Signaling Contours by Retinal Wave Propagation

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Abstract

We describe a neuromorphic retina that signals a luminance edge as a spike. In a fast process, the luminance profile of the receptor layer determines the membrane potential of the ganglion cells and their individual, adjustable spiking thresholds. In a slower process, a wave-propagation process, the charge of ganglion cells with high membrane potential will propagate toward neighboring cells with low membrane potential and low spiking threshold, thus signaling the edge as a spike. Following that, the signaled edge (or contour) actively propagates across the retinal map. The retinal signal can be used for a contour-integration or a contour- propagation approach.

1 Introduction

The retina transforms the luminance profile of our visual environment into a neural code suitable for computation in higher visual areas like the lateral geniculate nucleus (LGN) or the primary visual cortex (V1). Yet, there exists no specific neuronal implementation to date yet that simulates this tranformation for a grey-scale image for example.

We present here a model, that signals the contours in a grey-scale image employing a wave process. The wave process is motivated by the fact, that there exist traveling waves in the retina, e.g.(Jacobs and Werblin, 1998). Figure 1 shows the idea. In a luminance profile, an edge is a large difference between two receptor potentials (figure 1a). The problem to be solved by the retinal circuitry is then the following: how is such a large difference between two neighboring receptor potentials signaled by a single spike? This differential should be signaled independent of the absolute luminance level because in a contour profile (of our environment) these differences can be at any level and the level itself often varies along a contour. We suggest, that there are two processes at work to solve this problem, a fast and a

slow one. In the fast process, a receptor potential determines the initial membrane potential and the adjustable spiking threshold of its successor (spiking) neuron, which we call ganglion cell now. For reason of simplicity, the 2nd layer (bipolar and horizontal cells) is omitted. The adjustable spiking threshold of the ganglion cells is set above the initial potential with a fixed offset. In the slow process, see figure 1b, the charge spreads laterally through the network of connected ganglion cells (time steps 1 and 2). The spiking thresholds stay fixed during this slow process. The charge of a high potential ganglion cell will spread towards its neighboring ganglion cell with a lower potential as well as a lower spiking threshold, and cause it to fire (time step 2).

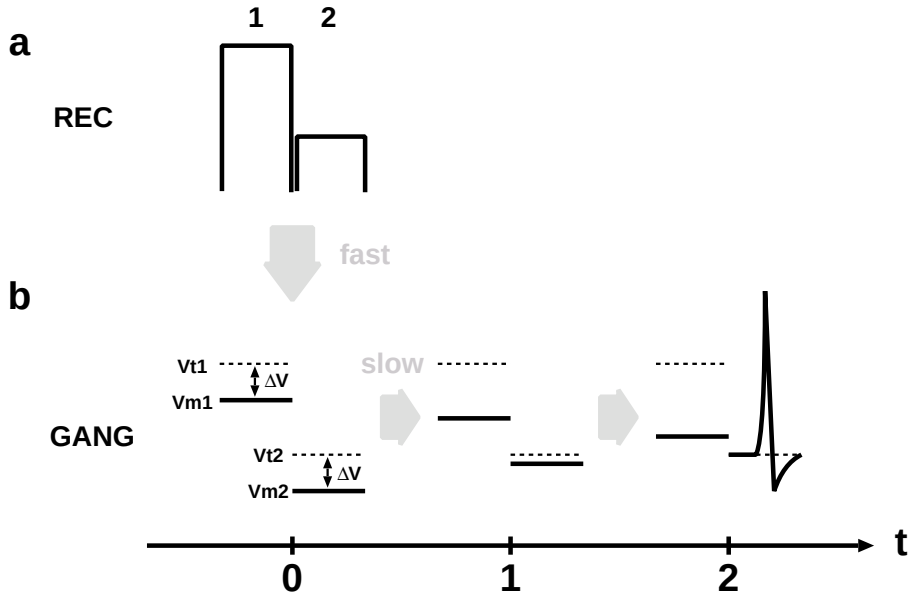

Figure 1: Receptor potentials and ganglion cell dynamics. a. Two receptor potentials with large luminance difference. b. Corresponding two ganglion cells with the membrane potential, Vm, and the spiking threshold, Vt, set above Vm with some fixed voltage offset, ΔV. Both are initially determined by the receptor potential in a fast process. In a subsequent, slower process, the charge will spread (time steps 1 and 2) and Vm will even out (spiking thresholds stay fixed), and cause the neighboring cell to fire, thus signaling the edge.

The motivation for the fast process is that receptors directly determine the membrane potential in their successive ganglion cells, whereas they indirectly determine their adjustable spiking threshold through an extra-cellular process. For example it has been shown for vari-

ous brain cell cultures, including retinal preparations, that calcium waves can spread quickly through the gap-junctions of the glia network (Charles, 1998). These calcium waves can alter the extracellular calcium concentration rapidly and substantially, and could therefore have a significant effect on the electrical behavior of neurons within short time.The charge propagation is motivated by the fact that there are traveling waves in the retina (Jacobs and Werblin, 1998).

2 Methods and Results

We have software-simulated the processes using simplest dynamics. The network is a two-dimensional sheet of integrate-and-fire neurons, which are connected by horizontal resistances forming so a continuous excitable membrane. The gray-scale values of an image are directly taken as the membrane potential for each corresponding neuron and a fixed offset is added to each value to set the spiking threshold. The subsequent horizontal propagation of activity already signals contours. Figure 2 shows results of such a simulation for two objects (desk and chair) for time steps 1, 2 and 4. The bottom row shows the results from a popular computer vision algorithm for comparison (Canny, 1986). Once the contours have been signaled, they continue to propagate across the excitable membrane. Most contours are signaled in an early phase, followed by a late phase, during which contours propagate into both directions across the retinal map, until they are canceled out due to collision with other contour-propagating waves (not shown), or until they have reached the border of the retinal map.

3 Discussion

We regard our retinal model of contour signaling as an abstraction of the wave process that may take place in the retina. The idea of an excitable membrane has already been exploited by Glaser and Barch to model motion detection (Glaser and Barch, 1999). The current simulation should foster the thinking that neural waves may have a computational function and they are not only of passive nature.

How can the output of our retina be decoded? There are basically two approaches possible: the classical, popular contour-integration approach and the less pursued contour-propagation approach.

The idea of the contour-integration approach is to integrate line pieces to form an object percept (reviewed in (Rolls and Deco, 2002)). The contour signaled during the early phase can be read out by cortical cells using a single-spike output as response, similar to Thorpe's scheme (Thorpe, 1990). Would the late phase, the contour propagation signal, interfere with the contour-integration scheme? Contour-integration may occur so rapidly, due to pure feedforward integration for instance (e.g. (Thorpe, 1990; Riesenhuber and Poggio, 1999)), that the late phase is not interpreted anymore and would be overshadowed by the fast, initial percept. Alternatively, the rapid integration in cortex may inhibit the late phase at the thalamic or cortical level via the abundant feedback loops.

The contour-propagation approach is an idea rooting in the Gestaltist's thinking of self-interacting shape (e.g. (Koffka, 1935)). The most influential, computational proposal was made by Blum (Blum, 1973), which he called the symmetric-axis transform. Some believe that the SAT - or similar region-encoding operations - is carried out in cortical areas (e.g. (Kovacs and Julesz, 1994; Burbeck and Pizer, 1995)).

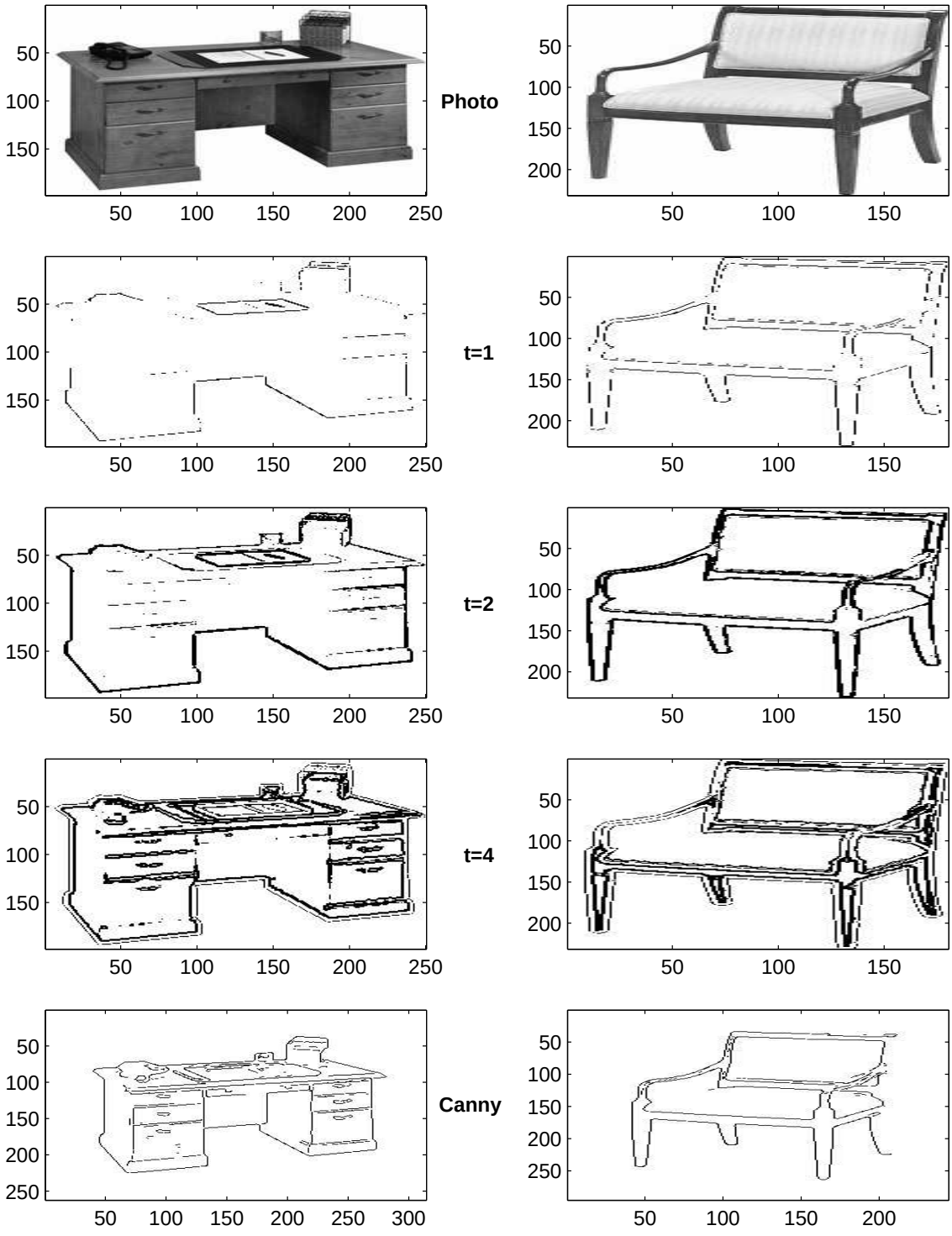

Figure 2: Contour signaling and propagation. Top row: photos. 2nd, 3rd and 4th row: Contour propagation after 1, 2 and 4 time steps, respectively. ΔV=0.5. Black lines and dots represent spikes. Bottom row: Contours obtained from the Canny algorithm (finest scale).

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