mediated by the SC, can occur in the virtual absence of activity in TR(S)N cells.

In conclusion, evidence offered in support of the 'moving hill' model is either inconsistent with this model or subject to plausible alternative explanations.

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Reply

We recorded from antidromically identified tecto-reticular and tecto-reticulospinal neurons that together are called TR(S)Ns] in alert cats trained to perform simple visuomotor tasks[1–4]. Cells were recorded in animals whose heads were unrestrained. We found that TR(S)Ns could be divided into two classes: 'orientation' TR(S)Ns [oTR(S)Ns] and 'fixation' TR(S)Ns [fTR(S)Ns]. Orientation TR(S)Ns discharged a burst of action potentials before the onset of gaze shifts of a specific amplitude and direction that defined each cell's 'preferred' movement vector. Each oTR(S)N also discharged during all gaze shifts larger than this optimal vector; however, the peak in each cell's discharge envelope occurred later, after the onset of the gaze shift. The time between the onset of gaze and the peak in neural activity increased with gaze amplitude. Thus, oTR(S)Ns discharged maximally during a gaze shift whenever instantaneous gaze motor error (difference between desired and current positions of gaze) was a fixed value similar to each cell's optimal vector. Each gaze shift terminated as the fTR(S)Ns were reactivated and continued to discharge until the programming of the next gaze shift. On the basis of these observations, we hypothesized that during a gaze shift there appeared to be a continuous sequence of activation – beginning with the oTR(S)N having the appropriate optimal vector and ending with fTR(S)Ns – of cells in the TR(S)N layer as gaze was driven toward the target. This continuous activation can be thought of as a 'moving hill' of activity on the spatial motor map of the superior colliculus (SC).

We argued that the continuously moving activity of oTR(S)Ns was correlated to gaze motion (eye + head) and not to movement of just the eye alone nor of the head alone. The reasons are as follows. We analysed only single-step gaze shifts. Gaze shifts containing small corrective saccades or made up of a series of coalesced saccades were excluded from the analysis. A cell whose optimal amplitude was 15° discharged at the onset of gaze shifts of that amplitude, but discharged after gaze shifts that were larger. While it is true that all gaze shifts larger than about 20° were accompanied by a 12–15° eye movement, the discharge of oTR(S)Ns was not related to the eye component. For 15° gaze shifts, the activity of a cell with a 15° 'preferred' movement vector occurred around the onset of the gaze shift (i.e. at the start of the eye movement; see Figs 7A,B of Ref. 4). However, during large single-step gaze shifts (e.g. >60°), the discharge of the same cell occurred later in the movement, after the eye saccade had reached its maximum deviation (see Fig. 7D of Ref. 4). Therefore, the cell was clearly not modulating the ocular saccade alone. Likewise, oTR(S)N activity was not related exclusively to head motion since we could record similar activity from oTR(S)Ns regardless of whether

the head was fixed or free (see Figs 1, 2, 12 of Ref. 4). The peak discharge of the cell in our example (see Figs 7, 9 of Ref. 4) occurred when the gaze motor error reached about 15°, irrespective of the eye position in the orbit. Therefore, we concluded that TR(S)N activity was not correlated to eye motion but was best correlated to gaze motion.

That the peak of spike activity is delayed with respect to movement onset is a fact emphasized both above and in our Fig. 9D (Ref. 4), which shows for cell Q24 a linear regression line relationship linking the time difference between gaze onset and peak of spike activity with the amplitude of the gaze shift. Dr Sparks considers cell Q24 for which we state that the optimal gaze motor error is 15°. Since our Fig. 7 shows that the peak of cell Q24's discharge does not precede the 15° gaze shift, but rather occurs shortly after its onset, he argues that peak discharge cannot be driving dynamic motor error and it is not legitimate to use the timing of this peak discharge to argue for 'moving hills'. This criticism is founded on the absolute precision of 15° as this cell's 'preferred' vector. The value of 15° arose from measurement of cell Q24's preparatory tonic discharge that preceded gaze shifts[3]. Our Fig. 7 (Ref. 4), on the other hand, suggests that the optimal vector is somewhere between 10° and 15°. The point here is that the exact value of the optimal gaze amplitude and timing of the peak discharge are subject, naturally, to errors in estimation as shown by the scatter about the regression line of Fig. 9D. Although each point is subject to error, the regression coefficient is 0.88, showing unequivocally that the peak in neural discharge is delayed as amplitude increases. It is inappropriate to criticize, as Sparks does, the data on the basis of one datum point (for a 15° gaze amplitude) contributing to that regression line. Sparks also neglects other compelling evidence supporting the 'moving hill' hypothesis, namely the plots of instantaneous firing frequency versus instantaneous gaze motor error (Fig. 9J of Ref. 4). Lines for different amplitude gaze shifts superimpose on each other suggesting that a 'hill' of constant height is moving along the oTR(S)N layer within the cat SC.

Furthermore, the peak discharge of a single oTR(S)N cannot be thought of as acting alone in determining gaze motor error. A gaze shift is driven as a response to a population discharge. For a given cell, part of the activity preceding the peak probably coincides with the onset of the motor drive, but it is difficult to assess when in the burst the motor drive really begins. This can be shown by electrical stimulation with a train of pulses where the latency to movement onset is 20 ms (Fig. 5C of Ref. 4), but applying two pulses of stimulation during a gaze shift affects eye saccade trajectory after a latency of 10 ms (see Fig. 6 and Table I of Ref. 4). Instead of using the peak in activity as the reference point in the discharge, we could have chosen activity beginning an arbitrary time before the peak. This would have resulted in a shift to the left of the regression line in Fig. 9D (Ref. 4), but would not have changed the conclusions about the moving hill.

Sparks suggests that the moving neural activity we presume to represent dynamic motor error could in fact reflect sensory responses of the neurons: i.e. the sequential activation of cells by a visual stimulus, such as a part of the laboratory surrounds, entering the receptive field of each TR(S)N. An obvious control experiment is to have the animal generate the same amplitude of movement in the absence of all sensory cues. In some of our experiments, ambient illumination was provided by a stroboscope flashing at 100 Hz (see Ref. 3). During some trials the stroboscope was unexpectedly turned off for 1–5 s, usually immediately before the cat initiated a large amplitude gaze shift. In these situations we recorded the same discharge patterns from TR(S)Ns. Because of journal space limitations, we reported results of this control experiment in a footnote (see note #11 of Ref. 1).

We presented other evidence against the sensory response hypothesis. Some neurons whose discharge characteristics during gaze shifts to predictive targets supported the 'moving hill' hypothesis, did not discharge during gaze shifts made spontaneously by the cat in the same visual surrounds[3].

Sparks does not appear to have read our article with care. We failed to find any oTR(S)Ns, even in the rostral SC, that had a distal border to their movement fields. This is stated explicitly in Ref. 4 (p. 1655), with reference to Fig. 12. Furthermore, some saccade-related cells have now been identified in the monkey SC that have a similar characteristic; their movement fields are only bounded at their central edges, the distal border does not exist[5].

Sparks emphasizes our early short communication[6] and downplays our later major report[4]. In the latter we had a much larger database and we modified slightly the conclusions arrived at in the short communication regarding oTR(S)N activity during spontaneous gaze shifts. Spontaneous gaze shifts made by cats are characterized by being very slow, and seldom exceed 30° in amplitude. During these spontaneous movements we finally concluded that only a few oTR(S)Ns discharged. We offer two explanations. First, these movements were controlled by the SC and, because fewer cells were active, the velocity was much lower. In support of this option, deactivation of a small region of the monkey SC has a profound effect on saccade velocity[7]. Second, some spontaneous movements may be generated and controlled by extracollicular circuitry.

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