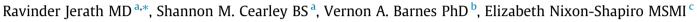
Medical Hypotheses 96 (2016) 20-29

Contents lists available at ScienceDirect

Medical Hypotheses

journal homepage: www.elsevier.com/locate/mehy

How lateral inhibition and fast retinogeniculo-cortical oscillations create vision: A new hypothesis



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ARTICLE INFO

Article history Received 1 April 2016 Accepted 21 September 2016

Keywords: After-image Human vision Lateral inhibition Phototransduction Physiological oscillations

ABSTRACT

The role of the physiological processes involved in human vision escapes clarification in current literature. Many unanswered questions about vision include: 1) whether there is more to lateral inhibition than previously proposed, 2) the role of the discs in rods and cones, 3) how inverted images on the retina are converted to erect images for visual perception, 4) what portion of the image formed on the retina is actually processed in the brain, 5) the reason we have an after-image with antagonistic colors, and 6) how we remember space. This theoretical article attempts to clarify some of the physiological processes involved with human vision. The global integration of visual information is conceptual; therefore, we include illustrations to present our theory. Universally, the eyeball is 2.4 cm and works together with membrane potential, correspondingly representing the retinal layers, photoreceptors, and cortex. Images formed within the photoreceptors must first be converted into chemical signals on the photoreceptors' individual discs and the signals at each disc are transduced from light photons into electrical signals. We contend that the discs code the electrical signals into accurate distances and are shown in our figures. The pre-existing oscillations among the various cortices including the striate and parietal cortex, and the retina work in unison to create an infrastructure of visual space that functionally "places" the objects within this "neural" space. The horizontal layers integrate all discs accurately to create a retina that is pre-coded for distance. Our theory suggests image inversion never takes place on the retina, but rather images fall onto the retina as compressed and coiled, then amplified through lateral inhibition through intensification and amplification on the OFF-center cones. The intensified and amplified images are decompressed and expanded in the brain, which become the images we perceive as external vision. *Summary:* This is a theoretical article presenting a novel hypothesis about the physiological processes in vision, and expounds upon the visual aspect of two of our previously published articles, "A unified 3D default space consciousness model combining neurological and physiological processes that underlie conscious experience", and "Functional representation of vision within the mind: A visual consciousness model based in 3D default space." Currently, neuroscience teaches that visual images are initially inverted on the retina, processed in the brain, and then conscious perception of vision happens in the visual cortex. Here, we propose that inversion of visual images never takes place because images enter the retina as coiled and compressed graded potentials that are intensified and amplified in OFF-center photoreceptors. Once they reach the brain, they are decompressed and expanded to the original size of the image, which is perceived by the brain as the external image.

We adduce that pre-existing oscillations (alpha, beta, and gamma) among the various cortices in the brain (including the striate and parietal cortex) and the retina, work together in unison to create an infrastructure of visual space that functionally "places" the objects within a "neural" space. These fast oscillations "bring" the faculties of the cortical activity to the retina, creating the infrastructure of the space within the eye where visual information can be immediately recognized by the brain. By this we mean that the visual (striate) cortex synchronizes the information with the photoreceptors in the retina, and the brain instantaneously receives the already processed visual image, thereby relinquishing the eye from being required to send the information to the brain to be interpreted before it can rise to consciousness.









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The visual system is a heavily studied area of neuroscience yet very little is known about how vision occurs. We believe that our novel hypothesis provides new insights into how vision becomes part of consciousness, helps to reconcile various previously proposed models, and further elucidates current questions in vision based on our unified 3D default space model. Illustrations are provided to aid in explaining our theory.

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Introduction to the retina

The retina is the area in the back of the eye that contains light sensitive neurons, and is the first stage of processing visual information and sending it to the brain [5–7]. Within the retina are five types of neurons: photoreceptors, bipolar cells, ganglion cells, horizontal cells, and amacrine cells [5]. Graded electrical activity is converted into action potentials in the retina, and is transmitted to the optic nerve. Since there is a relatively short distance for information to travel within the retina, action potentials are not needed, and instead, graded potentials mediate the information the retina processes [5].

In the retina there are approximately 100 million rods, 5 million cones, and only 1 million ganglion cells [8]. The horizontal and amacrine cells are responsible for the lateral interactions, which assist the visual system in detecting contrast in intensity of light [5,9,10]. The human retina is inverted to require light from projected images to pass through layers of tissue to reach the photoreceptors [11]. These layers of tissue contain various cells which cause light scattering [12] (Fig. 1). Muller cells are funnel-shaped glial cells in the retina that are essential for the transmission of light due to their unique shape, orientation, and refractive index [13]. These cells act as conduits that enable light to reach the photoreceptors with minimal scattering [13], and in mammals, every Muller cell is generally coupled with a partner cone cell [14].

The photoreceptors are rods and cones, and the infoldings in cones contain the pigment that makes up nearly 80% of the total disc protein, trapping photons as they travel through the outer segments [15]. Rods are highly sensitive to light, insomuch that only seven are needed to detect a single photon of light in a dark environment [15], whereas, cones are less sensitive and cannot detect light if photons are fewer than 100 [5]. The fovea consists of a concentrated amount of elongated cones for color vision acuity in bright light (which communicate with midget bipolar and midget ganglion cells), and extra-foveal regions for acuity in dimmer light, yet no rods exist within this area [8].

Ganglion cells synchronize the internal circadian rhythm with the external light-dark cycle, without attaching to the individual photoreceptors because they are reserved for image processing [16]. Our theory proposes that retinal layers and cells: 1) code distances, 2) compress the information about the images and distances, and 3) integrate the information with the striate cortex. Since the retina is considered to be part of the brain, we consider it as a smaller, compressed space that projects information into the larger neural space within our brains, bringing the brain to the eye. The retina is also the place where images are coiled and compressed as right side up images *before* being sent to the brain to be decompressed and expanded, according to our theory.

Images we see are processed in the retina [17], through biochemical cycles, resulting in amplification and intensity of images and their color contrasts [17]. When light enters our eyes through the cornea, it is focused in the retina on the rods and cones causing the initial image—which we do not see—to fall on the ON-center cells [5]. Photoreceptors synapse with horizontal cells and bipolar cells forming a triad, inhibiting the surrounding receptor cells, enhancing contrast, and hyperpolarizing or depolarizing bipolar cells [18]. The chain of interactions that takes place through parallel processing of visual information starts with input received from retinal photoreceptors sent via their interconnecting bipolar cells to reach the ganglion cells. Glutamate release at ribbon-type synapses allows for the transfer of information by photoreceptors and bipolar cells [19,20].

In the vertebrate retina, glutamate is not only the major neurotransmitter [21], it is also the used by the first visual synapse [22]. Glutamate depolarizes OFF-center ganglion cells and hyperpolarizes ON-center ganglion cells because of their ionotropic receptors. Reduction in glutamate has an opposite effect—hyperpolarizing OFF-center cells, and depolarizing ON-center cells [5]. ON-center ganglion cells respond to light by producing a burst of electrical activity, but the response to light in OFF-center ganglion cells results in decreased electrical activity. The cells respond with a burst of action potentials when the light is turned off [5] due to

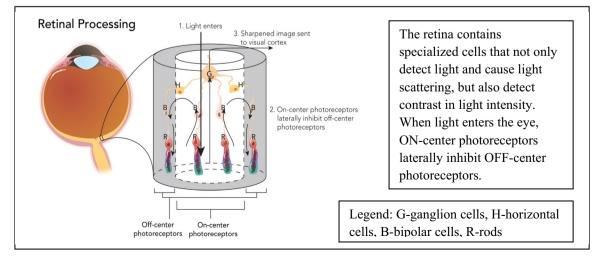


Fig. 1. Retinal Processing.

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