Cognitive Neuropsychology

ISSN: 0264-3294 (Print) 1464-0627 (Online) Journal homepage: http://www.tandfonline.com/loi/pcgn20

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To cite this article: Jared Medina & H. Branch Coslett (2016): What can errors tell us about body representations?, Cognitive Neuropsychology, DOI: 10.1080/02643294.2016.1188065

To link to this article: http://dx.doi.org/10.1080/02643294.2016.1188065

Published online: 07 Jul 2016.

COGNITIVE NEUROPSYCHOLOGY, 2016
http://dx.doi.org/10.1080/02643294.2016.1188065

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What can errors tell us about body representations?

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ABSTRACT

In this review, we examine how tactile misperceptions provide evidence regarding body representations. First, we propose that tactile detection and localization are serial processes, in contrast to parallel processing hypotheses based on patients with numbsense. Second, we discuss how information in primary somatosensory maps projects to body size and shape representations to localize touch on the skin surface, and how responses after use-dependent plasticity reflect changes in this mapping. Third, we review situations in which our body representations are inconsistent with our actual body shape, specifically discussing phantom limb phenomena and anesthetization. We discuss problems with the traditional remapping hypothesis in amputees, factors that modulate perceived body size and shape, and how changes in perceived body form influence tactile localization. Finally, we review studies in which brain-damaged individuals perceive touch on the opposite side of the body, and demonstrate how interhemispheric mechanisms can give rise to these anomalous percepts.

ARTICLE HISTORY
Received 24 February 2016
Revised 4 May 2016
Accepted 5 May 2016

KEYWORDS
Touch; localization; body representation; somatosensation; synchiria

Introduction

A number of theoretical advances in cognitive psychology and neuropsychology have been driven by the analysis of error patterns, using mistakes made by intact and damaged brains to infer the organization of functional subsystems (McCloskey, 2003). Although there have been a wealth of studies using error patterns in domains such as language, vision, memory, and motor control, there has been relatively little work on this topic in the somatosensory system. A number of advances in our understanding of somatosensory systems have come from neural recording studies in non-human primates. These studies have provided fascinating insights into the topography of somatosensory maps, cortical dynamics, and neural plasticity. Very little work with non-human primates, however, has examined the perceptual correlates of such changes. By examining errors in tactile processing made by brain-damaged individuals, as well as biases in neurologically intact individuals, we can build upon non-human primate research to develop a fuller understanding of how the mind represents the body.

In this review, we examine how evidence from errors (or, non-veridical sensory experiences) in somatosensory processing can be used to understand body representation in four sections. First, we discuss whether tactile detection and localization are serial processes, or whether individuals with numbsense provide evidence that detection and localization are parallel processes. The next three sections discuss localization of tactile stimulation, focusing on how specific types of error patterns provide evidence for different processes utilized in localizing touch. In the second section, we discuss how information from primary somatosensory maps needs to be linked to a higher order, secondary representation of body size and shape in order to localize touch (body form). However, as primary somatosensory map topography expands (after use-dependent plasticity), shifts, and contracts (after brain damage), errors may occur in the mapping between these representations. We discuss how localization performance, both after plasticity and in conditions of uncertainty, can inform us regarding body representation. In the third section, we focus on conditions in which body form representations are atypical, including when they persist when the body is not there (amputees who experience phantom limb phenomena) and manipulations (anaesthetization, muscle vibration) that alter perceived body size and shape. We then examine

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how tactile sensations may be mapped to these atypical body form representations. Finally, we review one additional category of non-veridical tactile sensation – errors in which participants feel touch on the side opposite stimulation. We review evidence from individuals with brain damage and discuss how it can inform us regarding interhemispheric communication of tactile information.

Tactile detection and localization – serial or parallel processes?

When a tactile stimulus is presented, an individual perceives both whether a stimulus was presented or not, and the location of the stimulus. Different models have been proposed to explain the relationship between processes for tactile detection and localization. In a serial processing model, sensory input is first processed to determine whether the participant detected the stimulus, followed by a second, separable process utilized for tactile localization. However, in a parallel processing model, tactile input is assumed to be processed separately for tactile detection and localization. The primary evidence in support of a parallel processing model is from brain-damaged individuals with numbsense – the ability to localize, without being able to consciously detect, tactile stimuli. There are only two detailed reports of this condition, as well as a brief mention by Lahav (1993). In the first reported case, Paillard, Michel, and Stelmach (1983) examined an individual who suffered left parietal and occipital damage, which resulted in severe right-sided hemianaesthesia, such that she did not report any sensation after stimulation along her entire right hand and forearm. In one experiment, the individual was blindfolded and was asked to point with the opposite hand to one of 18 locations (over three blocks) on the tested hand. Within these tactile stimulation trials were 10 "catch" trials in which the participant was not stimulated but was asked to make a localization judgment. Even though she was not able to consciously report tactile stimuli presented to her hemianaesthetic right hand, she made localization judgments on 90% of trials in which she was stimulated. Furthermore, she did not respond during any catch trials, ruling out a response bias towards making a localization judgment whenever prompted to respond. The participant was able to make localization judgments to these stimuli, and the authors claimed that she was able "to point approximately to the locus of stimulation". However, her localization ability was quite crude, with the majority of responses made towards the centre of the hand. The patient was within 2 cm of the target on only 26% of trials, compared to 92% in an age- and sex-matched control.

A second case of numbsense (J.A.) was reported by Rossetti and colleagues (Rossetti, Rode, & Boisson, 1995, 2001). This individual presented with complete right hemianaesthesia due to a left thalamic lesion and was completely unaware of tactile stimulation on the right hand during clinical testing. The experimenters first used a pencil tip to depress the skin surface at one of six locations (the five fingertips and the centre of the palm) and left it there until the patient made either a blindfolded pointing (motor) response, or a forced-choice verbal response as to which location was stimulated. There were no catch trials in this experiment. Using a criterion according to which any response on the stimulated hand segment counted as a correct pointing response (i.e., a response anywhere on the index finger would be correct for stimulation of the index fingertip, any response on the palm would be correct for stimulating the palm centre), J.A. was correct on 18/40 pointing responses, versus only 4/21 correct with verbal responses. In further testing, they found that J.A. was more accurate at pointing to his own hand than to a hand drawing, when making an immediate versus delayed (2–4-second) pointing response, and when making a pointing response compared to a combined pointing and verbal response. However, as observed in Paillard et al.'s (1983) case, even "correct" pointing responses were moderately inaccurate, and far below expected normal performance. In both cases of numbsense, there may be evidence for a crude somatotopy in localization judgments. However, localization performance is far poorer than in neurologically intact individuals.

Cases of numbsense have been provided, along with cases of tactile detection without intact localization (Halligan, Hunt, Marshall, & Wade, 1995; Rapp, Hendel, & Medina, 2002), as part of a double dissociation that provides support for a parallel processing model. However, supporting evidence for dissociations of tactile detection and localization have not been found in studies of neurologically intact individuals. Harris, Thein, and Clifford (2004)

presented a brief tactile stimulus to one finger followed by a vibrotactile mask to all four fingers and asked participants to report whether they detected the stimulus (using a yes–no response) and whether they could localize the stimulus (using a four-alternative forced-choice paradigm). On trials in which the participant failed to detect the stimulus, localization performance was significantly above chance (approximately 45% across multiple comparisons/conditions, with chance being 25%). Although these results are similar to cases of numbsense that demonstrated crude (but above chance) localization without detection, the authors noted that yes–no responses are susceptible to biases in decision criteria. If someone sets a conservative threshold for a "yes" response, they may report that they do not feel a stimulus even if there is enough information to detect and localize the stimulus. To examine this further, the authors compared their experimental data to simulation data that modelled three processes – a shared process for both detection and localization, a serial process for tactile detection, followed by tactile localization, and parallel processing of detection and localization. They found that the serial processing model was most consistent with their data. However, given that individuals with numbsense used manual pointing responses as opposed to forced-choice responding, one possibility is that numbsense responses are driven by a separate "somatosensation for action" system (e.g., Dijkerman & de Haan, 2007) that was not directly examined with verbal forced-choice responses. Harris and colleagues (Harris, Karlov, & Clifford, 2006) used the same backwards masking paradigm as before, with manual as opposed to verbal forced-choice tactile localization responses, and found no evidence for accurate localization of tactile stimuli that were not detected. These results provide evidence that past performance from individuals with numbsense could be explained by differences in response criteria. Individuals with numbsense may use a conservative response criteria for yes/no detection responses, but still have access to some sensory information in making a forced-choice response and crude localization judgments. Given that there have been no studies providing evidence for a separate localization without detection pathway, and that previous cases of numbsense could be explained by differences in response criteria, this evidence is not consistent with a parallel processing account. However, it is important that any future cases of numbsense explicitly examine whether their findings are simply due to biases in response criteria, or some unknown alternative mechanism.

Localizing touch: Plasticity and uncertainty

To interact with the environment, it is obviously important not only to detect touch, but to localize touch on the skin surface. Although there is a point-to-point topology between cortical maps in primary somatosensory cortex (S1) and the skin surface, this topology is non-uniform with larger cortical representations for skin surfaces frequently used in discriminative touch (e.g., the fingers versus the back). Given this non-uniform topology, an extremely simplistic process in which the perceived distance between two tactile stimuli was calculated solely by the cortical distance between activation peaks in primary somatosensory maps would clearly result in non-veridical percepts. One way to solve this problem is via a second representation that maps activity from these non-uniform maps in primary somatosensory cortex to a more veridical representation of the skin surface. Given that the trunk is not perceived as being smaller than the fingers, even though the finger representation is larger than the trunk representation in S1, this suggests some mapping process. Interestingly, an examination of tactile distance judgments provides some evidence that this mapping process to a secondary representation has systematic biases that reflect the non-uniform topology in S1. For example, Taylor-Clarke et al. (2004) stimulated participants with two simultaneous tactile stimuli on one skin surface (e.g., face), followed by two simultaneous tactile stimuli on a second skin surface (e.g., forearm), and were asked to report which distance was greater in a forced-choice paradigm. Unbeknownst to the participant, the distance between the two tactile stimuli was equal on both skin surfaces. However, biases in distance judgments emerged as a function of cortical representation, such that tactile distances on skin surfaces that are more densely represented (e.g., face, fingers) were felt as longer than those on less densely represented skin surfaces (e.g., forearm, back). The authors proposed the need for a secondary, veridical representation of the skin surface upon which information from these non-uniform primary somatosensory representations could be mapped.

The biases reported are probably caused by consistent error in this scaling process.

A second piece of evidence supporting the existence of a higher order representation for mapping tactile information comes from examinations of cortical plasticity. A number of studies in non-human primates have demonstrated that primary somatosensory maps are plastic, changing after central damage, amputation, and differential usage (for reviews, see Buonomano & Merzenich, 1998; Feldman & Brecht, 2005; Kaas, 1991). These studies provide evidence regarding the relationship between input and activation in primary somatosensory maps – but not evidence regarding how activity patterns in these maps are interpreted as tactile sensation on a particular skin surface. For example, individuals have been reported who can still feel touch after complete damage to the traditional "hand area" of S1 (e.g., Rapp et al., 2002). The fact that they can still feel touch on the hand provides evidence for plasticity, such that other undamaged regions now represent the hand. If localization of somatosensory stimuli involved a simple, fixed readout from activity in primary somatosensory maps (e.g., activation of neurons in cortical location X always results in sensation on skin surface Y), then damage to the hand area would result in the individual never feeling touch on the affected hand. As they can still feel touch on the hands, this provides evidence that the mechanisms for readout from S1 change as well, such that whatever activation is caused by stimulation of skin surface Y can now be interpreted as sensation on the hand. These and other results (see Medina & Coslett, 2010, for a review) provide evidence for a secondary representation of the size and shape of the body (*body form*) that is necessary to convert information from these plastic, distorted maps to a veridical representation of the location of touch on the skin surface.

There are a number of potential problems that need to be solved in mapping activity from primary somatosensory representations to a body form representation. First, primary somatosensory maps are plastic, with map topography changing subsequent to differential usage (use-dependent plasticity). We first review how the somatosensory system may deal with changes from use-dependent plasticity. Second, in processing sensory signals, inputs can be noisy due to either peripheral (near-threshold stimulation, nerve damage, decreased mechanoreceptors) or central (brain damage) factors, resulting in mislocalization errors. How do somatosensory systems deal with uncertainty? We examine patterns of tactile mislocalization after brain damage, on relatively low-acuity skin surfaces, and after modulating stimulus intensity, and discuss potential mechanisms for representing tactile location as noise increases.

Localization and use-dependent plasticity

One of the major factors in shaping the topography and characteristics of primary somatosensory maps is use-dependent plasticity (Detorakis & Rougier, 2012; Recanzone, Merzenich, Jenkins, Grajski, & Dinse, 1992). Neurons representing skin surfaces in area 3b of S1 that are typically stimulated together frequently have overlapping receptive fields, whereas "neighbouring" skin surfaces that are infrequently stimulated together are less likely to have overlapping receptive fields. Based on this, the borders separating finger representations in area 3b are relatively sharp at a given point in time, such that neurons on either side of this functional boundary have receptive fields for only one particular finger. However, these borders shift after amputation (Merzenich et al., 1984), stroke (Xerri, Merzenich, Peterson, & Jenkins, 1998), syndactyly (Allard, Clark, Jenkins, & Merzenich, 1991), and use-dependent training (Jenkins, Merzenich, & Recanzone, 1990). Given that these borders can shift such that a neuron in area 3b may represent one digit in one state, but a different digit after use-dependent changes, one might expect that tactile mislocalizations on the fingers would be quite common if the mapping to body form did not change in concert with S1 plasticity.

Neurologically intact individuals who have been shown to demonstrate use-dependent plasticity include three-finger Braille readers. Three-finger Braille readers have been shown to have larger finger representations than single-finger Braille readers and control participants (Elbert, Pantev, Wienbruch, Rockstroh, & Taub, 1995). However, only multi-digit Braille readers have been reported to demonstrate higher rates of tactile mislocalization (Sterr, Green, & Elbert, 2003; Sterr et al., 1998a, 1998b). Although these errors by multi-finger Braille readers could be interpreted as a mismapping from primary somatosensory to body form representations, these individuals frequently stimulate the skin surface of multiple fingers at the same time, leading to

an increase in multidigit receptive fields in area 3b after tactile coactivation (Allard et al., 1991; Mogilner et al., 1993). The observed tactile mislocalizations in multidigit Braille readers (Sterr et al., 2003; Sterr et al., 1998a, 1998b) are thought to be due to the increase in multidigit receptive fields from tactile coactivation. Given that tactile stimulation of multiple fingers leads to the development of multidigit receptive fields (Godde, Spengler, & Dinse, 1996), Schweizer, Braun, Fromm, Wilms, and Birbaumer (2001) examined tactile mislocalization of near-threshold stimuli after 20 hours of simultaneous tactile stimulation of the left thumb and little finger – two non-adjacent fingers typically not stimulated together. When stimulating one finger involved in coactivation training (e.g., the thumb), they found a significant increase in the number of errors made to the other coactivated finger (e.g., the little finger) after coactivation training compared to baseline (see also Braun, Schweizer, Elbert, Birbaumer, & Taub, 2000). However, in groups with larger S1 representations that use their fingers independently (violinists, pianists), no such mislocalizations have been reported. This suggests that the mislocalizations observed in multifinger Braille readers are not due to a mismapping from S1 to body form, but instead due to the emergence of multidigit receptive fields early in the somatosensory system. Furthermore, this suggests that given relatively normal usage patterns, the mapping from primary somatosensory representations to a sensory readout can change in tandem, resulting in veridical tactile localization even with somatosensory map expansion.

However, there are conditions in which changes in primary somatosensory maps result in non-veridical percepts. Craig (1993) examined the perceptual correlates of changes in somatosensory maps after prolonged tactile stimulation. In this study, four participants wore tactile stimulators positioned near the proximal end of the volar surface of their non-dominant forearm during waking hours for months. Tactile stimulation consisted of a 10-second series of pulses presented to the three stimulators every 48 seconds. After weeks of wearing the stimulators, three of the four participants reported anomalous sensations when localizing tactile stimuli. One participant, after wearing the stimulators for 36 days, reported double sensations on 14/24 forearm stimulation trials, with these double sensations often being mislocalized to the palm or upper arm. Interestingly, large mislocalizations and double sensations persisted for several weeks after the tactile stimulators were removed, and localization judgments on the forearm were mislocalized towards the centre of the forearm. These referred sensations even occurred when the participant was viewing touch, suggesting that top-down information did not influence performance. Craig proposed that cortical reorganization was probably responsible for the observed performance. Tactile mislocalizations occurred at locations neighbouring the forearm (palm, upper arm), suggesting a possible expansion of the forearm representation into neighbouring cortical regions. Furthermore, these results provide evidence that reorganization can occur in a manner that leads to mislocalizations, suggesting that changes in map topography are not always correctly accounted for. This is in contrast to other populations who use a skin surface extensively, but do not report these anomalous sensations (e.g., violinists and pianists without focal dystonia), suggesting that there may be limits to cortical reorganization that result in consistent mislocalizations and referred sensations under certain conditions.

Studies of non-human primates and humans have provided evidence for cortical plasticity, such that representations of the skin surface change over a period of months (Jenkins et al., 1990) or years (Pons et al., 1991). Although the time course of plastic changes has been examined in neural recording studies with non-human primates, we are aware of only one study that has examined perceptual changes in tactile localization over time after stroke. Birznieks, Logina, and Wasner (2016) examined tactile localization in an individual with left-hemisphere damage. Nine months after the stroke, the participant made tactile localization errors towards the centre of the hand. However, these localization errors diminished substantially 21 and 60 months after stroke, providing evidence for improvement, perhaps mediated by changes in cortical map topography consistent with the non-human primate literature.

After stroke there is evidence for decreased intracortical inhibition (Brown, Aminoltejari, Erb, Winship, & Murphy, 2009; Murphy & Corbett, 2009; Redecker, Wang, Fritschy, & Witte, 2002) that continues well after the acute stage (Blicher, Jakobsen, Andersen, & Nielsen, 2009; Manganotti, Acler, Zanette, Smania, & Fiaschi, 2008), providing conditions that favour reorganization after brain damage. Furthermore, changes in cortical map topography after stroke are thought

to be mediated by use-dependent plasticity. Given previously observed changes in map topography along with decreased inhibition after stroke, we (Medina & Rapp, 2014) hypothesized that post-stroke somatosensory representations may be more labile than those in normal individuals, even years after stroke. Therefore, we examined two individuals with damage to left primary somatosensory cortex who could feel, but not accurately localize, tactile stimuli presented to their contralesional hand. We presented tactile stimuli to either the hand only, or stimulated points on both the hand and forearm (mixed condition). In the mixed condition, we varied the number of forearm stimuli that immediately preceded stimulation of the hand. Forearm stimulation on the preceding trial shifted percepts of stimuli on the hands towards the forearm. Importantly, these proximal shifts were observed after only a single touch on the forearm, providing evidence that the location of the previous stimulus directly influenced the perceived location of the following stimulus. Use-dependent changes in somatosensory map topography in S1 are thought to be mediated by a competitive process in which increased stimulation of a particular skin surface results in an expansion of its cortical representation (Pearson, Finkel, & Edelman, 1987). Given decreased inhibition in the post-stroke representation, we proposed that the reorganized map is even more labile, resulting in an unstable map topography that is easily influenced by prior stimulation. Importantly, if the function that maps primary somatosensory maps to a representation of body form does not adjust for these rapid changes, then shifts in perceived tactile location should occur.

Localization and uncertainty

The dorsal surface of the hands, palm, and the rest of the arm differ from the palmar surface of the fingers in terms of receptor density (the ventral surface of the palm has approximately 40% of the mechanoreceptor density of the fingers; Johansson & Vallbo, 1979), the skin surface to S1 representation ratio (Jain, Catania, & Kaas, 1998; Jain, Qi, & Kaas, 2001), and morphology (with fingers being segregated while the palm is one continuous surface). Being that the palm and dorsal side of the hand are far less represented than the fingers, one might expect increased error in localizing touch on surfaces other than the fingers. Furthermore, the error patterns produced for localization on these surfaces can provide information regarding tactile processing. When stimulated on regions with lower acuity, is it simply the case that responses reflect some processing of stimulus location with random noise? Or, are there specific biases in tactile localization, providing evidence for how the brain represents location with limited information?

There is evidence that tactile localization accuracy is influenced by body structure – specifically, individuals are more accurate for localization judgments nearer to body part boundaries. Previous studies have found that localization judgments are more accurate for tactile stimulation near the borders than near the middle of the forearm (Hamburger, 1980). Cholewiak and Collins (2003) examined tactile localization accuracy along different body parts by placing an array of seven equally spaced tactors along the arm, asking participants to indicate which one was stimulated. When placing the stimulators along the volar (hairy) forearm, participants demonstrated a U-shaped curve for accuracy, with the best performance at the ends versus the middle of the tactor array. The tactors at the ends of the array were also at the ends of the forearm. Therefore, the better performance at the array terminus could occur because they were at the end of the array itself, or because the array termini were also at the borders of a defined body part (the forearm). If participants used body part boundaries as a reference point, one would predict improved performance at body part borders regardless of array placement. Therefore, in a second experiment, the authors placed the seven tactors (with the same spacing) from the middle of the forearm to the middle of the upper arm. Even though the stimulator at the middle of the forearm was now at the end of the tactile array, accuracy was the poorest at this location, providing evidence that the previously observed U-shaped curve was not based on array position. Furthermore, participants were most accurate for the localization point at the elbow border, providing evidence that body part borders serve as an anchor point for making tactile localization judgments.

Body part boundaries have also influenced performance on tactile distance judgment tasks. De Vignemont, Majid, Jola, and Haggard (2009) examined distance judgments for two tactile stimuli presented along the proximodistal axis of the hand and arm. Distance judgments when the two tactile stimuli were

presented within either the hand or forearm were consistently underestimated, whereas performance was significantly more accurate when the two tactile stimuli were presented across the hand and forearm (crossing the wrist). This increased accuracy near the wrist could be because the wrist provides an anchor point to be used for localization judgments – the farther from the anchor point, the more inaccuracy in localization judgments. However, a second potential explanation is that tactile acuity is simply better at the wrist than at the hand and/or forearm. Therefore, Knight and colleagues (2014) presented participants with pairs of two tactile stimuli, one pair presented along the mediolateral axis with the second pair presented along the proximodistal axis. If participants demonstrate increased acuity at the wrist, then judgments of tactile distance should be more veridical at the wrist regardless of pair orientation. However, if distance judgments are more accurate across body part boundaries, then the more veridical responses should only be observed when the tactile stimuli cross the wrist boundary along the proximodistal axis, and not the mediolateral axis. Although nearly every location was perceived as biased, such that mediolateral distances were judged as shorter than proximodistal distances (see Longo & Haggard, 2011), this bias was the smallest across the wrist, providing evidence that these more veridical responses occur near body part boundaries (see also Cody, Garside, Lloyd, & Poliakoff, 2008). Overall, these results provide evidence that tactile localization for suprathreshold stimuli is influenced in part by body part boundaries, suggesting a mechanism by which these boundaries are referenced in making a localization judgment.

Many individuals with damage to somatosensory cortex are able to detect and localize tactile stimuli (Corkin, Milner, & Rasmussen, 1970), though they often demonstrate errors in localizing suprathreshold stimuli. Although a number of studies have examined constant and variable tactile localization error in populations of brain-damaged individuals (e.g., Connell, Lincoln, & Radford, 2008; Corkin et al., 1970; Semmes, Weinstein, Ghent, & Teuber, 1960), very few have examined the spatial distribution of tactile mislocalization errors after stroke. After damage to somatosensory cortex via stroke, there is substantial reorganization that allows for (in some cases) re-emergence of the cortical representations that were ablated by stroke. However, given the reduction in representational space due to brain damage, the new representation has less cortex available for tactile processing – resulting in a much lower resolution representation due to central damage. Biases in tactile mislocalization after stroke can provide evidence regarding neural reorganization, plasticity, and the role of body part boundaries in localization with limited neural substrate.

Rapp et al. (2002) examined the tactile localization abilities of two individuals with left-hemisphere lesions – both with damage to the "traditional" hand area of left primary somatosensory cortex. To assess tactile localization, both participants were presented with a tactile stimulus to one of 22 locations on their hand (eyes shut) and responded by pointing to where they were stimulated. Although tactile detection was intact for both individuals, they consistently mislocalized tactile sensations. In particular, both participants shifted localization judgments of finger stimuli proximally towards the centre of their hand, whereas localization judgments of stimuli presented to the palm were shifted distally. However, even with these biases, the relative somatotopy of these localization judgments remained largely intact. On average, localization judgments for the distal segment of the finger were more distal than those for the medial segment of the finger, and so on. Importantly, both participants were quite accurate at localizing touch on the ipsilesional hand, providing a within-subject control for potential confounds such as visually guided reaching errors.

As discussed earlier, for tactile localization to occur, there is probably a mapping process from distorted somatosensory maps to more veridical representations of body size and shape. In neurologically intact individuals, there is evidence that distances on skin surfaces with a higher cortical representation to skin surface ratio (e.g., fingertips, lips) are represented as slightly longer than those with smaller cortical representation to skin surface ratios (e.g., forearm, forehead; see Taylor-Clarke, Jacobsen, & Haggard, 2004). After somatosensory damage, there is a significantly smaller representation of the affected skin surface after reorganization that probably projects to a representation of body size and shape. These mislocalization errors towards the centre of the hand likely reflect a mismapping between these representations. However, why do these errors seem to be made

towards the centre of the stimulated body part? One possibility is that these errors reflect general mechanisms in which central localization biases emerge where there is uncertainty about stimulus location. For example, Huttenlocher, Hedges, and Duncan (1991) proposed two sources of information for remembering location in spatial representations – an unbiased estimate of the exact location of the stimulus (fine-grained information) and two estimation processes that take into account categorical information about potential stimulus locations. First, to minimize error in conditions of uncertainty, participants demonstrate a bias towards the prototypical centre of the categorical space (prototype bias). Second, when remembering locations near category borders, potential responses outside of the category boundaries are truncated, also resulting in a bias away from the borders (Huttenlocher, Hedges, Lourenco, Crawford, & Corrigan, 2007; Huttenlocher, Hedges, & Prohaska, 1988). They then developed a model in which categorical information is weighted more strongly as the inexactness for fine-grained information increases. This model predicted localization bias in a task where individuals reproduced the remembered location of a visually presented dot in a circle.

We suggest that similar processes may be used, not only in spatial memory, but by sensory systems in situations with increased sensory noise. For example, somatosensory damage results in less neural substrate available to represent stimulus location, resulting in a noisier position estimate. However, sufficient neural substrate probably remains to localize the touch as within the boundaries of the hand, thus limiting the potential response space to a location within the hand. If so, these biases towards the centre of the hand may reflect increased weighting towards the centre of the hand with increased sensory uncertainty. Furthermore, as the representation of stimulus position becomes more inexact (due to brain damage), one would predict increased central bias. Interestingly, similar central biases in tactile mislocalization have been reported in neurologically intact individuals, depending on stimulus intensity. Steenbergen, Buitenweg, Trojan, and Veltink (2014) asked individuals to judge the location of tactile stimuli of varying intensity presented along the longitudinal axis of the dorsal surface of the forearm. As expected, as stimulus intensity decreased, localization judgment variability increased. Interestingly, they also found that (a) localization judgments erred towards the centre of the forearm, even at the highest intensity level, and (b) this central bias increased as stimulus intensity decreased. These results suggest that these categorical biases influence tactile localization judgments. However, future work will be necessary to understand what processes may be contributing to these central errors, and what these errors may reveal specifically regarding tactile processing and representation.

Touch and distortions in body form: Evidence from amputees, anaesthesia, and illusions

Approximately 60–80% of individuals (Ehde et al., 2000; Kooijman, Dijkstra, Geertzen, Elzinga, & van der Schans, 2000; Wartan, Hamann, Wedley, & McColl, 1997) with an amputated limb experience phantom limb phenomena – in which they report the sensation of a phantom limb even after amputation. Furthermore, some amputees experience referred sensations, such that touch on an intact part of their body results in tactile sensation on the phantom limb itself. These phenomena are interesting for at least two reasons relevant to this review. First, the existence of phantom limbs suggests the existence of a representation of the size and shape of the body that persists even after the limb is gone. Second, localization patterns of referred sensations on the phantom limb can inform us regarding mappings from primary somatosensory maps to body form after peripheral damage. In this section, we critically examine evidence regarding referred sensations after amputation. Next, relatively little research has been done examining how the brain represents body size and shape itself. Therefore, the second half of this section reviews what experiments designed to distort body size and shape tell us about body form representation.

Referred sensations in amputees

As opposed to infrequent reports in individuals with stroke (Aglioti, Beltramello, Peru, Smania, & Tinazzi, 1999; Ramachandran & Rogers-Ramachandran, 2000; Turton & Butler, 2001), referred sensations have often been reported in amputees who experience phantom limbs. Although there are a few cases of phantom referred sensations after stimulating other body parts (breast; Aglioti, Cortese, & Franchini, 1994), the majority of reported cases are after

amputation of the lower arm. In some of the well-known studies by Ramachandran (Ramachandran, 1993; Ramachandran, Rogers-Ramachandran, & Stewart, 1992; Ramachandran, Stewart, & Rogers-Ramachandran, 1992), the experimenter used a cotton swab to brush selected locations on the amputee's face and asked the participant to report the location(s) of the sensation. According to Ramachandran, multiple participants reported feeling touch on their phantom hand after stimulation of the ipsilateral face. Furthermore, there was a direct and consistent mapping for stimulating specific regions of the skin surface and sensation on a particular location of the phantom hand, such that (in the case of one amputee, V.Q.) the area above the lip would elicit referred sensations to the ipsilateral phantom index finger, whereas below the outer lip would elicit referred ipsilateral phantom little-finger sensations. The modality of the stimulus was often, though not always, the same in the referred sensation – that is, drops of water and heat were felt as wet and hot on the phantom hand. Finally, these individuals frequently felt both the actual stimulus presented to the face along with the referred sensation on the phantom limb.

Evidence from human amputees and studies mapping primary somatosensory cortex in non-human primates led to the development of the "remapping hypothesis of referred sensations" (Ramachandran & Hirstein, 1998), inspired by neural recording studies in non-human primates. Examining primary somatosensory maps 2–3 months after third-digit amputation in owl monkeys, Merzenich and colleagues (1984) found that the region of primary somatosensory cortex that represented the amputated digit came to represent neighbouring digits (e.g., digit 2, digit 4). However, this reorganization was spatially limited to 1–2 mm along the cortex, with "silent zones" in the cortex (areas that did not respond to tactile stimulation of the hand) still visible 8 months after amputation. In support of larger scale reorganization over a longer period of time, Pons et al. (1991) examined four macaques who received limb deafferentations at least 12 years before testing. In macaques, the arm representation is bordered by a representation of the trunk and the face. They found that the region in primary somatosensory cortex that previously represented the upper limb was now active for stimulation of the face, with no expansion of the trunk representation into the cortical region that previously represented the upper arm. Furthermore, this new face representation maintained a somatotopic organization that was similar to face maps in neurologically intact macaques (see also Florence & Kaas, 1995; Florence, Taub, & Kaas, 1998). Based on this work, Ramachandran's remapping hypothesis proposed that cortical reorganization over time resulted in changes in primary somatosensory regions such that the neighbouring face area invades the previously silent arm area. As neurons that previously represented the limb now were active for the face, the remapping hypothesis assumes that activation of these neurons results both in veridical sensations and in feeling touch on the phantom limb. This suggests the existence of two mappings from altered primary somatosensory maps to a secondary representation – one that correctly interprets activation subsequent to face stimulation as sensation on the face, and a second mapping that interprets activity in these same neurons (now active for face stimulation) based on their former, pre-amputation mapping – resulting in sensation on the phantom limb.

The remapping hypothesis has been the primary account explaining referred sensations in amputees. However, the primary assumptions and evidence that support this hypothesis have been questioned. First, the remapping hypothesis is predicated on the fact that there are shifts in cortical representations in amputees. Studies using magnetoencephalography in amputees have shown that the dipole centres for face and upper arm stimulation are closer in the affected than in the unaffected hemispheres, providing indirect evidence that the face and arm representations have expanded into the former lower arm region (Knecht et al., 1996; Yang et al., 1994). However, this pattern of cortical reorganization has not been found in more recent functional imaging studies of amputees. Makin, Scholz, Slater, Johansen-Berg, and Tracey (2015) used functional magnetic resonance imaging (fMRI) to examine the location of sensorimotor maps in upper limb amputees and controls. To assess shifts in map topography, amputees and controls were instructed to move specified body parts (e.g., flexion and extension of the fingers, elbows or toes, and limb smacking) for either the intact or the "phantom" limb, in a task designed to activate both motor and somatosensory regions. First, there was evidence for some reorganization in

amputees, as the unfolded cortical distance from the lip representation to the foot representation was significantly smaller in their affected than in the unaffected hemisphere, and compared to controls. However, the lip representation in these individuals did not shift *into* the hand area in the affected hemisphere. Instead, the region medial to the lip area still represented the phantom limb (see also Makin et al., 2013; Raffin, Richard, Giraux, & Reilly, 2016). Given that takeover of the lower arm area by representations of other body parts (upper arm, face) would be predicted for a remapping hypothesis, recent functional neuroimaging evidence would be inconsistent with such an account.

Do these findings provide strong evidence against the remapping hypothesis? In the Makin study (Makin et al., 2013), the authors use changes in the distance of the lip representation as a measure of reorganization of the face area. In the traditional somatosensory homunculus, the unfolded cortical distance from the lip area to hand area is farther than for most other skin surfaces on the face (e.g., eyes, nose, upper face). One possibility is that skin surfaces that are closer to the hand area (e.g., eyes, nose) would show substantially more reorganization, possibly extending fully into the hand representation; whereas areas of the face representation more distant from the limb representation (e.g., the lips) would demonstrate less reorganization. Future studies with amputees should map the entire face area (e.g., Huang & Sereno, 2007) to more fully characterize somatosensory reorganization in amputees.

A second concern for the remapping hypothesis is the quality of evidence for consistently experienced, somatotopically organized referred sensations. First, a number of studies on referred sensation (e.g., Halligan, Marshall, & Wade, 1994; Halligan, Marshall, Wade, Davey, & Morrison, 1993; Ramachandran, 1993; Ramachandran, Rogers-Ramachandran, et al., 1992; Ramachandran, Stewart, et al., 1992) did not present tactile stimuli in a pre-selected, randomized manner, nor did they report any statistics on the within-session replicability of these referred sensations – in many cases simply claiming that patterns were identical within a session. Demand characteristics, in which participants realize the purpose of the experiment and respond in the expected manner, could lead a participant to experience referred sensations in a consistent and "somatotopic" manner. To account for this, future studies should clearly identify the stimulation sites ahead of time, randomly present stimuli to these sites (without repetition at a location), and have a clear method for reporting and recording the location of the referred sensations. Second, studies that have used preselected stimulation points with random selection either do not report a somatotopic mapping of referred sensations, or present findings that are inconsistent with the remapping hypothesis. For example, Knecht and colleagues (1996) stimulated 30 different locations on the body of eight arm amputees. Although referred sensations were elicited in seven of these participants, a somatotopic relationship between stimulation points and phantom sensations was reported in only one participant. Grusser and colleagues (2004) reported two individuals in whom referred sensations to the digits occurred after stimulation of the intact limb, along with stimulation of both the ipsilateral and contralateral feet. Given that the foot representation is quite distant from the hand and arm representation, it is unlikely that stimulation of the foot should result in phantom sensations on the hands based on a remapping hypothesis.

Third, nerve fibre stimulation and tactile acuity studies are not consistent with a remapping hypothesis. For example, Schady, Braune, Watson, Torebjörk, and Schmidt (1994) used microelectrodes to stimulate the nerve fibres that previously innervated the amputated limb. If remapping occurred, stimulation of these nerve fibres should activate remapped cortical representations that were previously active for the amputated limb (e.g., face representation). However, stimulation of these nerve fibres resulted simply in sensations on the phantom hand, and not on any other body parts (see also Moore & Schady, 2000). Finally, consistent with non-human primate studies, the remapping hypothesis would predict that digit amputation would result in substantial expansion of the amputated digit representation by the neighbouring finger representations. For example, Merzenich and colleagues (1984) found a 65–80% increase in the size of the index and ring finger representations after amputation of the middle finger. Given a higher cortical representation to skin surface ratio due to this remapping, one may predict higher tactile acuity on the neighbouring fingers after amputation. However, Vega-Bermudez and Johnson (2002) found no differences in tactile acuity on the finger

neighbouring the amputated finger compared to the same finger on the non-amputated hand (see also Frey, 2015, Figure 21.1). In summary, although there is evidence for referred sensations in amputees, there are a number of questions regarding whether the evidence supports the traditional remapping hypothesis. As understanding how cortical reorganization influences perception in amputees has definite clinical and cognitive implications, future research should aim to test the remapping hypothesis along with other alternatives.

Representing body size and shape

We have proposed the existence of a representation of body size and shape (body form) necessary to map the location from primary somatosensory representations to a representation of the skin surface. What are some of the characteristics of this representation? And how do changes in perceived body size and shape change the perceived location of tactile stimuli? To examine the perceptual effects of anaesthesia, Gandevia and Phegan (1999) anaesthetized the thumb or lip and asked participants to report the perceived size (via both motor responses and template matching) of the anaesthetized body part, its cortical neighbours (e.g., the index finger subsequent to thumb anaesthetization), and the opposite homotopic body part (see also Walsh, Hoad, Rothwell, Gandevia, & Haggard, 2015). Local anaesthesia to the thumb resulted in an approximately 60–70% increase in the perceived size of the anaesthetized thumb, with a similar increase in perceived lip size, and no perceived change in the index finger neighbouring the anaesthetized thumb, nor in the unanaesthetized thumb. Anaesthetization of the lip resulted in a significant increase in perceived lip size, with only a slight increase in perceived thumb size (see also Türker, Yeo, & Gandevia, 2005, for similar findings after lip and teeth anaesthetization). In explaining these results, the authors note that removal of peripheral input does not result in a sense of a missing body part, providing evidence for a persistent representation of body size and shape even when peripheral input is removed. The authors propose that the increase in receptive field size after anaesthetization (see Calford & Tweedale, 1988, 1991) results in an increase in tonic background somatosensory activity that is interpreted as an increase in body size.

Changes in perceived body size have also been observed after removing input to entire limbs. Paqueron and colleagues (2003) found that nerve block of the upper and lower limb always resulted in a change in the perceived width of the body part, with some participants (11/30) reporting a perceived change in limb length as well. Interestingly, four out of 10 participants with upper limb block (versus zero out of 10 with lower limb block) reported swelling of the lips, providing evidence for some relationship between perceived changes in body size and somatosensory map topography. Given that amputation (Rasmusson, Webster, & Dykes, 1992) and nerve section (Dykes & Lamour, 1988) can result in increased spontaneous activity in somatosensory cortex, the authors suggest that increases in background somatosensory activity may lead to changes in perceived body size. Paqueron et al. (2003) noted that changes in perceived limb shape typically occurred earlier than changes in perceived limb position. Silva et al. (2010) specifically analysed the time course of kinaesthetic illusions after upper arm nerve block and found that changes in perceived body size always preceded changes in perceived body position in all 20 tested individuals, with changes in perceived body size occurring (on average) 15 minutes before changes in perceived body position. Overall, these findings after anaesthetization suggest that tonic activity – neuronal activation without presynaptic input – somehow mediates perceived body size and shape, such that removal of input results in increases in body size. Furthermore, removal of input may also influence perceived body size in parts neighbouring the anaesthetized region on the somatosensory map. However, the mechanisms that underlie these effects are unknown.

A second manner in which illusory changes in limb size can occur is via muscle vibration. Vibration of muscle tendons results in reflexive contraction of the muscle – known as the tonic vibration reflex. If this reflexive movement is resisted, and the participant does not have vision of the vibrating limb, illusory motion of the stimulated limb is experienced. It is thought that muscle vibration results in high levels of activity in muscle spindles and Golgi tendon organs that is interpreted as stretch, resulting in illusory movement. To examine the contributions of higher order representation to body perception, Lackner (1988) stimulated the muscles of a limb

while the participant was touching another body part. Not only did participants experience illusory movement of the stimulated limb, but they also experienced changes in the perceived shape of the other touched body part. For example, stimulation of the biceps while holding the nose resulted in the sensation of an elongated nose, or the "Pinocchio illusion". This illusion provides strong evidence for the existence of a higher order representation of the size, shape, and geometry of the body. Furthermore, it also shows that to resolve potential conflicts, the representation of body size and shape is malleable and can result in a distorted body shape representation. The Pinocchio illusion has been used to understand the relationship between perceived body size and shape and tactile localization. De Vignemont, Ehrsson, and Haggard (2005) used a variant of the Pinocchio illusion to create illusory lengthening of a participant's finger. To examine whether the perception of tactile stimulus position references this distorted representation of an extended finger, the authors presented two tactile stimuli at the same distance to either the participant's forehead or their finger and asked participants to choose whether the finger distance was longer than the forehead distance. They found that distances on the finger were perceived as longer when the finger was perceived as elongated. Similar changes are also observed when the visual size of the body is altered. Taylor-Clarke et al. (2004) found that perception of tactile distance on the forearm increased when viewing the forearm as elongated, and decreased when viewing the forearm as shortened.

Interestingly, there are few reports from brain-damaged individuals exhibiting distortions of perceived body size and shape. Macrosomatognosia and microsomatognosia (perceiving body parts and larger/smaller) have been reported subsequent to migraine auras (Podoll, Muhlbauer, Houben, & Ebel, 1998; Podoll & Robinson, 2000, 2002). We know of only one case of macrosomatognosia after stroke, in which an individual with a lateral medullary stroke and left facial anaesthesia perceived swelling of the mid-face (Rode et al., 2012). The relative rarity of changes in perceived body size after brain damage may reflect the inherently multisensory nature of these representations. The Pinocchio illusion and illusory changes in body size after anaesthetization does not occur when participants have vision of their own body. Even if somatosensory and/or proprioceptive inputs are damaged, information from other senses (such as vision) may compensate, leading to maintenance of a coherent, veridical body schema.

Localizing touch: Mirror errors and external space

In this final section, we review patterns of tactile mislocalization that are quite distinct from the errors reviewed so far. Although relatively underreported, some individuals report dyschiric phenomena in which tactile sensations are localized to the same location on the opposite side of the body. In this section, we review evidence from individuals with different dyschiric phenomena (including allochiria and synchiria) and discuss how these and other findings provide evidence for interhemispheric involvement in representing touch (see also Tame, Braun, Holmes, & Pavani, 2016). Next, synchiric phenomena and other aspects of tactile sensation are influenced by body position in external space (for a review, see Badde & Heed, 2016). We briefly discuss how representations of external space may influence tactile performance, focusing on evidence from brain-damaged individuals.

Mirror errors – dyschiric phenomena

The term *dyschiria* covers a number of separate phenomena that encompass an inability to accurately report which *side* of the body has been touched (for a discussion of terminology, see Meador, Allen, Adams, & Loring, 1991). Individuals with tactile *achiria* (Janet, 1898) or "simple allochiria" (Jones, 1908) can correctly localize the body part that was stimulated, but have no knowledge regarding which side of the body was stimulated. Mislocalization of tactile stimuli to the homologous location on the opposite side of the body is known as *allochiria*. An individual with tactile *synchiria* (Janet, 1898) will, when presented with a tactile stimulus on the ipsilesional side of the body, report sensations both where the stimulus was presented and at the homologous location on the opposite, contralesional side of the body.

Allochiria has been observed both after spinal cord and after cerebral damage. In a review of dyschiric phenomena in the late 19th and early 20th century,

Jones (1908) reported 29 cases of dyschiria, two demonstrating a form of synchiria, one reporting achiria, and the remaining subjects showing allochiria. The vast majority of the allochiric cases in this report were subsequent to tabes dorsalis (sclerosis of the posterior columns of the spinal cord; see also Obersteiner, 1881). In more recent literature, Kawamura and colleagues (1987) tested 123 acute patients with cerebral haemorrhage and found 20 (18 with damage to the right putamen) that demonstrated allochiria to strong pinch stimuli. These allochiric sensations were temporary and were not observed approximately three weeks after haemorrhage. Some, but not all, participants also demonstrated allochiria when presented with light touch, hot, cold, and vibratory stimuli. Allochiria has also been observed in response to non-painful stimuli in a few cases (Bender, Shapiro, & Teuber, 1950; Young & Benson, 1992). To our knowledge, there are no cases of tactile allochiria (e.g., mislocalization of touch presented on one side of the body to the other side of the body) in the absence of brain damage. However, Marcel and colleagues (2004) have reported that when two different tactile stimuli are presented in the homologous location on each side of the body (e.g., a tap on one side vs. a "drum" on the other side), the location of the type of tactile stimulus can be mislocalized.

Although there have been many cases of tactile allochiria in the literature, there have been few reports of tactile synchiria, with the majority being cases published over 100 years ago. Janet (1898) reported a subject that, when pinched on one side of the body, initially reacted by tightening the pinched location on both sides of the body. Drinkwater (1913) reported a 13-year-old subject with a family history of motor synchiria and no apparent signs of brain damage. When the subject was instructed to make a voluntary movement with one side of the body, that movement was made with both sides of the body. Furthermore, when "every variety of cutaneous stimulus" was delivered to either limb (and only the limbs), the subject reported feeling the stimulus on both sides of the body (see also Kramer, 1917). More recently, Sathian (2000) reported six individuals with brain damage due to infarction or resection that led to a deficit in light touch and pinprick sensation on the contralesional hand. Sathian presented stimuli to the participant's ipsilesional hand while in a mirror box, such that it looked like both hands were being touched. Pressure, but not pinprick or cold, stimuli presented to the ipsilesional hand resulted in sensations on both the ipsilesional and the contralesional hand. The majority of participants (5/6) also demonstrated these phantom contralesional percepts when the hand was not in a mirror box. These phantom sensations were referred to the correct finger, but not finger segment, whereas stimulus characteristics such as stimulus orientation, Braille patterns, or letters pressed onto the skin surface, could not be distinguished in the phantom sensations. Sathian suggested that the referred sensations are mediated by callosal connections between higher order somatosensory areas. Synchiria has also been observed in some individuals with chronic regional pain syndrome (Acerra & Moseley, 2005; Krämer, Seddigh, Moseley, & Birklein, 2008).

To explore the potential mechanisms that could give rise to these phantom percepts, Medina and Rapp (2008) examined synchiric phenomena in an individual (D.L.E.) with an extensive left-hemisphere lesion that included a large portion of somatosensory cortex and lateral, anterior thalamus (probably sparing the ventroposterior nucleus, which is involved in somatosensory processing). Surprisingly given the extent of his lesion, D.L.E. could detect light touch presented to his contralesional hand – although his tactile localization ability was highly inaccurate. Regardless of where tactile stimuli were presented on his contralesional hand, D.L.E.'s responses typically clustered on his third and fourth finger. Interestingly, when stimulated on his ipsilesional hand, he frequently reported sensations on both the ipsilesional and the contralesional hand. These phantom synchiric percepts were frequent, highly localizable, and "accurate" – that is, localized to the homologous location of the ipsilesional stimulus. In fact, these phantom synchiric percepts on the right contralesional hand were more accurately localized than actual stimulation of the right hand. When two stimuli were presented simultaneously to different locations on each hand (e.g., index finger of left hand, little finger of right hand), D.L.E. reported contralesional sensation in the location of ipsilesional stimulation on 82% of trials (see also Satomi, Kinoshita, Goto, Sakai, & Ito, 1989, for another patient with both tactile extinction and synchiria).

What mechanisms could give rise to dyschiric phenomena? Subsequent to tactile stimulation, activation travels through the spinal cord and then crosses over to the contralateral thalamus and then to contralateral primary somatosensory cortex (Jones & Powell, 1970; Kaas, Nelson, Sur, Dykes, & Merzenich, 1984). However, in addition to this well-known contralateral pathway, there is extensive evidence for ipsilateral primary somatosensory activity (see Tame et al., 2016, for a discussion). This has been observed with magnetoencephalography (MEG; Kanno, Nakasato, Hatanaka, & Yoshimoto, 2003; Korvenoja et al., 1995; Schnitzler, Salmelin, Salenius, Jousmaki, & Hari, 1995) and fMRI (Hansson & Brismar, 1999; Korvenoja et al., 1999; Nihashi et al., 2005) after median nerve stimulation or natural tactile stimulation of the skin surface. There is evidence for two pathways that could lead to ipsilateral activity – one type that is subcortical, and a second type that is transcallosal.

Evidence for ipsilateral activation via a non-callosal pathway has been found in studies examining somatosensory evoked potentials (SEPs) to median nerve stimulation in primary somatosensory cortex. SEPs to median nerve stimulation were recorded from 41 epilepsy patients via subdural electrode grids (Noachtar, Luders, Dinner, & Klem, 1997). Along with standard contralateral SEPs, four subjects also generated detectable ipsilateral SEPs that were of lower intensity and longer latency than the contralateral SEPs. As the latencies between contralateral and ipsilateral SEPs were shorter than those observed during transcallosal transmission, it is unlikely that the ipsilateral SEPs were generated via transcallosal transmission. In a different study, one participant with extensive right S1 and S2 damage showed activation in ipsilateral S2 using MEG (Forss, Hietanen, Salonen, & Hari, 1999). As there was no right S1 or S2 to send activation via the corpus callosum, the authors suggested that the ipsilateral activity was due to a direct, non-callosal ipsilateral pathway (see also Kanno, Nakasato, Nagamine, & Tominaga, 2004). One candidate pathway for this observed activation is a direct connection from the thalamus to ipsilateral sensorimotor cortex (Brus-Ramer, Carmel, & Martin, 2009; Gonzalez et al., 2004).

A second pathway utilizing the corpus callosum may be involved in tonic inhibition of somatosensory areas. Calford and Tweedale (1990) anaesthetized or amputated a body part (for example, the right thumb) and recorded from S1 (area 3b) in both somatosensory cortices in the flying fox and macaque. After anaesthetization of the right thumb, the receptive fields for left-hemisphere S1 neurons immediately expanded to represent regions of skin surface that were adjacent to the right thumb. Without anaesthetization, tonic activation from baseline sensory activity on the right thumb results in intrahemispheric inhibition that limits the size of the neuron's receptive field. However, when input from the anaesthetized and/or amputated body part is removed, intracortical inhibition is also removed, thus leading to immediate expansion of this neuron's receptive field. Interestingly, a similar, immediate expansion of receptive field size was also observed in the hemisphere *ipsilateral* to the anaesthetization. The authors concluded that tonic activity within the same somatosensory cortex via an interhemispheric pathway leads to inhibition of contralateral (right-hemisphere) somatosensory cortex. When the right thumb is anaesthetized and/or amputated, there is decreased tonic activity in the left-hemisphere somatosensory thumb representation and, via the interhemispheric pathway, decreased inhibition of the right-hemisphere thumb representation, leading to larger receptive fields for "thumb" neurons in both the left and right hemisphere (see also Clarey, Tweedale, & Calford, 1996). Consistent with these findings, Lipton, Fu, Branch, and Schroeder (2006) use recordings from a linear array of multielectrodes in area 3b and found a strong ipsilateral inhibitory response subsequent to median nerve stimulation (see also Reed, Qi, & Kaas, 2011).

Evidence for ipsilateral inhibition after tactile stimulation has also been observed in humans. After stimulation of the fingers of the right hand using balloon diaphragms, Hlushchuk and Hari (2006) found an increase in blood-oxygen-level-dependent (BOLD) response in Brodmann area 3b in contralateral S1. However, they also found a decrease in BOLD signal in the homotopic region in ipsilateral area 3b in somatosensory cortex. Kastrup and colleagues (2008) showed that this decrease in ipsilateral BOLD signal in S1 was strongly correlated with increases in stimulation thresholds on the limb that was not stimulated (see also Brodie, Villamayor, Borich, & Boyd, 2014; Klingner, Hasler, Brodoehl, & Witte, 2010; Schäfer et al., 2012). Finally, after stimulation of the right median nerve in individuals with right-hemisphere

somatosensory damage, Forss et al. (1999) found prolonged left-hemisphere N20 m components – an ERP marker of early somatosensory processing – compared to controls. These results are consistent with an account in which removal of tonic inhibition due to damage in the lesioned hemisphere results in stronger somatosensory activation in the undamaged hemisphere (see also Reisecker, Witzmann, & Deisenhammer, 1986).

Tactile synchiria may be explained in light of previous findings regarding callosal interhemispheric inhibition. We propose that after stimulation of the ipsilesional hand, activation related to that stimulation travels from primary somatosensory cortex to higher level integrative representations of the body in the intact hemisphere. Given that there are no transcallosal connections between Brodmann area 3b in somatosensory cortex, whereas there are extensive connections for higher processing stages, it is likely that information regarding the tactile stimulus is transmitted to the damaged hemisphere via callosal connections between these higher order regions. In the participants we studied with synchiria (Medina & Rapp, 2008), both superior parietal cortex and the angular gyrus were intact and may have mediated this transmission – though we note that future neuroimaging work is needed to examine this further. In these cases, we propose that mechanisms demonstrating inhibition of ipsilateral somatosensory activation discussed in the previous paragraph are damaged – perhaps as a result of the removal of tonic inhibition. Removal of this inhibition may give rise to synchiric individuals feeling touch on both sides of the body when only stimulated on the ipsilesional side.

Localization error and external space

The location of touch can be represented in a manner that does not take into account stimulus position relative to other body parts, objects, and so on. However, knowledge about where touch occurs relative to other body parts and the environment is clearly important (see Badde & Heed, 2016, for an extensive discussion of this topic). A number of studies with brain-damaged individuals have found that changes in body position modify tactile percepts. For example, the frequency of D.L.E.'s phantom synchiric percepts (Medina & Rapp, 2008) was modulated based on hand position relative to his trunk and head. D.L.E. demonstrated significantly more phantom synchiric percepts when his hands were positioned in contralesional than in ipsilesional space in a trunk-centred or head-centred frame of reference, providing evidence that inhibitory of ipsilateral somatosensory activation operates in multiple, external reference frames. Furthermore, a number of studies have shown that hand position in external space modulates detection rates in individuals with tactile extinction, with less sensation on the contralesional hand when it is in contralesional versus ipsilesional space (Auclair, Barra, & Raibaut, 2012; Bartolomeo, Perri, & Gainotti, 2004; Berti et al., 1999; Moro, Zampini, & Aglioti, 2004; Moscovitch & Behrmann, 1994; Peru, Moro, Sattibaldi, Morgant, & Aglioti, 2006; Smania & Aglioti, 1995; Vaishnavi, Calhoun, & Chatterjee, 2001; Valenza, Seghier, Schwartz, Lazeyras, & Vuilleumier, 2004). These results are typically explained by an attentional deficit that affects stimulus detection in the contralesional side of external space. Interestingly, the body itself may also influence the allocation of tactile attention. Coslett and Lie (2004) reported two individuals with left tactile extinction whose performance improved when the ipsilesional hand was in contact with the stimulated contralesional skin surface. This improvement in performance only occurred when the ipsilesional hand touched the contralesional hand, as was not observed when the skin surfaces were separated by a cloth, or near but not touching. Coslett and Lie suggested that self-touch may serve to automatically draw attention and, in these cases, ameliorate tactile extinction.

Are there also changes in the perceived *location* of tactile percepts on the skin surface due to changes in body position? Coslett (1998) examined the ability of three individuals with brain damage to report which finger was stimulated with their fingers positioned close together versus far apart. One participant (J.M.) made significantly more identification errors (with both verbal and motor responses) on the contralesional hand when the fingers were close than when they were far apart, suggesting an impairment in the coding of tactile location in external space. Consistent with this hypothesis, J.M. made significantly more errors on adjacent than on non-adjacent fingers when fingers were grouped (e.g., index/middle finger and ring/little finger grouped) in a follow-up experiment (see also Schwoebel, Coslett, &

Buxbaum, 2001). Similar effects of finger position on tactile localization have been observed in neurologically intact individuals. Haggard and colleagues (2006) presented individuals with suprathreshold tactile stimuli to participants' unseen fingers, with the hands positioned either vertically one on top of the other, or in the same vertical posture with the fingers of each hand interwoven. Across a number of manipulations, they found that hand position did not influence identifying which finger was stimulated, but did find that participants were poorer at knowing which hand was stimulated in the interwoven versus vertical postures. They proposed that identifying the stimulated finger does not need to reference the position of the body in external space – it is essentially a somatotopic task. Identifying which hand was stimulated involves mapping the presented touch on a finger to the position of a hand in external space. When the fingers are interleaved, this mapping process is more difficult, thus resulting in more hand identification errors. Overvliet, Anema, Brenner, Dijkerman, and Smeets (2011) presented near-threshold tactile stimuli to the fingertips with the fingers close together or spread apart, or with the fingers from each hand interleaved. Instead of asking participants which hand or finger was stimulated, they had participants respond using a forced-choice paradigm. They found that localization accuracy was most accurate with the fingers apart when compared to when the fingers were either close together or interwoven. Given that performance was most accurate when the fingers were farthest apart in external space, with no changes in a somatotopic representation, these results suggest that representations of external space play a role in tactile localization.

Conclusion

In this review, we have focused on how perceptual errors, both in neurologically intact and in brain-damaged individuals, can inform us regarding how the brain represents touch and the body. First, does the brain have separable, parallel pathways for tactile detection and localization? Although evidence from individuals with numbsense seems to suggest parallel processes, our review of the evidence suggests that tactile detection and localization are serial processes, with detection being necessary for localization. Second, primary somatosensory maps are both plastic (changing subsequent to central damage, peripheral damage, and usage patterns) and non-uniform (with more cortex dedicated to more relevant skin surfaces). Given changes in map topography, how does the brain deal with these changes in an attempt to accurately localize tactile sensation? We proposed the existence of a representation of body size and shape (body form) that is used to scale information from distorted primary maps to a veridical representation of the skin surface. Mislocalizations may occur when changes in primary somatosensory maps due to use-dependent plasticity are either too fast or too extensive to be properly interpreted by higher order body form representations. Furthermore, in conditions with uncertainty about stimulus position, we suggest that information about body part boundaries along with general tendencies to err centrally contribute to errors in tactile localization. Third, individuals can have experiences in which their perceived body size and shape are not consistent with their actual bodies. Examples include amputees who experience phantom limb phenomena and individuals who experience changes in perceived body size and shape due to anaesthesia or illusions. How does the brain map activation in primary somatosensory representations onto these atypical body form representations? We discuss several concerns regarding the traditional "remapping hypothesis" – in which changes in map topography after amputation are misinterpreted in a consistent manner. We propose that future studies of amputees systematically and carefully assess referred sensations to refine our understanding of this issue.

Finally, neuropsychological studies can often provide serendipitous results that lead to the discovery of mechanisms that would not be easily predicted. In our final section, we discussed how evidence from individuals who feel touch on the opposite side of stimulation provide evidence for interhemispheric connections between somatosensory areas, as well as evidence for inhibitory mechanisms that, when damaged, result in illusory percepts on the opposite side of the body. Although there is much to be known regarding how the mind represents the body, we hope that this review highlights the contributions of evidence from brain-damaged individuals and how careful examination of error patterns from both brain-damaged and neurologically healthy individuals can be used to develop testable models of body representation.

Disclosure statement

No potential conflict of interest was reported by the authors.

Funding

This work was supported by the National Institute of Neurological Disorders and Stroke (NINDS) [grant number NS089084]; and the National Institute of General Medical Sciences (NIGMS) [grant number GM104941] of the National Institutes of Health (NIH); along with a grant from the University of Delaware Research Foundation.

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