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Neuronal correlates of subjective sensory experience

Victor de Lafuente & Ranulfo Romo

When a near-threshold stimulus is presented, a sensory percept may or may not be produced. The unpredictable outcome of such perceptual judgment is believed to be determined by the activity of neurons in early sensory cortex. We analyzed the responses of neurons in primary somatosensory cortex, recorded while monkeys judged the presence or absence of threshold stimuli. We found that these responses did not covary with the monkeys' perceptual reports. In contrast, the activity of frontal lobe neurons did covary with trial-by-trial judgments. Further control and microstimulation experiments indicated that frontal lobe neurons are closely related to the monkeys' subjective experiences during sensory detection.

A fundamental goal of neuroscience is to understand how sensory experiences arise from activity in the brain. The detection of sensory stimuli is among the simplest perceptual experiences and is a prerequisite for any further sensory processing. Studies on the neuronal correlates of sensory detection showed that, in the case of vibrotactile stimuli, the responses of neurons in primary somatosensory cortex (S1) account for the measured psychophysical accuracy[1]. However, imaging and physiological studies show that, in addition to sensory cortices, areas of the frontal lobe are also active during sensory detection and discrimination[2–5]. This evidence raises an important question: what are the specific functional roles of primary sensory cortices and association areas of the frontal lobe in perception?

We addressed this question by recording from single neurons in S1 and medial premotor cortex (MPC; neurons from this frontal lobe area are involved in decision processes during somatosensory discrimination)[3], while trained monkeys reported the presence or absence of a mechanical vibration of varying amplitude applied to the skin of one fingertip. Here we report that the activity of S1 neurons covaried with stimulus strength, but not with the animals' perceptual reports. In contrast, the activity of MPC neurons did not covary with stimulus strength, but did covary with the animals' perceptual reports. We wondered whether, in addition to these neuronal correlates associated with the animals' perceptual reports, the animals could also perform the detection task if their MPC neurons were activated (artificially) with electrical microstimulation, instead of with the mechanical vibrations delivered to one fingertip. This would provide unequivocal proof that the activity of MPC neurons is directly involved with a specific cognitive function. Psychophysical performance with artificial stimuli was almost identical to that measured with the mechanical stimuli delivered to the fingertips. These results suggest that perceptual judgments arise in the activity of frontal lobe neurons but not in sensory cortices.

RESULTS

Two monkeys (*Macaca mulatta*) were trained to perform a detection task (Methods). In each trial, the animal had to report whether the tip of a mechanical stimulator vibrated or not (**Fig. 1a**). Stimuli were sinusoidal, had a fixed frequency of 20 Hz and were delivered to the glabrous skin of one fingertip; crucially, they varied in amplitude across trials. Stimulus-present trials were interleaved with an equal number of stimulus-absent trials in which no mechanical vibrations were delivered (**Fig. 1a**). Depending on the monkeys' responses, trials could be classified into four types: hits and misses in the stimulus-present condition, and correct rejections and false alarms in the stimulus-absent condition (**Fig. 1b**). Stimulus detection thresholds were calculated from the behavioral responses (**Fig. 1c**).

S1 responses during vibrotactile detection

First, we simultaneously characterized the activity of S1 neurons and the monkeys' psychophysical performance by recording the extracellular spike potentials of single S1 units while the monkeys performed the vibrotactile detection task (Methods). Thus we obtained each monkey's psychometric curve and the spike trains of an S1 neuron in the same trials (**Figs. 1c** and **2a**). The firing rate of this neuron varied smoothly as a function of stimulus amplitude, and no clear modulations in its firing rate could be appreciated during the stimulus-absent trials.

To test whether the responses of S1 neurons accounted for the monkeys' psychophysical performance, we calculated neurometric detection curves and compared them with the psychometric curves (**Fig. 2b–d**). The proportion of 'yes' responses for neurometric curves was defined, for a given amplitude, as the proportion of trials in which the neuron's firing rate reached or exceeded a criterion value[6,7] (Methods). For each neuron, this criterion was chosen to maximize the number of correct responses (**Fig. 2b**). The shape of the mean neurometric curve resulting from the activity of the S1 neurons ($n = 59$) showed close correspondence with the shape of the mean psychometric curve (**Fig. 2c**). Pairwise comparisons of detection thresholds, obtained from logistic fits to the simultaneously obtained neurometric and psychometric data, showed that the detection thresholds of individual S1 neurons were not significantly different from the animals' psychophysical thresholds (**Fig. 2d**; Wilcoxon signed rank

Instituto de Fisiología Celular, Universidad Nacional Autónoma de México, 04510 México, D.F., México. Correspondence should be addressed to R.R. (rromo@ifc.unam.mx).

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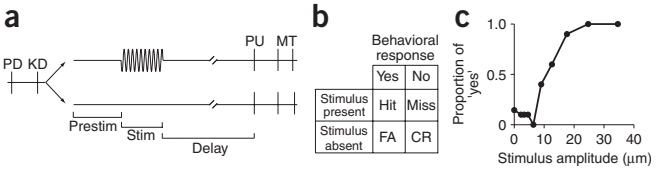

Figure 1 Detection task. (**a**) Trials began when the stimulator probe indented the skin of one fingertip of the right, restrained hand (probe down, PD). The monkey then placed its left, free hand on an immovable key (key down, KD). On half of the randomly selected trials, after a variable pre-stimulus period ("Prestim",1.5 s to 3.5 s), a vibratory stimulus ("Stim", 20 Hz, 0.5 s) was presented. Then, after a fixed delay period ("Delay", 3 s), the stimulator probe moved up (probe up, PU), indicating to the monkey that it could make the response movement (MT) to one of the two buttons. The button pressed indicated whether or not the monkey felt the stimulus (henceforth referred to as 'yes' and 'no' responses, respectively). (**b**) Depending on whether the stimulus was present or absent and on the behavioral response, the trial outcome was classified as a hit, miss, false alarm or correct rejection. Trials were pseudo-randomly chosen; 90 trials were stimulus-absent (amplitude = 0), and 90 trials were stimulus-present with varying amplitudes (9 amplitudes with 10 repetitions each). (**c**) Classical psychometric detection curve obtained by plotting the proportion of yes responses as a function of stimulus amplitude.

test[8], $P = 0.15$) and that the two threshold measures highly covaried (Pearson's correlation coefficient[9], $r = 0.6$, t-test: $P < 0.01$).

S1 responses do not covary with perceptual reports

We then studied whether the activity of S1 neurons covaried with the perceptual 'yes' or 'no' judgments that the monkeys made on a trial-by-trial basis. To test this, we compared the mean normalized activity during hit and miss trials for the near-threshold stimulus, as well as the corresponding activity during correct reject and false-alarm trials in the stimulus-absent condition (Methods). We found no significant differences in the activity of S1 neurons between hits and misses (**Fig. 2e**, upper left panel; t-test: $P = 0.47$), nor between correct rejections and false alarms (**Fig. 2e**, upper right panel: t-test, $P = 0.59$). This indicated that the activity of individual S1 neurons did not predict the monkeys' behavior. To further quantify this, we calculated a choice probability index, which estimates the probability with which the behavioral outcome can be predicted from the neuronal responses[3,4,10]. The results indicated that there were no significant differences between hits and misses or between correct rejections and false-alarm trials (**Fig. 2e**,**f**).

The low choice probability values are consistent with a detection model in which the activity of S1 serves as input to an additional processing stage(s) to determine whether a stimulus has occurred or not. According to this hypothesis, correlation between S1 activity and the final decision about the stimulus presence or absence is highly dependent on the amount of correlated noise among sensory neurons[11]. We found that the mean ± s.e.m. noise correlation coefficient across pairs of S1 neurons was 0.16 ± 0.02 ($n = 51$; see Methods). This amount of correlated noise is similar to those reported in previous studies[12–14], and is also consistent with the near-chance choice probability values reported here. These results further support a detection model in which, to judge the stimulus presence or absence, a central area(s) with internal fluctuations must track the activity in S1.

MPC responses covary with perceptual reports

To test whether the neuronal correlates of the perceptual decisions associated with detection might reside outside S1, we recorded the responses of neurons in the MPC (**Fig. 3a**), a frontal cortical area known to be involved in the evaluation of sensory information and in decision-making processes[3]. We found that, in contrast to the graded dependence on stimulus amplitude observed in S1, MPC neurons responded in an all-or-none manner that was only weakly modulated by the stimulus amplitude (**Fig. 3b**,**c**) but that closely correlated with yes and no behavioral responses (**Fig. 3b**). The mean normalized activity across the 50 MPC neurons was strong and sustained and, with near-threshold stimuli, it was clearly different for hit and miss trials (**Fig. 3d**, upper left panel, t-test: $P < 0.001$; and **Fig. 3e**).

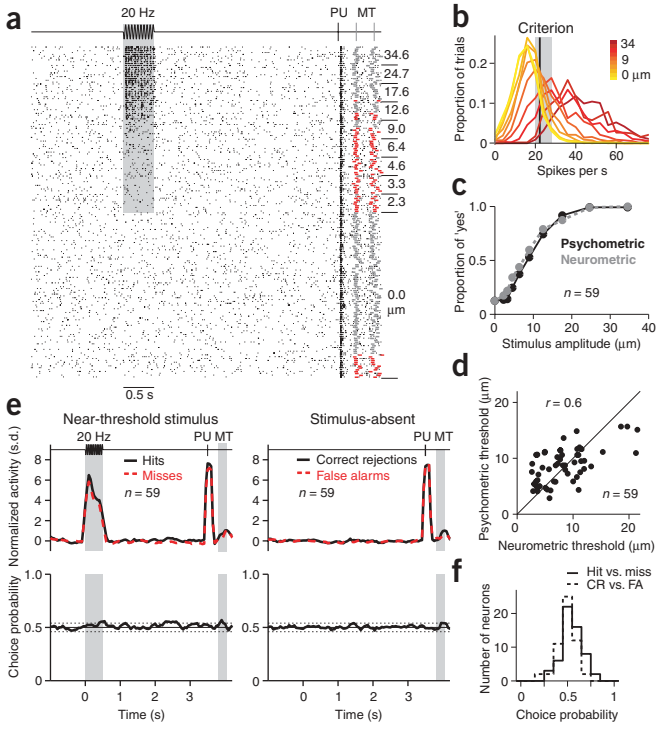

Figure 2 Activity of S1 neurons during the detection task. (**a**) Raster plot of the activity of an S1 neuron during the detection task. Each dot marks the time of spike occurrence, and each row is a trial. Trials are arranged by stimulus amplitude, shown at right. Red markers at the end of the trial denote misses in stimulus-present trials and false alarms in stimulus-absent trials. Gray box marks the time of stimulus presentation. (**b**) Activity distributions of the 59 neurons recorded in S1, grouped by stimulus amplitude (see calibration bar). Black vertical line marks the median criterion value (22 spikes per s) used to produce the neurometric proportion of yes responses for each neuron. Gray box indicates inter-quartile range. (**c**) Mean psychometric and neurometric detection curves (590 trials for each stimulus amplitude data point; 5310 trials for zero-amplitude data point). (**d**) Comparison of psychometric and neurometric detection thresholds, obtained from logistic fits (data not shown) to the proportion of yes responses for the neuronal and behavioral data obtained simultaneously. Diagonal marks the identity line; correlation coefficient, $r = 0.6$. (**e**) Comparison of normalized neuronal population activity (s.d., standard deviation) during hits and misses for near-threshold stimuli, and during correct rejections and false alarms in stimulus-absent trials. Lower panels show the choice probability index as a function of time. Dotted lines mark significance levels (Methods). (**f**) Distributions of indices across the population of S1 neurons, calculated between the activity of hit versus miss trials (mean ± s.e.m.: 0.54 ± 0.02) and correct rejection versus false alarm trials (mean ± s.e.m. = 0.50 ± 0.02 s.e.). n, number of neurons; CR, correct rejection; FA, false alarm.

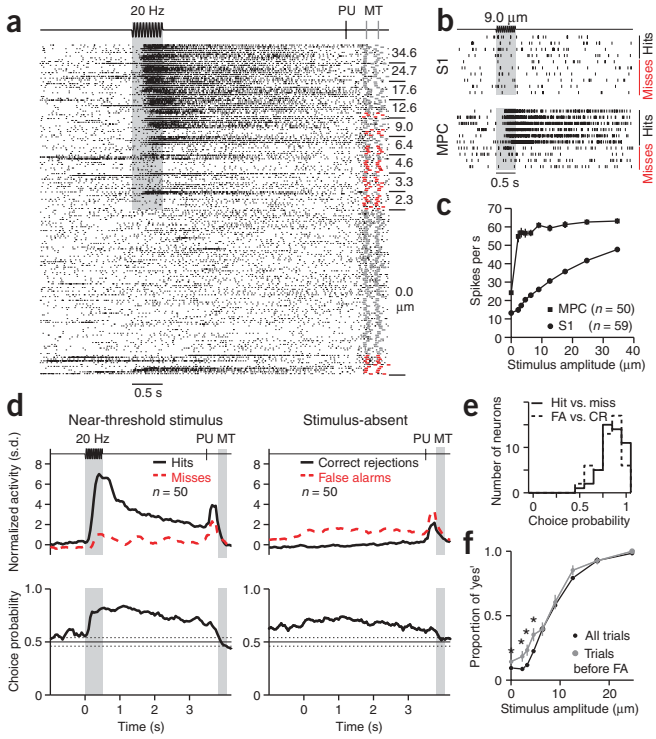

Figure 3 Activity of MPC neurons during the detection task. (**a**) Raster plot of the activity of an MPC neuron during the detection task; same conventions as in **Figure 2a**. (**b**) Responses of the S1 neuron shown in **Figure 2a** and the MPC neuron shown in **Figure 3a**, at near-threshold stimulus (9 μm). (**c**) Mean firing rates of hit trials for S1 ($n = 59$) and MPC ($n = 50$) neurons. (**d**) Comparison of normalized activity (s.d., standard deviation) during hits and misses in near-threshold trials, and during correct rejections and false alarms in stimulus-absent trials. Lower panels show the average choice probability index as a function of time. Dotted lines mark significance levels (Methods). (**e**) Distributions of indices across the population of MPC neurons ($n = 50$), calculated between the activity of hit and miss trials (hit vs. miss, mean ± s.e.m.: 0.85 ± 0.02) and between correct rejection and false alarm trials (CR versus FA, mean ± s.e.m.: 0.81 ± 0.02). (**f**) Detection curves resulting from all trials (overall performance, black circles; except for stimulus amplitude 34.6 μm) and from trials that preceded false-alarm responses (gray circles). Asterisks indicate significant differences in the probability of 'yes' responses (Fisher's exact test, $P < 0.01$)[32]. n = number of neurons; FA, false alarm; CR, correct reject.

Moreover, almost 70% of the false alarm trials were predicted from increases in neuronal activity in stimulus-absent trials (**Fig. 3d**, upper right panel, t-test: $P < 0.001$; and **Fig. 3e**). We also found that the MPC activity preceding stimulus onset was higher during hits than during misses (**Fig. 3d**, upper right panel). These early increases in activity predicted detection success significantly above chance (**Fig. 3d**). Although we do not know the role of this increased pre-stimulus activity, we speculate that it might be associated with trial history during a run. To investigate this conjecture, we analyzed the behavioral responses on trials preceding false-alarm responses. We found that the probability of a yes response was increased in trials preceding a false alarm, supporting the notion that monkeys were biased toward yes responses. We speculate that, because yes responses to three sub-threshold amplitudes (**Fig. 3f**) were rewarded, monkeys could have been encouraged to respond yes in the next trial, producing a false-alarm response. The results indicate that, for all MPC neurons studied, increased responses were associated with stimulus presence or with false alarms: that is, with yes responses. We did not find neurons that increased their activity during no responses. We do not know the reason for this, but we speculate that no is a default response that the stimulus presentation needs to override.

The close association between neuronal responses and behavioral responses, and the weak relationship between activity and stimulus amplitude, supported the interpretation that MPC neurons do not code the physical attributes of stimuli, but rather represent perceptual judgments about their presence or absence (**Fig. 4a**). As the monkeys reported their decisions by a motor act, a key question needed to be answered: was the MPC activity truly related to stimulus perception, or was it simply reflecting the different motor actions associated with the two response buttons? To test this, we designed a control task in which the correct response button was illuminated at the beginning of every trial[3,4]. In this variant of the detection task, the monkeys simply had to wait until the end of the trial to push the illuminated button, without needing to attend to the presence or absence of the mechanical

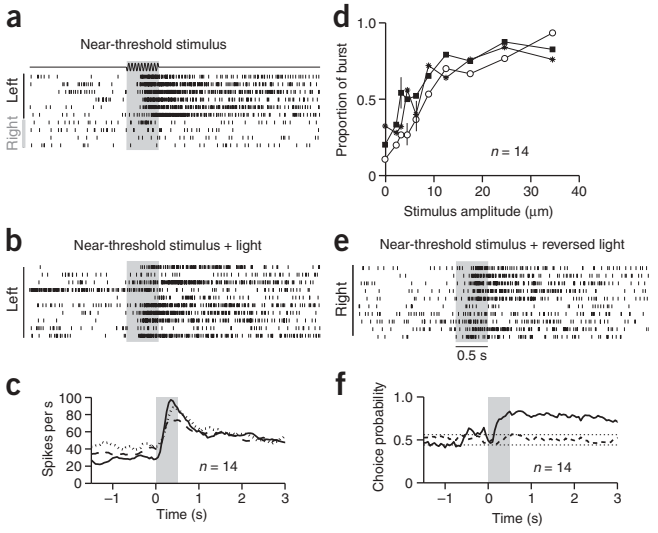

Figure 4 Sensory versus motor activity. (**a**) Responses of the MPC neuron shown in **Figure 3b**, to 10 repetitions of the 9 μm stimulus during the detection condition. (**b**) Responses of the same neuron to the same stimulus, but in a control condition in which the correct response button (left button) was illuminated at the beginning of each trial. In stimulus-absent trials, the right button was illuminated, so in this case the monkeys also knew the correct response button in advance (responses not shown). (**c**) Mean responses to a near-threshold stimulus (9 μm) during the standard detection task (continuous line) and during the two control conditions (dashed line, near-threshold stimulus + light; dotted line, near-threshold stimulus + reversed light). Each line is the mean of 140 trials from 14 neurons studied in these conditions (panels **a**, **b** and **e**). Responses to the stimuli were not significantly different across conditions (Kruskal-Wallis[8], $P = 0.11$). (**d**) Probability of burst response as a function of the stimulus amplitude during the detection task (open circles), detection task + light (squares) and detection + reversed light (asterisks). Symbols and small vertical bars, mean ± s.e.m. (**e**) Responses of the same neuron shown in **a** when the same correct response button (right button) was illuminated at the beginning of each trial. In this case, the positions of the buttons were reversed compared to the condition in **b**. (**f**) Choice probability indices for the population of neurons ($n = 14$) tested in the condition shown in **a** (continuous line); dotted line denotes choice probability indexes of the same neuronal population in the condition shown in **b** versus the condition shown in **e**. n = number of neurons.

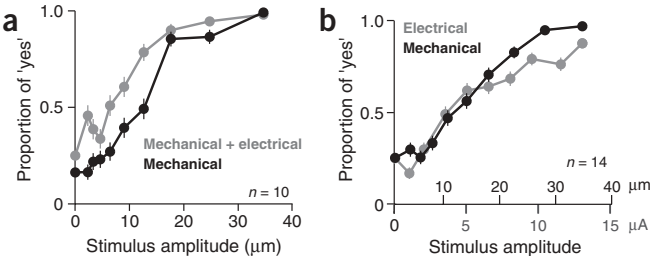

Figure 5 Detection curves during microstimulation experiments. (**a**) The mean detection curves for mechanical stimuli (black) and for mechanical-plus-electrical stimuli (gray). Mechanical and mechanical-plus-electrical trials were randomly interleaved (Methods). Ten experiments were conducted with this protocol, and each experiment consisted of 10 repetitions of each kind of stimulus; thus, each point is the mean of 100 stimulus-present trials and 900 stimulus-absent trials . (**b**) The mean detection curves for mechanical stimuli (black) and for electrical stimuli (gray). Mechanical and electrical stimulation trials were randomly interleaved (Methods). Fourteen experiments were conducted with this protocol, and each experiment consisted of 10 repetitions of each kind of stimulus; thus, each point is the mean of 140 stimulus-present trials and 1260 stimulus-absent trials. n = number of runs; symbols and small bars, mean ± s.e.m.

vibration. Raster plots of the neural activity for an example neuron showed that the responses to the stimulus in this control condition (**Fig. 4b** and dashed line in **Fig. 4c**) were very similar to the responses in the standard detection task (**Fig. 4a** and continuous line in **Fig. 4c**). In this test condition, all-or-none activity was still observed in relation to the near-threshold stimulus, and the probability of activation depended on the stimulus amplitude as in the standard detection task (**Fig. 4d**). Given that in the control test, the monkeys did not have to choose a response button based on the vibratory stimulus, the results are consistent with the interpretation that the activity of these MPC neurons is related to the subjective perception of sensory stimuli, rather than to the selection of the motor plan.

To further examine whether MPC activity was associated with the preparation of movements in different directions, we did a second control experiment in which the correct response button was illuminated at the beginning of every trial, as before. In this case, however, we switched response buttons so that the yes button was now illuminated during stimulus-absent trials and, conversely, the no button was illuminated during stimulus-present trials. The results showed that reversing the direction of the arm movements did not change the all-or-none character of the evoked MPC activity (**Fig. 4e** and dotted line in **Fig. 4c**). To test whether the direction of movement had an influence on the responses of MPC neurons, we calculated the choice probability index between the activities observed during the light (left movement; **Fig. 4b**) versus reversed light (right movement, **Fig. 4e**) conditions. The analysis shows that the choice probability values of MPC neurons were close to chance levels (dotted line in **Fig. 4f**), suggesting that these activities were not associated with the animals' hand and arm movements. Had these neurons participated primarily in movement choice or movement generation, their firing rates should have been consistently higher for one movement but not for the other. The observation of all-or-none responses during these control tasks favors the hypothesis that this MPC population reflects the failure or success of the near-threshold stimulus in triggering a sensory percept.

Microstimulation of MPC triggers perceptual reports

Given the close association between MPC activity and the behavioral reports of stimulus detection, we wondered whether artificial activation of MPC neurons through electrical microstimulation[15,16] would increase the monkeys' probability of detecting the vibratory stimuli. To test this, we injected a weak electrical current through the recording electrode in randomly selected stimulus-present and stimulus-absent trials (Methods). The resulting detection curves, separated into mechanical-plus-electrical and mechanical-only curves (**Fig. 5a**), show that monkeys tended to answer yes more often on microstimulation trials than with mechanical stimuli only. The increased probability of yes responses observed during microstimulation trials agreed with the hypothesis that MPC activity is related to perceptual judgments. Microstimulation experiments in the dorsal premotor cortex ($n = 5$) using the protocol described above did not produce significant effects on the behavioral performance (data not shown).

To further test whether artificial activation of MPC neurons could mimic neuronal activity related to sensory percepts, we did an experiment in which the mechanical vibrations were substituted by electrical stimuli of varying current strengths (Methods). We plotted detection curves for purely electrical stimuli, together with the detection curves for the mechanical stimuli that were randomly interleaved (**Fig. 5b**). Although it is difficult to compare these two stimulus quantities, the results show that psychometric performance based on microstimulation of MPC resembled that based on vibrotactile stimuli delivered to the skin. The same microstimulation protocol was used in dorsal premotor cortex ($n = 6$), but in this case monkeys always reacted as in the stimulus-absent trials (data not shown).

DISCUSSION

Our results suggest that sensory and frontal lobe neurons have significantly different roles during perceptual judgments. The activity of MPC—but not S1—neurons covaried with the reported sensory percepts during the vibrotactile detection task. Therefore, the functional role of S1 in this and other perceptual tasks may be mainly to generate a neural representation of the sensory stimulus, for further processing in areas central to S1[7,12,13,17,18]. However, a previous study found that functional magnetic resonance imaging (fMRI) signals in primary visual cortex (V1) reflect the percepts of human subjects, rather than the encoded stimulus features[19]. This result suggests that, in V1, top-down signals (non-sensory inputs delivered to visual cortex via feedback projections) can be combined with bottom-up (sensory) information and contribute to sensory percepts[19]. Our S1 data did not show evidence for this type of neural interaction; rather, it indicated that S1 represents the physical properties of stimuli and contributes little to near-threshold percepts. The discrepancy could be due to fundamentally different organizations across sensory cortices or to differences between species. Another possibility to consider is that the modulation revealed through fMRI may have an effect that is invisible from the point of view of single neurons. This would happen if, for instance, such modulation acted only to synchronize the spikes of multiple target neurons[20,21].

On the other hand, frontal lobe neurons, which are involved in decision-making[3,4,22,23], working memory[3,4,17,24] and motor planning[25,26], did seem to be fundamental to perceptual judgments during sensory detection. This is consistent with the idea that perceptual judgments result from the interaction between internal signals (working memory, expectation, attention) and sensory inputs[3–5,17,19], because the MPC is ideally situated to integrate these different types of information[27]. For the same reason, it is possible that other circuits of the frontal[3,4,18] and parietal lobes[17] also contribute to perceptual judgments in a similar way. An important clue about this interpretation is that the electrical current injected into MPC led to behavioral responses that resembled those elicited by mechanical vibrations,

suggesting that although the artificial stimulus did not originate in S1, it was still interpreted as sensory evidence. We do not know whether microstimulation in MPC evoked the same somatosensory sensation as that evoked by natural stimuli, but it produced the same behavioral reactions. Another possibility is that microstimulation of MPC does not produce any somatic sensation but, instead, activates a task rule such as 'a stimulus is present'. In this manner, varying the microstimulation strength could vary the probability of engaging a population of MPC neurons associated with this rule and, therefore, produce a psychometric detection curve similar to that produced by varying the mechanical stimulus strength.

The contribution of different cortical areas to perceptual processing has also been investigated using binocular rivalry and other protocols in which a fixed but ambiguous visual stimulus gives rise to multiple, alternating percepts; that is, the same sensory input is consistent with multiple perceptual interpretations[28,29]. These studies agree with the present data in that high-order cortices show much stronger correlations with behavioral (perceptual) reports than do primary sensory areas.

METHODS

Detection task. Stimuli were delivered to the skin of the distal segment of one digit of the restrained hand, via a computer-controlled stimulator (BME Systems; 2-mm round tip). Initial probe indentation was 500 μm. Vibrotactile stimuli consisted of trains of 20 Hz mechanical sinusoids with amplitudes of 2.3–34.6 μm (**Fig. 1**). These were interleaved with an equal number of trials where no mechanical vibrations were delivered to the skin (amplitude = 0). Animals pressed one of two buttons to indicate stimulus-present (left button) or stimulus-absent (right button). They were rewarded with a drop of liquid for correct responses. Performance was quantified through psychometric techniques[1]. Animals were handled according to institutional standards of the US National Institutes of Health and the Society for Neuroscience.

Recording sessions and sites. Neuronal recordings were obtained with an array of seven independent, movable microelectrodes[3,4] (2–3 MΩ) inserted into S1 and MPC. Recordings in S1 were made in areas 3b and 1, contralateral to the stimulated hand and ipsilateral to the responding hand and arm (two monkeys). Initially, we recorded S1 neurons with cutaneous receptive fields with quickly adapting or slowly adapting properties, but we found that the neurons with slowly adapting properties showed weak modulation in their firing rate during the stimuli (data not shown). We therefore focused on the quickly adapting neurons. Recordings in MPC (pre-supplementary motor area)[3,27] were made in both hemispheres. Electrodes were advanced into the MPC to find neurons that responded during the task. MPC neurons preferentially responded during the stimulus and delay periods of the task. Recording sites in S1 and MPC changed from session to session. The locations of the electrode penetrations in S1 and MPC were confirmed with standard histological techniques.

Data analysis. We analyzed the responses of 59 S1 neurons (area 3b: $n = 28$; area 1: $n = 31$). All the S1 neurons had small cutaneous receptive fields located in the distal segment of one digit (distal segments of fingertips 2, 3 or 4) and had quickly adapting properties. Stimuli were delivered to the center of the neuron's cutaneous receptive field while the monkeys executed the detection task (**Fig. 2a**). A total of 127 responsive neurons were recorded in the MPC of both hemispheres during the detection task. These neurons were sorted in two groups according to their response dynamics: one that had transient responses lasting up to 1 s after stimulus offset ($n = 40$), and another that showed persistent activity starting during the stimulus onset and continuing throughout the full delay period until "probe up" (PU) triggered the hand and arm movement ($n = 87$). For analysis, we used 50 of the 87 neurons that had sustained activity (because recordings were stable for these 50); during the detection task, we collected 10 repetitions per stimulus amplitude and 90 repetitions of the stimulus-absent trials (**Fig. 3a**). The neurons that had transient responses ($n = 40$) also had bimodal activity and generally behaved similarly, albeit for a limited time (data not shown).

To calculate response distributions and the neurometric detection curves of S1 neurons (**Fig. 2b–d**), on each trial we obtained the maximum firing rate in a 500-ms window that was displaced every 1 ms in the period between 1.5 s before and 3.5 s after stimulus onset (the same period was used for stimulus-absent trials). Neurometric curves were calculated as the proportion of trials in which the maximum firing rate reached or surpassed a criterion level. For each neuron, this criterion was chosen to maximize the number of hits and correct rejections (that is, correct trials). From logistic fits, we calculated psychometric and neurometric detection thresholds as the probability that the proportion of 'yes' responses would be 0.5.

For 59 S1 neurons and 50 MPC neurons, we calculated the firing rate as a function of time, using a 200-ms window displaced every 50 ms (**Figs. 2e** and **3d**). Normalized activity was calculated by subtracting the mean activity and dividing by the standard deviation of the activity from a 200-ms window of the pre-stimulus period. (The same results were obtained using the raw data.) Normalized activity shown in **Figure 2e** was based on 370 hits and 370 misses (left panels) and 620 false alarms and 620 correct rejections (right panels), collected during the study of the 59 S1 neurons. Normalized activity shown in **Figure 3d** was based on 312 hits and 312 misses (left panels) and 494 false alarms and 494 correct rejections (right panels), collected during the study of the 50 MPC neurons. We used trials with 12.6, 9.0 and 6.4 μm stimulus amplitude. Choice probability index was calculated using methods from signal detection theory[4,6,17]. This quantity measures the overlap between two response distributions: in this case, between hits and misses and between correct rejections and false-alarm trials. Dashed lines in **Figures 2e** and **3d** indicate $P = 0.01$ significance limits, bootstrap technique[30]. To determine the differences between hit and miss responses and between correct rejection and false alarm responses, we used the two-tailed t-test on the distributions of the number of spikes found in a 500 ms window during the stimulus period for S1 neurons, and during a 500 ms window starting 250 ms after stimulus onset for MPC neurons.

To estimate the amount of correlated noise activity across S1 neurons, the Pearson's correlation coefficient was calculated for each pair of simultaneously recorded S1 neurons ($n = 51$)[9]. We first standardized the firing rates of the two neurons by subtracting the mean and dividing by the standard deviation across the ten stimulus repetitions. Trials were sorted by stimulus amplitude. For each pair of neurons, we obtained a correlation coefficient as function of stimulus amplitude. The mean correlation coefficient, across the 51 pairs of neurons and across stimulus amplitude classes, was 0.16 ± 02 (mean ± s.e.m.). We found no relation between correlation coefficient and stimulus amplitude or trial type (hit, miss, correct rejection or false alarm).

Trials in the control light task proceeded exactly as described in **Fig. 1a**, except that at the probe down, the correct target button was illuminated (**Fig. 4b,e**). Vibrotactile stimuli were delivered while the light was kept on; then the probe was lifted off from the skin (PU) and the light was turned off. The monkey was rewarded for pressing the previously illuminated button. Hand and arm movements were identical to those in the somatosensory detection task but were cued by visual stimuli. Under this condition, the choice probability indices (**Fig. 4c**) and burst proportion (**Fig. 4d**) were calculated by comparing response distributions for left versus right button presses.

To estimate the proportion of bursts as a function of stimulus amplitude (**Fig. 4f**), we used a Poisson spike analysis[31] that determined whether or not a burst occurred on each trial. First, we counted the spikes across the whole trial and divided them by the trial duration to obtain the mean trial firing rate. Second, we counted the number of spikes in a 500-ms window, beginning 250 ms after stimulus onset. Finally, using the Poisson cumulative density function, we estimated the probability of obtaining a firing rate equal or larger than that observed in the 500-ms response window[31], given the mean firing rate across the whole trial. We considered that a burst occurred in a given trial if this probability was less than 0.05.

Microstimulation. A computer-controlled pulse generator (Coulbourn), in series with an optical stimulus isolation unit, produced biphasic current pulses with the cathodal phase leading. Each phase lasted 0.2 ms, with 0.05 ms

between phases. Microstimulation consisted of 5-μA biphasic current pulses, delivered at 200 Hz and superimposed over the period that corresponded to either the stimulus-present or stimulus-absent mechanical trials (**Fig. 5a**). By stimulating in both stimulus-present and stimulus-absent trials, we did not reinforce the monkeys to answer yes in microstimulation trials. In fact, because of the more frequent false-alarm responses (non-rewarded trials), overall performance during microstimulation trials was slightly, but significantly, lower than performance during normal detection trials.

In a second microstimulation protocol, mechanical stimuli were randomly substituted by electrical stimuli of 200 Hz with varying amplitudes (1–12 μA) in one half of the stimulus-present trials (**Fig. 5b**). In this experiment, monkeys were rewarded for answering yes in microstimulation trials. We cannot discard the possibility that microstimulation generated a non-natural sensation that could be used by the subjects to indicate detection. We believe, however, that this is unlikely because from the very first microstimulation trials, monkeys generated yes responses even if they were not rewarded for this behavioral report.

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COMPETING INTERESTS STATEMENT

The authors declare that they have no competing financial interests.

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