Dorsal and Ventral Stream Interaction: Contributions from Optic Ataxia

Marc Himmelbach and Hans-Otto Karnath

Abstract

In monkeys and humans, two functionally specialized cortical streams of visual processing emanating from V1 have been proposed: a dorsal, action-related system and a ventral, perception-related pathway. Traditionally, a separate organization of the two streams is assumed; the extent of functional interaction is unknown. After lesions of the dorsal stream in patients with optic ataxia, it has recently been shown that the ventral perception-related system might contribute to visuomotor processing if movements rely on remembered target positions. The ventral pathway thus seemed to participate in goal-directed movements, a function that previously has been assigned exclusively to the dorsal stream. We wondered whether different types of pointing movements are controlled by switching between two separated cortical pathways or whether a variable interaction of interconnected systems should be assumed. Our study investigated two acute stroke patients with optic ataxia following lesions of the dorsal stream in a delayed pointing task. The delays ranged from 0 to 10 sec. The patients’ pointing error decreased in a linear manner with the length of time. The finding suggests a gradual change between dorsal and ventral control of reaching behavior, rather than a sudden switch between two separated cortical processing streams. Although our observations with two patients require further validation, the results suggest that the ventral and dorsal systems interact closely in the sensorimotor control of reaching behavior.

INTRODUCTION

Various models of dissociated anatomical pathways processing visual information in the primate brain have been proposed (Ungerleider & Mishkin, 1982; Schneider, 1969; Trevarthen, 1968). Milner and Goodale (1995) suggested a dissociation of two visual pathways depending on the purpose of information processing. Whereas visual information dedicated to the guidance of action was supposed to be processed in a dorsal occipito-parietal stream, visual information processing for the purpose of perceptual tasks was suggested to take place in a ventral occipito-temporal stream. Their proposal was based on findings from single neurological patients with lesions to one of the two cortical pathways. One of these patients suffered bilateral lesions of the ventral stream and demonstrated “visual form agnosia,” the inability to identify familiar objects or estimate objects’ sizes and shapes. In contrast, this patient was able to perform normal, unimpaired grasping movements of the same or similar objects (Goodale, Milner, Jakobson, & Carey, 1991; Milner et al., 1991). On the other hand, it has been shown that patients with lesions of the posterior parietal cortex, that is, with an affected dorsal pathway, demonstrate severe visuomotor deficits (e.g., Perenin & Vighetto, 1988; Ratchliff & Davies-Jones, 1972). These patients show gross deviations of visually guided hand movements landing far from the respective target. This deficit, termed optic ataxia, is typically restricted to movements aiming at targets in the visual periphery. Goal-directed movements to foveated targets are largely spared. In contrast to patients with ventral lesions and visual form agnosia, patients with optic ataxia can reliably detect and localize visual stimuli in their environment and produce reliable perceptual estimates of objects’ shape, size, and orientation (Milner et al., 2001; Jeannerod, Decety, & Michel, 1994; Perenin & Vighetto, 1988).

Milner and Goodale (1995) also suggested temporal differences of information processing in the two visual pathways. Information processing in the dorsal stream, dedicated to control of action, should be accomplished within a short time scale as the relevant information about the environment changes very fast with respect to the position and orientation of our limbs and/or eyes. Perceptual processing in the ventral stream on the other hand does not critically rely on a fast updating of visual information. In comparison to action control, object recognition and memory are relatively independent of certain views or positions. Evidence supporting the assumption of this dissociation of temporal characteristics between the dorsal and the ventral stream has been observed in 1 patient with visual form agnosia (Milner, Dijkerman, & Carey, 1999; Goodale, Jakobson, University of Tübingen, Germany
and Keillor, 1994; Milner et al., 1991) and 3 patients with optic ataxia (Milner, Dijkerman, McIntosh, Rossetti, & Pisella, 2003; Revol et al., 2003; Milner et al., 2001; Milner, Paulignan, Dijkerman, Michel, & Jeannerod, 1999). It has been shown that the patient with bilateral lesions of the occipito-temporal cortex and severe visual form agnosia lost her ability to adjust the hand aperture to different object sizes if the movements were delayed for 2 sec (Goodale et al., 1994). Corresponding to this delayed grasping deficit, the same patient showed an increase of pointing errors to remembered target positions after a delay of 10 sec, which was significantly higher than the respective change in healthy controls (Milner, Dijkerman, et al., 1999). On the other hand, misreaching in patients with optic ataxia is considerably improved when a delay of 5 sec is introduced between target presentation and movement onset (Milner et al., 2003; Revol et al., 2003; Milner, Paulignan, et al., 1999).

In the view of Milner et al. (2003), the improvement of goal-directed movements in patients with optic ataxia following such a delay is mediated by the spared processing in ventral stream areas. The control of movements toward remembered targets was supposed to be guided by the system primarily dedicated to conscious recognition and memory (i.e., the ventral system). This interpretation is also sufficient to explain the pathological decrease of delayed movements’ accuracy in the patient with visual form agnosia and bilateral occipito-temporal lesions.

Following this assumption, the question arises how the interaction of the two systems is implemented in the human brain. Does the control of visually guided reaching just switch from the dorsal to the ventral stream, that is, is there an abrupt takeover by the intact ventral system when targets are not longer visible? If so, one would assume that after an initial improvement because of the disappearance of a target, the performance of patients with dorsal lesions should be stable, independent of the length of the additional delay interval. Alternatively, one could assume that the contribution of the ventral system increases as the delay between target presentation and movement execution gets longer. Thus, movement errors in patients with dorsal lesions should decrease with delays of increasing duration.

Recently, Westwood and Goodale (2003) investigated these alternatives in a sample of healthy subjects and concluded that the change from dorsal to ventral control of grasping movements takes place once the target disappears. A delay between target presentation and movement onset does not affect that change. The authors thus concluded that the dorsal system, namely the superior parietal cortex, does not store target information, not even for very brief delays. They proposed that the dorsal system is not involved in the spatial control of memory-guided movements.

To investigate these assumptions on the two visual systems, we examined the pointing accuracy of two patients (G.H. and U.S.) with optic ataxia in a delayed pointing task with four different delays (0, 2, 5, and 10 sec). The subjects sat in front of a vertical board. They were instructed to make an accurate pointing movement to a target, randomly chosen from 8 possible target positions located on the left and right side of the board (Figure 1). The subjects were instructed to start their hand movements upon an auditory signal, which was presented at the end of the delay.

**RESULTS**

**Baseline Condition**

We computed the absolute error of the pointing movements as the distance between the tip of the finger and the respective target at the end of the movement. Large errors were observed in both patients when pointing to visible targets was requested (Figure 2; baseline condition). This corresponded with the clinical findings (Table 1). The patient with unilateral lesions (G.H.) showed optic ataxia in her right visual half-field (Figure 2), where the mean absolute error (4.6 cm)
exceeded 2 SDs of the mean error of the control group (1.6 ± 0.5 cm). In contrast, her mean error on the left side was comparable with that of the control group. The patient with bilateral lesions (U.S.) showed huge pointing errors on both sides of the visual field that exceeded 2 SDs of the control group data by far (Figure 2). For patient U.S., movements to targets in both visual half-fields were thus included in the following analysis. For G.H., only movements to targets located on her right side were analyzed because optic ataxia was present only in her right visual half-field (Figure 2).

**Experimental Condition**

To address directly the hypothesis that the disappearance of a target might lead to significant improvement of movement accuracy independent of additional delays (Westwood & Goodale, 2003), we compared the patients' baseline performance with their movement errors following a delay of 0 sec. In both patients, one-tailed paired t tests did not reveal significant improvements (U.S., \( t = .11, p = .455 \); G.H., \( t = .86, p = .200 \)). In contrast, the same comparison revealed a significant decrease of accuracy in controls (\( t = −2.24, p = .036 \)) (Figure 3).

Further, to test whether increasing delay times give rise to a systematic decrease of movement errors, we performed individual repeated measures analyses of variance (ANOVs) for each patient using factor “delay.” The analyses revealed a significant effect for patient U.S. (\( F_3 = 3.14, p = .029 \)); it was not significant for patient G.H. (\( F_3 = 1.36, p = .261 \)). In the case of U.S., linear contrasts revealed significant differences between the 0-sec delay and the 5- and 10-sec delay, respectively (5 sec,
It is possible that the behavioral differences associated with successive delay intervals might be small and thus would require a high number of trials to become significant. Because a high number of trials could not be performed in the acute patients, we tested for such a possible trend carrying out regression analysis. We computed linear regression coefficients of the subjects’ “absolute pointing error” on the variable “delay time.” In both patients, the errors significantly decreased with increasing delay times between target offset and movement onset (Figure 3). We revealed negative linear regression coefficients, which were significantly smaller than zero in both cases (U.S., \(t = -2.70, p = .004\); G.H., \(t = -1.73, p = .044\)). In contrast, the regression coefficient measured in the control group was not significantly different from zero (\(t = .04, p = .967\)) (Figure 3). We also computed the individual regression coefficients for each control subject. The mean of these coefficients was \(b = .0005 (SD = .04)\). The regression coefficients of both optic ataxia patients thus were more than 2 SDs smaller than the mean coefficient of the control group.

To substantiate that the observed significant negative slopes of the regression analyses represent a gradual decrease of the movement error with successive delay intervals, we arranged the patients’ data of the delays 2, 5, and 10 sec in such a way that it suited best with the alternative assumption, namely, with the assumption of a sudden performance improvement between 0- and 2-sec delays. We permuted the data from the 2-, 5-, and 10-sec delay measurements following the criterion that the permuted data of these delays results in an individual mean value not more than 1% larger or smaller than the overall mean of all 3 delays (U.S., delay 0 = 9.31; delay 2 = 6.52; delay 5 = 6.52; delay 10 = 6.52; G.H., delay 0 = 4.01; delay 2 = 3.14; delay 5 = 3.14; delay 10 = 3.14). For each patient, we then computed linear regression analyses on 10 different permutations of the original data. In both patients, the outcome of all analyses was insignificant.

**Movement Time and Acceleration**

The patients could have improved the accuracy of pointing movements in the delay conditions by decreasing their velocity and taking more time for small adjustments. To investigate this possibility, we computed the bivariate correlation between movement time (MT) and the absolute error in both optic ataxia patients and in the control group for the data obtained in the experimental conditions. For the control group, we revealed a significant negative correlation (\(R = -.55, p = .013\)), indicating that their mean error was smaller with longer MTs. In contrast, patient U.S. showed no significant correlation (\(R = .09, p = .325\)), whereas patient G.H. demonstrated even an inverse correlation (\(R = .44, p < .001\)), indicating that she was even less accurate with longer MTs. The bivariate correlation analysis thus showed that the optic ataxia patients did not improve accuracy of pointing in the delay condition just by decreasing the velocity of the movements.

Still, the patients could have included more adjustments in the same time. To analyze this possibility, we determined the number of movement corrections by calculating the number of zero crossings of the movement acceleration function for each subject. The control subjects produced a biphasic acceleration pattern in the majority of their movements. The mean number of zero crossings was close to one (delay 0 = 1.08, SD = 0.08; delay 2 = 1.06, SD = 0.07; delay 5 = 1.08, SD = 0.11; delay 10 = 1.12, SD = 0.14) and was not significantly correlated to the length of the time delay in the experimental conditions (\(R = .18, p = .440\)). Patient G.H. also demonstrated a biphasic acceleration in most trials but nevertheless had a higher number of zero crossings in comparison to controls, slightly exceeding the interval of 2 SDs for delays between 0 and 5 sec (delay 0 = 1.33, SD = 0.75; delay 2 = 1.38, SD = 0.79; delay 5 = 1.38, SD = 0.79; delay 10 = 1.33, SD = 0.75). In contrast to final movement accuracy (see above), the number of acceleration periods was not significantly correlated to delay time (G.H., \(R = .05, p = .600\)). Unlike the control subjects and patient G.H., patient U.S. showed jerky movements throughout all conditions. Although the resulting velocity did not meet the kinematic criterion for movement end, a high number of distinct acceleration periods was observed (delay 0 = 6.32, SD = 5.33; delay 2 = 4.47, SD = 2.57; delay 5 = 6.87, SD = 5.63; delay 10 = 6.94, SD = 3.85). However, also in patient U.S., the number of acceleration periods and thus the number of corrections was not correlated with delay time (\(R = .118, p = .193\)).

**DISCUSSION**

The focus of the present study was to investigate how the dorsal and the ventral systems interact in the control of reaching behavior. Investigating two patients with dorsal stream lesions, we could not find evidence for the hypothesis that the disappearance of a target might lead to significant improvement of movement accuracy independent of additional delays (Westwood & Goodale, 2003). Linear regression analysis revealed a clear difference between the two patients with optic ataxia and controls. The findings argued for a decrement of patients’ pointing errors depending on the amount of time that had passed since the disappearance of a visual target. This result suggests a gradual change between dorsal and ventral control of reaching behavior, rather than a sudden switch between two separated cortical pathways.
However, the ANOVAs did not support the assumption of such a gradual change. Significant differences were not observed for pairs of successive delays. Nevertheless, the bilaterally lesioned patient demonstrated a significant decrease of errors for delays longer than 2 sec. Based on the ANOVA alone, we thus cannot decide whether the movement errors decrease suddenly at a certain delay time or continuously. However, it is possible that the behavioral differences associated with successive delay intervals might be small and thus would require a high number of trials to become significant. Because a high number of trials could not be performed in the acute patients, this might be a possible reason why the observed effects from the regression analysis could not be verified using ANOVA.

Both the observed clinical deficit and the effect of the delays were more prominent in the patient with optic ataxia and bilateral lesions than in the patient with unilateral damage. What might be the reason for this difference? A possible explanation could be that there was residual ipsilateral processing in the unilateral but not the bilateral case. Such ipsilateral processing is suggested by the typical distribution of errors with respect to the moving hand and the visual field of target presentation in optic ataxia patients with unilateral lesions. Whereas left hemisphere lesions lead to a deficit restricted to the contralesional hand and frequently also to the contralesional visual field, lesions of the right hemisphere lead to a deficit of both hands in the contralesional visual field (Revol et al., 2003; Perenin & Vighetto, 1988). Considering this pattern, the right dorsal system might be capable of ipsilateral movement control. Thus, at least in patients with unilateral left hemisphere lesions, visuomotor processing by the right hemisphere might in part compensate deficient processing of the left dorsal system. However, observing only two patients cannot provide conclusive evidence for such speculations. The behavioral differences observed in the two patients might likewise be attributed to individual differences with respect to lesion site and size.

Our findings seem to argue against recent assumptions by Westwood and Goodale (2003). The authors induced a size-contrast illusion in healthy subjects changing the apparent size of the target object while its physical dimensions remained constant. Such a change of apparent size is known to have an impact on perceptual judgments and delayed grasping but not on immediate grasping of target objects (Hu & Goodale, 2000). Beside a delay between target presentation and movement onset, Westwood and Goodale (2003) varied the visual feedback during the movement. They compared full visual feedback and the complete occlusion of the hand and the target. The authors found that the influence of the flanker object on grasping movements depended entirely on the occlusion of vision, whereas the delay had no effect. The authors thus assumed that the ventral visual system provides the decisive spatial information that is necessary to control hand movements if no immediate visual information about target size and position is available.

Other behavioral experiments in healthy subjects yielded different results. For example, it has been demonstrated that the impact of a visual illusion on pointing accuracy increases significantly with longer time delays (Bridgeman, Gemmer, Forsman, & Huemer, 2000). Thus, even if there would be a dramatic shift between the dorsal and the ventral systems instantly after the target has disappeared as shown by Westwood and Goodale (2003), there still seems to be a progressive change depending on the time delay between target presentation and movement onset. Such a progressive change clearly points to residual dorsal processing in delayed movements. In addition to this behavioral evidence, several findings from single-cell recordings in monkeys and neuroimaging studies in healthy subjects suggested that areas of the dorsal system are critically involved in delayed movement tasks. Single-cell recordings revealed visual and visuomotor neurons in the anterior part of the intraparietal (AIP) sulcus, which demonstrated sustained activity during the delay following the presentation of an object that subsequently had to be grasped (Murata, Gallese, Kaseda, & Sakata, 1996). Similar findings of delay activity have also been reported by Snyder, Batista, and Andersen (1997). These authors demonstrated increased levels of single-cell activity in the medial intraparietal area, which were specific for the planning of hand movements. Whereas Snyder et al. (1997) applied delays between 1 and 1.6 sec, Murata et al. (1996) applied delays of 2 sec; thus, both experiments were confined to short intervals in comparison to the delay times used in our present experiment. Using substantially longer delays, neuroimaging studies in healthy subjects revealed a specific increase of activity in the superior parietal cortex correlated with the planning of delayed hand movements (Connolly, Andersen, & Goodale, 2003; Lacquaniti et al., 1997). Connolly et al. (2003) found sustained activity in the superior parietal cortex during a delay of 9 sec between target presentation and movement execution.

These findings support the assumption that the dorsal system is involved not only in immediate but also in delayed pointing movements. It seems as if the dorsal stream stores spatial information on target position during interfering delays between target presentation and movement execution. However, these studies were designed to search for correlations between superior parietal activity and delayed movement tasks. They cannot answer the question whether this activity in superior parietal cortex is necessary to perform memory-guided movements. This question rather is addressed investigating the performance of brain-damaged patients with dorsal lesions. It is clear from previous reports (Milner et al., 2003; Milner, Paulignan, et al., 1999) as
well as our present data (Figure 3) that such patients do not reach the level of performance of healthy subjects in memory-guided actions even after long delays. Damage to the dorsal system thus indeed seems to have an impact on the planning and/or execution of delayed movements. Findings from a recent transcranial magnetic stimulation (TMS) study using a memory-guided pointing task further support this conclusion (Smyrnis, Theleritis, Evdokimidis, Mür, & Kandareas, 2003). The application of a single TMS pulse over the posterior parietal cortex as early as 300 msec after target presentation had a significant effect on the accuracy of hand movements executed 3000 msec after target offset. This indicates that the early encoding of the target stimulus or the early planning of hand movements is associated with undisturbed posterior parietal cortex function.

The present work did not distinguish between grasping and pointing movements. In fact, the majority of reported optic ataxia patients present a defect of hand preshaping as well as disturbed reaching and pointing movements (Milner et al., 2003; Milner et al., 2001; Perenin & Vighetto, 1988). On the other hand, cases have been reported showing dissociations between a grasping deficit and intact reaching/pointing movements (Binkofski et al., 1998; Jeannerod et al., 1994; Jakobson, Archibald, Carey, & Goodale, 1991). However, in these studies, target objects were placed at the subjects midline, presumably in the line of sight, a situation where most patients with optic ataxia show the least impairments (Milner, Dijkerman, et al., 1999; Perenin & Vighetto, 1988). In fact, it has been shown by Milner, Paulignan, et al. (1999) that the patient investigated by Jeannerod et al. (1994) with preserved reaching for targets under foveal vision demonstrated prominent misreaching when targets were located in the visual periphery. Nevertheless, studies employing temporary inactivation of a parieto-frontal circuit composed of AIP and the prefrontal area F5 in nonhuman primates clearly demonstrated isolated deficits of grasping movements, whereas reaching to the position of target objects was not affected (Fogassi et al., 2001; Gallese, Murata, Kaseda, Niki, & Sakata, 1994). These data argue for dissociated pathways or circuits subserving grasping movements on the one hand and reaching and pointing movements on the other hand. Although the aforementioned AIP–F5 circuit presumably constitutes the substrate of grasping, another parieto-frontal network consisting of the ventral intraparietal area and F4 is assumed to mediate the spatial organization of reaching movements (Rizzolatti & Matelli, 2003). Such a dissociation could explain some of the contradicting results obtained from behavioral studies in humans. For example, the aforementioned work by Westwood and Goodale (2003) on the one hand and by Bridgeman et al. (2000) on the other could be reconciled if we assume that reaching and grasping pathways are affected by visual illusions differently. However, other authors favor a strong functional coupling between distinguishable dorsal networks (see Galletti, Kutz, Gambarini, Breveglieri, & Fattori, 2003, for a review). They attenuate the assumption of a strict segregation of processes related to grasping and to reaching/pointing. In summary, these findings and interpretations of anatomical and behavioral data suggest that the previously proposed dichotomous view of visual perception and visuomotor action related to two visual processing streams requires further refinement and concise definitions of the behavior we are looking at.

Our present results suggest that the compensation of misreaching in patients with dorsal stream lesions does not rely exclusively on a switch between a deficient (dorsal) visuomotor and a spared (ventral) visuoperceptove processing system in the control of movements to visible targets and movements to remembered target positions, respectively. They contradict the hypothesis that the disappearance of a target might lead to significant improvement of movement accuracy independent of additional delays (Westwood & Goodale, 2003). Instead, the dorsal and ventral systems seem to closely interact in the sensorimotor control of delayed reaching movements. We observed a trend that this interaction is correlated with the length of the time delay between target presentation and movement onset. In conclusion, previous work and the present data on the reaching behavior of brain-damaged patients, TMS-induced effects in healthy subjects, and the evidence derived from neuroimaging studies and single-cell recordings argue for a decisive role of the dorsal system (which might consist of more than one pathway) not only for immediate movements if visual information about a target is available, but also for the control of delayed reaching movements.

METHODS

Subjects

We investigated two patients with optic ataxia (U.S. and G.H.: 62 and 57 years) 8 and 5 days after an ischemic stroke. A group of 6 non-brain-damaged neurological patients (age: median = 65 years, range = 16; 6 women) served as controls. All patients gave their informed consent to participate in the study, which has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. Patients were classified as having optic ataxia when reaching for objects in peripersonal space was grossly ataxic and remained uncorrected, as confirmed by the following clinical test. With head and trunk aligned, the patient grasped for targets at random locations. Ten reaches were recorded in each of 8 test conditions produced by crossing target side (left vs. right hemispace), with reaching hand (left vs. right hand), and with fixation condition (peripheral vs. foveal vision of the target). Patients with optic ataxia...
perform with large spatial errors when reaching for objects in peripheral vision. The majority of these ataxic reaches remain uncorrected. Table 1 presents the frequency of those trials in which the patients missed the target and did not correct their errors (ataxic reaches). U.S. showed severe optic ataxia in both visual half-fields with her left hand; G.H. showed a hand and field effect, with the worst performance in the right visual half-field using her right hand.

U.S. was a 62-year-old, left-handed woman. She was admitted after sudden onset of right hemiplegia and mild difficulties in word finding. Magnetic resonance imaging (MRI) showed bilateral lesions of the parietal cortices (Figure 4). In addition, left hemisphere damage comprised the occipito-temporal cortex, the left inferior frontal gyrus, as well as smaller portions of the post- and precentral gyri and underlying white matter. Neuropsychological testing using hierarchical letter stimuli (Navon, 1977) and pictures of complex scenes (e.g., the Cookie Theft Picture from the Boston Diagnostic Aphasia Examination) revealed simultanagnosia. U.S. further demonstrated right-sided spatial neglect omitting 28 of 30 targets on the right side of the Letter Cancellation Task (Weintraub & Mesulam, 1985) and 12 of 15 right-sided targets in the Bells Test (Gauthier, Dehaut, & Joanette, 1989) (omissions on the left side: 7/30 letter cancellation; 5/15 bells test).

G.H. was a 57-year-old, right-handed woman. MRI scanning revealed a lesion of the left medial parietal cortex mainly involving the precuneus (Figure 4). Beyond optic ataxia, the patient showed simultanagnosia. She did not show signs of spatial neglect. Clinical examination of both patients revealed no signs of apraxia, no visual field defects, no paresis of the dominant hand/arm as well as normal smooth pursuit and saccadic eye movements.

**Procedure**

Subjects sat at a distance of 45 cm in front of a vertical board. Their midsagittal plane was aligned with a green LED positioned in the middle of the board at eye level (Figure 1). Four horizontally arranged red LEDs were located left and right of the central green LED. The distance between the LEDs was 5 cm, resulting in a distance of 20 cm from the central green LED to the most peripheral red LEDs on either side. The board was covered with black textile; therefore, the LEDs were invisible until they were lighted. The head of the subjects was fixed with a helmet in a position aligned with the body midsagittal plane. The subjects were instructed to hold their gaze on the central green LED throughout the whole experiment. Accurate fixation was monitored using an infrared eye-tracking device that provided online display of eye position. Trials with eye movements were discarded and repeated during the experiment. The patients were required to make an accurate movement at a comfortable speed with the dominant ataxic left hand (U.S.) and the dominant ataxic right hand (G.H.) to the presented targets. Accordingly, the control subjects also pointed to the targets using their dominant, right hand. We measured 6 movements to each target per condition in patient G.H. and controls, whereas only 4 movements per target position and condition were measured in U.S. because of fatigue and concentration problems. The movements of the hand were recorded with an ultrasonic 3-D tracking device (Zebris, Zebris Medical, Isny, Germany) using one marker at the

![Figure 4](http://example.com/four.png)
tip of the index finger with a sample rate of 150 Hz. Data were digitized and stored on a PC for off-line analysis. They were filtered with a low-pass Butterworth filter and a cutoff frequency of 10 Hz. Missing data points of the finger marker were interpolated linearly for a period up to 50 msec when the marker got lost (e.g., because of occlusion of the marker). Kinematic start and end of movements were defined as the time when hand velocity reached 5% of tangential peak velocity or a velocity of less than 50 mm/sec. The following 2 conditions were conducted blockwise in an ABBA design.

**Experimental Condition**

At the beginning of each trial, the subjects placed the hand on a starting position located in front of them at their body midline (Figure 1A). After a random delay between 1000 and 2000 msec, one of the peripheral red target LEDs appeared and was visible for 2000 msec. Following a delay of 0, 2, 5, or 10 sec after target offset, an auditory signal indicated the start of the pointing movement to the remembered target position.

**Baseline Condition**

Differing from the experimental condition, target presentation was repeated after a delay of 5 sec, simultaneously with the auditory start signal (Figure 1B). The target was visible for 5 sec (i.e., throughout the whole pointing movement of the subject).

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Reprint requests should be sent to Marc Himmelbach, Section Neuropsychology, Department of Cognitive Neurology, Hertie-Institute for Clinical Brain Research, University of Tübingen, Hoppe-Seyler-Strasse 3, D-72076 Tübingen, Germany, or via e-mail: marc.himmelbach@uni-tuebingen.de.

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