Phantoms on the hands: Influence of the body on brief synchiric visual percepts

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ABSTRACT

Recent studies have found preferential responses for brief, transient visual stimuli near the hands, suggesting a link between magnocellular visual processing and peripersonal representations. We report an individual with a right hemisphere lesion whose illusory phantom percepts may be attributable to an impairment in the peripersonal system specific to transient visual stimuli. When presented with a single, brief (250 ms) visual stimulus to her ipsilesional side, she reported visual percepts on both sides – synchiria. These contralesional phantoms were significantly more frequent when visual stimuli were presented on the hands versus off the hands. We next manipulated stimulus duration to examine the relationship between these phantom percepts and transient visual processing. We found a significant position by duration interaction, with substantially more phantom synchiric percepts on the hands for brief compared to sustained stimuli. This deficit provides novel evidence both for preferential processing of transient visual stimuli near the hands, and for mechanisms that, when damaged, result in phantom percepts.

1. Introduction

Evidence from neuropsychological and brain stimulation studies has shown that the area on or around the body (peripersonal space) is distinctly represented compared to the area away from the body (Ladavas et al., 1998; Makin et al., 2008). For example, di Pellegrino et al. (1997) reported an individual in whom ipsilesional visual stimuli would extinguish the perception of contralesional tactile stimuli – but only when the ipsilesional visual stimulus was near the participant’s ipsilesional hand. Multiple brain regions in posterior parietal cortex, premotor cortex, and putamen have neurons with bimodal visuotactile receptive fields that are yoked to a location on the skin surface and then project to the immediately surrounding peripersonal space (Colby et al., 1993; Graziano and Gross, 1996; Graziano et al., 1994). More recently, a number of studies with neurologically intact individuals have found evidence not only for the existence of peripersonal representations, but that visual stimuli in peripersonal space are processed differently from visual stimuli away from the body (e.g. extrapersonal space). Differential effects for processing visual stimuli near versus far from the hands have been observed for a number of visual attention tasks (Abrams et al., 2008; Cosman and Vecera, 2010; Davoli et al., 2012a; Reed et al., 2006), change detection (Tseng and Bridgeman, 2011), visual perceptual grouping (Huffman et al., 2015), visual memory (Davoli et al., 2012b), visual sensitivity (Dufour and Touzalin, 2008), visual target detection (Kao and Goodale, 2009), and temporal fusion in object substitution masking (Goodhew et al., 2013).

It has been hypothesized that the processing of visual space around the body may be associated with specific visual pathways. Starting with retinal ganglion cells, there is evidence for segregation of visual information from the lateral geniculate nucleus into M (magnocellular) and P (parvocellular pathways). The magnocellular (M) pathway is sensitive to brief, moving, low contrast stimuli, while the parvocellular (P) pathway is sensitive to longer duration, high contrast stimuli (Livingstone and Hubel, 1988). Some have proposed a distinction between dorsal visual vision for action (M) and ventral vision for perception (P) pathways (Milner and Goodale, 1995), whereas others have proposed that the M-pathway is utilized more for transient visual stimuli and the P-pathway is used more for sustained visual stimuli (McCloskey, 2009). As objects near the hands are more likely to be acted upon, researchers have proposed a link between peripersonal representations of space near the hands and magnocellular processing. Magnocellular cells are more sensitive to low- versus high-frequency stimuli (Callaway, 1998), and individuals are more sensitive to detecting orientation changes of low- versus high-spatial-
frequency Gabor patches when presented near versus far from the hands (Abrams and Weidler, 2014). These and other results (see Goodhew et al. (2015) and Taylor et al. (2015) for reviews) have provided initial evidence supporting a link between magnocellular processing and peripersonal space near the hands. However, outside of studies of neurologically intact individuals, there is no additional supporting evidence for a link between peripersonal space and magnocellular processing. Furthermore, there is little known about the functional organization of such a system.

We present a case study of an individual with synchiria to further examine the relationship between different aspects of visual processing and peripersonal space around the hands. Synchiria is a rare disorder subsequent to brain damage, in which stimulation on the ipsilesional side of the body results in the perception of stimuli on both the ipsilesional and contralesional side. In a related deficit – visual allesthesias – individuals see objects presented in the ipsilesional visual field as appearing in the contralesional visual field. However, in the vast majority of these cases, the phantom visual images were palinoptic, persisting for a significant amount of time (up to 15 min) after the original object was out of view (see also Ardila et al., 1987; Jacobs, 1980; Kasten and Poggel, 2006; Murakami et al., 2014). Visual allesthesias is rare, and most previous accounts were clinical case reports, making it difficult to relate these impairments to a specific functional locus.

Although synchiria has been reported in other modalities (Diamond and Bender, 1965; Medina and Rapp, 2008), there are no similar cases in the visual literature.

We encountered an individual (K.G.) who demonstrated a striking case of visual synchiria. In informal testing, we presented a variant of the standard visual finger confrontation task to K.G., in which the experimenter extends his arms and briefly flexes and extends his left finger, right finger, or both fingers, with the participant reporting the movements she saw. With the experimenter standing directly in front of K.G. with the experimenter’s hands located over a table, K.G demonstrated mild extinction. However, when K.G.'s hands were on the table, with the experimenter's fingers directly over her hands, K.G. frequently reported seeing both of the experimenter’s fingers move when the experimenter only moved the finger over her right, ipsilesional hand. In a previous case of tactile synchiria that we reported (Medina and Rapp, 2008), we found that hand position in external space influenced the frequency of phantom tactile percepts; with more synchiria when the hands were in contralesional versus ipsilesional space in both trunk- and head-centered reference frames. Studies in human and non-human primates have provided evidence for ipsilateral activation in primary somatosensory cortex that is actively inhibited, likely by tonic inhibition from the opposite hemisphere (Calford and Tweedale, 1988). We proposed the existence of mechanisms for inhibition of ipsilateral activation that, when damaged, give rise to phantom synchiric percepts.

Given that (a) previous studies of synchiria have shown that representations of external space influenced the frequency of synchiric percepts and (b) that there is evidence linking representations of peripersonal space to magnocellular processing, we predicted that K.G.’s synchiria would be modulated by stimulus position and stimulus duration. More specifically, we expected that K.G. would demonstrate significantly more synchiric percepts for visual stimuli on versus off of the hands. Furthermore, as the M-pathway has been associated with brief versus sustained stimuli, we predicted that K.G.’s would demonstrate more synchiric phantom percepts for brief stimuli on the hand versus off the hands. Finally, given that the P-pathway is not associated with peripersonal space, we predicted that more sustained visual stimuli would not elicit phantom percepts either on or off of the hands.

2. Materials and methods

2.1. Case history

K.G., a 50-year-old female, suffered a large right middle cerebral artery stroke five years before testing. A CT scan showed that her damage encompassed parts of the frontal, parietal, and temporal lobes, with no damage to the primary visual system (see Fig. 1). K.G. was not eligible for an MRI due to scanner contraindications. At the time of testing, a neuropsychological examination showed that K.G. exhibited left-sided hemiparesis, mild neglect, and visual extinction. In additional testing to characterize tactile performance after the stroke, we presented a light touch with her eyes closed (using a flat, 5 mm diameter rubber cylinder) to the dorsal surface of her left hand (10 trials), right hand (10 trials), or both hands simultaneously (10 trials); there were also 6 catch trials on which no stimulus was presented. Five blocks were presented on the distal segment of her middle finger and four blocks on the center of her palm. As there were no differences between stimulus locations, we collapsed over stimulation sites in reporting her performance. When touched on her contralesional left hand, she failed to report the stimulus on 30% (27/90) trials, and when touched on both hands, she reported touch on the ipsilesional hand on only 25% of the trials (26/90), both consistent with damage to somatosensory cortex and/or attentional brain regions. Interestingly, when touched on her ipsilesional right hand, K.G. reported sensation on both hands on 30% of trials (27/90) – demonstrating tactile synchiria.

2.2. Methods

In the following experiments, K.G. was seated at a projection apparatus with a table and a semi-silvered mirror positioned 25 cm above the table. A video projector (connected to a PC running E-Prime 2.0, Psychological Software Tools, Pittsburgh, PA) and screen were positioned above the mirror, such that the images from the projector were cast onto the mirror surface. A small light was on below the mirror, such that the participant could easily see the table. When viewing through the semi-silvered mirror, visual images from the projector appeared to be located on the table or on the participant’s visible hands (if they were positioned on the table).

K.G. was seated such that her trunk and head were aligned with the center of the semi-silvered mirror, with eye movements monitored by visual inspection. Before each trial, a fixation point appeared at the center of the display for 1000 ms. Next, a visual stimulus (a 3 cm diameter red circle, 30 cm lateral to fixation) was presented for 500 ms either left of fixation (10 trials/block), right of fixation (10 trials/block), or on the center of fixation (10 trials/block). Trial order was randomized for each block. After each trial, K.G. was instructed to verbally report whether she perceived the visual stimulus on the left side, right side, both sides, or no stimulation. K.G. was tested in nine blocks over two sessions separated by approximately one month. In the five

![Fig. 1. CT scan for K.G. showing her lesion location (in dark).](image-url)
“on hands” blocks, K.G.’s hands were comfortably positioned on the table, and visual stimuli looked as if they were projected onto the center of her hand(s). In the four “off hands” blocks (one of which was not completed due to computer malfunction), K.G.’s hands were positioned at her sides, with visual stimuli seen as on the table. The visual stimuli were in the same location, relative to fixation, in both “on hands” and “off hands” blocks.

For Experiment 2, the apparatus, stimulus types, and potential responses were the same as in Experiment 1. To examine the relationship between stimulus duration and atypical percepts, each stimulus type was presented at five durations (50, 100, 250, 500, and 1000 ms). Each block consisted of 8 trials per stimulus type/stimulus duration,1 along with 32 no stimulus trials (144 trials/block). K.G. was tested in two ABBA sessions, each consisting of four blocks counter-balanced for stimulus position (on hands, off hands).

3. Results

3.1. Experiment 1 – Influence of stimulus position on synchiric percepts

Given the categorical nature of the responses, we used generalized linear models with logistic regression (logit model) using the glm function in R 3.1.3 to examine the influence of hand position on the frequency of three types of atypical percepts (synchiria: responding “both” when stimulated on the ipsilesional right side; neglect: responding “none” when stimulated on the contralesional left side; extinction: responding “right” when stimulated on both sides). As typical with logit models, differences between conditions and confidence intervals will be reported using odds ratios (OR). We found a significant effect of stimulus position for synchiric responses, as K.G. was more likely to report phantom synchiric percepts when on her hands (27/50, 54%) versus off (11/36, 30.6%) her hands (z = 2.13, p = .033, estimated OR: 2.67, 95% CI: 1.10–6.75). There was no main effect of stimulus position for neglect or extinction responses.

We noted that K.G. never reported experiencing visual phantoms in everyday life. There are a few rare cases of individuals who make visual reflection errors, a potentially related deficit in which stimuli presented on one side are seen on the opposite side. For example, McCloskey and colleagues (McCloskey, 2004; McCloskey and Rapp, 2000; McCloskey et al., 1995) reported an individual (A.H.) who perceived visual stimuli presented on one side as in the opposite location across an attention-centered frame of reference. Interestingly, her visual reflection deficit was substantially attenuated with brief versus sustained, flickering versus constant, and moving versus stationary visual stimuli, providing evidence for impairment to a sustained/parvocellular (not transient/magnocellular) visual system. Given related case reports of mirror mislocalizations that occur primarily for sustained/parvocellular visual stimuli (McCloskey et al., 1995; Pfugshaupt et al., 2007), we surmised that K.G.’s phantom mislocalizations may also be modulated by stimulus duration. However, given the proposed link between transient/magnocellular visual systems and space around the body, we predicted an opposite pattern of performance – increased atypical percepts for brief, not sustained, visual stimuli. In the next experiment, we examined the relationship between her phantom synchiric percepts, stimulus position, and stimulus duration.

3.2. Experiment 2 – Influence of stimulus duration on synchiric percepts

We ran logit models examining the frequency of atypical percepts (synchiria, neglect, and extinction), examining main effects and interactions for stimulus position (on hands, off hands) and stimulus duration, with trials grouped into short (50–250 ms) and long (500–1000 ms) duration bins. For synchiric percepts, there was a main effect of stimulus position, as K.G. was significantly more likely to report synchiric percepts on (96/160, 60.0%) versus off (38/160, 23.8%) the hands (z = 2.80, p = .005, estimated OR: 2.35, 95% CI: 1.30–4.32). Importantly, there was a highly significant stimulus position by stimulus duration interaction (z = 3.67, p < .001, estimated OR: 7.31, 95% CI: 2.58–21.82). K.G. reported synchiric percepts on 80.2% (77/96) of short duration trials presented on her hands, compared to only 25.4% (57/224) for all other conditions (see Fig. 2).

Next, we examined whether the observed synchiric responses were specific to when stimuli were presented to her ipsilesional side. Examining K.G.’s response profile for contralesional, left-sided stimuli, she rarely reported seeing stimuli on the opposite side (3/320 “left” or “both” responses, < 1% of all trials), demonstrating that the synchiric percepts were specifically generated by ipsilesional visual stimulation. Another question is whether the stimulus duration by location interaction was specific to generating synchiric percepts, or also influenced other aspects of perception. As typical for individuals with right parietal damage, K.G. demonstrated neglect (37.2%) and extinction (15.2%). Furthermore, as typically observed in individuals with attentional deficits, K.G. made more neglect and extinction responses for short versus long duration stimuli (main effect of stimulus duration; neglect: z = 4.18, p < .001, estimated OR: 5.62, 95% CI: 2.60–13.3; extinction: z = 3.54, p < .001, estimated OR: 6.62, 95% CI: 2.49–21.0). However, for neglect responses, there was no main effect of stimulus position (p > .250) nor a stimulus position by stimulus duration interaction (p > .250), providing evidence that the influence of body position on atypical responses was specific to synchiric percept generation. Interestingly, there was a significant stimulus position by stimulus duration interaction for extinction responses (z = –2.52, p = .012, estimated OR: 1.24, 95% CI: .023–.611), though in the opposite direction of what was observed for synchiric responses. For short duration stimuli, K.G. showed extinction on 35.9% (23/64) of trials off the hands, compared to only 7.8% (5/64) of short duration trials on the hands, with minimal extinction for long duration stimuli (5/64 off the hands, 6/64 on the hands). These results are consistent with K.G.’s pattern of performance on right-sided trials. When brief, bilateral stimuli are presented off of the hands, K.G. demonstrates visual extinction. However, when these stimuli are presented on the hands, we propose that the ipsilesional, right-sided stimulus produced a synchiric percept on the left side, thus masking her extinction and resulting in paradoxically “improved” performance (see Medina and Rapp (2008) for a similar finding in tactile synchiria).

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1. Due to a programming error, no 100 ms “both” trials were presented.
4. Discussion

We report an individual who, subsequent to a right hemisphere fronto-temporo-parietal lesion, reported phantom synchiric percepts on the contralesional left side when presented with visual stimuli on the right side. Interestingly, these phantom visual percepts were significantly modulated by ipsilesional stimulus duration and position, occurring most frequently when ipsilesional stimuli were brief (250 ms or less) and on the body. These phantom percepts were not observed after contralesional stimulation. Furthermore, this pattern of performance (increased atypical responses for brief stimuli on the body) was specific to generating synchiric percepts. To our knowledge, this is the first reported case of visual synchiria for brief stimuli (contrasting palinopsia), and the first case showing that these phantom visual percepts are modulated by proximity to the body. Our results provide novel evidence for inhibitory processes for visual stimuli on the body that, when damaged, result in phantom visual percepts. Furthermore, our results also provide converging evidence from a brain-damaged individual supporting the link between representations of the space on and around the hands and visual systems for processing brief, transient stimuli.

What functional architecture could account for visual synchiria modulated by proximity to the hands? Given the rare nature of synchiria in any modality, along with the paucity of neural data to constrain potential interpretations, what we present must be regarded as speculative. We note that there could be several alternative explanations for these findings. Our account is guided by explanations of previous individuals with synchiria along with evidence regarding the relationship between visual cortex and tactile/peripersonal representations. In this account, we will assume that the synchiric percepts reported by K.G. are a result of ipsilateral (right hemisphere) visual cortex activity after presentation of a stimulus in the right visual field. For this to occur, there would need to be the following: (a) a pathway from visual cortex to ipsilateral peripersonal and/or tactile representations in both hemispheres, (b) callosal connections to allow transmission of contralesional visual activity to the ipsilesional hemisphere and (c) a mechanism that, subsequent to brain damage, allows for the phantom synchiric percepts to occur.

First, there is clear evidence that information about the body, either via tactile stimulation or peripersonal representations, influences processing in visual cortex. Macaluso et al. (2000) found that presenting a tactile stimulus on the same side as a visual target enhanced activity in visual cortex. Furthermore, they also found that areas in visual cortex associated with increased activity with a same-sided tactile stimulus showed functional connectivity with supramarginal gyrus and inferior parietal lobe, regions that have been associated with peripersonal representations of space. Presenting tactile stimuli results in lower TMS-induced phosphene thresholds when the hand is in the same location in external space as the perceived phosphenes (Ramos-Estebanez et al., 2007). This enhancement effect loses its spatial specificity after TMS of posterior parietal cortex, providing additional evidence that posterior parietal regions are involved in visuotactile integration. Larger N1 ERP components, indicative of increased processing in occipital brain regions, have been generated for visual stimuli presented on versus above the hands (Simon-Dack et al., 2009). These studies provide evidence that posterior parietal regions involved in representing touch and the body interact with visual areas, resulting in changes in visual perception.

Second, for synchiria to occur, there needs to also be transmission of stimulus information from the contralesional hemisphere to the ipsilesional hemisphere. Given the lack of such connections (apart from those near the vertical meridian, e.g. Hubel and Wiesel, 1967) in V1, it is more likely that any callosal transmission would occur in areas outside of primary visual cortex. Studies of individuals with crossmodal extinction (Ladavas et al., 1998), crossmodal congruency tasks (Maravita et al., 2003) and non-human primates studies (Graziano and Gross, 1993) have provided evidence for bimodal visuotactile representations of peripersonal space. Peripersonal space is thought to be represented in two regions – posterior parietal and ventral premotor cortex (Makin et al., 2007). Interestingly, both posterior parietal cortex (Caminiti and Sbriccoli, 1985) and ventral premotor cortex (Boussaoud et al., 2005) have callosal connections between homotopic regions, allowing for the possible transmission of information about the stimulus between hemispheres. Therefore, we suggest that crosshemispheric information transfer is likely to occur between representations of peripersonal space, as opposed to the primary visual system.

Third, given that neurologically-intact individuals do not experience visual synchiria, there also needs to be a mechanism that allows for bilateral activation (subsequent to ipsilateral stimulus stimulation) to be suppressed. We propose two possibilities. One is that synchiria is caused by impairment of inhibitory mechanisms that typically serve to suppress ipsilateral activation, similar to what has been found in the somatosensory system. Past studies have found dense callosal connections between higher order primary somatosensory regions (e.g. Brodmann areas 1 or 2) and/or secondary somatosensory cortex (Fabri et al., 2001; Krubitzer et al., 1998). Furthermore, there is evidence that tactile stimulation on one side results in activation of contralateral primary somatosensory regions along with concurrent suppression in the homotopic ipsilateral somatosensory areas (Hlushchuk and Hari, 2006; Lipton et al., 2006). We have proposed that tactile synchiria is caused by damage to mechanisms that suppress ipsilateral activity after stimulation, resulting in the perception of bilateral stimuli subsequent to unilateral stimulation (Medina and Rapp, 2008; Sathan, 2006). Interestingly, in one case of tactile synchiria (Medina and Rapp, 2008), these inhibitory mechanisms were modulated based on body position in external space, as the participant demonstrated significantly more synchiric percepts when their hands were in contralesional versus ipsilesional space in head- and trunk-centered reference frames. One possibility is that a similar inhibitory mechanism, based on stimulus position in a peripersonal frame of reference, may also exist. An alternative mechanism is that, due to callosal connections between peripersonal representations, a right-sided visual stimulus on the hands may activate right hemisphere peripersonal representations in a subthreshold manner. Importantly, the absence of input from primary somatosensory cortex to these peripersonal representations could result in a stronger weighting for visual information in the peripersonal representation, such that any activation from visual areas would be amplified. This amplified signal could then result in suprathreshold phantom visual percepts.

K.G.’s visual synchiria is strongly influenced by stimulus duration, with significantly more phantom percepts for short (50–250 ms) versus long (500–1000 ms) visual stimuli. Our results demonstrating a relationship between short visual stimuli and the space around the hands are consistent with hypotheses in which the M-pathway is tied to processing transient visual stimuli, whereas the parvocellular system is preferentially involved in sustained visual processing (Breitmeyer and Ogmen, 2000; McCloskey, 2009). Evidence for a transient/sustained distinction comes from reaction time distributions for low- versus high-spatial frequency targets in humans and non-human primates (Tolhurst, 1975), visual backwards masking (Breitmeyer and Ganz, 1976) and individuals with developmental deficits or brain damage (McCloskey, 2004; McCloskey et al., 1995; Pflugshaupt et al., 2007). We suggest that K.G.’s deficit is caused by damage to mechanisms that inhibit transcallosal activation that originates in
peripersonal representations. Furthermore, given the links between peripersonal space around the hands and the M-pathway (Abrams and Weidler, 2014; Goodhew et al., 2014), we believe that this disinhibited activation reflects the nature of the peripersonal representation—primarily magnocellular, and biased strongly towards brief, transient visual stimuli as well as tactile stimuli.

A second interpretation is that enhanced performance on visual tasks performed near the body is caused by increased attention to the space around the hands (Cosman and Vecera, 2010; Reed et al., 2006). Is it possible that K.G.’s synesthetic percepts are caused by damage to attentional mechanisms for the area around the hands? We believe this is unlikely for the following reasons. K.G. demonstrates an attentional deficit (neglect) that is not modulated by stimulus position on the hands, suggesting that her neglect and synchiria are caused by damage to different mechanisms. Although it is possible that K.G.’s synchiria is also caused by attentional mechanisms separate from what cause her neglect, other evidence suggests that this is unlikely. In most attentional deficits, individuals are more likely to fail to respond to brief versus longer duration visual stimuli, whereas K.G. demonstrated the opposite pattern (more visual stimuli perceived at brief durations). It is also unclear what attentional mechanism would, when damaged, result in an increase in visual percepts. At the moment, damage to inhibitory mechanisms for representations of transient stimuli near the body is likely a more parsimonious account. Although our results do not seem to have a functional locus in the attentional system, we are not claiming that attentional accounts for improved visual performance near the hand are in error. One possibility, consistent with ERP work on the topic (Reed et al., 2013), is that there may be multiple processes that contribute to enhanced visual processing around the body.

Previous studies have shown that neurologically-intact individuals experience visual stimuli presented to embodied rubber hands as eliciting tactile sensations (Durgin et al., 2007). Given that K.G. demonstrates tactile, as well as visual synchiria, is it possible that K.G. “feels” the visual stimuli presented to her hands and that her visual synchiria utilizes tactile pathways? We note that K.G. never spontaneously reported feeling touch after visual stimulation, nor did she ever say that she saw the ipsilateral visual field, as she felt the visual stimuli. Given that the deficit involved peripersonal processing, which is intimately tied to bimodal visuo-tactile representations, we believe it is possible that removal of peripersonal inhibition may result in both visual and tactile synchiria. However, more evidence will be needed to support this conjecture.

Finally, we note that K.G. was unavailable for further testing, and that there were a number of additional questions that we hoped to explore. These included whether her deficit was multimodal (e.g. ipsilesional tactile stimuli produce contralesional tactile percepts; ipsilesional visual stimuli produce contralesional visual percepts) or crossmodal (ipsilesional tactile/visual stimuli produce contralesional visual/tactile percepts), whether sustained stimuli associated with the magnocellular system (e.g. low spatial frequency) would result in synchiria, and whether synchiria would occur for visual stimuli near her unseen hands. Given that K.G.’s lesion is not out of the ordinary, we believe that others may have a similar deficit, and that it may simply be understudied. We hope that this report will encourage neuropsychologists and neurologists to examine their patients for this interesting phenomenon, and address these and other questions in future research.

Acknowledgments

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References