# REVIEW

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# **Optic ataxia revisited:**

# Visually guided action versus immediate visuomotor control

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Abstract Optic ataxia and visual agnosia have been proposed to constitute a double dissociation which provides the main argument for the assimilation of the anatomical distinction between a dorsal and a ventral visual stream to the functional distinction between perception and action. In the present review, we argue that insufficient evidence has been collected to argue for this double dissociation. Several criteria are reviewed: (1) exploration of the visuomotor behavior in central versus peripheral vision has not been matched for the two types of patients; (2) the temporal constraints of visual processes that are impaired in the two neurological conditions appear to play a crucial role in the apparent dissociation; (3) the necessary reductionism of experimental conditions used to study action has led to an overconsideration of optic ataxia as a global deficit for action. Altogether optic ataxia appears to result from a specific impairment of immediate visuomotor control rather than of visually guided action as a whole. These results are discussed in the light of recent research on optic ataxia and on motor control, and directions for future research are proposed.

Keywords Optic ataxia  $\cdot$  Visual agnosia  $\cdot$  Peripheral vision  $\cdot$  Central vision  $\cdot$  Visual stream  $\cdot$  Visuomotor transformation

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#### **History and definition**

Balint (1909), followed by Holmes (1918), described patients with large lesions in the posterior parietal cortex (PPC) who exhibited a complex pattern of impairment (now referred to as Balint syndrome), including characteristic inaccuracy when they reach or grasp visual objects. Balint (1909) suggested that this inaccuracy was due to a visuomotor disconnection, which he called optische ataxie, whereas Holmes (1918) considered it rather resulted from a global impairment in spatial perception, which he called visual disorientation. Subsequent studies (Garcin et al. 1967; Ratcliff and Davies-Jones 1972; Vighetto 1980; Vighetto and Perenin 1981; Perenin and Vighetto 1983, 1988) have progressively argued and demonstrated that visuomotor deficits for peripheral targets can occur independently from perceptual disorders, rather supporting the optic ataxia definition of Balint (1909). The term ataxie optique was initially used in French (Garcin et al. 1967) to describe pure cases with unilateral lesions and deficit restricted to the peripheral visual field; whereas optische ataxie was used for patients with Balint syndrome, where praxic aspects are difficult to disentangle from ataxic symptoms, including visuomotor problems in central vision. Garcin et al. (1967) have proposed a number of conditions necessary for diagnosing optic ataxia: (1) the visual field should be spared in the area concerned by the visuomotor deficit; (2) proprioception should be spared; (3) there should be no intrinsic motor (and oculomotor) and no cerebellar deficit. Interpretation of optic ataxia as a specific visuomotor disorder was particularly reinforced by the careful study of reaching behavior by Vighetto (Vighetto 1980; Perenin and Vighetto 1988). First, testing auditory-guided movements showed that optic ataxia appears as a modalityspecific reaching impairment. Second, although verbal discrimination of dot position was impaired in some patients, a direct causal link between these subtle deficits in visual space perception and the gross misreaching errors was excluded by the authors. Finally, patients showed reaching errors related both to a visual field effect and to a

hand effect. This combination of sensory and motor influences, as well as the localization of the underlying lesion in-between the visual and the motor areas, supports the idea that the deficit lies just at the visuomotor interface rather than solely at either the sensory or motor level. Superimposition of the lesions of six left brain-damaged and two right brain-damaged patients with pure optic ataxia using CT scans (Perenin and Vighetto 1988) has revealed a symmetrical converging region that includes the superior parietal lobule, in and above the intraparietal sulcus and sparing the human inferior parietal lobule. The localization of the lesion leading to optic ataxia led to this disorder being considered an impairment of the visual dorsal stream.

#### **Current dominant theory**

In the anatomical context of a dorsal (occipitoparietal) versus a ventral (occipitotemporal) visual stream, the visuomotor deficit of optic ataxia has been used as the main argument to attribute the How function to the dorsal stream. Indeed, such patients demonstrate an exaggerated and poorly scaled grip aperture compared with healthy subjects, although they are not impaired in object recognition (Jeannerod 1986; Perenin and Vighetto 1988; Goodale et al. 1991; Jeannerod et al. 1994; Milner and Goodale 1995). On the other hand, patients with lesion of the inferotemporal cortex (ventral stream) are impaired in object recognition (visual agnosia) but remain able to reach and grasp these same objects that they cannot describe. The reciprocal patterns of impairment found in optic ataxia and in visual agnosia have been considered as a functional double-dissociation (see Rossetti and Revonsuo 2000), leading to the interpretation of these two visual streams as a dorsal visual pathway for goal-directed action and a ventral visual pathway for perception (Goodale and Milner 1992; Jeannerod and Rossetti 1993, Milner and Goodale 1995; Rossetti 1998). In contrast with these early interpretations, it is rather important to note that patients with pure optic ataxia can perform everyday actions, whereas patients with visual agnosia are strongly impaired in their daily behavior.

The neuroanatomical basis for a dissociation between perception and action can also be guestioned. A recent review of anatomical networks between primary visual area V1 and primary motor area M1 has underlined that the ventral stream, as well as the dorsal stream, projects onto the frontal motor areas, and consequently the dorsal stream has no private pathway toward M1 (see Fig. 1; Rossetti et al. 2000; Rossetti and Pisella 2002). Furthermore, no strict segregation between two visual streams appears within the complex and very interconnected visuomotor network. It is a temporal dissociation that can allow dissociation of the two visual streams on the basis of functional rather than anatomical criteria (Rossetti and Pisella 2002). Parietal areas exhibit shorter response latencies to visual stimuli than ventral areas (fastconducting magnocellular dorsal pathway versus mainly

slow-conducting parvocellular ventral pathway), and the dorsal pathway has numerous shortcuts and is organized in a more parallel manner than the ventral stream (justifying the name proposed by Nowak and Bullier, 1997: the fast brain).

At the behavioral level, recent analyses of visuomotor performances of patients with bilateral optic ataxia have led to a distinct interpretation of optic ataxia and thus of the function of the dorsal stream in human. Pisella et al. (1999, 2000) and Gréa et al. (2002) have shown that a patient with bilateral optic ataxia shows a specific impairment of on-line visuomotor control rather than a global impairment of goal-directed actions (see Fig. 3). These results are discussed below together with those of other studies that indicate that visuomotor impairments in optic ataxia are restricted to specific experimental conditions in which fast and immediate actions are directed into the contralesional peripheral visual field. In addition, important clinical observations of the difficulties faced by patients with optic ataxia and visual agnosia in



Fig. 1 Overview of the visual-to-motor network. Cortical neuronal networks allowing visual inputs to be transformed into motor output: the possible substrates for dissociation and interactions between ventral and dorsal pathways driving information from primary visual cortex (V1) to primary motor cortex (M1). The dorsal and the ventral streams are depicted here in green and red, respectively, as well as their efferences. Blue arrows arise from areas receiving convergent dorsal and ventral inputs, either directly or indirectly. Further projections from areas receiving these mixed convergent inputs have also been represented in *blue*. Even though the posterior parietal cortex and the inferior temporal cortex receive a single direct projection from each other, they were not considered as mixed recipient areas. By contrast, areas in the frontal lobe receive parallel dorsal, ventral, and mixed projections. (AIP Anterior intraparietal area, BS brainstem, Cing. cingulate motor areas, d dorsal, FEF frontal eye field, FST floor of the superior temporal sulcus, Hipp. hippocampus, LIP lateral intraparietal area, MIP mesial intraparietal area, PIP posterior intraparietal area, MST medial superior temporal area, MT mediotemporal area, PF prefrontal cortex, PM premotor cortex, SC superior colliculus, SEF supplementary eye field, SMA supplementary motor area, STS superior temporal sulcus, STP superior temporal polysensory area, TE temporal area, TEO temporo-occipital area, v ventral, VIP ventral intraparietal area.) (Adapted from Rossetti et al. 2000-derived from Morel and Bullier 1990; Schwarz 1994; Schall et al. 1995; Tanné et al. 1995; Van Hoesen 1982)

everyday life will be considered. Altogether this review will shed new light on the interpretation of optic ataxia and leads us to reconsider and to modify the dominant theory about the functions of the dorsal and the ventral visual streams.

### Foveal vision versus peripheral vision

The concept of two visual streams was mostly developed in the late 1960s by Schneider (1969) and Trevarthen (1968; for previous accounts see Jeannerod and Rossetti 1993). It was based on the functional and phylogenetic distinction of two visual pathways processing visual information received on the retina. On the one hand, an older tectal pathway for ambient vision is involved in localization of stimuli in space and orientation behavior (see also Goodale 1983). On the other hand, a more recently evolved geniculostriate pathway for central vision participates in visual stimulus identification. The distinction between these two visual functions was initially interpreted as a subcortical versus cortical dissociation and then evolved toward the dissociation within the cortex of the dorsal versus ventral visual streams (Ungerleider and Mishkin 1982; reviews by: Goodale and Milner 1992; Jeannerod and Rossetti 1993; Milner and Goodale 1995; Pisella and Rossetti 2000; Rossetti and Pisella 2002). Since subcortical visual structures are now known to project onto cortical areas, the two types of interpretation are not necessarily mutually exclusive.

In the early writings about the ventral versus dorsal anatomical dissociation, specialization of the dorsal stream for peripheral vision and of the ventral stream for central vision was suggested and even emphasized (review by Paillard, 1982). In the context of motor control, the dissociation between the two streams was correlated with the functional segregation between position and movement, i.e., between static and kinetic visual cues. Consequently, they were supposed to process two kinds of error signals and to be involved in fast versus slow corrective visuomotor loops (Bard et al. 1985; review by Paillard, 1996).

In recent decades, the dorsal stream has been mostly considered as the vision-for-action (or How) system and it is rarely emphasized that most patients with optic ataxia are able to guide accurate actions toward objects in central vision. This pattern of result has, however, been confirmed by all studies which have compared movements aimed at central and peripheral targets in cases of pure optic ataxia. Out of the ten patients studied by Vighetto (1980; Perenin and Vighetto 1983, 1988), only three had a deficit in central vision. This pattern of results should not be neglected, because it is not as easy to explain as it appears. In patients with unilateral optic ataxia, this could still be interpreted in terms of visual fields: since central vision is analyzed by both hemispheres, the good performance at fixation could be attributed to the spared hemisphere, as is the case for the visual field following occipital lesions. However, patients with pure bilateral optic ataxia also

exhibit normal visuomotor performance in central vision in many experimental conditions involving either a pointing or a grasping response (Milner et al. 1999, Pisella et al. 2000, Gréa et al. 2002). Figure 2 depicts A.T.s pointing accuracy reported by Milner et al. (1999). The accuracy of this bilateral patient in central vision is close to that of control subjects. Figure 3 shows I.G.s grasping trajectories (from Gréa et al. 2002), which are not kinematically different from controls for stationary objects in free vision. In the case of primary visual areas, bilateral lesions are known to suppress the contralesional macular sparing that is observed with unilateral lesions, which demonstrates that central vision is processed in the two occipital lobes. But, in the case of optic ataxia, it is rather difficult to propose a satisfactory explanation of the sparing of visuomotor performance in central vision following a bilateral lesion of the PPC. To explain this simple fact, one has to argue that a different system has to be recruited for action in central vision, or at least that actions directed to centrally viewed objects must be processed through visuomotor channel(s) that bypass(es) the dorsal stream. As a consequence, the specific implication of the dorsal stream in action would be restricted to movements guided in peripheral vision. Under natural conditions, objects are most often first captured visually, since central vision allows them to be perceptually analyzed, identified, and selected, before it is decided to guide an action toward them. The only exceptions can be found in particular cases of familiar objects in a familiar environment. But it is reported in the literature that patients with optic ataxia exhibit far less visuomotor deficits when they reach and grasp familiar objects (Jeannerod et al. 1994; Milner et al. 2001). Given that in everyday life it is rather exceptional to direct actions toward unfamiliar objects that have never been seen in central vision, the claim that the dorsal stream is the main support to our actions clearly has to be reconsidered.



Fig. 2 Central versus peripheral vision. Pointing accuracy of a patient with a bilateral optic ataxia (A.T.) compared with normal performance. Mean pointing error is plotted (in millimeters) for various target positions (in degrees). As is seen in most patients with pure optic ataxia, pointing accuracy in central vision falls in the same range as normals. (Redrawn from Milner et al. 1999)

Fig. 3 Online correction deficit. When a grasping movement is directed to objects presented in free vision in a central (C) or right (R) location, a patient with a bilateral optic ataxia can reach accurately to and grasp them. However, she exhibits strong difficulties in updating her trajectories on-line in response to a target displacement from C to R (CR) triggered at movement onset. In this situation, artificially raising the need for online corrections, I.G. completed her initial movement before initiating a second movement to the final position of the object



#### Programming versus real-time visuomotor guidance

Since Perenin and Vighetto (1988), who argued that optic ataxia consists of a general impairment of visuomotor transformations, many arguments have raised the necessity to determine more precisely the exact visuomotor process that is impaired in these patients and thus the precise role of the human dorsal stream in goal-directed action. From a kinematic analysis of ataxic grasping movements, Jakobson et al. (1991) have proposed that the basic deficiency of optic ataxia is a programming deficit. In a more recent study, we tried to determine what processing specifically characterizes action guided toward peripheral targets and whether we could reveal a related impairment in central vision (Pisella et al. 2000). A patient with bilateral optic ataxia (I.G.) due to a bilateral PPC lesion was shown to be able to reach accurately to targets presented in free vision (not only for pointing, but also for grasping; Milner et al. 2001; Gréa et al. 2002). However, I.G. was strongly impaired in the same free-vision condition, when these targets or objects were experimentally displaced at the onset of her pointing or grasping movement (see Fig. 3). She could eventually reach the final target position only after completing her initially programmed movement and using slow and intentional corrective processes (Pisella et al. 1999, 2000; Gréa et al. 2002). In addition, the fast, unwilled corrections produced by normal subjects in response to the target jumps (i.e., by what has been called the hands automatic pilot by Pisella et al. 2000) were not observed in I.G. Results in normal subjects using transcranial magnetic stimulation also support the idea that the dorsal stream is necessary for on-line motor control (Desmurget et al. 1999). To summarize, converging results (Desmurget et al. 1999; Pisella et al. 2000; Gréa et al. 2002) now demonstrate the crucial role of the dorsal stream in real-time automatic adjustments but not in movement programming, nor in slow intentional motor control.

We will thus argue that a specific impairment in realtime motor control may explain the pattern of deficit observed in optic ataxia with respect to peripheral/central vision (Pisella et al. 2000) and with respect to closed-loop versus open-loop conditions. When motor programming is

realized on the basis of foveal visual information, on-line visuomotor guidance participates in goal-directed actions only for minor final adjustments. Conversely, when an action is programmed on the basis of imprecise peripheral visual information, on-line visuomotor control appears essential to quickly adjust the parameters of the ongoing action in order to succeed in reaching or grasping the goal (Pélisson et al. 1986; Goodale et al. 1986). Target jumps occurring at movement onset similarly engage early and important on-line corrective control. Such on-line processes have also been described for pointing responses to stationary targets in visual open-loop (i.e., without visual feedback) conditions (Prablanc et al. 1986; Pélisson et al. 1986; Goodale et al. 1986). Vighetto (1980) has tested three patients with unilateral optic ataxia in open-loop versus closed-loop pointing conditions. He has shown that lack of vision of the hand during movement execution dramatically affects pointing accuracy in these patients (see also Fig. 4). Vighetto (1980) has also reported that, in the closed-loop condition, patients spontaneously increase their movement duration. It could be suggested that slowing the movement allows patients to use slow feedback loops based on the visual reafference of hand position, which consequently improves the final pointing performance. Vighetto (1980) has also tested the effect of viewing the hand at starting position. Pointing accuracy is improved significantly in conditions where vision of the hands starting position is provided, probably again because this condition allows a better movement programming ad hence reduces the need for on-line corrective processes (Rossetti et al. 1994). Interestingly the view of Paillard (1996) leads to the same hypothesis, that the dorsal system is specialized for the fast corrective loop and would predict as well that its need increase when action is guided to peripheral targets. Indeed the dorsal system allows quick adjustment of movement trajectory based on the kinetic feedback of the moving arm seen in peripheral vision, which becomes more important during reaching to peripheral targets, since action is directed away from eye fixation.

It is interesting to reconsider the arguments found in the literature in favor of a programming deficit in optic ataxia in the light of these recent results. We will see that most



**Fig. 4** Open loop versus closed loop. Pointing accuracy of patient O.K. with unilateral optic ataxia following a lesion of the right posterior parietal cortex. This patient exhibits errors due to a combination of a hand effect and a field effect (see Revol et al. 2003). When pointing with the right (ipsilesional) hand, ataxic errors in the left visual field due to the field effect mainly expressed in terms of variable errors (not represented here). Lateral error is plotted here when pointing to targets at different eccentricities with the right hand in experimental conditions where visual feedback of the hand is available during movement execution (*Closed-Loop*) or not (*Open-Loop*). The open-loop condition reveals the deficit of the patient in terms of lateral errors in the left visual field (including central vision)

data interpreted as reflecting a programming deficit can also be seen as consequences of a deficit in real-time motor control. For example, the abnormally long deceleration period and large grip aperture described in optic ataxia patients (Jeannerod 1986; Jakobson et al. 1991) could be considered as compensation for the lack of online control processes, which are applied toward the end of the movement. Similarly, the increase in hand movement latency observed after a lesion of area 7 in monkeys (Faugier-Grimaud et al. 1985) and in man (Vighetto 1980; Jeannerod 1986; Jakobson et al. 1991) may be explained by the need to refine the programming of movements in order to compensate for the on-line control deficit. This is consistent with the recent evidence detailed below that delaying action can improve visuomotor performance in optic ataxia.

Altogether the above data provide strong evidence for a deficit of real-time visuomotor guidance, but the definitive demonstration of an additional programming deficit remains to be done. Recent results may indeed suggest that the early kinematics of reaching movements are impaired in some patients with optic ataxia (Roy et al. 2002; Milner et al. 2003). These results could be related to the impairment of reaching observed following parieto-occipital (PO) lesion in the monkey (Galletti et al. 2003). In the same way as it is specifically the fast (versus the slow), on-line motor corrections that are impaired in optic ataxia, it remains to be determined whether an impairment of visuomotor programming might only be observed when temporal constraints are specifically imposed on movement initiation (see Milner et al. 2003; and below).

#### Immediate versus delayed movement

The classical distinction between programming versus online control referred to in the previous section is currently being reconsidered, and these two aspects of motor control are progressively conceived as more and more interdependent (review by Desmurget et al., 1998; see also Figs. 4-15 in Rossetti and Pisella 2002). Accordingly, they might be alternatively described as a distinction between fast immediate visuomotor transformations and slow immediate visuomotor transformations (Rossetti and Pisella 2003). As mentioned earlier, the microscopic and functional dissociation between the two main visual streams are more important to consider than only their relative anatomical segregation. This functional dissociation is mainly based on the temporal properties of transfer of visual information along the two streams (Nowak and Bullier 1997). These properties allow the prediction that, unlike healthy controls, patients with lesion of the PPC would perform delayed action better than immediate action and that the residual visuomotor abilities of patients with visual agnosia should be dramatically affected by a memory delay. Both of these predictions have been supported by neuropsychological observations. Milner et al. (1999) have shown that patients with bilateral optic ataxia are more accurate in delayed pointing than in immediate pointing to peripheral targets. The same paradoxical improvement of visuomotor performance in these patients (contrary to what happens with delay in normal subjects) was also observed for the grasp component (Milner et al. 2001, 2003). Note that these results strongly challenge the view that the integrity of the dorsal stream is necessary for performing actions. They suggest that the impairment found in the peripheral visual field of patients with optic ataxia is specific to fast visuomotor processes, as already suggested by Pisella et al. (2000). Interestingly this paradoxical pattern of results is clearly dissociated from the disruption of visuomotor functions found in visual agnosia in delayed tasks (Goodale et al. 1994). This clear dissociation could be interpreted as the most demonstrative argument for a double dissociation between visual agnosia and optic ataxia (see Milner et al. 2003). However, the visuomotor performance of the two types of patients have not yet been tested in similar conditions, especially with respect to the distinction between central and peripheral vision.

If one considers the ecological usefulness of delayed sensorimotor reactions, then another pattern of dissociation may be proposed. In many areas of life within a social environment, it may be more productive not to react immediately to attractive stimuli. Therefore the immediate reaction systems have to be supervised by more complex levels of cognition. That is exactly what patients with environment-dependency syndrome (Lhermitte 1986) appear to lack. This syndrome results in the release of immediate reactions to visual stimuli and is consecutive to frontal lesion. Therefore the issue of immediate versus delayed action suggests that optic ataxia might be better dissociated from patients with frontal lesions than with lesion of the ventral stream (see Rossetti and Pisella 2003). In this framework, optic ataxia would be considered as a specific deficit for immediate (i.e., nonmediated) visuo-motor responses.

#### **Everyday life versus experimental conditions**

Action, in contrast to movement or behavior, refers to the ability to produce a deliberate sequence of motor activation that produces a desired effect on the body-environment relationship. Therefore action should be associated with intention (see Revonsuo and Rossetti 2000), and immediate motor control can be regarded as a local function of the whole system (Fig. 5). This definition of action is to be contrasted with its frequent use in neuroscience and experimental psychology. The frequent association of voluntary with action should be regarded as a pleonasm, but it also results from the overuse of action terminology. Action is broadly used whenever a motor response is required from the subject. However, it is rather obvious that simple key-pressing tasks used in most laboratories cannot be considered as action. The only use of reaction time measurements in such experiment reflects the fact that reactions rather than actions are processed in these tasks. Most experimental conditions designed to explore voluntary actions actually investigate reactions to an expected stimulus. These reactions are most often overlearned in everyday life (e.g., pointing or grasping, key-pressing) or are investigated after a learning session in the case of more complex tasks. Therefore the intention of the subject may be restricted to the will to perform the task or to comply to the instructions. Clinical observation and examination may provide a less constrained way to explore intentional aspects of behavior.

The most important and relevant aspects to mention about everyday consequences of neurological deficit are the spontaneous complaints from patients with optic ataxia and visual agnosia. Most of the patients with unilateral optic ataxia do not complain of any action deficit in everyday life. In the group study by Vighetto and Perenin (1981), half of the patients had never complained of functional deficit before the systematic search for movement guidance in peripheral vision, and the other half of the patients included cases with other symptoms such as mild sensory deficit or praxic problems. Patients with a bilateral lesion often complain about functional deficits, from which it is difficult to disentangle the impact of pure optic ataxia and that of symptoms due to other parietal or more remote damage. This is evident in the case of Balint syndrome.

Reports about the functional deficit of patients with optic ataxia in everyday life are usually brief. Little mention of functional deficit has been provided even for patients with a bilateral lesion (e.g., Milner et al. 1999, 2001; Pisella et al. 2000). Jeannerod et al. (1994) described in more detail the case of the bilateral patient A.T.:



Fig. 5 Visually guided action versus immediate visuomotor control. Action refers to the ability to produce a deliberate sequence of motor activation. Therefore action should be associated with both intention and perceptual input, both factors contributing to initial decision process and to the planning of action. Immediate motor control can thus be regarded as local regulation of the whole system

Visual recognition of shape, texture, depth, colour, faces had always been normal. AT never complained of any difficulty concerning perception of movement....Despite some recovery with the passing years of rehabilitation programs, AT still presented a severe disorientation. Indeed she was hampered in her everyday life for actions like dressing, cooking, ironing, sewing or drivingHowever, it is not clear whether the whole deficit exhibited by A.T. can be accounted for by her optic ataxia, since she also exhibited several problems with more perceptual functions (which is also compatible with her rather large lesion; see Michel and Hénaff, in press). The observation made by Jeannerod et al. (1994) was that A.T. was mostly impaired in peripheral vision when she had to grasp unfamiliar objects. It was later shown that another bilateral optic ataxia patient (I.G.) could learn to size her grip to unfamiliar objects across several experimental sessions (Milner et al. 2001). These two observations show that the grasping deficit observed in peripheral vision should not be related to a gross visuomotor deficit but rather to a deficit in determining the grip size appropriate to unfamiliar objects, i.e., when only metric cues are available in the visual environment.

In our clinical experience, patients such as I.G. with bilateral lesions (Pisella et al. 2000) may, for example, exhibit difficulties in taking the first step on a staircase. They also have problems navigating in a complex dynamic environment (e.g., a train station), in that they have to stop repeatedly and slow down their walking speed considerably. One other typical complaint of bilateral patients is of a slowness and clumsiness in writing. I.G.s deficit thus appears to be mostly confined to situations where she has to react to visual information in real time or when facing a complex environment.

In contrast, patients with visual agnosia spontaneously complain of huge difficulties in everyday life. They cannot find familiar objects or select the appropriate tool for daily tasks. They have to rely on specific cues such as context, touch, and noise, and strategically put objects in fixed, predetermined places so that they can find them without recognizing them.

The patients difficulties are so obvious that descriptions often focus on the few residual functions that patients can perform in everyday life. The famous case D.F., who was initially described by Milner and Heywood (1989), had a constructional apraxia. However, she could manage some everyday activities such as shopping and cooking by 2 years postonset (Milner et al. 1991). As in most patients with visual agnosia, one ultimate consequence of the deficit was a loss of spontaneity in her behaviour (Milner et al. 1991, p. 410). A more recent description by Lê et al. (2002) emphasized the fact that their patient (S.B.) could move in space without apparent difficulties and grasp moving objects (table tennis balls), i.e., when no goal selection was involved. But, for example, S.B. could not even choose his food at the cafeteria, as is typically observed in other patients with visual agnosia. Therefore the cluster of residual functions maintained in visual agnosia appears to be mostly related to intact, on-line motion-processing abilities and their use in controlling movement (e.g., Lê et al. 2002).

Taken altogether these clinical considerations allow us to draw the conclusion that, for everyday-life actions, patients with visual agnosia are impaired at least as much as patients with optic ataxia. Of course this does not contradict the fact that under experimental conditions where so-called actions are limited to reactions to a visual target or object—presented in peripheral vision—patients with optic ataxia are more impaired than patients with visual agnosia. But one has to be aware that it is an obvious constraint of these experimental conditions that they emphasize the specific need for on-line visuomotor guidance whereas the contribution of other aspects of action control is artificially reduced.

As mentioned earlier, another line of thought has been to propose a contrast between optic ataxia and the environment-dependency syndrome observed following frontal lesions (Lhermitte 1986; Pisella et al. 2000; Rossetti and Pisella 2003). This loss of autonomy is observed both for social stimuli (eliciting imitation behavior) and for physical stimuli (eliciting utilization behavior; Lhermitte et al. 1986). Rather, the patients behavior appears as though implicit in the environment was an order to respond to the situation in which the patients find themselves (Lhermitte 1986). In other words these patients appear to be reacting immediately to external stimuli without being able to shift their action in time or in space, which may appear to be the opposite

pattern to that described in optic ataxia. As already proposed by Pisella et al. (2000), Frith et al. (2000), and Rossetti and Pisella (2003), it may be that optic ataxia can be better dissociated from this environmental-dependency syndrome than from visual agnosia.

# Discussion

We have reviewed several axes along which dissociations between optic ataxia and visual agnosia have been proposed, and arguments for a dissociation between perception and action have been made. The issues of foveal versus peripheral vision, the temporal constraints of visual information processing, and the necessary reductionism of experimental conditions used to study action are the key factors that should be kept in mind before arguing for a double dissociation between optic ataxia and visual agnosia, as well as between vision for action and vision for perception. It is also obvious that many visual streams can contribute to action, none of them disrupting all types of action. This is compatible with the variety and the complexity of projection streams that can be described between the visual input and the visual output (Rossetti and Pisella 2002).

In contrast to local sensorimotor processes, a true action involves many aspects of perception. Complex actions imply a continuous perceptual control of the movement sequence and of possible side effects. Simple actions imply that an appropriate goal is selected in a rich environment. In this framework the postulated double dissociation between optic ataxia and visual agnosia can obviously not be supported by a simple distinction between global perception and global action. The former idea of a double dissociation between perception and action was based on the assumption that all visual perception was altered in visual agnosia (which is mostly the case in at least D.F.) but also considered optic ataxia as being an action disorder. More recent studies have shown that the aspects of action that are impaired in optic ataxia are quite restricted. Optic ataxia patients can perform all types of natural actions and are impaired only when a time constraint is imposed: their on-line motor updating is altered and their reaches toward peripheral targets are impaired when they have to produce immediate responses toward unknown objects. The strongest argument for a dissociation between optic ataxia and visual agnosia is now grounded on temporal parameters. Our main argument here is that optic ataxia does not appear as a general deficit of action but rather as a specific deficit localized at a restricted level of action organization that is immediate visuomotor control. A crucial role appears to be played by real-time visual processing of both the goal and the arm for this limited aspect of action that is impaired in optic ataxia. Several higher levels of action organization can be affected by other syndromes such as apraxias, unilateral neglect or frontal syndromes, resulting from the unilateral lesion of functions with high hemispheric lateralization. In contrast to optic ataxia, affecting a low-level of the interface between visual input and motor output, these cognitive disorders often produce deficits for both hands and in both hemifields.

Three clear lines of investigation emerge from the present review. First, to build a strong argument for a double dissociation between optic ataxia and visual agnosia, further studies should either test visuomotor performance of optic ataxia patients in central vision (as is done for visual agnosia) or study visuomotor performance of visual agnosia patients in peripheral vision (as is done for optic ataxia). The visuomotor performance of patients with visual agnosia are never explored in peripheral vision as is the case in most reports about optic ataxia, and the perceptual performance of optic ataxia patients have not been described in peripheral vision to allow comparison with their motor impairment. A second crucial area of investigation should explore the important role of time in the dissociations found within the visual system. This area of research is currently growing fast. The third line of investigation concerns the cluster of characteristics that are impaired in optic ataxia: it would be interesting for future studies to investigate the functional links between fast, online visual processing, immediate visuomotor control, and peripheral vision. One possible link could be that these situations are mainly realized using automatic control processes.

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