Modelling Short-Latency Disparity-Vergence Eye Movements Under Dichoptic Unbalanced Stimulation

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Vergence eye movements align the optical axes of our two eyes onto an object of interest, thus facilitating the binocular summation of the images projected onto the left and the right retinae into a single percept. Both the computational substrate and the functional behaviour of binocular vergence eye movements have been the topic of in depth investigation. Here, we attempt to bring together what is known about computation and function by evaluating a biologically inspired model of horizontal and vertical vergence control [5] and comparing the model's performance to that of human observers.

The Computational Substrate

There is a direct relationship between the sensory and motor aspects of binocular vision since vergence control shares the same computational resources of stereopsis in primary visual area V1 [6, 1]. Simple and complex V1 cells integrate visual information from the two eyes. The response of complex cells can be modelled as the squared sum of a quadrature pair of simple cells with Gabor-like receptive fields [3]. The sensitivity to retinal disparity derives from either a phase or a position difference between the left and right eye receptive fields. By responding to local matches between the left and right eye images, V1 complex cells exhibit a tuning curve which is modulated by the stimulus disparity. V1 complex cells, which are thus characterized by different orientation, frequency and disparity sensitivity, provide a distributed representation of disparity information which can be exploited by higher visual areas to perceive depth and plan eye movements.

Vergence Behaviour

The performance of the primate short-latency vergence control has been extensively tested by dichoptically varying the visual stimuli [1, 7, 8]. Vergence eye movement are evoked if the left and right eye images are correlated but not horizontally aligned. The gain of the vergence control decreases with decreasing interocular correlation and becomes zero if no correlation is present [7]. Moreover, anti-correlated stimuli initiate vergence responses in the opposite direction of disparity [6], coherently with the response of complex cells [1]. Furthermore, since binocular vision requires the vertical alignment of optical axes, the horizontal vergence control system only tolerates a limited amount of vertical disparity within the binocular images. Beyond this limit, the correlation between the two images decreases enough to disrupt the horizontal control [8]. Finally, vergence behaviour to interocular contrast differences has seldom been investigated. Divisive normalization, which accounts for the invariance of the responses of complex cells to the contrast of the input images [2], suggests that vergence gain control should be unaffected by interocular contrast differences.

The Vergence Model

The model relies on a network of V1 simple and complex cells tuned to different disparities along different orientations [5]. Model parameters are: receptive field size $\approx 1.5^\circ$, frequency 3.5 cyc/deg, seven phase shifts $\in (-\pi, \pi)$, eight orientations $\in (0, \pi)$. The horizontal and vertical vergence controls are obtained by directly combining the foveal complex cell responses without the explicit computation of a disparity map. The model includes both monocular and binocular divisive normalization stages, originally introduced in the binocular energy model to explain the invariance to interocular contrast found in the responses of complex cells [2]. The approach has been already proven to be effective in correctly driving vergence movements in natural conditions on a robotic stereo head [4].

Experimental Procedures

The performance of the model was evaluated using naturalistic yet highly controllable "dead-leaves" stimuli with different disparity steps. We tested the model with varying amounts of interocular correlation, interocular contrast, and vertical disparity. To assess the influence of divisive normalization, all testing was repeated as we sequentially removed each normalization stage. We further compared the model to human observers who were asked to perform a simple vergence task on the same dichoptic "dead-leaves" stimuli employed when testing the model.

Results

When testing the model with dichoptic stimuli we found that its vergence gain was modulated by the amount of correlation and reverses for negative correlation (Fig. 1, top). Furthermore, the model's performance was maximum when the contrast in each eye was the same. The model's performance did not vary as a function of interocular contrast if a monocular normalization stage was included. If the monocular normalization stage was not included, performance degraded with increasing contrast differences between the left and the right eye (Fig. 1, middle). Lastly, vertical disparities disrupted horizontal vergence responses to horizontal disparities (Fig. 1, bottom).

The performance of human observers at the vergence task was affected by all experimental manipulations. Small interocular contrast differences did not affect human vergence traces, however large differences strongly degraded performance, contrary to what is predicted by the model implemented with a monocular normalization stage.

Conclusions

We implement a biologically plausible vergence control strategy and test its response to different classes of dichoptic stimuli. The model provides a qualitative explanation of psychophysiological data. The normalization stages solely influenced the model's response to stimuli with different interocular contrast. The normalization circuits are implemented to explain the robustness and effectiveness of the primate neural mechanisms to the unpredictable and changeable lighting conditions of natural environments. Specifically, the monocular normalization mechanism essentially resolves the imbalance between the left and right eye, thus preventing the vergence control to be modulated by interocular contrast. Despite the functional advantage, human vergence differs from this behaviour. Whereas this observation suggests that the proposed disparity-vergence model may be improved to account for human behaviour, it also highlights how dichoptic unbalanced stimulation can be used to investigate the significant but neglected role of sensory processing in motor planning of eye movements in depth.

References

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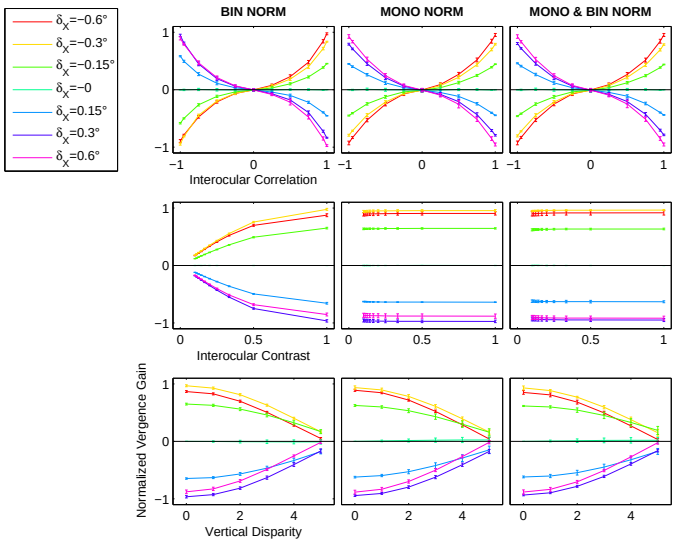

Figure 1: Normalized vergence gain produced by the vergence model as a function of interocular correlation (first row), interocular contrast (second row) and vertical disparity (third row). Different model configurations are presented in the different columns. disparity steps are color-coded as in legend. Data from the model implemented with the binocular, monocular, and or both (right). The experiment was repeated for varying values of (top) interocular correlation (normalized for interocular correlation 1), (middle) interocular contrast (normalized for interocular contrast 1) and (bottom) vertical disparity (normalized for 0 vertical disparity)