

Supplementary Fig. S1. Properties of saccade-related P-cells (n=149) in the vermis lobule VIa and VIIa-c. **A**. Distribution of average firing rates for simple spikes (blue) and complex spikes (right). The bar at the top indicates mean and standard deviation. **B**. Waveforms for the simple and complex spikes. The waveforms were normalized by setting the cell's mean voltage to 0 and the maximum negative going simple spike deflection to -1. Error bars are standard deviation. **C**. Within-cell interactions between simple and complex spikes. The blue curve shows the firing rate of simple spikes at time t, given that the cell produced a simple spike at time zero, labeled as SS(t) |SS(0). The red curve shows the firing rate of simple spike rates for each P-cell were normalized with respect to average simple spike firing rate as computed over the entire recording session. Error bars are standard deviation. **D**. Suppression period of simple spikes following production of a complex spike. Suppression period for each P-cell was defined as the duration of time after a complex spike that was required before the simple spike firing rate recovered 63% of its pre-complex spike value. The bar at the top indicates mean and standard deviation.



Supplementary Fig. S2. Spike timing properties of a sample pair of simultaneously recorded P-cells. These are the same cells as in Fig. 1. **A**. Simple and complex spike waveforms. Error bars are standard deviation. **B**. The curve Pr(SS1(t)|SS1(0)) quantifies the probability of a simple spike in P-cell 1 at time t, given that P-cell 1 produced a simple spike at time zero. This quantifies the simple spike refractory period. The curve Pr(SS1(t)|CS1(0)) quantifies the probability of production of a simple spike in P-cell 1 at time t, given that P-cell 1 produced a complex spike at time zero. This indicates the complex spikes induced suppression of simple spikes. **C**. The curve Pr(SS1(t)|SS2(0)) quantifies the probability of production of a simple spike in P-cell 1 at time t, given that a simple spike in P-cell 1 at time t, given that a simple spike in P-cells 2 at time zero. **D**. The curve Pr(SS1(t)|CS2(0)) quantifies the probability of production of a simple spike in P-cell 1 at time t, given that a complex spike was produced by P-cells 2 at time zero. Bin size is 1 ms in the probability plots.



Supplementary Fig. S3. Modulation of simple spikes in individual P-cells during saccades. **A**. Data from a single P-cell, quantifying the change in simple spike rates, aligned to saccade onset. Range was defined as the maximum change in firing rate in the pre- to post-saccade period. The light brown curve shows the average saccade velocity (peak value is 475 deg/s). Error bars are standard deviation computed via bootstrapping. **B**. Strength of saccade-related modulation of each P-cell was defined via a z-score. The z-score computed the mean of the range of the firing rates divided by the standard deviation of the range (7.5±0.3 mean±SEM). Strongly modulated P-cells were those that had a z-score greater than 3, composing 96% (142 out of 149) of the cells in the population. **C**. Change in simple spike rates with respect to baseline for the bursters (red) and pausers (blue). Baseline firing rate is defined as the average firing rate as measured during the entire recording session.



Supplementary Fig. S4. To check whether the increased synchronization index during saccades was an artifact of the change in firing rates of P-cells, we performed a simulation of spiking neurons that burst and paused like the cells in our population. However, the simulated cells had independent probabilities of spiking. **A.** The simulated population consisted of 11 neurons ranging from bursting to pausing in their activity. The plots show the construction of the average firing rates. We started with a unimodal function of various amplitudes, added the response in the middle row to each neuron, producing the data on the right, representing the instantaneous average firing rate $\overline{r_i}(t)$ of each simulated cell. **C**. The population firing rate of the simulation. **C.** Joint probability, and synchrony index of 55 pairs of cells (11 cells taken 2 at a time). Joint probability reflected modulation of firing rates, but because spike timing in each neuron was independent, the synchrony index remained at chance level.



Supplementary Fig. S5. Quantifying the possibility that synchronization among simultaneously recorded P-cells may be corrupted by a common noise source. **A**. Theoretical model. We recorded from neurons S_1 and S_2 , but our recordings $\tilde{S_1}$ and $\tilde{S_2}$ could include spikes generated by a third neuron N. How large is this common noise source in our data, and how does it affect our estimate of synchronization index? **B** and **D**. Because of the common noise, the estimated synchronization index will always be larger than the actual synchronization. In the presence of common noise, an increase in the firing rates of neurons S_1 and S_2 should reduce the estimated synchronization index. The greater the common noise, the larger the effect of change in firing rates on the synchronization index. However, in the actual data, before saccade onset there is a large increase in firing rates, but no changes in synchronization index. This suggests that there is little common noise in the recordings. **C**. Isolation quality of P-cells in our simultaneously recorded data set, as assessed by $\Pr(SS(t)|SS(0))$. The probability of a spike at 1 ms is an estimate of the size of the noise. The distribution of the noise in the 42 pairs is shown in the lower figure. The median common noise is 0.2 Hz.



Supplementary Fig. S6. Parameter values for fitting the vigor equation (Eq. 9) to the velocity and amplitude relationship of saccades in each subject.