nature neuroscience

The proprioceptive representation of eye position in monkey primary somatosensory cortex

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The cerebral cortex must have access to an eye position signal, as humans can report passive changes in eye position in total darkness, and visual responses in many cortical areas are modulated by eye position. The source of this signal is unknown. Here we demonstrate a representation of eye position in monkey primary somatosensory cortex, in the representation of the trigeminal nerve, near cells with a tactile representation of the contralateral brow. The neurons have eye position signals that increase monotonically with increasing orbital eccentricity from near the center of gaze, with directionally selectivity tuned in a Gaussian manner. All directions of eye position are represented in a single hemisphere. The signal is proprioceptive, because it can be obliterated by anesthetizing the contralateral orbit. It is not related to foveal or peripheral visual stimulation, and it represents the position of the eye in the head and not the angle of gaze in space.

Humans can report passive changes in eye position in total darkness[1], indicating that the cerebral cortex must have access to an eye position signal. Furthermore, visual responses in posterior parietal[2–4] and prestriate[5] cortex, as well as the superior colliculus[6] and even the lateral geniculate nucleus[7], are modulated by eye position. The source of the cortical and subcortical eye position information is unknown. In the 19th century, Wundt and Hering independently postulated that the sense of eye position arose from sensors in the eye muscles, a signal now called 'inflow'. Helmholtz, on the other hand, thought that the muscle sense was too inaccurate to use for calibrating vision. He postulated that the 'effort of the will' (Willensanstrengung), more recently called 'outflow', 'corollary discharge' or 'efference copy', was used by the brain to determine eye position (see ref. 8 for a description of this debate).

Researchers have assumed for decades that the cortical eye position signal arises from an outflow signal[2,3], although the possibility that this signal could arise from proprioception has also been raised[9]. However, neither a proprioceptive representation of eye position nor corollary discharge of a motor command for eye position (as opposed to one for saccadic eye displacement[10,11]) has been demonstrated unambiguously in the cerebral cortex.

There is a somatotopic representation of the entire body in the primary somatosensory cortex (SI), called the 'homunculus' in the human. In the homunculus and its equivalent in the monkey (the 'simiunculus'), the leg is represented on the medial surface of the cortex, the hand more laterally on the surface of the cortex and the face most laterally. There are parallel representations of superficial and deep sensors. In particular, muscle spindles, the receptors that describe muscle length, are represented in area 3a, in the depth of the central sulcus[12].

Until now there has been a hole in the homunculus where the eye muscles should be. The monkey extraocular muscles do have a sensory structure, the palisade ending or myotendinous cylinder, which resembles the skeletal muscle spindle[13–15]. This structure presumably measures the length of the muscles, and by extension the position of the eye in the orbit. Like the skeletal muscle spindles, the myotendinous cylinder is a fusimotor structure whose sensitivity is controlled by specialized muscle fibers that adjust the tension of the stretch receptor. Neurons in monkey eye muscles project to the spinal trigeminal nucleus and the nucleus cuneatus[16], but their cortical projection has never been determined. The most comprehensive study of area 3a in the macaque[12] found no signals from the ophthalmic branch of the trigeminal nerve at all, but did not study the possible contributions of the extraocular musculature. Here we report a sensory representation of eye position in monkey primary somatosensory cortex, dependent on signals from the contralateral orbit.

RESULTS

Neurons in somatosensory cortex represent eye position

We trained two rhesus monkeys (*Macaca mulatta*) to fixate on a spot of light and make saccades for liquid reward[17]. We implanted a 2-cm recording cylinder over the somatosensory cortex, and searched in the cylinder until we found multiunit activity with tactile receptive fields on the monkey's brow. We then advanced the electrode deep into the sulcus until we found multiunit activity that had eye position sensitivity, at a depth of 9.5 mm or more. The eye position neurons were in a thin layer, with only one or two cells isolable in each electrode penetration. We found eye position cells in a 4 mm × 4mm area of the cylinder. Once we found the area, we could

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Received 5 January; accepted 8 March; published online 1 April 2007; doi:10.1038/nn1878

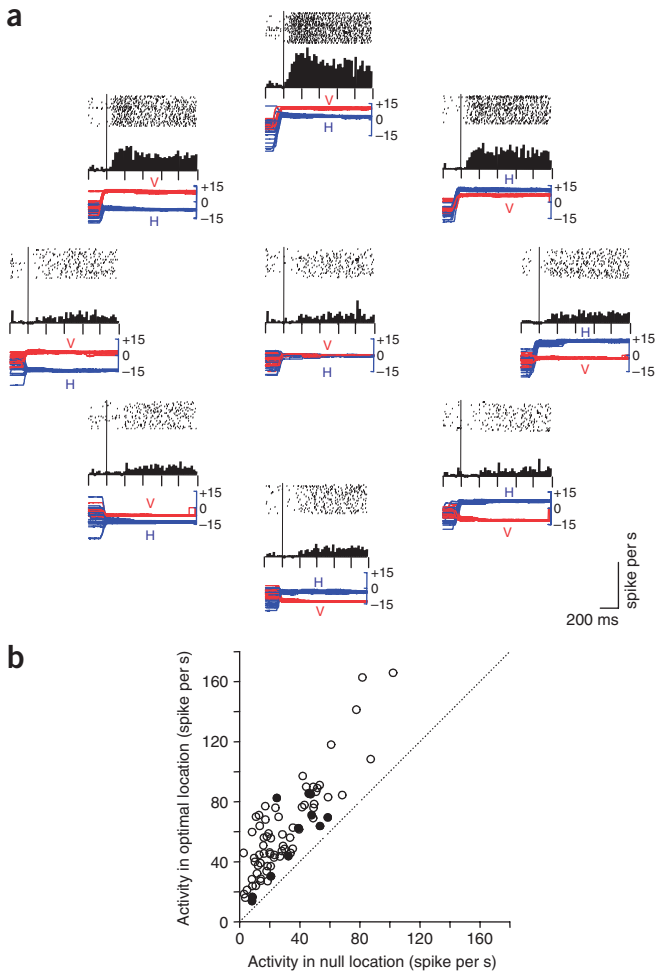

Figure 1 Activity of a tonic eye position neuron in monkey SI. (**a**) Nine raster diagrams, one at the center of the orbit and eight others positioned radially 15 ° from the center. The position of the raster is related to the position of the eye in the orbit. Each tick is an action potential, and each line is a trial. Lines are synchronized on the end of the foveating saccade. Because the trial began with the appearance of the fixation point, the eye position before the saccade was uncontrolled. The histograms beneath each raster average, without smoothing, the activity of the raster above, with a bin width of 25 ms. Eye positions for each trial are superimposed beneath each raster (horizontal, blue; vertical, red). (**b**) Mean activity for the first 1,000 ms after the end of the foveating saccade in the optimum (ordinate) plotted against mean activity in the null (abscissa) direction of each of 76 cells with a significant ($P < 0.05$ by *t*-test) difference between average activity over the first 1,000 ms after the end of the saccade in the optimal and null directions (open circles: cells fit to a Gaussian with $R^2 \geq 0.7$; filled circles: cells fit to a Gaussian with $R^2 < 0.7$).

reliably find eye position neurons in almost every penetration. Not every penetration included neurons with obvious tactile receptive fields on the face.

We tested the eye position sensitivity of each neuron by requiring the monkey to fixate on nine different points, one at the center of the screen and the others in a radial array of points 15° from the center of the screen (example in **Fig. 1a**). We found 88 neurons (33 from monkey C, 55 from Monkey W) that informally appeared to have eye position sensitivity. Of these, 81 neurons had a significant ($P < 0.05$) main effect of eye position by ANOVA (66 significant at a $P < 0.01$ level). Seventy-six neurons had a significant difference ($P < 0.05$) between maximal and minimal responses by *t*-test (65 significant at $P < 0.01$ level). We plotted the optimum eye position response against the minimum response for every cell with a significant response ($P < 0.05$ by *t*-test, **Fig. 1b**).

We did T1-weighted magnetic resonance imaging (MRI) of a tungsten electrode that was left in place at the site of an eye position neuron in each monkey (**Fig. 2a**). The eye position area was in the depth of the central sulcus, in a position consistent with area 3a and the representation of the ophthalmic branch of the trigeminal nerve[12].

All the cells were tuned for a particular radial direction of movement. Sixty-five cells fit a Gaussian distribution for the direction of eye position from the center of gaze with $R^2 > 0.7$, which was significant ($P < 0.05$ level, **Fig. 2b**). All of the directions of eccentric position were represented in a single hemisphere without any apparent directional preponderance. We plotted the tuning maxima of the Gaussian for all cells which fit a Gaussian with $R^2 > 0.7$ (**Fig. 2c**). We were unable to discern any topographic organization or map of orbital position preference.

All position signals increased monotonically with increasing orbital eccentricity. We studied the amplitude tuning of 12 cells using 7 points along their preferred direction. All cells had a measurable baseline activity in the off direction, and began to increase their discharge as the eye passed near the midline of the orbit (**Fig. 2d**). We determined this

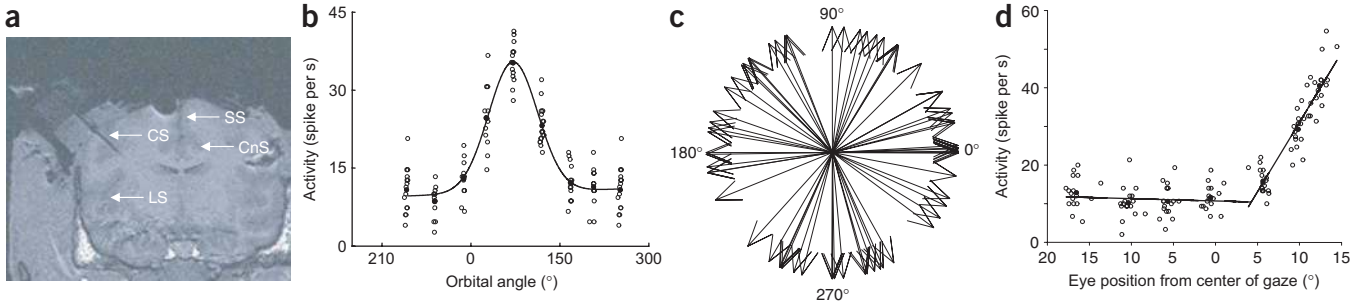

Figure 2 Location and tuning of eye position neurons. (**a**) T1-weighted coronal MRI of a tungsten electrode at the site of an eye position neuron in S1. The electrode tip is in the floor of the central sulcus (arrow, CS; LS, lateral sulcus; CnS cingulate sulcus; SS sagittal sulcus). A titanium screw MRI artifact can be seen medial and superior to the electrode. (**b**) Example of Gaussian tuning of directional activity. Each open symbol is the mean activity of the neuron in one trial in the first 1,000 ms after the foveating saccade, and the filled circles are the averages ($R^2 = 0.82$, $P < 0.001$). (**c**) Optimal position tuning direction, calculated as the peak of the Gaussian to which the neuronal responses (mean of activity in first 1,000 ms after the saccade) were fit, for each neuron ($n = 65$) that fit a Gaussian with $R^2 > 0.7$. (**d**) Example of linear tuning with eccentric position of a single cell ($R^2 = 0.8261$, slope = 3.59).

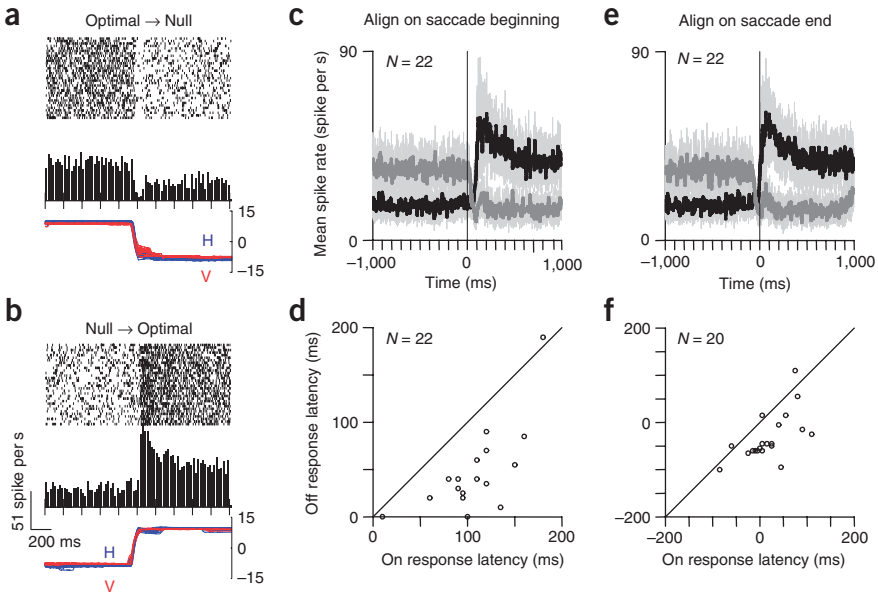

Figure 3 Tonic and phasic responses of S1 eye position neurons. (**a**) Single neuron example. The monkey makes a saccade from an optimal to a null orbital position. Horizontal (blue) and vertical (red) eye position traces are superimposed. The rasters were synchronized on the end of the saccades. (**b**) The monkey made a saccade from a null to an optimal position. (**c**) Average responses (dark) and 95% confidence limits (light) of neurons when the monkey made a saccade from the null to optimal (black) and the optimal to null (gray) orbital positions. Averages were calculated in 5-ms bins, not smoothed, and synchronized on the beginning of the saccade (**d**) Latency of off-response from saccade beginning (ordinate) plotted against on-latency of on-response from saccade beginning ($N = 22$, $R^2 = 0.43$, $P = 0.0009$). (**e**) Average response, displayed as in b, calculated from end of saccade. (**f**) Latency of off-response from saccade end (ordinate) plotted against on-latency of on-response from saccade end ($N = 20$, $R^2 = 0.37$, $P = 0.0043$). For two cells in the sample averaged in **c**, we were unable to determine a satisfactory latency.

inflection point by eye, and fit different straight lines to each segment. The fit to a straight line in the responsive region was significant for 10/12 cells, with the fits of 8 cells significant at $P < 0.0001$. There was no correlation between eye position and signal in the off region (**Supplementary Table 1** online).

The signal was tonic in that it was maintained for as long as we required the monkey to fixate, with the exception of one neuron in which the activity began to decay after 600 ms of fixation. However, roughly 70% of the neurons also exhibited a phasic excitation in addition to the tonic signal when the monkey made a saccade toward the preferred orbital position of the neuron, and a phasic suppression of response when the monkey made a saccade away from the preferred position (**Fig. 3**). We measured the phasic (0 to 300 ms from the end of the saccade) and tonic (300 to 1,000 ms from the end of the saccade) responses of 22 neurons as the monkey made saccades from a consistent fixation point. In this sample, 6/22 had no phasic responses. Suppression had a much shorter latency than excitation: the mean latency of the off-suppression was 30 ms from the beginning of the saccade (**Fig. 3b**) and the mean latency of the on-excitation was 80 ms from the beginning of the saccade. The latencies were correlated (**Fig. 3e**, $N = 22$, $R^2 = 0.43$, $P = 0.0009$). When calculated from the end of the saccade (**Fig. 3c**), the mean latencies were 5 ms before the end of the saccade for the off-suppression and 30 ms before the end of the saccade for the on-excitation. Latencies were correlated with this alignment as well (**Fig. 3f**, $N = 20$, $R^2 = 0.37$, $P = 0.0043$).

The eye position signal is sensory and not a motor corollary

The location of the eye position signal, in a region of somatosensory cortex dedicated to muscle proprioception, suggests a proprioceptive origin. However, the eye position signal could also have arisen from an efference copy or corollary discharge of a motor fixation signal. To distinguish between these two alternatives, we temporarily anesthetized the contralateral orbit by a retrobulbar block while we recorded the activity of an eye position neuron[18] (**Fig. 4**). This procedure caused ptosis, conjunctival and corneal anesthesia, and a complete or partial paralysis of the eye, but had no effect on the movement of the contralateral eye. The monkey continued to make accurate eye movements in one eye despite the paralysis of the other eye. When the eye

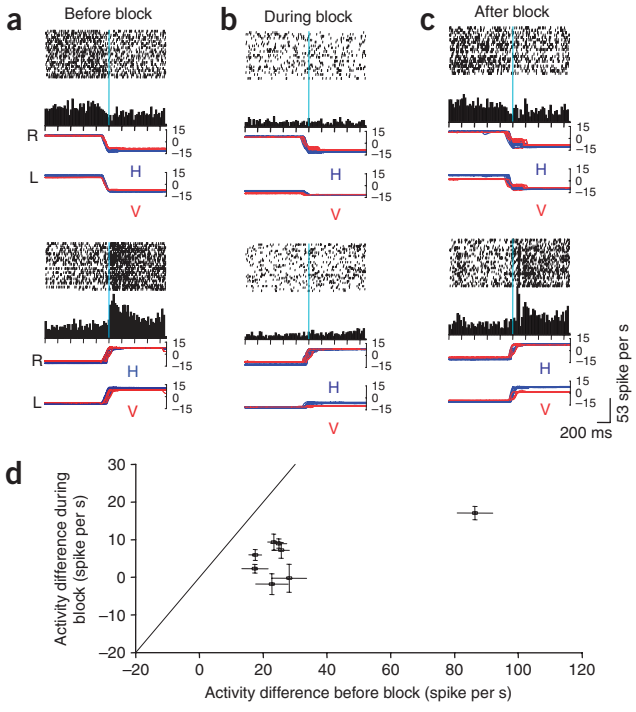

Figure 4 Effect of retrobulbar block on neural activity. (**a**) Neural activity when the monkey made a saccade from on-region to off-region (top) and off-region to on-region (bottom). Right (R) and left (L) eye traces are shown (eye positions: horizontal, blue; vertical, red) . (**b**) Loss of activity after retrobulbar block of the left eye. The left eye moved little, if at all, although the right eye moved normally. (**c**) Recovery of neural activity when the eye regains movement, even though the ptosis was still present. The activity was less than before the block, but the eye had not entirely recovered. (**d**) Difference in activity before and after a saccade before the block (ordinate) plotted against the difference in activity before and after a saccade during the block (abscissa). Error bars are s.e.m. All points were significant by *t*-test, the largest *P* value being 5.875×10^{-6}.

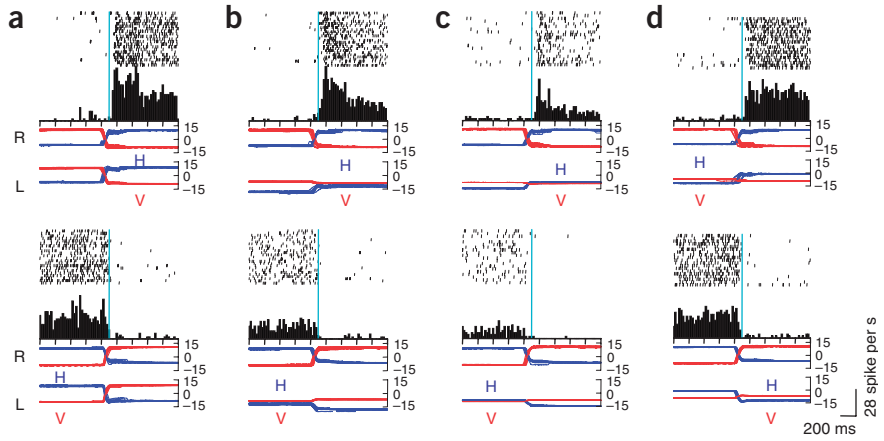

Figure 5 Gradual effect of partial block on the eye position signal. (**a**) Neural activity when the monkey made a saccade from an off-position to an on-position (top), and from an on-position to an off-position (bottom) before block. Labels as in **Figure 4**. (**b**) . Activity soon after the block, with a partial, but significant ($P = 2.7169 \times 10^{-6}$ by *t*-test) decrease. (**c**) Activity later, with a further partial, but significant ($P = 3.0551 \times 10^{-10}$ by *t*-test), decrease. (**d**) Recovery of eye movement and signal. This activity is not significantly different from the activity before the block ($P = 0.5986$, by *t*-test).

movement was completely or nearly abolished, the eye position signal was completely abolished (**Fig. 4b**). When the block wore off, the eye began to move again, and the eye position signal returned (**Fig. 4c**). We did this experiment on four cells in each of two monkeys. When the eye movement was reduced, but not totally abolished, the eye position signal was diminished (**Fig. 4d**). For each experiment, there was a significant difference in the responses before and during the block ($P = 5.8 \times 10^{-6}$ for the worst case, $P = 6 \times 10^{-9}$ for the median case).

Of course, one possibility is that the procedure agitated the monkey enough that we lost the cell. This was never the case. For some cells, the block took effect over a time course of several minutes, and the signal gradually decreased. It then returned after the eye movements recovered (**Fig. 5**). In this example, the block predominantly affected vertical movements, but the cell was tuned in a horizontal direction, and neither the horizontal eye movements nor the signal were completely affected. However, the clear maintenance of activity after the onset of the block, and its subsequent diminution and recovery demonstrate that artifactually losing the cell as a result of the block is not an explanation for the effect of the block (**Supplementary Table 2** online).

The eye position is not visual or related to gaze in space

The activity of the cells was dependent on the angle of the eye in the orbit and not on the angle of gaze in space. For nine cells, we rotated the monkey's head roughly 10° so that a gaze angle that originally was related to an ineffective eye position (**Fig. 6a**) now was related to an effective eye position (**Fig. 6b**). The signal that had previously been low was increased, as the eye was now in a more eccentric position in the orbit, even though the position of the eye in space was unchanged. For the far eccentric eye position, the monkey found it difficult to achieve fixation, so the eye position is not as regular as it is for the more central fixation. Rotating the head moved the eye out of the optimum position for the coil, so the eye calibration was unreliable. The population

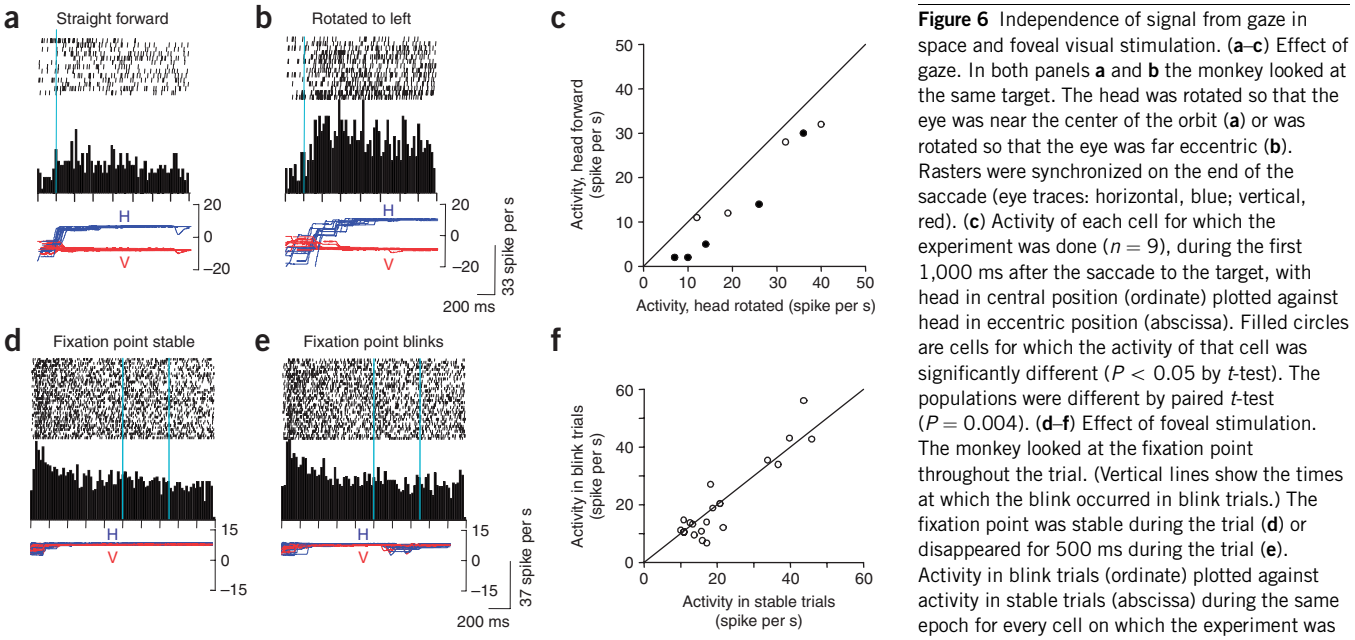

Figure 6 Independence of signal from gaze in space and foveal visual stimulation. (**a**–**c**) Effect of gaze. In both panels **a** and **b** the monkey looked at the same target. The head was rotated so that the eye was near the center of the orbit (**a**) or was rotated so that the eye was far eccentric (**b**). Rasters were synchronized on the end of the saccade (eye traces: horizontal, blue; vertical, red). (**c**) Activity of each cell for which the experiment was done ($n = 9$), during the first 1,000 ms after the saccade to the target, with head in central position (ordinate) plotted against head in eccentric position (abscissa). Filled circles are cells for which the activity of that cell was significantly different ($P < 0.05$ by *t*-test). The populations were different by paired *t*-test ($P = 0.004$). (**d**–**f**) Effect of foveal stimulation. The monkey looked at the fixation point throughout the trial. (Vertical lines show the times at which the blink occurred in blink trials.) The fixation point was stable during the trial (**d**) or disappeared for 500 ms during the trial (**e**). Activity in blink trials (ordinate) plotted against activity in stable trials (abscissa) during the same epoch for every cell on which the experiment was done ($N = 22$, **f**). Each symbol is the averaged activity of a single neuron during the 500-ms blink (**d**) or its 500-ms equivalent epoch during the stable trials. There was no difference between them ($P = 0.949$, by paired *t*-test).

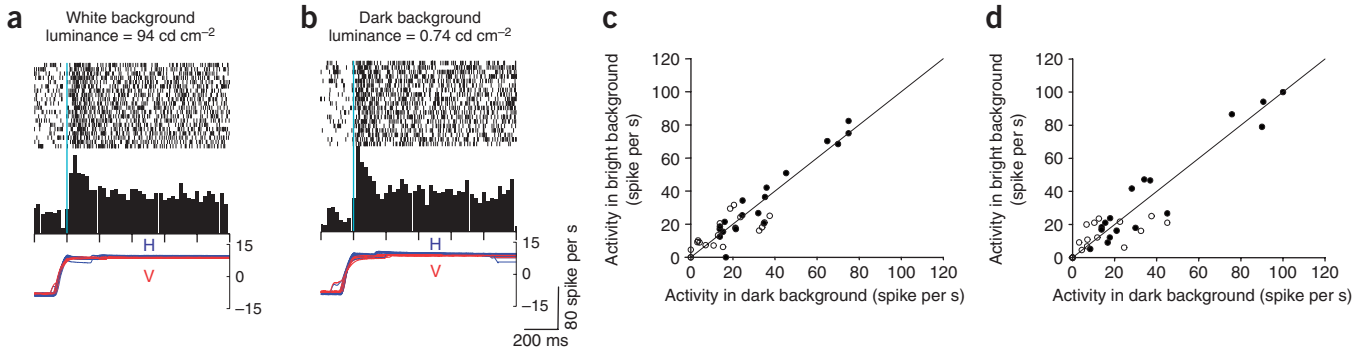

Figure 7 Independence of activity from background illumination. (**a**) The monkey made a saccade from a null to an optimal position across a bright background (94 cd m^{-2}) on which the various details of the recording booth, such as the screen border and the field coils, were highly visible (eye traces: horizontal, blue; vertical, red). (**b**) The monkey made the same saccade as in **a** across a dark background (0.74 cd m^{-2}) on which the details of the recording booth were obscure. There was no difference in the response for the first 1,000 ms after the end of saccade ($P = 0.3528$, by paired *t*-test). (**c**) Tonic activity (mean activity from 300 to 1,000 ms after end of saccade) of 19 neurons at optimal (filled circles) and null (open circles) orbital positions in a bright environment (ordinate) plotted against activity in a dark environment (abscissa). There is no difference between saccades across bright and dark background ($P = 0.924$ by paired *t*-test). (**d**) Phasic activity (mean activity from 0 to 300 ms after end of saccade) for the 17 of the 19 neurons in **b** which had phasic activity, plotted under the same conditions as in **b** ($P = 0.9323$ by paired *t*-test).

showed a significantly ($P = 0.004$ by paired *t*-test) higher response in the rotated case (**Fig. 6c**). Five of the cells for which we did this experiment individually showed a significantly higher response after head rotation ($P < 0.05$ by *t*-test), and the others showed a trend in the direction (**Fig. 6c**).

The cells were not visually responsive. For 22 cells, we searched for a foveal response by extinguishing the fixation point for 500 ms while the monkeys attempted to maintain fixation. The monkeys' fixation wavered slightly near the end of the epoch, but compared to an identical epoch in which the fixation point remained lit (**Fig. 6d,e**), there was no significant difference in the average neuronal response of a single cell ($P = 0.3528$) nor was there for the population (**Fig. 6f**, $P = 0.95$ by paired *t*-test). The monkey's eye position began to waver slightly during the end of the blink period, but this small change in eye position did not affect the neural response.

We could not find any evidence for peripheral visual responsiveness. For 19 cells, we had the monkeys make saccades to a black fixation point across a bright white background of average luminance 94.03 cd m^{-2} (**Fig. 7a**) and on randomly intermixed trials to a white fixation point across a black background of average luminance 0.74 cd m^{-2} (**Fig. 7b**). These different visual environments had no effect on the eye position responses ($P = 0.4078$ by paired *t*-test). Across the population background, illumination made no difference for either the phasic component of the response (**Fig. 7c**, $P = 0.924$ by paired *t*-test) or the tonic component of the response (**Fig. 7d**, $P = 0.9323$ by paired *t*-test). We never found a visual response by presenting anecdotal visual stimuli on the screen, moving a hand-held light across the screen or approaching the monkey with a treat.

DISCUSSION

In these experiments, we have identified a proprioceptive representation of the position of the contralateral eye in the primary somatosensory cortex of the monkey. Here we discuss the nature of signal and its implication for the neural processes underlying spatial perception and action.

Eye position as a somatic sensation

We found the neurons that represent eye position in the depth of the central sulcus, in the lateral area dedicated to the representation of the ophthalmic branch of the trigeminal nerve[12]. This area is consistent with area 3a, the portion of the somatosensory cortex in which the skeletal muscle spindles are represented[19]. The eye position area is relatively broad, accessible from a surface area 4 mm × 4 mm. Some, but not all, of the electrode tracks had easily obtainable tactile receptive fields on the brow. This relatively large area is in keeping with the observation that compared with overlying area 3b, area 3a has a much larger and more diffuse topographic map[12].

The signal arises from proprioceptors in the contralateral orbit. When the ocular paralysis was complete, the signal was totally abolished. When the ocular paralysis was partial, the signal was partially abolished. We did not inject the ipsilateral eye, but neurons in area 3a can be driven exclusively from the contralateral body, even in anesthetized monkeys[12]. One possible explanation for our data is that the process of inducing the block made the animal so agitated that we lost the neuron. This is unlikely for two reasons. First, the response never disappeared entirely; instead the neurons maintained a weak background signal. Second, when the block took a few minutes to take effect, the neurons gradually lost their eye position signal and gradually redeveloped the signal as the block wore off. Retrobulbar block does not abolish the signal in the abducens nucleus[18], which projects to the anesthetized eye, and because the monkey continued to make eye movements with its normal eye, the block could not have affected any of the more central motor processes associated with eye movements. The only possible interpretation of these data is that the signal arises from some proprioceptive mechanism in the eye, for which the best candidate is the fusimotor myotendinous cylinder.

The eye position response is consistent with a fusimotor response. The neurons have a relatively short latency phasic response and a slower tonic response that could easily arise from dynamic and static gamma signals[20], both of which are present in eye muscles[13]. It is possible that the phasic response could also represent a half-wave–rectified representation of eye velocity, but we do not have enough data to evaluate that possibility.

Little is known about the cortical processing of signals from fusimotor receptors. There have been few recordings from the cortical representation of skeletal muscle spindles, let alone oculomotor proprioceptors in awake, behaving monkeys. During passive, one-dimensional ramp flexion and extension movements and maintained positions of the ankle, area 3a neurons show a combination of phasic and tonic activity[21] with a time course similar to that which we found

in the eye position neurons. A separate study[22] showed a neuron in area 3a that responded during wrist flexion, was slightly suppressed by extension, and could be excited by cutaneous pressure over the bellies of two functionally linked muscles, the extensores carpi radialis brevis and longus.

The neurons show a wide distribution of tuning maxima. This is in marked contrast to ocular motor neurons, whose isofrequency curves are parallel[23], suggesting that their tuning curves in polar coordinates are also bunched tightly together. Assuming that the receptor tuning maxima should be aligned with the muscles whose length they monitor, if the cortical neurons received signals from only one muscle, one would expect that their tuning maxima should also be bunched together, similar to those of the motor neurons. The wide and almost uniform distribution of tuning maxima suggests that the signal on the cortical eye position neurons arises from the integration of the signals from several muscles. We suggest that this integration enables area 3a to provide a richer, more accurate and more nuanced representation of limb, neck and eye position than would be provided by the representation of single or functionally linked muscles alone.

The function of the proprioceptive eye position signal

Much evidence suggests that oculomotor proprioception is not necessary for on-line processing of visual space for action. Thus, sectioning the ophthalmic branches of both trigeminal nerves has no effect on single- or even double-step saccades, nor does it derange the spatially accurate saccades made between the flash of a target and the derangement of eye position evoked by electrical stimulation of the superior colliculus[24]. Similarly, lesions of the trigeminal nerve do not derange the accuracy of limb movements, even for open-loop pointing in which the target, but not the limb, is visible[25]. The neural processes underlying spatially accurate movement presumably compensate for saccadic eye movement by using the more rapid process of a corollary discharge of the saccade, which can even precede the physical eye movement[10], giving enough time for neural calculation. However, other studies do raise the possibility of a small contribution to movement and perception by oculomotor proprioception[26].

Corollary discharge, however, must be calibrated for the corollary signal to represent the actual movement reliably. Some movements can be calibrated visually, but not all movements are made to visual targets. Consistent with this, oculomotor proprioception is critical for long-term calibration of the motor system[27]. Thus the accuracy of vergence and saccadic movements gradually decays after trigeminal section in monkeys with muscular paresis[28] that had compensated stably before the nerve section. In humans, open-loop pointing is not affected immediately by strabismus surgery[29] or the injection of botulinum toxin into eye muscles[30], both of which would be expected to disrupt oculomotor proprioception. However, open-loop pointing becomes less accurate over a period of weeks. This slow decay of the accuracy of the limb and eye motor systems as a result of proprioceptive damage suggests that oculomotor proprioception is needed for calibration[27].

An obvious physiological use for the oculomotor proprioceptive signal that we have discovered would be to provide the eye position signal in the parietal cortex, which could then be used for calibration[27] or for the sensory perception of eye position[1]. This signal manifests in two different ways: a pure, nonlight-sensitive eye position signal[2] and an eye position modulation of visual responsiveness, noted both in neurons with foveal[2] and peripheral[3] receptive fields. The foveal visual and nonvisual fixation neurons respond after saccades with phasic and tonic responses, with the activity often taking several hundred milliseconds to decline to the tonic level. The time course of these responses is notably different from that of the signal on oculomotor neurons[31]. The oculomotor neuronal signal also has two components, but one is tightly locked to eye position and the other is tightly locked to eye velocity, with the phasic velocity component leading the eye velocity by a few milliseconds. This is unlike the activity of the parietal eye position neurons; their activity resembles both the eye position signal we have described in SI and skeletal muscle spindle afferents. Both of these signals have a much longer phasic component, which lags effector velocity. It is therefore not unreasonable to speculate that the parietal eye position signal arises from eye proprioceptors, just as the head position signal that modulates parietal visual responses probably arises from neck proprioceptors[32].

Many computational models of spatial processing for action rely on eye-position–modulated visual responses, the gain fields, as an intermediate step in the neural computation of target position[33–35] in a supraretinal coordinate frame. However, if the parietal eye position signal arises from oculomotor proprioception, then the gain fields would be unimportant, as would be oculomotor proprioception itself, for on-line spatial processing for action, and instead would have a slower, more calibratory and, perhaps, perceptual role. Nonetheless, the brain must have a rapid and accurate estimate of eye position, both to coordinate visually guided movements of the limbs and to compensate for the dissonance in retinal and oculomotor vectors for eye movements that originate far from the center of gaze[36]. The corollary discharges of eye position and displacement may be adequate for providing a stable representation of the world for immediate action despite a moving eye[10,11,24,37].

METHODS

General. Both the New York State Psychiatric Institute and Columbia University Medical Center Institutional Animal Care and Use Committees approved all animal procedures and certified their compliance with the *NIH Guidelines for the Care and Use of Experimental Animals*. We prepared monkeys for physiological recording using standard sterile surgical techniques, ketamine induction and isofluorane endotracheal anesthesia, and by implanting a head-holding socket, 2-cm recording chambers (positioned at 20 mm A, 27 mm L) and a subconjunctival magnetic search coil in each eye[38]. We trained the monkeys to make saccades and perform visual fixation for liquid reward, using the REX system for behavioral control[39], and monitored eye position and the time-stamping of digitized action. A Hitachi CPX275 LCD projector running the VEX open GL-based graphics system (available by download from lsr-web.net) rear-projected stimuli onto a screen. We measured image luminance using a Minolta photometer. We recorded neurons using glass-coated tungsten electrodes (Alpha-Omega), and commercially available amplification (FHC or Alpha-Omega) and filtering (Krohn-Heit) equipment. We measured eye position using a two channel Riverbend Phase Detector. Data from the recording electrodes were sorted and digitized by the MEX system (available by download from lsr-web.net)[40].

Retrobulbar blocks. Using a described technique[18], we held down the animal's eyelid and passed an Atkinson needle through the upper lateral lid into the upper retrobulbar space. We then injected 2 ml of 2% lidocaine without epinephrine and withdrew the needle. If necessary, we injected another 0.5 ml of 2% lidocaine into the lower retrobulbar space. The animals tolerated the procedure. Every second during the procedure they received a free liquid reward, which they consumed avidly, despite having a needle in their subconjunctival space. Once we withdrew the needle, they resumed working immediately. The monkeys worked as efficiently when only one eye moved as they did when both eyes moved. Presumably, the lidocaine in the orbit prevented their feeling any possible orbital sensation, and the ptosis prevented diplopia, which might have been an uncomfortable result of the retrobulbar block.

Head rotation. The monkeys sat in standard Crist primate chairs, with the head secured to a head post, which was itself secured to the chair with a large hex nut. To rotate the head, we attached a pair of vice-grip pliers to the post, loosened the nut, rotated the head with the pliers, and tightened the nut again. This prevented vertical movement of the head and resulted in a head rotation between 10° and 15°. It is important to emphasize that the rotation, centered on the neck and not on an eye, moved the eye coil out of the portion of the magnetic fields for which it had been calibrated, and the eye position in the eccentric head position was measured only approximately.

MRI imaging. We first recorded neural activity, locating a cell with an eye position signal. The electrode was cut so that it was shorter than the height of the recording cylinder, and the guide tube removed with the electrode still in place. We then tranquilized the monkey with ketamine and atropine, removed it from the primate chair, transported it to the MRI lab, anesthetized it with endotracheal isofluorane, and put it in a Kopf MRI compatible stereotaxic instrument. The images were taken with a GE Excite HD 1.5 T scanner, and we analyzed the data with DicomWorks.

Data analysis. We wrote the analysis programs in Matlab, using curve-fitting and statistics toolboxes (MathWorks). Unless otherwise stated, all neural values were calculated from the first 1,000 ms after the end of the saccade. We determined neural latencies using our modified Poisson method[41].

Note: Supplementary information is available on the Nature Neuroscience website.

ACKNOWLEDGMENTS

We are grateful to H. Eggers for teaching us how to perform retrobulbar blocks, G. Duncan for electronic and computer support, Y. Pavlova for dedicated animal maintenance, M. Osman and G. Asfaw for veterinary care, S. Dashnaw and J. Hirsch for MRI, and L. Palmer for facilitating everything. This research was supported by grants from the James S. McDonnell Foundation, the US National Eye Institute (1 R01 EY014978-01 and 1 R24 EY015634-01), and the Whitehall, Keck, Dana and Kavli Foundations.

AUTHOR CONTRIBUTIONS

X.W. and M.W. recorded all the neural activity, did much of the data analysis and made the figures. I.S.C. wrote many of the data analysis programs and did some of the preliminary data analysis. M.E.G. dreamed up the project, worked out the retrobulbar block technique with H. Eggers and supervised the entire project.

COMPETING INTERESTS STATEMENT

The authors declare no competing financial interests.

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