The Demystification of Autoscopic Phenomena: Experimental Propositions

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Autoscopic phenomena (AP) are rare, illusory visual experiences during which the subject has the impression of seeing a second own body in extrapersonal space. AP consist of out-of-body experience, autoscopic hallucination, and heautoscopy. Recent neurologic reports support the role of multisensory integration deficits of body-related information and vestibular dysfunctions in AP at the temporo-parietal junction. A caveat to test the underlying neurologic and cognitive mechanisms of AP has been their rare and spontaneous occurrence. Recent evidence linked AP to mental own-body imagery engaging brain mechanisms at the temporo-parietal junction. These recent observations open a new avenue for testing AP-related cognitive mechanisms in selected clinical and normal populations. We review evidence on several clinical syndromes (psychosis, depression, anxiety, depersonalization, body dysmorphic disorder), suggesting that some of these syndromes may relate to AP-proneness, thereby leading to testable propositions for future research on body and self processing in addition to AP.

Introduction

The self as an entity distinct from other human conspecifics may be described as an enduring and spatial entity (the feeling that we are the same person across time and space) to which certain mental events and actions are ascribed (the feeling of agency; being author of one's own thoughts and actions) and which is distinct from the environment [1]. The many concepts of the self have been influenced by theology, philosophy, and psychology [2–4,] but also by clinical observations from the fields of neurology and psychiatry [1,5–7,8••]. One group of clinical phenomena may be especially fruitful in this respect: autoscopic phenomena (AP). During AP, the individual sees a second own body in extrapersonal space, associated to varying degrees by a separation of the self from the body. AP challenge our notions about the experienced unity of self and body, localization of the self, and agency [2,4,9•]. This review describes recent scientific approaches to AP and body and self processing and makes testable predications for future research.

Autoscopic phenomena generally are classified among disorders of somatognosia and include various short-lasting (usually), illusory experiences about the location and position of one's body or body parts [10–12]. They generally occur in patients with posterior brain damage and are characterized by illusions that only affect a certain body part (body-part illusions) or affect the entire body (body illusions or AP) [8••,10,12]. Recently, phenomenologic, functional, and anatomic mechanisms of AP and their importance for mechanisms of self processing have been reported [5,8••,13,14••]. It has been speculated that these phenomenologic characteristics point to similar and distinct neurocognitive mechanisms in the three main forms of AP [5,8••].

Definition of the Three Main Autoscopic Phenomena

During an out-of-body experience (OBE), people seem to be awake and feel that their "self," or center of awareness, is located outside of the physical body and is somewhat elevated (disembodiment). It is from this elevated extracorporeal location that the subjects experience seeing their body and the world (for overview see [8•,13,14••,15,16]). The subjects' reported perceptions are organized in such a way as to be consistent with this elevated visuo-spatial perspective. Therefore, an OBE can be defined as the presence of disembodiment, distanced and elevated visuo-spatial perspective, and autoscopy.

During an autoscopic hallucination (AH), a person experiences seeing his double in extracorporeal space without leaving his own body (no disembodiment). As compared with OBEs, individuals with AH experience seeing the world from their habitual visuo-spatial perspective and experience their "self," or center of awareness, inside their physical bodies.

Lastly, during a heautoscopy (HAS), the individual also has the experience of seeing a double of himself in extracorporeal space. However, it is difficult for the subject to decide whether he is disembodied and whether the self is localized within the physical body or in the autoscopic body [8••]. In addition, the subjects often report seeing

the world simultaneously or in an alternating fashion from the physical body and the double's body. For additional details see Brugger [14••] and Blanke *et al*. [8••].

Neurology

Etiologic mechanisms

Autoscopic phenomena have been reported in various diseases of the central nervous system and may be attributable to generalized disease (meningitis, encephalitis, intoxications, generalized epilepsies) or focal disease (focal epilepsy, traumatic brain damage, migraine, vascular brain damage, neoplasia) [5,8••,13,17]. In regard to focal brain damage, these studies primarily implicated posterior brain regions, including the temporal, parietal, or occipital lobes [5,13]. More recently, Blanke and Arzy [18], Blanke *et al.* [8••,19] and Maillard *et al*. [20] suggested that AP may be related primarily to damage at the temporo-parietal junction (TPJ) of either hemisphere.

Functional mechanisms

Many different functional mechanisms have been proposed in the study of AP, including visual (hallucinatory) mechanisms, proprioceptive and/or kinaesthetic mechanisms, and vestibular mechanisms (for discussion see Devinsky *et al*. [13] and Brugger *et al*. [5]). More recently, Blanke *et al*. [8••] suggested a differential implication of vestibular processing in the different forms of AP. These authors suggested systematic differences in the strength of a vestibular dysfunction in AH, HAS, and OBE. The role of the vestibular system for AP also is supported by descriptions of vestibular sensations during AP in healthy populations [15,16,21]. Blanke *et al.* [8••] suggested that OBEs were associated with a gravitational, otolithic, vestibular disturbance, whereas the vestibular dysfunction in patients with HAS is more variable and often is characterized by rotational components. Vestibular dysfunction was absent in patients with AS. Based on this neurologic evidence, these authors suggested that AP may relate to dysfunctional multisensory integration at the TPJ [8••]. Additional vestibular dysfunctions may be irrelevant for AH, but are increasingly important in HAS and OBE. Although these propositions seem promising, the rarity of AP in clinical and healthy populations renders their experimental investigation difficult.

There are several studies that provided theoretical propositions for experimental studies with respect to AP [14••,15,22,23]. Concretely, these studies suggested that brain mechanisms engaged during mental own-body transformations may rely on similar brain mechanisms to those underlying AP. We recently did a series of experimental studies to test some of these propositions [9•]. In an evoked potential mapping study, we showed the selective activation of the TPJ at 330 to 400 ms after stimulus onset when healthy volunteers imagined themselves in the position and visual perspective generally reported by people experiencing spontaneous OBEs. In an independent study sample, we showed that interference with the TPJ by transcranial magnetic stimulation at that time impaired the mental transformation of the own body in healthy volunteers relative to transcranial magnetic stimulation over a control site at the intraparietal sulcus. No such inference was observed for imagined spatial transformations of external objects, suggesting the selective implication of the TPJ in mental imagery of one's own body. Lastly, in an epileptic patient with OBEs originating from the TPJ, we showed partial activation of the seizure focus during mental transformations of her body and visual perspective mimicking her OBE percept. These results suggested that the TPJ is a crucial structure for the conscious experience of the normal self mediating spatial unity of self and body.

Taken together, the aforementioned clinical and experimental findings suggest that deficient multisensory integration (involving visual, somatosensory, and vestibular processing) and mental own body imagery (involving visuo-spatial perspective taking, self-location, and spatial unity) seem crucial for our understanding of deviant self-processing as occurs during AP. In addition, the use of mental own-body transformation using paradigms implicating imagery perspective changes and imagery self-location may allow linkage of lower level multisensory processing with higher level processing in regard to body, self, and AP.

Yet, given that AP occur spontaneously and rarely in normal, neurologic, and psychiatric populations and rarely in neurologic patients, we propose that it may be favorable to additionally screen and investigate individuals from healthy and psychiatric populations who may have an enhanced risk and/or chance to experience an AP. This is additionally developed in the remainder of this article. First, based on the illusory character of AP and the link between epilepsy and psychiatry [24,25], we discuss scientific evidence regarding psychotic populations as a potentially "AP-prone" population. We also discuss other AP-prone populations (individuals with depression, anxiety, depersonalization, and body dysmorphic disorders), who may be likely candidates for AP because of previously reported multisensory integration deficits and vestibular dysfunctions (We refer mainly to recent studies for two reasons. First, these studies use current diagnostic criteria [*Diagnostic and Statistical Manual of Mental Disorders, International Classification of Diseases of the World Health Organization*]. Second, an extensive historical overview is beyond the scope of this article. The reader can obtain more information in the cited articles). We also discuss the potential implication of the TPJ in these syndromes.

Psychiatry

Psychosis

Aberrant bodily experiences have been described as core features in schizophrenia [26,27] (see [28–30] for recent overviews). Schneider [31] described delusional perceptions and somatic and passivity experiences as first-rank symptoms of schizophrenia; they also have been referred to as a loss of "Meinhaftigkeit" (mineness). Recent descriptions of body-image aberrations in patients with schizophrenia include perceptions of alterations in the size and shape of the own body, feelings of bodily unreality, merging of the body with external objects, and of the own body as not belonging to oneself [28]. Röhricht and Priebe [30] described enhanced body sensations of numbness and/or stiffness, desomatization, abnormal pain, emptiness, heaviness, lightness, falling and/or sinking, levitation and/or elevation, diminution, shrinking, enlargements, or constrictions (see also [27]). A major part of these symptoms also have been described by neurologic patients with AP [5,8••].

Angyal [27] presented a patient with schizophrenia perceiving an "alter ego" (or Doppelgänger). APs have been considered visual pseudohallucinations [8••,14••] and may be conceptualized as just another positive symptom in psychosis [32•]. However, Blackmore [33] and Röhricht and Priebe [29] found no evidence for a higher prevalence of OBEs in patients with schizophrenia when compared with healthy control subjects.

A recent study reporting somatic delusions in psychosis included AP as a distinct category [32•]. The authors assessed somatic delusions in many patients with depression, mania, chronic schizophrenia, and acute schizophrenia, respectively. The finding most relevant to this article is the observation that only acute psychotic patients reported loss of boundary (3.2% of psychotic patients; e.g. "other bodies intermingled with mine") and AP (2% of psychotic patients; "standing outside myself looking at myself" [OBE], or "can see inside myself from a height "[OBE-like]). These findings from McGilchrist and Cutting [32•] may point to a link between AP and schizophrenia, but only when patients have acute psychotic symptoms.

Further support for a link between positive psychotic symptoms and AP has been provided from healthy schizotypal individuals [34,35]. These authors observed that healthy individuals, who have experienced an OBE at least once in their lifetime, also reported higher positive, but not negative, schizotypal thoughts. Moreover, those who experienced OBEs also were those who were more prone to experience other positive "psychotic-like" experiences, such as hallucinations and involuntary imagery in situations of mild sensory limitation and physical relaxation compared with subjects who never experienced an OBE [34]. The relationship between OBEs and positive "psychotic-like" experiences is additionally supported by studies that have shown that people with OBEs also had elevated paranormal belief scores [36] and reported hallucination-like experiences more frequently [37••] (additional information linking OBE with personality, behavior, or drug use can be found elsewhere [38,39]).

Based on the previously described neurologic data, we also expected to find multisensory and vestibular deficits specifically related to positive psychotic or schizotypal symptoms and/or to the TPJ. Unfortunately, reports on multisensory integration in schizophrenia (see [40] for review), particularly multisensory integration of bodily information, are relatively sparse (see [41,42] for reviews). In the study by Spence *et al*. [41], patients showing passivity symptoms (such as loss of agency and/or alien control) were asked to move a joystick with the right hand to the sound of auditory stimuli. Compared with normal control subjects and patients without passivity symptoms, this patient population showed a hyperactivation of right inferior parietal lobule and cingulate gyrus. Farrer *et al*. [42] tested action attribution in patients with Schneiderian first-rank symptoms. The task required self-other decisions about seen hand movements on a computer screen. Crucially, the spatial match between own hand position and the one seen on the screen was distorted gradually. Supporting previous reports that the right inferior parietal lobule is involved in the attribution of action to another agent [43], increased brain activity in the right angular gyrus was observed in the normal participants with increased deviance of seen hand positions from their own hand positions. In first-rank patients, this relationship was absent or much weaker and correlated positively with first-rank symptoms. The authors also reported that the lack of increase in activation caused by increasing degrees of distortion among in these patients was associated with an abnormally high level of activation in the perfectly matching condition (an increased activity that only appeared for the "other" condition). Therefore, the patients showed high activity in the right angular gyrus when they experienced being the agent of their actions, whereas this activation in normal control subjects is only seen when agency is allocated to another person.

Even less is known about vestibular dysfunction in schizophrenia. Early reports described a reduced reactivity of the vestibular system in (mainly catatonic) patients with chronic schizophrenia [44]. However, a subsequent review disqualified vestibular dysfunctions of peripheral or central origins in schizophrenia [45]. However, as argued for a relationship between psychosis and AP, vestibular dysfunctions may only be relevant when patients show acute symptoms [46]. To our knowledge, there are no research findings applying contemporary methodologies to study the vestibular system in positive psychotic patients.

The lack of consistent reports of AP in schizophrenia may be explained as follows. First, AP have not yet been related systematically to positive symptoms in schizophrenia because studies rarely distinguished between acute and chronic symptoms and medication. Second, the lack of reports of AP in psychosis may be the result of the fact that

clinicians do not ask their patients about these not well-known experiences (see [47] for a similar argument on olfactory hallucinations). Lastly, for patients with active psychotic symptoms, AP experiences may be just another odd experience that they do not consider worth mentioning.

Depression, anxiety, depersonalization, and body dysmorphic disorders

As will be shown in more detail below, depression, anxiety, depersonalization, and body dysmorphic disorders are frequent comorbidities [48–50]. Moreover, they also have been linked to (acute) psychosis and aberrant body experiences. Therefore, within the scope of the current report, it seems appropriate to treat them together. Our goal is to elucidate whether these syndromes may represent "AP-prone" subpopulations and whether multisensory and vestibular processing deficits and an implication of the TPJ have been described.

A link between AP and the latter syndromes has been hypothesized previously [17,51–54]. Chapman *et al*. [28] noted that patients with schizophrenia do not only have more body image aberrations as compared to normal control subjects, but that body image aberrations increased as a function of individuals' scores on the Beck's depression inventory. Dening and Berrios [17] reviewed patients with AH and HAS and found that more than 50% of patients studied suffered from depression followed by schizophrenia. Anxiety was comorbid in approximately 30% of patients with depression. Although information about depersonalization was not available for many patients, 27% of the AP patients for whom this information was provided received the additional diagnosis of depersonalization.

Aberrant body experiences are phenomenologically reminiscent of AP and are common in patients with depersonalization and body dysmorphic disorders. Murray and Foxe [55] have shown that healthy subjects with OBEs score higher on the somatoform dissociation scale and the body satisfaction scale. Depersonalization and body dysmorphic disorders were formerly classified as anxiety disorders, whereas today, depersonalization is classified as a dissociative disorder and body dysmorphic disorders are classified as somatoform disorders [56]. Criteria of depersonalization include persistent or recurrent experiences of feeling detached from one's mental processes or body [56]. Criteria for body dysmorphic disorders include obsession about the size and shape of different body parts [56]. Although they are considered to be distinct syndromes, both are closely related with anxiety and depression [30,50,57]. Röhricht *et al*. [30], for instance, reported a significantly increased amount of body dissatisfaction, high amount of somatic complaints, somatic depersonalization, and boundary loss in patients with depression and anxiety as compared with normal control subjects. In a study testing a large population of patients with depersonalization disorders, Axis I comorbidity of anxiety and depression were the most prevalent [58].

With respect to multisensory and vestibular deficits in these syndromes, multisensory integration deficits have been reported in patients with state and trait anxiety [59,60]. Vestibular dysfunctions have been associated with depression, anxiety, and depersonalization [49,61,62]. Anatomically, anxiety, depression, depersonalization, and body dysmorphic disorders have been linked with the heteromodal association cortex such as the TPJ [57,63–67]. Simeon *et al*. [57] tested brain metabolism in eight patients with depersonalization and 24 healthy control subjects using positron emission tomography. The most important finding was that patients showed higher metabolic rates in parietal and temporo-parietal cortices. Moreover, there was a positive correlation between the relative glucose metabolism and the degree of dissociation and/or depersonalization. Using lesion analysis, Sierra *et al*. [63] observed depersonalization symptoms in a patient subsequent to a right subdural hematoma predominating in the right parietal lobe. Lastly, Osuch *et al*. [65] used positron emission tomography in medication-free patients with depression. Their results showed that these patients' state anxiety correlated inversely with metabolic rate at the TPJ (at the angular gyrus).

Multisensory integration deficits, vestibular dysfunctions, and an implication of the TPJ have been reported in patients with anxiety, depression, depersonalization, and body dysmorphic disorders. This phenomenologic and experimental evidence suggests that these syndromes may be promising research targets to further our understanding of the functional mechanisms (multisensory and vestibular processing) and neural mechanisms of AP at the TPJ and other brain areas. Unfortunately, the comorbidity of the syndromes (including psychosis) makes a more refined selection of AP-prone individuals almost impossible. For instance, anxiety has been related to schizophrenia [26,68] schizotypy [69], depression [70], dissociation and/or depersonalization [58], and body dysmorphic disorders [71]. Furthermore, depression and depersonalization are associated with the schizophrenia spectrum [72,73], especially for patients during an acute phase of their illness [74]. However, it may be suggested that anxiety is the strongest predictor for AP given the important role of vestibular dysfunctions in anxiety [62]. Given the presumable role of anxiety as discussed previously, we suggest that anxiety is most closely linked to the form of AP that is characterized by complete disembodiment (as in OBE), also attributable to the vestibular involvement in anxiety and OBEs. However, AH may instead be linked to depersonalization and body dysmorphic disorders, in which multisensory body-related information processing is disturbed without the significant implication of vestibular dysfunctions.

Conclusions

The current review indicates that a more detailed questioning of patients with AP by neurologists and psychiatrists is mandatory. To differentiate between the different forms of AP and their respective incidence in the different syndromes, future studies need to refine the questions that are asked about AH, HAS, and OBEs. Most studies to date only used Palmer's question ([75] question 23): "Have you ever had an experience in which you felt that "you" were located "outside of" or "away from" your physical body; that is the feeling that your consciousness, mind, or center of awareness was in a different place from your physical body?" McCreery and Claridge [37••] questioned the specificity of Palmer's question, stating that this question includes a wider range of unusual experiences different from OBEs. Therefore, to differentiate between AH, HAS, and OBE, future investigators should not only specify disembodiment, but also ask about autoscopy (Does the subject experience seeing her own body during the AP?) and the subject's visuo-spatial perspective during the experience (Does the subject experience seeing her own body and space from her habitual body-centered perspective or from an elevated and distanced extracorporeal perspective?). The accurate description of these three main phenomenologic characteristics of AP will not only allow to differentiate between the different AP-forms (AH, HAS, and OBE), but also between AP and other phenomena such as depersonalization or the feeling of a presence [76].

In science the most challenging phenomena often are the ones we take for granted in our everyday lives. Excellent examples are the self and the experienced spatial unity (between self and body). Folk and psychologic notions are challenged by AP. The reviewed evidence from neurologic and psychiatric patients experiencing these striking dissociations between self and body suggests that AP are culturally invariant phenomena that can be investigated scientifically. The neuroscientific study of the self is in its infancy and there are currently no established models, very little data, and often not even the vocabulary to describe neuroscientific notions of the self [1]. The investigation of AP in specific neurologic and psychiatric populations and their neural mechanisms may allow improvement of our neuroscientific models of self and corporeal awareness.

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