

Vision of the body modulates processing in primary somatosensory cortex

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ABSTRACT

Viewing the body affects somatosensory processing, even when entirely non-informative about stimulation. While several studies have reported effects of viewing the body on cortical processing of touch and pain, the neural locus of this modulation remains unclear. We investigated whether seeing the body modulates processing in primary somatosensory cortex (SI) by measuring short-latency somatosensory evoked-potentials (SEPs) elicited by electrical stimulation of the median nerve while participants looked directly at their stimulated hand or at a non-hand object. Vision of the body produced a clear reduction of the P27 component of the SEP recorded over contralateral parietal channels, which is known to reflect processing in SI. These results provide the first direct evidence that seeing the body modulates processing in SI and demonstrate that vision can affect even the earliest stages of cortical somatosensory processing.

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Vision of the body alters somatosensation, even when entirely non-informative about stimulation. Viewing the body speeds tactile reaction time [59], improves tactile acuity [32], reduces intensity of acute pain [33], and heightens therapeutic effects of tactile training on chronic pain [40]. It also modulates amplitude of both tactile somatosensory evoked potentials (SEPs [17,50,54]), and nociceptive laser evoked potentials (LEPs [33]). The neural mechanisms underlying such effects, however, are unclear. Transcranial magnetic stimulation (TMS) applied to contralateral primary (SI) – but not secondary (SII) – somatosensory cortex reduced the visual enhancement of touch [16], suggesting that vision may modulate SI processing. However, similar disruption has also been reported from TMS applied over more posterior parietal regions, such as the intraparietal sulcus [48], known to be involved in visuo-tactile interactions [35] and related to tactile acuity [52].

Allison et al. [1–3] distinguished *short-* and *long-latency* SEPs. Short-latency components, up to around 40 ms, are generated exclusively within area 3b of SI [2]; long-latency components, after 40 ms, are generated by several areas in addition to SI [3] (for review, see [2]). Previous studies of seeing the body on SEPs [50,54] used tactile stimuli, which commonly fail to produce clear components before 50 ms, and thus have demonstrated effects only on long-latency components. Taylor-Clarke et al. [54] found modulation of the N80 and N140 components, while Sambo et al. [50]

found that seeing the hand modulates effects of tactile attention on the P100 and N140. Further, we [33] found that seeing one's hand reduces the amplitude of the N2/P2 LEP complex (peaking between 200 and 400 ms). This complex has been observed in intracranial recordings from SI [41], but is also associated with other brain regions such as the anterior cingulate and SII [20]. Thus, while existing findings are consistent with modulation of SI, none implicate SI directly.

We investigated the effects of seeing the body on processing in SI using short-latency SEPs. Participants looked either directly at their right hand or a non-hand object (a piece of wood) while we stimulated their right median nerve. We investigated the effects of vision on the early 20 and 30 ms potentials, specifically the N20 and P27, commonly taken to unambiguously implicate SI [1,2,63].

Fourteen healthy volunteers (13 males) between 18 and 30 years of age were tested. All but one were right-handed as assessed by the Edinburgh Inventory [42]. Data from two additional participants were excluded due to excessive noise in the EEG. Procedures were approved by the local research ethics committee.

Square-wave electrical pulses (amplitude 10 mA) were delivered transcutaneously to the right median nerve at the wrist using a neuromuscular stimulator at 4 Hz. Pulse duration was manipulated so stimuli generated small, but clear, thumb twitches (M : 138 μ s; range: 70–210).

To ensure maintained tactile attention, participants performed a tactile discrimination task interleaved with electrical stimulation. Tactile stimuli were square-wave gratings applied manually to the right index fingertip. Participants judged whether the grating ran *along* or *across* the finger [60]. Thresholds were obtained

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for each participant starting with the largest ridge-width (1.5 mm) and working down until accuracy was approximately 60%. A cardboard skirt was placed around stimuli so that orientation could not be seen.

Participants sat at a table with their right arm resting palm-up. In the *view hand* condition, participants focused visual attention and gaze directly on their stimulated right hand. A baffle blocked view of the experimenter. In the *view object* condition, the baffle was moved in front of the hand and a small wooden block (approximately hand-sized) was seen and fixated.

One thousand median nerve stimuli were delivered in each block. Twenty square-wave gratings (half *along*, half *across*) were applied periodically during the block. Nerve stimulation stopped during the tactile stimuli. The number of electrical stimuli between touches was varied (30, 40, 50, 60, or 70) to make the timing of touch unpredictable. The experimenter held the grating directly above the finger and saw a timer countdown, so touch began immediately following nerve stimulation, preventing participants from waiting until after nerve stimulation to orient tactile attention. In the *view object* condition, a second grating was held above the object and pressed down at the same time as the actual stimulus, so temporal cueing was equivalent across conditions. Participants made unspeeded judgments of grating orientation by pressing one of two buttons with their left hand. The grating remained pressed to the fingertip until the response, when electrical stimuli resumed.

Blocks alternated between *view hand* and *view object* conditions (initial condition counterbalanced across participants). Participants completed up to four blocks (mean 3.2 blocks) of each visual condition (some chose to end the experiment before completion).

A SynAmp amplifiers system and Scan 4.3 software (Neuroscan, El Paso, TX) were used to record electroencephalographic (EEG) data. Recordings were obtained from 14 scalp electrodes, (Fz, C3, Cz, C4, CP5, CP3, CPz, CP4, CP6, P3, Pz, P4, O1, and O2), placed according to the 10–20 System. Horizontal electroculogram was recorded bipolarly from electrodes on the outer canthi of each eye, and vertical electroculogram was recorded from an electrode below the right eye. The reference electrode was AFz. Electrode impedances were kept below 5 k Ω . EEG signals were amplified, bandpass filtered from 0.05 to 1000 Hz (slope: 12 dB/octave), and digitized at 5 KHz.

EEG data were analyzed with EEGLAB [10]. Data were segmented into epochs timelocked to electrical pulses (–10 to 95 ms) and visually inspected to remove obvious artifact. Because the large electrical artifact induced by stimulation affected digital filtering, the first 6 ms after stimulation in each epoch were cut and linearly interpolated. Data were then re-referenced to the average of the left and right mastoids, digitally low-pass filtered at 400 Hz, and the interval between –10 and 0 ms was used for baseline correction. Epochs in which voltage exceeded $\pm 80 \mu\text{V}$ at any channel were eliminated ($M = 10.3\%$ of trials rejected, $SD = 9.9\%$).

We focused on the short-latency 20 and 30 ms components. Both pairs of components reverse polarity across the central sulcus, resulting in N20 and P27 (sometimes called P30) over parietal channels and P20 and N30 over frontal channels [2,11]. The relation between frontal and parietal components is controversial. Some researchers argue that they reflect opposite poles of single tangential generators in SI [1,2,4,38,63]; others claim they are generated by separate, radial generators in SI and motor cortex, respectively [11,37,43,49,61]. Clearly, this latter ‘dual-radial’ hypothesis contradicts the suggestion that short-latency SEPs unambiguously implicate SI. Crucially for present purposes, however, these hypotheses differ only with regard to the frontal P20 and N30; both agree that the parietal N20 and P27 originate in area 3b. Thus, we focus on the N20 and P27, widely agreed to reflect SI processing.

We computed mean and peak amplitude for each participant in both visual conditions for the N20, P27, and the later P45 component. N20 amplitude was calculated as the mean (or minimum) voltage between 17 and 23 ms, the P27 as the mean (or maximum) between 22 and 32 ms, and the P45 as the mean (or maximum) between 40 and 50 ms. For simplicity, we averaged the three contralateral parietal channels (CP3, CP5, and P3). We also analyzed these same components, with opposite valence, at Fz, to assess ‘frontal’ SEP components.

Figs. 1 and 2 show scalp maps and SEPs from contralateral parietal channels. There was no significant difference in N20 amplitude when viewing the hand compared to the object, *mean amplitude*: $-.33$ vs. $-.20 \mu\text{V}$, $t(13) = -1.57$, $p > .10$; *peak amplitude*: -1.23 vs. $-1.11 \mu\text{V}$, $t(13) = -1.34$, $p > .10$. However, seeing the hand did produce a clear reduction of P27 amplitude, *mean amplitude*: $.14$ vs. $.43 \mu\text{V}$, $t(13) = -2.67$, $p < .02$; *peak amplitude*: 1.05 vs. $1.37 \mu\text{V}$, $t(13) = -2.39$, $p < .05$. There was also a marginally significant reduction of P45 amplitude, *mean amplitude*: 1.21 vs. $1.41 \mu\text{V}$, $t(13) = -2.00$, $p = .067$; *peak amplitude*: 1.93 vs. $2.15 \mu\text{V}$, $t(13) = -2.05$, $p = .062$. Fig. 2 (right panel) shows a difference waveform; the negativity produced by seeing the hand peaks in the time window of the P27, but extends substantially later, though this difference is only marginally significant by the time window of the P45.

No effects of vision were apparent on SEPs measured at Fz, for the N20, P27, or P45 time windows (all $ps > .20$).

Analysis of grating orientation judgments revealed that performance had, indeed, been kept near 60%, but that participants were above chance both when looking at the hand (61.2% correct), $t(13) = 3.57$, $p < .005$, and the object (58.5% correct), $t(13) = 3.66$, $p < .005$. Performance did not differ significantly between the two conditions, $t(13) = .82$.

Seeing the body modulates processing in primary somatosensory cortex. The short-latency P27 SEP component was reduced when participants looked directly at their stimulated hand, compared to an object. These results are consistent with previous findings of modulation of longer-latency SEPs [50,54] and LEPs [33], but show for the first time effects on a component unambiguously implicating SI. Our findings also complement the finding [16] that TMS applied to SI reduces visual enhancement of touch.

Could our results reflect processing in SII? Most studies have failed to identify SII activation prior to 60 ms, whether with intracranial recordings [1,18], scalp EEG [24,61], or MEG [25,26,30,36]. Two studies, however, report SII activations before 50 ms, one using intracranial recordings [7] one with MEG [31]. This fits into a larger debate concerning whether the organization of SI and SII is serial and hierarchical [28,29,45] or largely parallel [44,46,64], which is beyond the scope of this paper. Nevertheless, we consider it unlikely that SII activation underlies the present effects. First, such early signals are small and only infrequently observed (never to our knowledge with EEG). Second, the early activity observed in SII with MEG [31] was an increase in root mean-squared “system noise,” not evoked responses. Such activation would not show up on averaged SEPs. Indeed, Karhu and Tesche [31] reported evoked activation in SII only around 40 ms, considerably later than our effects. Similarly, evoked-responses in other regions, like the pre-SMA [6], have not been reported before 50 ms.

Can our results be explained by spatial attention? While participants fixated their index fingertip in the *view hand* condition, there was no specific focus in the *view object* condition. We consider it unlikely, however, that spatial attention can account for our results. Studies of spatial attention on SEPs report that directing within-hand spatial attention to specific fingers modulates the P100 and subsequent activity, but *not* earlier components [15]. Further, directing attention to the hand generally produces voltage increases in the P27 time window [12,19], exactly opposite to the

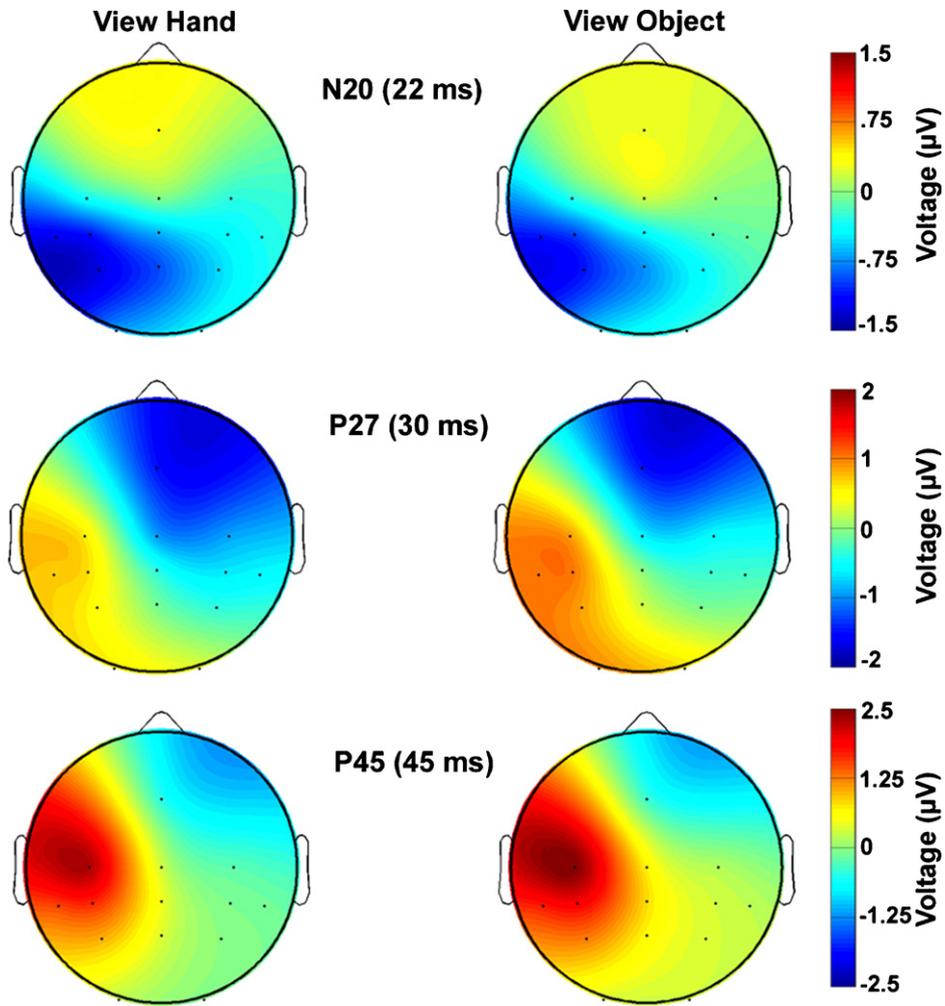


Fig. 1. Topographic maps of EEG activity at the peak of the N20, P27, and P45 components.

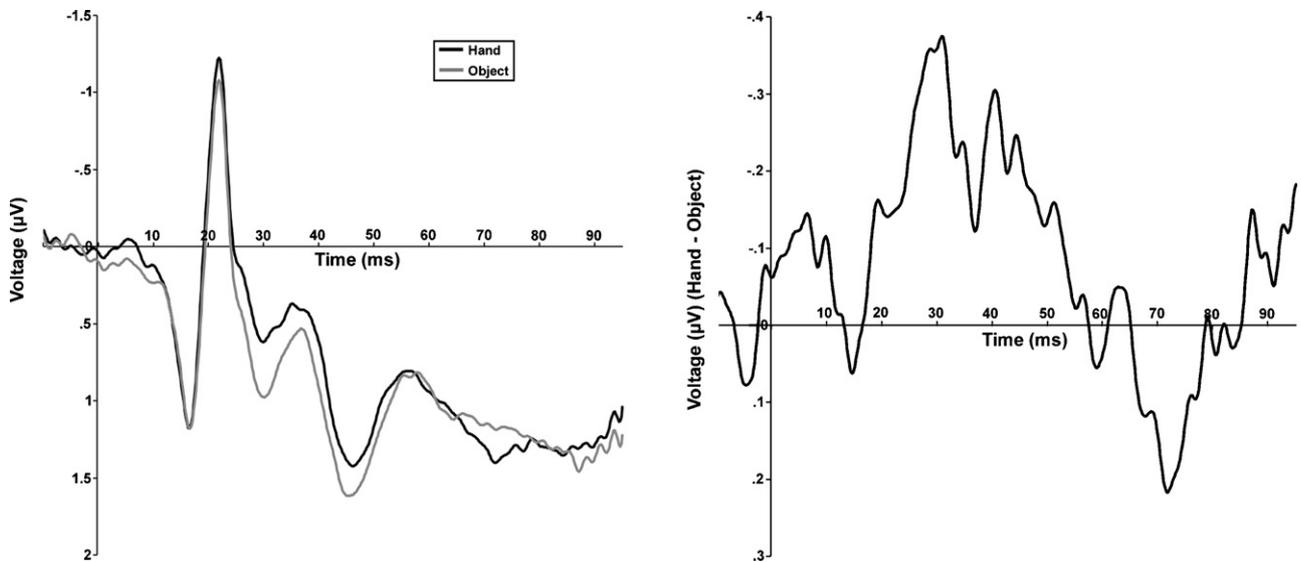


Fig. 2. Left panel: grand mean SEPs in the two visual conditions over contralateral parietal channels (average of CP3, CP5, and P3). Right panel: difference waveform (Hand-Object) showing the time course of differences between visual conditions.

decreases we report. Further, a recent study found that seeing the hand eliminated effects of within-hand spatial attention on SEPs entirely [22]. These findings suggest that it is unlikely that the early effects we observe result from spatial attention.

Similarly, gaze angle differed slightly between conditions. Some studies report effects of gaze on somatosensation [17,27]. Thus, it is possible that eye gaze may contribute to the present effects. Nevertheless, we consider it unlikely that gaze direction plays an important role. First, gaze effects have generally involved a categorical difference in gaze direction, such as looking at one hand vs. the other hand (e.g., [17]), while the present study involved only a slight difference. Second, Forster and Eimer [17] found that while the combined effect of vision and gaze modulated relatively early SEP components (e.g., the P45), effects of gaze alone were restricted to the substantially later N140 component, suggesting that gaze direction alone does not modulate early SEPs.

Recent studies have reported multisensory modulation of early SEPs [8,39]. Bernier et al. [8] found reduction of the P27 when participants manually traced stimuli with mirror-reversed, rather than direct vision. Meehan et al. [39], similarly, reported gating of this component during attention to spatially coincident visual and vibrotactile stimuli. While the exact relation between those studies and the present results is unclear, it is notable that in each case, vision selectively reduces the P27, without corresponding effects on the N20.

Why does seeing the body selectively *reduce* – rather than enhance – P27 amplitude? One possibility is that seeing the body affects somatosensation by modulating inhibitory interneurons in SI [23,33]. For example, seeing the hand reduces the spatial gradient of tactile masking [23], suggesting it shrinks the effective size of tactile receptive fields (RFs). Modulation of RF size depends on intracortical inhibition [5,14], and reduction of RF size could account for heightened tactile acuity from seeing the body [32].

Consistent with this interpretation, several lines of evidence suggest that P27 amplitude is inversely related to SI intracortical inhibition. First, increased P27 amplitude (or decreased P27 suppression from paired stimuli) is found in conditions involving impaired intracortical inhibition such as focal hand dystonia [53,55] and carpal tunnel syndrome [58]. Second, increases of P27 amplitude are found following deafferentation of either adjacent parts of the stimulated limb [56,57] or the contralateral limb [62], which are known to reduce intracortical inhibition [9,65]. Third, increased intracortical inhibition induced by administration of the GABA agonist benzodiazepines reduced P27 amplitude [47,51]. Thus, the reduced P27 amplitude we observed may reflect increased intracortical inhibition in SI induced by viewing the body.

While several studies have found increased tactile acuity when viewing the body [16,32,34,54], we found only a non-significant effect in the same direction. The reason for this difference is unclear. One possibility is stimulating the median nerve, which innervates the index finger, may interfere with touch, or distract participants. Indeed, sensory effects such as paraesthesia commonly last several second after the offset of nerve stimulation and so overlapped touch. Previous studies report substantial subject-to-subject variability in visual modulation of tactile acuity [34]. Thus, visual modulation of SI in this study may be functionally unrelated to behavioural VET effect observed previously.

In conclusion, our results demonstrate that seeing the body modulates short-latency SEPs known to originate in area 3b of SI [1,2,63]. These results confirm and extend previous findings showing effects of seeing the body on somatosensation [33,50,54] by directly linking such effects to the earliest stages of cortical somatosensory processing. The involvement of SI is also consistent with the hypothesis that visual modulations of somatosensation are mediated by inhibitory interneurons in SI [23,33]. More generally, the finding of visual modulation of SI provides further evidence of

the multisensory nature of even early, “sensory-specific” primary cortices [13,21].

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References

- [1] T. Allison, G. McCarthy, C.C. Wood, T.M. Darcey, D.D. Spencer, P.D. Williamson, Human cortical potentials evoked by stimulation of the median nerve. I. Cytoarchitectonic areas generating short-latency activity, *J. Neurophysiol.* 62 (1989) 694–710.
- [2] T. Allison, G. McCarthy, C.C. Wood, S.J. Jones, Potentials evoked in human and monkey cerebral cortex by stimulation of the median nerve, *Brain* 114 (1991) 2465–2503.
- [3] T. Allison, G. McCarthy, C.C. Wood, P.D. Williamson, D.D. Spencer, Human cortical potentials evoked by stimulation of the median nerve. II. Cytoarchitectonic areas generating long-latency activity, *J. Neurophysiol.* 62 (1989) 711–722.
- [4] T. Allison, C.C. Wood, G. McCarthy, D.D. Spencer, Cortical somatosensory evoked potentials. II. Effects of excision of somatosensory or motor cortex in humans and monkeys, *J. Neurophysiol.* 66 (1991) 64–82.
- [5] K.D. Alloway, P. Rosenthal, H. Burton, Quantitative measurements of receptive field changes during antagonism of GABAergic transmission in primary somatosensory cortex of cats, *Exp. Brain Res.* 78 (1989) 514–532.
- [6] C. Barba, M. Frot, M. Guénot, F. Mauguère, Stereotactic recordings of median nerve somatosensory-evoked potentials in the human-supplementary motor area, *Euro. J. Neurosci.* 13 (2001) 347–356.
- [7] C. Barba, M. Frot, F. Mauguère, Early secondary somatosensory area (SII) SEPs. Data from intracerebral recordings in humans, *Clin. Neurophysiol.* 113 (2002) 1778–1786.
- [8] P.-M. Bernier, B. Burle, F. Vidal, T. Hasbroucq, J. Blouin, Direct evidence for cortical suppression of somatosensory afferents during visuomotor adaptation, *Cereb. Cortex* 19 (2009) 2106–2113.
- [9] J.P. Brasil-Neto, J. Valls-Solé, A. Pascual-Leone, A. Cammarota, V.E. Amassian, R. Cracco, M. Hallett, L.G. Cohen, Rapid modulation of human cortical motor outputs following ischemic nerve block, *Brain* 116 (1993) 511–525.
- [10] A. Delorme, S. Makeig, EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis, *J. Neurosci. Meth.* 134 (2004) 9–21.
- [11] J.E. Desmedt, G. Cheron, Somatosensory evoked potentials to finger stimulation in healthy octogenarians and in young adults: wave forms, scalp topography and transit times of parietal and frontal components, *Electroenceph. Clin. Neurophysiol.* 50 (1980) 404–425.
- [12] J.E. Desmedt, N.T. Huy, M. Bourguet, The cognitive P40, N60 and P100 components of somatosensory evoked potentials and the earliest electrical signs of sensory processing in man, *Electroenceph. Clin. Neurophysiol.* 56 (1983) 272–282.
- [13] J. Driver, T. Noesselt, Multisensory interplay reveals crossmodal influences on ‘sensory-specific’ brain regions, neural responses, and judgments, *Neuron* 57 (2008) 11–23.
- [14] R.W. Dykes, P. Landry, R. Metherate, T.P. Hicks, Functional role of GABA in cat primary somatosensory cortex: shaping receptive fields of cortical neurons, *J. Neurophysiol.* 52 (1984) 1066–1093.
- [15] M. Eimer, B. Forster, The spatial distribution of attentional selectivity in touch: evidence from somatosensory ERP components, *Clin. Neurophysiol.* 114 (2003) 1298–1306.
- [16] M. Fiorio, P. Haggard, Viewing the body prepares the brain for touch: effects of TMS over somatosensory cortex, *Euro. J. Neurosci.* 22 (2005) 773–777.
- [17] B. Forster, M. Eimer, Vision and gaze direction modulate tactile processing in somatosensory cortex: evidence from event-related brain potentials, *Exp. Brain Res.* 165 (2005) 8–18.
- [18] M. Frot, F. Mauguère, Timing and spatial distribution of somatosensory responses recorded in the upper bank of the Sylvian fissure (SII area) in humans, *Cereb. Cortex* 9 (1999) 854–863.
- [19] L. Garcia-Larrea, H. Bastuji, F. Mauguère, Mapping study of somatosensory evoked potentials during selective spatial attention, *Electroenceph. Clin. Neurophysiol.* 80 (1991) 201–214.
- [20] L. Garcia-Larrea, M. Frot, M. Valeriani, Brain generators of laser evoked potentials: from dipoles to functional significance, *Neurophysiol. Clin.* 33 (2003) 279–292.
- [21] A.A. Ghazanfar, C.E. Schroeder, Is neocortex essentially multisensory? *Trends Cogn. Sci.* 10 (2006) 278–285.
- [22] H. Gillmeister, C.F. Sambo, B. Forster, Which finger? Early effects of attentional selection within the hand are absent when the hand is viewed, *Eur. J. Neurosci.* 31 (2010) 1874–1881.
- [23] P. Haggard, A. Christakou, A. Serino, Viewing the body modulates tactile receptive fields, *Exp. Brain Res.* 180 (2007) 187–193.

- [24] H. Hämäläinen, J. Kekoni, M. Sams, K. Reinikainen, R. Näätänen, Human somatosensory evoked potential to mechanical pulses and vibration: contribution of SI and SII cortices to P50 and P100 components, *Electroenceph. Clin. Neurophysiol.* 75 (1990) 13–21.
- [25] R. Hari, J. Karhu, M. Hämäläinen, J. Knuutila, O. Salonen, M. Sams, V. Vilkmann, Functional organization of the human first and second somatosensory cortices: a neuromagnetic study, *Eur. J. Neurosci.* 5 (1993) 724–734.
- [26] R. Hari, K. Reinikainen, E. Kaukoranta, M. Hämäläinen, R. Ilmoniemi, A. Penttinen, J. Salminen, D. Teszner, Somatosensory evoked magnetic fields from SI and SII in man, *Electroenceph. Clin. Neurophysiol.* 57 (1984) 254–263.
- [27] J. Honore, M. Bourdeaud'Hui, L. Sparrow, Reduction of cutaneous reaction time by directing the eyes towards the source of stimulation, *Neuropsychologia* 27 (1989) 367–371.
- [28] K. Inui, X. Wang, Y. Tamura, Y. Kaneoke, R. Kakigi, Serial processing in the human somatosensory system, *Cereb. Cortex* 14 (2004) 851–857.
- [29] Y. Iwamura, Hierarchical somatosensory processing, *Curr. Opin. Neurobiol.* 8 (1998) 522–528.
- [30] R. Kakigi, Somatosensory evoked magnetic fields following median nerve stimulation, *Neurosci. Res.* 20 (1994) 165–174.
- [31] J. Karhu, C.D. Tesche, Simultaneous early processing of sensory input in human primary (SI) and secondary (SII) somatosensory cortices, *J. Neurophysiol.* 81 (1999) 2017–2025.
- [32] S. Kennett, M. Taylor-Clarke, P. Haggard, Noninformative vision improves the spatial resolution of touch in humans, *Curr. Biol.* 11 (2001) 1188–1191.
- [33] M.R. Longo, V. Betti, S.M. Aglioti, P. Haggard, Visually induced analgesia: seeing the body reduces pain, *J. Neurosci.* 29 (2009) 12125–12130.
- [34] M.R. Longo, S. Cardozo, P. Haggard, Visual enhancement of touch and the bodily self, *Conscious. Cogn.* 17 (2008) 1181–1191.
- [35] E. Macaluso, C.D. Frith, J. Driver, Selective spatial attention in vision and touch: unimodal and multimodal mechanisms revealed by PET, *J. Neurophysiol.* 83 (2000) 3062–3075.
- [36] F. Mauguière, I. Merlet, N. Forss, S. Vanni, V. Jousmäki, P. Adeleine, R. Hari, Activation of a distributed somatosensory cortical network in the human brain. A dipole modelling study of magnetic fields evoked by median nerve stimulation. Part I. Location and activation timing of SEF sources, *Electroenceph. Clin. Neurophysiol.* 104 (1997) 281–289.
- [37] F. Mauguière, J.E. Desmedt, J. Courjon, Astereognosis and dissociated loss of frontal or parietal components of somatosensory evoked potentials in hemispheric lesions, *Brain* 106 (1983) 271–311.
- [38] G. McCarthy, C.C. Wood, T. Allison, Cortical somatosensory evoked potentials I. Recordings in the monkey *Macaca fascicularis*, *J. Neurophysiol.* 66 (1991) 53–63.
- [39] S.K. Meehan, W. Legon, W.R. Staines, Spatiotemporal properties modulate intermodal influences on early somatosensory processing during sensory-guided movement, *Clin. Neurophysiol.* 120 (2009) 1371–1380.
- [40] G.L. Moseley, K. Wiech, The effect of tactile discrimination training is enhanced when patients watch the reflected image of their unaffected limb during training, *Pain* 144 (2009) 314–319.
- [41] S. Ohara, N.E. Crone, N. Weiss, R.D. Treede, F.A. Lenz, Cutaneous painful laser stimuli evoke responses recorded directly from primary somatosensory cortex in awake humans, *J. Neurophysiol.* 91 (2004) 2734–2746.
- [42] R.C. Oldfield, The assessment and analysis of handedness: the Edinburgh inventory, *Neuropsychologia* 9 (1971) 97–113.
- [43] D. Papakostopoulos, H.J. Crow, The precentral somatosensory evoked potential, *Ann. N.Y. Acad. Sci.* 425 (1984) 256–261.
- [44] M. Ploner, F. Schmitz, H.-J. Freund, A. Schnitzler, Parallel activation of primary and secondary somatosensory cortices in human pain processing, *J. Neurophysiol.* 81 (1999) 3100–3104.
- [45] T.P. Pons, P.E. Garraghty, D.P. Friedman, M. Mishkin, Physiological evidence for serial processing in somatosensory cortex, *Science* 237 (1987) 417–420.
- [46] T. Raij, J. Karhu, D. Kicic, P. Lioumis, P. Julkunen, F.H. Lin, J. Ahveninen, R.J. Ilmoniemi, M. Hämäläinen, B.R. Rosen, J.W. Belliveau, Parallel input makes the brain run faster, *NeuroImage* 40 (2008) 1792–1797.
- [47] D. Restuccia, M. Valeriani, E. Grassi, S. Mazza, P. Tonali, Dissociated changes of somatosensory evoked low-frequency scalp responses and 600 Hz bursts after single-dose administration of lorazepam, *Brain Res.* 946 (2002) 1–11.
- [48] T. Ro, R. Wallace, J. Hagedorn, A. Farnè, E. Pienkos, Visual enhancing of tactile perception in the posterior parietal cortex, *J. Cogn. Neurosci.* 16 (2004) 24–30.
- [49] P.M. Rossini, R. Traversa, P. Boccasani, G. Martino, F. Passarelli, L. Pacifici, G. Bernardi, P. Stanzione, Parkinson's disease and somatosensory evoked potentials: apomorphin-induced transient potentiation of frontal components, *Neurology* 43 (1993) 2495–2500.
- [50] C.F. Sambo, H. Gillmeister, B. Forster, Viewing the body modulates neural mechanisms underlying sustained spatial attention in touch, *Eur. J. Neurosci.* 30 (2009) 143–150.
- [51] T.B. Sloan, M.L. Fugina, J.R. Toleikis, Effects of midazolam on median nerve somatosensory evoked potentials, *Br J. Aesthet.* 64 (1990) 590–593.
- [52] R. Stilla, G. Deshpande, S. LaConte, X. Hu, K. Sathian, Posteromedial parietal cortical activity and inputs predict tactile spatial acuity, *J. Neurosci.* 27 (2007) 11091–11102.
- [53] Y. Tamura, M. Matsushashi, P. Lin, B. Ou, S. Vorbach, R. Kakigi, M. Hallett, Impaired intracortical inhibition in the primary somatosensory cortex in focal hand dystonia, *Move. Disord.* 23 (2007) 558–565.
- [54] M. Taylor-Clarke, S. Kennett, P. Haggard, Vision modulates somatosensory cortical processing, *Curr. Biol.* 12 (2002) 233–236.
- [55] M. Tinazzi, A. Priori, L. Bertolasi, E. Frassin, F. Mauguière, A. Fiaschi, Abnormal central integration of a dual somatosensory input in dystonia: evidence for sensory overflow, *Brain* 123 (2000) 42–50.
- [56] M. Tinazzi, T. Rosso, G. Zanette, A. Fiaschi, S.M. Aglioti, Rapid modulation of cortical proprioceptive activity induced by transient cutaneous deafferentation: neurophysiological evidence of short-term plasticity across different somatosensory modalities in humans, *Eur. J. Neurosci.* 18 (2003) 3053–3060.
- [57] M. Tinazzi, G. Zanette, A. Polo, D. Volpato, P. Manganotti, C. Bonato, R. Testoni, A. Fiaschi, Transient deafferentation in humans induces rapid modulation of primary sensory cortex not associated with subcortical changes: a somatosensory evoked potential study, *Neurosci. Lett.* 223 (1997) 21–24.
- [58] M. Tinazzi, G. Zanette, D. Volpato, R. Testoni, C. Bonato, P. Manganotti, C. Miniussi, A. Fiaschi, Neurophysiological evidence of neuroplasticity at multiple levels of the somatosensory system in patients with carpal tunnel syndrome, *Brain* 121 (1998) 1785–1794.
- [59] S.P. Tipper, D. Lloyd, B. Shorland, C. Dancer, L.A. Howard, F. McGlone, Vision influences tactile perception without proprioceptive orienting, *NeuroReport* 9 (1998) 1741–1744.
- [60] R.W. Van Boven, K.O. Johnson, The limit of tactile spatial resolution in humans: grating orientation discrimination at the lip, tongue, and finger, *Neurology* 44 (1994) 2361–2366.
- [61] T.D. Waberski, H. Buchner, M. Perkhun, R. Gobbelé, M. Wagner, W. Kücker, J. Silny, N30 and the effect of explorative finger movements: a model of the contribution of the motor cortex to early somatosensory evoked potentials, *Clin. Neurophysiol.* 110 (1999) 1589–1600.
- [62] K.J. Wehrhahn, J. Mortensen, R.W. Van Boven, K.E. Zeuner, L.G. Cohen, Enhanced tactile spatial acuity and cortical processing during acute hand deafferentation, *Nature Neurosci.* 5 (2002) 936–938.
- [63] C.C. Wood, D. Cohen, B.N. Cuffin, M. Yarita, T. Allison, Electrical sources in human somatosensory cortex: identification by combined magnetic and potential recordings, *Science* 227 (1985) 1051–1053.
- [64] Q.H. Zhang, M.K. Zacharian, G.T. Coleman, M.J. Rowe, Hierarchical equivalence of somatosensory areas I and II for tactile processing in the cerebral cortex of the marmoset monkey, *J. Neurophysiol.* 85 (2001) 1823–1835.
- [65] U. Ziemann, M. Hallett, L.G. Cohen, Mechanisms of deafferentation-induced plasticity in human motor cortex, *J. Neurosci.* 18 (1998) 7000–7007.