The role of self-touch in somatosensory and body representation disorders after stroke

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Somatosensory impairments occur in about half of the cases of stroke. These impairments range from primary deficits in tactile detection and the perception of features, to higher order impairments in haptic object recognition and bodily experience. In this paper, we review the influence of active- and self-touch on somatosensory impairments after stroke. Studies have shown that self-touch improves tactile detection in patients with primary tactile deficits. A small number of studies concerned with the effect of self-touch on bodily experience in healthy individuals have demonstrated that self-touch influences the structural representation of one’s own body. In order to better understand the effect of self-touch on body representations, we present an informal study of a stroke patient with somatoparaphrenia and misoplegia. The role of self-touch on body ownership was investigated by asking the patient to stroke the impaired left hand and foreign hands. The patient reported ownership and a change in affect over all presented hands through self-touch. The time it took to accomplish ownership varied, based on the resemblance of the foreign hand to the patient’s own hand. Our findings suggest that self-touch can modulate impairments in body ownership and affect, perhaps by helping to reinstate the representation of the body.

Keywords: body ownership; active touch; somatoparaphrenia; misoplegia; body image; body schema

1. INTRODUCTION

Deficits in somatosensory function after stroke are relatively common, occurring in about half of the patients [1]. They are not a unitary phenomenon and impairments range from deficits in basic detection and in perception of features such as shape and texture, to tactile agnosia and body disownership. In this review, we will describe these impairments and particularly discuss the role of active touch in these deficits. The somatosensory system involves serial as well as parallel processing of sensory input and this is reflected in the deficits observed after stroke [2,3]. Serial processing can be found in terms of deficits in primary somatosensory deficits that affect higher order functions such as haptic object recognition. At the same time, the presence of selective deficits in, for example, haptic object recognition with intact recognition of body parts by touch suggests a parallel organization.

In this review, we first focus on primary somatosensory deficits. Second, deficits in haptic perception are described. Haptic perception is the process of recognizing objects through somatosensory input and involves a combination of sensory perception of patterns on the skin surface (e.g. edges, curvature and texture) and proprioception of hand position and conformation. A stroke can give rise to a range of impairments in bodily representation that can be further subdivided. That is, certain deficits can affect the use of somatosensory input for the guidance of action, whereas others involve bodily perception and experience. This is the subject of §4. In §5, the role of active self-touch in modulating somatosensory deficits after stroke is discussed. Several studies have shown that self-touch improves perceptual detection for the affected body parts. The circumstances in which this occurs and the underlying mechanisms are discussed. In §6, a case study of a stroke patient with impairments in bodily experience is described. In this patient, the role of self-touch on body ownership was systematically investigated and the possible underlying mechanisms are discussed.

2. PRIMARY SOMATOSENSORY IMPAIRMENTS

Primary somatosensory impairments are probably the most frequently reported somatosensory deficits after stroke [4,5]. They consist of an inability to discriminate somatosensory aspects, such as the loss of pressure sensitivity, elevated spatial acuity as measured by two-point discrimination, a loss of vibratory sense or deficits in proprioception. Deficits in primary somatosensory perception have been reported most often after damage to the (hand area of the) contralateral primary somatosensory cortex, the thalamus or the subcortical somatosensory pathways [6]. Impairments occur in the subacute phase in about half of the
patients [1,4]. Deficits in different primary somatosensory submodalities can be selectively affected and as such can be considered independent of each other [4,6]. Proprioception not only appears to be more frequently impaired than tactile input [4], but also appears to recover faster [7]. Several somatosensory test batteries have been developed that test this range of primary somatosensory functions [4,5].

3. HAPTIC OBJECT RECOGNITION DEFICITS AFTER STROKE

(a) Feature discrimination
A higher level in the hierarchical processing of somatosensory input concerns the discrimination of the haptic features of an object. These features include texture, substance, size, shape, weight and the hardness of a stimulus. Extraction of these features may be separate for the micro- and macrogeometrical properties of an object [8]. The microgeometrical properties correspond to the surface of the object (texture, roughness or hardness) and are associated with activation in the parietal operculum [9–13]. The macrogeometrical properties correspond to the length of axes of the object (size and shape) and seem to be processed predominantly by the anterior part of the intraparietal sulcus. Micro- and macrogeometrical feature perception may be selectively impaired after stroke. For example, amorphognosia (disorders in discriminating the size or shape of an object) and ahylognogasia (disorders in discriminating the texture, weight or thermal properties of objects) are dissociable disorders [14–16]. Macrogeometrical feature perception deficits may involve more posterior cortical lesions compared with microgeometrical feature perception deficits [14,17].

(b) Object exploration
We frequently make use of tactile perception in order to recognize objects in our daily lives, for example, when retrieving keys from our pockets. Recognizing objects by touch is usually not a passive process [18]. The stimulus is typically explored actively using finger and hand movements to build a perceptual representation of the object. Psychophysical studies suggest that the finger and hand movements made are not arbitrary but depend on the object characteristics that need to be identified. Lederman & Klatzky [19] (see also this volume) observed that when subjects were asked to discriminate a particular dimension (e.g. texture, hardness and weight), different types of hand movements, so-called exploratory procedures (EPs), could be identified. The EPs used depend on the dimension to be discriminated. For example, texture was mainly explored through sideways movements between skin and object surface (lateral motion), whereas hardness was determined by pressing the object. When identifying objects, sequences of EPs are executed that allow building a representation that can be matched with object representations in memory [20]. The selection of an EP for a particular property depends on a number of factors including its duration, breadth of sufficiency (number of features that can be extracted through this particular EP) and compatibility with other EPs. In the context of this review, it is important to note that the ability to perform these EPs needed for haptic object recognition can be selectively disturbed. This phenomenon is referred to as tactile apraxia and occurs particularly in the hand contralateral to frontal and posterior parietal lesions [12,21]. Posterior parietal lesions seem to be associated particularly with a decrease in frequency and regularity of EPs and haptic object recognition impairments, but not with deficits in non-manipulative alternating finger movements. In contrast, frontal lesions result in impaired non-exploratory finger movements as well as impaired EPs, but with intact haptic recognition and sensory abilities. Considering the close link between EPs and perceptual feature extraction, it may not be surprising that difficulties in the haptic exploration of an object can lead to problems in object recognition [21,22].

(c) Object recognition
Forming a representation of an object depends not only on EP execution and extraction of sensory features, but also on the integration of those features. An example is the discrimination between a key and a coin in a pocket by touch. To recognize the key, information about its thermal properties, the weight, and its size and shape have to be extracted by purposeful hand movements and needs to be integrated into a coherent object representation. Subsequently, the representation of the object, in this case the key, is used to access the semantic properties (its use and function). An inability to recognize objects haptically despite relatively preserved primary somatosensory function is called tactile agnosia [23]. Identification of the object through other modalities is usually preserved. In tactile agnosia, the type of abnormality that occurs in the information processing can vary. First, problems concerning the perception and integration of the micro- and/or macrogeometrical properties can arise [3,17,24]. This is also referred to as tactile apperceptive agnosia (astereognosis), which can be feature-specific (for example, selective deficits in integrating texture information). Clinical reports of pure apperceptive agnosia (i.e. without somatosensory or motor deficits) are rare and often associated with right hemispheric damage. As the right hemisphere is associated with supra-modal spatial perception, some studies suggest that higher order tactile disorders are primarily a disorder in spatial skills [25,26]. Indeed, somatosensory deficits often co-occur with deficits in higher order spatial processing such as neglect [27]. However, more recent studies found that tactile agnosia can exist without spatial deficits [28,29]. The second type of abnormality associated with tactile agnosia concerns tactile associative agnosia and arises when the representation of the (correctly extracted) features fails to generate the semantic knowledge of the object. Thus, patients with associative tactile agnosia can draw what an object looks like (e.g. a metal object with an irregular side in case of a key) but are not able to name the object [30]. In order to recognize objects, stored semantic knowledge about the object is needed [31]. In associative agnosia, this semantic information can no longer be accessed [32]. Interestingly, in apperceptive agnosia, prior semantic knowledge about an object improves tactile recognition performance, confirming that top-down mechanisms are involved in tactile processing [3].

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The somatosensory system is not only capable of recognizing objects, but importantly, also provides primary information about the body. Disorders in bodily experience after damage to the central nervous system have been widely reported. In §4, an overview of these deficits is given.

4. BODY-RELATED DISORDERS
Information about the position of the different parts of our body relative to each other is based on an integration of visual, vestibular, proprioceptive and tactile input. Some authors have proposed that multiple representations of our body coexist. An early and highly influential proposal in this respect came from Head & Holmes [33]. They suggested three different type representations: (i) the ‘body schema’ as ‘a combined standard against which all subsequent changes of posture are measured . . . before the changes of posture enter consciousness’, (ii) the ‘superficial schema’ as a central mapping of somatotopic information derived from the tactile information, and (iii) the ‘body image’ as an internal representation in the conscious experience of visual, tactile and motor information of corporal origin [34]. More recently, the most common distinction made is that between body image and body schema [34, 35]. The body image represents a conscious perceptual identification of body features. It may be more (but not entirely) visually based and is influenced by stored knowledge about the body structure and semantics. The body schema is related to the position of body parts in space and is mainly based on proprioceptive input combined with tactile information. Information about the body schema is continuously updated as our body moves or changes and is primarily involved in sensory-guided actions. The cerebral basis of the body schema is still unclear, though a central role for the superior part of the posterior parietal cortex has frequently been suggested [2]. Disorders in the body representations may include features of both types. In §4a,b, we will describe the most well-known examples from the large variety of disorders that exists.

(a) Body representation disorders
Disorders of body representations can affect the entire body or be limited to specific body parts. An example of the latter is finger agnosia, in which a person is unable to identify his fingers, despite a preserved ability to use them and detect touch on their fingers. Finger agnosia is possibly the most common of all body representations and is most often present for the middle three fingers of both hands [36]. It can be mainly considered as a body image deficit in which a structural perceptual representation of part of the body is disturbed, whereas sensorimotor control remains intact [37, 38]. Another body representation disorder concerns left–right disorientation, where an ability to identify the right and the left side of one’s body is disturbed while other spatial concepts (e.g. up–down or front–back) and left–right identification of other objects or persons, are preserved [39]. Left–right disorientation and finger agnosia often occur after lesions to the (left) inferior parietal lobe. Together with dyscalculia and dysgraphia, they constitute Gerstmann’s syndrome [40]. Two final examples of a body representation disorder in which structural representation of the body appears to be impaired are autotopagnosia in which a person is not capable of localizing their own body parts and heterotopagnosia in which problems arise in localizing somebody else’s body parts [41–43]. These disorders are also associated with left posterior parietal lesions.

(b) Body awareness disorders
For disorders concerning body awareness, a distinction can be made between awareness with respect to a deficit, and awareness towards one’s own body. In the first category, a prominent example is anosognosia, where there is a lack of awareness of any physical (or cognitive) deficit. In the case of denial, patients reject the idea of physical impairment, while patients with a lack of insight admit the existence of their deficit but underestimate the severity and the implications of their physical impairment (anosodiaphoria) [44]. These types of disorders occur in about 18 per cent of patients with first ever stroke [45] and 32 per cent of right-hemisphere stroke patients [46]. However, most patients recover quickly [46]. The disorders are generally associated with right insular lesions [46–48], but not exclusively. Interestingly, it was found that patients with anosognosia for hemiplegia are significantly slower in performing an inhibition task with deficit-related sentences than a control group of aware hemiplegic patients. This suggests that in anosognosic patients, implicit knowledge of the deficit is present while explicit awareness is not [47,49].

In the second category of body awareness disorders, there are several disorders relating to awareness towards one’s own body. In asomatognosia, patients feel that parts of their body are ‘missing’ or have disappeared from corporal awareness [50,51]. For example, the loss of awareness of one arm, which may or may not be paralyzed. Another disorder related to disturbances in body ownership is somatoparaphrenia, where patients actively deny the ownership of a paralysed hand, arm or foot. Patients might give alternative interpretations to explain their affected functions, for example, by believing that the affected limb belongs to someone else, that it is an animal or that it is part of a rotting corpse. A reverse interpretation is also possible, meaning that patients may identify the body parts of another person as their own [52–54]. The duration of symptoms of asomatognosia and somatoparaphrenia can range from minutes to months. Presenting evidence that contradicts the illusion reduces the denial only temporarily, after which the asomatognosia or somatoparaphrenia returns. Misophasia is a more severe form of a body awareness disorder and is defined as a hatred for the affected limb, with offensive behaviours towards the limb as a result [55]. Disorders associated with deficits in ownership are often observed after extensive right-hemispheric lesions, where premotor, parietal and posterior insular damage are particularly implicated (for review see Vallar & Ronchi [54]).

Problems concerning the ownership of limbs can also be present when the particular limb is no longer a physical part of the body, as in the case of phantom
limb phenomenon. This can be defined as ‘the persistent experience of the postural and motor aspects of a limb after its physical loss’ [56]. The phantom limb phenomenon occurs in approximately 95 per cent of patients who undergo amputation of a limb [57]. Much less frequent is the phantom limb phenomenon after cerebral damage. This is referred to as the supernumerary phantom limb (SPL), and is defined as ‘the awareness of having an “extra limb” in addition to the regular set of two arms and two legs’ [58,59]. Patient studies describe multimodal involvement, regarding the tactile (feel objects with their phantom arm), visual (visually perceive their phantom limb) and motor components (generate action) present in SPL. Neural correlates in the brain areas that represent these modalities have been found in the right hemisphere [60]. However, SPL may occur among patients with left-hemispheric stroke as well, especially when the thalamus is involved and the patient suffers from spastic paresis on the right side [59].

Apart from anosognosia, body awareness disorders after brain damage occur relatively infrequently and recover over time. As a consequence, in-depth research of the underlying neural mechanisms is sparse. However, these disorders are of great interest as they can provide insight into the mechanisms underlying bodily awareness, body ownership and self-other distinctions.

5. SELF-TOUCH AND SOMATOSENSORY FUNCTIONING

Accumulating evidence shows that actively touching one’s own body may involve different sensory mechanisms compared with being touched passively. The different mechanisms underlying self-touch and touch by others have been observed both in healthy individuals [61,62] and in patients with somatosensory deficits. In this section, we review the literature on self-touch and elementary tactile deficits after stroke. Furthermore, we present a case study in which self-touch modulated higher order processes, that is, ownership over the subject’s affected arm.

(a) Self-touch and tactile perception

(i) Self-touch enhancement

The effect of active self-touch in modulating somatosensory perception after stroke was first reported by Weiskrantz & Zhang [63]. A right-hemispheric stroke patient with clear sensory deficits in the contralesional hand, who was unable to detect tactile stimulation administered by the experimenter, was described. In contrast, the patient was able to feel tactile stimulation on the affected hand when it was touched by the own unaffected hand or by a probe that was held by this hand. Thus, tactile sensitivity was enhanced by active self-touch. This finding was replicated more recently in a larger sample of stroke patients in which 22 out of 39 patients showed self-touch enhancement [64]. Most of the patients with enhanced self-touch (17/22) suffered right hemisphere lesions. The mechanisms underlying the self-touch modulation have not yet been established, although several mechanisms have been proposed. These include (i) a heightened attention towards the spatial region of the affected hand, (ii) the use of proprioceptive information of the administering hand, and (iii) the use of predicted sensory consequences of the generated action.

The influence of attention on somatosensory perception has been widely investigated in healthy individuals. Several studies have focused on how visual attention modulates somatosensory perception. They showed that orienting the eyes towards a body part facilitates detection of touch, which could not be explained by proprioceptive information or visual information of the target [65]. In addition, other studies showed that tactile extinction diminished when a visual stimulus was presented close to the affected hand [66]. Besides directing visual attention, other studies demonstrated that non-visual attention also changes perceptual processing. For example, Coslett & Lie [67] described two patients who suffered from tactile extinction as a result of a right-hemispheric brain lesion. The ability to detect tactile stimulation with the affected hand improved by direct passive skin contact with the ipsilateral hand. It was suggested that the unaffected hand provided a focus of attention to the extinguished side.

Another contributing factor to self-touch modulation in patients is the use of proprioceptive information, which provides the patient with information about whether the administering hand is close to the affected hand and is touching it. Studies on healthy participants have demonstrated the interaction between touch and proprioception with the so-called Pinocchio illusion. This illusion is observed when the passive biceps tendon is vibrated while participants hold the tip of the nose between finger and thumb. As a result of this stimulation, a proprioceptive illusion is induced in which the elbow extends and the nose elongates [68–70]. The role of proprioception in tactile enhancement is investigated with experiments in which proprioceptive input is non-informative during detection of touch. For example, Valentini et al. [64] and Weiskrantz & Zhang [63] controlled the proprioceptive input by having the patient’s affected hand digititerated with the examiner’s hand. Sometimes, the patient would touch their own affected hand; in other trials, the experimenter’s fingers were touched. Self-touch enhancement was reliably observed only when their own affected hand was touched, suggesting that proprioception is not the primary contributor to self-touch. In line with this result, White et al. [71] stated that neither proprioceptive information nor attention towards the spatial region of the affected hand offers a sufficient explanation for self-touch enhancement. Instead, they showed that a temporal delay between administration and actual tactile stimulation eliminated the self-touch enhancement, suggesting an important role for temporal expectation. Thus, action with the administering hand provides a precise temporal cue for focusing attention on the affected hand.

(ii) Self-touch attenuation

Although some studies show that self-touch increases the sensitivity for tactile stimulation, other studies report attenuation for tactile stimulation during self-administered touch. For example, Weiskrantz et al. [62] and Blakemore et al. [72] investigated the issue of ‘why we can not tickle ourselves’ and concluded...
that self-generated action diminishes the intensity of the tactile stimulation that accompanies the action. These findings are in concordance with the 'forward model' that is thought to make a prediction about the sensory consequences of an action [73–76]. This prediction allows self-produced stimuli to be attenuated, giving the opportunity to signal external events that provide information that is novel. Jackson et al. [77] demonstrated in a functional magnetic resonance imaging study that a somatosensory stimulus delivered to a hand that is being prepared for movement is perceived later than when the same stimulus is delivered to a stationary hand for which no movement is prepared. In addition, during stimulation on the hand prepared for motion, reduced activation was observed within the bilateral parietal operculum and insula compared with the non-moving hand. The authors interpreted the perceived delay as a result of an increased somatosensory threshold.

Thus, self-touch has been associated with both enhanced and reduced tactile sensitivity. The discrepancy between self-touch enhancement and self-touch attenuation is as yet unexplained. Jackson et al. [77] pointed out that the two different theories, i.e. the attenuation of tactile perception by the forward model and the enhancement of tactile perception by attention, might in fact be consistent with each other. The estimate of the sensory consequences of a self-generated action is largely irrelevant to behaviour and therefore, attenuation of these stimuli may be beneficial to monitor other signals. White et al. [71] proposed that an accurate temporal anticipation is the crucial factor underlying self-touch enhancement, allowing enhancement of processing resources for the anticipated stimulus.

(b) **Self-touch and body representation**

Although there is considerable evidence for modulation of tactile perception by self-touch, less is known about self-touch modulation and higher order somatosensory processing. A recent report demonstrated a right-hemispheric stroke patient who was able to detect somatosensory stimulation on the left-hand but was not able to localize the stimulus [78]. This suggests that the patient had an impaired representation of the affected hand. Interestingly, localization on the affected hand improved when the patient reached with the right hand to the left of the own affected hand while having the illusion of self-touch. The authors hypothesized that self-touch might have altered the patients’ representation of the affected hand. The effect of self-touch on the structural body representation is also investigated in a study with healthy individuals [79]. In this elegant experiment, subjects were asked to touch several fingers of one hand with fingers of the other hand. They induced a discrepancy between the number of fingers touched on the active and on the passive hand by interleaving the experimenter’s fingers between the fingers of the passive hand. When asked to name the number of fingers inbetween fingers, participants showed an underestimation that was specific for the self-touch conditions. This suggests that self-touch influences the structural representation of one’s own body. In a recent study, Kammers et al. [80] investigated the influence of touch on paradoxical pain using the thermal grill illusion. They induced the thermal grill illusion by placing the participant’s index and ring finger in hot water and the middle finger in cold water. This results in paradoxical feeling of painful heat in the middle finger. It was shown that the painful heat induced by the illusion was reduced when the fingers of the other hand touched the fingers used to induce the illusion. They suggested that this self-touch effect is not owing to low-level touch–temperature interactions, but that an increase in the coherence of body representations is involved.

These reports have provided accumulating evidence that self-touch has more extensive representational effects rather than the sensory-attentional enhancement reported previously [64]. Although they demonstrated that self-touch modulates body representation in healthy participants, the question remains whether self-touch can be beneficial for patients who suffer from body representation impairments. While White et al. [71] suggest that self-touch can enhance tactile localization, so far, the effects of self-touch on bodily awareness and body ownership disorders have not been reported. Here, we describe an informal case study in which we investigate the effect of self-touch on body representation.

6. **SELF-TOUCH AND DISORDERS IN BODY REPRESENTATION**

(a) **Case history and observation**

Patient GE is a 60-year-old woman who had a large right intra-axial haemorrhagic stroke affecting the right parietal and frontal lobes (figure 1). As a consequence, she suffered from left hemiplegia, hemianaesthesia and left hemispatial neglect. There was evidence of anosognosia for hemiplegia. Moreover, GE reported that she had problems in identifying her left hand and arm as her own. The hospital staff also noticed that GE occasionally mistook the hand of someone else for her own (somatoparaphrenia). GE reported that she had experienced negative feelings towards her left arm (misoplegia) since her stroke. These included the feeling of hatred and the urge to harm or punish the arm. These negative feelings were no longer present at the time of assessment two weeks later. A striking observation was that the patient was stroking her left arm and hand with great care and affection. GE reported that this was because she felt the stroking in the affected hand and this helped her to regard the arm as a part of herself.

GE was assessed two weeks after her stroke. Her speech was fluent and informative. She was able to understand questions and instructions and was motivated to cooperate with the investigations. A neuropsychological assessment revealed impairments in several cognitive domains, including long-term memory, working memory, visual perception, visuoconstruction and executive functioning. Furthermore, the patient demonstrated finger agnosia, proprioceptive impairments and astereognosis. In addition, evidence for peripersonal neglect was found. Besides these impairments, GE was oriented in person, place and time. Also, her attention span was unimpaired. At the

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time of the assessment, GE did not report problems regarding the sense of ownership or the affect for her contralesional limbs.

GE was not able to detect somatosensory input on the left side of her body when the experimenter administered it. However, the patient reported the sense of touch in her contralesional hand when the right ipsilesional hand generated the touch (self-touch). Interestingly, this was regardless of whether she perceived direct skin contact with her ipsilesional hand since she felt the touch as well when her ipsilesional hand administered touch by a paintbrush. This observation is consistent with the idea that temporal expectation is an important mechanism for self-touch enhancement [71].

(b) Hypothesis I: active (self-)touch enhances ownership

Our main investigation aimed to test whether the self-touch could have helped the patient in ‘keeping the arm as a part of herself’ as she spontaneously suggested. In other words, does self-touch affect disorders of body ownership? To investigate this, GE was asked to stroke her own arm and different arms that were not her own, referred to as foreign arms. The experimental design and set-up are shown in figures 2 and 3. The following foreign arms were used: (i) a left rubber arm, (ii) the left arm of the experimenter, (iii) a right rubber arm, (iv) a left rubber arm in an anatomically implausible position. GE was asked to stroke the dorsal surface of the arm (own arm or foreign arm) for 3 min, and to comment on the arm that she was stroking. In particular, she was asked whether she thought the stroked arm belonged to her or someone else. The time it took to report ownership over the arms was recorded. Immediately after the first stroke and 3 min after stroking, she was asked to rate the emotional valence towards her arm, ranging from 0 (negative) to 5 (excessive positive emotional valence). First, GE stroked her own arm for 3 min and after the emotional valence rating GE started to stroke the first foreign arm (i.e. the left rubber arm) followed by the emotional valence rating. The conditions ‘own arm’ and ‘foreign arm’ alternated four times.

Stroking of the arms resulted in an increased sense of ownership over the particular arm. More specifically, GE achieved a sense of ownership over all four foreign arms by stroking. Interestingly, during stroking, her attitude towards the arm changed noticeably from a dislike and rejection towards a more affective stroking and increasing belief that the foreign arm was a part of her body. Although the patient achieved the sense of ownership over all the foreign arms, the stroking time that was needed to achieve the sense of ownership varied between the different arms. This variation possibly depended on the level of similarities between the real arm of the patient and the foreign arm (figure 4). For example, to achieve a sense of ownership over the experimenter’s arm, the stroking time that was needed was 15 s, whereas 125 s of stroking was needed to achieve a sense of ownership over a right rubber arm. This suggests a top-down component in which a structural representation of her own arm influences the tendency to regard another arm as belonging to her. In healthy individuals, somewhat similar top-down influences have been observed when inducing the rubber hand illusion (RHI) [81].

Figure 1. Three images of an axial plane CT-scan of patient GE showing a large haemorrhagic stroke in the right hemisphere. The images follow the radiologist convention of right hemisphere represented on the left.

Figure 2. Experimental design. The patient stroked different arms alternating her own arm and foreign left (l) and right (r) arms. The stroking time for each arm (own arm and foreign arms) was 3 min. Within the 3 min, the time it took to achieve ownership over the arm was recorded.
However, while the RHI induction fails when a right rubber arm is stroked synchronously with a left real arm, or when the rubber arm was placed in an anatomically incongruent position, in the current case study ownership over these rubber arms was eventually achieved. These results confirmed our hypothesis that active (self-)touch indeed enhances ownership.

Another result of this investigation was the change in affect towards the arms. For all arms, the affect increased during stroking of the foreign arms (figure 5). However, similar to the time differences in achieving ownership, the level of affect seems related to the resemblance to the structural aspects of her own arm. For example, the emotional valence towards the experimenter’s arm is more positive compared with other foreign arms. In addition, the positive valence towards her own real arm was highest and similar to the emotional valence GE reported about her other limbs. The positive valence towards her own real arm did not differ between the first stroke and after 3 min. Intriguingly, when we showed the patient her own arm after stroking a foreign arm, she did not visually recognize this arm as her own and regarded her arm as unpleasant. However, when she touched her own arm with her ipsilesional hand, she immediately acknowledged that the arm she was holding was hers, which coincided with a positive affect. This suggests that although ownership over her own affected arm was weakened and therefore could be extinguished by stroking a different arm (active touch), merely touching the affected arm reinstated ownership and re-established

Figure 3. Experimental setup. The patient was asked to stroke the dorsal surface of her own hand. During stroking of the foreign arms, the position of the own arm remained unchanged, except that a pillow covered the own arm. The foreign arms were positioned similar to the own arm except that they were positioned on top of the pillow. The foreign arms in the anatomically implausible position did not match the position of the own arm. While stroking, the patient was asked whether she thought the stroked arm belonged to her or to someone else.

Figure 4. The effect of stroking condition on the time needed to obtain ownership. The results show that the stroking time that was needed to achieve the sense of ownership varied, based on the level of similarities between the real arm of the patient and the foreign arm. Note that the stroking time for the foreign arms was recorded once, whereas the stroking time for the patient’s own arm is an average of four trials.
normal positive affect appreciation. This observation also suggests that stroking a foreign arm is followed by a sense of disownership of her real arm. To investigate this further, a second investigation was conducted.

(c) Hypothesis II: stroking a foreign arm disowns the real arm

The second investigation aimed to assess whether stroking a different arm caused disownership of her real arm as was suggested by the first investigation. In addition, we wanted to ascertain that stroking the affected hand only induced changes in ownership when performed by the subject herself rather than by someone else, the differentiation between active self-touch, passive self-touch and passive touch.

Three stroking conditions were used: (i) stroking her contralesional hand with her ipsilesional hand (active self-touch), (ii) stroking her contralesional hand by the experimenter (passive touch), and (iii) stroking a left rubber arm by her ipsilesional hand (active touch). These stroking conditions were administered three times each for duration of 90 s. The conditions were administered in a counterbalanced order. After each condition, five questions were asked to assess whether different possible deficits regarding her hemiplegic hand were present (table 1). The five questions aimed to target signs of five different deficits, that is, change in affect, misoplegia, anosognosia for hemiplegia, asomatognosia and somatoparaphrenia. The questions targeting these deficits could be answered by a four-point scale, ranging from 1 (not present) to 4 (present).

The results showed that after GE stroked a rubber arm (active touch), there were clear changes in affect towards her own arm (figure 6). In addition, she showed signs of asomatognosia by rejecting her own arm as belonging to her body. These changes did not occur when she had stroked her own arm (active self-touch), or when the experimenter had stroked her arm (passive touch). As expected, based on the previous observations, GE reported that she did not feel the stroking when the experimenter stroked her arm, whereas she did feel the stroking in her contralesional arm when she was the agent of the stroking. The findings of changes in affect and ownership over her own arm following the stroking of the rubber arm indicate that stroking a rubber arm induces deficits in the sense of ownership over her real arm. Possibly, GE obtained an enhanced sense of ownership over the rubber arm by stroking, similar to the first experiment. As hypothesized in the first experiment, stroking helped her to re-establish the representation of her arm. However, with stroking a rubber arm, the representation that is built differs from the representation of her real arm. As a result, her real arm does not
match the newly built representation and is therefore rejected. As hypothesized, these results suggest that ownership over a rubber arm disowns the real arm.

(d) Limitations
Before we draw conclusions from these investigations, several limitations need to be considered. Similar to other patients with body ownership disorders, GE’s haemorrhagic stroke affected several cognitive domains and resulted in increased fatigue. GE suffered from deficits in long-term memory, working memory, visual perception, visuoconstruction and executive functioning. One could assume that these cognitive deficits resulted in an overall confusion that caused the changes in the attitude towards her arm. However, her attention span as well as her orientation to place, time and person was unimpaired, preserving the ability to direct and hold her attention towards the investigations. Furthermore, an overall fatigue or confusion could not explain the differences in stroking time that was needed to achieve ownership (hypothesis 1).

Another limitation of the present study is that the sense of ownership was measured by verbal response of GE, and therefore, an objective measure of ownership is missing. As a result, the possibility cannot be ruled out that the patient had a tendency to comply with the expectation of the experimenter and reported having a sense of ownership over a foreign arm when this was not the case. However, besides verbal response, a change in the attitude of GE was also observed. For all foreign arms, the patient was reluctant to stroke the foreign arm at first. Her attitude towards the arms changed from rejection to more affective—almost cuddling—stroking of the foreign arm. In addition, again the variation in stroking time between the different arms suggests that her response reflects a reliable sense of ownership rather than a random tendency to accept every arm as her own. Nevertheless, to rule out these uncertainties and to test our newly raised hypotheses, more controlled trials are required. A more fundamental point of criticism that we have to take into account is whether the body ownership problems of the patient might have interfered with the questions we asked about her own arm. As hypothesized, the patient achieved a sense of ownership over the rubber arm after stroking. If this hypothesis was true, then how do we know for certain that the questions we asked about her real arm were not conceived as questions about the rubber arm? The changes in affect and sense of ownership would therefore concern the rubber arm, instead of her own. This would be in line with the findings of the first experiment, in which a less-positive affect for the rubber arm compared to her own arm is found. For future research, it would be interesting to investigate whether a newly achieved sense of ownership automatically results in a sense of disownership of the own limb, as implied by the second investigation.

7. DISCUSSION
The overall aim of the present paper was to review the role of active and self-touch on the variety of somatosensory deficits ranging from elementary, primary deficits to impairments in the bodily experience. Most studies on self-touch have focused on primary tactile perception, and demonstrated changes in the detection threshold as a result of self-touch in patients with primary somatosensory deficits. Recently, a few studies have also demonstrated changes in the structural and cognitive aspects of body representation following self-touch in healthy participants [79]. The present study extends the knowledge of the effect of self-touch by demonstrating the role of self-touch on bodily experience. It was shown that active touch and active self-touch modulate the sense of ownership in a patient with prominent disorders in body ownership. Moreover, the affect towards the limb became more positive during stroking. Although several limitations restrict the implications of the results, this study provides an indication that self-touch, especially active self-touch helps reinstate the body representation and the sense of ownership. This suggests that self-touch not only plays a role in primary somatosensory deficits, but can modulate impairments in the bodily experience as well. These findings raise a new hypothesis for future studies on the role of self-touch in bodily experience.

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