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Report

# Neurological and Robot-Controlled Induction of an Apparition

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## Summary

**Tales of ghosts, wraiths, and other apparitions have been reported in virtually all cultures. The strange sensation that somebody is nearby when no one is actually present and cannot be seen (feeling of a presence, FoP) is a fascinating feat of the human mind, and this apparition is often covered in the literature of divinity, occultism, and fiction. Although it is described by neurological and psychiatric patients [1, 2] and healthy individuals in different situations [1, 3, 4], it is not yet understood how the phenomenon is triggered by the brain. Here, we performed lesion analysis in neurological FoP patients, supported by an analysis of associated neurological deficits. Our data show that the FoP is an illusory own-body perception with well-defined characteristics that is associated with sensorimotor loss and caused by lesions in three distinct brain regions: temporoparietal, insular, and especially frontoparietal cortex. Based on these data and recent experimental advances of multisensory own-body illusions [5–9], we designed a master-slave robotic system that generated specific sensorimotor conflicts and enabled us to induce the FoP and related illusory own-body perceptions experimentally in normal participants. These data show that the illusion of feeling another person nearby is caused by misperceiving the source and identity of sensorimotor (tactile, proprioceptive, and motor) signals of one's own body. Our findings reveal the neural mechanisms of the FoP, highlight the subtle balance of brain mechanisms that generate the experience of "self" and "other," and advance the understanding of the brain mechanisms responsible for hallucinations in schizophrenia.**

## Results and Discussion

Descending with his brother from the summit of Nanga Parbat, one of the ten highest mountains in the world, Reinhold Messner felt a third climber "descending with us, keeping a regular distance, a little to my right and a few steps away from me, just outside my field of vision" [10]. Messner "could not see the figure" but "was certain there was someone there," sensing "his presence" [10]. This apparition, the sensation that somebody is nearby when no one is actually present, is called the feeling of a presence (FoP) and has been described during periods of physical exhaustion [1, 3, 4, 11, 12] and has influenced occult literature and fiction [13]. Although people do not see the "presence," they may describe its spatial location and frequently turn around or offer food to the invisible presence [14, 15]. Although the FoP has been described in psychiatric [1, 2, 15, 16] and neurological patients [2, 16], its neural origin is unknown. A single case report showed that electrical stimulation in temporoparietal cortex induces the FoP, suggesting that disturbed sensorimotor processing (tactile, proprioceptive, and motor cues) is important [17]. However, this has not been confirmed in other patients, and the significance of these findings for the FoP in healthy subjects is unclear.

### Neurology and the FoP

We performed lesion analysis and analyzed the associated hallucinations and neurological symptoms in 12 FoP patients (Table 1; Figure S1 available online). The presence was felt in all cases in close proximity to and behind the patient's body ($p < 0.01$). The presence was lateralized ($p < 0.01$) in contralesional space ($p < 0.01$) and equally often in right or left hemispace (not significant, n.s.; Table 1). Sensorimotor deficits ($p < 0.01$) and the experience of illusory movements of the presence during movements of the patient (n.s.) were frequent symptoms (Supplemental Experimental Procedures). For lesion analysis, we used a multimodal imaging approach, relying on combined functional and structural neuroimaging data to determine anatomical regions of maximal lesion overlap [18–20]. This approach, which combined functional and structural lesion data, was necessary because many patients suffered from epilepsy, and in several patients, FoP was induced by electrical stimulation, and because the FoP is rare. Projecting all lesions onto the left hemisphere, lesion overlap analysis highlighted three cortical regions: insular cortex, frontoparietal cortex, and the temporoparietal cortex (Figure 1A). We next compared lesion extent within these three cortical regions between FoP patients and control patients matched for complex hallucinations, etiology, and sensorimotor deficits (Figure 1B; Supplemental Experimental Procedures): lesion extent did not differ between both groups in Brodmann area 22 ($p = 0.18$) and 48 ($p = 0.68$), whereas FoP patients had significantly larger lesions in Brodmann area 7 ($p = 0.01$). These results show that although FoP is associated with insular, temporoparietal, and frontoparietal lesions, only frontoparietal lesions (Brodmann area 7) were specifically associated with the FoP.

### Robotically Induced Bodily Illusions

In order to study the FoP in healthy subjects, we designed a master-slave robotic system [21] and investigated sensorimotor signals and their role in inducing FoP experimentally by integrating our findings with principles from other body illusions [5] (informed consent was obtained, and all the studies

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Table 1. Clinical Data for FoP Group: Clinical Data Are Summarized for Each FoP Patient

| Patient | Diagnosis/Etiology | Lesion | Lesion Analysis | Neurology/Neuropsychology | Semiology |
|---|---|---|---|---|---|
| FoP a | neurocystcercosis | frontoparietal cortex (R) | MRI | gait disturbance/mild executive deficits | presence of a person while walking, to his right, behind |
| FoP b | epilepsy, status post (s/p) ischemic stroke, vasculitis | occipitoparietal cortex, frontoparietal cortex (R) | MRI, EEG | left-sided sensorimotor deficit | presence behind left shoulder, a silhouette, like a shadow of the same proportions; echopraxia; unpleasant; most frequently perceived while walking |
| FoP c | epilepsy | frontoparietal cortex (L) | MRI, EEG, PET, SPECT, iEEG | right-sided weakness/ postictal aphasia | presence of a "black person" behind her, no lateralization, unpleasant |
| FoP d | epilepsy, s/p resection of capillary angioma in the left insula | insula, frontoparietal cortex (L) | MRI, EEG, PET, SPECT, cortical stimulation | right-sided numbness/ executive deficits | presence of a man, behind to her right, in peripersonal space, fear and anxiety |
| FoP e | intracerebral hematoma, ischemic stroke | temporal lobe, frontal lobe, parietal lobe, insula (R) | MRI | left-sided sensorimotor deficit/anosognosia, reduplicative paramnesia | presence of daughter about 50 cm behind, to the right |
| FoP f | epilepsy, cerebral histiocytosis | thalamocapsular-caudate region, insula (R) | MRI, EEG | left-sided dysmetria/left spatial neglect | presence of "a person's black shadow" to her left, same position and posture as the patient, close family member |
| FoP g | epilepsy, s/p capsulolenticular haemorragic stroke | insula, capsulolenticular region (R) | MRI, EEG | right-sided paraesthesia and hemiparesis/neglect, apraxia | presence of four people in mostly left frontal space, family members |
| FoP h | epilepsy, hemiplegic migraine | insula, parietooccipital cortex (L) | MRI, EEG | right-sided paraesthesia and weakness/aphasia | presence of a person's "shadow" to his right, behind |
| FoP i | epilepsy | mesial temporal lobe, anterior temporal lobe (L) | MRI, PET, SPECT, iEEG | normal/postictal aphasia | sensation of somebody's presence, behind to the left, anxiety |
| FoP j | epilepsy, s/p resection of a left temporal dysplastic lesion | temporoparietal cortex (L) | MRI, EEG, PET, SPECT, cortical stimulation | normal/aphasia, anomia | presence of a male shadow, behind to the right, same position, echopraxia |
| FoP k | epilepsy | posterior temporal lobe (L) | MRI, cortical stimulation | normal/aphasia | presence behind to the right, strictly unilateral, unpleasant, no echopraxia |
| FoP l | epilepsy, intracerebral hematoma | temporoparietooccipital cortex (L) | MRI, EEG | right sided sensorimotor deficit/aphasia, paraphasia, agraphia, alexia | presence of a person ("shadow of a female person"), on her right side (20–30 cm), behind, while standing and walking, echopraxia |

were conducted in conformity with the Declaration of Helsinki; Supplemental Experimental Procedures). We investigated whether the FoP is associated with illusory touch sensations (questionnaire) and mislocalization of the body ([20, 22]; Supplemental Experimental Procedures) (Figure 2A; Figure S2; Movie S1). While standing and blindfolded, participants moved their arms and thereby moved the master device (via their inserted right index fingers) in front of them. These movements were sent to the slave robot, which applied tactile stimuli in real time to the participants' backs (Figure 2A; Movie S2) [7]. Participants moved the master robot for 3 min while they received tactile cues on their backs (by slave robot) and their right fingertips (by master robot; Movie S2). Stroking was applied either synchronously or asynchronously (500 ms delay), with or without somatosensory force feedback at the hand (2 × 2 factorial design).

During synchronous, but not asynchronous, stimulation, participants (study 2; Supplemental Experimental Procedures) experienced the sensation of touching themselves (self-touch), despite extending their arms in front of their bodies ($p < 0.01$; Figure 2B). Synchronous stimulation and stimulation with force feedback were further associated with a drift of the subject's body toward the front position, where they felt their hands ($p < 0.05$; Figure 2C; Movie S2; Supplemental Experimental Procedures). Thus, sensorimotor signals from the fingertip (forward-extended arm) while a tactile cue is applied to the subject's back induce the illusory feeling of touching one's own back with one's own finger (self-touch) and bias self-location toward the fingertip. These findings extend earlier illusions due to sensory conflicts between two hands [5] or between two hands and the nose [23] to an illusion between hand and trunk (see also [6, 7]).

## Robotically Induced FoP

More interesting effects were observed during stronger sensorimotor conflicts; during asynchronous stimulation, participants showed a drift in self-location in the opposite, backward direction ($p < 0.01$) and reported higher other touch than self-touch. Moreover, during postcondition debriefing, five subjects reported to have experienced a FoP (Supplemental Experimental Procedures). In study 3 (Supplemental Experimental Procedures), we investigated whether we could induce the FoP experimentally, predicting that under asynchronous stimulation without somatosensory force feedback (fingertip), subjects would feel the presence of a person that is touching them, associated with a backward drift in self-location (toward the presence). Figure 3A shows that participants experienced being in the presence of another person in the asynchronous versus synchronous stimulation condition ($p < 0.01$) and experienced being touched by that invisible presence behind them ($p < 0.01$). Asynchronous stimulation induced a backward drift in self-location toward the position of the presence ($p < 0.05$; Figure 3B; Movie S3).

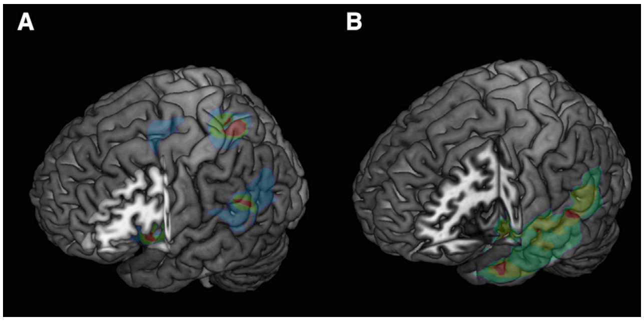

Figure 1. Lesion Analysis in Study 1

(A) Lesion overlap analysis for the FoP group revealed three regions where overlap was maximal. These regions were as follows: temporoparietal cortex (five patients; Brodmann area 22; Montreal Neurological Institute [MNI] x = 58, y = −51, z = 22), frontoparietal cortex (five patients; Brodmann area 7, MNI x = −32, y = −54, z = 62), and insula (five patients; Brodmann area 48; MNI x = −43, y = 8, z = −4). The color scale indicates the following: blue represents three patients, green represents four patients, and red represents five patients. Five patients had a right lesion, and seven patients had a left lesion; for analysis, all lesions were projected onto the left hemisphere.
(B) Maximal lesion overlap for the control group. The color scale indicates the following: green represents four patients, yellow represents five patients, and red represents six patients. Five control patients had a right lesion, and seven control patients had a left lesion; for analysis, all lesions were projected onto the left hemisphere.

To exclude that the FoP was caused by explicit questioning or related mechanisms, we designed a person numerosity task that tested implicitly the presence of another person close to the participant (study 4). While using the robot in synchronous and asynchronous stimulation, participants estimated the number of people that they felt were close to them in the testing room (the following question was asked: "how many people do you feel close to you?"). Data show that during the asynchronous (FoP-inducing) condition, participants judged a significantly higher number of people as being close to them (mean = 2.0) as compared to the synchronous condition (mean = 1.6; $p < 0.01$; Figure 3C).

Our neurological data reveal that the FoP is caused by focal brain lesions and that the FoP is most often experienced unilaterally, within peripersonal space behind the body, and associated with illusory own-body perceptions. FoP patients also show frequent somatosensory-motor deficits that were contralateral to the lesion on the same side as the presence. Compatible with the variability in lesion location across earlier clinical studies, we found that lesions associated with the FoP were focal but were linked to temporoparietal, frontoparietal, and insular cortex (of either hemisphere). Previous work showed that brain interference or lesions in FoP patients were in temporoparietal cortex [17] and frontoparietal cortex [2]. The present data also highlight that the FoP follows insular lesions and indicated lesion location with greater precision than previous work. Additional analysis in control patients (matched for complex hallucinations, etiology, and sensorimotor deficits) revealed that from the three lesion overlap zones, only the frontoparietal site was specifically associated with the FoP, highlighting the importance of the latter region in the FoP. Interestingly, temporoparietal cortex [20], insula [24], and frontoparietal cortex [5, 25] have been associated with bodily self-consciousness and are areas that integrate sensorimotor or multisensory bodily signals, as shown in human [26] and nonhuman primates [27, 28], compatible with the sensorimotor deficits we observed. The present findings highlight that the FoP is characterized by its own distinct phenomenology (compared to out-of-body experiences [OBEs], heautoscopy, and autoscopic hallucinations) and interference with frontoparietal cortex. All latter conditions have been linked to a single and hemisphere-specific lesion site [18, 20] and to disorders of multisensory integration that do not involve the sensorimotor system. OBEs are attributed to visuosomatosensory-vestibular disintegration [20, 29], heautoscopy is attributed to visuosomatosensory-interoceptive disintegration, and autoscopic hallucinations are attributed to visuosomatosensory disintegration [18, 30]. Instead, the present FoP data give most importance to abnormal integration of sensorimotor signals caused by frontoparietal lesions of either hemisphere. We note that these lesion overlap data have to be regarded with caution, as we included different types of brain lesions and included functional (intracranial electroencephalogram [EEG], cortical stimulation, and PET) and structural (MRI) lesion data. Moreover, our lesion overlap analysis also associated temporoparietal cortex and the insula with the FoP, but this was not corroborated by comparison with control patients. More work is needed to understand how these three regions differ in their involvement in the FoP.

The robotic data corroborate and apply our neurological findings to healthy subjects and show that sensorimotor conflicts using well-controlled bodily stimulations are sufficient to induce the FoP (albeit more weakly than in neurological patients). Based on clinical data and previous body illusion work [5, 7, 23], our robotic data show that the FoP can be induced when exposed to conflicting sensorimotor signals that are spatially and temporally incompatible with physical self-touch. Joining sensorimotor signals from forward-extended arms without force feedback at the fingertips (motor-proprioceptive cues), with delayed tactile feedback at the subjects' backs, was sufficient to induce the FoP. Under such stimulation, subjects reported being in the presence of another person behind them and being touched by that invisible presence. This was associated with a backward drift in self-location toward the presence and with elevated person numerosity judgments, corroborating our experiential findings behaviorally. The robotically induced FoP thus mimics the FoP in clinical populations and healthy subjects and is associated with abnormal perception of one's own body. These are major quantitative achievements because previous reports consisted of post hoc anecdotal accounts occurring far away from the research laboratory and because the FoP has never before been induced experimentally [1, 3, 4, 11, 12].

A prominent model for motor control and bodily experience posits that efferent copy signals from the sensorimotor system are used to make predictions about the sensory consequences of movement and that such integration is fundamental for normal bodily experience [8, 31]. Predicted sensory consequences based on motor commands are compared with the reafferent sensory inputs during motor execution. A match between the predicted sensory information and the actual sensory information is considered to be self-generated, whereas differences between predicted sensory consequences and the reafferent signals are indicative of the influence of an external object or another agent. Our master-slave robot generated a spatiotemporal mismatch between our participants' arm movements (motor-proprioceptive signals) and their sensory consequence (tactile feedback on their back), which was delayed and spatially incompatible with respect to the arm-related signals. This spatiotemporal conflict was resolved by our participants generating the illusory experience that the felt touch was not caused by themselves but by another person behind them who was touching their backs. This was revealed by subjective evidence, that is, a decrease in the reported feeling of touching one's own body, an increase in the feeling of being touched by somebody else, and an increase in feeling the presence of another person under asynchronous stimulation. Such reductions in self-touch and agency for one's actions have been reported before (visual-motor, audio-motor, and somatosensory-motor conflicts [31–34]). Our data are the first to induce such changes in association with the apparition or presence of another agent. Based on the present findings, earlier data using trunk stimulation [30], and theoretical considerations [35], we argue that the sensorimotor arm-trunk conflict in association with strong spatial incompatibility of self-touch induced the FoP.

In addition to explaining a fascinating phenomenon with a rich cultural history, the present data are also of relevance for the understanding of schizophrenic symptoms. Abnormal integration of sensorimotor signals and their cortical representations has been described in schizophrenic patients [36] and has been associated with positive hallucinatory and delusional symptoms [37, 38]. According to this view, positive schizophrenic symptoms, such as alien voices and delusions of control, are caused by central deficits in integrating predicted sensory consequences of own movements and the respective reafferent signals. As a consequence, schizophrenic patients under certain conditions may not perceive self-generated sounds and movements as such but may misperceive them as being generated by external agents (as in the experience of alien voices or control of own movements by others), and this is corroborated by behavioral and neuroimaging investigations [37, 39, 40]. The present data not only account for a loss of agency in such patients but also show that a conflict between proprioceptive-motor signals and tactile feedback at a physically impossible position induced the feeling of being in the presence of an alien agent and being touched by that invisible person. Furthering the mechanistic insight into the functional brain processes generating hallucinations and delusions, we show that simple sensorimotor conflicts induced, in healthy subjects, an experience that shares crucial aspects with positive, first-rank symptoms in schizophrenia, including the apparition of the alien agent [40, 41].

The FoP has fascinated mankind from time immemorial across all cultures, impacting the literature of divinity, occultism, and fiction. The phenomenon continues to fascinate humans today, as testified by several recent case collections [4] and documentaries [13]. Collectively, the present neuroimaging and robotics data provide a solid scientific explanation

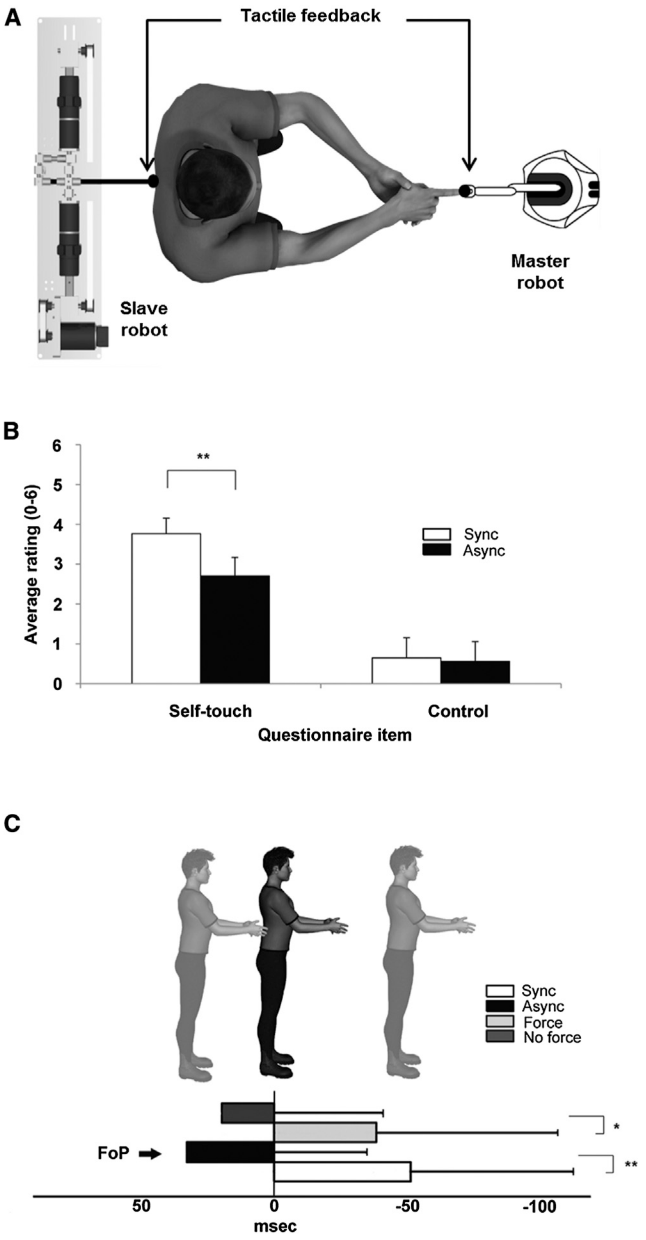

Figure 2. Master-Slave Robotic System and Tactile Full-Body Illusion in Study 2

(A) A schematic view of the master haptic interface (Phantom Omni; SensAbleTechnologies) and the slave robot are shown (see Movie S1; Figure S2; Supplemental Experimental Procedures). Position of the slave robot is controlled by the master robot, resulting in a total correspondence between the movements of the two devices (intrinsic delay $< 1$ ms). The participant moved the master robot via his right index finger (that was attached to the robot), which actuated the movements of the slave robot that applied touches to the participant's back. In order to test the impact of robotically controlled sensorimotor conflicts to induce changes in bodily self-consciousness, we tested the following four experimental conditions: (2 × 2 factorial design) (1) synchronous with force feedback, (2) asynchronous with force feedback, (3) synchronous with no force feedback, and (4) asynchronous with no force feedback. During the asynchronous conditions, the movements performed at the master device were delayed by 500 ms before being transmitted to the slave device (factor delay: synchronous or asynchronous). A "virtual back" in front of participants was created in order to have a mechanical stop (occurring synchronously or asynchronously) to the touch that the participant received on the back (factor force feedback: force or no force).

(B) Ratings for illusory touch and control questions are shown. Note that illusory self-touch is significantly larger in the synchronous versus asynchronous condition ($p < 0.01$) and also significantly larger than ratings of the control items ($p < 0.01$).

(C) Participants showed a drift in self-location toward the virtual back (toward the fingertip) that was larger during the synchronous than asynchronous conditions ($p < 0.01$) and was larger in the condition with versus without somatosensory force feedback to the participants' fingertip ($p < 0.05$) (Movie S2). Self-location was quantified using the mental ball throwing task, during which participants were asked to estimate (by pressing a button) the time that a ball they were holding in their hands would take to reach the wall if they were to throw it (Supplemental Experimental Procedures). The condition in which five subjects spontaneously noted a FoP is indicated with an arrow.

Error bars show the SEM.

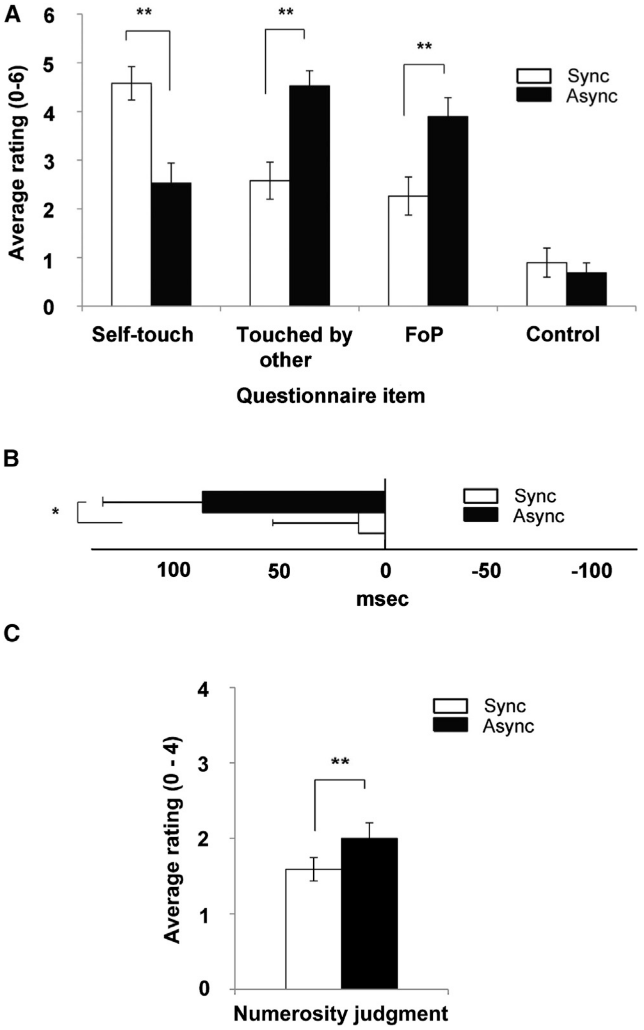

Figure 3. Robotically Induced FoP in Studies 3 and 4

(A) FoP questions, touched-by-other questions, illusory self-touch questions, and control questions are shown. As predicted, during asynchronous stimulation, participants experienced another person standing behind them (FoP; $p < 0.01$), touching them (touched by other; $p < 0.01$). As in study 2, synchronous stimulation induced illusory self-touch ($p < 0.01$).

(B) A significant difference in self-location was found between the asynchronous (backward direction, associated with FoP) and synchronous condition in study 3 ($p < 0.05$; Movie S3).

(C) Number of people (0–4) that participants judged as being close to them (the following question was asked: "how many people do you feel close to you?"; person numerosity task) during synchronous and asynchronous sensorimotor stimulation. As predicted, participants reported a significantly higher number of people during the FoP condition (asynchronous) than the synchronous condition ($p < 0.01$). Note that during the experiment, nobody was ever close to the participants.

Error bars show the SEM.

for the FoP and link a phenomenon that appears strange and complex at first sight to basic mechanisms of sensorimotor signal integration in a cortical network centering in frontoparietal cortex and to a prominent account of positive symptoms in schizophrenia. Apart from explaining a fascinating phenomenon and its potential clinical impact, the present data reveal the fine balance between the distributed cortical brain mechanisms in humans that generate the experience of "self" and "other," which, if distorted, give rise to the FoP.

## Supplemental Information

Supplemental Information includes Supplemental Experimental Procedures, two figures, one table, and three movies and can be found with this article online at http://dx.doi.org/10.1016/j.cub.2014.09.049.

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