Instructed Delay Discharge in Primary and Secondary Somatosensory Cortex Within the Context of a Selective Attention Task

El-Mehdi Meftah,1 Stéphanie Bourgeon,1 and C. Elaine Chapman1,2

1Groupe de recherche sur le système nerveux central, Département de physiologie and 2École de réadaptation, Faculté de médecine, Université de Montréal, Montreal, Quebec, Canada

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Meftah E-M, Bourgeon S, Chapman CE. Instructed delay discharge in primary and secondary somatosensory cortex within the context of a selective attention task. J Neurophysiol 101: 2649–2667, 2009. First published February 18, 2009; doi:10.1152/jn.91121.2008. The neuronal mechanisms that contribute to tactile perception were studied using single-unit recordings from the cutaneous hand representation of primate primary (S1) and secondary (S2) somatosensory cortex. This study followed up on our recent observation that S1 and S2 neurons developed a sustained change in discharge during the instruction period of a directed-attention task. We determined the extent to which the symbolic light cues, which signaled the modality (tactile, visual) to attend and discriminate, elicited changes in discharge rate during the instructed delay (ID) period of the attention task and the functional importance of this discharge. ID responses, consisting of a sustained increase or decrease in discharge during the 2-s instruction period, were present in about 40% of the neurons in S1 and S2. ID responses in both cortical regions were very similar in most respects (frequency, sign, latency, amplitude), suggesting a common source. A major difference, however, was related to attentional modulation during the ID period: attentional influences were almost entirely restricted to S2 and these effects were always superimposed on the ID response (additive effect). These findings suggest that the underlying mechanisms for ID discharge and attention are independent. ID discharge significantly modified the initial response to the standard stimuli (competing texture and visual stimuli), usually enhancing responsiveness. We also showed that tactile detection in humans is enhanced during the ID period. Together, the results suggest that ID discharge represents a priming mechanism that prepares cortical areas to receive and process sensory inputs.

INTRODUCTION

It is now firmly established that a number of cortical areas involved in motor planning and execution show preparatory discharge during experiments involving an instructed delay (ID) period. In such studies, monkeys are trained to interpret symbolic sensory stimuli (visual or, in some cases, auditory) presented during the instruction period to guide the planning and execution of a subsequent movement. The discharge that develops during the ID period is frequently predictive of the subsequent movement, encoding for example the desired direction of movement or other movement parameters. Interestingly, ID discharge is present even when learned movements are observed and not actually performed (Cisek and Kalaska 2004). ID discharge begins within several 100 ms of cue onset (Crammond and Kalaska 1989; Weinrich and Wise 1982; Weinrich et al. 1984) and is generally sustained over the duration of the ID period, which can extend for many seconds. Whereas ID discharge is most prominent in motor areas of the frontal lobe (Cisek and Kalaska 2005; Weinrich and Wise 1982; Weinrich et al. 1984; reviewed in Johnson et al. 2001), it is also seen in posterior parietal cortex, including area 5 (reviewed in Andersen et al. 1997; Colby and Goldberg 1999; Kalaska 1996; Platt and Glimcher 1999). ID responses in these areas, depending on the paradigm, have been variously ascribed to motor set, movement preparation or sensorimotor transformation, movement suppression, attention to salient features, and working memory (Andersen et al. 1997; Johnson et al. 2001; Kalaska and Crammond 1995; Murray et al. 2000; Romo et al. 1999; Shadlen and Newsome 2001). The challenge is to distinguish among these different possibilities in different paradigms and contexts.

Although much emphasis has been placed on the importance of ID discharge for planning and executing movements, there is now growing evidence that primary sensory cortical receiving areas themselves can be activated by preparatory state in relation to perceptual tasks. Imaging studies in humans have shown that there are baseline shifts in activity in striate and extrastriate cortices associated with selective attention to visual stimuli (Kastner et al. 1999; Ress et al. 2000), with activity increasing even in the absence of visual stimulation. Driver and Frith (2000) proposed that the baseline changes in visual areas seen with voluntary attention may be analogous to the increased activity associated with motor imagery; i.e., subjects may be imagining the upcoming stimulus in anticipation of its presentation. They also considered how such a baseline shift might contribute to attention-related changes in stimulus-evoked discharge, including moving cells into an optimal dynamic range or priming the internal representation of the target stimulus in a winner-take-all competition.

With respect to the somatosensory cortex (S1 and S2), there have been only a few reports of changes in baseline discharge. Zhou and Fuster (1996) reported that some S1 neurons develop prolonged changes in discharge within the context of a delayed haptic match-to-sample task. Discharge during the lengthy retention period of this task, 14–20 s, was suggested to form the neuronal basis for short-term haptic working memory. When the haptic cue was replaced by a symbolic visual cue (visuohaptic matching task), Zhou and Fuster (1997) reported that the visual cues also provoked activity (including during the retention period). Although these are interesting observations, other factors may have contributed to their findings. For example, their retention period was long, making it difficult to ensure that modest shifts in the animal’s posture, changes in fusimotor bias (given that the task involved haptic perception),
and/or motor set/preparation, did not contribute to the observed results. Consistent with the suggestion that motor preparation might have contributed to the results of Zhou and Fuster, Liu et al. (2007) reported preparatory activity in S1 neurons during the ID period of a motor task. In contrast, Crammond and Kalaska (1989) found no evidence for ID discharge in area 2 of S1 during a visuomotor task. Finally, using a purely tactile task (two-sample vibrotactile frequency discrimination) and shorter delay intervals (1–3 s), Romo and collaborators found some evidence for discharge in S2, but not S1, neurons during the delay period that separated two sequences of vibration (Salinas et al. 2000). This discharge was short lasting and limited to only 13% of the S2 sample. Using this same task, these investigators showed prominent delay period discharge elsewhere, notably in the prefrontal cortex and premotor cortical areas (Hernández et al. 2002; Romo et al. 1999). Thus the existence, let alone the function, of baseline changes in S1 or S2 remains controversial.

We previously reported that directed attention modifies the discharge frequency of 20% of S2 neurons in the cutaneous hand representation during the final 500 ms of an ID period that preceded a directed-attention task (Meftah et al. 2002). We also noted changes in discharge, independent of attention, during the instructed delay period but did not analyze these. The goal of this report was to determine the extent to which the presentation of the symbolic light cues themselves (signals that cued the animal for the modality to discriminate in the upcoming trial, tactile or visual) modulated cell discharge rates during the delay period relative to background firing. For this, we used a slightly expanded version of our original database. We also examined the functional importance of this discharge, with analyses that addressed the hypothesis that ID discharge modulates evoked responses to tactile stimulation, priming somatosensory cortical areas to receive and process tactile inputs. A complementary psychophysical study in humans investigated the importance of priming to perception, by measuring tactile detection during the ID period.

Preliminary reports of the results were previously published (Bourgeon et al. 2004; Meftah and Chapman 2004).

**METHODS**

**Single-unit recordings in monkeys**

**BEHAVIORAL TASKS.** Experiments were performed in the same two adult monkeys (Macaca mulatta; G, 8.5 kg; I, 9.2 kg) as used in our previous studies (Chapman and Meftah 2005; Meftah et al. 2002). Details of the behavioral tasks, data acquisition, histological controls, and the general characteristics of the database were previously described. All procedures were approved by the institutional animal care and use committee and followed the guidelines specified by the Canadian Council on Animal Care.

In brief, monkeys were trained to perform a cross-modal attention task (Fig. 1). Each trial started with an instruction period (2 s) in which a colored light indicated the modality to attend and discriminate in the upcoming stimulation period: green for the tactile modality (texture-discrimination task) and red for the visual modality (visual-discrimination task). The instruction period was followed by the stimulation period (3 s) (Note that the instruction light remained on throughout the latter period.) The tactile task consisted of discriminating a change in tactile roughness: a standard surface (2-mm spatial period [SP]) was scanned at 50 mm/s under the immobile finger tips, D3/4, and at a variable delay (~1 or 1.7 s) SP was incremented, corresponding to a subjective increase in roughness (Meftah et al. 2000). Given the complex nature of the overall attention task, large increases in SP (to 3.7 or 4.7 mm) were used. The monkeys were trained to signal the change in roughness by releasing a response lever with the nonstimulated hand; time constraints (reaction time [RT] windows of 200–700 ms) were imposed for a juice reward to be given. The visual task was similar: a standard light intensity (array of yellow light-emitting diodes) appeared at the same time as the tactile stimulus.

![FIG. 1. Schematic representation of events during a trial (see text for details), showing the epochs in which cell discharge frequency was calculated on a trial-by-trial basis. For 139 cells (complete instructed delay [ID] recording), ID discharge was measured over the entire 2-s period (shaded region); discharge during the Hold period served as the reference value for defining an ID response. For the remaining 102 cells, only the final 500 ms of the ID period was recorded (hatched region); discharge during the Static period (right, final 500 ms of the trial) served as the reference for these cells.](http://jn.physiology.org/doi/abs/10.1152/jn.00551.2008)
stimulation began. This was followed at variable delays (0.9, 1.3, or 1.7 s) by an increase in light intensity (low, medium, or high). Again, the monkey signaled the perceived change in light intensity by releasing the response lever; responses were rewarded only if they fell within the RT window (100–400 ms; the RT windows were longer for the tactile trials to compensate for the time that it took for the texture change to traverse the 18-mm aperture).

The tactile and visual tasks were interleaved during data acquisition, so that attention to the relevant modality had to be oriented on a trial-by-trial basis. During the intertrial interval, the tactile stimulator (40-cm-diameter cylindrical drum) was repositioned to the start of the segment of surface to be presented in the upcoming trial. A brief tone signaled that the surface was in position. Once the nonstimulated hand was in position on the lever and the stimulated digits were in contact with the surface, data acquisition was initiated. Following a 500-ms hold period (Fig. 1), the instruction light appeared, with the color indicating the modality to attend and discriminate in the upcoming stimulation period. Two seconds later, the stimulation period began (3-s duration): there was an initial period of standard stimulation (2-mm SP texture + standard light intensity) followed, at variable delays, by a change in intensity of the salient modality (tactile or visual). In 50% of the trials, there was a change in both modalities (never simultaneous). In this case, the monkeys had to ignore changes in the unsignaled modality in favor of the signaled modality (see Fig. 1). [Note: as reported previously (Mefthah et al. 2002), success rates were high in both monkeys (93 and 96% correct) and not different for the trials with one or two changes, consistent with attention having been successfully directed to the signaled modality.] At the end of the stimulation period, drum rotation stopped; the visual stimulus and the instruction cue were also extinguished. Data acquisition continued for an additional 0.7 s to provide a baseline value, as the monkey sat quietly with its digits in contact with the now immobile surface (static period).

DATA RECORDING AND ANALYSES. Extracellular activity of single neurons in S1 and S2 cortex was recorded with a glass-coated tungsten microelectrode. Complete testing of each cell (116 in S1, 125 in S2) in the attention task required about 120 trials: 60 tactile and 60 visual trials (order quasi-random). The focus of this report is on the discharge seen during the 2-s instruction period and its relationship to the discharge seen in the subsequent stimulation period.

In our initial recordings from one monkey (G), data collection from the ID period was restricted to the final 500 ms of the ID period (hatched region during the instruction period; Fig. 1: S1, n = 67; S2, n = 35) along with the subsequent stimulation and static periods (700 ms after the end of the stimulation period; lights and drum rotation off). After noticing that discharge was modulated during the ID period, data acquisition was extended to include the entire 2-s-long ID period along with the preceding 500-ms hold period [S1, n = 49 (monkey I); S2, n = 90 (G, 26; I, 64)].

During data acquisition, we carefully monitored the position of the stimulated digits, rejecting the occasional trial in which the monkey did not leave its digits immobile and in contact with the surface throughout the trial. Off-line, we also inspected the vertical contact force records and rejected trials in which contact force varied by more than ±0.2 N prior to the time of the lever response. This led to the rejection of <5% of trials from the analysis.

Patterns of discharge were examined using rasters and perievent histograms. Mean cell discharge frequency was measured in a number of epochs on a trial-by-trial basis. Quantitative analyses were restricted to rewarded trials, thus ensuring that attention was correctly directed to the signaled modality. The measured epochs included (Fig. 1): 1) the hold period [first 500 ms of the trial (only cells with a complete ID period)]; 2) the ID period [2 s (complete) or only the final 500 ms (incomplete)]; 3) the stimulation period (3 s); 4) the standard period (first 700 ms of the stimulation period); 5) the salient change (Δ) in texture (200 ms before and after the Δ in texture, so including the response to the standard 2-mm SP surface and the modified surfaces, 3.9 or 4.9 mm SP); and 6) the static period (final 400 ms of the trial; drum rotation ended and lights extinguished).

Cells were first classified into two groups according to the presence or absence of an ID response. All trials (tactile, visual) were pooled for these analyses, but the results were verified for each modality separately. A minimal difference of 2.5 impulses (imp)/s in mean discharge rate was required for the response to be considered physiologically significant. For cells with a complete ID recording (139/241), the discharge rate in each trial during the entire 2-s-long ID period was compared with the discharge rate in the corresponding initial hold period (paired t-test, P ≤ 0.01). For cells with only the final 500 ms of the ID period recorded (102/241), discharge in the final 500 ms of the ID period was compared with that measured in the static period at the end of the trial. Incomplete cells were classified as showing an ID response if the discharge rate was significantly different both overall (3 SPs pooled) and when the comparison was restricted to the 2-mm SP, corresponding to the texture on which the digits rested during the ID period. To validate the analysis used for the incomplete cells, the same analysis was applied to the cells with a complete ID recording: the results confirmed those obtained in the main analysis (hold vs. ID) in 87% of the cells. The distribution of the errors was such that there were equal numbers of false positives and false negatives, indicating that the overall estimate of the prevalence of ID discharge was accurate. In the cases in which the results were not confirmed, the result (false positive or negative) was explained by poststimulation modulation of discharge associated with the end of the drum rotation, corresponding to the end of the period of dynamic tactile stimulation.

For all cells with an ID response, the sign of the ID response was assigned to be either positive [ID > reference discharge (hold or static)] or negative (ID < reference discharge). We also categorized the sign of the response evoked during the initial 700 ms of the stimulation period (standard period) relative to the discharge rates measured over the final 500 ms of the ID period. In some instances the evoked response was mixed: phasic increase and decrease (or vice versa); in these cases, the sign of the initial evoked response was used to categorize the sign of the response during the standard period.

The onset latency of the ID response was computed from the pooled frequency histograms for each task (complete recordings only). This approach was taken because the ID response was sometimes variable (absent or small), especially in S1. A sustained change in discharge frequency (more than or less than ±2 SE of the hold discharge) needed to persist for at least three successive 10-ms bins; the earliest bin was defined as the onset of the response.

The variability of the ID response was assessed by first sorting trials according to the discharge rate during the instruction period (low, high). In the trials with the lowest or highest discharge rate [n = 20 or n = 10, depending on whether the tactile and visual trials were pooled (respectively, absence or presence of attentional modulation during the ID period)], the discharge rate in the ID period was compared with that in the corresponding hold period (paired t-test) to determine whether an ID response was present.

Attention sensitivity during the ID period was determined using the same approach used previously (Chapman and Mefthah 2005; Mefthah et al. 2002); the presence of a significant difference in discharge between the tactile and visual tasks (independent t-test, P ≤ 0.01).

These analyses were applied to all cells: using the discharge rate during the entire ID period (2 s, complete cells), as well as the discharge during the final 500 ms of the ID period (all cells). Differences in mean rate had to be ≥2.5 imp/s to be considered physiologically significant. These results were confirmed using a distribution-free statistic, the area of the curve under the receiver operating characteristic (ROC) curve obtained by plotting the probability that discharge rate in the tactile trials was greater than a variable criterion response level (number of impulses/trial, increments of one impulse) as a function of the probability that, for the same cell,
discharge in the visual trials was greater than the criterion (Britten et al. 1992; Puré and Wurtz 2001). Attention sensitivity in later periods of the trials was reported previously (Meftah et al. 2002). Of relevance here are the results from measures in two periods—standard stimulus and salient Δ in texture (see earlier text)—related to the present findings.

HISTOLOGICAL METHODS. Near the end of the experiment, the monkeys were sedated (ketamine 15 mg/kg) and electrolytic lesions made in selected tracks to delimit the recording region. After the final session, the monkeys were killed with an overdose of pentobarbital and perfused through the heart with formol-saline solution. Electrode tracks were reconstructed from 50-μm parasagittal sections stained with cresyl violet. For S1, the cytoarchitectonic boundaries were determined using the criteria described by Powell and Mountcastle (1959) and Jones et al. (1978). In S2, the criteria described by Jones and Burton (1976) were used. The histology for monkey G has been published (Fig. 4; Meftah et al. 2002). The histology for monkey I is shown in Fig. 2. The hatched regions (Fig. 2B) show the corresponding regions sampled from monkey G (recordings made only in the right hemisphere).

Psychophysical experiment in humans

Ten paid volunteers (5 men, 5 women; 22–30 yr of age; all but one right-handed for writing) participated in the experiment, which consisted of one single session (2-h duration). The results from one subject were omitted because she could not perform the perceptual tasks. The experimental protocol was approved by the institutional ethics committee and all subjects gave their written informed consent. Subjects performed the selective-attention task described earlier (170 trials altogether), along with a tactile detection task whereby a weak, near-threshold stimulus was presented at three delays during the ID period: 200, 1,000, and 1,500 ms. In half of the trials, a weak electrical stimulus [70% detected (P70), 0.38–0.86 mA, rectangular pulse, 2-s duration] was delivered via surface electrodes to the glabrous surface of the middle and proximal phalanges of the left D3, corresponding to

FIG. 2. Histology from the left (A) and right (B) hemispheres of monkey I. Surface reconstruction (top) of the entry points of the recording tracks along with 2 parasagittal sections at different laterality (middle, bottom). Large dots show the tracks from which the cells in the current database (monkey I) were recorded; small dots show tracks that did not contribute data to this report. CS, central sulcus; IPS, intraparietal sulcus; LS, lateral sulcus; PCS, postcentral sulcus. In B, the hatched region shows the general region in which recordings were made in monkey G (taken from Meftah et al. 2002). These regions were aligned on the basis of the receptive fields of the units encountered superficially in S1 (D1 lateral; D5 medial), and the major sulci.
the finger in contact with the textured surfaces in the main task. In the other 50% of trials, no electrical stimulus was presented. Subjects were instructed to perform the main task (detect the change in tactile or visual stimulus, according to the color of the instruction cue), and also report verbally at the end of the trial whether a weak electrical stimulus was perceived. They were also asked to rate their confidence in this latter decision, so as to calculate a bias-free measure of detectability, $d'$ (Green and Swets 1988; details in Chapman et al. 2005), for each delay tested. The results were analyzed using a repeated-measures ANOVA.

**RESULTS**

**Single-unit recordings in monkeys**

Data are presented here from two monkeys. Recordings were made from the right hemisphere of monkey G and both hemispheres of monkey I, after a pause to train the animal to switch arms for the task. All cells had a cutaneous receptive field on the tips of the stimulated digits (D3 and/or D4) and showed a significant modulation of their discharge (increased or decreased) during the stimulation period of the selective-attention task as compared with the discharge at rest. All cells were recorded from the hemisphere contralateral to the stimulated digits. The database included 116 S1 cells (67 from monkey G and 49 from monkey I) and 125 S2 cells (G, $n = 61$; I, $n = 64$). These represented a slightly expanded database from that used previously (Meftah et al. 2002): 14 S1 and 49 S2 additional cells in monkey I.

Table 1 summarizes the localization of the S1 sample relative to cytoarchitectonic areas. The majority of the sample was localized to areas 3b ($n = 36$) and 1 ($n = 45$), along with a small number ($n = 18$) assigned to the 3b/1 border. A small number of neurons was recorded in area 2 ($n = 17$).

The histology for monkey I is shown in Fig. 2 (region sampled in monkey G superimposed in Fig. 2B; see legend for details). Of 49 cells recorded in S1 of monkey I that met the criteria for inclusion in this report, the majority were localized to area 1 ($n = 33$). The remaining cells were in area 3b ($n = 7$) or the 3b/1 border region ($n = 9$).

The adaptation rate of most cells to punctate, manually applied stimuli was determined (236/241). Cells were classified as either rapidly adapting (S1, 55/114; S2, 50/122) or slowly adapting (response to a maintained stimulus lasted $> 2$ s) (S1, 59/114; S2, 72/122). Single-digit receptive fields were common in S1 (35/114) and all cells had a contralateral receptive field. S2 was characterized by larger receptive fields covering multiple digits (120/125), as well as by bilateral receptive fields (59/125). In both S1 and S2, the majority of cells were sensitive to light touch (respectively, 98/116 and 105/125); the rest (16%) required more vigorous stimulation and/or moving touch for activation.

### Table 1. ID discharge as a function of cytoarchitectonic area in S1 ($n = 116$) and the sign of the ID response

<table>
<thead>
<tr>
<th>Area</th>
<th>ID+ ($n = 39$)</th>
<th>ID− ($n = 9$)</th>
<th>ID Absent ($n = 68$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>3b ($n = 36$)</td>
<td>13</td>
<td>0</td>
<td>23</td>
</tr>
<tr>
<td>3b/1 ($n = 18$)</td>
<td>5</td>
<td>1</td>
<td>12</td>
</tr>
<tr>
<td>1 ($n = 45$)</td>
<td>16</td>
<td>7</td>
<td>22</td>
</tr>
<tr>
<td>2 ($n = 17$)</td>
<td>5</td>
<td>1</td>
<td>11</td>
</tr>
</tbody>
</table>

+, increase; −, decrease relative to the reference period.

In the following sections, the general characteristics of the ID responses in primate S1 and S2 are described using single-cell examples (Figs. 3–5) and population histograms (Fig. 6). These are followed by further examples and analyses addressing the links between ID responses and neuronal sensitivity to tactile inputs, behavior, and attention.

**SINGLE-CELL EXAMPLES: S1.** Rasters and perievent histograms in Fig. 3 illustrate representative single-cell examples from area 1. Only rewarded trials are shown, ensuring that attention was correctly directed to the signaled modality in all trials. Overall, 41% of S1 cells (47/116) showed evidence of either an increase, ID+ (e.g., Fig. 3A), or decrease, ID− (e.g., Fig. 3B), in discharge rate during the ID period (shaded region), compared with discharge during the reference period (initial hold period for these examples, left of the shaded region). These examples were representative of the larger population since the ID responses developed within the first 200 ms of the instruction onset and persisted throughout the instruction period.

The majority of S1 cells with an ID response showed a pattern of increased levels of discharge during the ID period compared with the reference period, ID+ (A: 81%, 38/47). The rest showed the opposite response, ID− (B: 19%, 9/47). The two examples in Fig. 3, A and B share several common features. First, a comparison of the summary histograms (below) shows that the magnitude of the response during the instruction and standard periods (right of the shaded region) was similar for both tasks, tactile and visual. Thus the ID response in these examples was independent of attentional influences during these periods, as found in the overwhelming majority of S1 cells (Meftah et al. 2002). Second, the sign of the response evoked during the stimulation period was the same as that seen in the ID period, an increase in discharge in A, and a decrease in discharge in B. Overall, the sign of the ID response predicted the sign of the response evoked during the subsequent stimulation period in most S1 cells with an ID response (38/47, 81%). Third, in these and all other cells (S1 and S2), the ID discharge was not associated with any systematic changes in contact force with the textured surface (see mean force trace labeled “drum”). Likewise, there was no change in contact force with the response lever during the ID period (the nonstimulated, contralateral hand rested on the response lever throughout the trial, up until the lever was released when the monkey perceived the change in the signaled modality).

Figure 3C shows an example of an S1 cell that showed no evidence of an ID response and so was representative of a sizable proportion of the S1 cells (69/116, 59%). Such neurons, however, were not insensitive to the standard stimuli. In this example, there was a robust increase in discharge at the onset of the standard stimulation (2-mm SP texture scanned at 50 mm/s and the standard light intensity). This was again independent of the direction of attention (see histograms, bottom).

**SINGLE-CELL EXAMPLES: S2.** The proportion of S2 cells with an ID response (38/125, 46%) was similar to that found in S1; moreover, ID+ responses also predominated here (74%). Typical examples are shown in Fig. 4 (A, ID+; B, ID−). These cells illustrate several characteristics shared with S1: ID responses developed within around 200 ms of the cue onset, were sustained throughout the ID period, and were similar in amplitude to the responses in S1. In a proportion of S2 cells
including these examples), the ID response amplitude and timing did not vary as a function of the task (tactile vs. visual). Thus the direction of attention did not modify the ID response. As also found in S1, the sign of the ID response often predicted the sign of the response evoked during the subsequent standard period (increased discharge, Fig. 4A). Finally, the no-ID cells in S2 (see Fig. 4C) were, as in S1, also responsive to the standard stimuli. Several differences were noted in S2 cells compared with S1 cells. First, the link between the sign of the ID response and the subsequent discharge evoked during the standard period (i.e., standard texture and visual stimuli) was not as straightforward as that in S1. About 31% (18/58) of S2 cells behaved like the cell shown in Fig. 4B (see also Fig. 5B); i.e., the sign of the ID response (ID–) did not predict the sign of the response during the subsequent standard period (standard +: discharge increased relative to the level measured in the final 500 ms of the ID period; note, however, that in this example this was superimposed on a continuing suppression relative to the initial hold period). This was less often observed in S1 (19%). Second, the S2 sample contained subjectively more dramatic examples of ID discharge than did S1, usually because attentional effects were superimposed. The cell shown in Fig. 5A is an example. This cell showed a ramp increase in discharge during the ID period, but the ID response was much larger in the tactile trials than that in the visual trials; i.e., the cell’s discharge was differentially modulated by the direction of attention. Moreover, this attentional effect carried over into the subsequent standard period, so that discharge rates were substantially higher in the tactile trials. This is discussed further in the following text. A final feature of the S2 cells is illustrated by the neuron shown in Fig. 5B. This neuron showed a phasic increase in discharge that appeared to be linked to the onset of the instruction cue (latency, ~300 ms) combined with a distinct,
sustained response suppression that began earlier (195 ms) and continued, albeit irregularly, throughout the ID period. A total of 8 S2 cells showed a similar pattern of discharge, consisting of an early transient excitatory burst (mean latency, 110 ms; range, 40–300 ms) along with an associated long-lasting change in discharge during the instruction period (usually a decrease in discharge, 7/8). All of these cells (referred to as complex cells) showed a pattern of increased discharge during the subsequent standard period, i.e., as the textured surface was moved under the finger tips.

POPULATION ANALYSES: S1 AND S2. Overall, ID responses were found with equal frequency in S1 (41%) and S2 (46%; see Table 2) ($\chi^2$ test, $P = 0.36$). Cells with an ID response were found throughout S1, but their distribution varied across the three areas containing the cutaneous representation of the digits. Close to half of the cells with an ID response were localized to area 1 (Table 1), where they represented 50% of the area 1 sample (23/45). Lower proportions of cells with an ID response were found in areas 3b, 3b/1, and 2 (35%, 25/71). In addition, ID− cells were almost exclusively found in area 1 (7/9). For S2, cells with an ID response were colocalized with cells lacking an ID response in both the anteroposterior and the mediolateral dimensions of the recordings in both monkeys. The only indication of some topographical localization in S2 concerned the small group of cells categorized as having a complex response pattern (e.g., Fig. 5B): 7 of 8 were located rostrally in S2 (4 cells from each monkey).

Figure 6 shows population histograms for the ID discharge seen in S1 (A) and S2 (B), grouped according to the sign of the ID response and the sign of the subsequent standard response (+ or − relative to the discharge rate during the final 500 ms of the ID period). For comparison, we also show the discharge of no-ID cells (only the standard + cells are shown here because these made up the majority of no-ID cells in S1 and S2; see Table 3). Separate averages were generated for the tactile (black) and visual (red) trials. As detailed in Table 3, the sign of the response during the ID period predicted the sign of the response during the subsequent standard period in the majority of cells in both S1 and S2 (+/+ or −/−). For these cells, there was little difference across S1 and S2 in the population histograms, with the exception that attentional influences were evident only for the S2 cells (+/+, tactile > visual during both the ID and standard periods). The opposite pattern (+/− or −/+−) was less frequently observed (Table 3) and tended to be particularly characteristic of S2 (illustrated here). Finally, the no-ID/standard + cells in both S1 and S2 had significantly weaker responses during the standard period than did the corresponding ID+/standard + cells ($P < 0.01$) (see following text).

Inspection of Fig. 6 indicates that the changes in discharge frequency during the ID period were modest in both S1 and S2,
but sustained throughout the instruction period. The absolute change in discharge rate was calculated relative to the reference period (hold, complete ID recorded; static, incomplete ID); for this, all rewarded trials were pooled (tactile, visual). For the 47 cells with an ID response in S1, the median change in discharge was modest, 5.7 imp/s (range, 2.5 to 39.5 imp/s). The median change in discharge in S2 was similar to that seen in S1, 5.4 imp/s (2.5 to 26.5 imp/s, Mann–Whitney, $P = 0.73$). When the tactile
and visual trials were considered separately, however, S2 cells showed a wider range of changes than did S1, reflecting the contribution of attentional influences (Fig. 5A; also see the following text). For S1 and S2, the magnitude of the ID response corresponded to, respectively, 28 and 39% of the subsequent response to the tactile stimulus during the standard period (analysis restricted to cells with increased discharge during the ID and standard periods).

Finally, latencies for the appearance of an ID response are summarized in Fig. 7. The median latencies were similar in S1 (230 ms) and S2 (200 ms). A comparison across the two areas revealed no difference (Mann–Whitney, \( P = 0.46 \)); however, S2 was characterized by a wider range of onset latencies including the shortest latency encountered, 40 ms, and the longest, 1,420 ms. For neither region was there any difference overall in the latencies during the tactile and visual trials (S1, \( P = 0.53 \); S2, \( P = 0.88 \)), although individual cells sometimes showed a difference.

**Influence of ID discharge on neuronal responsiveness to scanned texture**

At a general level (population histograms, Fig. 6), there was evidence that the presence of an ID response modified the

### Table 2. Database from S1 \((n = 116)\) and S2 \((n = 125)\)

<table>
<thead>
<tr>
<th></th>
<th>S1 (Texture)</th>
<th>S1 (Not-Texture)</th>
<th>S2 (Texture)</th>
<th>S2 (Not-Texture)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ID present</td>
<td>24 (14)</td>
<td>23 (9)</td>
<td>32 (23)</td>
<td>26 (20)</td>
</tr>
<tr>
<td>ID absent</td>
<td>26 (14)</td>
<td>43 (12)</td>
<td>27 (16)</td>
<td>40 (31)</td>
</tr>
</tbody>
</table>

Numbers in parentheses indicate the number of cells recorded with a complete ID period (S1, 49/116; S2, 90/125).

### Table 3. Sign of the neuronal response during the ID period (relative to the reference level) and during the standard period (relative to the final 500 ms of the ID period) in S1 and S2

<table>
<thead>
<tr>
<th>ID/Standard</th>
<th>S1 ((n = 116))</th>
<th>S2 ((n = 125))</th>
</tr>
</thead>
<tbody>
<tr>
<td>ID cells</td>
<td></td>
<td></td>
</tr>
<tr>
<td>+/+</td>
<td>35</td>
<td>37</td>
</tr>
<tr>
<td>+/-</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>+/0*</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>–/+</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>–/0*</td>
<td>6</td>
<td>11</td>
</tr>
<tr>
<td>–/0*</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>No-ID cells</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0/+</td>
<td>58</td>
<td>54</td>
</tr>
<tr>
<td>0/–</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>0/0*</td>
<td>6</td>
<td>3</td>
</tr>
</tbody>
</table>

*For these cells, there was no significant modulation during the standard period (standard vs. reference); all showed modulated discharge during later periods of the trials.

**Fig. 6.** Population histograms for cells in S1 (A) and S2 (B). Cells were grouped on the basis of the sign of the ID response relative to the discharge during the initial hold period (from left to right: ID+, ID−, and no-ID) and the sign of the response evoked during the Standard period (+ or − relative to the final 500 ms of the ID period). Data from the tactile and visual tasks are shown superimposed: black, tactile; red, visual (bin width, 25 ms). Attentional effects during the ID period, mainly in S2, were mixed (tactile > visual or vice versa) so that the pooled data show little separation across the 2 tasks.
subsequent neuronal responsiveness to the scanned texture during the standard period. Thus the standard responses in both S1 and, especially, S2 were larger in cells with an ID+ response than in cells without an ID response (compare far left and right histograms in Fig. 6). For the S1 data, peak discharge was identical but discharge in the period after the initial phasic response was significantly lower in the no-ID cells (ID+, 36 imp/s; no-ID, 25 imp/s; \( P = 0.009 \)). For the ID− cells, there was a similar trend for the evoked suppression during the standard period to be greater than that found for no-ID cells, showing a pattern of suppressed discharge during the standard period (not shown).

At a single-cell level, trial-by-trial analyses for individual neurons showed that the response evoked by the standard stimuli often covaried with the level of discharge in the preceding ID period. Examples are shown in Figs. 8 (S1) and 9 (S2); quantitative analyses are summarized in Fig. 10. The rasters in Figs. 8 and 9 have been rearranged in increasing order (from top to bottom) of the mean discharge rate during the instruction period. The area 1 cell in Fig. 8 had larger-amplitude responses to the standard stimuli in trials in which the ID response was well developed, compared with those trials with a weak or absent ID response (compare bottom two histograms, right). This observation was quantified using linear regression analyses (mean discharge rate during the standard period vs. ID period in each trial). The results (Fig. 10A) indicated that this cell had a significant positive correlation (\( P < 0.0005 \)). In contrast, the S2 cell illustrated in Fig. 9 showed no obvious change in the amplitude of the discharge during the standard period as a function of the preceding ID response (compare bottom two histograms, right), even though there was a twofold variation in the magnitude of the ID response. For this neuron, the linear regression was nonsignificant (Fig. 10F). Significant relations, however, were obtained in other S2 cells (e.g., Fig. 10E, corresponding to the cell shown in Fig. 5A).

This regression analysis was extended to all cells (complete ID recording) and the frequency histograms in Fig. 10, C and G summarize the distributions of the coefficient of determination, \( r^2 \) (tactile and visual tasks pooled), which gives a measure of the proportion of variability in cell discharge accounted for by the regression. For each cortical region, the analysis was applied to cells with an ID present as well as those without an ID response. For S1 (Fig. 10C), cells with an ID response were more likely to show a significant relation (filled area) than those without an ID response (74 vs. 42%; \( \chi^2 \) test, \( P = 0.026 \)). Nevertheless, some S1 cells with an ID response, as found in S2, failed to show a significant relation (Fig. 10B, ID response shown in the inset). Similar results were obtained for the S2 cells (Fig. 10G), although the difference was less marked (respectively, 77% of the ID cells vs. 55% of the no-ID cells, \( P = 0.033 \)). For S1, but not S2, the \( r^2 \) values were higher for the ID present cells compared with ID absent (Mann–Whitney tests, \( P = 0.003 \) and \( P = 0.56 \), respectively).

Although the ID response modified the initial response to the standard textured surface (2-mm SP) + standard light intensity, this effect did not carry over into the salient texture period (Fig. 10, D, S1 and H, S2), corresponding to the period in which the digits encountered the modified texture (3.9- or 4.9-mm SP). Few cells in either S1 (7/49) or S2 (14/90) showed a significant relation between the discharge rates during the salient texture \( \Delta \) period and the corresponding ID period. This is reflected in lower values for \( r^2 \).

The above-cited analyses were based on correlating cell discharge rate measured during the entire 2-s ID period with that measured during different epochs in the subsequent stimulation period. Use of the entire ID period could be criticized because, conceivably, one could argue that it was the final portion of the ID period that was of greatest behavioral significance. To address this, we repeated the aforementioned analyses (Fig. 10), this time restricting the ID analysis window to the final 500 ms of the ID period (i.e., just prior to the onset of the standard stimuli). The results showed that \( r^2 \) values were significantly lower than those for the analysis based on the entire 2-s ID period (\( P < 0.0005 \) for S1 and S2). Consistent with this, the proportions of cells with a significant relation were reduced, although the differential remained, with more ID cells showing a significant relationship with the evoked responses than the no-ID cells. Given the lower \( r^2 \) values, this approach was not retained for further analyses.

One question about the relation between the initial response to the textured surface and the ID response, however, was the potential link with the overall excitability of the cell as reflected in the level of spontaneous discharge during the reference period (initial hold, Fig. 1). On a population level, we found no significant difference in baseline discharge rates during the reference period in S1 and S2 (respectively, 12.7 and 15.3 imp/s, \( P = 0.26 \)). Nevertheless, the ID discharge of...
some cells (area 1 cell illustrated in Fig. 8) covaried with the reference discharge \((P < 0.0005)\). In contrast, the S2 cell shown in Fig. 9 showed no such dependence \((P = 0.16)\). This analysis was extended to all cells (complete ID recording). A high proportion of cells showed a significant relation between the discharge during the ID period and the reference discharge, with S1 cells being more likely to show a significant relation than S2 \((S1, 42/49;\ S2, 60/90;\ \chi^2\ test,\ P = 0.015)\). These results suggested that changes in the overall excitability of the cortical cells may have contributed to the results. But close to one half of the cells with a significant relation (“ID” vs. baseline) were no-ID cells \((S1, 20/42;\ S2, 27/60)\). Furthermore, we found no evidence that the presence of a significant relation during the ID period versus reference—subsequently predicted the occurrence of a significant relation with the evoked discharge during the standard period \((\chi^2\ test\ applied\ to\ the\ pooled\ data\ from\ S1\ and\ S2,\ P = 0.95)\). Together we suggest that simple changes in cortical excitability cannot explain the present findings.

Behavior and the ID response

In both S1 and S2, there was considerable variability in the magnitude of the ID response across the rewarded trials. S1 cells were characterized by the frequent absence of the ID response in many trials. A typical example is shown in Fig. 8: there was no obvious ID response in the trials at the top of the main raster (trials rearranged in increasing order of the discharge rate during the ID period); other trials (bottom) showed a well-developed ID response. S2 cells, in contrast, tended to show more consistent responses during the ID period, but even so response amplitude often varied (twofold change in the example shown in Fig. 9). Overall, S1 was characterized by the frequent absence of an ID response in the trials with the lowest discharge rate \((17/23, 74\%)\). In contrast, only 21% of S2 cells failed to show any modulation during the ID period in such trials \((\chi^2\ test,\ P < 0.0001)\). Taken together, these observations suggest that correct behavioral responses were not dependent on the presence or absence of the ID response.

This suggestion was supported by the results of linear regression analyses applied to the cells with an ID response to determine whether RT to the signaled modality varied systematically with the level of discharge during the ID period (data from the two tasks were analyzed separately because of systematic differences in RT). No significant relation was observed either in the single-cell example shown in Fig. 8 (tactile, \(P = 0.65\); visual, \(P = 0.13\)) or across the population of S1 ID cells. Similar results were obtained in S2, both for the example shown in Fig. 9 (tactile, \(P = 0.65\); visual, \(P = 0.41\)) and for the S2 sample of ID cells. Together, the results indicated that the level of discharge during the ID period did not systematically modify RT to the signaled modality.

Further evidence that ID discharge was not predictive of behavior comes from analyses of the small numbers of error trials recorded in these tasks. As previously reported (Meftah
et al. 2002), these highly trained monkeys made few errors (7% of trials in monkey G, 4% in monkey I). For the example shown in Fig. 8, there were three unrewarded trials recorded; all represented errors in the timing of the motor response (too early or late). Inspection of the raster (top left) and histogram (top right) suggests that the ID response was present in at least one of these trials. This observation reinforces the suggestion that the ID response in S1 was not directly linked to correct detection of the signaled modality. For the S2 cell illustrated in Fig. 9, two types of error trials were recorded (top two rasters): trials with no response, and those with incorrectly timed responses. The ID response was less evident in the no response trials, but arguably still present. Moreover, ID discharge appeared to be better developed in the error trials with unrewarded responses than in some of the correct trials (compare middle two histograms, right). Thus even in S2, there was some evidence that ID discharge did not predict correct responses.

**Attention sensitivity during the ID period**

For all cells, discharge rates during the ID period were compared across the tactile and visual trials to determine whether cell discharge was differentially modulated by the direction of attention. For the analyses restricted to the final 500 ms of the ID period (entire database) and as previously reported in Meftah et al. (2002), only a fraction of S1 cells (7% of cells with an ID response, 3/44; 2.6% of the entire sample, 3/116) were classified as attention sensitive during the ID period (independent t-test). Consequently, the population hist-
tograms for the tactile (black) and visual (red) trials are virtually identical (Fig. 6A). Indeed, only one cell contributing to these histograms was categorized as attention sensitive in the ID period (tactile/H11022 visual, ID/H11001).

In contrast, a higher proportion of S2 cells (30%, 17/56 of the ID cells, or 14% of the entire sample, 17/125) were attention sensitive during the ID period. Considering the cells with an ID/H11001 response pattern, the population histograms (Fig. 6B, left) show a trend for discharge rates to be higher in tactile than in visual trials, but this effect was modest and more obvious in the subsequent standard period. The difference did not show clearly on the population histograms because the effects were mixed in sign: of the ID/H11001/Standard/H11001 cells contributing to the population histograms in Fig. 6B, 50% were attention sensitive (12/24), with 8 showing higher discharge rates when attention was directed to the tactile modality and 4 showing the opposite pattern. For the ID/H11002 cells (middle), the large majority of cells were not sensitive to attention (10/13). The presence or absence of attentional effects in each cell was confirmed using a distribution-free statistic based on the area under the ROC curve (see METHODS). The exact time course for attentional modulation will be addressed in a subsequent publication, but when attentional effects were present, a clear separation was generally evident over the final 1 s of the 2-s-long ID period (also evident in the population histograms).

Our analysis criteria modestly underestimated the proportion of attention-sensitive S2 cells because we required that the effect be present in the 500-ms period immediately preceding the stimulation period, reasoning that this was likely of greatest behavioral significance. In fact, a further four cells were transiently attention sensitive in earlier portions of the ID period (from 250 to 1,500 ms).

Figure 11 summarizes the distribution of attention-sensitive cells in S1 and S2 (shaded region) in relation to their categorization as ID/H11001, ID/H11002, or no-ID. For comparison, we also show the distribution of attention-sensitive responses in the subsequent two periods of the task (same database): the standard and salient Δ texture periods (Chapman and Meftah 2005; Meftah et al. 2002).

As reported previously, attentional effects were rare in S1 and mainly concentrated in the salient Δ texture period. S1 cells with an ID response showed a tendency to be more frequently attention sensitive in this critical period than did the no-ID cells ($\chi^2$ test,
Having the final histology from one of the monkeys now, we report that attentional effects in S1 were concentrated in area 1: 70% of neurons with attentional effects in one of the three analysis periods were located in area 1 (83% if area 3b/1 cells are included). As a proportion of the total number of cells recorded, 35.6% (16/45) of area 1 cells and 16.7% (3/18) of 3b/1 cells were attention sensitive, compared with only 8.3% (3/36) of area 3b and 5.8% (1/17) of area 2 cells. Attentional effects were more frequent in S2 than in S1, but followed the trend of being more frequent in the standard and salient texture periods than in the ID period (Meftah et al. 2002). Only S2 cells with an ID response showed attentional modulation during the instruction period and these effects were superimposed on the ID response (example in Figs. 5A and 10F). S2 cells with an ID response were more frequently attention sensitive during the two subsequent task periods (standard, salient texture) than those that did not show an ID response (χ² tests, P < 0.01 in each case).

Overall, cells with an ID response were more likely to be attention sensitive in at least one of the task periods than cells without an ID response (χ² tests: S1, P = 0.026; S2, P = 0.001). This finding suggests that there is a link between the presence of an ID response and attention sensitivity during later epochs of the trial in both S1 and S2, but the presence of an ID response did not predict attention sensitivity.

Finally, we examined the data to see the extent to which texture sensitivity was associated with the presence or absence of an ID response (Table 2). In S1 and S2, texture sensitivity was equally likely in cells with or without an ID response (χ² tests, S1, P = 0.097; S2, P = 0.153). Texture sensitivity in S1, however, was not uniform: area 1 contained the highest proportion of texture-sensitive cells (58%, 26/45), and area 2 the lowest proportion (18%, 3/17). Areas 3b (33%, 12/36) and 3b/1 (39%, 7/18) were intermediate.

Psychophysical experiment in humans: tactile detection during the ID period

Our analyses of the neural data indicated that the discharge during the ID period modified responses to the subsequent standard texture, but the effects did not carry over to the salient Δ texture period. It seemed logical to think that the ID discharge might enhance detection of tactile stimuli presented during the instruction. This hypothesis was investigated by testing the ability of human subjects (n = 9) to detect weak electrical stimuli during the ID period of this same cross-modal attention task. Stimuli were applied to D3, the same digit to which the textured surfaces were presented. The results are shown in Fig. 12A: the detectability index d' is plotted as a function of the stimulus delay and the modality to which attention was directed. Detection of the near-threshold electrical stimulus was better when attention was directed to the tactile modality (black), mean d' = 1.803, compared with the visual modality (gray), mean d' = 1.323. Performance in the visual trials was close to 75% detected (d' = 1.35, dotted line) (the experimental design was such that 50% corresponds to performance at chance levels). A repeated-measures ANOVA showed that modality (tactile vs. visual), but not delay, was a significant factor (respectively, P = 0.037 and 0.861). The threshold estimate (P70, intensity at which 70% of the electrical stimuli were detected; measures

FIG. 11. Summary of the relative numbers of ID (+ or −) and no-ID cells in S1 and S2 in relation to the presence (shaded) or absence (unshaded) of attentional modulation during 3 analysis periods: Instruction, Standard, and Salient Δ texture.
made outside the context of the task) showed no change across the experimental session (Fig. 12B). These results are consistent with the existence of a process that was present within 200 ms of the instruction onset and lasted throughout the 2-s instruction period, i.e., very similar to the time course for the ID discharge described in the recordings from S1 and S2 cortex.

**Discussion**

During the 2-s instruction period that preceded a directed-attention task, we found that roughly 40% of S1 and S2 cells with a cutaneous receptive field on the stimulated finger tips developed a sustained increase or decrease in discharge that continued up to the start of the subsequent tactile or visual task. This discharge began as early as 40 ms after the onset of the instruction cue (medians of 200 and 230 ms) and modified neuronal sensitivity to the subsequent standard stimuli. ID responses, however, were not tightly linked with attentional modulation, suggesting that the underlying mechanisms for ID discharge and attention are independent. Given that tactile detection in humans was also increased over the same time period, this discharge may represent a priming mechanism that prepares cortical areas to receive and process sensory inputs, with the enhanced detection seen during the tactile trials reflecting the added action of selective attention.

**Instructed delay discharge in somatosensory cortex**

One of the most striking observations here was that the instructed delay responses seen in S1 and S2 were very similar in most respects (frequency, sign, latency, amplitude), strongly suggesting that this discharge shares a common origin (discussed further in the following text). This finding contrasts with our previous results in these same monkeys, to the effect that attention has differential effects on neuronal discharge in S1 and S2 with attentional influences being more frequent, larger, and more complex in S2 than in S1 (Chapman and Meftah 2005; Meftah et al. 2002).

There were two major differences in the ID response in S2 compared with S1. First, one response pattern was unique to S2, the complex response pattern. This consisted of an initial phasic response to the instruction cue, which appeared to be stimulus related, and so presumably visual in nature, along with a sustained suppression during the ID period (Fig. 5B). These cells were frequently sensitive to both attention and texture (6/8), suggesting that they may play an important role in signaling task-relevant information. Second, the ID responses in S2 were less variable than those in S1. This could not be explained by any systematic difference in general neuronal excitability since discharge rates during the reference period were similar for S1 and S2. Although this observation might argue against a common source for the ID discharge, we believe that the result reflects the general absence of attentional effects in this period in S1 versus S2 (7 and 30% of the ID cells). The attentional influences appeared to be additive to the ID response (e.g., Figs. 5A and 10F). In support of the latter suggestion, we found no cells in either S2 or S1 with a response during the ID period uniquely associated with one of the instruction cues (tactile or visual).

There was also evidence for regional localization of the ID responses. In S1, such responses were particularly frequent in area 1, where 50% of the cells had an ID response. Taken together with observations that attention sensitivity in S1 was almost entirely restricted to cells in area 1 and the adjacent 3b/1 border zone (83%), and that the proportion of texture-sensitive cells was highest in area 1 (58 vs. 31% elsewhere), these results suggest that area 1 must play a privileged and higher-order role in the analysis of tactile inputs compared with area 3b. As for area 2, our sample size was too small for any speculation. ID responses in S2, in contrast, were not obviously localized, apart from the complex S2 cells that were, in both monkeys, restricted to the more rostral part of the sampled region, possibly within the parietal ventral (PV) somatosensory area (Krubitzer et al. 1995), rather than in S2 proper. Such cells most likely receive visual input relayed through area 7b (Disbrow et al. 2003; Friedman et al. 1986), leading to the suggestion that this region of the lateral sulcus should be regarded as multisensory.

**Comparison with previous studies**

This study, in contrast with previous work (see Introduction), examined ID discharge within the context of sensory discrimin-
ination tasks. Earlier studies were more restricted (S1 only) and were, in contrast to this study, undertaken in relation to motor tasks: sensory cues presented during the ID period provided information that aided the animal to plan and execute a movement following a (later) GO signal. Published results are contradictory. Crammond and Kalaska (1989) found little evidence for ID discharge in S1: only 5% of S1 cells (area 2) developed an ID response, compared with 55% in the adjacent area 5 (same animals). In contrast, we found that 35% of area 2 cells had an ID response. More recently, Liu et al. (2007) reported that 65% of S1 cells (location not specified) show modulated discharge during an instructed delay period that preceded a motor task. These effects were different from those reported here in that a pattern of decreased discharge predominated, around 60%, compared with the opposite finding here (increased discharge predominated in S1 and S2). In addition, some of their trials had no explicit instruction given at the start of the ID period and yet discharge was modulated during the “delay” period that preceded the GO cue. This makes it likely that the ID discharge was more likely related to planning the upcoming movement. It is not clear how these results can be reconciled with those of Crammond and Kalaska (1989), but several factors may have contributed, including differences in the S1 regions sampled (e.g., hand vs. shoulder representations; areal localization of the recorded cells) and differences in the design of the motor tasks.

We believe that the ID discharge reported here was specific to these sensory discrimination tasks performed in the context of a directed-attention task and not directly related to the motor response that was generated in each trial. The strongest evidence for this came from a consideration of the ID responses in S1. For the cell shown in Fig. 8, for example, the ID response was clearly absent in many of the rewarded trials, i.e., trials in which the motor response was made. There was also some suggestion of an ID response in the no response trials shown for the S2 cell in Fig. 9. We cannot, however, exclude the possibility that the animal had planned to respond in these few error trials. Further experiments are needed—and this using formal GO and NO-GO trials as used in studies of ID responses within the context of the planning and execution of voluntary movements (Kalaska and Crammond 1995; Petrides 1986; Wise et al. 1983). If the ID discharge seen here simply reflected motor planning, however, we would have expected that RT to the signaled modality would vary with the level of ID discharge, but this was not found. Our suggestion that the ID response was specific to this directed-attention task is supported by observations (S. Bourgeon, A. Dépeault, E.-M. Meftah, and C. E. Chapman, unpublished data) made during S1 recordings in two other monkeys. These animals performed either a simple texture- or visual-discrimination task: both tasks were preceded by a noninformative warning period (2-s-duration light) and both tasks required a motor response at the end of the presentation of a moving textured surface. There was virtually no evidence of discharge during the warning period in either animal.

Baseline changes in discharge rate

The present results are complementary to several previous reports that unimodal sensory cortical areas show evidence of baseline changes while anticipating the arrival of sensory stimuli. Luck et al. (1997) reported maintained changes in the firing rate of V2 and V4 neurons while monkeys attended the appearance of a visual stimulus in the cell’s receptive field. Interestingly, the proportion of modulated cells was of the same order as found here (30%). Although they found no modulation in V1, more recent imaging studies in humans have reported baseline effects in V1 (Kastner et al. 1999; Ress et al. 2000). The latter studies also confirmed that these effects were widespread, involving other visual processing areas (e.g., V2 and V4), as well as other regions (superior parietal lobule, frontal eye field, and supplementary eye field). Although the baseline effects shared some similarities with visual attention (spatially selective dependence on task difficulty), Kastner et al. (1999) suggested that the baseline and attentional effects on visually evoked responses were not tightly coupled and argued for different sources for these effects. The present results provide direct support for this suggestion since the attentional effects in this task were independent of underlying changes in baseline discharge rate (i.e., the ID response). Moreover, the amplitude of the ID response here, 28–39% of the response evoked by the subsequent tactile stimulus, is of the same order of that reported by Kastner et al. (35–50% of the response to the subsequent visual stimuli). This suggests that the basic underlying phenomena are likely the same.

Independence from attentional influences

Although ID responses were found with equal frequency in S1 and S2, attentional influences during the instruction period were almost entirely restricted to S2 cells. Moreover, these attentional influences were always superimposed on the baseline changes in discharge associated with the presentation of the instruction cues. Thus whereas there was a link between the two effects in that attentional influences were never seen in isolation from the ID response, the differential distribution of the attentional effects (S2 >> S1) suggests that the underlying sources of these influences (ID, attention) were independent.

We also found that cells with an ID response were more likely to be attention sensitive in later epochs of the trial, including the standard and salient Δ texture periods. Nevertheless, attention sensitivity later in the trials was not restricted to cells with an ID response (Fig. 11). Together the results suggest that these two mechanisms target partly overlapping populations of cells in S1 and S2. The discharge of all of these cells was modulated during the stimulation period of the task—i.e., while tactile and visual stimuli were presented. Thus it seems reasonable to suggest that these two mechanisms, ID discharge and attentional modulation, work together to enhance the neuronal processing of tactile inputs in these two somatosensory cortical regions. Since ID responses were not restricted to texture-sensitive cells (Table 2), the interactions are likely to be complex.

Cross-modal responses in unimodal sensory areas

S1 and S2 cortex are both regarded as unimodal sensory areas and presumed to be exclusively engaged in processing somesthetic stimuli. Why would they respond to visual instruction cues? Whereas earlier studies of cross-modal integration focused largely on higher level association cortices and midbrain structures (e.g., superior colliculus) as potential targets
for interactions across modalities (Stein 1998; Stein and Wallace 1996), more recent evidence indicates that interactions can occur in what are regarded as unimodal sensory areas (reviewed by Ghazanfar and Schroeder 2006; Kayser and Logothetis 2007). For example, somatosensory and visual inputs have been shown to activate neurons in auditory association cortex (Fu et al. 2003; Schroeder and Foxe 2002; Schroeder et al. 2001). Indeed there is now anatomical evidence that visual association cortices project to areas 3b and 1 in the marmoset (Cappe and Barone 2005), thus providing a direct anatomical substrate for the present findings. Posterior parietal cortex is a likely source of visual inputs to S2 (Disbrow et al. 2003; Friedman et al. 1986). By virtue of the reciprocal connections between S2 and S1 (reviewed in Burton 1986), visual inputs may also be relayed indirectly to S1.

Although earlier single-unit recordings in S1 found little or no evidence that S1 neurons are activated by either visual or auditory stimuli, these recordings were made in the context of tasks in which the sensory cue triggered either a movement or a tactile exploration (Ageranitoti-Belanger and Chapman 1992; Bioulac and Lamarre 1979; Chapman and Ageranioti-Belanger 1991; Nelson 1988). Since the sensory stimulus served as the GO cue, there was little chance to see the types of responses reported here since their latencies are relatively long, close to the RTs expected for movements triggered by teleceptive cues (Spidalieri et al. 1983).

Recently there have been several reports of discharge in S1 elicited by visual stimuli presented well in advance of the subsequent task, thus providing enough of a delay to see the responses independent of any other event. Zhou and Fuster (1996, 1997, 2000) reported that 30–50% of S1 cells (recorded mainly in the hand representation) responded to an informative visual cue that preceded a visuohaptic delayed match-to-sample task. The sign of modulation was similar to that seen here (70% increased discharge; 30% decreased discharge) and, as also found here, many of these cells were modulated during the subsequent haptic exploration. Further comparisons with the present results, however, are hampered by the lack of information regarding the areal location of the cells tested with the visuohaptic task, the amplitude of the observed responses, along with the receptive field properties of the cells (modality, location). Based on their observation that a proportion of these cells (~14% of the sample) showed sustained changes in discharge during the lengthy delay period (~20 s) that preceded the haptic exploration, they suggested that the discharge represented a short-term memory trace, similar to that seen in a wide range of other cortical regions, including inferotemporal, posterior parietal, and prefrontal cortex (de Lafuente and Romo 2006; Fuster 1990). Their suggestion, however, has been challenged by the results of Salinas et al. (2000) who found no evidence for S1 discharge during the delay period that separated two observation intervals in a vibration-discrimination task.

**ID responses and behavior in monkeys**

The results showed that task performance (success or failure, RT) was independent of the presence or absence of an ID response, particularly in S1 where ID discharge was frequently absent in many rewarded trials. This observation was supported by anecdotal observations of the few error trials recorded in these monkeys.

The absence of any link between the ID responses in S1 and behavior is consistent with this area being situated at a relatively low level in the hierarchical processing of tactile inputs, and thus minimally involved in the sensory decision process. Thus a number of previous studies have found little or no link between S1 tactile responsiveness and success or failure in sensory-discrimination tasks (e.g., Ageranioti-Belanger and Chapman 1992; Chapman and Ageranioti-Belanger 1991; Salinas et al. 2000; Tremblay et al. 1996). In contrast, there is persuasive evidence that S2 is more closely linked to the sensory decision process (Jiang et al. 1997; Pruett et al. 2001; Romo et al. 2002), so that the lack of any link here was more surprising. Clearly, the recordings need to be extended to a situation in which more error trials occur (e.g., using smaller increments in SP for the textured surfaces) to address this important issue. Alternately, and given the relatively long delay between the end of the instruction period and the time of the earliest change in stimulus intensity, it could be that the ID period—corresponding to the time taken to perceive and respond to the change in surface texture—might be better predicted by some form of weighted function of discharge levels during the ID period and the amplitude of the evoked tactile signal.

**Function of ID discharge**

In the present study, S1 neurons responding to the visual instructions rarely showed differential responses related to the direction of attention toward the tactile or the visual task. Interestingly, few S1 cells (~4% of the sample) in the studies by Zhou and Fuster (1996, 1997, 2000) showed differential discharge in relation to the initial visual cues that instructed the animal as to the tactile stimulus that would be rewarded in the task. Importantly, our task had no memory requirement because the instruction cue was on throughout the entire trial. One element, however, was common to this study and those of Zhou and Fuster: the instruction period preceded a sensory-discrimination task. We suggest that the results can be reconciled if the discharge during the ID period reflects not a tactile memory, but rather priming of somatosensory cortex within the context of a sensory-discrimination task to enhance neuronal sensitivity to tactile stimuli, or what could be termed “sensory set.” This mechanism is different from selective attention, because it is nonselective, independent of the cued modality. Indeed, we predict that similar results would be obtained if the modality of the instructions were changed, such as replacing the colored lights with different frequencies of auditory cues. We favor the idea that the modulation is driven by the activation of a “top-down” effect triggered by the instruction, but cannot exclude the possibility that there is an element of multisensory integration involved as well (see following text).

Results from the complementary psychophysical experiments here support the idea that priming assists sensory processing: we showed that tactile detection was significantly increased throughout the ID period, with detection being enhanced within 200 ms after the onset of the instruction cue. This latter observation was surprising since the median onset latency for the ID response in S1 and S2 was 200–230 ms; although earlier ID responses were observed (40 ms), these were rare and not well developed. It should be stressed,
however, that the detection process requires some finite time so that this modest discrepancy can be resolved in favor of the existence of a priming mechanism if the ID discharge acted to enhance later neuronal processes involved in tactile perception. Such a suggestion would be compatible with our psychophysical results since the subjects did not make their report until after the end of the trial (~5 s after the electrical stimulus). The added observation that detection of the near-threshold electrical stimuli was specifically enhanced during the ID period of the tactile trials is consistent with our observation of attentional influences during the ID period. Attentional effects during the ID period, however, were almost entirely restricted to S2. Taken together, these observations are consistent with the view that S2 is located at a hierarchically higher level than S1 in the processing of tactile stimuli (Chapman and Meftah 2005; Jiang et al. 1997; Romo et al. 2002).

In our directed-attention task, we believe that the ID discharge reflects preparation to perform the appropriate sensory discrimination task, with the animal being totally engaged in the task once the competing stimuli began (onset of the standard period). The ID discharge significantly modified the initial response to the standard stimuli in both S1 and S2; surprisingly, this effect did not extend into the salient texture Δ period. Moreover, texture-sensitive cells were not more likely to have an ID response than were the non-texture-sensitive cells (Table 2). Although the latter finding may reflect an underestimation of the numbers of texture-sensitive cells (small range of SPs tested), it seems more likely that not all of the texture-sensitive neurons actually contributed to the performance of the task. Since we also found that cells with an ID response were more frequently attention sensitive than those without an ID response in both S1 and S2, these observations, taken together, may well reflect the fact that only relatively small groups of neurons are essential for the performance of this texture-discrimination task.

Texture is a complex multidimensional stimulus dependent on the physical characteristics of multiple tactile elements (e.g., grains of sand for abrasive papers or, as used here, raised dots). Moreover, all of the cutaneous mechanoreceptive primary afferents involved in discriminative touch are coactivated when textures are scanned, providing rich but complex signals. One major question is to determine how such complex stimuli are represented in the brain. Although elementary features, such as orientation in the visual system, are thought to be encoded by a population code, it is not clear how the information is combined into a single unambiguous percept (the binding problem). More complex features are thought to be encoded by small numbers of highly selective cells (e.g., face-specific cells in inferotemporal cortex; Gross et al. 1969, 1972), or what is referred to as sparse coding. This is an attractive idea since information is unambiguously encoded. Moreover, cells may have discharge properties consistent with both types of coding—e.g., sensitive to certain faces, but also providing general information about face shape (Reddy and Kanwisher 2006). There is already evidence for the existence of highly selective cells in S1, e.g., area 2 shape-sensitive cells (Iwamura and Tanaka 1978). One interesting speculation is that the priming mechanism may be one of several mechanisms to recruit neurons that are functionally important for the task.

Another possibility is that the ID discharge may represent a central signal related to the expected input. These were highly trained animals that had experienced these same sensory inputs over thousands of trials. In this scenario, the ID response might reflect a process of mentally rehearsing the expected input, thus representing an internal model of the expected input (Chelazzi et al. 1993). This model might then be compared with the actual sensory inputs to update the current estimate of the sensory state and thus generate the discrimination response. This suggestion, inspired by similar such proposals in the motor field (e.g., Vaziri et al. 2006), leads to the prediction that the behavioral response should reflect some combination of the ID response and the tactile feedback. Further experiments, using near-threshold increments in SP, are necessary to further address this issue.

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