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Review Paper

Neural stimulation for visual rehabilitation: Advances and challenges

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ABSTRACT

Blindness affects tens of million people worldwide and its prevalence constantly increases along with population aging. In some pathologies leading to vision loss, prosthetic approaches are currently the only hope for the patient to recover some visual perception. Here, we review the latest advances in visual prosthetic strategies with their respective strength and weakness. The principle is to electrically stimulate neurons along the visual pathway. Ocular approaches target the remaining retinal cells whereas brain stimulation aims at stimulating higher visual structures directly. Even though ocular approaches are less invasive and easier to implement, brain stimulation can be applied to diseases where the connection between the retina and the brain is lost such as in glaucoma and could therefore benefit to patients with different pathologies. Today, numbers of groups are investigating these strategies and the first devices start being commercialized. However, critical bottlenecks still impair our scientific efforts towards efficient visual implants. These challenges include electrode miniaturization, material optimization, multiplexing of stimulation channels and encoding of visual information into electrical stimuli.

Contents

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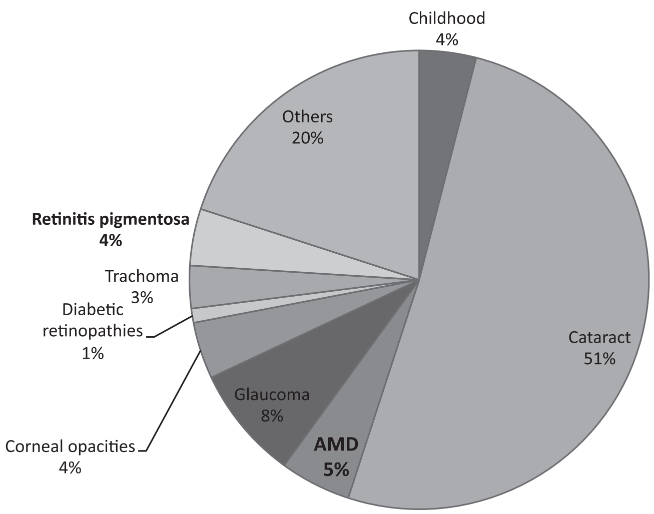

Fig. 1. Global causes of blindness. Percentages of blindness causes in 2010 (AMD: Age-related Macular Degeneration). Retinitis pigmentosa and age-related macular degeneration account for 9% of blindness cases. Data from the World Health Organization (2010).

1. Introduction

Blindness is one of the most debilitating sensory impairment affecting 39 million people worldwide. The leading cause of this sensory disability is cataract accounting for 51% of cases, however, it can be treated very efficiently, but access to treatment remains an issue in most underdeveloped countries. The remaining causes of acquired blindness are glaucoma, age-related macular degeneration (AMD), diabetic retinopathy, and retinitis pigmentosa (RP). Fig. 1 summarizes the global causes of blindness and their prevalence. In these diseases, different cell types can be deficient and degenerate, thereby triggering blindness. For instance, photoreceptors are degenerating in RP and AMD whereas retinal ganglion cells sending visual information to the brain are lost in diabetic retinopathy or glaucoma. For some of these diseases, there is currently no efficient treatment for preventing severe visual loss or blindness. This is the case for RP accounting for 1 million patients worldwide. In these pathologies, photoreceptor degeneration leads to a progressive reduction of the visual field often declining to legal blindness. For the past 40 years, tremendous efforts towards visual rehabilitation through electrical stimulation of the neural tissue with implanted electrodes have been conducted. We will present here the recent developments and our latest advances in the field of visual prosthetics and the current technological and conceptual bottlenecks that will need to be overcome to restore functional vision in blind patients.

Fig. 2. LeRoy's experimental procedure for ocular stimulation in 1755. A conductive wire was place around the head of a patient blind from cataract with another one around his leg. The discharge of a Leyden jar passed through the eye of the patient evoking vivid flashes of light.

2. Prosthetic rehabilitation

In 1755, Charles LeRoy applied the electric discharge of a Leyden jar – an ancestor of capacitors – on the ocular surface of a patient blind from cataract (Fig. 2). This stimulation elicited vivid flashes of light or phosphenes, reported by the patient. It was the starting point of visual prosthetics and from this point, various strategies have been investigated to restore visual perception through electrical stimulation.

Electrical stimulation of the visual system can be performed on multiple locations along the sensory pathway. First, the retina can be stimulated in case of ganglion cell survival and preservation of the information flow through the optic nerve. Stimulating the optic nerve directly is also possible, although the high density of nerve fibers is an issue for stimulation control. And finally, it is possible to stimulate brain structures such as the lateral geniculate nucleus (LGN) or the visual cortex directly, even in case of complete retinal degeneration or optic nerve injury. However, these strategies are much more invasive. In all cases, the device consists in a photosensitive part – i.e. camera – a processing stage and an array of electrodes in contact with the targeted structure (Fig. 3).

We will present here the latest advances in visual implants and the major challenges that need to be addressed.

2.1. Epiretinal implants

Epiretinal implants electrically target the ganglion cell layer. A matrix of electrodes is directly fixed on the surface of the retina with a tack and connected to a stimulator receiving data and power through coil–coil interaction and radio-frequency (RF) signal.

Humayun et al. were the pioneer of epiretinal implants (Humayun et al., 2003, 2009, 2012). The first epiretinal device to be chronically implanted in patients – the Argus I – developed by Second Sight Medical Products was composed of 16 Pt electrodes (Humayun et al., 2003; Caspi et al., 2009). Their report confirmed that light perception could be achieved through epiretinal stimulation. The implanted patient was able to recognize shapes, gratings orientations, and had a restored visual acuity of 20/3240.

The next generation of their epiretinal device named Argus II was designed to reach a higher resolution. Fig. 4 describes the Argus II device containing a 6×10 electrode matrix implanted in 30 subjects from 2007 to 2009.