

2 **Vestibular inputs modulate somatosensory cortical processing**

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7 **Abstract** The vestibular system is unique among the  
8 senses because of the entirely multisensory nature of its  
9 cortical projections. Neuroanatomical and neuroimaging  
10 studies show that vestibular stimulation activates somato-  
11 sensory areas, and particularly the so-called parieto-insular  
12 vestibular cortex (PIVC) in the monkey, while deactivating  
13 visual areas. Further, recent psychophysical studies showed  
14 that vestibular stimulation facilitates detection of electro-  
15 cutaneous stimuli, suggesting a vestibular-somatosensory  
16 perceptual interaction. However, the functional mechanism  
17 underlying this perceptual facilitation remains unclear. We  
18 therefore recorded somatosensory potentials evoked by  
19 left median nerve stimulation, before and immediately after  
20 left cold caloric vestibular stimulation (CVS), in a small-  
21 scale study of eight healthy volunteers. CVS selectively  
22 enhanced the N80 component recorded over both ipsilat-  
23 eral and contralateral somatosensory areas, without  
24 significantly affecting earlier or later components. Inter-  
25 estingly, the N80 component has been localised to the  
26 parietal operculum, which includes the human homologue  
27 of the monkey PIVC, and is thus a prime neuroanatomical  
28 candidate for vestibular-somatosensory convergence. As a  
29 control, we measured visual evoked potentials to reversing

checkerboard patterns and found no effects of vestibular 30  
stimulation. This rules out explanations based on indirect 31  
effects of vestibular modulations, such as general arousal 32  
or supramodal spatial attention. We believe our results 33  
provide the first clue linking brain structure to function for 34  
the interaction between vestibular and somatosensory 35  
systems. 36

37  
38 **Keywords** Vestibular system · Somatosensory evoked  
39 potentials · Visual evoked potentials · Multisensory  
40 interaction

41 **Abbreviations**

42 CVS Caloric vestibular stimulation  
43 SEPs Somatosensory evoked potentials  
44 VEPs Visual evoked potentials  
45 SII Secondary somatosensory cortex  
46  
47

48 **Introduction**

49 The vestibular system plays a continuous role in most  
50 everyday adaptive behaviours, including motion percep-  
51 tion, posture control and orientation in the surrounding  
52 space (Berthoz 1996). These complex functions require  
53 integration of vestibular inputs with signals from other  
54 sensory modalities, such as vision and somatosensation.  
55 Interestingly, no unimodal vestibular cortex has been found  
56 so far in the mammalian brain. Rather electrophysiological  
57 studies identified a complex vestibular network whose core  
58 area is the parieto-insular vestibular cortex (PIVC). This  
59 area lies in the posterior parietal operculum extending into  
60 the posterior insular lobe (Akbarian et al. 1988; Grüsser  
61 et al. 1990; Guldin and Grüsser 1998).

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62	Recently, human functional neuroimaging studies	Subjects with a history of motor, somatosensory, vestibular	111
63	revealed a pattern of cortical and sub-cortical activations in	or auditory disorders were excluded. Informed consent was	112
64	response to caloric vestibular stimulation (CVS, Bottini	obtained prior to participation in the experiment. The	113
65	et al. 1994; Fasold et al. 2002; Naito et al. 2003), including	experimental protocol was approved by the University	114
66	the posterior parietal operculum and the retroinsular and	College London research ethics committee and the study	115
67	insular cortex. Vestibular activations were also found in the	was designed according to ethical standards of the Decla-	116
68	primary and, more prominently, in the secondary somato-	ration of Helsinki.	117
69	sensory cortex (SII) (Fasold et al. 2002; Bottini et al. 1994;		
70	Emri et al. 2003).	Design and caloric vestibular stimulation procedure	118
71	Evidence from brain-damaged patients is consistent with		
72	a functional link between vestibular and somatosensory	Data from each participant were gathered in a single ses-	119
73	systems. A temporary remission of tactile hemianaesthesia	sion. Our interest focused on specific changes in somato-	120
74	has been described in right and left brain-damaged patients	sensory and visual cortical processing between two	121
75	immediately after CVS was administered to the left ear	experimental conditions: one before CVS (Pre-CVS) and	122
76	(Vallar et al. 1990, 1993). However, the possibility that	one immediately after CVS (Post-CVS). CVS was per-	123
77	CVS influences touch indirectly, via a supramodal atten-	formed by slowly pouring 30 ml of cold (iced) water into	124
78	tional mechanism, cannot be definitively ruled out by these	the external left auditory canal close to the tympanic	125
79	patient studies (Vallar et al. 1990, 1993). Recent psycho-	membrane using a 50-ml syringe with a short piece of	126
80	physical studies provide stronger evidence that vestibular	elastic tubing attached. The participant's head was posi-	127
81	inputs influence somatosensory processing directly, and not	tioned 30° backward from the horizontal plane, placing the	128
82	only via vestibular effects on supramodal spatial attention.	lateral semicircular canal in the vertical orientation. CVS	129
83	We showed that vestibular stimulation facilitates detection	effectiveness was verified by the experimenter by the	130
84	of faint somatosensory stimuli also in healthy volunteers,	presence of ipsilateral slow-phase nystagmus. Care was	131
85	not only on the left (Ferrè et al. 2011a), but also on the	taken to ensure both electrophysiological recordings were	132
86	right (Ferrè et al. 2011b) hand. However, the exact func-	completed within 15 min following CVS since CVS effects	133
87	tional mechanism underlying this perceptual facilitation	on the vestibular system are limited to around 30 min	134
88	remains unclear.	(Bottini et al. 1995; Ngo et al. 2007).	135
89	To resolve this issue, we recorded somatosensory	During electrophysiological recordings participants were	136
90	evoked potentials (SEPs) by left median nerve stimulation,	seated with their left arm resting palm-up in a dimly lit	137
91	before and immediately after left CVS. Since vestibular	room. SEPs and VEPs were recorded before and immedi-	138
92	and somatosensory inputs converge in multimodal areas	ately after CVS (Pre-CVS and Post-CVS conditions). The	139
93	(Bottini et al. 1995; Fasold et al. 2002, Eickhoff et al.	order of SEP and VEP testing was counterbalanced between	140
94	2006), we hypothesized that CVS would predominantly	participants (ABBA order). Square-wave electrical pulses	141
95	affect long-latency components generated by higher	set at 10 mA of amplitude were delivered transcutaneously	142
96	somatosensory and multisensory areas rather than short-	to the left median nerve at the wrist at 4 Hz. Participants	143
97	latency components arising from primary somatosensory	focused visual attention and gaze directly on a fixation point.	144
98	cortex (Allison et al. 1991). We also measured visual	Pulse duration was manipulated so stimulation generated	145
99	evoked potentials (VEPs) to reversing checkerboard pat-	just detectable visible thumb twitches in each participant.	146
100	terns. VEPs served partly as a control for non-specific,	Experimental stimulation was then set at 120 % of this value	147
101	supramodal effects of CVS, such as changes in level of	(mean pulse width: 144 µs). 1200 median nerve stimuli were	148
102	arousal or spatial attention. In addition, we were interested	delivered over five separate blocks, lasting around 1 min	149
103	in possible dissociations between vestibular influences on	each. To record VEPs, a black and white full-screen	150
104	somatosensory and visual processing given neuroimaging	checkerboard pattern was displayed on a computer screen	151
105	reports of visual cortex deactivation due to vestibular	with a refresh rate of 60 Hz at 100 cm viewing distance.	152
106	stimulation (Bense et al. 2001; Naito et al. 2003).	Mean luminance of the stimulus was 100 cd/m <sup>2</sup> with 98 %	153
		contrast. The checkerboard had a spatial frequency of one	154
		cycle/degree and reversed every 500 ms.	155
107	<b>Materials and methods</b>	Electrophysiological recordings and data analysis	156
108	Participants		
109	Eight naïve paid participants volunteered in this experi-	A SynAmp amplifiers system and Scan 4.3 software	157
110	ment (three female, mean age = 24.7, SD = 4.6 years).	(Neuroscan, El Paso, TX) were used to record electroen-	158
		cephalographic (EEG) data. Recordings were obtained	159

160 from 14 scalp electrodes, (F1, Fz, F2, C3, Cz, C4, CP3,  
161 CPz, CP4, P3, Pz, P4, O1, and O2), placed according to the  
162 10–20 System. Horizontal electroculogram (EOG) was  
163 recorded from bipolar electrodes placed on the outer canthi  
164 of each eye, and vertical EOG was recorded from bipolar  
165 electrodes placed above and below the right eye. The refer-  
166 ence electrode was AFz. Electrode impedances were kept  
167 below 5 k $\Omega$ . EEG signals were amplified, bandpass filtered  
168 from 0.05 to 1000 Hz (slope: 12 dB/octave), and digitized  
169 at 5 kHz. Data were analysed using Matlab ([http://www.  
170 TheMathworks.com](http://www.TheMathworks.com)), and EEGLAB (Delorme and Makeig  
171 2004).

172 To identify SEP components, epochs were extracted  
173 from 50 ms before each shock to 200 ms afterwards. The  
174 signal between 2 and 13 ms after electric shock onset was  
175 linearly interpolated to remove electrical artefacts of  
176 stimulation. To identify VEP components, epochs of  
177 250 ms were extracted from the raw EEG data from 50 ms  
178 before each reversal of the checkerboard to 200 ms after-  
179 wards. Data were then digitally low-pass filtered at 70 Hz.  
180 Trials with eyeblinks (horizontal or vertical EOG exceed-  
181 ing  $\pm 80$   $\mu$ V) or with voltage exceeding  $\pm 120$   $\mu$ V at any  
182 channel between -50 and 200 ms relative to each shock  
183 were eliminated. Components of the evoked response were  
184 identified by inspecting grand averages. The peak ampli-  
185 tudes for each component were then calculated by identi-  
186 fying maxima/minima in individual subject averages in  
187 each condition (Pre-CVS and Post-CVS) in the time win-  
188 dow appropriate for established components in the grand  
189 average, based on previous literature.

## 190 Results

191 Inspection of SEP topography showed broadly consistent  
192 components across contralateral central and parietal leads.  
193 Four short-latency components were identifiable (N20,  
194 P27, N30 and P50) (see Fig. 1a). Three longer-latency  
195 components were identifiable; N80, P100, and N140. Peak  
196 amplitudes for each component were calculated across  
197 contralateral sensory cortex electrode (C4), and Pre-CVS  
198 and Post-CVS amplitudes were compared using paired  
199 T-tests. Amplitudes of short-latency SEPs were not influ-  
200 enced by CVS (N20:  $t_{(1,7)} = 0.421$ ,  $p = 0.686$ ; P27:  
201  $t_{(1,7)} = 0.416$ ,  $p = 0.690$ ; N30:  $t_{(1,7)} = 0.406$ ,  $p = 0.697$ ;  
202 P50:  $t_{(1,7)} = 1.94$ ,  $p = 0.094$ ). Among the long-latency  
203 components, the N80 component showed a clear difference  
204 between the experimental conditions: the N80 amplitude  
205 recorded over contralateral sensory cortex (channel C4),  
206 was greater for the Post-CVS condition than the Pre-CVS  
207 condition ( $t_{(1,7)} = 2.907$ ,  $p = 0.02$ ). Vestibular stimulation  
208 did not affect P100 ( $t_{(1,7)} = 0.526$ ,  $p = 0.616$ ) and N140  
209 ( $t_{(1,7)} = 1.263$ ,  $p = 0.247$ ) components.

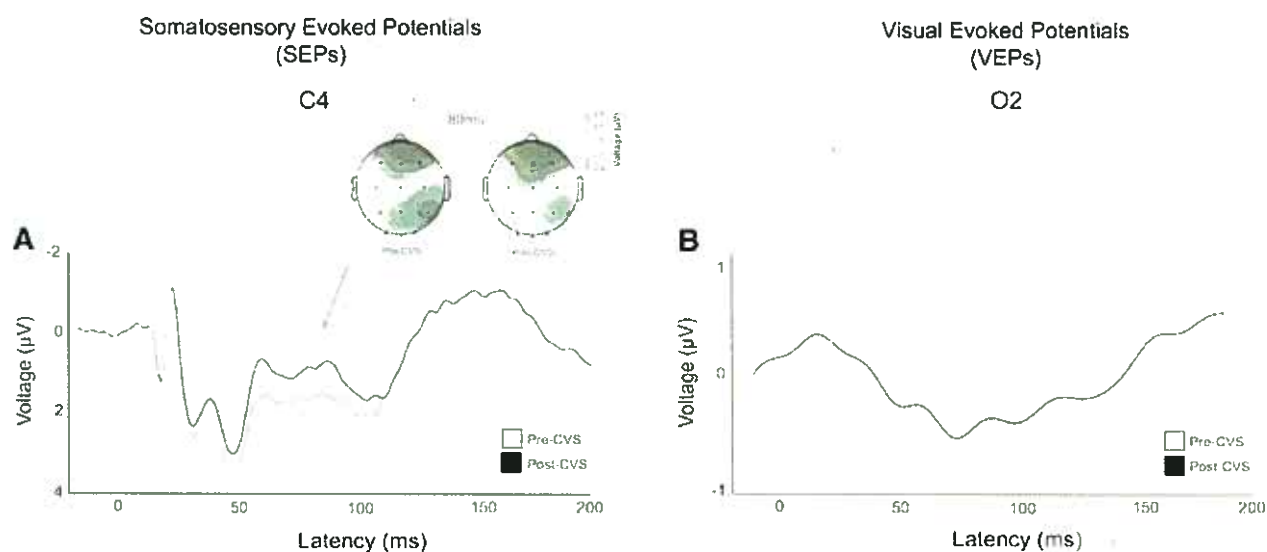
The N80 component has been interpreted in different 210  
ways. Allison et al. (1991) suggest it reflects a second, 211  
recurrent wave of processing within contralateral primary 212  
somatosensory cortex (Allison et al. 1991). Others suggest 213  
it reflects processing in operculum region and SII (García- 214  
Larrea et al. 1995), based on its bilateral pattern (Jung et al. 215  
2009). Therefore, we further investigated the lateralisation 216  
of CVS effects on the N80 component in a factorial follow- 217  
up analysis, with factors of Stimulation (Pre-CVS/Post- 218  
CVS) and Hemisphere (contralateral C4/ipsilateral C3). 219  
We found significant main effect of Stimulation ( $F_{(1,7)} =$  220  
10.349,  $p = 0.015$ ) and Hemisphere ( $F_{(1,7)} = 11.155$ ,  $p =$  221  
0.015), but no interaction between Stimulation and Hemi- 222  
sphere ( $F_{(1,7)} = 2.043$ ,  $p = 0.196$ ). 223

For pattern reversals, the VEPs (see Fig. 1b) showed N75, 224  
P100 and N135 peaks at occipital electrodes, resembling 225  
those reported previously. Peak amplitudes for each com- 226  
ponent were calculated across occipital cortex electrodes 227  
(O1, Oz and O2). No statistically reliable effects of CVS on 228  
any VEPs component were found in any channels (O1 229  
channel: N75:  $t_{(1,7)} = -0.258$   $p = 0.804$ ; P100:  $t_{(1,7)} =$  230  
 $-0.459$ ,  $p = 0.660$ ; N135:  $t_{(1,7)} = -0.032$ ,  $p = 0.975$ ) 231  
Oz channel: N75:  $t_{(1,7)} = -0.624$   $p = 0.552$ ; P100:  $t_{(1,7)} =$  232  
 $-1.470$ ,  $p = 0.185$ ; N135:  $t_{(1,7)} = -1.109$ ,  $p = 0.304$ ; O2 233  
channel: N75:  $t_{(1,7)} = -0.7521$   $p = 0.477$ ; P100:  $t_{(1,7)} =$  234  
 $-1.180$ ,  $p = 0.276$ ; N135:  $t_{(1,7)} = -1.317$ ,  $p = 0.229$ ). 235

## Discussion 236

The vestibular system has widespread interactions with 237  
multisensory cortical networks, including somatosensory 238  
areas. Our results provide preliminary evidence regarding 239  
how vestibular input might influence somatosensory cortical 240  
processing. SEPs evoked by left median nerve stimu- 241  
lation showed CVS-induced modulation in the N80 242  
component recorded over both ipsilateral and contralateral 243  
somatosensory areas. The vestibular modulation was spe- 244  
cific to this component since neither earlier nor later SEP 245  
components were affected by CVS. Moreover, the effect 246  
was specific to somatosensory processing. VEPs to 247  
reversing checkerboard patterns were not influenced by 248  
CVS, ruling out explanations based on indirect vestibular 249  
effects mediated by general arousal or supramodal 250  
attention. 251

Different origins have been proposed for the N80 SEP 252  
component. Allison et al. (1991) suggested this compo- 253  
nent represented a recurrent, second wave of processing 254  
within SI. Others localised this component to SII (García- 255  
Larrea et al. 1995; Hari et al. 1984). Investigations of SII 256  
latency confirm that responses to nerve stimulation are not 257  
found in this area prior to 60 ms post-stimulus (Allison 258  
et al. 1989; Frot and Mauguière 1999; Hämäläinen et al. 259  
1990; Hari et al. 1984; Hari et al. 1993; Karhu and Tesche 260



**Fig. 1** Vestibular modulation of somatosensory processing. **a** Grand average SEPs recorded over contralateral channel C4. Note significant enhancement of N80 in Post-CVS condition compared with Pre-CVS. **b** Grand average VEPs recorded over right occipital channel

(O2). No significant difference was found between experimental conditions (Pre-CVS and Post-CVS) although the traces are visually distinct

1999; Kakigi 1994; Waberski et al. 1999). Crucially, the N80 component has similar amplitudes contralaterally and ipsilaterally (Jung et al. 2009). This strongly supports the hypothesis of an SII origin for this component, given the bilateral organisation of this area (Iwamura et al. 1994).

More recent source analysis studies localised the N80 component in the parietal operculum (Jung et al. 2009; Eickhoff et al. 2010). Specifically, the source lies immediately adjacent to the neuroanatomical site of vestibular-somatosensory convergence in the human homologue of the monkey PIVC, identified by combined functional imaging and vestibular stimulation (Eickhoff et al. 2006). Functional responses and topographic characteristics suggest that area OP 2 is the key vestibular projection within PIVC. This area lies deep within the Sylvian fissure at the junction of the posterior parietal operculum with the insular and retroinsular region (Eickhoff et al. 2006). Further OP 2 lies adjacent to area OP 1, which has been identified as the secondary somatosensory cortex in primates. Eickhoff et al. (2006) claimed that vestibular activations were not found in the human OP 1 region, but we note that their study used galvanic vestibular stimulation, which generally has weaker effects than the CVS used in our study. Jung et al. (2009) used EEG source analysis to confirm opercular origin of the N80 and N110 SEP components, although they suggested that N80 could arise from either the frontal or the parietal operculum. Based on this anatomical and functional evidence, the N80 modulations in our experiment may reflect vestibular-induced changes in

somatosensory processing within the parietal operculum, possibly including the secondary somatosensory cortex.

Traditionally, CVS-induced behavioural effects in brain-damaged patients have been interpreted as modulations of supramodal spatial attention (Vallar et al. 1990). However, our SEP results do not strongly support an attentional interpretation. Attentional modulations of SEPs are classically found for the N140 component and are not generally reported in components occurring before 100 (Eimer and Forster 2003). Additionally, we did not find any reliable evidence of CVS-induced modulation of VEPs. Interestingly, Bense et al. (2001) showed that vestibular stimulation bilaterally deactivated the occipital visual cortex (BA 17–19). We failed to find any modulation of visual components consistent with either increased or reduced visual excitability.

Could our effects be due to some aspect of the CVS procedure, other than vestibule-somatosensory interactions? Evoked potentials were recorded not during CVS itself, but a few minutes after irrigation. By this time, nystagmus and vertigo have subsided (Miller et al. 2000; Ngo et al. 2007, 2008). However, it has been demonstrated the activation of vestibular projections lasts over the oculomotor reflex for about 30 min. We can therefore exclude effects of both vestibular-induced gaze modulation and the transient non-vestibular effects of the irrigation (e.g., the cold sensation in the ear). Moreover, the specific enhancement of the N80 SEP component cannot be explained by general, non-vestibular aspects of the stimulation.

Our study is preliminary, and limited in several ways. First, the number of participants is small, though comparable with other somatosensory evoked response studies (Simões and Hari 1999). Our results therefore require replication in a larger study. Because of its small size, our study therefore has only low statistical power to detect effects of CVS. While this does not undermine positive findings, it means that absence of statistical significance should be treated with particular caution. For example, our study cannot conclude that SEP components other than the N80 are not modulated by CVS since the probability of a type II error is high in a small study. In the same way, we urge caution in interpreting the absence of significant VEP modulations. Thus, our results cannot exclude the possibility that vestibular stimulation has some non-specific, supramodal effects, in addition to its specific somatosensory effects. Second, we used a limited number of electrodes and so may have missed some cortical sources. However, our scalp topographies (see Fig. 1a) show sensitivity over areas classically associated with somatosensory evoked potentials. Third, the order of the experimental conditions (Pre-CVS and Post-CVS) was obligatorily the same in all participants. Fourth, and finally, we have stimulated only the left ear with CVS, and the left hand with median nerve stimulation. We chose this combination because the strongest effects of vestibular stimulation have generally been found when stimulating the left vestibular organ, and thus the right hemisphere (Vallar et al. 1990, 1993; Ngo et al. 2007). Future, larger studies might factorially combine the side of vestibular and somatosensory stimulations to investigate this question more fully.

To summarize, our findings demonstrate that vestibular stimulation modulates somatosensory cortical processing. We found enhancement of the bilateral N80 SEP component, which has been localised to the parietal operculum, including the secondary somatosensory cortex. The convergence of vestibular and somatosensory information in the parietal operculum suggests that vestibular modulation of somatosensory perception (Ferrè et al. 2011a, b) may occur in the circuits that generate this SEP component. More precise localisation is required to identify whether multisensory interactions within PIVC are sufficient to explain our result or whether vestibular input also interacts with somatosensory areas other than PIVC that may have been missed in this small-scale study. Nevertheless, we believe our results provide the first evidence linking brain structure to function for the interaction between vestibular and somatosensory systems.

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