Optic ataxia is not only 'optic': Impaired spatial integration of proprioceptive information

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Optic ataxia is considered to be a specific visuo-manual guidance deficit, which combines pointing errors due to the use of the contralesional hand ("hand effect") and to the presentation of the visual target in the contralesional field ("field effect"). The nature of the hand effect has not been identified. The field effect is acknowledged as an impaired spatial integration of visual target location. However, spatial integration of proprioceptive information from the arm has never been experimentally tested in these patients. Here, we specifically investigated the capacity of two patients with unilateral optic ataxia in tasks requiring different levels of proprioceptive integration from primary information processing to proprioceptivo-motor integration. In a first experiment -proprioceptive pointing with the ipsilesional hand toward the index finger of the contralesional hand- revealed a large mislocalisation of the ataxic hand accounting for the hand effect. In a second experiment -proprioceptive pointing with the ataxic arm toward the finger of the ipsilesional hand- revealed reaching errors for non-visual targets, i.e. optic ataxia is not specific to 'optic' targets. Altogether, the present results call for a redefinition of this neurological condition in the framework of parietal functions.
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Introduction

Following a lesion of the dorsal part of the posterior parietal cortex (PPC), optic ataxia (OA) is classically defined by manual misreaching errors specifically for visual (optic) targets, in the absence of primary sensory or motor deficit. It is therefore considered as a human model of the PPC involvement in visuo-manual reach guidance. As revealed by its name, *optic* ataxia has constantly been studied with the aim of dissociating the dorsal and ventral streams in visual information processing (Jeannerod and Rossetti, 1993; Milner and Goodale, 1995; Holmes, 1918; Ratcliff and Davies-Jones, 1972; Pisella et al., 2000; Khan et al., 2005) as a visuo-motor deficit, i.e. as an impaired spatio-motor integration of visual targets locations.

However, the PPC, including the reach-related areas (Calton et al., 2002; Galletti et al., 2003; Medendorp et al., 2005), is known to be a multisensory region located between the visual (occipital) and the proprioceptive (anterior parietal cortex) primary cortices. It receives visual and proprioceptive information in such a way that an antero-posterior somatic-to-visual gradient can be described with reciprocal connections with the frontal cortex that match this functional organisation (Battaglia-Mayer et al., 1998; Burnod et al., 1999). Moreover, it has been shown that the PPC also integrates information about the position of the hand to plan the movement (Medendorp et al., 2005). Therefore, the study of OA as a model of the parietal implication in visuo-motor transformation could consist in studying visuo-proprio-motor integration. Here, we specifically investigated the capacity of two patients with unilateral optic ataxia in tasks requiring different levels of proprioceptive integration from primary information processing to proprioceptivo-motor integration.

Patients with unilateral lesion exhibit pointing errors linked to the presentation of a visual target in their contralesional visual field (field effect) and to the use of their contralesional (ataxic) hand to perform the movement (hand effect) (Perenin and Vighetto, 1988). The visual field effect (Holmes, 1918; Ratcliff and Davies-Jones, 1972; Khan et al., 2005) has been explored much more than the hand effect (Perenin and Vighetto, 1988). Accordingly, it is often highlighted that the misreaching errors are restricted to peripheral vision and to the contralesional visual field. However, the few experiments in which patients were tested for visual pointing in the absence of visual feedback of the hand have shown an increase of errors and especially their appearance in central vision (Levine et al., 1978; Brouchon et al., 1986; Jeannerod, 1986, Vighetto, 1980). In the first experiment, we assessed the nature of the hand effect by first testing the prediction that this increase in pointing errors in the dark is specifically related to the use of the
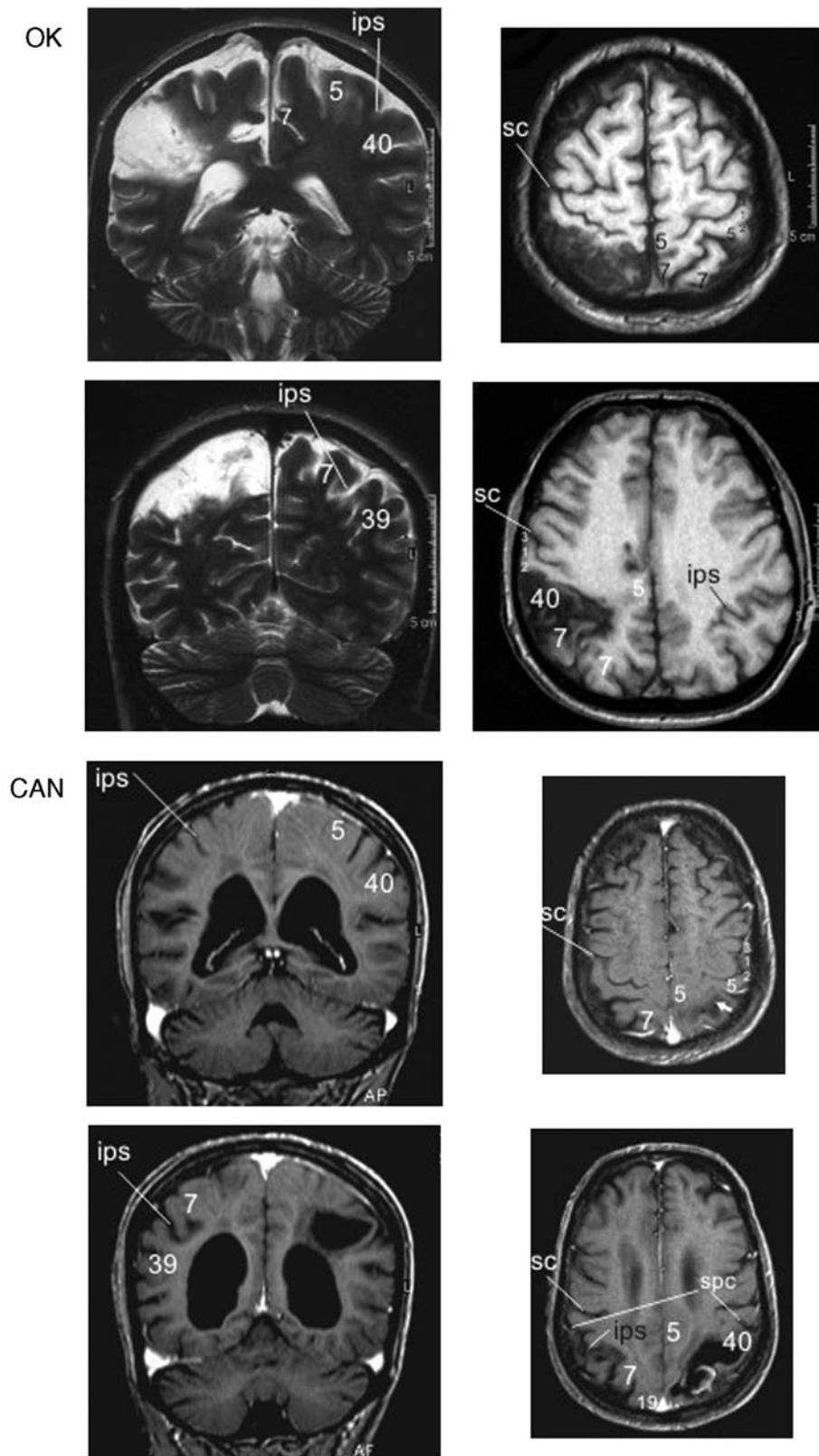
Fig. 1. MRI scans for patient OK and patient CAN. Frontal and horizontal sections of T1 MRI scans for the two patients, except the frontal views of patient OK (T2). Left hemisphere is presented on the right of the MRI image and vice versa. When the lesion site is ambiguous it is indicated by a white arrow. SC: central sulcus, ips: intraparietal sulcus, spc: post-central sulcus, numbers indicate Brodmann's areas (BA). *Patient OK right PPC damage*: BA 7 in its lateral and medial aspects with a slight extension into BA 39, 40 and 5. *Patient CAN left PPC damage*: lateral aspect of BA 7, slight extension in the deep part of BA 39 and 40.

contralesional hand. No quantitative study of the field and the hand effect has been done in this open loop condition. Secondly, a proprioceptive pointing task of -pointing toward the contralesional hand in the dark- with the ipsilesional hand specifically tested the hypothesis of a deficit in extracting the spatial location of the ataxic hand from multi-joint proprioceptive information.

The classical clinical examination of OA patients' ability to realise a movement to a proprioceptive target, consisting of grabbing their thumb or pointing their nose, claims for the absence of any deficit. Since the PPC is known to be a multisensory region integrating not only visual information, but also other sensory modalities, mainly somatosensory, this statement is surprising. However, this clinical test, performed with eyes closed in personal space, is very different from the visual pointing task in which patients maintain fixation and produce movements in the peri-personal space. In order to check for the preservation of proprioceptive pointing capacity of OA patients, we judged it necessary to test them in a condition comparable to the one that reveals their visual pointing deficit (pointing toward visual targets in the dark, see Fig. 2). In a second experiment, we therefore performed a proprioceptive pointing task of -pointing toward the ipsilesional hand in the dark- with the ataxic hand to verify whether, contradictory with the very definition of *optic* ataxia, misreaching errors can be observed for non-visual targets.

Methods

Participants

Six neurologically healthy controls (age range=25–60, M=38) and two patients with unilateral optic ataxia gave their informed consent to take part in the study. Patient OK is a right-handed 39-year-old male patient with right PPC damage (Brodmann's area 7 in its lateral and medial aspects with a slight extension into areas 39, 40 and 5 and into the right posterior corpus callosum) extended toward the parieto-occipital junction, caused by an ischaemic stroke involving the posterior branch of the right sylvian artery. Patient CAN is a right-handed 76-year-old male patient who suffered from left PPC damage (lateral aspect of BA 7, slight extension in the deep part of areas 39 and 40) extended toward the parieto-occipital junction, caused by a hemorrhagic stroke. The lesions of both patients include the areas that have been recently shown to be critical for optic ataxia (Karnath and Perenin, 2005). Clinical evaluation controlled for the absence of any purely motor, somatosensory or visual deficits and of any sign of neglect. The two patients spontaneously expressed no complaint that could concern the proprioceptive function. The clinical evaluation of the static and dynamic proprioception of the upper limbs consisted of applying a slow passive movement in flexion or extension (the test included 25% catch trials) on each joint serially (index, wrist, elbow, shoulder), whilst patients kept their eyes closed. We asked them 1) whether they perceived a movement, 2) in which direction and 3) to reproduce the single joint angles with the other limb (Rivermead Assessment of Somatosensory Performance (RASP) subtests). Coherent with the sparing of the anterior parietal cortex in the two patients (Fig. 1), no primary proprioceptive deficit was revealed for either limb. Coherent with the large sparing of Brodmann area 5 (Fig. 1) and contrary to the patient of Wolpert et al. (1998), neither of the two patients presented any symptoms of fading somatosensory sensation or of fading grip force, nor any specific inaccuracy for slow movements. As usually observed in

optic ataxia, patient OK has been instead shown to be more accurate with delayed movements than with immediate ones (Revol et al., 2003).

Data acquisition

In this study, subjects and patients underwent two main pointing paradigms with central eye fixation in the dark: a classical visual pointing paradigm (pointing toward peripheral visual targets) and a proprioceptive pointing paradigm (pointing toward peripheral proprioceptive targets) performed either with the ipsilesional hand or with the ataxic hand. Patient CAN additionally performed the visual pointing paradigm in the light to assess the effect of providing the visual feedback of the pointing hand location on the reaching errors. Patient OK was additionally tested with two extra gaze fixation positions in one condition of the proprioceptive pointing task (pointing toward the ataxic hand/with two visual fixations).

In both paradigms, subjects were sitting in the dark, facing a vertical frontal pointing screen at 25 cm from their eyes and fixating on a phosphorescent dot at the centre of the screen. Eye movements were monitored using an infrared camera. The pointing movements were recorded by the VICON system (3D trajectories recording system) from which the final positions were extracted and compared with the target position to obtain pointing errors in the screen plane (2D).

Pointing toward peripheral visual (V) and proprioceptive (P) targets were performed in blocked sessions with the left (L) or right (R) hand within an ABBA design (order: P-L V-R P-R V-L). Three pointing movements were performed per target presented in random order toward 5 target locations per hemifield. Target positions with respect to the fixation were as follows: (x=12.5 cm, y=0 or 32° of visual angle), (x=12.5 cm, y=±7.5 cm or 36°) and (x=19.5 cm, y=±7.5 cm or 47°).

Pointing hand starting position was always at the right bottom corner of the screen for the right hand and left bottom for the left hand. Subjects and patients were asked to point toward the target as quickly and as precisely as possible. In the visual pointing paradigm, the pointing movements were performed toward small luminous dots made from a laser (see cartoon in Figs. 2A and B). In the proprioceptive pointing paradigm, subjects pointed with one index finger on one side of the vertical screen toward the other hand's index fingertip, which was passively positioned on the other side by the experimenter. The target hand was contralesional in the first proprioceptive pointing condition (see cartoon in Figs. 3A and B) and ipsilesional in the second proprioceptive pointing condition (see cartoon in Fig. 4).

For the condition of proprioceptive pointing toward the ataxic hand/with two visual fixations (fixation up: 15 cm from central fixation=+31° and fixation down: −15 cm from central fixation= −31°; see cartoon in Fig. 3B) testing the influence of the eye position on patient OK, 3 proprioceptive target positions instead of 5 were used per hemifield (those at 47° and 32°) but were presented 6 times.

Data analysis

Absolute pointing errors were measured as the difference between the target position and the patients' and subjects' end point. Because of a lack of homogeneity between the variances of the pointing errors, non-parametric statistical tests (Mann–Whitney U test) were used to compare each patient with the control
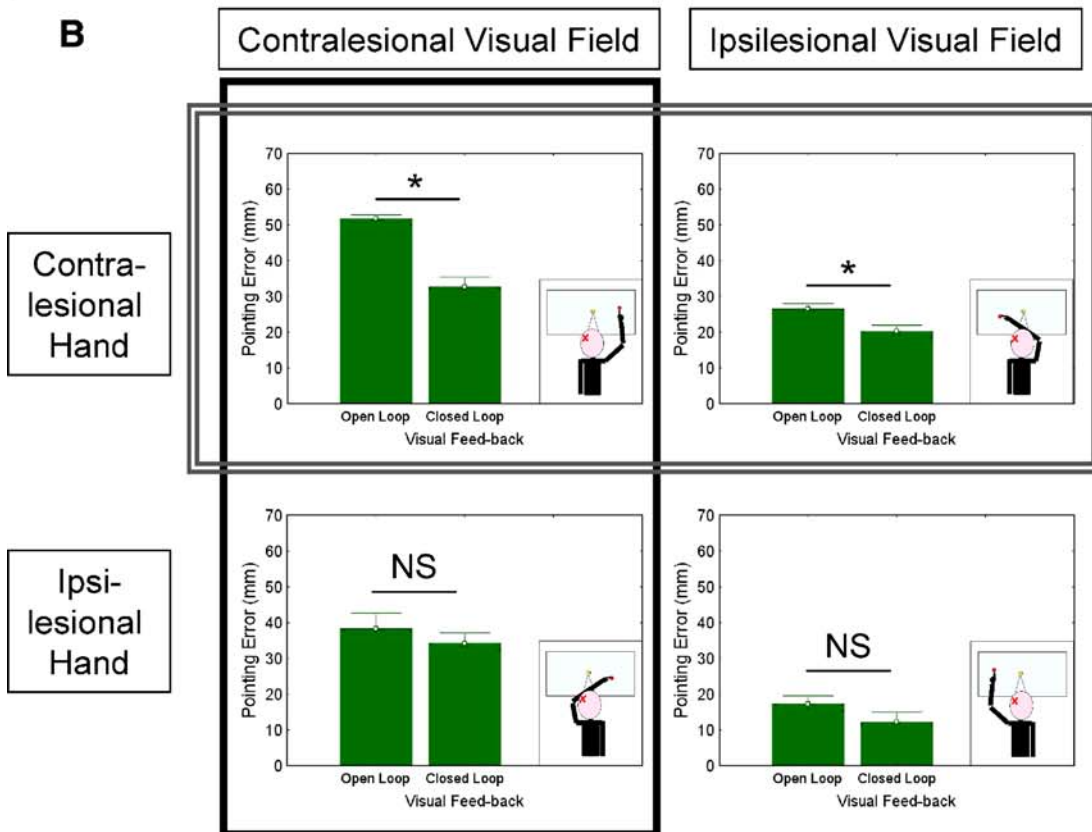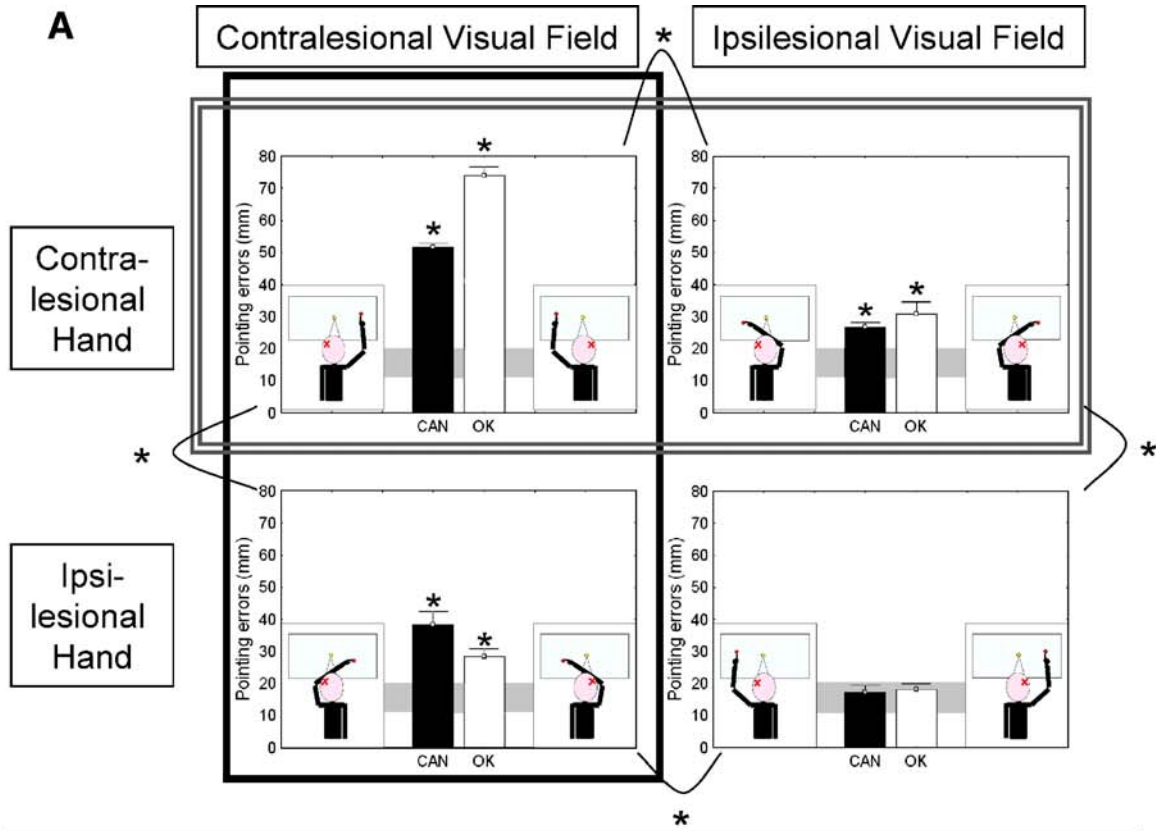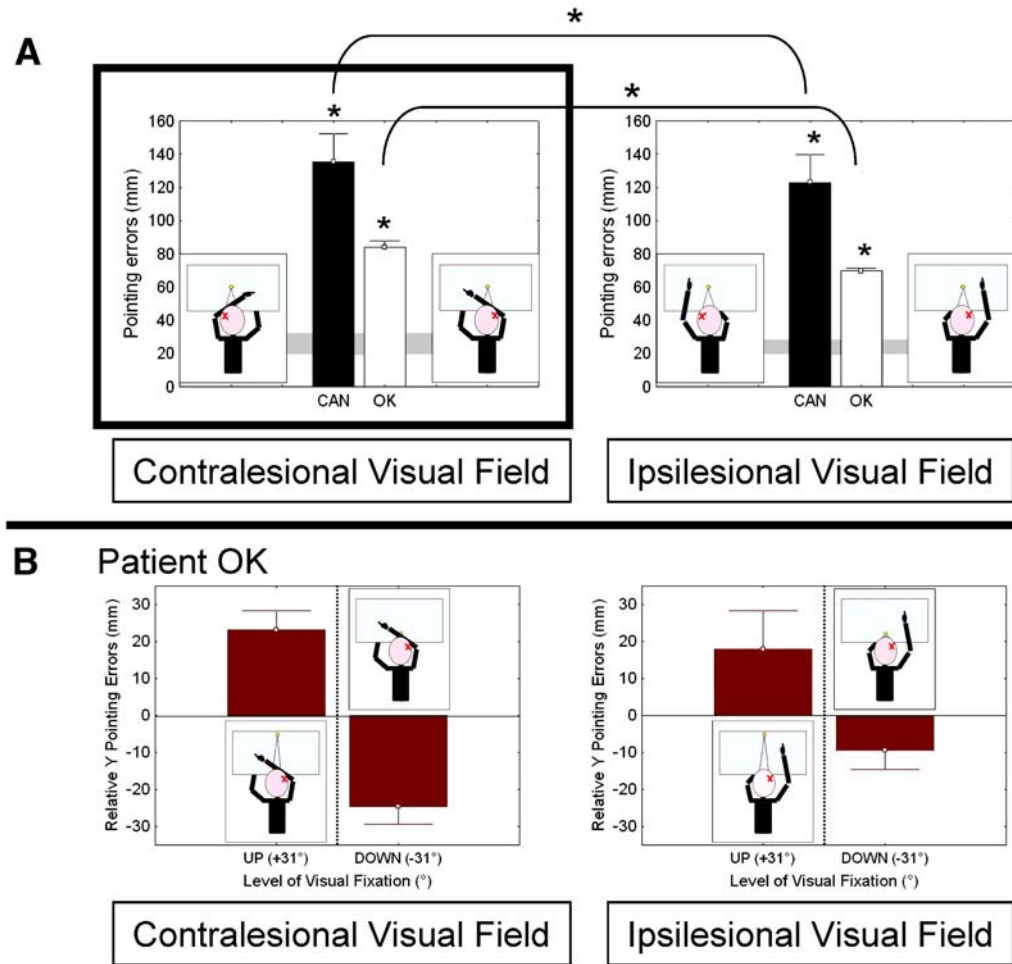
Fig. 3. Pointing to proprioceptive targets with the ipsilesional hand. (A) Pointing toward the ataxic hand: central fixation. Columns represent the means and standard deviations of the end points errors for the two patients when they pointed to their contralesional hand with their ipsilesional hand in the two hemifields. The grey zone corresponds to the range of pointing errors made by control subjects (mean plus/minus standard deviation). The stars indicate significant differences between visual fields for each patient and for each condition compared to controls. (B) Pointing toward the ataxic hand: with fixation up or down. Columns represent the means and standard deviations of the relative (to central fixation) vertical errors for patient OK when he pointed with his ipsilesional hand toward his contralesional hand passively positioned in the two hemifields with two different eye positions (up: 15 cm=+31° and down: −15 cm=−31°).

performance as well as within patient performance. We adjusted the Z and p values in regard to small sample statistics.

Results

Experiment 1: identifying the nature of the hand effect

In addition to the classical clinical diagnosis, the visuo-manual deficits of both patients were first evaluated by a task of -pointing toward peripheral visual targets- in the dark in the left and right visual fields and using both the left and right hand. Both patients

exhibited comparable patterns of pointing errors (Fig. 2A) that combined field and hand effects (Perenin and Vighetto, 1988), such that pointing with the contralesional hand in the contralesional visual field produced the largest absolute errors (5.2 cm for CAN and 7.5 cm for OK on average, compared with the mean pointing accuracy of the control group: 1.7 cm). Conversely, pointing with the ipsilesional hand in the ipsilesional field resulted in negligible errors (1.7 cm for CAN and 1.8 cm for OK on average). The two crossed combinations showed in intermediate performance. Except for the condition in which the ipsilesional hand pointed toward the ipsilesional hemifield (Z adjusted>0.6

Fig. 2. Pointing to visual targets. (A) In the dark—field and hand effects. Columns represent the means and standard deviations of the end points errors for each four combinations of hemifield and pointing hand for the two patients. The grey zone corresponds to the range of pointing errors committed by control subjects (mean plus standard deviation). The stars indicate significant differences between conditions (hand and field) for each patient and for each condition compared to controls. (B) In the light—effect of visual feedback. Columns represent the means and standard deviations of the end points errors when pointing in the dark and in the light for each four combinations of hemifield and pointing hand for patient CAN. The data from the dark condition are the same as already illustrated in panel A. The stars indicate significant differences between the dark and light pointing task performances.

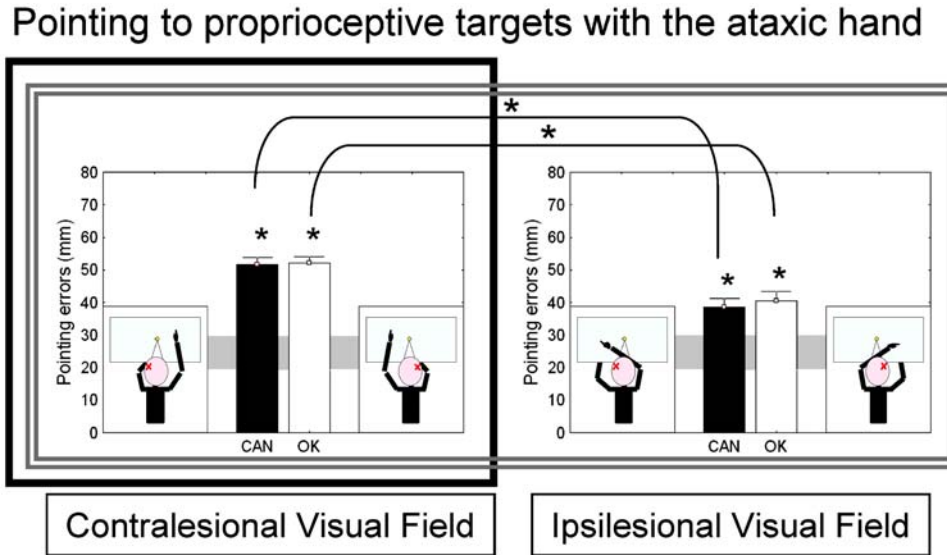## Pointing to proprioceptive targets with the ataxic hand

Fig. 4. Pointing to proprioceptive targets with ataxic hand. Columns represent the means and standard deviations of the end points errors for the two patients when they pointed toward their ipsilesional hand with their contralesional hand in the two hemifields. The stars indicate significant differences between visual fields for each patient and for each condition compared to controls. The grey zone corresponds to the range of pointing errors committed by control subjects (mean plus standard deviation).

and $p > 0.2$ for both patients), the mean pointing errors were significantly larger than the standard deviations of the control group represented by the grey rectangles (Z adjusted < -2.35 and $p < 0.05$ in the three conditions and for both patients). These mirrored deficits are likely explained by the extensive unilateral lesion of BA 7 in the right hemisphere for patient OK and in the left hemisphere for patient CAN (Fig. 2A).

Patient CAN also performed the same four conditions of -pointing toward peripheral visual targets in the light- allowing him to see his pointing hand (visual feedback). Pointing errors significantly decreased in the light (with respect to the dark) when movements were performed with the contralesional hand (in both hemifields: $Zs = 2.61$; $ps < 0.01$) but not with the ipsilesional hand ($Zs < 0.95$; $ps > 0.05$). This set of results (Fig. 2B) shows that pointing in the dark appeared to mainly increase the errors made with the contralesional hand, i.e. the 'hand effect'.

Searching for the underlying mechanisms of the hand effect would therefore consist in asking what is specific to visuo-manual reaching in the dark: the visuo-motor transformation has to rely on the felt position of the arm. Since the patients have no primary proprioceptive deficit, one can hypothesise that the hand effect more specifically corresponds to impairment in extracting the spatial position of the contralesional hand from the multi-jointed proprioceptive information. In order to test this precise proprio-ceptivo-spatial transformation, we designed a task of -pointing toward the ataxic hand in the dark- in which the fingertip of the contralesional (ataxic) hand was the proprioceptive target (passively positioned by the experimenter the vertical screen: see Methods). Large absolute pointing errors (11 cm away from the target in the contralesional field and 10 cm in the ipsilesional field on average, 2.6 cm in average for controls) were observed in the two patients. These errors were much larger for the OA patients than for the control subjects ($Zs > 2.80$; $ps < 0.005$ for both patients) and clearly revealed a deficit in localising the felt position of the contralesional (ataxic) hand.

Patients' errors were larger when the target hand was located in the contralesional than in the ipsilesional hemifield ($Zs = 2.19$; $ps < 0.05$ for both patients). Even with smaller amplitudes, the difference found here between the two hemifields for unseen proprioceptive targets is reminiscent of the field effect observed for visual targets (illustrated in Fig. 2A), and recently shown to depend on an eye-centred representation of the position of the visual target (Khan et al., 2005). We further explored this aspect by asking patient OK to perform an additional task of -pointing toward the ataxic hand in the dark/with visual fixation up or down- i.e. two different vertical eye positions (up: 15 cm from central fixation = $+31°$ and down: -15 cm from central fixation = $-31°$). If the eye-centred reference frame (demonstrated for visual targets) is also used in the visuo-proprioceptive integration of information, then pointing errors should be modified with respect to eye position. This was confirmed by the results illustrated in Fig. 3B. An obvious modification of the y-direction of the error vectors across both hemifields was observed when the position of fixation varied from the top to the bottom of the screen (Fig. 3B; $Z = 2.88$; $p < 0.01$).

We designed a control task directly comparable to our pro-prioceptive pointing task. The two patients were asked to reproduce with one arm the multi-joint postural configuration of the other, which was passively positioned by the investigator in postures similar to those used in the proprioceptive pointing task. This control task was correctly performed by the two patients.

Experiment 2: revealing a multimodal field effect

The latter results suggested that the hand effect corresponds to a deficit in extracting the spatial position of the ataxic hand from proprioceptive information of arm joints. Misreaching errors due to this hand effect would then be observed for pointing toward proprioceptive targets as well as visual ones in OA patients. To verify this prediction contradictory to the definition itself of *optic* ataxia that excludes any misreaching toward targets in other modalities than visual, we performed a second proprioceptive

pointing task of -pointing toward the ipsilesional hand in the dark-with the ataxic hand (see Fig. 4). Significantly large pointing errors (compared to controls) were observed for both patients whenever the ipsilesional target hand was positioned in either hemifield (Mann–Whitney U test: all adjusted Zs > 2.80; all ps < 0.005 for both patients). In addition, the task revealed a field effect for non-visual targets ($Z = 2.19$; $p < 0.05$ for both patients). These results seem to suggest a similar pattern when the ataxic hand seems to follow the same error pattern for proprioceptive targets as is classically observed for visual targets: a combination of a hand effect and a field effect (however less important than is visual pointing task) as well as an influence of the gaze position on pointing errors.

Discussion

Optic ataxia is considered to be a human model of the PPC involvement in visuo-manual reach guidance. When pointing to peripheral visual targets, unilateral optic ataxia patients exhibit a misreaching pattern which combines pointing errors linked to the presentation of a visual target in their contralesional visual field (field effect) and to the use of their contralesional (ataxic) hand (hand effect) (Perenin and Vighetto, 1988). Coherent with the study of optic ataxia as the typical consequence of the damage of the visual dorsal stream (in contrast to visual agnosia, a typical consequence of the damage of the visual ventral stream; see Milner and Goodale, 1995; Pisella et al., 2006), only the visual field effect has been identified and largely documented (Ratcliff and Davies-Jones, 1972; Khan et al., 2005; Prado et al., 2005). Rather, the PPC can be viewed as a visuo-proprioceptive interface (Burnod et al., 1999) as well as a visuo-motor interface (Perenin and Vighetto, 1988), the aim of this study was to investigate the spatial integration of proprioceptive information in optic ataxia in order to identify the nature of the hand effect (experiment 1) and to verify the specificity of the misreaching for visual (optic) targets (experiment 2).

Studies that investigated optic ataxia patients' pointing performance in the dark tended to show an increase of errors in this condition. This could be attributed to a general increase of the task difficulty but our -pointing toward peripheral visual targets in the dark versus in the light- task allows us to conclude that the absence of visual feedback of the pointing hand specifically affects pointing movements performed with the ataxic hand. This result is the first step in proposing the hypothesis of a high-level proprioceptive integration deficit underlying the hand effect as we know that without the vision of the pointing hand, the visuo-manual transformation has to rely more on its proprioceptive localisation.

In the -pointing toward the ataxic hand- task, we therefore asked patients to point toward their contralesional hand. Surprisingly large mislocalisation errors were observed (11 cm on average). Another surprising result was the increase of mislocalisation errors when the ataxic hand was positioned in the contralesional field. This "field effect" confirmed that the deficit has no primary origin and rather corresponds to a mislocalisation of the fingertip of the ataxic hand in peri-personal space from proprioceptive information, probably in an eye-centred reference frame. This possible extraction of eye-centred location from proprioceptive information is reinforced by the results of the -pointing toward the ataxic hand in the dark/with visual fixation up or down- task showing a strong modulation of pointing errors with

shifts of eye position. The same observation has been reported in proprioceptive pointing tasks performed by normal subjects (Blangero et al., 2005; Pouget et al., 2002).

This result cannot be attributed to a primary proprioceptive deficit excluded from the diagnosis of optic ataxia by definition during clinical examination and from the proprioceptive subtests of the RASP battery. Moreover, the two patients performed well in the posture reproduction test, which excludes a basic deficit in the perception of multi-joint configuration. Superior parietal lobule lesions in the macaque have been shown to cause deficits in a proprioceptive reaching task (Rushworth et al., 1997a) and again the deficits seem to be spatial in nature rather than due to an inability to reproduce a particular configuration of joint angles (Rushworth et al., 1997b). A patient with primary somatosensory deficit would have failed to report passive joint angle movement in the clinical evaluation and would have also failed to reproduce the posture. In the proprioceptive pointing task, we would have seen large errors, but probably no field effect associated. This pattern of pointing errors is also different from the other high-level parietal deficits known. A patient with profound apraxia may show deficit in the posture reproduction but not in the pointing task. Astereognosia, which is also a high level somatosensory deficit, could have induced errors but again, with no field effect. This effect is a key point to conclude to a spatial integration deficit linked to an eye-centred representation.

If the hand effect is due to proprioceptive mislocalisation of the ataxic hand for aiming movements, then patients should exhibit misreaching errors when pointing with their ataxic hand toward targets of other sensory modalities. This was the hypothesis tested in experiment 2 by the -pointing toward the ipsilesional hand- task. This task revealed misreaching when the ataxic hand pointed toward proprioceptive targets, i.e. patients' reaching deficit is not specific to visual pointing, in contradiction with the classical definition of *optic* ataxia.

The usual exclusion of reaching deficits for proprioceptive targets in OA is grounded in clinical examinations of the patient's ability to grasp (with their ataxic hand) the thumb of their healthy hand or their nose with eyes closed. Accordingly, the clinical examination of patients OK and CAN did actually fail to detect this high-level proprioceptive deficit, they were included in these experiments as representative of optic ataxia patients. The reverse condition of grasping the thumb of the ataxic arm with the healthy hand (which is in fact the most impaired condition based on the present results) is rarely tested. Moreover, this test calls for grossly locating targets in body coordinates, whereas the present experiment allowed us a more quantitative evaluation of the errors committed in the peri-personal space and calls for a specific impairment in the proprioceptivo-spatial transformation in OA.

Moreover, the task tested in experiment 2 reveals not only that errors due to the hand effect appear when the ataxic hand points toward targets of different modalities but also that an additional field effect can be observed when non-visual targets are presented in the contralesional visual field. It was previously shown that OA visuo-motor deficit does not depend on the visual location of the targets but on a higher-level eye-centred spatial representation (Khan et al., 2005). The present results extend this conclusion suggesting that this eye-centred spatial representation is multi-modal (at least vision and proprioception). The same results have also been shown for pointing to auditory target in normal subjects (Lewald, 1998; Lewald and Ehrenstein, 1996) and a case of auditory ataxia can be found in the literature (Guard et al., 1984).

Further experiments may test whether this functional representation corresponds to physiological properties of the "multimodal eye-centred representation" revealed by monkey electrophysiological data and on which pointing toward auditory targets has also been shown to depend (Cohen and Andersen, 2002).

Conclusion

The demonstration of high-level proprioceptive impairment in OA allows us to interpret misreaching following PPC damage from the point of view of the spatial integration of both visual (target) and proprioceptive (hand) position information. The present results further demonstrate that the eye-centred representations used for visuo-manual planning within the PPC (Medendorp et al., 2005; Buneo et al., 2002) can integrate proprioceptive as well as visual information and thus correspond to visuo-proprioceptive interfaces.

In summary, we may have provided a possible identification of the underlying mechanism of the OA hand effect. In addition, the combination of a field effect and a hand effect was observed for pointing in the dark with eyes fixed toward "peripheral" proprioceptive targets, as well as for pointing toward peripheral visual targets. These results suggest the need to re-define and even rename this deficit. We propose "parietal ataxia" to parallel and also distinguish with "cerebellar ataxia".

Acknowledgments

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