CHAPTER

18

Disorders of body representation

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Introduction

Our brain constantly receives and sends a flow of multisensory information including proprioceptive, tactile, visual, vestibular, auditory, olfactory, visceral, and motor-related signals. The integration of these signals into multisensory representations is responsible not only for the way our body is represented but also for the way it is consciously experienced. Conscious body experience includes the experience that a "real me" "resides" in "my" body and perceives the world from the perspective of that body, a phenomenon called bodily self-consciousness or corporeal awareness.[1,2] Recent studies in cognitive neuroscience have shown that it is possible to modulate bodily self-consciousness by experimentally manipulating these multisensory bodily signals (see Chapter 8). During such manipulations, healthy individuals may transiently experience (1) ownership of another body or body part (i.e., self-identification), (2) changes in where they feel their body located in space (i.e., self-location), or (3) modulation of the perspective from where they perceive the world (i.e., first-person perspective). These different experimental protocols have provided valuable insights into the neural mechanisms that generate and modulate bodily self-perception in the normal brain.

Another strategy consists in studying the phenomenology of individuals presenting altered perceptions of their bodies. Disorders of body representation take various forms and have a rich phenomenology, with impacts on distinct body parts and different levels of disorder awareness. Disorders may be limited to the upper limb, relate to internal organs, or involve the whole body. Moreover, patients may report an "absence" of a body part,

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describe supernumerary phantom limbs, or perceive a double of themselves in extrapersonal space. Finally, some patients notice distorted or unusual body representations, sometimes being critical and able to rationally describe how they perceive their body as abnormal.[3] Others are rather indifferent (i.e., anosognosia) or hold false beliefs regarding the very existence of an alteration (i.e., delusion).

In this chapter, we will review the main clinical alterations of body representation. First, we will describe instances of altered body representations in neurological conditions, either constrained to a specific part or impacting the whole body. In the second part, we will present body representation disorders associated with other diseases, namely chronic pain and psychiatric conditions.

Neurological disorders of body representation

Unilateral disorder of body representation

One of the most common cases of altered body representation in neurology is the perceived absence of a body part, as if it was not part of the body, or at least not completely. This entails inattention toward a given body part (i.e., personal neglect), the vivid sensation that a body part has disappeared (i.e., the feeling of amputation), or the misattribution of a limb to someone else (i.e., somatoparaphrenia). In contrast to these cases in which body perception is diminished, some disorders of body representation imply abnormally increased bodily percepts, like sensations in a nonexisting limb (i.e., phantom limbs and supernumerary phantom limbs) or overestimation of perceived body size (macrosomatognosia). In the following section, we describe the main unilateral disorders of body representation.

Personal neglect

The term personal neglect was coined by Zingerle[4] in reference to a neuropsychological disorder characterized by the inattention toward one part or an entire half of the body[5,6] (see also Chapter 19). Personal neglect typically concerns the left body side and is associated with right hemispheric brain lesions. The clinical manifestations indicative of personal neglect are indifference, forgetfulness, or unawareness for the left hemi-body. Classically, patients forget to comb, shave, or make up the left side of their face or leave their left foot out of the wheelchair rest. Although inattention is striking at the behavioral level, patients are not aware of their deficit and do not report the unattended body part as missing from their body representation. In contrast to somatoparaphrenic patients (see next section), patients with personal neglect do not manifest disownership for the affected hemi-body, and acknowledge under request that the disregarded body part belongs to them, even if they behave as if it did not exist.

Lesion analysis in patients with personal neglect revealed the role of the right inferior parietal cortex including the supramarginal and postcentral gyri.[6] Lesions were also found in the underlying white matter, suggesting that neglect may result from a disconnection between the postcentral gyrus coding for proprioceptive and somatosensory signals, and areas linked to more abstract body representations. Subsequent lesion analyses have confirmed the importance of parietal regions and underlying white matter, extending to temporal areas.[7,8]

Feeling of amputation, hemi-depersonalization

Neurological patients may experience the sensation from a body part as numbed or completely absent. As opposed to personal neglect, patients fully appreciate the illusory nature of their sensation. This disorder is considered as the reverse of the well-known phantom limb sensation experienced by most amputees (see below). Other related phenomena include the feeling that a limb is no longer attached to the rest of the body, as if it were floating at some distance, or the feeling that the whole body is split into two halves.[9,10] These symptoms are usually of short duration and appear mostly during epileptic seizures, migraine events, or vascular stroke affecting premotor, primary motor, or parietal cortex, as well as subcortical structures of either hemisphere.

Somatoparaphrenia

The term somatoparaphrenia was introduced by the neurologist Joseph Gerstmann[11] in reference to patients presenting an abnormal sense of disownership for their contralesional hemi-body. Somatoparaphrenic patients claim that their own limb does not belong to them, and more explicitly that it belongs to someone else like the doctor, a nurse, a roommate, or some undetermined person.[12] Somatoparaphrenia is characterized by a distal-to-proximal gradient, with greater prevalence for the hands, followed by an entire limb (arm/leg) and only rarely the whole hemi-body. Patients can display strong emotional reactions and develop feelings of hostility against the affected body part, manifested as verbal or physical aggressive behaviors (i.e., misoplegia). Most of the reported cases of somatoparaphrenia involve extensive frontotemporoparietal lesions, with a prominent role of the temporoparietal junction (TPJ) in the genesis of the delusion.[12] More sporadically, deep cortical regions such as the insular cortex or subcortical regions including the basal ganglia have also been involved.[13,14]

Phantom limbs and supernumerary phantom limbs

The majority of amputees experience persistent and vivid sensations in their physically absent limb, referred to as a "phantom limb".[15–17] The phantom limb is usually clearly perceived and is similar in shape, size, and posture to the physical limb before amputation, although distorted perception of the phantom limb can also occur (see Ref.18 for review). In rare cases, "supernumerary" phantom limbs are experienced by nonamputated patients, described as an additional body part, felt as an entity, and sharing the properties of the real body.[19–21] Supernumerary limbs are mostly perceived on the same side as a paralyzed limb and typically remain immobile although movements have been occasionally described.[22] Supernumerary phantom limbs have been reported following lesions of the basal ganglia,[23] capsulolenticular region,[22,24] thalamus,[19] supplementary motor area,[25] bilateral parietal lobe,[26] spinal cord,[27] and following motor cortex stimulation.[28] In all cases, the reduplicated physical body part is always injured, deafferented, or paretic. Some authors have proposed that supernumerary phantom limbs are due to a mismatch between the perceived paretic or deafferented limb and its brain representation.[29]

Macro- and microsomatognosia

In rare occasions, some patients misperceive the size and weight of specific body parts. Microsomatognosia refers to the subjective experience of perceiving a body part as smaller

than usual, whereas macrosomatognosia is used for patients describing a limb that is increased in size and often in weight.[30] Frederiks[30] proposed that such misperception is typically paroxysmal, occurs in both halves of the body, and occurs in an unclouded mind. Similarly to what is observed for supernumerary phantom limbs, patients with macro- or microsomatognosia are usually fully aware of the illusory nature of their percepts. Typical causes include migraines and epileptic seizures.[31] Rare cases have been reported following toxoplasmosis or typhoid infections, mesencephalic lesions, and damage to sensorimotor structures in either hemisphere.[30]

Global body representation disorder

Most of the body representation disorders described so far can conceptually be extended to the full body. For instance, macrosomatognosia can concern the entire body in patients with Alice in Wonderland syndrome, who have an erroneous perception of their whole body size with respect to the external environment.[32] Similarly, extreme forms of depersonalization in which patients claim to be nonexistent or dead (i.e., Cotard's syndrome)[33] can be considered as an equivalent of the feeling of amputation described earlier, but concerning the whole body.

In the next section, we will focus on a particular form of full-body hallucination in which patients experience illusory duplications of their own body. Most duplications are predominantly visual, commonly referred to as "autoscopic phenomena" (i.e., autoscopy, heautoscopy, and out-of-body experience (OBE)), but can also be nonvisual, like in the feeling of presence (sensorimotor phenomenon; i.e., Ref. 34).

Autoscopic hallucinations

During an autoscopic hallucination, people experience seeing an image of themselves in the extrapersonal space (i.e., the space that is far away from the subject and that cannot be directly acted on by the body), as if they were looking into a mirror, without the experience of leaving their body (i.e., no disembodiment). Patients with autoscopic hallucinations see the world from their habitual perspective and their "self" is perceived as located inside their physical body. Therefore, autoscopic hallucinations are mostly visual phenomena (with multisensory components), with no change in the bodily self. They usually last a few seconds or minutes and may occur repeatedly. A few case studies have reported persistent autoscopic hallucinations over time with a visual double present over months and even years.[35,36]

Visual field deficits and visual hallucinations are frequently associated with autoscopic hallucinations.[37,38] Based on this observation, it has been proposed that autoscopic hallucinations relate to visual deficits including abnormal visual imagery or defective plasticity following a lesion in the visual cortex.[36] Others have proposed that this phenomenon is linked to defective multisensory integration of signals from vision, proprioception, and touch.[37,39,40]

Autoscopic hallucinations can occur after neurologic disorders such as migraine and epilepsy as well as brain damage in the occipital and/or parietal lobe.[36,40,41] A recent quantitative lesion analysis study investigated the brain correlates of autoscopic hallucinations in a group of seven patients. The authors found that damage affecting the superior occipital gyrus and the cuneus in the visual cortex of the right hemisphere was involved.[42]

Heautoscopy

Unlike autoscopic hallucinations, people experiencing heautoscopy self-identify with a body seen in the extrapersonal space. It is usually difficult for the observer to determine whether he/she is disembodied or not and whether the center of conscious experience (i.e., the self) is localized within the physical body or in the autoscopic body.[41] During heautoscopy, patients may even experience so-called bilocation (i.e., the feeling of existing at two places at the same time), often associated with the experience of seeing from different visuospatial perspectives (i.e., from the physical and autoscopic bodies), in an alternating or even simultaneous fashion.[41–45] This phenomenon can be considered as an intermediate form between autoscopic hallucination (where the self is located in the physical body) and OBE (see next section) (where the physical body is completely abandoned by the self).

Heautoscopy has been reported in patients suffering from parietal or temporal lobe epilepsies, neoplastic lesions of the insular cortex, and migraine in association with a psychiatric disorder.[44,46–49] A recent lesion study found that heautoscopy was associated with damage to the left posterior insula.[42] Patients with heautoscopy often present altered perception of visceral information such as palpitation,[45] which is in line with the involvement of the insular cortex and its role in interoceptive processing and the encoding of emotionally relevant information for self and other.[50–52] Further corroborating the link between insular cortex, interoception, and heautoscopy, a recent report described a patient with a selective right insular tumor in whom a mild form of heautoscopy, including bilocation and body reduplication, could be experimentally induced based on cardiovisual stimulation (i.e., participants observe a virtual body illuminated in synchrony with their heartbeat).[53]

Out-of-body experience

An OBE can be defined as a waking experience combining disembodiment (i.e., the feeling of being outside one's physical body), elevated perspective (i.e., the perceived location of the self at a distanced and elevated visuospatial perspective), and autoscopy (i.e., the experience of seeing of one's own body from this elevated perspective). Subjects experiencing OBEs always localize the self outside their physical body, usually as if located in an elevated position looking at the physical body on the bed or the ground.[41,54]

OBEs have been reported predominantly in patients with epilepsy and migraine.[54] Although many brain regions have been linked to OBEs (e.g., frontotemporal cortex,[48] parietal lobe,[55] temporal lobe[56]), the TPJ seems to play a crucial role[40,41,41a], with a right hemispheric predominance.[57] Notably, electrical stimulation of the right TPJ induced an OBE in a patient presenting with intractable epilepsy.[58] Other cases of OBEs induced by brain stimulation of the TPJ have been reported.[59] Importantly, OBEs are not only found in clinical populations but also appear in approximately 5%-10% of the healthy population across the majority of the world's cultures.[60] A variant of OBE called the full-body illusion can be experimentally induced in healthy volunteers by providing conflicting multisensory signals (Ref. 61; see Chapter 8, for further details). Brain imaging during this illusion confirmed the role of the TPJ for OBEs.[62]

With respect to other autoscopic phenomena, OBEs are characterized by specific vestibular sensations.[41,63] These are feelings of elevation, floating, and a 180 degrees inversion of the

body and its visuospatial perspective in extrapersonal space. Otolithic dysfunctions are therefore likely to contribute to OBEs.[1] In addition to these vestibular disturbances, OBEs are sometimes accompanied by paroxysmal visual body–part illusions such as supernumerary phantom limbs and illusory limb transformations.[41,48,55,58] These observations suggest that visual illusions of body parts and autoscopic phenomena may share similar neural origins.[45] Based on the association of OBEs with visuo-somatosensory illusions, abnormal vestibular sensations,[63] and the well-known role of the TPJ in multisensory integration,[63a,63b] it has been proposed that OBEs are caused by disturbed multisensory integration of bodily signals.[1,41]

Feeling of presence

Initially described by the psychiatrist Karl Theodor Jaspers,[64] the "feeling of a presence" (FoP) refers to the distinct feeling of the physical presence of another person or "being" in the near extracorporeal space although nobody is actually around.[45] Importantly and in contrast to autoscopic phenomena, this illusion is not experienced visually as the person is "sensed" but usually not seen. This "presence" can be felt behind, sideways, or in front of one's physical body and may even involve multiple entities.[47] Authors have named this illusion of a sensorimotor double "hallucination du compagnon,"[65] idea of a presence[66] or presence hallucination[66a]. The FoP has been described in several psychiatric conditions,[34,64,66–68] neurological patients suffering from epilepsy, stroke, or Parkinson's disease[67,69,70] and healthy individuals mostly during periods of physical exhaustion.[64,66,67]

The mechanisms underlying the FoP are the topic of recent research, highlighting the role of multisensory integration and body representation. Electrical stimulation of the TPJ induced FoP in a single case study during presurgical investigations.[71] This finding was recently confirmed by a lesion analysis study in 12 FoP patients: focal brain lesions overlapped in the temporoparietal, frontoparietal, and insular cortex (of either hemisphere).[72] Additional analysis in control patients revealed that from the three lesion-overlap zones only the frontoparietal site was specifically associated with the FoP. Interestingly, the temporoparietal cortex,[62] insula,[73] and frontoparietal cortex[74] are known to integrate multisensory bodily signals and are considered as neural loci of bodily self-consciousness. As for OBEs, mild forms of FoP can now be induced noninvasively in healthy volunteers. Using a robotic system generating specific sensorimotor conflicts, Blanke and collaborators were recently able to experimentally induce the FoP and related illusory own-body perceptions.[72] In this experiment, blindfolded participants moved a master robotic device in front of them while receiving delayed tactile stimuli on their back. During such spatiotemporal mismatch between motor–proprioceptive signals (participant's movements in front of them) and their sensory consequences (tactile feedback on their back) subjects reported being in the presence of another person behind them and being touched by this invisible presence. A prominent model for motor control and bodily experience posits that in self-generated movement, efference 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 self-generated experience.[75,76] Collectively, these suggest that the FoP might be the consequence of a misperception of the source and identity of signals of one's own body.[72]

Body representation disturbance in chronic pain

More than any other sensation, pain is inextricably linked to the body, which constitutes the reference and the object of any painful sensation.[77] Neuroimaging studies have shown that the link between body and pain underlies a partial overlap and mutual connections between central pain representations (the so-called pain matrix; i.e., a network of brain areas activated by nociceptive inputs including brainstem and thalamic nuclei, primary and secondary somatosensory areas, insular, and anterior cingulate cortices) and central body representations (i.e., the body matrix, a network of multisensory regions processing bodily related inputs, such as the posterior parietal cortex, the somatosensory cortices, and the insula).[78] This link at the neural level is supported by behavioral evidence in patients experiencing pain over a prolonged period and beyond the expected time for healing (i.e., chronic pain), who also demonstrate abnormalities in their body representation. Indeed, patients with chronic pain often misperceive their affected body part in size or shape, reporting feelings of foreignness, strangeness, or even hostility toward the painful limb. In the following section, we present and discuss the main changes in body representation occurring in three different chronic pain states, namely complex regional pain syndrome (CRPS), phantom limb pain (PLP), and spinal cord injury (SCI).

Complex regional pain syndrome

CRPS is a chronic pain condition usually affecting one limb, characterized by pain in combination with sensory, autonomic, trophic, and motor abnormalities.[79] Body perception disturbances are frequent in such patients who, for instance, report their affected limb to be larger than it really is.[80] In addition to size distortions, some patients also demonstrate disturbances in how they perceive the shape of their affected body part, for instance, describing a missing segment in the affected limb or having difficulties in determining its position.[81] Moreover, patients with CRPS have reduced abilities to determine the laterality of pictured hands, implying the existence of underlying altered spatial representations; the degree of this disturbance is directly influenced by the intensity of pain.[82,83]

Patients with CRPS show an important cortical reorganization with reduced representation of their affected limb in the primary sensory and motor cortices. Studies in the CRPS population revealed that the amount of cortical reorganization directly correlates with pain intensity and that these cortical changes are normalized during recovery.[84–86] However, the directionality of this link is unclear, and whether these cortical changes cause or are caused by chronic pain remain to be tested.

An interesting clinical feature of CRPS is that patients tend to neglect their affected limb and report finding their hand "foreign," "strange," or "as if someone had sewed a foreign hand on it."[87–89] This clinical manifestation observed in more than half the patients is called "neglect-like syndrome" and shares similarities with symptoms observed following right parietal damage. However, (1) this feeling of foreignness is observed independently of the affected side, (2) is not associated with hemispatial deficit (e.g., patients show no bias in the bisection task, see Chapter 19), and (3) patients are typically fully aware of their deficit and realize the irrational nature of their feeling.[87] Together, these three points make the

neglect-like syndrome distinct from the conditions of personal neglect and somatoparaphrenia described in Unilateral disorder of body representation section.

Phantom limb pain

A striking example of body misperception in a clinical population is phantom limb sensation, defined as the sensation that a missing body part is still present. PLP occurs in up to 80% of amputees.[15,90] Over the past decades, several studies reported multiple cortical changes in PLP. Since seminal animal studies, it is indeed well established that cortical reorganization occurs following amputation, with an invasion of adjacent body part representations into the cortical representation of the deafferented body part.[91] For instance, upper limb amputees show a shift of their facial representations in somatosensory and motor cortex into the digit and hand area.[92,93] More controversial is the relation between such cortical reorganization and chronic pain. Some authors reported that amputees with PLP have a greater shift of their mouth cortical representation into the hand area in motor and somatosensory cortex than amputees without pain.[94] Moreover, this cortical reorganization appeared to be correlated with the level of pain.[95] Based on the relation between the degree of cortical reorganization and the level of pain, Flor and colleagues have proposed maladaptive changes as the neural basis of PLP.[96] More recently, it has been claimed by Makin and colleagues that PLP maintains local cortical representations but disrupts interregional connectivity.[97–99] For instance, it was reported that functional connectivity between the representation of the missing limb and the rest of the sensorimotor network is decreased.[98] A recent study furthermore showed that somatosensory regions are functionally disconnected from the posterior parietal cortex in amputees, the latter being a key region for the integration of multisensory bodily signals.[100] Collectively, these results underline the role of cortical body representations (unimodal or multimodal) in PLP.

As for the previous disorders, there is a clear link between PLP and altered body representation. For instance, amputees typically report their missing limb as heavy, swollen, stuck in a given position, or shortened.[18,101] The feeling of telescoping is a commonly reported symptom with significant association with PLP, where patients experience their phantom has shrunken with just the more distal portion floating near, attached to, or "within" the stump.[16,96,102] It is estimated that about 50% of amputees perceive their phantom limb to be telescoped; the telescoping process generally begins within the first few weeks postamputation.[15,103] Some authors have proposed that telescoping originates from the disparity in brain representation of the different limb segments, with an overrepresentation of distal (i.e., the hand) compared with proximal parts.[15] Neuroimaging data showed that telescoping is associated with cortical reorganization in which distal representations invade brain regions representing proximal body parts. For instance, imaginary movement of a completely telescoped phantom arm induces activity in the shoulder area.[96]

Based on the hypothesis that cortical reorganization and phantom pain are related, a range of novel therapies have been developed to diminish PLP by targeting maladaptive cortical reorganization. These include sensory[104] and motor training,[105] peripheral[106] or cortical stimulation,[107] or combined visuomotor stimulation using a mirror box setup[92] (see Chapter 20).

Spinal cord injury

Spinal cord damage can cause permanent loss of sensorimotor function and, in about 65% of patients, chronic neuropathic pain.[108,109] Similarly to amputees, SCI patients may experience vivid phantom sensations in the deafferented body part.[17,110,111] However, they commonly describe their phantom occupying anatomically unrealistic and unnatural postures: for example, patients may feel that their legs are "twisted" or "blown up." They may perceive their "toes turned down under the bottom of the foot" or their digit somehow twisted so that "each toe pointed in a different way."[110] Moreover, patients often report their phantom to be larger than the actual in size or in movement.[110–112] This is in contrast with phantom limb sensations in amputees, which occur in a plausible body space and are reduced in size (i.e., telescoping).[18,111]

Several studies have demonstrated that functional and structural cortical reorganizations occur following SCI.[113–115] These changes are in line with the modifications described in amputees, that is an invasion of the adjacent cortical representation into that of the deafferented body part. Neuroimaging studies showed shifts of functional motor and sensory cortical representations that relate to the severity of SCI.[116] Moreover, these cortical changes also appear significantly correlated with ongoing pain intensity levels in SCI.[117] Recently, Scandola and colleagues meticulously examined bodily misperceptions in a group of 49 patients with SCI.[118] They reported various corporeal illusions involving body form (sensations of body loss and body-part misperceptions), body motion (illusory motion), and body ownership (disownership-like feelings and somatoparaphrenia-like feelings) that were related to neuropathic pain. The authors hypothesized that these body misrepresentations reflect uncontrolled neuroplastic changes.

Based on the observation that multisensory processing and body representation are impaired in SCI patients,[119,120] a recent study investigated how body ownership and neuropathic pain can be modulated by multisensory stimulation. Using immersive virtual reality (VR), Pozeg and colleagues manipulated the sense of leg ownership and global body ownership in SCI patients applying synchronous visuotactile stimulation (i.e., creating a virtual leg illusion[121] or full-body illusion[61]). Compared with healthy subjects, SCI patients showed reduced sensitivity to multisensory stimulation inducing illusory leg ownership but preserved ability in global ownership manipulation. In addition, leg ownership decreased with time since SCI. This study, among others, suggests that manipulations of bodily self-consciousness are likely to be of high relevance to alleviate pain, given that these effects were achieved after even short periods of multisensory VR exposure.

Body representation disturbance in psychiatric disorders

Representations of the body are altered in a number of psychiatric conditions. Here, we review studies on anorexia nervosa (AN) and schizophrenia (SZ), two conditions for which a great deal of research has been conducted on body representation. We will also briefly discuss alterations in body representation seen in gender dysphoria (GD).

Anorexia

Patients with AN show extreme dissatisfaction with their body size, despite being underweight. There is a long-standing debate about whether this dissatisfaction is purely cognitive-affective or whether there is also a perceptual distortion of body size.[122] This distinction fits with current conceptualizations of body representation in the brain as shaped both by bottom-up sensory input and by top-down cognitive, semantic, and affective representations[123]. An abundance of data confirm differences in the former, "attitudinal," component of body image in AN.[124] Across studies of AN, attitudinal body dissatisfaction shows a larger effect size than visual distortion[125] and is observed in more studies and more patients.[126,127] Yet there is evidence of perceptual body distortion in AN as well. We will focus on this perceptual component of body representation, while acknowledging that the affective component plays a prominent role in AN.

The majority of research on body representation in AN has probed visual body representation. Numerous studies have examined visual estimations of body size by asking patients to draw the width of their body, select a body outline matching their shape, or adjust a photograph, mirror, or video image until it is perceived to be the patient's size. A number of studies report visual body size differences in AN[125]. Some observe these measures to be positively correlated with attitudinal measures of body dissatisfaction,[125,128,129] suggesting a causal relationship of some kind between these components of the body image. Quite a few other studies, however, do not observe distortions in the visual body image or observe it only in a subset of patients.[127,130] The presence of visual body distortion in AN is thus controversial and certainly not universal, suggesting that it is not the primary cause of body image dissatisfaction. AN patients have been found to show selective deficits in visually processing upright—but not upside-down—bodies, suggesting difficulties with configural processing that may be related to a more detail-oriented approach to viewing bodies.[131]

Brain imaging has also been used to investigate visual body representation in AN. An occipitotemporal pathway including the extrastriate body area (EBA) and fusiform body area is key to detecting body-related information, while a parietofrontal pathway is closely linked to body identification and self-other discrimination.[124] Differences in processing visual images of bodies have been found in individuals with AN in the body-shape processing network,[132–134] as well as in the insula, for self-images.[135,136] Mohr and colleagues suggest that difficulty retrieving multimodal body image representations from the precuneus and posterior parietal cortex may underlie deficits in body size estimation.[135] In addition, visual body shape comparison tasks show more activation of right hemisphere sensorimotor regions in AN, including hyperactivation of the insula, but hypoactivation of the anterior cingulate cortex. This finding may relate to altered interoceptive or motivational processes in AN.[124] Finally, alterations in the structure of the EBA, located in the lateral occipital cortex, have been observed.[137]

While researchers have traditionally focused on visual body distortion in AN, more recent research efforts have turned to somatosensory body representation. Most studies of primary tactile perception do not find deficits in AN, although slight deficits in more difficult versions of a finger identification task have been documented in AN patients before treatment.[138] However, several studies have reported differences in secondary tactile perception, which involves perceptual scaling of a tactile stimulus to compute and represent its size (and other

characteristics).[139] Gaudio and colleagues review evidence from 13 studies examining nonvisual multisensory alteration of body perception in AN and conclude that there are tactile and proprioceptive differences that may be associated with alterations in parietal cortex functioning in AN patients.[140]

Patients with AN overestimate the distances between points applied both on the arm and on the abdomen, suggesting an enlarged tactile body representation. Tactile overestimation is correlated with body dissatisfaction, suggesting a connection between tactile body maps and attitudinal aspects of body image.[141,142] To see whether deficits in primary tactile perception might underlie this effect, Keizer and colleagues studied touch detection and found a higher threshold for two-point discrimination on the arm and abdomen in AN, as well as a lower pressure detection threshold on the abdomen in patients with AN.[142] These inconsistent findings suggest alterations in primary tactile perception that may impact tactile body distortion. More recently it was found that tactile overestimation occurs only in the horizontal direction of the body, suggesting a warping of tactile body image by specific cultural body fears.[143]

Sensory information also comes from the inside of the body, through interoception. Several studies document difficulties with interoceptive awareness in AN. These difficulties include reduced sensitivities to sensations of hunger and satiety,[144–146] difficulty recognizing signs of physiological stress such as an increased heart rate[147,148] and altered processing of taste and pain.[149,150]

Patients with AN also show differences in integrating visual and proprioceptive information. The size-weight illusion (SWI) arises from visual and haptic comparison of two objects of equal weight but different physical size. Typically, the smaller object feels heavier due to an implicit expectation that weight is proportional to size. AN patients show a reduced SWI despite normal discrimination of mass, suggesting decreased integration of visual and proprioceptive information in AN.[151] This result could imply that individuals with AN have more difficulty taking their appearance (visual feedback) into account when judging their body size and might rely to a greater extent on internal sensory cues.

Haptic perception involves active sensorimotor exploration of the surface of an object. Deficits in integrating visual and haptic information are reported by Grunwald and colleagues, who found that patients with AN had difficulty drawing objects that they explore through touch[152] and reproducing angles through haptic perception.[153] Patients in this study also showed reduced parietal activation during this task.[152] In contrast, no deficits have been observed in haptic recognition of simple shapes.[154,155]

The rubber hand illusion (RHI) involves integrating visual and tactile input (see Chapter 8). Patients with AN show a stronger RHI than controls. Greater proprioceptive drift and greater embodiment of the hand both correlated with symptoms of AN.[156] The authors suggest these results indicate that the bodily self is more plastic in individuals with an eating disorder. Indeed, heightened malleability of the body persisted beyond recovery, suggesting a trait phenomenon.[157]

There is also evidence of altered sensorimotor and spatial orientation representations of the body in AN. Individuals with AN judged they would be unable to fit their body through an aperture that was easily wide enough,[158] showing distortions in body schema. Nico and colleagues found that AN patients showed selective distortions of their left body boundary when judging whether an approaching visual stimulus would contact their body. This performance paralleled that of right—but not left—parietal patients, suggesting alterations in right

hemisphere processing of the body schema.[159] Other investigators[155 160] demonstrated an effect of body tilt on the visual and tactile sense of verticality in AN patients, showing deficits in integrating visual, tactile, and gravitational information and using the body as a frame of reference. In contrast, another study[138] found no differences on cognitive and body-related spatial tasks in AN patients after treatment, and during acute illness found differences only when tasks required an executive function load in additional body schema–related processes. Body schema dysfunction may thus reflect broader cognitive dysfunction during acute states of AN. Stimulation of the vestibular system alters representation of body parts. Noting the high comorbidity of vestibular dysfunctions and psychiatric symptoms, Mast et al. postulate that the vestibular system plays an integral role in multisensory coordination of body representation and may also play a role in AN.[161]

In sum, individuals with AN show significant affective bodily dissatisfaction but also evidence of perceptual distortions in body representation. There is evidence of distorted bodily perception in visual, tactile, and motor domains as well as altered multisensory body representations. The causality of these distortions for affective body dissatisfaction and progression of AN is unclear.

Schizophrenia

SZ is a severe psychological disorder characterized by abnormal social behavior and unusual or confused thoughts. Common symptoms include "positive symptoms" such as hallucinations and delusions as well as "negative symptoms" such as reduced movement and emotional responsiveness. Cognitive neuroscience approaches to SZ have amassed evidence that core features of SZ may arise from cognitive dysfunction.[162] Cognitive and perceptual declines are found in most individuals with SZ; indeed, cognitive impairment is more common in SZ than psychotic symptoms.[163] Accordingly, disruptions in multisensory body perception may underlie certain symptoms of SZ (see also Chapter 17 by Cascio et al., this volume).

SZ is strongly associated with anomalous self-perception. Patients with SZ often experience problems with self-recognition and self-attribution of thoughts and actions.[164] A theme of blurred boundaries between self and other ties together many symptoms of SZ including auditory hallucinations, thought insertion, thought broadcasting, and the influence of others on the patient's thoughts, actions, or emotions. With regard to body perception, there is evidence of altered body structural description in SZ.[165,166] In addition, patients more frequently report feelings of strangeness toward their faces than healthy controls.[167] Bodily delusions and hallucinations are also not uncommon in SZ.

Perception of bodily touch in patients with SZ reveals altered multisensory representations and impairment of self-other distinction. Patients with SZ show reduced ventral premotor cortex response to observed touch of the body and abnormal responses to bodily touch and observed touch in the posterior insula.[168] The RHI has been found to be affected in SZ, with studies differing with regard to being stronger[169] or weaker[170] in SZ patients than in healthy controls, suggesting, at the very least, altered mechanisms of body representation that require further study. Multisensory perception of bodily movement is also disrupted in SZ. Results from a number of studies suggest that patients experiencing hallucinations or delusions of control frequently misattribute their own actions to others.[171] In healthy controls,

tracking of self- versus other-generated hand movements activates the angular gyrus and insula. SZ patients do not show this pattern, suggesting abnormal tracking of self-generating movement.[171] This may relate to the frequent experience in SZ of personal actions not feeling under one's control. Indeed, SZ patients experiencing feelings of alien control of self-generated movements show hyperactivity in the right inferior parietal lobule.[172]

Multisensory integration is a foundational capacity for a normal experience of self. A bottom-up account of SZ postulates that perceptual deficits impact higher-level cognitive processes whose disruption leads to symptoms of SZ.[173] For examples, Postmes and colleagues suggest that failures of multisensory integration may underpin disrupted experiences of self commonly seen in SZ such as depersonalization, diminished feelings of agency, and loose associations.[174] Many examples of deficits in multisensory integration have been found in SZ (see chapter by Cascio et al., this volume). Patients with SZ show reduced audiovisual binding and deficits in the network subserving audiovisual integration.[175,176] They also exhibit reduced facilitation of reaction time for detecting bimodal targets relative to unimodal targets, and those with more negative symptoms show the least degree of benefit from bimodal cues.[177] Relatedly, patients with SZ show impairments in recognizing whole-body expressions and impairments in integrating affective visual and vocal cues from the same source (such as a face or body along with a human vocalization).[178,179] The bottom-up account of SZ is also supported by functional brain imaging data showing disrupted resting state networks that particularly affected visual, auditory, and crossmodal binding networks. These disruptions were correlated with negative symptoms, positive symptoms, and hallucinations in individuals with SZ.[173]

In sum, patients with SZ show altered bodily perception and difficulties relating to distinguishing self from other. Differences in visual, tactile, and sensorimotor representation of the body have been observed. Multisensory integration is also altered. Problems with sensory binding correlate with many clinical symptoms of SZ and may play a causal role in these symptoms.

Gender dysphoria

People who are transgender experience a marked discrepancy between their experienced or expressed gender, and the gender assigned to them at birth. When this discrepancy causes significant distress or problems in functioning, it may be diagnosed in the DSM-5 as GD.[180] The biological mechanisms of GD are not known, and research in this area is nascent. Most studies have explored differences in brain structure in FtM (female to male) and MtF (male to female) individuals. Overall, these studies show a mixed pattern of masculine and feminine cortical thickness and white matter tracts, different from both cisgender men and women.[181] The incongruence between the perceived and physical body frequently leads to body dysphoria and body-related avoidance, such as avoidance of looking in the mirror.[180,182,183] For FtM individuals, breasts and genitals cause the greatest dissatisfaction.[184–186] Problematic areas for MtF individuals include genitals, face, and hair.[187] In contrast,[184] identify socially visible characteristics such as voice, hair, and muscularity as most predictive of overall body satisfaction. Most transgender individuals feel more like "themselves" and experience a more positive body image after physically transitioning their body to better align with their

gender.[188] Numerous studies document improved quality of life for transgender individuals following hormone therapy and gender-confirming surgery.[189]

Initial work on GD by Ramachandran and colleagues has found evidence of altered body representation aligned with gender identity. Some presurgical FtM individuals reported the feeling of having a penis, despite being clearly aware it is not physically present.[190] Ramachandran and McGeoch note a parallel to the experience of a phantom limb after amputation, suggesting that just as the neural representation of a body part lingers after it is removed, body maps in the brain might be altered to align with gender identity in individuals with GD. Indeed, FtM and MtF individuals may have lower rates of phantom breasts and penises after they are removed during a gender-confirming surgery than do cisgender individuals who have these body parts removed for other medical reasons, suggesting altered neural representation of these body parts before their removal.[190,191]

To test whether somatosensory processing is altered for incongruent-feeling body parts, Case and colleagues compared processing of tactile input to the breast in presurgical FtM individuals compared with cisgender female participants.[192] Breasts were rated as highly incongruent for all FtM men and genderqueer individuals in the study, but not for the cisgender women. Magnetoencephalography recordings of brain responses to tactile stimulation of the breast showed reduced response to the tactile input in the supramarginal gyrus and secondary somatosensory cortex, but increased activation at the temporal pole, near the amygdala, in the FtM group. No such differences were seen following tactile stimulation of the hand. These results suggest reduced sensory integration and more anxiety or alarm for sensation from this body part. Furthermore, altered white matter connectivity (measured by diffusion tensor imaging) was found in these same brain areas, suggesting that altered sensory processing could be related to underlying structural differences in these brain regions. These results suggest that the experience of bodily incongruence may include altered integration of tactile sensation.

Several groups have now examined differences in resting state connectivity in transgender individuals, as related to body representation. Lin and colleagues found that transgender participants showed higher centrality of the primary somatosensory cortex and superior parietal lobule, as well as greater recruitment of visual and auditory regions in the body network.[193] These results suggest greater multisensory influences on body representation in transgender individuals. Manzouri and colleagues found evidence that FtM individuals may have weaker connections between body perception networks and body self-ownership networks as well as reduced functional connectivity between regions involved in body perception and emotion.[194] A similar attempt to characterize functional connectivity in adolescents with GD identified sex-atypical connectivity patterns within the visual network, the sensorimotor network, and the posterior default mode network (DMN). Interestingly, these networks, which are sexually dimorphic between cisgender male and female adolescents, did not differ between prepubertal children with and without GD.[195] Feusner and colleagues also attempt to identify neurobiological correlates of the subjective incongruence between body and self in FtM individuals.[196] They report decreased connectivity within the DMN in FtM individuals as well as decreased connectivity in occipital and temporal regions. Furthermore, they report correlations between higher ratings of "self" for gendered body images and greater connectivity within the anterior cingulate cortex in FtM individuals. Similar to an earlier report,[192]

this study[196] suggests that individuals with GD may not incorporate physical traits of their assigned birth into their neural self-representation.

In sum, individuals with GD show high levels of body dissatisfaction, related particularly to sexually dimorphic body features. Individuals with GD show evidence that multisensory neural body representation is altered in the brain and is less connected with areas related to emotions and representations of "self." Further work is needed to investigate the neural representation of the desired body form and its impact on body image and body schema.

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