



# Reaching with cerebral tunnel vision

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(Received 12 September 1995; accepted 7 May 1996)

**Abstract**—We studied reaching movements in a 48-year-old man with bilateral lesions of the calcarine cortex which spared the foveal representation and caused severe tunnel vision. Three-dimensional (3D) reconstruction of brain MR images showed no evidence of damage beyond area 18. The patient could not see his hand during reaching movements, providing a unique opportunity to test the role of peripheral visual cues in limb control. Optoelectronic recordings of upper limb movements showed normal hand paths and trajectories to fixated extrinsic targets. There was no slowing, tremor, or ataxia. Self-bound movements were also preserved. Analyses of limb orientation at the endpoints of reaches showed that the patient could transform an extrinsic target's visual coordinates to an appropriate upper limb configuration for target acquisition. There was no disadvantage created by blocking the view of the reaching arm. Moreover, the patient could not locate targets presented in the hemianopic fields by pointing. Thus, residual nonconscious vision or 'blindsight' in the aberrant fields was not a factor in our patient's reaching performance. The findings in this study show that peripheral visual cues on the position and velocity of the moving limb are not critical to the control of goal directed reaches, at least not until the hand is close to target. Other cues such as kinesthetic feedback can suffice. It also appears that the visuomotor transformations for reaching do not take place before area 19 in humans. Copyright © 1996 Elsevier Science Ltd.

**Key Words:** visual cortex; motor control; optic ataxia; sensorimotor transformations.

## Introduction

Reaching and grasping external objects is a fundamental human activity that demands the coordination of several different nervous system functions. To accomplish this task, the brain transforms a target's visual coordinates to body-centered space, plans a hand path and trajectory (the sequence of hand positions and velocities to target), and computes multiple joint torques, especially around the shoulder and elbow. It also specifies the necessary limb segment orientations from among many possibilities, and activates appropriate muscle groups and inhibits others to meet those specifications. The sensory feedback, frames of reference and neural mechanisms used to solve these complex motor control problems are active research topics [5, 18, 19, 21, 37].

The initial direction of a reach is thought to depend on firing of populations of directionally tuned movement neurons [13]. Such neurons have been identified in a network of interconnected structures including the motor cortex, premotor cortex, basal ganglia, cerebellar cortex

and deep nuclei, and area 5 of parietal cortex in primates [12, 13]. Even so, the initial command mediated by these neurons may be inaccurate or else a target may have moved. Consequently, once a movement has been initiated, corrections may be needed. Visual feedback, conscious or not, on the position of the reaching limb is one mechanism of potential importance in such correction. Kinesthetic feedback and monitoring of the efferent motor command are others.

The eye has the possibility of updating the brain on the arm's progress throughout a reach. The shoulder, which is the theoretical origin of a visual coordinate system for locating extrinsic objects in 3D space [36, 38] is often out of sight, but the hand can usually be seen with peripheral vision throughout much of a reaching movement. Visual cues on the position and velocity of the limb and its individual segments are increasingly available as the hand crosses the panorama toward a foveated target. Of what use are these visual cues to limb control and when do they come into play? Studies in subjects with acquired constriction of the visual fields (i.e. 'tunnel vision' or 'keyhole vision') can provide relevant details [35] to address these important motor control questions.

Here, we report on the perceptual, cognitive and reaching abilities in a subject with severe tunnel vision caused

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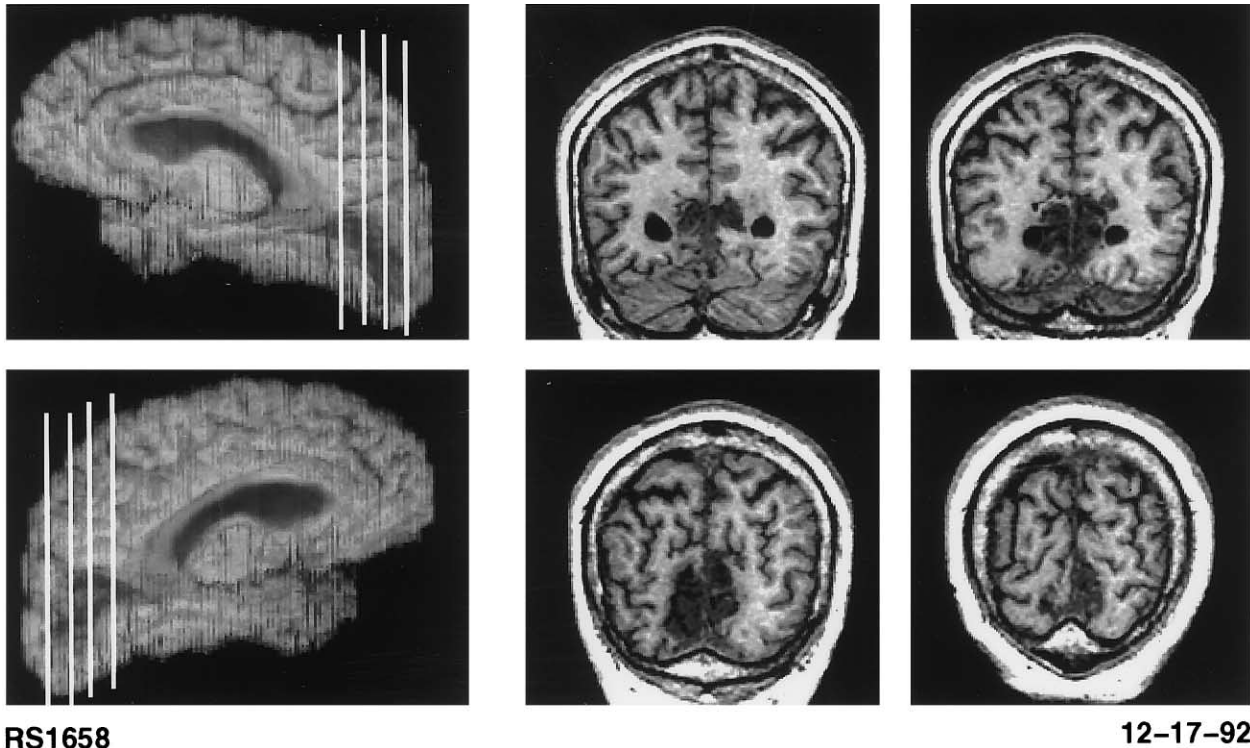


Fig. 1. A T1-weighted MR scan showing the occipital lobe in four coronal sections (the two middle and two right panels). The locations of these coronal sections are depicted on 3D-MR reconstructions of the mesial surface of the right hemisphere (top left panel) and left hemisphere (bottom left panel). The most anterior coronal section is the top middle panel, and the most posterior is the bottom right panel. The lesions appear dark, are bilateral and surround the anterior depths of the calcarine fissure. They do not appear to extend beyond area 18 of the visual association cortex or reach the tip of the occipital pole (courtesy of Hanna Damasio).

by bilateral lesions of the visual cortex. When a person in this condition reaches for a fixated target, kinesthetic cues are present throughout the reach, but visual cues are unavailable unless the hand is near the target. If peripheral visual cues of the moving limb are necessary to the feedback control of ongoing reaches, then cerebral tunnel vision should perturb the distance, direction or timing of limb movements to a visual target. Alternatively, kinesthetic input from the moving limb plus knowledge of visual target coordinates may be sufficient to update ongoing reaches, in the absence of visual input on the reaching limb. In this instance, the control parameters of reaching in tunnel vision should resemble those of normal control subjects.

The findings in cerebral tunnel vision have implications for understanding how the brain controls reaching movements to extrinsic targets in everyday life, as well as the specific role of visual field loss in optic ataxia, a disorder of reaching behavior first reported with extensive damage to the posterior sectors of both cerebral hemispheres [1, 8, 28]. The findings also provide evidence relevant to the 'blindsight' hypothesis that predicts residual visual guidance for reaches to targets presented within the aberrant or 'destriated' fields [7, 26, 27, 41].

## Methods

### Subject

A 48-year-old left-handed man (patient RS) was piloting a speed-boat when he suddenly lost all vision except for a small

area around the fixation point. There was no clouding of consciousness, numbness, weakness, double vision or slurred speech, and no swallowing or balance troubles. He managed to stop the boat safely, but required assistance to land it. The patient had had a stroke in the distribution of the calcarine branches of the posterior cerebral artery that affected both sides of the brain. He felt as if he were viewing the world 'through 2 straws'. His visual constriction never recovered.

Within his remaining vision, the patient could see fine details, recognize faces and reported normal color sensation. He could also still read, but preferred not to do so since it was laborious work and he would tend to get lost on a page. Similarly, if he turned on the television, he preferred to listen rather than to watch. He had trouble locating and reaching for objects in front of him as at his dinner setting. The patient also tended to bump into obstacles while walking, and consequently began using a cane for assistance. He could not resume automobile driving or return to his duties as an auto body shop manager.

Neurological examinations were performed on multiple occasions, including the day of the stroke and were normal each time, except for the finding of tunnel vision. On confrontational testing of the visual fields, the patient could not count fingers or detect or even point to the moving hand of the examiner. The patient was alert, oriented to time, place and personal information, and could relate a cogent history. Fundi were normal. There were no abnormalities of the lens, ocular media or retina. There was no diplopia, dysarthria or nystagmus. Ocular rotations were full. There was no weakness or pronator drift, and the patient could make rapid alternating movements with either hand. Light touch, pinprick, vibration, and proprioceptive sensation were intact. The patient had trouble locating objects, and would often misreach. This gave the appearance of optic ataxia, except that he could quickly and accurately acquire targets once he had them in direct sight. He also had trouble navigating between his appointments and would hold

the arm of the examiner and request verbal updates to avoid nearby obstacles.

### Anatomy

A T1-weighted MR scan performed 4 months after the ictus shows the occipital lobe in coronal sections (see Fig. 1). The lesions are bilateral and surround the calcarine fissure. They do not extend very far into the association cortex, and there was no evidence of any damage in area 19.

### Visual fields

Use of a Tangent screen at 2 m with both eyes open showed a double homonymous hemianopia. The remaining sectors of the visual field comprised two small half moons of vision extending approximately  $1.5^\circ$  from fixation on the left, and  $3^\circ$  on the right (Fig. 2). Goldmann perimetry showed that the patient could not detect the largest brightest Goldmann target (V4e:  $64 \text{ mm}^2$ , 1000 apostilb) outside his tunnel vision.

### Visual sensory function

We studied the patient's visuoperceptive abilities in detail (see Table 1). Visual acuity was between 20/15 and 20/20 with each eye. Spatial contrast sensitivity was likewise normal at 1.8 (Pelli-Robson). The Worth four-dot test (four lights seen) showed no evidence of diplopia. Stereoacuity was normal at 40 sec of arc (Randot). Color vision screened with the SPP2, was normal. Thresholds for the perception of a global coherent motion direction signal (tested at  $3^\circ/\text{sec}$  and  $11^\circ/\text{sec}$ ) and the perception of shape-from-motion were relatively normal in

foveal vision, tested by random dot cinematograms using standardized techniques in our laboratory [24, 33]. We also tested the perception of a coherent motion signal (up, down, right, left) in each of the four quadrants eccentric to fixation. The responses in each quadrant depended on a four-alternative-forced choice procedure and did not require the tunnel vision patient to assert the conscious appreciation of motion. With these procedures, the patient showed no ability to perceive a directional motion signal outside the fovea, compatible with his inability to detect the moving hand of the examiner or the motion of his own limb during reaching movements.

### Eye movements

The patient could initiate eye movements both voluntarily and to verbal command. Visual guided saccades were inaccurate until he knew the target's visual position. Eye movements were recorded using an infra-red device (Skalar Eye Position Meter 1500; Delft, The Netherlands) and digitized at 400 Hz. The results showed that ocular fixation was steady. The patient could also follow targets moving horizontally across the face of a videomonitor (see Fig. 3).

### Visual cognition

Neuropsychological testing showed normal orientation, language, and verbal memory and verbal intellect (WAIS-R VIQ = 101). Performance IQ was 82, in the normal range, but somewhat below expectations for the subject. The reduced performance was probably due to difficulties caused by the marked visual loss. Tactile naming and left-right discrimination were intact. The results from a variety of tests showed that the patient's visual cognitive abilities were generally intact (see Table 2).

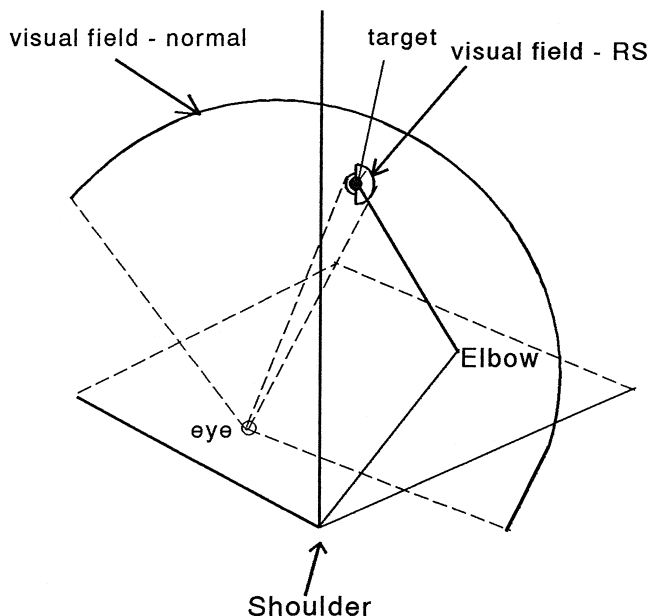


Fig. 2. The visual fields depicted in relation to movements of the arm. The patient had a double homonymous hemianopia. The remaining sectors of the visual field comprised two small half moons of vision extending approximately  $1.5^\circ$  from fixation on the left, and  $3^\circ$  on the right. The patient had no view of the moving limb until the hand was quite close to a fixated target [compare with Fig. 10 (A and B)].

### Testing the useful field of view

This procedure used a pointing task to test if the tunnel vision patient had any useful peripheral vision in the fields of his double homonymous hemianopia that was undetected by standard perimetry and motion perception testing. Such residual vision ('blindsight') would be important to detect since it might theoretically influence the subject's reaching behavior [7, 27] by providing 'unconscious' vision of the limb (and extrinsic targets). If present, such vision might alter the premises of the current study (i.e. that the patient's reaches were being made in the absence of peripheral visual cues).

We assessed the possibility of residual vision using a commercially available microprocessor-based instrument (Visual Resources, Inc., Bowling Green, KY). This instrument uses three subtests to measure the useful field of view (UFOV),

Table 1. Visual perception

Visual acuity	Better than 20/20
Spatial contrast sensitivity	1.8 (Pelli-Robson)
Worth four-dot test	Four lights seen, i.e. no diplopia
Stereoacuity (Randot)	40 sec of arc-normal
Color screening (SPP2)	Normal
Motion direction discrimination (random-dot cinematograms)	Normal
Perception of shape-from-motion	Normal

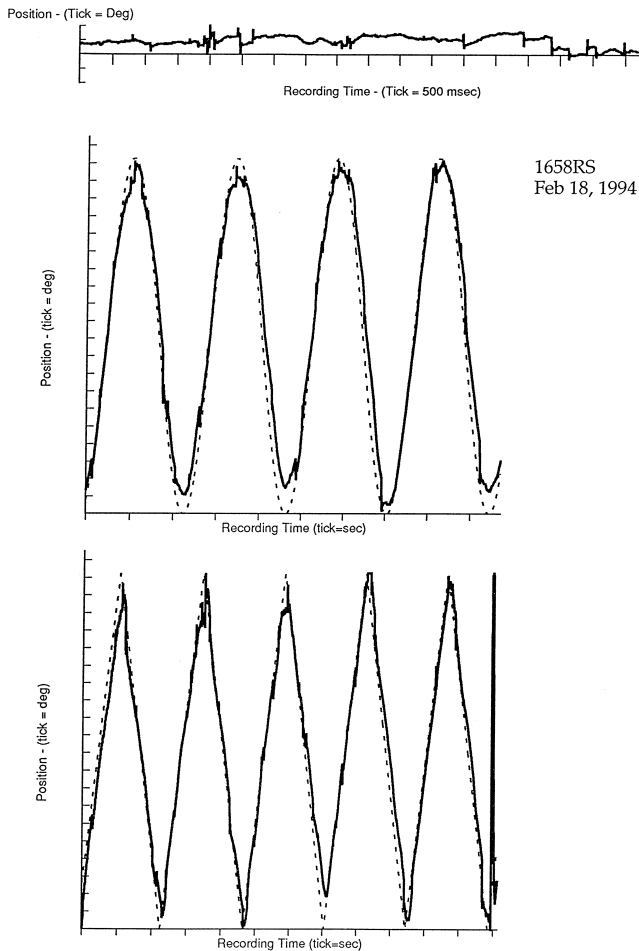


Fig. 3. Eye movements as recorded using an infra-red device (Skalar Eye Position Meter 1500) and digitized at 400 Hz. Top: the results showed that ocular fixation was steady. Middle and bottom: the patient (dark line) could follow targets (dotted line) moving horizontally across the face of a videomonitor. Upward displacement along the  $y$ -axis represents rightward movement. The  $x$ -axis represents time.

Table 2. Visual cognitive abilities

Benton facial recognition test	Score = 47 (71st percentile).
Hooper visual organization task	$T$ -score = 58 (normal)
Mooney faces	37/44 (normal)
Benton visual retention test	Six correct/five errors (normal)
Rey-Osterreith complex figure test-copy	32/36 (50th percentile)
3D block construction	29/29 (normal)
Judgment of line orientation	18 (5th percentile) mild defect score

expressed in terms of the percentage reduction (0–90%) of a maximum  $35^\circ$  radius field [3]. In all three subtests, subjects must identify a target of varying duration, presented at fixation. However, the second and third subtests require the localization of a simultaneously-presented peripheral target presented at one of 24 possible locations, 10, 20 or  $30^\circ$  out from fixation along one of eight radially equidistant imaginary axes (horizontal, vertical and oblique). In the third subtest, the peripheral target is embedded among distracters (triangles).

Importantly, the accuracy of the response in the second and

third subtests depends on a pointing response registered on a large touch screen (20 in diagonal; viewing distance of 23.5 cm). The subject had to reach toward the peripherally presented targets using the index finger of the preferred (left) hand. The factors of high contrast (99%), large target size ( $5.1 \times 3.2^\circ$  of visual angle) and rapid (instantaneous) rise-time are optimal for detecting residual vision [42]. Also, the subject was allowed to look to the location of the switched-off target for pointing, another factor of reputed importance in detecting blindsight in a pointing task [7].

## Results

For subtest 1, the minimum duration at which subjects can perform the task with 75% correct accuracy was determined. For subtests 2 and 3, the best fitting line reflecting the relationship between eccentricity and localization errors was computed for each test duration, and the size of the UFOV was defined as that eccentricity at which the subject could localize the peripheral target correctly 50% of the time. The results yield an overall score between 0 and 90, which represents the percentage reduction of a maximum  $35^\circ$  radius field [3]. Extensive age-related normative data have been collected for this task [2, 3].

The subject could discriminate between targets presented in central fixation on all three subtests. However, on subtests 2 and 3, the subject's pointing responses were unrelated to target location and were even directed to the wrong hemifield. The combined deficits on the three subtest scores gave an overall reduction in the UFOV of 90%. Thus, the size of the UFOV measured on this task was about  $3^\circ$ , a result in striking agreement with that of the Goldmann perimetry. He had no evidence of useful vision outside his tunnel vision, not even on a pointing task. We proceeded to test the role of this profound visual field loss on his reaching performance.

## Apparatus and procedures for studying reaching behavior

### Overview

Reaching behavior was studied using three different experimental procedures. These procedures were conducted 4 months and 12 months after the ictus. Procedure 1 assessed reaches to external targets under visual guidance. Procedure 2 tested the ability to transform visual target coordinates to limb coordinates for reaches to external targets. Procedure 3 assessed reaches between intrinsic and extrinsic targets.

In screening procedures, we noted that the patient could not locate any extrinsic object with his hand without looking directly at it. If the object fell outside his sighted area, he would grope, compatible with his inability to point to visual targets presented in the fields of his double homonymous hemianopia in the UFOV task. Consequently, in experiments using visual targets

(Procedures 1 and 2), the patient had to verbally affirm the sighting of a target before commencing a reach.

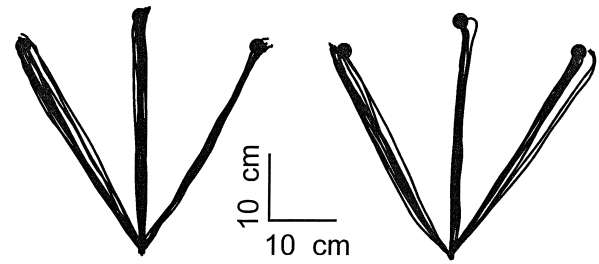
### Apparatus

Data acquisition relied on an optoelectronic technique. Infra-red (IR) emitting diodes were placed on the humerus, elbow, wrist, hand (2), dorsum of the index fingertip on each side, and on the forehead. The positions were recorded by three IR cameras (WATSMART System, Northern Digital, Waterloo, Ont.). Prior camera calibration, using a 3D frame and direct linear transformation software, allowed accurate determination of the 3D spatial coordinates of the body-mounted IREDs. IRED positions were digitized at 100 Hz. Reflection errors were excluded from the analyzed data using methods described in previous studies [34]. Plots of the position and speed of the fingertip movement could then be extracted from the smoothed (10 Hz low-pass Butterworth digital filter, zero phase shift) digitized data to determine movement onsets and endpoints from velocity thresholds.

### Procedure 1. Reaching to external targets under visual guidance

Four highly visible 1-cm diameter markers were placed on a black, non-reflective table surface to designate three targets and a start position. The start position was located directly in front of the subject. The three targets were 36 cm distant from the start position with the middle target being located directly anterior and the left and right targets 15 cm to either side of it. The subject was comfortably seated so that the start and middle targets were in line with the body midline. The ability to smoothly transport each hand from the start position, to touch with the index finger the right, left, or middle target, then return was evaluated. At least 10 round trips were made with each hand to each of these extrinsic visual targets. The subject was asked to make movements at a comfortable speed. The procedures were repeated with the subject reaching as quickly as possible.

**Results:** Plotting the hand paths to targets showed that reaching was on course to all targets with both hands (Fig. 4). The endpoint variabilities of the patient's reaching movements, represented by the area of an ellipse with axes in the A-P and mediolateral directions, were comparable to the results in four non-brain damaged, normally sighted control subjects (Fig. 5). Movements to the middle target showed slightly greater variability (to about  $1 \text{ cm}^2$ ). Yet target diameter was 1 cm, so that in most cases, RS was still on target. Moreover, his reaches were faster than those of controls (see Fig. 6), suggesting that the slightly greater variability was the consequence of a speed-accuracy tradeoff. The time course of hand position and velocity (the kinematics) for reaching move-



Left Hand Movements Right Hand Movements

Fig. 4. The paths of several reaching movements to left, middle and right targets with each hand. The patient is viewed from above. Left hand movements are shown in the left panel, and right hand movements are shown in the right panel. Reaching is on-course to all targets with both hands.

### Pointing to visual targets

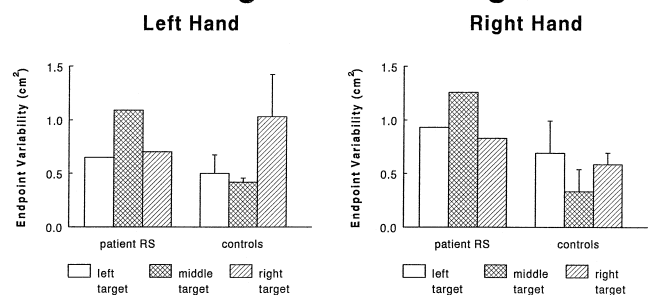


Fig. 5. The endpoint variability of the patient's reaching movements, represented by the area of an ellipse with axes in the A-P and mediolateral directions, is shown. The patient shows low endpoint variability with both hands, comparable to the mean endpoint variability in four non-brain damaged, normally sighted control subjects. The error bars represent 1 S.D.

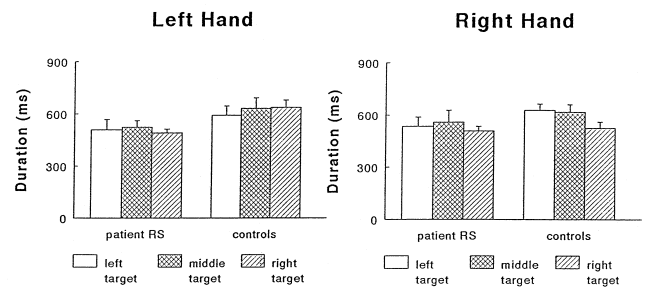


Fig. 6. Movement durations (msec) for comfortable speed reaches are shown. Error bars for patient RS represent 1 S.D. and for controls the means of the within-subject S.D.s. Again the results in the tunnel vision patient resemble those of controls.

ments to visual targets were also normal (Fig. 7). The same normal results were obtained whether the reaches were conducted at 'comfortable' or 'fast' speeds.

### Procedure 2. Transformations between external target coordinates and limb orientation

This procedure was designed to assess the subject's ability to transform a target's visual coordinates to body-

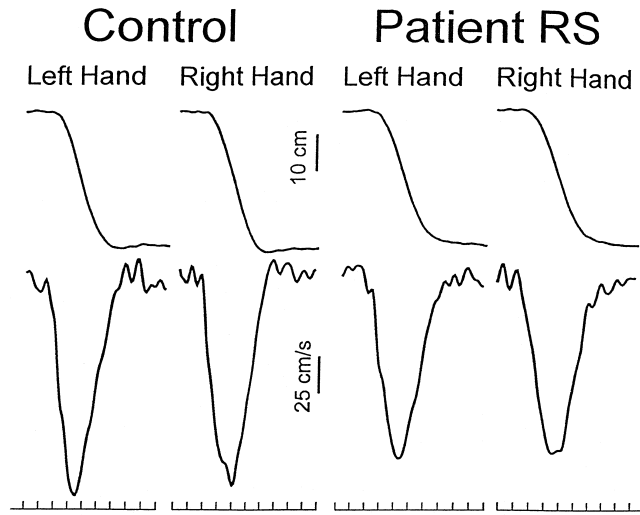


Fig. 7. The kinematics of reaching movements made by the tunnel vision subject (right panel) resembles that of a normal control (left panel). The top tracings show finger position vs time for movements along the anterior/posterior axis to the middle target. The time scale is 0.1 sec between vertical ticks. Movements toward the target are downward. The bottom tracings show the time course of velocity. Notice that the velocity tracings are fairly smooth and show a single major negative peak caused by the acceleration and deceleration of the hand.

The reaches were conducted at 'comfortable' speeds.

centered space. Targets were presented visually, at many different 3D spatial locations within arm's length by the experimenter. However, the subject was blindfolded immediately after spotting the target and before commencing the reach. Also, the target was removed. In this case, the only sensory feedback during the reach was kinesthetic ('open loop' with respect to vision). Sixty to 80 reaches were made with each arm. Comparing the endpoint coordinates of the subject's kinesthetically guided fingertip position following a reach with the actual coordinates of the visually presented target provided an index of the subject's transformation errors. Repeating the same procedures but with no blindfold ('closed loop' with respect to vision) allowed projection of the limb within the hemianopic fields. If these defective fields provided useful information for reaching, then the patient's performance would be expected to improve in this latter condition compared to the former 'open loop' condition.

**Results:** Fingertip and target positions were represented in a spherical coordinate system with its origin at the subject's shoulder [10, 36, 38]. In this system direction (azimuth, elevation) and radial distance give an object's 3D spatial location. Fingertip location can be plotted against target location for azimuth, elevation and distance. Perfectly accurate reaches occur when fingertip location matches target location at movement endpoint. Sensorimotor transformation errors cause deviations from these ideal reaches and can be represented as constant, variable and root mean square (rms) errors in azimuth, elevation and distance. Using this approach, we found that the rms errors for distance (cm) and direction

(azimuth and elevation; radians) were comparable to those of five normally sighted, non-brain-damaged control subjects (Fig. 8). Also, the subject's performance was no better in the visual closed loop than in the open loop condition (Fig. 9). This argues against hemianopic guidance of reaches.

The transformation of target coordinates into an upper limb configuration was examined through the relationship between arm and forearm orientation angles (yaw and elevation) and target location coordinates (azimuth, elevation and distance) [Fig. 10(A and B)]. In young, neurologically normal subjects, the relationships are linear with a coefficient of determination exceeding 0.8 [38]. Arm and forearm yaw angles depend mainly on target azimuth and distance, or upon target azimuth only [38]. Arm and forearm elevation depends on target elevation and distance. The results in our patient were generally comparable. For example, left and right arm yaw angles depended on target azimuth and distance with coefficients of determination of greater than 0.93. Arm elevation depended primarily on target elevation and distance (and in some cases, azimuth), although coefficients of determination were lower than for arm yaw (right arm elevation 0.86, left arm elevation 0.66).

### Procedure 3. Reaches between intrinsic and extrinsic targets

To start this procedure, the subject was blindfolded and the index finger was placed on an extrinsic target located about 40 cm in front in the midline. The subject was then asked to reach for an intrinsic target (the tip of the nose) and then to return the fingertip to the kinesthetically specified extrinsic target. The patient had access to kinesthetic feedback during the reach. Reaches were conducted at a comfortable speed. Five round trips were performed with each hand.

**Results:** Figure 11 depicts left-hand performance. The paths of the index finger movements from extrinsic target

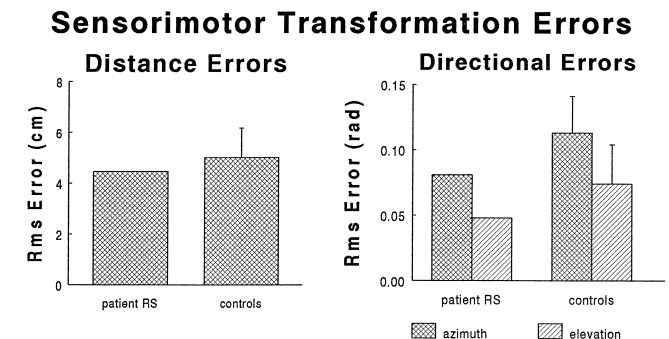


Fig. 8. Sensorimotor transformation errors. The r.m.s. errors (cm) for radial distance are shown (left panel). The r.m.s. errors (radians) for direction (azimuth and elevation) of fingertip and target positions are shown (right panel). The error bars for the control subjects represent 1 S.D. The performance of the tunnel vision patient is comparable to or better than that of controls.

# REACHES TO REMEMBERED VISUAL TARGETS

## LEFT HAND

## RIGHT HAND

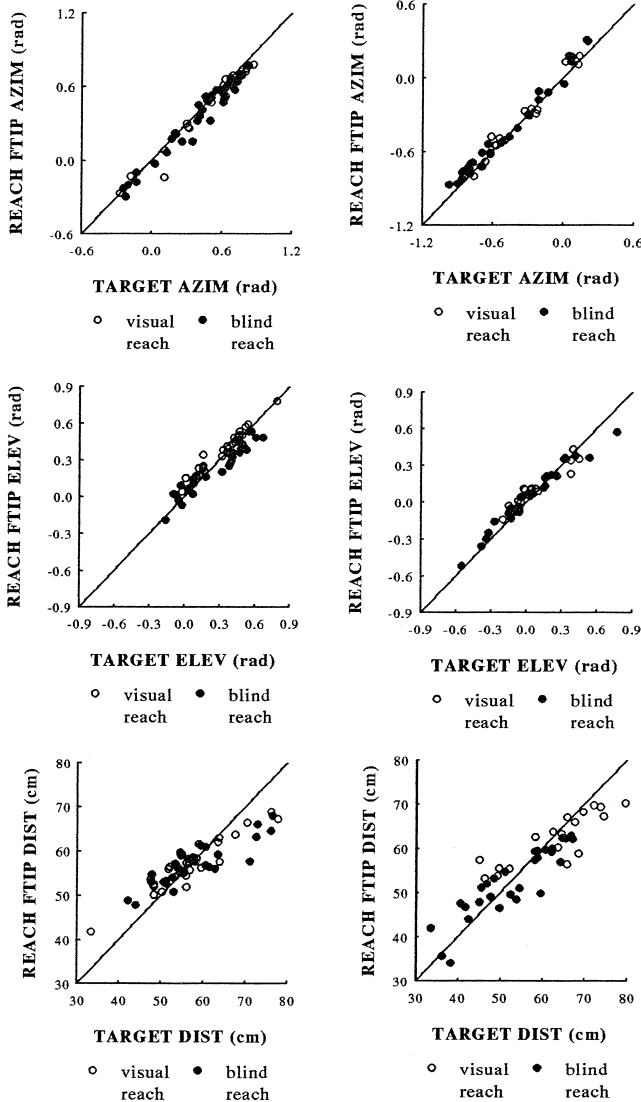


Fig. 9. The tunnel vision patient's performance for reaches to remembered visual targets (Experiment 2). Comparison of the endpoint coordinates of the subject's fingertip position (azimuth, elevation, distance) with the target coordinates provides an index of sensorimotor transformation errors. The results show highly accurate performance, whether the reaches are conducted with the subject blindfolded and no possible vision of the moving limb (black circles, 'open loop' with respect to vision, no 'blindsight' possible) and eyes open (open circles, 'closed loop' with respect to vision, 'blindsight' theoretically available). The lack of difference between the 'open loop' and 'closed loop' performances fails to support a role for blindsight (see text).

to the nose are shown in the left panel. The paths from the nose back to the extrinsic target are shown in the right panel. There was little variability in the hand paths or endpoint accuracy. Movement durations were comparable to those in control subjects. The patient per-

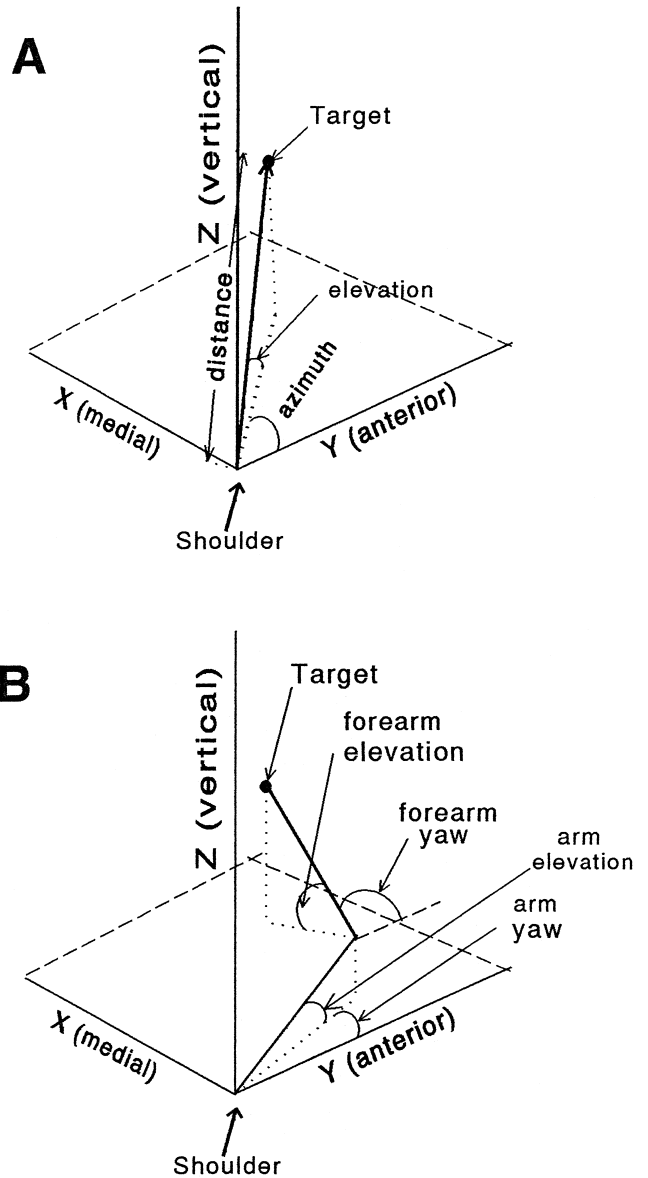


Fig. 10. (A) Visual target coordinates in a spherical coordinate system (azimuth, elevation, distance) with the origin at the shoulder. The fixation target is depicted as a black dot [the same target depicted in Figs 2 and 10(B)]. (B) The relationship between arm and forearm orientation angles (yaw and elevation) and visual target coordinates. Despite an inability to see the hand until it was very close to the fixated target (see Fig. 2), the tunnel vision patient had no trouble transforming visual target coordinates (A), to limb coordinates (B), to produce accurate reaches (see text).

formed equally well with the right hand. Thus, this patient's ability to move to a kinesthetically specified extrinsic target and to an intrinsic (body) target appeared to be normal.

### Discussion

Our patient had extensive bilateral lesions of the occipital lobe affecting the supracalcarine and infracalcarine

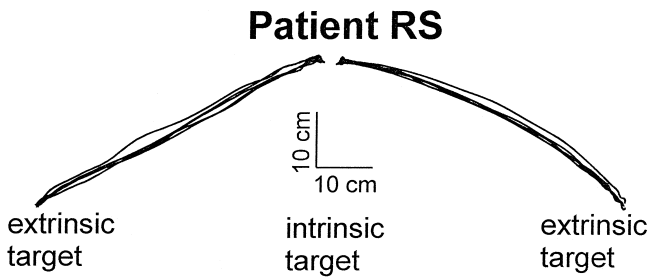


Fig. 11. The paths of the index finger movements from an extrinsic (midline) target to an intrinsic target (the nose) are shown in the left panel. The paths from the nose back to the extrinsic target are shown in the right panel. There was little variability in the hand paths or endpoint accuracy. The patient performed equally well with the right hand. Thus, this patient's ability to make hand movements to a kinesthetically specified extrinsic target and to an intrinsic (body) target appeared to be normal.

cortex along the depths of the calcarine fissure. The foveal representation was spared and there was no evidence of lesion extension into Brodmann's area 19. The result of this damage was a marked concentric reduction of the visual fields leaving a small area of preserved vision about fixation. Visual functions in this remaining keyhole of vision were remarkably preserved, including static visual acuity, spatial contrast sensitivity, color, stereopsis, motion direction discrimination, the perception of shape and structure-from-motion. Visual cognition and intellect were largely normal, as were eye movements and goal directed reaches.

In Procedure 1, our patient made normal reaches to fixated targets under visual guidance, whether these movements were conducted at 'comfortable' or 'fast' speeds. In Procedure 2, he showed the normal ability to transform visual target coordinates to limb coordinates for reaching. In Procedure 3, he generated normal movements to intrinsic and extrinsic targets under kinesthetic guidance. The speed, endpoint accuracy (for distance and direction), and the kinematics of reaches were within normal limits. Moreover, the hand paths were straight to target and there was no position or velocity tremor or ataxic movements.

Interestingly, our tunnel vision patient had to move the eyes and head with precision to foveate targets prior to reaching. This requirement may have enhanced the information available for matching the upper limb configuration to visual target coordinates. In any case, the experimental results are compatible with the hypothesis that monitoring of kinesthetic sensations and efferent commands together with knowledge of external target coordinates gave sufficient information for the guidance of reaching. Thus, we suggest that the underlying sensorimotor transformations for reaching do not occur before area 19 in humans. The relevant structures are likely to include Brodmann's areas 5, 7, superior 19, and 39, in dorsolateral parietal and parieto-occipital cortices, the site of the lesions described in optic ataxia by Bálint

[1] (see below) and subsequent investigators [8, 9, 28] and lately implicated in the control of prehension of visually inspected objects [15]. They also include Brodmann's areas 22, 37, and inferior 19. Lesions in these more ventromesial cortices have been reported lately to alter sensorimotor transformations for reaching [32, 34].

Several studies have tested normal subjects performing reaching movements with vision of the limb blocked while allowing vision of the target. Such visual 'open-loop' movements exhibit a high degree of accuracy, and show no differences [40] or only small differences in speed or accuracy [4, 20, 29] compared to visual 'closed-loop' reaches (vision of the limb available). Indeed, unseen hand movement can be corrected, even if the target is imperceptibly moved during the initial eye saccade toward the target [25, 30]. Jeannerod and Biguer [20] stated that blocking the view of the hand during movement caused reductions in the height to which the hand was lifted. However, this could have occurred simply because the subjects knew they could not see the hand and consequently did not bother to lift the hand into the line of sight. It seems that visual information on hand position in normal subjects is only marginally useful to reaching performance until the hand is close to the target [6] and moving sufficiently slowly [11]. These results are highly compatible with the results of the current study in cerebral tunnel vision.

Sivak and MacKenzie [35] measured the reaching and grasping of dowels in six students wearing pinhole goggles blocking all but the central 10° of vision. Movement direction was accurate and handpaths were straight to target, resembling the findings in our brain-damaged patient. Moreover, the grasp component and the coordination of transport and grasp were also still intact. However, the peak acceleration, velocity and deceleration were slightly slowed down and the movements took slightly longer, mostly due to prolongation of the deceleration phase. In contrast, our subject was not slowed in his movements. This slight difference may relate to the timing of testing in relation to the development of the visual deficit. In particular, our study patient had tunnel vision of chronic duration at the time of evaluation, whereas the Sivak and MacKenzie subjects were tested acutely, having had no chance to adapt to their simulated defect.

The adaptation to the sensory deprivation caused by tunnel vision might be explained by an adjustment of inputs from populations of neurons in the parietal lobe such as the arm projection and manipulation neurons identified in monkey [23]. These neurons project to premotor area (F5) which contains armfield areas and ultimately projects to primary motor cortex (F1) [19, 39]. The relevant parietal neurons are motor or visual dominant. Motor-dominant neurons are active during reaching in light or dark. Visual dominant neurons are activated by fixation of target objects prior to movement or else require vision of the moving arm [19, 39]. The latter population of neurons, assuming they exist in the human,



would be essentially deafferented in tunnel vision. Successful adaptation to this pathological state would require a reweighting of inputs to premotor and motor cortex from the remaining active (afferented) neuronal populations in the parietal lobe that can still service limb control. (We should emphasize that our patient had no evidence of damage in F1, F5 or the parietal lobe.)

Finally, we note that, while visual cues on the position and velocity of the moving limb did not appear to contribute to the control of reaching movements in our patient, they might contribute in certain settings. For example, such cues may serve as a backup to kinesthetic feedback mechanisms for the control of visually guided reaches in individuals who have lost position sense of their limbs and joints due to peripheral neuropathy [14], dorsal column disease, or damage to cortical somatosensory areas S1 or S2 or their connections.

#### *Pointing to targets in the defective field*

Individuals with damage to striate area V1 or underlying optic radiations, as our patient had, deny seeing the fields of their scotoma. Yet perhaps they have ‘blindsight’, a “visual capacity in a field defect in the absence of acknowledged awareness” [41]. Indeed, relevant demonstrations have relied on pointing tasks. Perenin and Jeannerod [26] reported that six adults with occipital lobe lesions could still point to afterimages induced by a light flash in the aberrant field. Yet, light scatter could not be eliminated, and ‘in most cases’, the patient reported a ‘bright flash’, sometimes perceived as ‘grossly circular’. Such conscious appreciation of targets strongly suggests conventional (geniculostriate) vision. Perenin and Jeannerod [27] later reported similar pointing behavior in two out of six hemispherectomy patients. However, fixation difficulties were observed, and target presentations of up to 2 sec increased the chances for fixation lapses, compatible with the observations of increased performance with increased target durations. Also, no neuroimaging procedures were conducted to verify the lesion loci in the preceding two studies.

Corbetta *et al.* [7] also studied pointing to brief light flashes presented to the hemianopic fields of four occipital lobe patients. The subjects performed mostly at chance. However, when the head and eyes were allowed to move freely after the target presentation, Case 4 showed a small increase in the regression coefficients (from  $r=0.23$  to  $r=0.37$ ) for the widely scattered data points relating finger and target position (their Fig. 6). The authors interpreted this as a meaningful improvement and wrote that, “in the free gaze position, patients could combine unconsciously acquired information on target position with the awareness of working space and of hand trajectories during the execution of pointing”. Interestingly, vertical errors were not reported so that a highly inaccurate response might count as a ‘hit’ if its horizontal projection lay somewhere near the target. Also “RTs...

longer than 900 ms (misses of retardations) were rejected” (p. 606), meaning that when a subject did not detect the target in the blind field, the trial did not count. This practice would seem to bias the findings toward residual vision.

Importantly, we found no evidence that our subject’s pointing behavior was guided by inputs from the defective fields either static or kinetic, overt or covert. Thus, in Procedure 2, there was no difference between reaches to visually specified targets conducted in the ‘closed loop’ (eyes open, ‘blindsight’ possible) as opposed to ‘open loop’ (blindfolded) condition. There was also no evidence of residual vision in the peripheral fields on the UFOV task in which the size of the useful fields based on a pointing response corresponded well with the determinations of conventional perimetry. These results were obtained despite optimal presentation strategy and target specifications for detecting residual vision [42] and an unrestricted opportunity to view the moving limb. Furthermore, testing of the defective fields using global coherent motion (random dot cinematogram) stimuli and forced choice procedures shows that our subject can no more detect a directional motion signal than the examiner’s moving hand or his own. The negative findings mirror other clinical observations of the patient’s overwhelming difficulties in highly visual tasks such as reading and navigation. If the tunnel vision patient had a ‘blindsight’, it was hardly useful. The results are compatible with the findings of Hess and Pointer [17] and Merigan *et al.* [22], who found no residual vision in the aberrant visual fields, respectively, of humans and monkeys with V1 lesions. Of course, absence of ‘blindsight’ cannot be proven. Proving any negative assertion is a logical impossibility.

#### **Comment on optic ataxia**

Finally, our tunnel vision patient provides important limiting conditions for assessing the role of visual field loss in optic ataxia. Bálint [1] described optic ataxia as a defect of hand movements under visual guidance, despite normal limb strength and position sense. The index case was a man with extensive cerebrovascular lesions of the posterior hemispheres. Yet, such patients often have extensive visual field defects, visuoperceptive and cognitive defects [31]. Their abnormal reaching behavior might be due to poor visual resolution or visual field defects causing inaccurate visual target localization, or rather to some mismatch between visual target coordinates and limb coordinates. Can these different effects be separated in patients with bilateral lesions?

In brief, Balint’s patient had trouble locating visual targets with the right hand but not supposedly, with the left [16, 31], although Balint provided few critical observations on the latter point. His patient showed a profound left visual hemineglect with visual attention supposedly displaced 35–40° to the right due to extensive

damage in the right inferior and superior parietal lobules. Right hemisphere lesions in these locations are likely to produce visuomotor difficulty in reaches conducted with the left hand to both visual fields ('hand effects') and with both hands to the left visual fields ('field effects') (cf. the modern reviews of Jeannerod [18], pp. 209–244, and Perenin and Vighetto [28]). Thus, there is reason to question Balint's assertion of normal left hand control, just as there is reason to doubt the claim that his subject could see only one object at a time, regardless of the size [31]. Anatomically speaking, Balint's idea that lesions of the angular gyri were responsible for his patient's syndrome was weakened by the presence of extensive bilateral lesions of the thalami (including, in the pulvinar, a critical station for visuospatial processing) and of the posterior white matter (in which optic radiations lie). The current case is most relevant to the potential visual field deficits caused by the latter lesions and their role in the behavioral manifestations of cases such as Balint's.

The patient in the present study had lesions of the early visual cortex in both hemispheres and near-complete visual field loss. However, his cognitive abilities and the visual functions in his residual visual fields were highly preserved. Moreover, while he was observed to grope for targets, this only occurred under the trivial circumstance in which the patient initiated a reach before he had a target in sight. Otherwise, the data show that he made apparently normal sensorimotor transformations between visual target and limb coordinate spaces. The clinical implication is that, in screening for optic ataxia, the examiner should always verify that the subject has foveated the target for a reach before drawing any conclusions on limb movement control.

*Acknowledgement*—This work was supported by NIH PO NS19632.

## References

- Bálint, R. Seelenlahmung des 'Schauens,' optische Ataxie, räumliche Störung der Aufmerksamkeit. *Monatsschrift für Psychiatrie und Neurologie* **25**, 51–181, 1909.
- Ball, K. K., Owsley, C., Sloane, M. E., Roenker, D. and Bruni, J. R. Visual attention problems as a predictor of vehicle crashes in older drivers. *Investigations in Ophthalmology and Visual Science* **34**, 3110–3123, 1993.
- Ball, K. K., Roenker, D. L. and Bruni, J. R., Developmental changes in attention and visual search throughout adulthood. In *The Development Changes in Attention and Visual Search Throughout Adulthood*, J. T. Enns (Editor), pp. 489–507. Elsevier–North-Holland, Amsterdam, 1990.
- Blouin, J., Teasdale, S., Bard, C. and Fleury, M. Directional control of rapid arm movements the role of kinetic visual feedback system. *Canadian Journal of Experimental Psychology* **47**, 678–696, 1993.
- Caminiti, R. and Johnson, P. B. Internal representations of movement in the cerebral cortex as revealed by the analysis of reaching. *Cerebral Cortex* **2**, 269–276, 1992.
- Carlton, L. G. Processing visual feedback information for movement control. *Journal of Experimental Psychology: Human Perception and Performance* **7**, 1019–1030, 1981.
- Corbetta, M., Marzi, C. A., Tassinari, G. and Agliotia, S. Effectiveness of different task paradigms in revealing blindsight. *Brain* **113**, 603–616, 1990.
- Damasio, A. R. and Benton, A. L. Impairment of hand movements under visual guidance. *Neurology* **29**, 170–178, 1979.
- Darling, W., Butler, A. and Rizzo, M. Increased sensorimotor transformation errors in a patient with inferior parietal lobe damage. *Society for Neuroscience Abstracts* **21**, 415, 1995.
- Darling, W. and Miller, G. Transformations between visual and kinesthetic coordinate systems in reaches to remembered object locations and orientations. *Experimental Brain Research* **93**, 534–547, 1993.
- Elliott, D. and Allard, F. The utilization of visual feedback information during rapid pointing movements. *Quarterly Journal of Experimental Psychology A* **37**, 407–425, 1985.
- Georgopoulos, A. P. On reaching. *Annual Review of Neuroscience* **9**, 147–170, 1986.
- Georgopoulos, A. P. Higher order motor control. *Annual Review of Neuroscience* **14**, 361–377, 1991.
- Ghez, C., Gordon, J. and Ghilardi, M. F. Impairment of reaching movement in patients without proprioception. II. Defects of visual information on accuracy. *Journal of Neurophysiology* **73**, 361–372, 1995.
- Goodale, M. A., Milner, A. D., Jakobson, L. S. and Carey, D. P. Neurological dissociation between perceiving objects and grasping them. *Nature* **349**, 154–156, 1991.
- Harvey, M. Translation of 'psychic paralysis of gaze, optic ataxia, and spatial disorders of attention' by Rudolph Balint. *Cognitive Neuropsychology* **12**, 261–282, 1995.
- Hess, R. F. and Pointer, J. S. Spatial and temporal contrast sensitivity in hemianopia. A comparative study of the sighted and blind hemifields. *Brain* **112**, 871–894, 1989.
- Jeannerod, M. *The Neural and Behavioural Organization of Goal-directed Movement*, pp. 209–244, Oxford University Press, Oxford, 1988.
- Jeannerod, M., Arbib, M. A., Rizzolatti, G. and Sakata, H. Grasping objects: the cortical mechanisms of visuomotor transformation. *Trends in Neuroscience* **18**, 314–320, 1995.
- Jeannerod, M., Biguer, B., Visuomotor mechanisms in reaching within extrapersonal space. In *Analysis of Visual Behavior*, D. J. Ingle, M. A. Goodale and R. J. W. Mansfield (Editors), pp. 387–409. MIT Press, Cambridge, MA, 1982.
- Kalaska, J. F. and Crammond, D. J. Cerebral cortical mechanisms of reaching movements. *Science* **255**, 1517–1523, 1992.

22. Merigan, W. H., Nealey, T. A. and Maunsell, J. H. R. Visual effects of lesions of cortical area V2 in macaques. *Journal of Neuroscience* **13**, 3180–3191, 1993.
23. Mountcastle, V. B., Lynch, J. C., Georgopolous, A., Sakata, H. and Acuña, C. Posterior parietal cortex of monkey: command functions for operations within extrapersonal space. *Journal of Neurophysiology* **38**, 871–908, 1975.
24. Nawrot, M., Shannon, E. and Rizzo, M. The relative efficacy of cues from 2-D shape perception. *Vision Research* **36**, 1141–1152, 1996.
25. Pelisson, D., Prablanc, C., Goodale, M. A. and Jeannerod, M. Visual control of reaching movements without vision of the limb. II. Evidence of fast unconscious processes correcting the trajectory of the hand to the final position of a double step stimulus. *Experimental Brain Research* **62**, 303–311, 1986.
26. Perenin, M. T. and Jeannerod, M. Residual vision in cortically blind hemifields. *Neuropsychologia* **13**, 1–7, 1975.
27. Perenin, M. T. and Jeannerod, M. Visual functions within the hemianopic field following early cerebral hemidecortication in man: I. Spatial localization. *Neuropsychologia* **16**, 1–13, 1978.
28. Perenin, M. T. and Vighetto, A. Optic ataxia: a specific disruption in visuomotor mechanisms. *Brain* **111**, 643–674, 1988.
29. Prablanc, C., Echallier, J. F., Komilis, E. and Jeannerod, M. Optimal response of eye and hand motor systems in pointing at a visual target. *Biological Cybernetics* **35**, 113–124, 1979.
30. Prablanc, C. and Martin, O. Automatic control during hand reaching at undetected two-dimensional target displacements. *Journal of Neurophysiology* **67**, 455–469, 1992.
31. Rizzo, M. Balint's syndrome and associated visuospatial disorders. *Clinical Neurology* **2**, 415–437, 1993.
32. Rizzo, M., Darling, W. and Damasio, H. Disorders of reaching with lesions of the posterior cerebral hemisphere. *Society for Neuroscience Abstracts* **21**, 269, 1995.
33. Rizzo, M., Nawrot, M. and Zihl, J. Motion and shape perception in cerebral akinetopsia. *Brain* **118**, 1105–1128, 1995.
34. Rizzo, M., Rotella, D. and Darling, W. Troubled reaching after right occipito-temporal damage. *Neuropsychologia* **30**, 711–722, 1992.
35. Sivak, B. and MacKenzie, C. L. Integration of visual information and motor output in reaching and grasping: the contributions of peripheral and central vision. *Neuropsychologia* **28**, 1095–1116, 1990.
36. Soechting, J. F. and Flanders, M. Sensorimotor representations for pointing to targets in three-dimensional space. *Journal of Neurophysiology* **62**, 582–594, 1989.
37. Soechting, J. F. and Flanders, M. Moving in three-dimensional space: frames of reference, vectors, and coordinate systems. *Annual Review of Neuroscience* **15**, 167–191, 1992.
38. Soechting, J. F. and Flanders, M. Errors in pointing are due to approximations in sensorimotor transformations. *Journal of Neurophysiology* **62**, 595–608, 1989.
39. Taira, M., Mine, S., Georgopolous, A. P., Murata, A. and Sakata, H. Parietal cortex neurons of the monkey related to the visual guidance of hand movement. *Experimental Brain Research* **83**, 29–36, 1990.
40. Vercher, J. L., Mages, G., Prablanc, C. and Gauthier, G. M. Eye-head-hand coordination in pointing at visual targets: spatial and temporal analysis. *Experimental Brain Research* **99**, 507–523, 1994.
41. Weiskrantz, L. *Blindsight. A Case Study and Implications*. Oxford University Press, Oxford, 1986.
42. Weiskrantz, L., Harlow, A. and Barbur, J. L. Factors affecting visual sensitivity in a hemianopic subject. *Brain* **114**, 2269–2282, 1991.