Non-spatial, motor-specific activation in posterior parietal cortex

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A localized cluster of neurons in macaque posterior parietal cortex, termed the parietal reach region (PRR), is activated when a reach is planned to a visible or remembered target. To explore the role of PRR in sensorimotor transformations, we tested whether cells would be activated when a reach is planned to an as-yet unspecified goal. Over one-third of PRR cells increased their firing after an instruction to prepare a reach, but not after an instruction to prepare a saccade, when the target of the movement remained unknown. A partially overlapping population (two-thirds of cells) was activated when the monkey was informed of the target location but not the type of movement to be made. Thus a subset of PRR neurons separately code spatial and effector-specific information, consistent with a role in specifying potential motor responses to particular targets.

Posterior parietal cortex (PPC) seems to process spatial information in a way that is strongly influenced by how that information will be used[1–3]. The response to a visual and auditory stimulus in a cell's receptive field is often enhanced when that stimulus is behaviorally relevant[3–9]. The enhancement is not merely all or none; activity in the lateral intraparietal area (LIP), for example, seems to be continuously modulated by the return (reward) associated with a given target[10].

Initially, PPC was thought to reflect a supramodal spatial saliency map whose output can be used by different effectors[3,4]. More recent experiments have shown that there are multiple maps with outputs associated with particular effectors[11–15]. This result does not replace the idea that behavioral enhancement in PPC reflects task relevance. Rather, it demonstrates that one aspect of task relevance is the choice of effector for the response. We call this an 'effector-specific intention'. For example, memory-related activity for a particular target in LIP is more robust when the subject will respond with an eye movement rather than with an arm movement, whereas the reverse pattern occurs in the parietal reach region (PRR)[12]. Thus, enhancement can depend on the choice of which effector to use.

Here we asked whether effector-specific intentions might by themselves, without spatial information, nonetheless drive sustained activity in PRR. The responses of neurons have been examined when both effector and spatial information were known[12,15]. Now we ask whether effector-specific intention is only modulatory, or if it can drive a response all on its own. A response from effector-specific intention alone would be unexpected, given the prevailing view that the dorsal visual pathway, of which PPC is a part, is dedicated to processing spatial information[16,17]. The presence of non-spatial, effector-specific information in PPC would also be at odds with its classical role as a purely sensory association cortex. In contrast, non-spatial, effector-specific intention activity might be expected if the dorsal stream is more active in sensorimotor transformations, and in particular, if PPC serves in part to combine relevant spatial information with specific motor intentions[11–15]. Such a role is accepted for the premotor cortex[18], which receives input from PPC[19–21], but the appropriate experiments have not been performed in PPC.

We also wished to know whether spatial information would drive activity without effector information. In previous studies, animals are often trained to use only one effector. When training involves multiple effectors, different movements are typically segregated in discrete blocks, which provides implicit effector information[22–25]. In some sense, effector information is absent in go/no–go tasks, in which animals receive a spatial target before the instruction to make or withhold a movement[26]. But even here, only a single effector is involved. Therefore we do not know whether spatial information, without a clear effector-specific plan, drives activity in PPC.

To assess the impact of effector information without spatial information and spatial information without effector information, we trained monkeys on a task where a delay was interposed between the instruction of what movement to make (reach or saccade) and the target location for the movement. On some trials, we first instructed the type of movement to prepare, and then after a delay we presented the target for the movement. This allowed us to test whether PRR neurons distinguished between reach and saccade trials, in the interval after receiving the effector-specific instruction but before receiving the target. On other trials, we revealed the location of the spatial target and after a delay gave the effector-specific instruction. This allowed us to assess the impact of knowing the spatial target of the movement, even though the animal did not know the type of movement to be per-

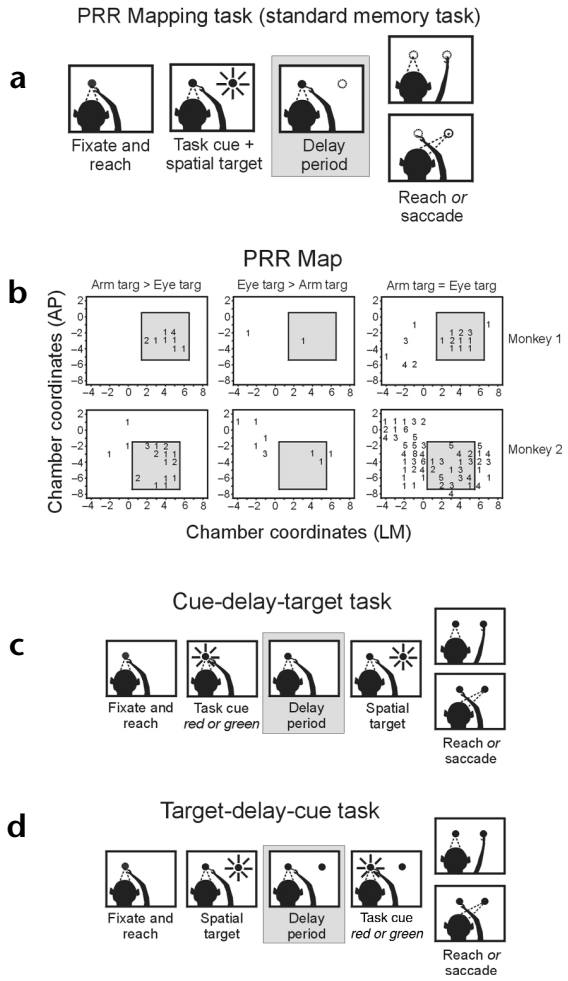

Fig. 1. Localization of the parietal reach region. (**a**) The standard memory reach and saccade tasks used to map out PRR[12]. Task type (reach or saccade) was indicated by the color of the target. Cells showing significantly higher activity during the delay period following a reach compared to a saccade task cue were identified. (**b**) PRR (shaded rectangle) was mapped as the region showing a high proportion of such cells. For each of two animals (top and bottom rows), left and center maps show the number of cells at each location (relative to –5 AP, 12 L) with greater delay activity on reach and saccade trials, respectively. Right, cells with similar responses on reach and saccade trials (*t*-test on activity 150–650 ms after target offset; $P < 0.05$). Coordinates in mm. (**c**) Cue–delay–target task. A cue instructs the type of movement (eye or arm), and after a variable delay (gray), a target both instructs a spatial location and triggers the movement. During the delay, the effector to be used is known, but the spatial goal for the movement is unknown. (**d**) Target–delay–cue task. Similar to (**c**), except the target and cue appear in reverse order. During the delay, the spatial target is known, but the effector is unknown.

formed. We found that many PRR cells were activated by the instruction to make a reach without the spatial goal, and that many cells were activated by the spatial instruction without the effector cue. Many cells were responsive to both types of instructions, but some cells carried only effector information, whereas others carried only spatial information.

Results

Delineation of PRR

PRR was defined functionally as the region of PPC containing visually responsive neurons, many of which show sustained firing during a standard memory reach task (**Fig. 1a**)[12,15]. These functional criteria were applied independent of the properties under study. Based on these independent functional criteria, we identified a cube of cortex, of similar anterior–posterior (AP) and medial–lateral (ML) dimensions, in each of two animals (**Fig. 1b**). Changes of 1–2 mm in these borders have minimal effect on our results and do not change our overall conclusions. We subsequently obtained high-resolution images of cortex in monkey 2 (M2) using magnetic resonance imaging and used those images to localize cells of interest (**Fig. 3**).

Effector specificity

We tested whether neurons in PRR were activated by the instruction to prepare a reach, without spatial information regarding the target of that reach. In cue–delay–target trials (**Fig. 1c**), a central color cue instructs the type of movement to be performed (reach or saccade) and then, after a variable delay, a peripheral target appears, which instructs the goal of the movement. The animal's task was to acquire the target using the instructed movement. The animal could move only after the movement was fully specified, that is, after both the cue and target had appeared. Typically, targets were either inside the receptive field of the recorded cell, or at a location on the opposite side of the fixation point. We compared delay period responses on reach and saccade trials.

This cue–delay–target task (**Fig. 1c**) differs in two important ways from the standard memory task that was used previously to establish effector specificity in LIP and PRR[12,15], and which was used in the present study to delineate PRR (**Fig. 1a** and **b**). First, the cue–delay–target task contains an interval in which the upcoming movement type (reach or saccade) is known, but the spatial goal of that movement is not known. This interval allows us to measure the influence of effector information on neuronal activity without spatial information. This contrasts with conditions in the standard memory task, in which the type of movement and the goal of the movement are instructed together at the same time.

A second difference is that in the cue–delay–target task, movements occur 'on-demand' with the appearance of the peripheral target. This contrasts sharply with the standard memory task, in which the movement is fully specified but must be withheld until a 'go' cue is provided. Outside the laboratory, animals often do not wait for a cue, but instead move as soon as a target appears. The cue–delay–target task, unlike a standard memory task, approximates this type of behavior inside the laboratory.

In a single PRR cell aligned on the presentation of the movement-type cue (**Fig. 2a**, left), a cue instructing a reach resulted in an increase in firing rate from baseline (10.5 ± 0.97 spikes/s, mean ± s.e.m.) to a delay period rate of 27.0 ± 1.2 spikes/s (two-tailed *t*-test, $P < 0.05$), whereas a cue instructing a saccade had a significant inhibitory effect (11.3 ± 1.1 to 8.2 ± 1.0 spikes/s; $P < 0.05$). The difference in delay period activity on reach and saccade trials was large and significant (27.0 versus 8.2 spikes/s; $P < 0.001$), despite no spatial information regarding target location, and despite no more than 1 degree of movement of the arm or eyes (see below). In previous experiments in parietal, premotor and motor cortex, activity related to an upcoming motor response has been called motor preparation, motor set or motor intention[27]. It is important to recognize, however, that in contrast to these earlier studies, we are describing a signal that occurs completely without spatial information.

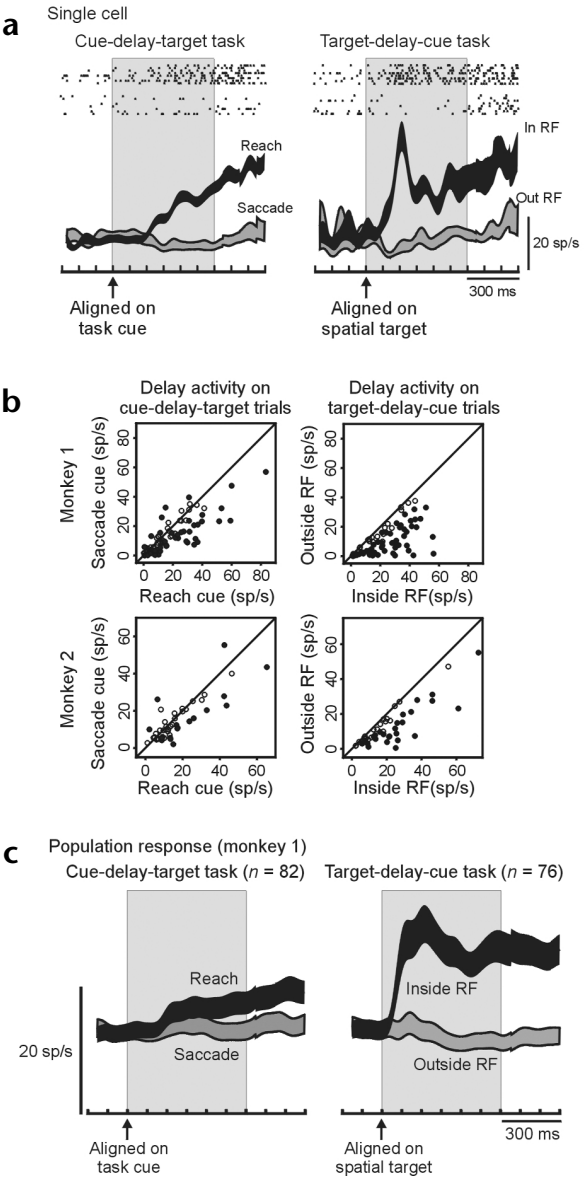

Fig. 2. Neuronal activity in PRR can be evoked by the intention to make an arm movement even without a spatial target, or by spatial information without effector information. (**a**) A single PRR cell increased its firing when instructed to prepare a reach (left, dark trace), but not when instructed to prepare a saccade (light trace). Note the absence of a spatial goal for the movement (cue–delay–target task). The same cell showed clear spatial tuning (right) on interleaved trials when the movement type had not yet been instructed (target–delay–cue task). Rasters plot the temporal sequence of individual action potentials occurring in each of the first eight trials. Traces and rasters are aligned on the presentation of the cue (left) or target (right). The subsequent delay period was 600, 900 or 1200 ms long. Shading indicates the first 600 ms. To avoid contamination by movement responses, data from trials with 600 ms delay periods are truncated at 650 ms, causing a slight discontinuity in the traces. An additional 300 ms of data are included from trials in which the delay was 900 or 1200 ms. Traces shown for cue–delay–target trials (left) were calculated using 64 reach and 64 saccade trials (8 trials × 8 directions for each cue). Traces shown for target–delay–cue trials (right) were calculated using 16 'in RF' (receptive field) and 16 'out RF' trials (8 trials × 2 effector cues for each direction). In this and subsequent figures, the origin of the *y*-axis corresponds to zero spikes/s. (**b**) Activity in most PRR neurons, measured in the final 300 ms of the delay period, was greater on reach than saccade trials (left; cue–delay–target task). Most cells fall to the right of the diagonal line. Cells with significant effects are plotted as filled symbols. For comparison, a similar scatter plot is shown for target–delay–cue responses (right; target–delay–cue task). (**c**) Across the population, PRR cells increased their firing when instructed to prepare a reach (left, dark trace) but not when instructed to prepare a saccade (light trace) without spatial information. For comparison, responses to spatial information without effector information are shown on the right. Trace thickness in (**a**) and (**c**) represents mean ± s.e.m.

Spatial responses

To determine the response to spatial information without effector-specific intention, a second trial type was randomly interleaved with the cue–delay–target trials. On target–delay–cue trials, the peripheral target appeared first and then, after a delay period, the movement cue was presented (**Fig. 1d**). The same cell that showed non-spatial, effector-specific responses in the cue–delay–target task also showed spatial responses in the target–delay–cue task (**Fig. 2a**, right). The cell responded to the peripheral stimulus when it fell inside the receptive field with a transient increase followed by a sustained elevation in firing (17.5 ± 3.0 spikes/s over baseline; $P < 0.05$). The cell was suppressed (albeit not significantly) when the stimulus fell outside the receptive field (3.7 ± 2.7 spikes/s below baseline, $P > 0.05$). These responses occurred even though the movement type—saccade or reach—had not yet been specified. Thus, this cell shows clear spatial tuning despite uncertainty regarding how that spatial information will be used.

Population responses

Like the example cell above, many cells in PRR were modulated by effector-specific intention despite the absence of spatial information (**Fig. 2b**, left), and many cells were spatially tuned despite the absence of effector information (**Fig. 2b**, right). In this graph, cells without effector-specific or spatial modulation would fall on the diagonal lines. Instead, most cells are to the right of this line, indicating a preference for reach cues over saccade cues (left) and the presence of spatial tuning (right).

As a measure of overall tendency and time course, responses were averaged across every cell recorded in PRR that had a task-related response of any sort and at any time (Methods; **Fig. 2c**, data from M1). Activity from 82 cells on cue–delay–target trials increased after an instruction to prepare a reach but not after an instruction to prepare a saccade (left, 3.6 ± 0.9 spikes/s increase and 0.9 ± 0.6 spikes/s decrease, respectively). On trials in which the delay period lasted longer than 600 ms, the differential effect was even larger. In the last 300 ms of the delay interval, firing had increased on average by 5.6 ± 1.0 spikes/s when a reach was instructed, but only by 0.1 ± 0.6 spikes/s when a saccade was instructed. Data from a second animal (M2) showed a similar pattern: an increase of 4.8 ± 1.1 spikes/s after a reach instruction, but only 2.1 ± 1.0 spikes/s after a saccade instruction (49 cells). These differences were significant in both animals ($P < 0.05$).

There was also robust spatial tuning on target–delay–cue trials (**Fig. 2c**, right). Targets inside the receptive field evoked 14.2 ± 1.7 spikes/s and 8.9 ± 1.2 spikes/s more activity in each of the two animals, respectively, than targets outside the receptive field. Comparing the results of cue–delay–target and target–delay–cue trials shows that over the last 300 ms of the delay period, non-spatial, effector-specific information was 39% (M1, 5.5 versus 14.2 spikes/s) and 30% (M2, 2.7 versus 8.9 spikes/s) as effective in evoking a neural response as spatial information without an effector instruction.

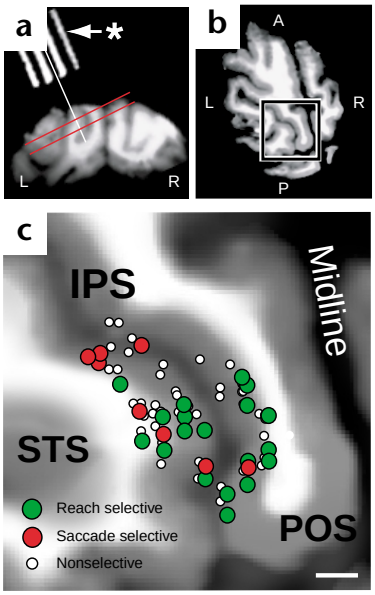

Fig. 3. Intention cells in the IPS. (**a**) A roughly coronal MRI section showing a schematic electrode (thin white line) passing through the center of the recording chamber (*) and into the IPS (animal M2). The section is aligned with the path of the electrode. (**b**) A roughly horizontal MRI section, perpendicular to the path of the electrode and 8.0 mm below the cortical surface (lower red line from **a**). A, anterior; P, posterior; L, left; R, right. (**c**) Expanded view of the square in (**b**), showing the recording sites of IPS cells with intention-related activity. Cells with reach-related intention activity (green circles) were found on both sides of the proximal portion of the IPS. Only cells between 3.5 and 8.0 mm in depth are shown (red lines in **a**). Within this range, electrodes traveled roughly perpendicular to the sulcus. Sites along the same track are jittered by up to 0.25 mm for clarity. IPS, intraparietal sulcus; POS, parieto-occipital sulcus; STS, superior temporal sulcus. Scale bar, 2 mm.

On a cell-by-cell basis, 37% of cells within PRR were significantly more active when the movement-type cue instructed a reach rather than a saccade (**Table 1**; data from two animals; Student's *t*-test, $P < 0.05$). Only 8% of cells showed the reverse preference. Thus, cells favoring the reach cue were significantly more common than cells favoring the saccade cue ($P < 0.001$, chi-square test). Very few cells showed the reverse pattern. Many cells were not activated in either condition, and some cells were activated equally in the two tasks. This contrasts with PPC cells that we recorded outside of PRR. Fewer of these cells were significantly modulated by the movement-type cue, and of those that were, cells preferring arm and eye movements were present in equal numbers (15% versus 14%, respectively; $P > 0.05$ chi-square test).

Localization

Using anatomical MR imaging, we reconstructed the locations of cells recorded in M2 relative to the intraparietal sulcus (IPS; **Fig. 3**). Effector-specific cells preferring arm movements were present in large numbers on both banks of the proximal one-third of the IPS. Of 33 reach cells in M2, 13 lay on the medial bank of the IPS and 16 lay on the lateral bank. (Two more lay on the mesial wall, and two were indeterminate.) For comparison, saccade cells were much more common on the lateral bank and generally lay in the middle third of the sulcus. Of 20 saccade intention cells in M2, 11 lay on the lateral bank of the IPS and only 2 on the medial bank. (Four were indeterminate, 2 lay on the mesial wall, and 1 lay in the STS.) This distribution of reach- and saccade-preferring cells is similar to that obtained using the standard memory task that was used to functionally map PRR, both in the same animals (**Fig. 1b**) as well as in a previous study in different animals (**Fig. 3** of ref. 12).

Alternative explanations: spatial prediction

We propose that effector-specific intention activity occurs even without explicit spatial information. This is a surprising finding, given the prevailing, well-supported notion that the dorsal pathway is dedicated to the analysis of spatial information[16,17]. It is therefore important to consider whether intention activity may reflect implicit rather than explicit spatial information. Might an expectation or prediction of target location substitute for explicit spatial information in our cue–delay–target task?

Cellular activity in LIP[6,10], superior colliculus[28–30] and the frontal eye fields (FEF)[31] can reflect the probability of a movement into the receptive field of the recorded cell. In the superior colliculus, the evoked activity is proportional to the probability that the target will appear in the receptive field[28–30], as if on any given trial a prior probability or likelihood ratio is associated with each of a number of potential spatial locations[32]. Perhaps effector-specific activity in cue–delay–target trials is a related phenomenon. In this case, the activity we observed would still be effector specific, but rather than occurring independently of spatial information, it would be related to a prediction of target location.

As a test of the spatial probability hypothesis, we examined whether delay period activity depends on the probability of the target appearing in the receptive field. Such a relationship is central to the spatial prediction hypothesis. Although most cells were tested in blocks of trials where the target might appear at one of only two possible locations, other cells were tested in blocks where the target could appear at any one of eight positions, and hence the probability of the target falling inside the receptive field was less for this second group of cells. The spatial anticipation hypothesis predicts that the difference in delay activity for arm com-

Table 1. Summary of neurons.

	Inside PRR			Outside PRR		
	M1	M2	Total	M1	M2	Total
Cue–delay–target task						
Cells tested	82	49	131	95	142	237
Arm > eye	36 (44%)	13 (27%)	49 (37%)	15 (16%)	20 (14%)	35 (15%)
Eye > arm	8 (10)	3 (6)	11 (8)	16 (17)	17 (12)	33 (14)
Eye = arm > baseline	18 (22)	12 (24)	30 (23)	29 (31)	37 (26)	66 (28)
Eye = arm = baseline	20 (24)	21 (43)	41 (31)	35 (37)	68 (48)	103 (43)
Target–delay–cue task						
Cells tested	76	48	124	79	142	221
Spatial tuned cells	55 (72%)	25 (52%)	80 (65%)	44 (56%)	72 (51%)	116 (52%)

Data are shown for two animals (M1 and M2). Of the 368 cells tested in the cue–delay–target, all but 25 were also tested in the target–delay–cue task. All but 2 cells in the target–delay–cue task were tested in the cue–delay–target task.

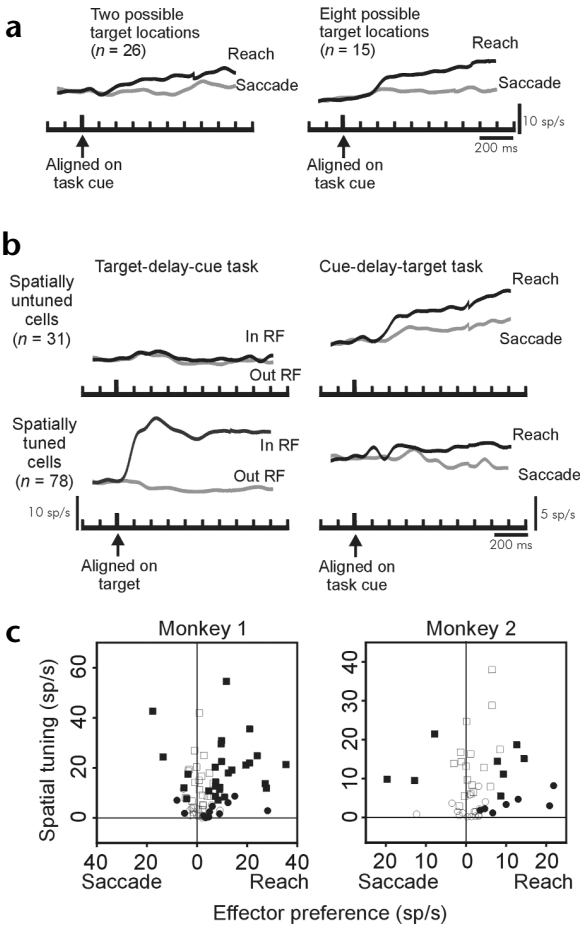

Fig. 4. Effector-specific activity in PRR is not driven by a prediction of upcoming target location. (**a**) Delay period activity showed a similar preference for reach compared to saccade trials, regardless of whether the cell was tested under conditions in which the target had a high probability (left) or low probability (right) of appearing in the receptive field, in contrast to spatial predictive signals in the superior colliculus[28–30]. Only spatially tuned cells are included. Traces show mean activity as a function of time. (**b**) Delay period activity in cells without spatial tuning (top row) was similar to delay activity in cells with strong spatial tuning (bottom row). Left column (target–delay–cue task) shows extent of spatial tuning; right column (cue–delay–target task) demonstrates intention activity, which, if anything, is more robust among untuned cells. Untuned cells include all those that failed a test of statistical significance for spatial tuning (44 cells), excluding those cells in which nonsignificant spatial tuning exceeded 3 spikes/s (13 cells). Tuned cells are all those with statistically significant spatial tuning. (**c**) A wide range of spatial tuning and effector-specific intention activity within individual cells. The ordinates show spatial tuning, quantified as delay activity on target–delay–cue trials: inside minus outside receptive field conditions. The abscissas show intention activity, quantified as delay activity on cue–delay–target trials: reach minus saccade conditions. Significant spatial tuning is indicated by square versus round; significant effector-specific intention activity is indicated by solid versus hollow. Many cells show just one effect, and there is no consistent relationship between intention activity and spatial tuning.

pared to eye trials in spatially tuned cells will be greater in the two- compared to the eight-target condition. Instead, the difference between arm and eye trials was 5.7 ± 2.0 spikes/s with two possible target locations, and 7.9 ± 3.3 spikes/s with eight possible target locations (**Fig. 4a**; 26 and 15 cells tested, respectively). These two values are not significantly different from one another ($P > 0.05$, one-tailed t-test). In contrast, the latency of eye movements in the two data sets showed a highly significant difference (169.8 ± 1.2 ms, $n = 486$ trials, with two possible target locations, and 178.6 ± 1.0 ms, $n = 806$ trials, with eight possible target locations; $P < 0.001$, Student's t-test). This indicates that the animals were indeed using the information contained in the probability structure to optimize their behavior, and yet the effect of this optimization was not apparent in the intention activity. This is direct evidence that intention activity does not reflect the strength of the prediction that a target will land in the receptive field of the cell.

As a second test of the spatial prediction hypothesis, we asked whether cells without spatial tuning showed effector-specific delay activity. If effector-specific activity reflects predicted spatial information, then it should not be present in cells that are not spatially tuned. Of 44 cells without spatial tuning in the target–delay–cue task, 41% showed effector-specific activity in cue–delay–target trials. This percentage was not significantly different from the 46% of the 78 cells with spatial tuning that were effector-specific ($P > 0.05$, chi-square test). In addition, the magnitude of effector-specific activity was similar for spatially tuned and untuned cells (**Fig. 4b**).

Interaction between effector and spatial signals

To understand what computations PRR performs, it is necessary to know the relationship between spatial and effector-specific signals. We showed that the presence or absence of spatial activity did not affect average effector-specific intention within PRR (**Fig. 4b**). Further, when the amplitude of spatial activity (target–delay–cue task) is plotted against the amplitude of effector-specific intention activity (cue–delay–target task), there are cells with only spatial activity, cells with only effector-specific intention activity, and cells with both types of responses (**Fig. 4c**). A statistical analysis reveals that the two properties occur

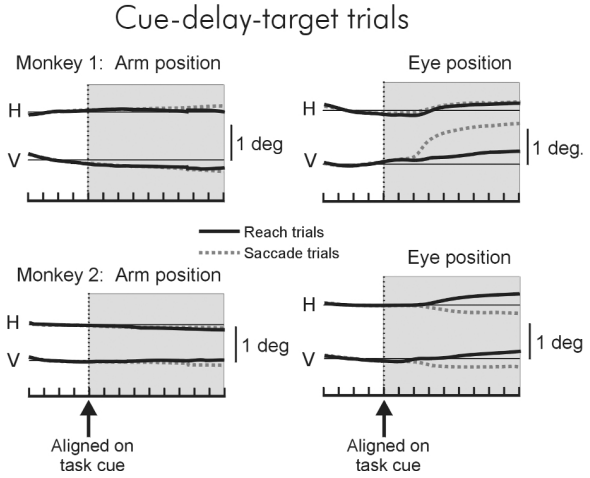

Fig. 5. Effector-specific activity in PRR is not driven by proprioceptive feedback from the arm or eyes. Left column, average horizontal and vertical arm position during the delay periods of cue–delay–target trials. For both animals, arm position was influenced little by the effector type that was cued: reach (dark traces) or saccade (light traces). Right column, similar plots for eye position. The cued effector had a small influence on eye position, although the difference between saccade and reach trials never exceeded 1 degree and is therefore unlikely to account for our results. Trace thickness represents mean ± s.e.m.

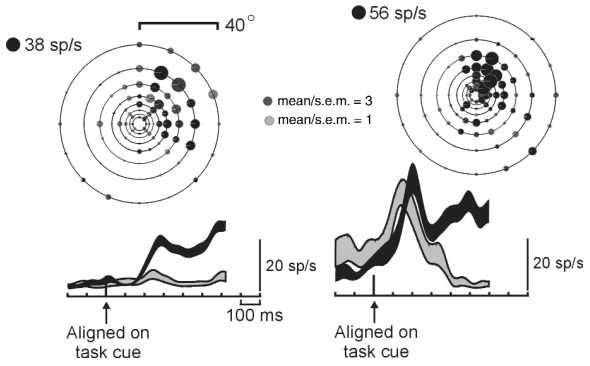

Fig. 6. Effector-specific activity in PRR is not driven by color-selective sensory responses. Receptive-field plots for two typical PRR cells (top). Filled circles represent the response to a flashed stimulus at each of 72 locations from 3.5 to 40 degrees from the fovea. Filled circle diameter indicates mean firing rate, and shading (grey to black) indicates the reliability of each mean. Traces (below) show mean ± s.e.m. of the responses on cue–delay–target trials. When the receptive field excludes the fovea (left), effector-specific activity begins ~200 ms after cue onset. This activity is different for saccade and reach instructions. When the receptive field includes the fovea (right), there is an increase in activity beginning ~100 ms after cue onset. In this case, the initial response is similar for saccade and reach instructions and probably reflects a simple sensory response to the foveal cue. After 200–300 ms, however, effector-specific activity emerges. The similarity of effector-specific responses in cells with and without foveal receptive fields suggests that effector specificity does not result from a learned color preference.

independently of one another. In particular, the probability of a cell having significant spatial tuning was independent of the presence or absence of effector-specific activity, and, as noted in the previous section, the probability of having significant effector-specific activity was independent of the presence or absence of spatial tuning ($P > 0.05$; chi-square tests). Thus, spatial information and effector information are coded by two partially overlapping sets of cells in PRR.

Alternative explanations: delay period movement

In addition to implicit spatial information, we considered other alternative explanations for effector-specific intention activity. Animals might make subtle movements during the delay period of cue–delay–target trials. These differences might differ systematically on reach compared to saccade trials. We therefore investigated whether a proprioceptive response to differential movement might explain effector-specific delay-period activity.

Arm movements greater than 3 degrees, even if made slowly, would be detected on-line automatically and result in the trial being aborted. Smaller movements, however, could be accepted. To test for slight but systematic differences in movement, we pooled behavioral data (absolute change in arm and eye position) across all trials (**Fig. 5**). This analysis provided a very large amount of statistical power, as thousands of trials were averaged together in each case.

Average arm position changed little over the course of the delay period, with the difference in arm position between reach and saccade trials always less than 0.2 degrees in each animal. For this change in position to account for the observed changes in firing (5.5 and 2.7 spikes/s in M1 and M2) would require sensitivities of 27 and 13 spikes/s per degrees of arm movement, respectively. Such a high sensitivity is extremely unlikely.

In contrast to the nearly identical arm movements, there was a small amount of differential eye movement (0.8 and 0.5 degrees in M1 and M2, respectively). In M1, the eyes were displaced up and to the right 200 ms after cue onset, with more upward movement on saccade trials. In M2, the eyes drifted down and to the left on saccade trials, and up and to the right on reach trials. In both animals, the drift was gradual.

Might the difference in PRR delay activity reflect this difference in eye movement, either as a result of proprioceptive feedback or efference copy? We believe this is unlikely for three reasons. First, the required eye movement sensitivity (5 spikes/s per degrees of eye movement) is large for parietal cortex[33,34]. Second, it is unlikely that there was any systematic relationship between preferred gain-field directions and differences in eye position; for example, when the eye position traces were realigned according to the preferred direction of each cell, all differences in eye position between reach and saccade trials disappeared (data not shown).

A third reason for rejecting a causal relationship between differential eye movement and differential firing is that the qualitative effects are dissimilar. In both animals, firing rate increased sharply on arm trials, but only slightly on eye trials (**Fig. 2c**). In M1, eye position shows the reverse pattern: a sudden sharp change on eye trials, but only a slight change on arm trials. Eye position in M2 shows yet another pattern: equal and opposite changes for eye and arm trials. In neither animal does the pattern of differential eye movement match the pattern of differential activity.

Alternative explanations: learned color preferences

It is possible that what we have identified as effector-specific intention instead reflects a learned receptive field preference for different colored stimuli[35]. To rule out this possibility, we carefully mapped sensory receptive field borders in 10 PRR cells showing effector-specific intention. Six cells had receptive fields that clearly excluded the fovea and parafovea, yet nonetheless showed clear effector-specific intention (**Fig. 6**, left). Effector-specific (and instruction-specific) effects typically emerged 200–300 ms after the appearance of the cue. The remaining four cells had receptive fields that included the fovea or parafovea (**Fig. 6**, right). We would expect that learned color preferences would manifest sooner and more strongly when the stimulus appears within the receptive field, compared to outside the receptive field. Yet this was not the case. Cells with central receptive fields, unlike those with peripheral fields, typically showed an initial non-specific increase in activity starting ~100 ms after cue onset, followed by an effector-specific divergence after 200–300 ms (**Fig. 6**, right). Thus effector-specific divergences occurred with similar frequency and at similar times in cells with and without parafoveal responses. This strongly suggests that the divergence does not reflect a simple learned color preference.

Across the population of cells, divergences in firing on cue–delay–target saccade and cue–delay–target reach trials almost never occurred within 100 ms of cue onset, and had a median latency of 233 ms. (For comparison, the visual response latencies in PRR, to targets presented in the receptive field, had a median latency of 104 ms). The long latencies for the divergence of activity on reach and saccade trials are consistent with the time required for processing symbolic cues, not color-selective sensory responses[36].

Discussion

Our results show that PPC neurons can be activated not only by spatial information without a motor plan, but also by the plan to use a specific effector without spatial information (**Fig. 2**). Some

cells carry both spatial information and effector-specific intention signals, but many cells carry just one signal or the other (**Fig. 4c**). The coding of spatial information without a motor plan is hardly surprising, given previous ideas of PPC as the region in which spatial information is processed[15]. In addition, PPC is organized into regions with privileged connections to particular effectors[37] such as eye movements[12], arm movements[12,20–26,38,39] and grasping movements of the hand[40,41]. Even so, the current finding that effector-specific intentions are encoded without spatial information is unexpected. On average, effector-specific intention signals drove cells about one-third as much as spatial signals. These findings strongly suggest that PPC is involved in specifying how an organism will respond to a particular target (motor intention).

Because we generally did not map receptive fields with high spatial resolution, we are likely to have underestimated the effects of spatial information. However, the few cells that we did map in high resolution showed broad tuning (**Fig. 6**), and therefore it is unlikely that finer-resolution mapping would greatly increase our estimate of population-averaged spatial modulation; hence we expect that it would not greatly change our estimates of the relative magnitudes of intention and spatial signals.

Conceivably, the effector-specific intention signals may be modulating implicit rather than explicit spatial information, in the form of an expectation or prediction about where a target will appear. Predictive spatial activity has been described in LIP[6,38,39]. However, this activity appeared when there was near certainty regarding where the target would appear, whereas we observed activity in the presence of as many as eight possible target locations (**Fig. 4a**). Predictive activity in the presence of eight possible target locations is observed in the superior colliculus[28–30]. However, the magnitude of predictive activity in the colliculus depends on the probability that a target will appear in the receptive field of the cell being recorded. This was not the case in PRR (**Fig. 4a**). Indeed, even cells without spatial tuning showed effector-specific activity, inconsistent with the idea that this activity is associated with any sort of implicit spatial information, including a spatially tuned predictive signal (**Fig. 4b**).

Finally, the timing of the effector-specific response in PRR is inappropriate for predictive spatial activity. In LIP, FEF and the superior colliculus, activity predictive of a target appearance arises shortly before the target is expected to appear[6,28–31]. In our experiments, cue–delay–target trials were interleaved with target–delay–cue trials. As a result, on half of all trials, the peripheral target was the first instructional stimulus to appear. Therefore one would expect a predictive signal to arise shortly before the first instructional stimulus on every trial. This expectation was not fulfilled. There was no increase in activity shortly before the first stimulus in cue–delay–target and target–delay–cue trials (**Fig. 2c**). Thus the delay period activity we observed is quite different from spatial predictive activity, and is unlikely to reflect a modulation of implicit spatial information by effector-specific intentions. Instead, PRR neurons encode effector-specific intentions even without spatial information.

Cells that can encode effector-specific information and target information in isolation or in combination have been reported elsewhere in the brain. In particular, activity in dorsal premotor cortex reflects both the position of a reach target (spatial information) and the arm to be used for the reach (effector information)[18]. As in PRR, either spatial information or effector-specific information can evoke activity without the other. Dorsal premotor cortex has extensive and reciprocal connections with many parts of PPC involved in reaching[19–21]. Perhaps this reciprocal connectivity explains in part why action selection occurs in both dorsal premotor and posterior parietal cortices. The finding that some PRR cells code effector-specific instructions, others code spatial instructions, and still others code both instructions, suggests that PRR, like dorsal premotor cortex, is involved in specifying how the animal will respond to a particular target.

Although PPC seems to be well organized with respect to effector system[12,13], there is only a coarse organization of spatial signals[42]. Thus, whereas effector-specific signals seem to be well localized within particular cortical regions such as PRR (**Fig. 1b**; **Table 1**), similar responses to visual targets can be found throughout much of the intraparietal sulcus (**Table 1**). This suggests that, without an effector-specific plan to respond to a target, the appearance of a target will result in diffuse, widespread activation of PPC. Such activation in multiple regions could be viewed as contingency plans for multiple potential movements[43].

Localization

Neurons with arm-specific intention activity lie on both banks of the proximal portion of the IPS. The proximal one-third of the medial bank is area MIP (medial intraparietal)[44,45], although this name is often misapplied to refer to the entire medial bank[46]. The proximal one-third of the lateral bank has been named area cIPS (caudal intraparietal sulcus)[47] or, more recently, zone LOP (lateral occipitoparietal)[48]. However, the boundaries of both areas are vague[48]. It is not known, for example, whether LIP and cIPS/LOP tile the entire proximal IPS or whether other areas are also present. Therefore, for the present we prefer the term PRR.

Our use of the term 'region' rather than 'area' is intended to highlight that these neurons have been grouped by function, and not by connectivity or histology. Their exact relationship to particular cortical areas remains uncertain, although they clearly overlap at least parts of both MIP and cIPS/LOP. Based on the clear and consistent differences in connectivity between the two banks of the proximal IPS[20], we do not expect PRR neurons to form a single homogenous functional population. For example, it would be worthwhile to investigate tuning for visual disparity, which has been demonstrated on the lateral bank[47] but has not been tested on the medial bank. In addition, even properties held in common by neurons on both banks may prove to have different functional consequences for the organism. However, neurons on both banks share many properties in common. Both MIP and cIPS neurons are visually responsive, and both are hypothesized to be involved in reaching or grasping[44,47]. Reach-selective memory activity for spatial locations has been demonstrated[12], and we now show arm-specificity without spatial information in both areas.

In conclusion, we have recorded from a localized region of PPC that codes not only spatial information about the goal of an upcoming movement, but also non-spatial information about the effector to be used to achieve that goal. These cells lie on both banks of the proximal portion of the IPS. The presence of non-spatial, task-specific information in PPC is at odds with its classical role as a sensory association cortex, and instead supports the notion that this region is active in sensorimotor transformations.

Methods

Recording procedures. Animals sat in a custom-designed monkey chair (Crist Instrument, Hagerstown, Maryland) with an open front that allowed reaches toward a visual stimulus. Stimuli were back-projected by a CRT projector onto a touch panel 25 cm in front of the animal. All experiments complied with the relevant laws and institutional guidelines, and were approved by the Washington University Institutional Animal Care and Use Committee.

Recordings were made from the left hemispheres of two adult rhesus monkeys. Recording chambers were centered at 5 mm posterior and 12 mm lateral (Horsley–Clarke coordinates) and placed flush to the skull. While we searched for cells, animals performed non-delayed, center-out combined eye and arm movements to 20 degrees peripheral targets in each of eight directions. Cells that changed firing rate at any point in the search task (for example, at target appearance or movement onset) were tested further. To map PRR, we tested 220 cells (40 M1, 180 M2) on a standard interleaved memory reach and saccade task[12] (**Fig. 1a**). After 500 ms of central fixation and touch, a red or green peripheral target appeared at one of eight positions, instructing either a reach or saccade to the target. After a delay (800 ms), the central stimulus disappeared, and the animal made a reach without moving its eyes, or a saccade without moving its arms, to the remembered location of the peripheral target. We delineated PRR based on neural activity during the delay period (**Fig. 1b**; M1, dimensions, ML × AP × DV, 5 × 6 × 8 mm; M2, 5 × 6 × 4 mm).

Behavioral tasks. Most cells were tested on cue–delay–target and target–delay–cue tasks (**Fig. 1c** and **d**). Each trial began with the monkey fixating and reaching for a blue central square. On cue–delay–target trials, after a variable delay of 500–800 ms, the color of the fixation point changed to green or red to instruct an arm or eye movement (color assignment was reversed between the two animals) and remained on for the duration of the trial. After a second delay of 600, 900 or 1,200 ms, a blue peripheral target appeared at one of eight equally spaced positions located 20° from the central reach/fixation stimulus. Animals were free to acquire the peripheral target as soon as it appeared, according to the previous movement-type instruction. They received a liquid reward after holding the target for 400 ms, and so were motivated to respond at short latency. Eye-movement latencies for the two monkeys averaged 171 ± 22 ms and 240 ± 74 ms (mean ± s.d.). Reach latencies averaged 292 ± 90 ms and 384 ± 93 ms, respectively.

Target–delay–cue trials were similar to cue–delay–target trials, except that the spatial target was presented first, and the foveal color change occurred second. The variable delay period was similar to that of cue–delay–target trials, and the animal was free to acquire the target once the foveal color change had occurred. Target–delay–cue and cue–delay–target trials were interleaved for 90% of recorded cells.

Arm and eye positions were monitored by a 43.2 cm touch panel (Keytec, Richardson, Texas) and a scleral search coil (CNC Engineering, Seattle, Washington), respectively. Trials were terminated if animals failed to maintain touch and fixation within a square window (side length ± 6° for touch, ± 3° for fixation) or moved their arm or eyes more than 3° in any direction during the delay period. Trials were also terminated and the data not analyzed if the eyes moved before the reward was delivered on a reach trial, or if the arm moved before the reward was delivered on a saccade trial. In practice, animals almost never moved the uninstructed body part after receiving the reward, but instead returned the peripherally displaced limb directly back to the central target to start the next trial. All tasks were performed in the dark. Animals typically performed over 80% of trials successfully.

Each task (cue–delay–target saccade, cue–delay–target reach, target–delay–cue saccade, target–delay–cue reach) was repeated 8–10 times using a peripheral target in one of two, four or eight interleaved directions (292, 9 and 67 cells, respectively). When testing in only two or four directions, we used a pilot run of two target–delay–cue trials in each of eight directions to establish the best direction. Receptive fields were plotted with finer resolution in only ten cells (**Fig. 5**). Unless otherwise mentioned, baseline and delay period firing rates were measured 0–300 ms before and 300–600 ms after cue onset, respectively, and significance was tested with a two-tailed t-test and a criterion value of $P < 0.05$. Traces were smoothed using a 191 point digital low-pass filter with a transition band spanning 20 to 32 Hz.

Anatomical localization. Anatomical MR images of M2 were acquired using a custom-designed surface coil (NOVA Medical, Wakefield, Massachusetts) in a Siemens Allegra MR system (Siemens Medical Systems, Erlangen, Germany) operating at 3 T. The anesthetized animal lay prone with his neck partially extended and supported by foam. With the surface coil positioned around the animal's chamber, we acquired eight three-dimensional datasets (Siemens multiplanar rapid acquisition gradient echo, T*R* = 14.3 ms, TE = 7.0 ms, TI = 15 ms, 0.8 mm isotropic voxels), which were averaged online for signal-to-noise enhancement.

To determine the orientation, depth, and scale of our recording grid relative to the MRI, we placed a custom-designed plastic cylinder (5 cm height, 1.5 cm diameter) containing MR contrast agent (0.015 mM gadoversedamide) into the animal's recording chamber. Bars at known locations within the cylinder (2 mm × 2 mm × 8 mm, 4.5 mm spacing) displaced the contrast agent (**Fig. 3a**), allowing us to reconstruct the position and orientation of the recording chamber and grid (Crist Instruments) through which our electrodes passed. From this, we were able to project our recording sites onto the MR image with an estimated accuracy of 1 mm or better (**Fig. 3c**).

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Competing interests statement

The authors declare that they have no competing financial interests.

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