



What makes somatosensory short-term memory maintenance effective? An EEG study comparing contralateral delay activity between sighted participants and participants who are blind

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ARTICLE INFO

Keywords:

Contralateral delay activity
Dorsolateral prefrontal cortex
Electroencephalogram
Sensory memory
Somatosensory cortex
Somatosensory memory

ABSTRACT

Somatosensory short-term memory is essential for object recognition, sensorimotor learning, and, especially, Braille reading for people who are blind. This study examined how visual sensory deprivation and a compensatory focus on somatosensory information influences memory processes in this domain. We measured slow cortical negativity developing during short-term tactile memory maintenance (tactile contralateral delay activity, tCDA) in frontal and somatosensory areas while a sample of 24 sighted participants and 22 participants who are blind completed a tactile change-detection task where varying loads of Braille pin patterns served as stimuli. Auditory cues, appearing at varying latencies between sample arrays, could be used to reduce memory demands during maintenance. Participants who are blind (trained Braille readers) outperformed sighted participants behaviorally. In addition, while task-related frontal activation featured in both groups, participants who are blind uniquely showed higher tCDA amplitudes specifically over somatosensory areas. The site specificity of this component's functional relevance in short-term memory maintenance was further supported by somatosensory tCDA amplitudes first correlating across the whole sample with behavioral performance, and secondly showing sensitivity to varying memory load. The results substantiate sensory recruitment models and provide new insights into the effects of visual sensory deprivation on tactile processing. Between-group differences in the interplay between frontal and somatosensory areas during somatosensory maintenance also suggest that efficient maintenance of complex tactile stimuli in short-term memory is primarily facilitated by lateralized activity in somatosensory cortex.

1. Introduction

Humans are constantly confronted with considerable sensory input. Maintaining and processing such information is indispensable for mastering everyday activities. Furthermore, bridging temporally separated sensory information is vital to fully perceive, understand, and interact with our environment (Goldman-Rakic, 1992). Research has accordingly focused on the maintenance of sensory information in short-term memory (STM), given its fundamental importance to complex cognition such as decision making and goal-directed behavior (D'Esposito, 2007;

Goldman-Rakic, 1992; Shah and Miyake, 1999). STM maintenance is generally considered a process which allows higher-order manipulations such as comparisons between sensations across time (working memory, Aben et al., 2013; Atkinson and Shiffrin, 1968; Cowan, 2008b) and extends beyond ultra-short-term unconscious sensory traces in modality-specific brain areas (Atkinson and Shiffrin, 1968; Gallace et al., 2008; Sperling, 1967). While STM capacity is limited to a few items, sensory memory can hold more (unconscious) information, with its capacity and maintenance differing across modalities (Cowan, 2008a).

In addition to visual and auditory inputs, STM maintenance of tactile information is essential for all humans, not only in terms of ob-

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<https://doi.org/10.1016/j.neuroimage.2022.119407>.

Received 2 March 2022; Received in revised form 27 May 2022; Accepted 21 June 2022

Available online 22 June 2022.

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ject recognition (Gallace and Spence, 2009; Rincon-Gonzalez et al., 2011), but also for motor learning; sensory and proprioceptive feedback during movement guides adaptational processes that minimize discrepancies between intended and actual motion paths (Krakauer et al., 2019; Rossi et al., 2021). In addition, tactile STM forms the basis for Braille reading. However, compared to visual and auditory memory, somatosensory STM receives less research focus (Gallace and Spence, 2009). A better understanding of both its characteristics and underlying neurophysiological processes would greatly assist with the development of new concepts for teaching Braille reading to people who are blind (Mašić et al., 2020).

Sensory recruitment models of general STM propose that sensory information is maintained through the activation and interplay between modality-specific brain regions recruited during initial sensory processing, and supramodal brain areas corresponding to higher-order processing (Jonides et al., 2008; Linden, 2007; Postle, 2006; Reuter-Lorenz and Jonides, 2007; Ruchkin et al., 2003; D'Esposito and Postle, 2015). The prefrontal cortex (PFC) in particular plays an important top-down (supramodal) role, by recruiting task-relevant neuronal networks, monitoring attention, and representing task goals (Chai et al., 2018; D'Esposito and Postle, 2015; Fuster, 2015; Lenk et al., 2014; Postle, 2006). Concerning the somatosensory modality in particular, non-human primate studies show activation in the (dorsolateral) PFC and somatosensory areas during tactile STM tasks (Pasternak and Greenlee, 2005; Resch et al., 1992; Romo and Salinas, 2003; Wang et al., 2015; Zhou and Fuster, 1996). Studies with humans have revealed similar activation patterns (Bender et al., 2007; Burton and Sinclair, 2000; Harris et al., 2002; Katus et al., 2015; Ohara et al., 2008; Savini et al., 2012; Spitzer and Blankenburg, 2011; Staines et al., 2002; Zhao et al., 2018). However, a number of key characteristics of somatosensory memory remain unclear, in particular the duration of tactile memory traces in sensory (haptic) memory and also STM capacity. Also, to date, no study has demonstrated a functional relevance to the sensory recruitment model with specific reference to somatosensory STM.

An exciting advancement in human neurophysiological research in STM involves contralateral delay activity (CDA). CDA is a slow-moving waveform recordable with non-invasive electroencephalography (EEG), which classically scales in amplitude with memory load during the maintenance period of visual change-detection tasks (McCollough et al., 2007; Vogel and Machizawa, 2004). More recently, Katus et al. (2015) report late contralateral negativity at lateral central electrodes during a tactile change-detection task. This so-called 'tactile CDA' (tCDA) also seems to be, within limits, sensitive to varying memory load (Katus and Eimer, 2018; Katus et al., 2015). Most researchers interpret (t)CDA as the shift of memory relevant attention (Berggren and Eimer, 2016; Hecht et al., 2016; Katus and Eimer, 2015, 2018; Lewis-Peacock et al., 2012).

For the present study, we accordingly designed a tactile change-detection paradigm to help address the above outstanding issues. In addition to memory arrays varying in terms of a wider range of load (two, four or six items), we compared the effects of a cue, which narrowed down the possible locations of impending change. The cue appeared either before, or at various stages after the memory array (S1), and always before the appearance of the target array (S2). The various latencies of cue onset allow us to identify how long after S1 cueing remains effective, and thus, for how long traces are likely held in haptic memory.

We present behavioral and EEG data recorded while both regular-sighted and human participants with blindness performed our paradigm. Due to their intense use of the tactile sense (e.g. for navigation and object recognition) and their experience in Braille reading, people who are blind have extraordinary expertise in somatosensory information processing (Fine and Park, 2018). Proficient Braille readers are especially intensively trained in perceiving, processing, and memorizing small-sized haptic stimuli at their fingertips. Previous work in the context of blindness-driven cortical plasticity demonstrates expanded neuronal representation of fingers used for Braille reading (Pascual-Leone et al.,

Table 1

Causes for blindness in $n = 22$ participants.

Causes for blindness	<i>n</i>	Age of onset
Retinopathy of prematurity	8	six at birth; 1.5; 13 years
Tumor disease	3	1; 3; 18 years
Genetic diseases (non-defined)	2	both at birth
Unknown	3	two at birth; 4 years
Optic nerve atrophy	2	1; 22 years
Macular degeneration	2	at birth; 12 years
Retinopathy (adolescent)	1	12 years
Incontinentia pigmenti	1	6 years

1993; Pascual-Leone and Torres, 1993; Sadato et al., 1998), a stronger functional connection between different regions in the somatosensory cortex (Heine et al., 2015), and the recruitment of occipital areas for the processing of nonvisual input (e.g., Fine and Park, 2018). Additional evidence supports the hypothesis of cross-modal compensation; individuals who are blind show enhanced sensory and cognitive function (Fine and Park, 2018; Kupers and Ptito, 2014; Withagen et al., 2013), and, in particular, hyperacuity in the senses of hearing and touch (Arnaud et al., 2018; Boven et al., 2000; Kauffman et al., 2002; Kupers and Ptito, 2014; Renier et al., 2014). We therefore expected participants who are blind to significantly outperform sighted controls behaviorally in our paradigm. The present study will therefore detail the neurophysiological underpinnings of this superior performance, adding to the compensation literature, while also using this de facto expert-vs-novice contrast across our wider analyses to ascribe functional relevance to neurophysiological findings related to somatosensory STM and the tCDA.

2. Materials and methods

2.1. Participants

We initially recruited 28 participants who are blind and 28 participants with normal or corrected-to normal vision. We approached various specialized social societies to contact and recruit people who are blind. For the latter sample we used advertisements on information boards and on the webpage of the University of Dresden. We excluded four sighted participants and three participants with blindness from analysis due to technical problems during EEG recording. We excluded three more participants who are blind during pre-processing. Two participants had substantial artefacts in their EEG data, such that removing artefact-laden epochs would have left no trials for further analyses. We removed the third participant with blindness due to performance below chance level. The final sample thus contained 22 participants who are blind (11 female, 11 male, mean age 35.7 ± 9.0 , range 23–57 years) and 24 sighted participants (15 female, 9 male, mean age 29.8 ± 10.3 , range 18–50 years). There were no significant differences between the two groups regarding age ($t(44) = -2.10, p = .08$) and sex ($t(44) = 0.84, p = .40$). None of the participants were diagnosed with a neurological or psychiatric disorder, and no participants were taking psychoactive medication at time of testing. Aside from one ambidextrous participant in the group of participants with blindness, all participants were right-handed as assessed by the Edinburgh Handedness Inventory (EHI, Oldfield, 1971). For assessing handedness in participants who are blind, we used an adjusted EHI, modifying three unsuitable items (e.g., 'reading a text in Braille' instead of 'writing'). As most people who are blind typically learn to read Braille with both hands, the cut-off score for right-handedness was consequently lowered from 0.4 to 0.35. One such person did not complete the EHI, but reported during anamnesis as being right-handed. Half of the participants with blindness were blind at birth; the other half had become blind later in life. All of them were blind for at least 16 years. Blindness etiologies are summarized in Table 1. Participants with blindness were either totally blind or visually strongly impaired, satisfying criteria for the category four (which permits rudimentary discernment

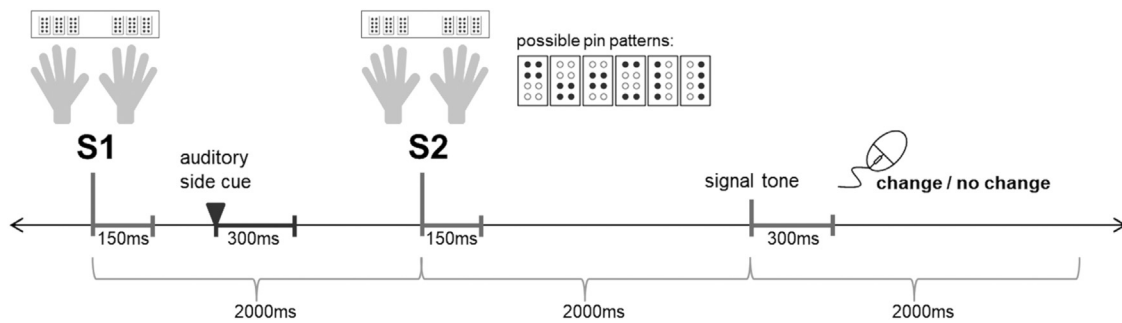


Fig. 1. Schematic illustration of one trial in the change-detection task with auditory cue. S1 = first stimulus, S2 = second stimulus.

of different levels of brightness) and five of the classification of blindness provided in ICD-10 (ICD-10-CM 2022, 2021). All participants met the inclusion criteria of at least one year of experience in Braille reading (mean 27.6 ± 8.7 years). None of the sighted participants had experience with Braille reading. In the supplementary material, we report additional analyses comparing participants who are congenitally blind with those who lost their sight later in life, and correlational analyses assessing influencing factors on performance rates in the group of participants with blindness. Results suggest that blindness onset was not a confounding factor in this sample. All participants gave informed consent and received €30 monetary compensation for their participation (plus reimbursement of travel expenses, if necessary). The study was approved by the ethics committee at the Technische Universität Dresden, and conformed to the Declaration of Helsinki.

2.2. Experimental protocol

Participants performed a change-detection task based on established paradigms for the visual system (e.g., Vogel and Machizawa, 2004), adapted for tactile stimuli. With their index, middle, and ring fingers of both hands placed on a Braille-reading device (InfoDot 40, Flusoft), participants were presented with a tactile stimulus (S1) for 150ms. Six distinguishable pin patterns were used as stimuli (Fig. 1). Stimuli did not represent Braille letters, but arbitrary patterns of four pins. After a retention interval of two seconds following S1 onset, a second stimulus (S2) was presented for 150ms. Two seconds after S2 onset, an auditory tone prompted participants to state via mouse click whether they perceived S1 and S2 to be the same, or whether they recognized a change. Changes occurred on 50% of trials, and always occurred only at a single finger. The nature of the pin combinations ensured that each change involved a minimum of two pins. Deferring the motor response avoided preparatory motor activity overlapping with somatosensory memory maintenance. Both the order of the hand experiencing a change and the combination of presented pin patterns were pseudo-randomized. A schematic illustration of one trial of the change-detection task is shown in Fig. 1.

We manipulated additional task parameters to address our core hypotheses. The first parameter related to an auditory cue, providing information about the hand at which change would occur ('left' or 'right'). Such a cue reduces memory load by half, if used effectively. To further assess whether the cue was most effective at different stages of trials, we altered the latency of its appearance (1500ms prior to S1, 150ms, 300ms, 500ms, 800ms, 1200ms after S1). A second parameter related to memory load, i.e., the number of presented pin patterns. This varied between two (one at each index finger), four (one at each index and middle finger), and six items (one at each index, middle, and ring finger).

The whole task consisted of 12 blocks with 40 trials each. One block, serving as a baseline condition without memory demands, was not included in the final analysis. Design for the remaining 11 blocks ascribed specific parameters to trials of a given block (blocked conditions). Five blocks respectively probed five levels of cue-latency effects (1500ms

prior to S1, 300ms, 500ms, 800ms, 1200ms after S1) and used a constant memory load of four items (i.e., four pin patterns) presented at S1. The other six blocks probed the interaction between memory load (two, four or six items presented at S1) and discrete cue effects (150ms post S1, or none). The inter-trial interval was pseudo-randomized trial-by-trial, and ranged from six to ten seconds. Due to the high number of blocked conditions, a completely counterbalanced block order was not possible across our participant sample. Nonetheless, every block condition appeared at each possible order position for at least two (and maximum six) times.

All participants completed two training sessions, one on the day of data acquisition, and another at an appointment beforehand. Training contained eight practice blocks with 12 trials each. A block was repeated when a performance rate above chance level was not achieved. No participants were excluded due to insufficient performance in the practice trials. All participants were instructed to keep their eyes closed during the whole task.

2.3. EEG recording and pre-processing

While participants performed the tactile change-detection task, we collected simultaneous EEG data at a sampling rate of 5000Hz with an online band-pass filter (DC as low cut-off and 1000Hz as high cut-off; BrainAmpDC amplifier; Brain Products GmbH, Gilching, Germany). We used a 64-channel electrode cap (equidistant layout, Easycap GmbH, Herrsching, Germany) with sintered Ag/AgCl electrodes. An electrode near Fpz served as the online reference, before transformation to an average reference during offline pre-processing. Three additional horizontal and vertical electrooculogram electrodes were applied; one lateral to the right eye and one above and one below the left eye (1cm distance). We kept impedances $< 10k\Omega$. We conducted offline pre-processing using BrainVision Analyzer 2.1 software (Brain Products GmbH, Gilching, Germany). We initially segmented the continuous data into epochs spanning 9500ms, starting 2000ms prior to S1, and created separate sets for correct and incorrect responses as well as for the auditory cues (left/right). We included only trials containing a response within 2000ms of the response-prompt signal tone for further analysis. Eye movement artefacts were removed by calculating the propagation factors for blinks and eye movements (Gratton et al., 1983). We removed DC trends by subtracting linear estimates fitted to intervals of 500ms at the beginning and end of the segments. By comparing averages with and without DC-detrend correction a systematic bias caused by the correction process was ruled out. We next used an automatic artefact rejection procedure to exclude segments with amplitudes $> 100\mu V$, and then low-pass filtered (30 Hz) remaining segments using a Butterworth filter (slope 8dB/octave). The time interval of 500ms prior to S1 served as baseline. We computed participant-level averages across all correct trials. We then calculated CDA following the rationale of the lateralized readiness potential (Coles, 1989): $CDA = [\text{Mean}(el2-el1)_{\text{left}} + \text{Mean}(el1-el2)_{\text{right}}]/2$, where el1 and el2 respectively correspond to the left and right lateralized electrode of a region-specific pair (see specific electrodes in Data

analysis section) and subscripts 'left' and 'right' respectively correspond to trials cued to a change at that hand, i.e., subtracting ipsilateral from contralateral activity, relative to hand. The average signal-to-noise ratio of all correct trials at the relevant electrodes was 45.5.

2.4. Data analysis

We selected a time window spanning from 700 to 900ms relative to cue onset to calculate the amplitude of tCDA. This window allowed time to process the auditory stimulus and focus attention on the respective hand, i.e. capturing activity during the plateau after the tCDA's initial rise. In the condition in which the cue was presented 1500ms prior to S1, we used the time window 700-900ms after S1. We used a shorter time interval than previous work (e.g., Vogel and Machizawa, 2004; Katus and Eimer, 2018; Katus et al., 2015) in order to obtain better temporal resolution and to avoid strong overlaps of the analyzed time windows across experimental conditions. Our selection of this specific time window of tCDA was guided by previous work on the sensory post-processing N700 component, which might be related to CDA and other memory-maintenance-related slow waves (Bender et al., 2007; Bender et al., 2010), and by work on early CNV (Bender et al., 2004).

On the basis of former research (Postle, 2006; Ruchkin et al., 2003) we investigated neural activity in the frontal and the somatosensory cortex, respectively representing the supramodal 'monitoring' area and the modality-specific region involved in somatosensory memory maintenance. Based on existing anatomical knowledge and in line with previous research, we selected pairs F5/F6 as electrode sites overlaying the dorsolateral prefrontal cortex (DLPFC; Gupta and Tranel, 2012; Kaiser, 2010) and pairs CP3/CP4 to capture cortical activity in the primary and secondary somatosensory cortex (SI and SII; Holmes and Tamè, 2019; Kaiser, 2010; Martuzzi et al., 2014). We a priori performed a data-driven principal component analysis (PCA) which confirmed the selected areas of interest (Kayser and Tenke, 2006; Pourtois et al., 2008). These target electrode sites also provided separation between frontal and somatosensory activity.

To conduct source analysis, we used low-resolution electromagnetic tomography (LORETA in BrainVision Analyzer 2.1), which provides current density estimates for 2349 voxels spanning the gray matter and hippocampus (Pascual-Marqui et al., 1999). We used LORETA to analyze the cortical distribution of tCDA in the respective time windows described above.

To test the hypothesis that tCDA reflects access of somatosensory representations, we tested whether the onset of site-specific tCDA varied in latency in accordance with cue latency. We used the inflection point of tCDA waves at CP3/CP4 and F5/F6 as a marker for when the gradient in the somatosensory and DLPFC sites were respectively maximal. This inflection point latency was automatically determined as the minimum of the second derivative during a 1000ms interval which extended from 50ms prior to the auditory cue to 950ms afterwards. We computed these gradients for both sites on participant-averaged waveforms, separately for each cue latency. For a reliable automatic inflection point detection the data was filtered with a high cut-off filter of 1Hz. All relevant parameters were exported to SPSS for further analysis.

2.5. Statistics

Statistical analyses were performed using the IBM SPSS Statistics 27 software (IBM Corp., Armonk, NY, USA; Version 27). The p -value for significance was set at .05. If not stated otherwise, the α -level was adjusted for all performed post-hoc t -tests using the Bonferroni-Holm correction for multiple comparisons. One-sided p -values are reported for post-hoc t -tests assessing directed hypotheses. We applied Greenhouse-Geisser correction when ANOVAs contained factors with more than two levels and sphericity was not met. The ANOVAs contained all possible interaction terms.

- 1) *Task performance (% correct) – Cue effects.* In order to examine the effects of participant group and cue latency on performance rate, we conducted a two-way mixed ANOVA with between-subjects factor GROUP (blind, sighted) and within-subject factor CUE LATENCY (seven conditions containing four items: no cue, cue 1500ms prior to S1, cue 150ms, 300ms, 500ms, 800ms, and 1200ms after S1). *Task performance (% correct) – Load effects.* In order to examine the effect of participant group, memory load and cue presence on performance rate we conducted a three-way mixed ANOVA, with between-subjects factor GROUP (blind, sighted) and within-subjects factors LOAD (two, four, and six items) and CUE (none, cued).
- 2) *Time course of tCDA – cue effects.* We next examined whether tCDA onsets in temporal accordance with the appearance of a cue detailing the upcoming location of task-relevant change, and whether this was localized to frontal or somatosensory sites. For this, we conducted a three-way mixed ANOVA on tCDA inflection point latencies, as a function of between-subjects factor GROUP (blind, sighted) and within-subjects factors CUE LATENCY (six conditions with four presented items and cues: 150ms, 300ms, 500ms, 800ms, and 1200ms after S1), and ELECTRODE (F5/F6, CP3/CP4). *Load effects.* To analyze whether the varying task difficulty was reflected in site-specific alterations in the amplitude of tCDA, and whether this was in turn modulated by blindness and tactile proficiency, we performed a three-way mixed ANOVA on the amplitude of tCDA as a function of between-subjects factor GROUP (blind, sighted) and within-subjects factors LOAD (two, four, and six items), and ELECTRODE (F5/F6, CP3/CP4). We restricted this analysis to cued trials. *Topographic comparison across cue conditions.* To analyze whether site-specific tCDA amplitude differed between sighted participants and participants with blindness, as a function of the latency of the cue, we performed a three-way mixed ANOVA with between-subjects factor GROUP (blind, sighted) and within-subjects factors ELECTRODE (F5/F6, CP3/CP4) and CUE LATENCY (1500ms prior to S1, 150ms, 300ms, 500ms, 800ms, and 1200ms after S1).
- 3) *Correlation between performance rates and tCDA.* We used Spearman's correlation coefficient rho to assess the general relationship between performance rates and tCDA measured at frontal and centroparietal electrodes (tCDA_F, tCDA_{CP}). In order to contrast 'load' and 'latency' conditions, correlation coefficients for each site were calculated separately for the blocks with four presented items and varying cue latencies (trials with cues 1500ms before S1, 150ms, 300ms, 500ms, 800ms, and 1200ms after S1), and the cued blocks with varying memory load (two, four, and six items). Four correlation analyses were performed in total: tCDA_F with performance in latency conditions, tCDA_F with performance in load conditions, tCDA_{CP} with performance in latency conditions, and tCDA_{CP} with performance in load conditions. In each of the four correlations, we regressed the n -element vector of individual subject means of tCDA amplitude for those given conditions, with the n -element vector of each subject's overall performance rate for those conditions, where n is the total number of subjects in the experiment, i.e., participants with and without blindness.

3. Results

3.1. Task performance

3.1.1. Cue effects in participants with and without blindness

A two-way mixed ANOVA revealed main effects for the factors GROUP ($F(1,44) = 10.35$; $p = .002$, $\eta_p^2 = .19$) and CUE LATENCY ($F(6,264) = 3.59$, $p = .002$, $\eta_p^2 = .08$). Participants who are blind performed significantly better than sighted controls ($t(44) = 3.22$, $p = .002$, $d = 0.95$; Fig. 2). For both groups, performance rates increased significantly when cues appeared 1500ms before, and 150ms and 800ms post

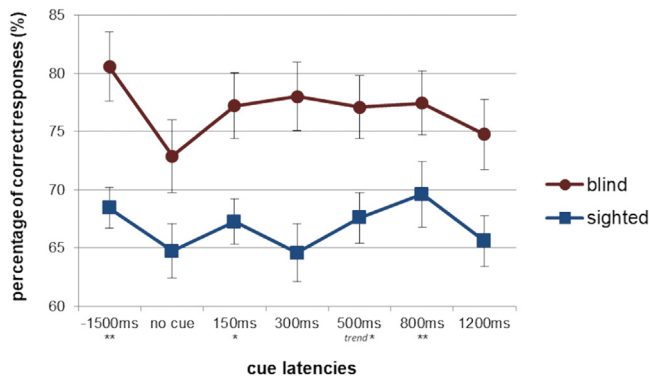


Fig. 2. Percentage of correct responses reported by sighted participants and participants who are blind for all cue latencies. Error bars indicate the 95% confidence interval. Asterisks below x-axis reflect post-hoc comparisons (collapsed across groups) with the no-cue condition. Participants who are blind $n = 22$, sighted participants $n = 24$, $*p < .05$, $**p < .01$.

Table 2

Post-hoc analyses of main effect of cue latency on task performance. $N = 46$, $*p \leq .05$, $**p < .01$.

	df	t	p	d
no cue vs -1500ms	45	-3.77	.001**	-0.55
no cue vs 150ms	45	-2.32	.050*	-0.34
no cue vs 300ms	45	-1.63	.11	-0.24
no cue vs 500ms	45	-2.16	.054	-0.32
no cue vs 800ms	45	-3.40	.004**	-0.50
no cue vs 1200ms	45	-1.05	.15	-0.15

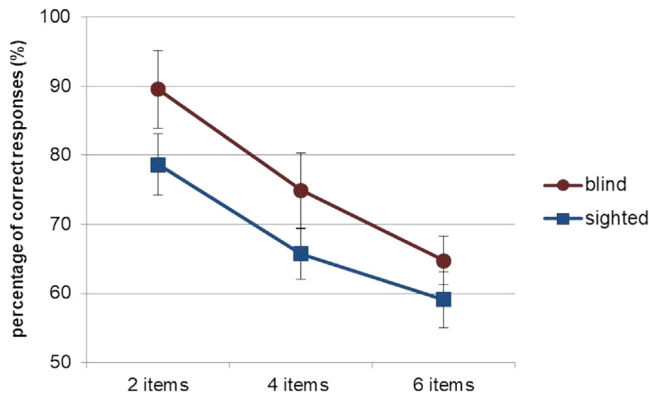


Fig. 3. Percentage of correct responses reported by sighted participants and participants who are blind for all load conditions (cued and uncued trials averaged). Error bars indicate the 95% confidence interval. Blind $n = 22$, sighted $n = 24$.

onset of S1, relative to no cue (Fig. 2; Table 2). For the 500ms condition, after Bonferroni-Holm correction for six comparisons, a trend in the same direction was still found ($p = .054$). For cues 300ms after S1, no overall trend emerged. Also, long-latency cues (1200ms) were not used effectively by the participants. There was no significant interaction between GROUP and CUE LATENCY ($F(6,264) = 1.01$, $p = .42$). Descriptive statistics can be found in supplementary Table 1.

3.1.2. Load effects

A three-way mixed ANOVA showed main effects of LOAD ($F(1.68, 74.06) = 186.90$, $p < .001$, $\eta_p^2 = .81$) and GROUP ($F(1,44) = 7.59$, $p = .008$, $\eta_p^2 = .15$; Fig. 3) on task performance. Post-hoc analyses respectively revealed that performance worsened with increased memory load (2 items – 4 items: $t(45) = 13.65$, $p < .001$, $d = 2.01$; 4 items – 6 items: $t(45) = 7.86$, $p < .001$, $d = 1.16$; 2 items – 6 items: $t(45) = 15.50$,

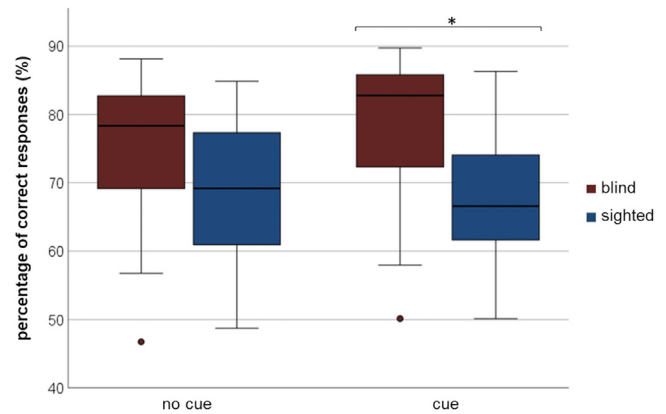


Fig. 4. Percentage of correct responses reported by sighted participants and participants who are blind with and without a cue. Each boxplot reflects the average scores across all load conditions (two, four, and six items). Blind $n = 22$, sighted $n = 24$, $*p = .002$.

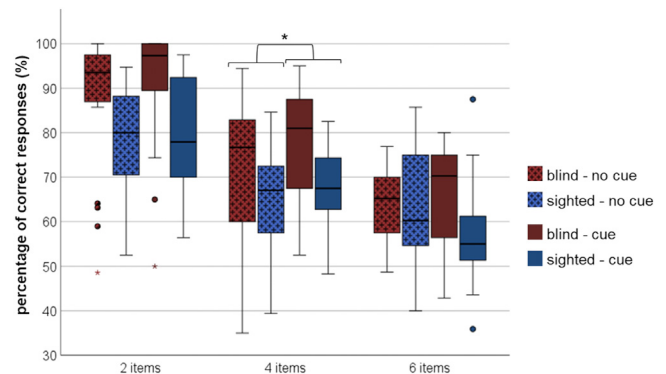


Fig. 5. Percentage of correct responses for each load condition reported by sighted participants and participants who are blind with and without cues. Blind $n = 22$, sighted $n = 24$, $*p = .038$.

$p < .001$, $d = 2.29$) and that participants who are blind were superior to sighted participants ($t(44) = 2.76$, $p = .008$, $d = 0.81$; Fig. 3). This ANOVA also returned a significant interaction between CUE and GROUP ($F(1,340) = 7.25$, $p = .01$, $\eta_p^2 = .14$). Post-hoc analyses suggest this interaction was driven by participants with blindness benefitting more from cues than sighted participants (blind – sighted, with cue: $t(44) = 3.48$, $p = .002$, $d = 1.02$; blind – sighted, without cue: $t(44) = 1.89$, $p = .07$, $d = 0.56$; Fig. 4).

This ANOVA also returned a significant interaction between CUE and LOAD ($F(2,88) = 3.38$, $p = .04$, $\eta_p^2 = .07$), suggesting cues had a different impact on performance rates depending on the amount of presented items. Post-hoc analyses reveal performance increased in trials with four items when a cue was present ($t(45) = 2.32$, $p = .04$, $d = 0.34$; Fig. 5), which was not the case for the other two load conditions (load 2: $t(45) = 1.27$, $p = .21$, load 6: $t(45) = -1.19$, $p = .21$). Descriptive statistics can be found in supplementary Table 2.

3.2. Time course, topography, and amplitudes of tCDA

3.2.1. Effects of cue latency on tCDA

A three-way mixed ANOVA with between-subjects factor GROUP and within-subjects factors CUE LATENCY and ELECTRODE showed an effect of CUE LATENCY on the onset of the tCDA over F5/F6 and CP3/CP4 ($F(4,176) = 221.63$, $p < .001$, $\eta_p^2 = .83$). As mentioned in the methods section, we used the inflection point, i.e., highest gradient in the waveform, as a proxy for component onset. The later the cue was presented, the later this inflection point emerged (see Table 3).

Table 3

Timing of inflection points in tCDA (tactile contralateral delay activity) measured at frontal and centroparietal electrodes as a function of cue latencies. Data are given in ms; standard deviation is displayed in brackets.

	All (N = 46)		Blind (n = 22)		Sighted (n = 24)	
	F5/6	CP3/4	F5/6	CP3/4	F5/6	CP3/4
150ms	424.9 (286.3)	469.1 (291.0)	424.6 (287.3)	336.1 (220.7)	425.1 (291.6)	591.1 (297.9)
300ms	530.6 (266.2)	611.9 (309.7)	479.0 (257.6)	528.4 (297.1)	577.9 (270.5)	688.6 (307.0)
500ms	817.4 (310.6)	877.0 (297.1)	768.0 (307.4)	850.2 (310.1)	862.6 (313.0)	901.4 (289.1)
800ms	1093.6 (290.6)	1152.0 (310.1)	1041.5 (257.9)	1092.1 (277.7)	1141.4 (315.5)	1206.9 (333.5)
1200ms	1677.4 (253.6)	1479.2 (238.6)	1650.3 (209.5)	1444.1 (213.4)	1702.2 (290.5)	1511.4 (259.9)

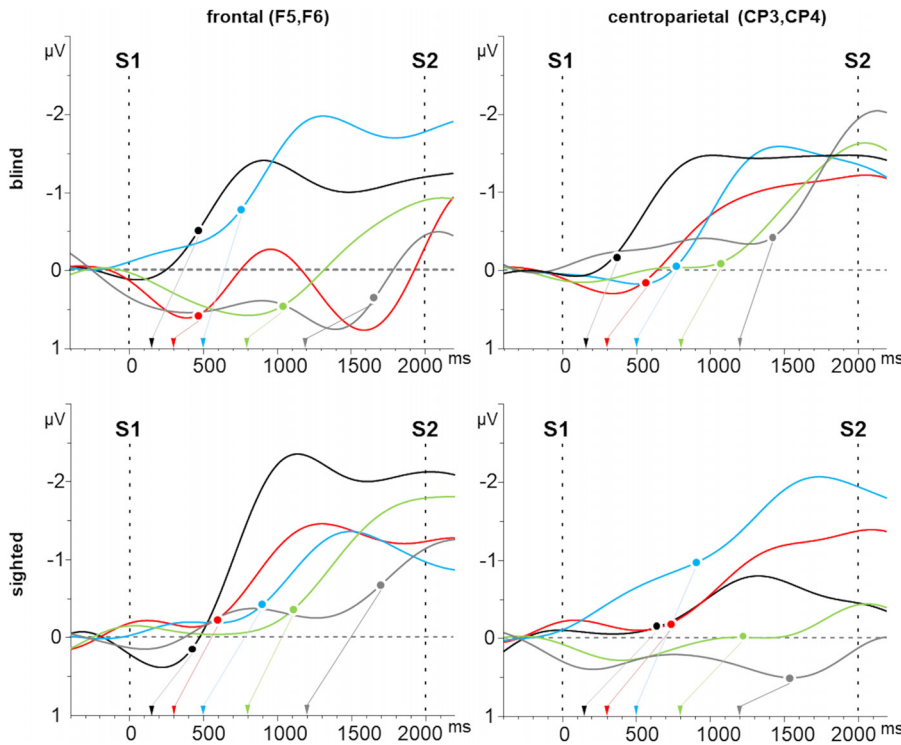


Fig. 6. tCDA (tactile contralateral delay activity) evoked by different cue latencies at frontal and centroparietal electrodes of sighted participants and participants who are blind. S1 = first stimulus, S2 = second stimulus. Colors indicate latency of cue: black = 150ms, red = 300ms, blue = 500ms, green = 800ms, grey = 1200ms. Dots in matching colors highlight the inflection points in each time course. Triangle markers along x-axis, using the same color scheme, show the timing of the corresponding cue on the time line. Blind $n = 22$, sighted $n = 24$. Note that the strong filter was only used for the purpose of a reliable automatic inflection point detection and better visualisation; analyses were all performed on wave forms as described in the methods section. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

This ANOVA also returned a main effect of GROUP ($F(1,44) = 10.38$, $p = .002$, $\eta_p^2 = .19$). The time courses of participants who are blind reached the inflection points approximately 100ms earlier compared to those of sighted participants ($t(44) = 3.22$, $p = .002$, $d = 0.95$). This ANOVA also returned a significant interaction between CUE LATENCY and ELECTRODE ($F(4,176) = 4.46$, $p = .002$, $\eta_p^2 = .09$). Post-hoc analyses suggest that when the cue was presented 1200ms after S1, centroparietal activity preceded frontal activity ($t(45) = 4.10$, $p < .001$, $d = 0.60$) while in conditions with an earlier cue (notably, where behavioral data indicate their strongest benefit), inflection points were more comparable at the two sites (all p -values $> .66$). Analyses using a different approach to identify the onset of the tCDA, i.e. automatic detection of the positive peak 500ms after cue onset, showed similar results (see supplementary Table 4 and 5). Fig. 6 visualizes the time courses recorded in participants with and without blindness. Note the pronounced rises in frontal electrodes in sighted participants and in centroparietal electrodes in participants who are blind. Descriptive statistics of the amplitudes, and t -tests comparing them to zero, can be found in supplementary Table 6. Parameters of the post-hoc t -tests are in supplementary Table 3.

3.2.2. Load effects on tCDA in frontal and somatosensory areas

A three-way mixed ANOVA on the tCDA amplitudes (restricted to cued trials) revealed a main effect of the factor ELECTRODE ($F(1,44) = 7.06$, $p = .01$, $\eta_p^2 = .14$) and a significant interaction between

ELECTRODE and GROUP ($F(1,44) = 10.12$, $p = .003$, $\eta_p^2 = .19$, Fig. 7). Post-hoc analyses reveal this interaction was driven by participants with and without blindness differing in the topographic distribution of neural activation over all load conditions; participants who are blind showed higher negative potentials over centroparietal areas compared to sighted participants ($t(44) = -3.10$, $p = .006$, $d = -0.92$). The influence of memory LOAD (two, four, or six presented items) on neural activation did not reach significance but trended towards it ($F(1.5,68.1) = 2.81$, $p = .08$, $\eta_p^2 = .06$). Amplitudes evoked by intermediate memory load seemed to be higher compared to those evoked by low and high memory load (4 – 2 items: $t(45) = -2.75$, $p = .018$, $d = -0.40$; 4 – 6 items: $t(45) = -1.89$, $p = .07$, $d = -0.28$). There was no significant interaction between LOAD and GROUP ($F(1.5,68.1) = 0.60$, $p = .51$, $\eta_p^2 = .01$). Descriptive statistics and t -tests against zero can be found in Table 7 in the supplementary material.

3.2.3. Topographic comparison across cue conditions

A three-way mixed ANOVA on tCDA amplitudes revealed an interaction between GROUP and ELECTRODE ($F(1,44) = 8.69$, $p = .005$, $\eta_p^2 = .17$). Post-hoc analyses reveal participants with and without blindness differed in topography; participants who are blind showed more negative tCDA amplitudes over centroparietal sites compared to sighted participants ($t(44) = -2.05$, $p = .046$, $d = -0.61$), while at frontal electrodes no difference was evident ($t(44) = 1.27$, $p = .21$). No main effect of cue latency was observed ($F(3,146) = 1.68$, $p = .17$), suggesting cues

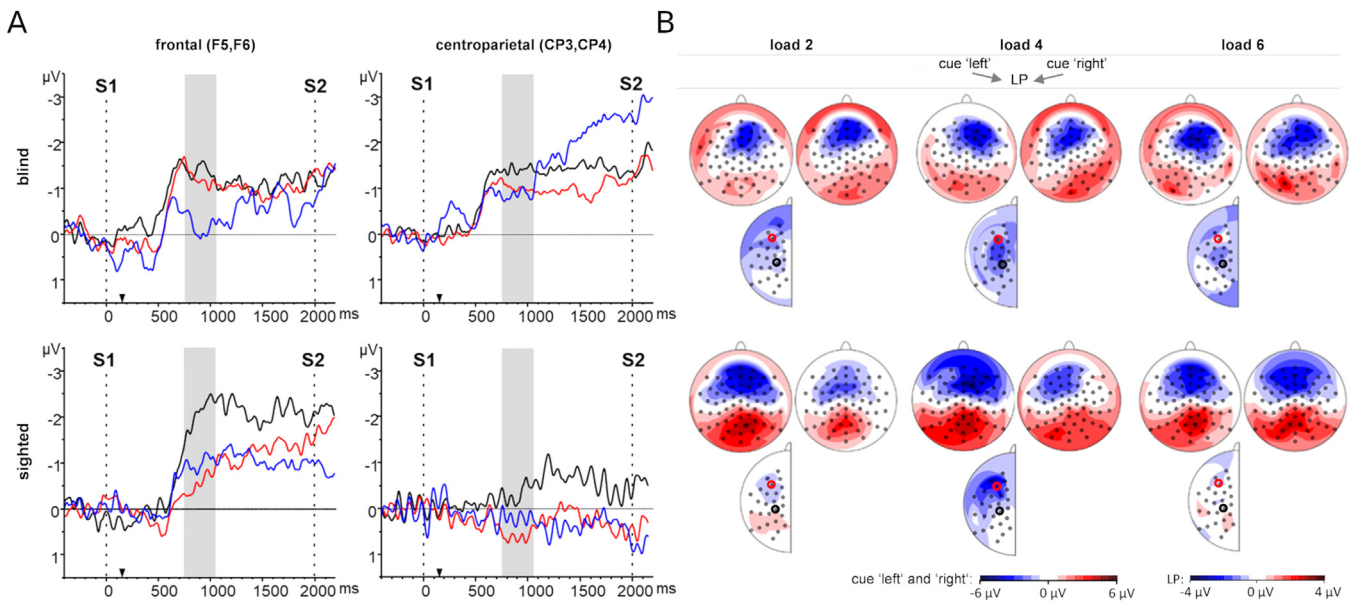


Fig. 7. A. Time course of tCDA and topographic distribution of neural activation for different load conditions for participants with and without blindness. Red line = two presented items, i.e., load 2, black = four presented items, i.e., load 4, blue = six presented items, i.e., load 6, S1 = first stimulus, S2 = second stimulus. Cue onset depicted by black marker on the x-axis, 150ms after S1 (time 0). B. Topographies. Each trio of topographies contains cues to the left hand, cues to the right hand, and, beneath them, the lateralized potential (LP; calculated as described in the method section). Topography maps are the average activity across time window 850-1050ms relative to S1 (shaded grey area in left panels). Blind $n = 22$, sighted $n = 24$. Note that the depicted waveforms have been filtered with a 5Hz high cut-off filter for a better visualisation; analyses were all performed on wave forms as described in the methods section.

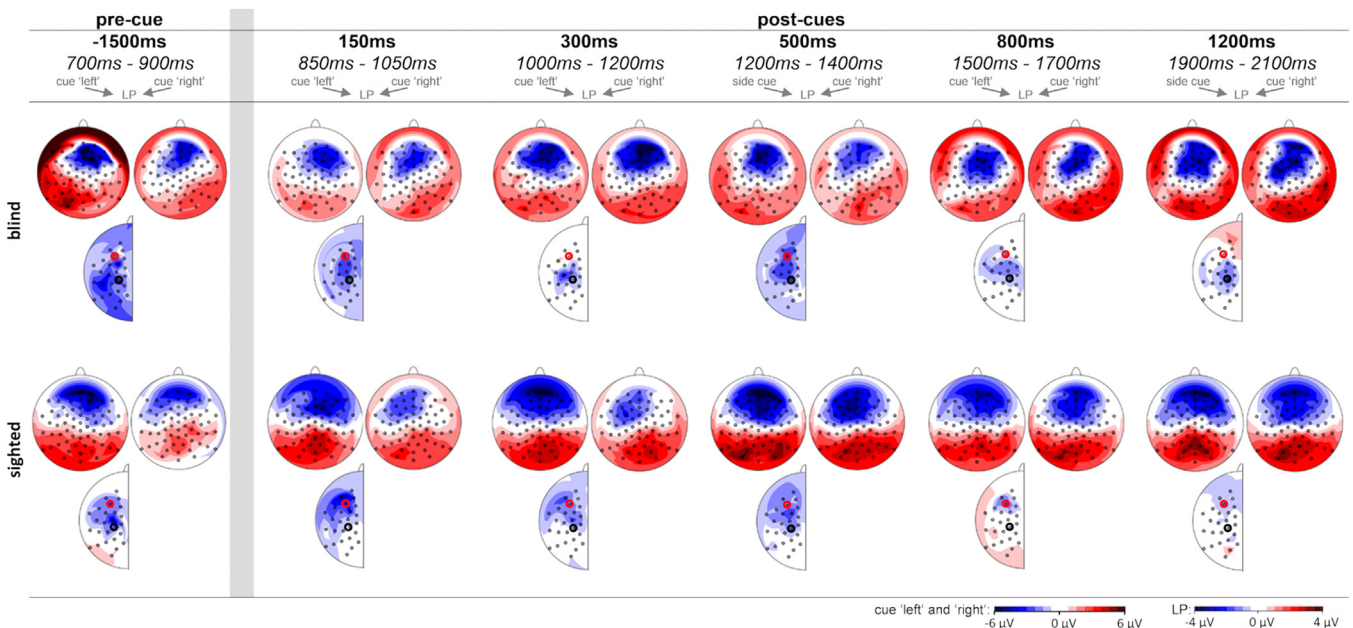


Fig. 8. Distribution of neural activation in participants with and without blindness for all cue latencies. LP = lateralized potentials, F5/F6 is marked in dark red, CP3/CP4 in black. Topographies are averaged across the window 700-900ms after onset of the relevant cue (or after S1 condition -1500ms). Time of these windows on the original time scale is written at the top of each column in italics. Blind $n = 22$, sighted $n = 24$.

appearing before or after S1 had no bearing on tCDA amplitudes. The topographies are visualized in Fig. 8.

3.2.4. Source analysis

LORETA analysis showed wide-spread frontal activity, including the dorsolateral and ventrolateral prefrontal cortex (DLPFC, VLPFC), and premotor areas (PMA), with a clear lateralization contralateral to the cued hand. Activation of somatosensory areas was also found. T -tests against zero with the amplitudes in the mentioned areas confirmed significant activation. In the condition with cues after 300ms, the p -value

concerning primary somatosensory cortex (SI; BA 1, 2, 3) was .021 for participants who are blind; all other p -values were $< .001$. Exemplarily for all other conditions, Fig. 9 shows the activity distribution during trials with cues after 800ms.

3.3. Correlation performance and tCDA

We conducted correlation analyses to probe the relation between tCDA and task performance across subjects, separately for different task conditions. In frontal areas, in conditions with four presented items and

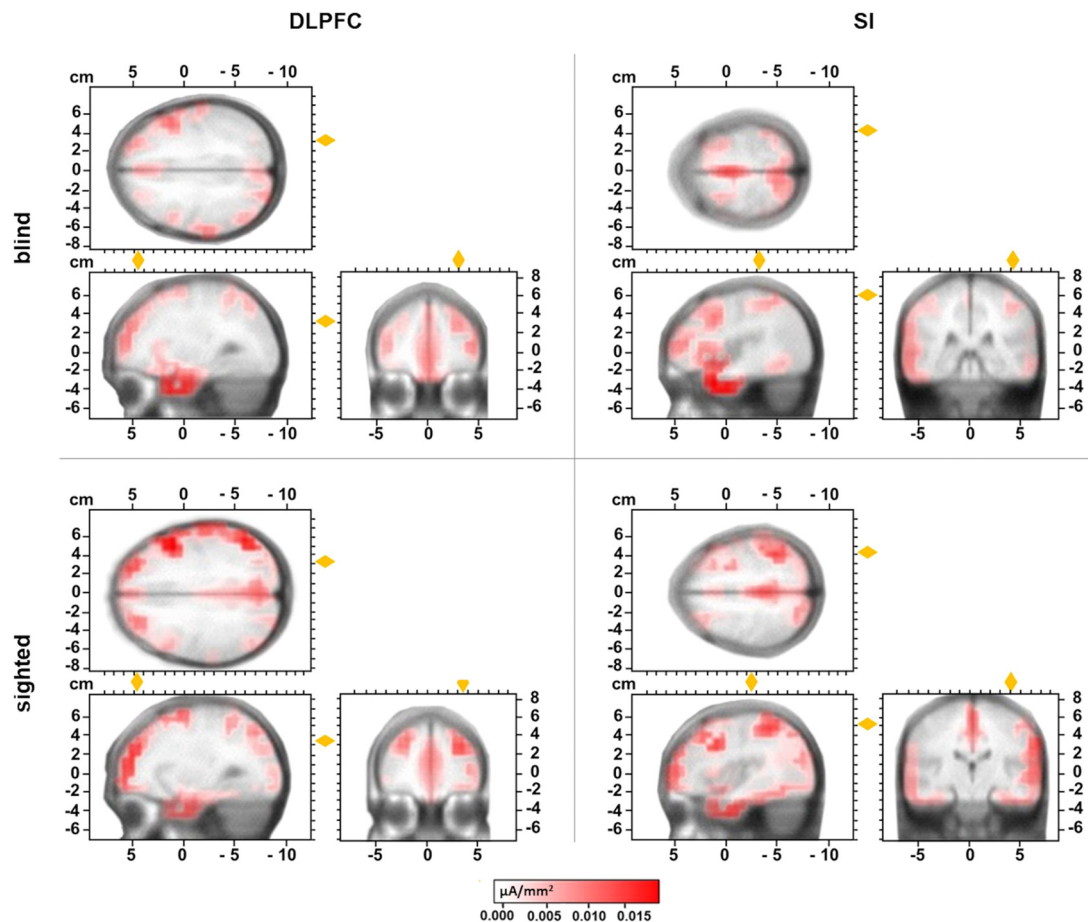


Fig. 9. Results of LORETA-source analysis (BrainVision Analyzer 2.1) for frontal and somatosensory areas, exemplarily displayed for trials containing the cue ‘left’ in the condition with cue presentation after 800ms. The time window of 700-900ms after the auditory cue was analyzed, i.e., 1500-1700ms after S1. Note the wide-spread frontal activity with prominent activity contralateral to the presented cue.

varying cue latencies, we observed no relation ($r = .25$, $p = .09$). There was similarly no correlation between performance and neural activation in centroparietal areas ($r = -.12$, $p = .42$). In the conditions with varying memory load, we found a negative correlation between performance rate and tCDA in the centroparietal area ($r = -.35$, $p = .02$). The nature of this correlation suggests that performance rate increases with increasing lateralized negativity at centroparietal electrodes. We observed no correlation between neural activation in frontal areas and performance rates ($r = -.03$, $p = .84$).

4. Discussion

The purpose of this study was to shed further light on the effects of visual sensory deprivation and a compensatory focus on somatosensory memory processes as reflected by tactile contralateral delay activity (tCDA). To that aim, we tested both participants with and without blindness performing a tactile change-detection task, and further explored the behavioral and neurophysiological effects of memory load and a cue, which narrowed down the possible locations of impending change. tCDA was seen in both frontal regions and somatosensory areas, consistent with sensory recruitment theories. tCDA amplitudes seemed to be highest with intermediate memory loads. We observed that participants who are blind, and with enhanced tactile proficiency due to their experience in Braille reading and intense usage of tactile information showed a number of key differences to sighted participants. They first showed better performance in the change-detection task, across all levels of load, and made better use of the cue. Secondly, they showed an earlier rise in tCDA in both frontal and somatosensory sites. Third, they showed more

pronounced lateralized negativity over somatosensory regions. Finally, we observed that across all subjects, better performance was associated with higher lateralized negativity over somatosensory areas in conditions with short cue latency, pointing towards a functional relevance of attention-based contralateral stimulus post-processing in primary and secondary somatosensory areas.

Separately, we additionally observed that the cue, appearing up to 800ms after the memorized array, improved task performance in both groups, providing evidence that somatosensory traces last for up to over a second after stimulus offset.

4.1. Differences in task performance between participants with and without blindness and experience in Braille reading

Participants who are blind outperformed sighted participants in the tactile change-detection task across all experimental conditions. Our data therefore reinforce the findings of previous research demonstrating enhanced sensory and cognitive function in people who are blind, and support cross-modal compensation in blindness with respect to STM (Boven et al., 2000; Fine and Park, 2018; Kauffman et al., 2002; Kupers and Ptito, 2014; Pascual-Leone et al., 1993; Pascual-Leone and Torres, 1993; Sadato et al., 1998; Withagen et al., 2013). The sense of touch is one of the main channels for people who are blind to perceive their environment, explaining why their processing of tactile information is greater than that of regular-sighted people. Our data make additional cognitive contributions to the compensation literature. We first observed that increasing memory load impaired task performance for both participants with and without blindness in a similar manner.

However, participants who are blind evidently profited more from cues, across a wider range of memory loads. Descriptive data suggested that sighted participants garnered no benefit from cues in low-load conditions, and even performed better in the high-load condition when no cue was presented. This surprising latter effect may have been due to cues being perceived as an additional distraction, which disrupted maintenance of the larger, more effortful array. Alternatively, cues may not have been helpful due to initial unsuccessful encoding outright. The unique ability amongst participants who are blind to utilize the cue under heavy load conditions demonstrates not just a potentially greater capacity in somatosensory STM, but also more efficient task-relevant tuning of this process by affiliated top-down resources.

More generally, deteriorating performance with increasing memory load is consistent with previous work across different STM modalities (Katus et al., 2015; Vogel and Machizawa, 2004). Prior studies using partial report procedures further report effective tactile STM to be limited to five items (Gallace and Spence, 2014). Our results suggest a similar capacity limitation of around four items, which might be slightly higher in participants who are blind.

4.2. Haptic memory: duration of somatosensory memory traces

Performance rates of participants with and without blindness improved with the presentation of cues up to 800ms after stimulus onset (i.e., 650ms after the offset). Participants apparently made use of these cues, which theoretically reduced their memory load by half (from two items at each hand to only two items at the relevant hand). Cues prior and post the sample stimulus (S1) led to performance improvement, suggesting that directing attention to task-relevant stimuli, makes the encoding and/or access of memory representations more efficient. The fact that cues 650ms after S1 offset still had an impact on performance presents evidence that initial somatosensory memory traces last at least this long. Given the duration of the cue, the time needed for its decoding and the ensuing redeployment of attention, somatosensory memory traces seem to endure up to one second. The positive effect of cues on performance disappeared only when they appeared 1200ms after S1. While this may reflect the temporal limit of somatosensory memory traces, an alternative explanation is that the trace was interrupted by the soon-following second stimulus (S2; 600ms after cue offset). Overlap between processing of the auditory cue and S2 is at least plausible, given that word recognition itself can take up to 500ms (Balass et al., 2010; Pykkänen and Marantz, 2003) and further experiments could include longer cue latencies and extend inter-stimulus intervals to disentangle these effects. Furthermore, additional cues with long latencies could help substantiate the limit of two seconds for somatosensory memory traces suggested by previous work (Bliss et al., 1966; Shih et al., 2009).

Notably, performance amongst participants did not seem to improve when cues appeared 300ms post S1, deviating from the broader pattern seen in the other conditions. Descriptive data suggested this was mainly due to sighted participants not using the cue effectively. As we controlled for order effects, and the SNR was not noticeably different from other latency conditions, with few outliers, the insignificant cue effect in this condition might be random. Haptic-auditory attentional blink effects might also be linked to the observed results. However, research on this topic focuses on the visual system and rarely concerns combined haptic and auditory stimuli (Dell'Acqua et al., 2006; Dux and Marois, 2009; Rau et al., 2020). Another possible explanation could be the cue overlapping with a still ongoing encoding process related to S1. Beneficial use of cues with latencies beyond 300ms likely depend on the development of a functional memory representations of S1, while with lower cue latencies, S1 and the cue might be temporally integrated (Brockmole et al., 2002; Coltheart, 1980). Participants who are blind were possibly faster to encode the bilateral sample array and therefore made use of the cue at this early latency, and thus did not show a drop in performance in the condition with cues 300ms after S1.

4.3. Characteristics of tCDA in participants with and without blindness

4.3.1. Source analysis

LORETA-analysis revealed wide-spread frontal activity, suggesting dorsolateral areas are not exclusively involved during somatosensory STM maintenance. The results instead propose a frontal network might be collectively activated during the maintenance of complex tactile information. Activity in PMA (Brodmann areas 6), and VLPFC (Brodmann areas 44, 45, and 47) was also evident in our data. Previous research reports activations in VLPFC during maintenance of tactile information encoded through passive perception (Kostopoulos et al., 2007; Spitzer et al., 2014). In addition, supplementary motor areas and PMA activate when presented tactile stimuli are complex (Savini et al., 2012). Cortical activation may further differ depending on the method of perception, i.e., active exploring versus passive perception (Miller, 1978; Savini et al., 2012; Simões-Franklin et al., 2011) which is worth consideration in future research.

Our source analysis also confirmed involvement of somatosensory regions, with source activity bilaterally distributed. As our reported three-dimensional cortical maps show general current density and do not consider polarity (Pascual-Marqui et al., 1994; Pascual-Marqui et al., 1999), they cannot confirm whether bilateral activity reflects inhibitory positivity ipsilateral to the cue, and excitatory contralateral negativity, or whether activity of the same valence was present on both sides of the brain. However, the topographies in Figs. 7 and 8 point towards the latter interpretation. The few extant EEG studies on tCDA did not apply source analysis (Katus and Eimer, 2015, 2018; Katus et al., 2015, 2015), however, their reported topographies show lateral activity distributed over SI and SII. Frontal lateral activity was rather small if evident at all. Apart from fundamental involvement of somatosensory areas, the processing of complex tactile stimuli apparently drives more frontal (i.e., supramodal) activity than that of simpler vibrotactile stimuli, as seen in Katus and Eimer (2015, 2018).

The source analysis also showed some activity in temporal and occipital regions. Both regions might be involved in a wider network contributing to memory maintenance. However, activity in the temporal lobe may also have been related to auditory processing of the mechanical noise created by presentation of the Braille pins. Visual cortex may also have been involved for group-specific reasons, i.e. blindness-driven cortical reorganization in participants who are blind (Fine and Park, 2018), and efforts to visualize the tactile input in sighted participants. An exciting avenue for future research would be to explore the wider network configurations and dynamic blindness-driven functional reorganization during somatosensory short-term memory with brain imaging techniques offering higher spatial resolution (e.g., functional connectivity of fMRI data).

4.3.2. Sensitivity of tCDA to cue latency and memory load

In the present study, the time courses of tCDA varied in temporal accordance with different cue latencies which further substantiates the interpretation of tCDA as an attention-based activation of memory representations (Berggren and Eimer, 2016; Hecht et al., 2016; Katus and Eimer, 2015, 2018; Lewis-Peacock et al., 2012). We also observed synchronicity between prefrontal and somatosensory activity. Only in the condition with cues 1200ms after S1 did we see tCDA in somatosensory regions precede its analogue in prefrontal areas. This might be explained by preparatory somatosensory activity in light of the impending S2 and further studies could tackle this issue by expanding the inter-stimulus interval. Nonetheless, simultaneous prefrontal and centroparietal activation in all other conditions provides evidence that tactile memory performance requires monitoring activity in DLPFC to support modality-specific regions (Chai et al., 2018; Fuster, 2015).

We observed tCDA's amplitude trending toward significant modulation by load conditions, hinting at its sensitivity to memory load and task difficulty. In the descriptive data of sighted participants, we observed an increase in tCDA amplitudes from two to four memory items.

In contrast, tCDA amplitudes registered by participants who are blind, particularly at prefrontal electrodes, did not differ considerably between the low- and medium-load condition. This suggests that in these conditions, task difficulty, and the corresponding cognitive effort required to solve the task, might have been lower for participants with blindness. Presumably their experience and enhanced skills with tactile processing might explain this difference, relative to controls. In previous studies with sighted participants and vibrotactile stimuli, amplitudes increased in line with increasing memory load from one to two items (Katus and Eimer, 2015, 2018, 2015), but no further increase occurred from two to three items (Katus and Eimer, 2018). Differences in features and the complexity of presented stimuli most likely account for these different results.

4.4. What makes somatosensory STM effective?

4.4.1. Interplay of frontal and somatosensory areas

Both participants with and without blindness showed dorsolateral prefrontal tCDA during the tactile change-detection task. Furthermore, source analysis confirmed activity in a wide-spread frontal network. Frontal activation may therefore be necessary for the maintenance of tactile information, although not related to levels of expertise in tactile processing. Previous research on somatosensory memory has also pointed to a contributing role of frontal areas to successful memory maintenance (Burton and Sinclair, 2000; Curtis and D'Esposito, 2003; Harris et al., 2002; Kostopoulos et al., 2007; Staines et al., 2002; Zhao et al., 2018). Furthermore, marginally lower prefrontal tCDA amplitudes in the descriptive data of participants who are blind suggest that they possibly did expend less cognitive effort to solve the memory task compared to sighted participants. A possible explanation might lie in the general enhancement in executive functions and an increased prefrontal efficiency, which some studies have recently attributed to participants who are blind (Fine and Park, 2018; Singh et al., 2018). Likewise, the slightly higher frontal amplitudes of sighted participants might be interpreted as an inefficient, effortful attempt to recruit modality-specific areas to fulfil the memory task.

However, frontal activity alone does not appear to be sufficient for successful somatosensory STM maintenance. As participants with blindness performed better than sighted controls and showed greater tCDA over somatosensory regions in all experimental conditions, parallel activation of frontal and somatosensory areas instead seem particularly relevant for behavior. The extent of attention allocated to tactile memory representations in modality-specific areas may therefore be crucial for efficient memory maintenance. Enhanced tCDA in somatosensory regions amongst participants who are blind can possibly be attributed to cortical plasticity driven by their everyday usage of the tactile sense for perception and navigation (Fine and Park, 2018). Adaptively altered task-related neuronal networks may drive better encoding of sensory inputs and easier activation of memory traces in the somatosensory cortex. Enlarged cortical representation of the fingers used for Braille reading (Pascual-Leone et al., 1993; Pascual-Leone and Torres, 1993; Sadato et al., 1998) and increased connectivity in somatosensory regions (Heine et al., 2015) could also contribute to higher tCDA amplitudes. For a clearer understanding of the functional relevance of tCDA in frontal and somatosensory regions, future studies could additionally focus on comparisons between correct and incorrect trials.

Prefrontal and somatosensory tCDA also onsetted earlier in participants who are blind, relative to controls. Higher levels of expertise and training in tactile and auditory perception might therefore also lead to faster processing in corresponding neuronal networks (Fine and Park, 2018; Heine et al., 2015), which possibly contributed to the enhanced performance of participants who are blind. However, seeing as sighted participants showed generally rather low tCDA amplitudes over somatosensory areas, this latency effect should be interpreted with caution as it cannot be separated from amplitude effects.

4.4.2. Relation of tCDA and performance rates

It is reasonable to hypothesize that the enhanced negativity in somatosensory areas in participants who are blind is part of the neuronal basis for their superior performance in the tactile change-detection task. The correlation analysis with the whole sample comparably showed a direct linear relationship between lateralized negativity at centroparietal electrodes and performance rates in conditions with varying memory load. More negative tCDA amplitudes in the somatosensory cortex were associated with better performance. This was particularly the case for memory tasks with cues appearing at short latencies after S1 (150ms), i.e., those used in blocks assessing load effects. In conditions with long cue latencies, the correlation was also negative, but not significant. These results augment our knowledge of the pivotal role that somatosensory regions play for tactile memory. Not only have single-unit recordings in primates demonstrated delay activity in the somatosensory cortex during tactile memory tasks (Romo and Salinas, 2003; Zhou and Fuster, 1996), imaging studies with humans have also identified SI and SII, along with the DLPFC and parietal areas, as relevant cortical regions for maintenance of haptic information (Burton and Sinclair, 2000; Staines et al., 2002). TMS studies have affirmed the functional relevance of SI and SII (Harris et al., 2002; Zhao et al., 2018), which is further substantiated with our observed correlation between tCDA in somatosensory areas and memory performance. Studies on the visual CDA have reported a comparable relation between CDA over lateral-occipital and posterior-parietal regions and performance (Adam et al., 2018), as well as lower CDA amplitudes in incorrect trials (McCollough et al., 2007). Similar neuronal mechanisms underlying STM processes across modalities have been suggested (Bender et al., 2010; Sreenivasan and D'Esposito, 2019). Analogous relations between performance and delay activity across modalities can be interpreted as further support for that assumption, as well as evidence for sensory recruitment models.

The correlation not consistently yielding significance in all experimental conditions might be due to the different timing of the cues. Cues presented shortly after S1 most likely interfere with the initial stimulus processing, in contrast to cues with long latency. Higher tCDA in early processing states could facilitate a more pronounced memory representation and, on that basis, better performance rates. Also, it is plausible that the additional challenge of the curtailed encoding creates the ideal context to see individual differences in both behaviour and underlying neuronal processes.

We did not observe statistically significant correlations between prefrontal tCDA and performance in conditions with varying cue latencies, nor in load conditions with short cue latency. However, activity in this region onsetting in tandem with centroparietal activity in blocks where cues help performance stresses its likely relevance for task accomplishment. If prefrontal tCDA contributes to efficient STM, it however does not have a direct linear relation with performance.

To conclude, the results tentatively suggest a direct relation between tCDA in somatosensory areas and performance. Especially given combined superior task performance and higher tCDA amplitudes in somatosensory regions of participants who are blind, the efficient activation of relevant sensory areas seems to be crucial for somatosensory memory tasks. In particular, high (negative) tCDA amplitudes in the somatosensory cortex, coupled temporally with an active frontal network, seem to facilitate good performance. The effectiveness of tactile STM seems to increase with expertise, and appears to be related to activity in modality-specific brain areas.

4.5. Limitations

To capture activity originating in the DLPFC and SI we employed a hypothesis-driven approach that selected electrodes implicated by previous studies as overlying these cortical areas. Nonetheless, deterministic correspondence between electrode sites and site-specific cortical activity is complicated by volume conduction effects. Activity measured at sites over specific cortical areas might also entail activity originating

from more distant regions. We addressed this issue by first performing a source analysis, which revealed no specific between-group shifts in cortical sources or surface topography. Second, a data-driven topographic a priori PCA analysis showed that waveform timeseries at our target electrodes correlated with timeseries of spatially related frontal and centroparietal components. Our hypothesis-driven electrode selection therefore seemed appropriate and unlikely to have impacted our core findings. However, further studies employing data-driven methods such as permutation-based clustering (Maris and Oostenveld, 2007) or multivariate pattern analysis (Fahrenfort et al., 2018) may help refine the relative contributions of different supramodal areas or the specific parts of the somatosensory system. Even though we did not find respective first indications in our data, such techniques may lend key information regarding wider plastic reorganization or compensatory recruitment beyond classic electrode sites (e.g., visual areas), or uncover relevant time frames harder to discover with more conventional methods (Fahrenfort et al., 2017).

Variation in the onset of blindness amongst our participants may also have resulted in heterogeneity regarding neural plasticity and brain development (Fine and Park, 2018; Sadato et al., 2002). However, in additional analyses we compared participants who are congenitally blind with those who lost their sight later in life and did not observe differences regarding performance rates or tCDA characteristics. We additionally did not see a correlation between blindness onset or disease duration with performance rates (see supplementary material). Nonetheless, future studies could attempt to reduce such tempero-etiological heterogeneity or potentially examine its effects more formally.

In this study, Braille-like patterns were used as tactile stimuli. Even though the stimuli did not represent Braille letters, experienced Braille readers might have nonetheless drawn upon top-down strategies to assist with encoding, such as connecting them to existing letter representations or applying strategies used while learning Braille to keep the Braille-like stimuli in mind. Thus, their superior performance might not solely be due to enhanced tactile processing. Also, Romo and Salinas (2003) argue that the additional spatial component of Braille patterns makes the stimuli too complex for investigations into basic tactile information processing, and propose vibrotactile stimuli as more appropriate. However, we chose the Braille-like stimuli in this study due to their high ecological validity; tactile information in daily life almost always entails spatial components. Also, Braille-like stimuli allow inferences about neural networks involved in the passive haptic perception of Braille letters, which might facilitate a better understanding of Braille reading and, in the long run, new learning approaches for Braille reading (Mašić et al., 2020). Furthermore, memory research shows that different stimulus features are memorized differently, involving different brain areas and conforming to individual time horizons across memory traces (Pasternak and Greenlee, 2005). The use of Braille patterns therefore holds the potential to lead to additional insights into more complex tactile memory processing. However, future study designs using vibrotactile stimuli will have better compatibility with existing literature.

5. Conclusion

During a tactile change-detection task, presented cues could be used efficiently to streamline memory performance up to 800ms after the onset of a tactile sample stimulus. In line with sensory recruitment models, tCDA was seen in both frontal regions and somatosensory areas. Reinforcing cross-modal compensation hypotheses and adding new insights on neurophysiological underpinnings of vision loss, participants who are blind outperformed sighted participants and made better use of the cue, while uniquely showing more pronounced lateralized negativity in the somatosensory cortex. These group differences, and additional correlational analyses, suggest a direct relation between tCDA in the somatosensory cortex and performance, which provides new evidence for the functional relevance of tCDA in somatosensory memory. Efficient STM maintenance of complex tactile stimuli depends on the activation

and interplay between a wide-spread neural network involving frontal areas and somatosensory regions, with a pivotal role of lateralized activity in the latter.

Funding

We acknowledge support for the Article Processing Charge from the DFG (German Research Foundation, 491454339).

Ethics statement

The study was approved by the ethics committee at the Technische Universität Dresden, and conformed to the Declaration of Helsinki.

Software availability statement

The following software packages were used, and are available via the corresponding websites: BrainVision Analyzer - Brain Products GmbH, Gilching, Germany; IBM SPSS Statistics 27 software - IBM Corporation, Armonk, NY, USA.

Author contributions

Breiterger: Formal analysis, Data curation, Validation, Visualization, Methodology, Writing- original draft
 Pokorny: Formal analysis and Visualization
 Biermann: Formal analysis and Visualization
 Jarczok: Methodology, Writing- Review & Editing
 Dundon: Writing- Review & Editing
 Roessner: Methodology, Resources, Project administration
 Bender: Conceptualization, Methodology, Project administration, Resources, Writing- Review & Editing, Supervision

Data availability statement

The ethics committee did not grant permission to share study data with third parties or to upload data in anonymized form.

Declaration of Competing Interest

None.

Acknowledgment

Thanks go to Franziska Wuttig for data acquisition.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.neuroimage.2022.119407.

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