Gordon et al. 1987) suggests an impairment in feedforward control. Third, the apparent absence of deficits in programming trajectories could have resulted from the relative simplicity of the processing required to specify the direction and extent of single-joint as opposed to multijoint movements (Flash 1987; Soechting 1989). The accurate performance of multijoint movements requires the nervous system to transform the spatial coordinates of the target, represented in extrinsic or retinotopic coordinates, into a complex set of commands specified in terms of an intrinsic coordinate system based on the controlled biomechanical variables of the limb (Atkeson 1989; Soechting 1989). In the case of even simple pointing and reaching movements, in which two or more limb segments must be rotated by multiple muscle groups acting in different ways, the task is biomechanically quite complicated. Therefore, it is reasonable to assume that specification of movement parameters requires precise information about the state of the limb prior to movement (Polit and Bizzi 1979; Hasan and Stuart 1988).

In the present studies, we used a multijoint reaching task in which subjects moved a hand-held cursor to different targets on a two-dimensional surface. Subjects did not view the cursor or the targets directly. Rather, target locations and cursor position were displayed on a computer monitor. This allowed us to control the visual information subjects obtained about the position of the arm and the results of their movements. In this paper, we first characterize the alterations in the trajectory of the hand that occur in the absence of proprioceptive input from the limb. We then present evidence that, without sensory input from the limbs, programs for reaching movements fail to compensate for anisotropies in the inertial properties of the arm. Finally, we examine how vision of the limb helps deafferented patients to improve their accuracy. We show that this improvement arises in large part by enabling a feedforward compensation for anisotropies in limb inertia. Some of the results have been reported previously in abstract form (Gordon et al. 1987; Ghez et al. 1988).

METHODS

Subjects. Subjects were four neurologically normal adults (three males and one female, aged 27–41) and three female patients (G.L., 39; M.A., 43; and M.B., 66). The patients had each developed severe large-fiber sensory neuropathies affecting both arms, the trunk, and, to a variable degree, both lower extremities. Onset was gradual, progressing over several months to 2 years. In the two younger patients, the etiology of the disease was unknown, and the disease had not progressed for several years. Both patients had complete loss of position sense in the hand and wrist and could only detect movements of the elbow and shoulders at the extremes of range. Temperature and pain sensation were preserved. Deep tendon reflexes were absent in all muscles of the upper extremities, but muscle strength was normal on clinical examination. The restriction of the neuropathy to large sensory fibers was confirmed by somatosensory evoked potentials, electromyography and nerve conduction studies, and, in one patient, by nerve and muscle biopsy. These two younger patients were intensively studied over a long series of testing sessions, and they became highly practiced in the tasks we asked them to perform. It should also be noted that, although muscle strength was normal, both patients were severely disabled and were unable to successfully perform many activities of daily living, such as dressing, drinking from a cup, or writing. One of the patients was confined to a wheelchair because of her difficulty in balancing. The other, although unsteady, was able to walk independently on a wide base. In the third, older patient we examined, the sensory neuropathy was secondary to a carcinoma of the lung. In this patient, sensory deficits were less severe at the shoulder and elbow. Nevertheless, this patient was also severely impaired in functional activities, and her deficits in our experiments were qual-

itatively similar to those of the other two. We were only able to examine this patient in one session.

Apparatus and tasks. Subjects were seated facing the screen of a computer (17 cm × 12 cm, Macintosh SE, Apple Computer) and moved a hand-held cursor on a digitizing tablet (42 cm × 30 cm, Numonics Corp.) with their dominant hand. The position of the cursor on the tablet (x and y coordinates) was sampled by the computer at 200 Hz and displayed on the computer screen as a cursor with the shape of a crosshair. The ratio of cursor movement on the tablet to cursor movement on the screen was approximately 2.4 to 1. In most experiments, the tablet was positioned at waist level, so that the upper arm was approximately vertical and the elbow was flexed at about 90°. In some experiments, to facilitate theoretical analysis and computer simulations, the tablet was positioned at shoulder level so that the entire arm moved in a horizontal plane. In this position, the subject's upper arm was supported by a sling and cable hanging from the ceiling. In all experiments described here, the tablet was directly in front of the subject, so that its center was aligned with the midsagittal plane of the subject.

In this paper, we present results from a reaching task, in which subjects were required to make a quick and accurate movement of the cursor from one point to another on the tablet without visual feedback. At the start of a trial, two small circles were displayed on the computer screen, a start circle and a target circle. During the initial alignment phase, subjects monitored their movements on the screen as movement of a crosshair cursor, and they used this feedback to position the cursor in the center of the start circle on the screen. After alignment was achieved, a "go" tone was presented; subjects were told to then move "when ready" and to make a "single, quick, and uncorrected movement" to attempt to reach the target circle. In most experiments, the screen cursor was blanked at the time of presentation of the tone, so that visual information could not be used to correct the movement trajectory. At the end of the movement, the trajectory was displayed to the subject to provide knowledge of results. Subjects were encouraged to try to be as accurate as possible and were provided with a running score of their performance. Targets were typically presented in a variety of locations (from 9 to 24 in different experiments), requiring movements in different directions and of different extents. The order of target presentation was varied in random fashion, and no target was presented twice in succession, to prevent subjects from progressively refining a stereotyped movement strategy for a specific target. In most experiments, vision of the hand and arm was blocked by the combination of a drape attached around the neck and a two-way mirror covering the hand. In some experiments, described below, vision of the hand was allowed either before trials or during trials, by illumination of the hand from underneath the mirror. In addition, in some experiments, subjects were allowed to visually monitor the

screen cursor during movement. These manipulations allowed us to assess the roles of different types of visual information in achieving accurate control of movement trajectories.

RESULTS

Deafferented Patients Make Increased Errors in Trajectory and in End Position

In general, subjects with normal sensation and motor control had little difficulty in producing relatively accurate movements to different targets without visual feedback, and the trajectories of their movements were stereotyped in form. Figure 1 shows typical trajectories of one control subject to nine different targets (three directions and three amplitudes). Movements are straight and terminate close to the required target. The precision with which direction is specified is evident from the overlap of paths to targets in the same direction. Since the direction of movement is specified from the very beginning of the trajectory, it must be largely preplanned and thus controlled by a motor program. In addition, movements are terminated cleanly with a stable end position. As illustrated for one patient in Figure 1, however, movements made by deafferented subjects were highly variable and inaccurate. Trajectories in this figure can be seen to be curved and frequently misdirected at their onset. Terminal endpoints are also unstable: Secondary movements and drifts often occur at the termination of the movements. It should be noted that the patients were typically unaware of such drifts and believed that their hand had come to a stable resting position.

Movement accuracy was assessed by characterizing the spatial distributions of endpoints at each of the target locations, as shown in Figure 2A for the control subject and deafferented patient of Figure 1. Movement endpoints here were defined as the point at which velocity reached zero or at which the movement sharply changed direction; thus, terminal drifts were not included. In this figure, each endpoint distribution is surrounded by a contour whose shape was computed using principal-components analysis and a fitting procedure based on the quartiles in the principal and orthogonal axes of the two-dimensional distribution. The fitted contours closely surround most of the points in each distribution and allow assessment of the shapes and sizes of the distributions. In the control subject, endpoint distributions have a characteristic elliptical shape with the long axis aligned close to the average direction of movement (defined using the center of the distribution). Thus, errors in direction are considerably smaller than errors in extent. The deafferented patient, however, shows markedly larger endpoint dispersions, with distributions that are more circular in shape or whose long axis is unrelated to the average direction of movement. The other deafferented patients showed similar distributions. Thus, the variable errors are larger in the patients than in controls. Moreover, errors in direction appear increased disproportionately to errors in extent.

To assess this trend quantitatively across subjects, we measured separately the "on-axis" errors (deviation of each endpoint from the average endpoint along the axis of the average movement direction) and "off-axis" errors (deviation from average endpoint along an axis perpendicular to average movement direction). The ratio of off-axis to on-axis errors then provides a measure of the shape of the endpoint distributions. In control subjects, this ratio varied from 0.3 to 0.4, indicating that the lengths of the endpoint distributions were on average 2.5–3 times larger than the widths. In deafferented subjects, the ratios ranged from 0.6 to 1.2, indicating that their endpoint distributions were more circular. The overall size of the distributions was measured as the average deviation of each endpoint from the average endpoint. Deafferented subjects showed an average deviation that was 2.4 times larger than the average deviations of controls (mean deviation for controls = 1.18 cm, mean for patients = 2.83 cm).

Average velocity profiles for responses to the 30° targets are shown in Figure 2B (direction angle is defined as increasing counterclockwise with 0° at the 3
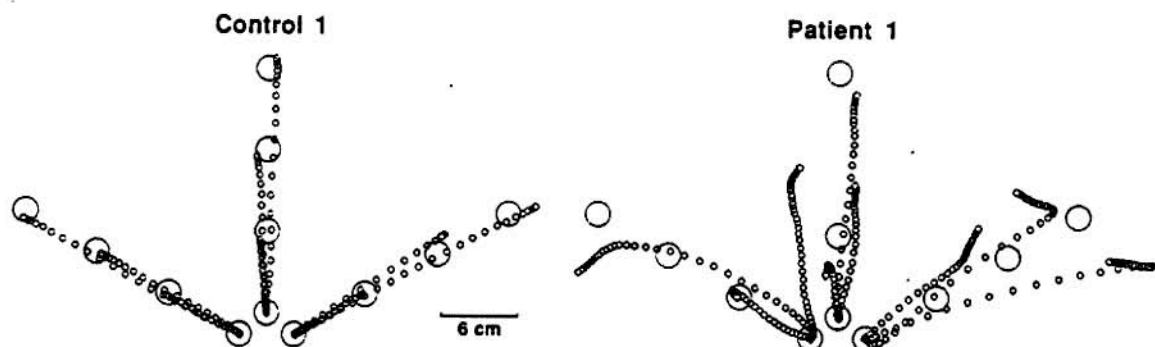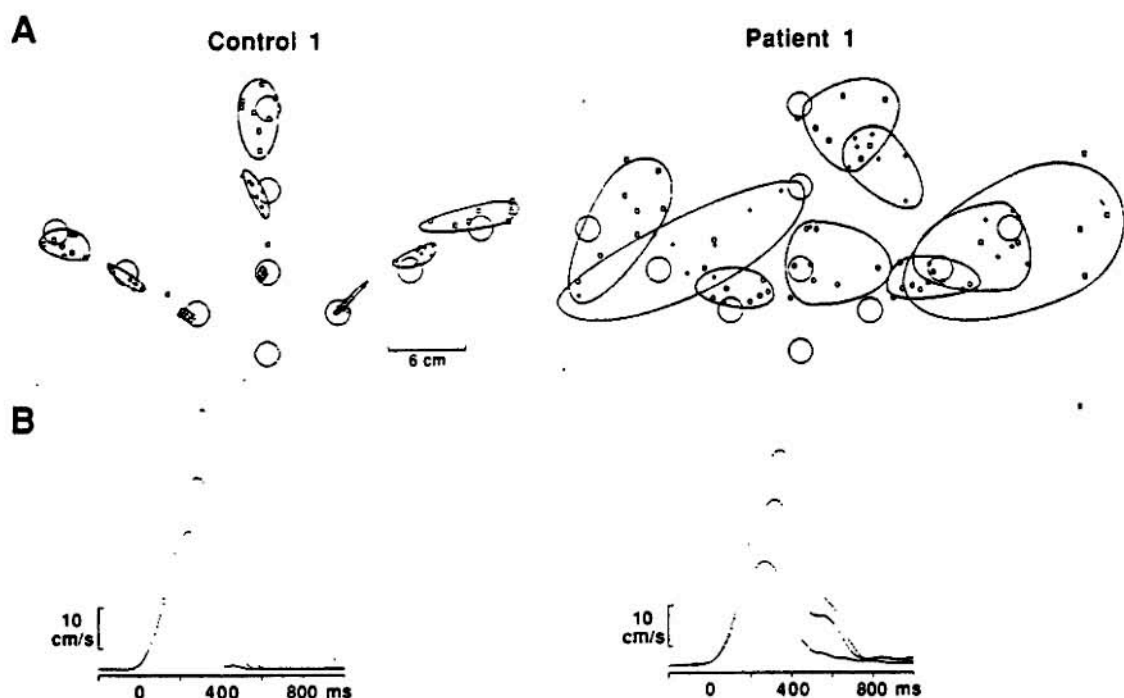
Control 1   **Patient 1**

Figure 1. Selected movement paths to each of nine targets made by a control subject (*left*) and a patient (*right*). Each path is plotted with small circles that correspond to x-y coordinates in bins spaced 20 msec apart. The large circles indicate starting points and targets. The starting points for movements in each of the three directions are superimposed. Actual starting points for movements in the three directions had a different spatial relationship to each other than that shown.

A

Control 1   Patient 1

6 cm

B

10
cm/s

0   400   800 ms

10
cm/s

0   400   800 ms

Figure 2. Endpoint distributions (*A*) and average tangential velocities (*B*) of movements made by a control subject (*left*) and a patient (*right*). (*A*) Endpoint distributions show terminations of movements made to nine different targets in three directions. Terminations are measured as locations where hand velocity reached zero or reversed in direction. Endpoints of movements to targets requiring smallest movement extent are designated by circles. medium extents as diamonds. and largest extents as squares. The distribution of endpoints to each target is surrounded by a contour whose orientation is computed by the method of principal components and whose size and shape are based on the interquartile range in each of the major axes. (*B*) Average tangential velocities of responses to the three targets in the 150° direction (requiring movements diagonally to the left). Each trace is the average of eight movements aligned on movement onset.

o'clock position). In general. both control subjects and deafferented patients showed bell-shaped velocity profiles. with increases in peak velocity and peak acceleration as greater distances are reached. These characteristically shaped velocity profiles are typically seen in relatively fast movements (Abend et al. 1982: Hollerbach and Flash 1982) and are taken to indicate that the extent of movement is programmed in advance (Brooks 1974: Ghez 1979).

In both the control subjects and the patients. peak velocities showed systematic differences for movements made in the three directions: responses made to the 30° target consistently reached higher peak velocities than the responses to the 90° and 150° targets (not shown). In parallel with these directional differences in trajectories. directionally dependent errors are evident in the endpoint distributions shown in Figure 2A. In both control subjects and patients, there are systematic errors in direction and extent that depend on the target direction: For example. movements made to the 30° target are typically hypermetric, whereas movements made to the 150° target are hypometric. Moreover. there are errors in direction that are consistent for each of the different target directions. This systematic dependence of extent and direction errors on target direction was observed consistently in all subjects. However.

although these errors were small in controls, they were frequently large in the patients.

Increased Systematic Errors in Deafferented Patients Result from Failure to Compensate for Mechanical Anisotropy

It is known from the work of Hogan and other investigators (Hogan 1985: Mussa-Ivaldi et al. 1985) that the inertial load at the hand differs for movements in different directions. With the arm oriented vertically, the 30° movement is largely carried out by rotating around the long axis of the humerus and thus involves principally the inertia of the forearm, whereas for movements in the 150° direction, the inertias of both upper arm and forearm resist movement. We therefore hypothesized that systematic differences between movements made in different directions could have resulted from a failure to program joint torques in accord with differences in inertia.

We analyzed the effect of the intended direction of movement on trajectory variables and on errors by presenting subjects with targets located in 24 different directions spaced 15° apart, all requiring a movement of the same extent (7.5 cm) from a central starting point. To simplify the mechanical conditions and the compu-

tations of the effects of inertia, the subject's arm was now placed in a horizontal plane (see Methods), restricting movement to 2 degrees of freedom (one at the shoulder and one at the elbow).

If subjects fail to compensate for direction-dependent variations in limb inertia, the initial acceleration of the movements should show a systematic dependence on direction. Figure 3 shows vector plots of the average peak accelerations and average movement extents for each of the 24 target directions. Responses of a control subject and of a patient are shown. Three important findings are illustrated in these plots. First, in both controls and patients, the magnitude of peak acceleration varies in an orderly way for movements in different directions. Peak accelerations are highest along an axis oriented at approximately 60°–240°, and they are lowest along the axis perpendicular to this (see dashed line in upper left of Fig. 3: Note that the axis of least inertia varies according to the exact position of the subject's arm). Second, in the patient, this systematic variation in acceleration is carried over into the final movement extents, which are greatest in the same axis in which peak accelerations are largest. The control subject, in contrast, shows only a slight residual effect of the acceleration anisotropy. Third, there are systematic directional biases that also depend on target direction.

The peak accelerations for movements in different directions thus appear to vary inversely with limb inertia. As pointed out by Hogan (1985), the variation in "apparent mass" of the hand takes the form of an ellipse, whose long axis is oriented very close to the long axis of the forearm. This is shown for the conditions of this experiment in Figure 4A: When the subject moves to a target in the 60°–240° axis, the movement is largely carried out by moving the forearm. Relatively little movement of the upper arm is required. This means that the inertial resistance to movement is relatively low in this direction compared to the orthogonal direction, in which the inertias of both upper arm and forearm resist movement.

To gain further insight into whether variations in peak acceleration derive from anisotropy in inertial resistance, we used standard equations for the apparent mass of a two-link manipulator (Hogan 1985; Mussa-Ivaldi et al. 1985) to compute the initial acceleration vectors that would result from a constant impulse of force applied in each of the 24 directions from the initial position of the subject's arm. The predicted peak acceleration vectors in the different directions form an ellipse whose long axis is approximately perpendicular to the long axis of the forearm (Fig. 4B). This vector plot is similar in shape and orientation to the acceleration vector plots of both the control and the patient shown in Figure 3. When these simulated peak accelerations are plotted as a function of initial movement

Figure 4. Anisotropy of the arm's inertial field. (A) Position of the subject in horizontal arm task. Superimposed ellipse shows calculated inertial field, which resists movements of the hand in different directions. Top part of figure shows average accelerations of a control subject for movements in the axis of highest inertia (left) and least inertia (right). (B) Calculated acceleration vectors for movements with a constant force applied at the hand in each of 24 equally spaced target directions. Compare to actual acceleration vectors in Fig. 3. (C) Calculated peak accelerations plotted as a function of initial movement direction. Compare to plots in Fig. 5.
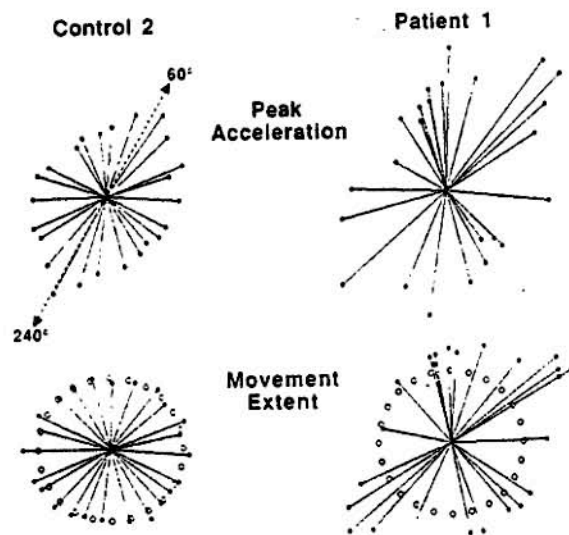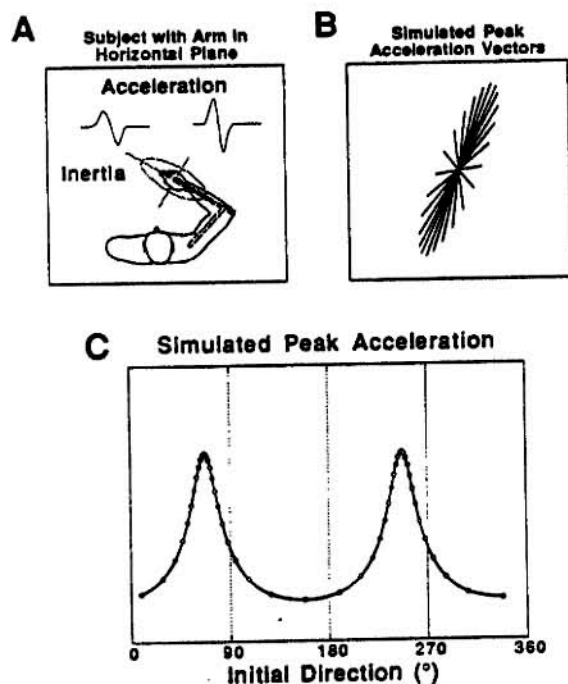

Figure 3. Vector plots of average peak acceleration (top) and average movement extent (bottom) of a control subject (left) and a patient (right). Each line represents the average of six trials aimed to a single target. Subjects were given 24 targets separated by 15° intervals (targets are shown as small circles in plots of movement extent). The acceleration vectors for each subject are scaled so that the average vector is the same length as the average extent vectors for that subject. The dashed line in the upper left plot indicates the approximate axis of least inertia.

direction (Fig. 4C), the plot shows two relatively sharp maxima and two broad minima. The peaks occur for directions in which inertia is minimal.

Figure 5 presents the measured peak accelerations by target direction and a line fitted to this distribution for the control subject and the patient whose responses were illustrated in Figure 3. As shown here, both control subjects and patients exhibit variations in peak acceleration that are quite similar in shape to the predicted values, with sharp maxima and broad minima close to those of the simulation. Therefore, peak acceleration varies inversely with the inertial resistance to

movement. This indicates that the initial net forces moving the hand, or, equivalently, the programmed joint torques, do not fully compensate for the inertial anisotropy of the limb.

Nevertheless, the control subjects largely compensated for the inertial anisotropy by the end of the movement by modulating movement time. The middle row of Figure 5 shows movement extent as a function of direction in a control subject and a patient, whereas the bottom row shows movement time, also as a function of direction. In the control subject, the plot of movement time is almost a mirror image of the peak acceleration
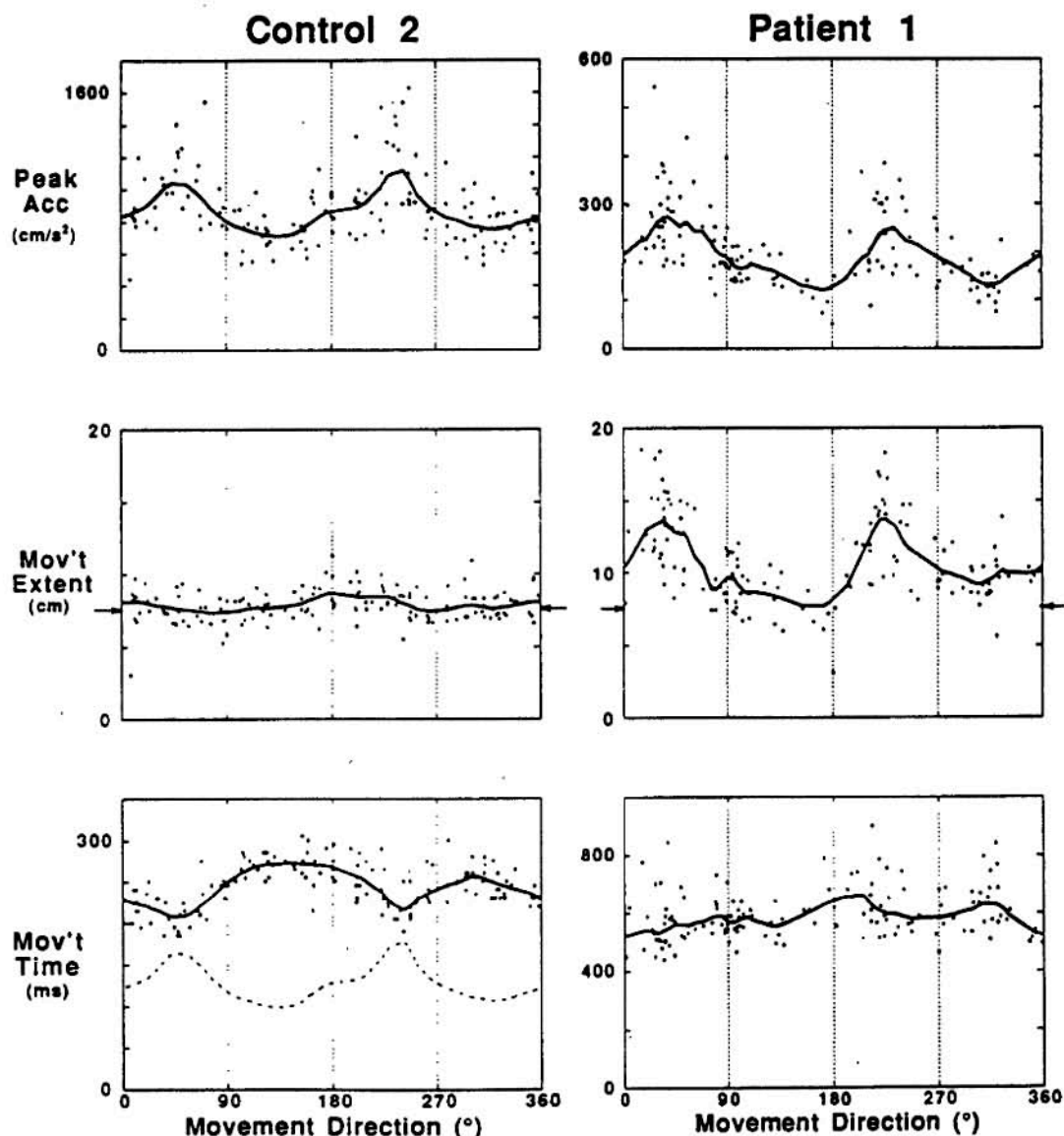
Figure 5. Directional anisotropies in a control subject (*left*) and a deafferented patient (*right*). All plots show movement direction on the horizontal axis. In top row, peak acceleration is plotted; in middle row, movement extent; and in bottom row, movement time is plotted. In all plots, a procedure called LOWESS (locally weighted scatterplot smoother) is used to fit a line to the scatterplots (Cleveland 1979). This procedure averages the data in local regions and gives less weight to outlying data points. The arrows on the sides of the movement extent plots in the middle row show target extent. The dashed line in the movement time plot for the control subject shows the best fitting line for peak acceleration (from the top row) to allow comparison.

plot. Thus, the control subject compensates for high peak accelerations by truncating movement time and for low accelerations by prolonging movement time. In contrast, the patient does not show this compensation, and there is relatively little variation in movement time as a function of direction. As a result, the variation in movement extent parallels the variation in peak acceleration.

In addition to variations in peak acceleration, the elliptical form of the arm's inertial field also results in a disparity between the direction of application of force and the direction of the resulting acceleration. As can be seen in the simulated acceleration vector plot (Fig. 4B), even though unit forces are applied in 24 equally spaced directions, the directions of the accelerations are not distributed uniformly. There is a higher concentration of accelerations directed toward the long axis of the ellipse, that is, the direction in which inertia is least. Figure 6 shows that in the patient this mechanical anisotropy leads to marked nonuniformities in the distribution of the directions of movement paths. Whereas the control subject shows relatively evenly spread
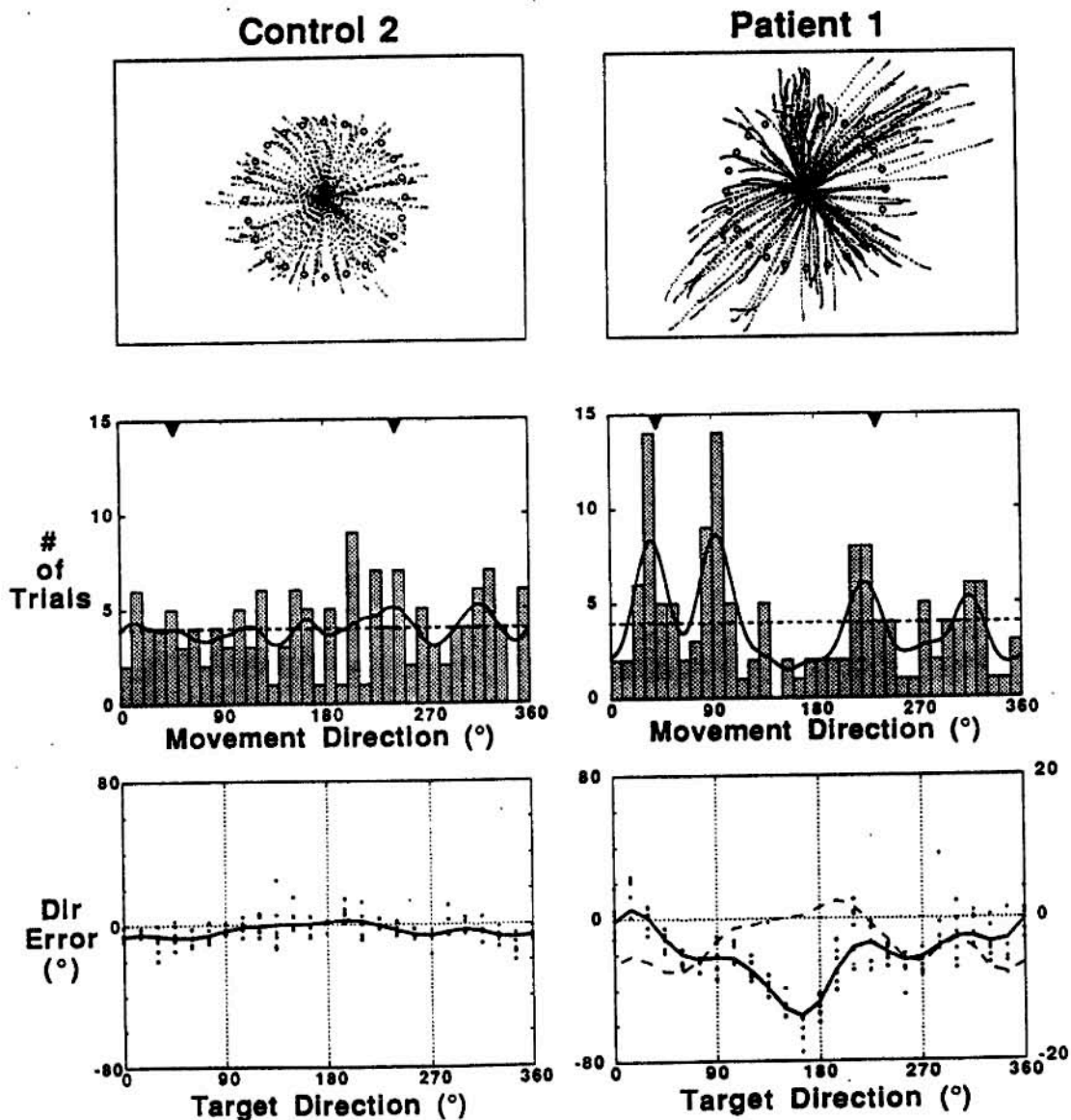

Figure 6. Biases in movement direction in a control subject (*left*) and a deafferented patient (*right*). Top plots show movement paths made to 24 targets spaced 15° apart (small circles indicate target locations). Each path is plotted as a series of dots; the dots represent locations of the hand every 5 msec. Drifts that occur after termination of movement are not shown. Middle plots show histograms of movement direction with local density superimposed. The dashed lines in these plots show expected density for a uniform distribution. Bottom plots show directional error plotted as a function of target direction. LOWESS is used to fit a line to the scatterplots (see Fig. 5). The dashed line in the directional error plot for the patient shows the best fitting line for the control subject (from the plot at left) displayed on a higher scale (see axis labels on right side of plot) to allow comparison.

paths. with only small gaps. the patient shows high densities of paths in some directions and large gaps in other directions. These nonuniformities become more apparent when movement directions are represented as histograms (with local density superimposed). as in the middle row of Figure 6. In this figure. it is apparent that the patient shows particularly high concentrations of movements in the directions in which peak acceleration is highest (indicated by triangles at top of histograms) as well as local concentrations in the directions approximately orthogonal to these. Both patients examined in this task showed qualitatively similar patterns of error. The bottom row of Figure 6 shows the directional. errors that are associated with these nonuniformities in the distribution of movement directions. Although the patient makes much larger errors on average than the normal. these errors do not simply result from greater variability in response direction. Rather, the systematic error is much greater in the patient. whereas the variable error is only modestly increased. Therefore, the deafferented patients make exaggerated errors in both extent and direction that are consistent with anisotropies in the inertial field. This suggests that the patients do not adapt the amplitudes and directions of applied forces to the actual mechanical properties of their limbs.

Visual Monitoring of the Limb Reduces Error by Improving Feedforward Control

It is well known that deafferented patients are able to reach for objects a great deal more accurately if they can see their hands. One explanation is that vision provides deafferented patients with feedback information to correct errors that develop in the course of movement. Thus. the difference between the position of the hand and the target could serve as an error signal for the correction of movement trajectory. An alternative explanation (but one that need not exclude the first) is that vision of the arm might improve performance by providing feedforward information to improve the programming of movement itself. Perhaps by updating an internal model of the limb, such information could enable the patient to more accurately specify the commands needed to move his limbs with the appropriate direction and extent. In this section, we analyze the extent to which vision contributes to both feedback and feedforward control, by providing subjects with different forms of visual information.

We first analyzed the effect of two forms of visual information. In one condition (WithFB). subjects were allowed to monitor the cursor on the screen during the movement. In another condition (PreVision). subjects
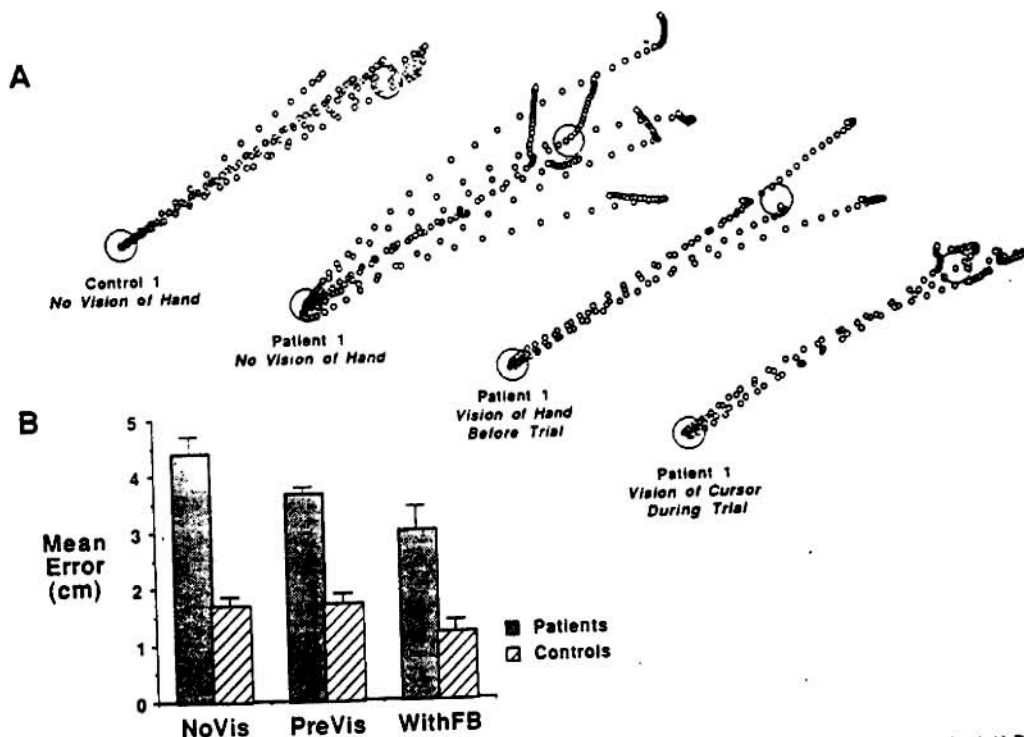

Figure 7. Effect of prior vision of the hand and vision of the cursor on errors in deafferented patients and controls. (A) Paths of all movements aimed to a single target are plotted for control subject with no vision ($n = 8$). deafferented patient with no vision ($n = 8$). patient with prior vision of hand ($n = 4$), and patient with vision of cursor on screen ($n = 4$). Each path is plotted as a series of small circles: the circles represent locations of the hand every 20 msec. The large circles show starting points and targets. (B) Mean error and standard error of the mean (distance from target to termination of movement) are shown by condition for patients (shaded bars. $n = 3$) and control subjects (hatched bars. $n = 3$).

were allowed to see their arms for a few seconds before each trial. We compared movements made under these two conditions with those in the standard condition of no vision of the arm or cursor (NoVision). For this experiment, we presented targets in three directions with three amplitudes in each direction. Figure 7A shows, as an example, movement paths to one target for a control subject in the NoVision condition and for a patient in all three conditions. PreVision substantially reduces error, primarily by decreasing the variability of the initial direction. Cursor feedback (WithFB) reduces error at both the initiation and the termination of movement. Figure 7B shows the average errors made by three control subjects and three patients in each of these conditions. In the control subjects, prior vision of the arm had little effect on accuracy, whereas cursor feedback produced modest but significant decreases in error. In the patients, on the other hand, both PreVision and cursor feedback led to significant decreases. In fact, in two of the three patients, PreVision was as potent in reducing error as cursor feedback.

These findings indicate that prior vision of the limb alone allows the deafferented patients to improve accuracy through a feedforward mechanism, that is, by improving the initial programming of the movement. This feedforward mechanism was further analyzed in two subjects by presenting them with 24 targets arranged, as before, in a circular array around a common origin. In addition to the blocks of no-vision trials, the subjects were now given blocks in which on alternate trials vision of the hand and arm was either allowed or prevented. In the trials with vision, the patients were able to see their arms in motion. However, since no two successive targets were the same, the visual information from these trials could not be directly used to program the following movements, which were made without vision. Any information gained on the trials with vision could only serve to improve the general rules used to plan subsequent movements or to calibrate an internal model of the limb on which such rules might be based.

The results of this experiment are shown for one patient in Figure 8. The three columns show perform-
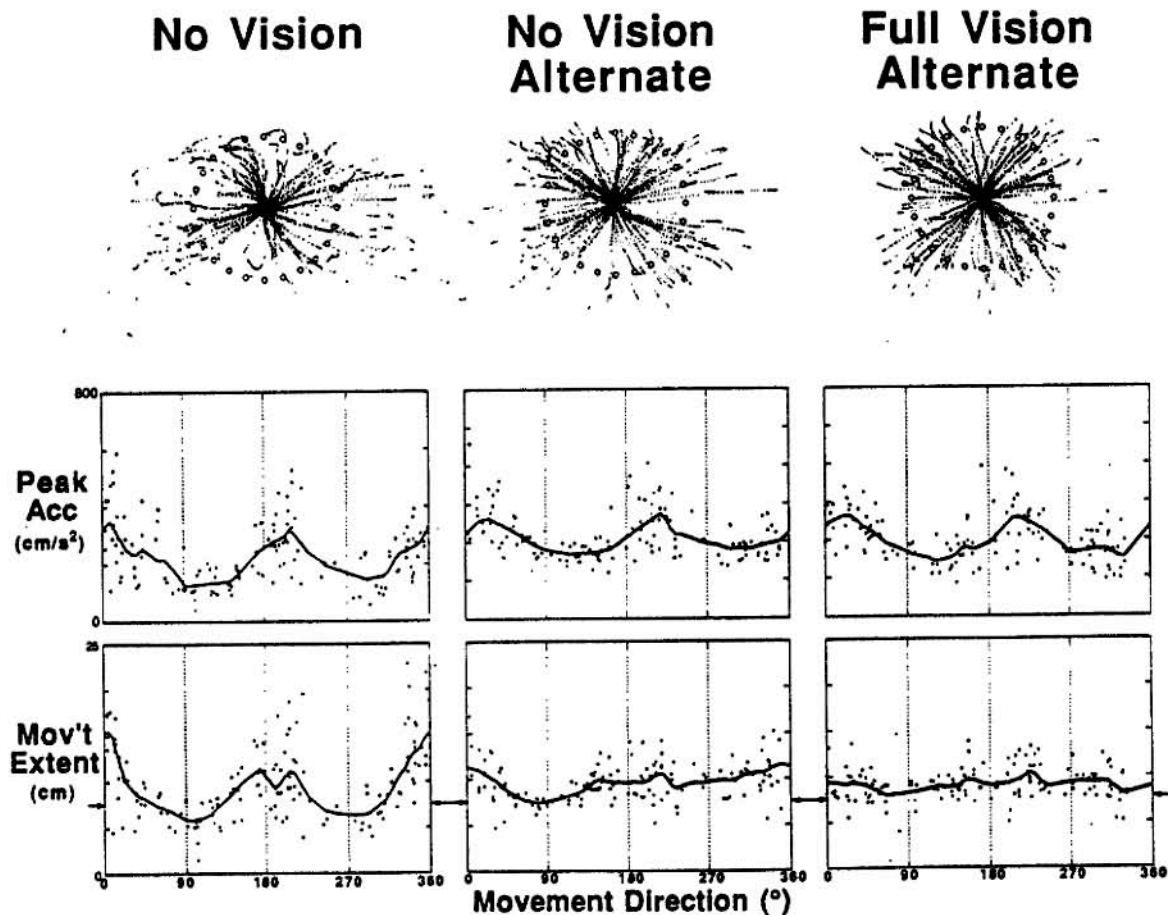
Figure 8. Responses of a deafferented patient (Patient 2) to 24 targets under three conditions: no vision (*left*), no vision alternate (*middle*), and full vision alternate (*right*). No-vision trials were run in separate blocks. No vision alternate and full vision alternate were run in the same blocks; trials with vision of the hand were followed by trials with no vision. Top row shows plots of movement paths (see Fig. 6 for explanation). Middle row shows plots of peak acceleration; bottom row shows plots of movement extent (see Fig. 6 for explanation).

ance in each of the three conditions: no vision, alternate no vision (trials with no vision that followed trials with vision of the arm), and alternate full vision (trials on which vision of the hand was allowed). The top row of plots shows the variation in acceleration as a function of movement direction. The middle row shows the variation in movement extent. The bottom row shows the movement paths to the 24 targets. The two groups of responses made without vision of the arm differ dramatically. In the blocks where the subject could never see her arm, there is again a failure of compensation for inertial anisotropy with fluctuations in movement extent that parallel the fluctuations in peak acceleration. In contrast, in the blocks of trials in which the subject had no vision of the arm but had previously viewed her arm during movement, the modulation in movement extent in different directions is considerably reduced. Finally, it can be seen that vision of the arm during movement provides only modest improvement over vision of the arm on the preceding movement. The other patient showed similar but less dramatic results. We therefore conclude that the major improvement in performance provided by vision during reaching is not, as might otherwise have been suspected, due to the availability of feedback signals. Instead, it results from improvement in the subject's ability to correctly program the forces needed to compensate for the variations in the inertia of the limb.

DISCUSSION

Our studies of reaching movements in normal and deafferented subjects lead to two main conclusions. First, information from the limb is necessary both to accurately program movement trajectory and to specify the patterns of muscle contraction necessary for the subsequent maintenance of posture. Because of the absence of proprioceptive input, deafferented patients are unable to compensate for the inherently non-uniform inertial properties of their limbs, as normals do, and they cannot maintain a steady position of the hand at the end position. Second, visual monitoring of the arm can partially substitute for deficient proprioceptive information. Although allowing for some feedback correction, vision of the limb acts principally by enabling patients to more accurately specify critical parameters of the motor programs to guide subsequent movements. This conclusion is based on the finding that prior vision of the limb, either in a static condition or while it is moving, is as effective as concurrent vision of the cursor in improving accuracy.

The fact that, under normal conditions, trajectories are straight and fairly accurate from their onset indicates that the direction of movement is planned to a substantial degree before the movement begins. Similarly, the normal scaling of initial acceleration to the distance to be traveled shows that movement distance is preprogrammed as well. The large errors in direction and extent of movement made by deafferented patients indicate that, contrary to conclusions derived from studies of single-joint movement, the programming of multijoint movement is critically dependent on proprioceptive information from the limb.

Although the large errors made by deafferented patients appear initially random, analysis of the initial kinematic features of movements shows that these errors are systematic and reflect anisotropy in the inertial field of the arm. A surprising finding of these studies was that anisotropy in limb inertia also influences the initial portions of the trajectories made by normal subjects. The systematic variations we find in peak acceleration with movement direction indicate that the motor program that specifies the trajectory of the limb does not take full account of the inertial anisotropy of the limb. A dramatic finding in the present study was that deafferented patients show severe directional biases that lead to gaps in the distributions of their movement paths. They appear as "motor scotomas," representing areas of the workspace that were rarely entered. Our preliminary evidence is that inertial anisotropy also contributes to these directional biases; however, their mechanism may be more complex.

Vision of the hand and arm at rest prior to movement or vision of the limb's motion in response to the subject's prior voluntary commands can substantially correct deficits in movement programming in deafferented patients. This intermittent information could only have acted through feedforward mechanisms, and not through feedback. These observations suggest that a fundamental role of proprioceptive information from the limb is to allow subjects to program the forces they exert in accord with the complex mechanical properties of their limbs. In deafferented subjects, knowledge of the current state and properties of the limb obtained through vision apparently can substitute for deficient somatosensory input and enable partial compensation for inertial anisotropy.

We propose that proprioceptive input is used by the central nervous system to update an internal representation, or model, of the mechanical properties of the limb. We hypothesize that such a model is used to specify both the general kinematic plan and the adjustments that are necessary to adapt this plan for movements in different directions. When somatosensory information is reduced or absent, as in our patients, the internal representation of the limbs is unstable and degrades with time, forcing patients to rely on visual input. This emphasizes the importance of precise and current information about the state of the limb in the control of movement. The sensory receptors in our muscles and joints, especially the spindles and tendon organs, appear well adapted to provide this information, for example, to enable the brain to take into account the varying inertial loads for movements in different directions. Finally, since the neural processes involved in using this state information to plan movements are distributed throughout many centers of the nervous system, it is likely that the deficits we have encountered in deafferented patients will have analogs in the deficits of patients with central lesions.

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