

Diseased optic nerves – the use of microchips

Ross G. Cooper¹, Andrew Tennett², Jason Cooper³

¹ Physiologist, 22 Kimble Grove, Pype Hayes, Erdington, Birmingham B24 0RW, UK

² Final year student in Mechatronics, Mechatronics Department, Faculty of Engineering, Nelson Mandela Metropolitan University, Port Elizabeth, South Africa

³ Consulting Optician FBDO CL (Hons.), Attleborough, Nuneaton, Warwickshire, UK

Key words: optic neuropathies, electrical stimulation of retina, retinal vision prostheses, microchips in the optic nerve

The eye is an extremely complex and efficient sense organ, endowed with a layer of receptors, a lens and a cornea that focus light onto the receptors, and an optic nerve that conducts nerve impulses to the visual cortex in the brain [1, 2]. In case of optic nerve damage, microelectrodes could be inserted from an embedded microchip, or a series of microchips, into fibre bundles within the nerve. The aim of this article, therefore, is to advocate the usefulness of microchips and their biocompatibility for the alleviation of optic nerve disease.

The optic nerve may be afflicted by vascular and/or ischemic disease, inflammatory or infectious disease, and degenerative disease [3]. Widely-reported optic nerve diseases include glaucoma, optic neuritis, and ischemic optic neuropathy. Optic nerve degeneration and mitochondrial dysfunction are associated with genetic and acquired optic neuropathies [4]. Such damage would require methods to assist damaged ganglion nerve fibres, including prosthetics targeting distal portions of the visual pathway [3]. Recently, a medial trans-conjunctival approach was used to implant a stimulating electrode around the intra-orbital section of the optic nerve, allowing sufficient exposure of the nerve after detaching one rectus muscle and performing a lateral canthotomy [5]. HIV⁺ individuals often express neuro-ophthalmological opportunistic pathology, including homonymous haemianopsia, caused by foci of cerebral toxoplasmosis, progressive multi-focal leucoencephalopathy or primary intra-cerebral malignant lymphoma [6]. Ocular congenital toxoplasmosis acquired through *Toxoplasma gondii* infection has numerous symptoms, including optic neuritis [7], optic atrophy [8] and necrotizing retinitis [9]. Damage to the macula densa results in severe loss of visual acuity¹⁰. Optic atrophy is the end process of many factors damaging the optic nerve, characterised by a loss of axons with glial proliferation and decreased vascularity of the nerve head [10]. The use of implanted microchips should utilise and stimulate healthy ganglion cells in the optic nerve directly.

It is important to recognise the biocompatibility of different materials within the eye. Microchips should be non-corrosive. A recent study evaluated the use of a flexible polyimide substrate with an integrated platinum electrode [11]. In that investigation, a parylene C coating was deposited on the

electronic components as an insulation layer, and silicone rubber used to encapsulate the electronics. Electrical stimulation of the retina evoked neuronal responses in the visual cortex and indicated the feasibility of the system approach for chronic use [11]. Poly (haema) hydrogels appear to be non-corrosive, are useful for carrying Schwann cells, and may provide a stable three-dimensional scaffold capable of supporting axonal regeneration in lesioned optic nerves [12].

Ideally, a microchip positioned on the optic nerve will elicit a visual response. However, finer optometrical details of light progression in the eye need further study. We have therefore proposed equations for determining the distance that light must travel in the eye. Given that the average eyeball is 24.5 mm long and the thickness of the retina 120 µm, thicknesses added due to implantation of microchips will be as small as ca. 0.05 mm per chip. Assumed variations, including those due to the angle of incidence and refraction of light, as well as attenuated speeds of light through the aqueous and vitreous humours, would require further determination. The approximate time taken for light to reach the electrodes attached to the optic nerve is expressed by:

$$\frac{x + y + zn}{s}$$

where:

x = length of the eye from the cornea to the retina;

y = thickness of retina;

z_n = thickness of the microchip;

n = number of microchips;

s = speed of light.

The variation of distance within the eye through which light must travel may be calculated using the following formula:

$$\int_b^a (x + y + zn)^{\frac{a}{b}}$$

where:

a = upper variable of number of microchips;

b = lower variable of number of microchips..

Further investigations should be performed to understand the pathology of congenital degeneration of the optic nerve and retinal function, which would lead to devising microchips that are non-corrosive, and capture and channel light energy. Thereafter, action potentials within the optic nerve would be stimulated. Additionally, more investigations on light

Corresponding author: Dr. Ross G. Cooper, Physiologist, 22 Kimble Grove, Pype Hayes, Erdington, Birmingham B24 0RW, UK.
E-mail: rgcooperuk@yahoo.com

Received: 7 January 2009; accepted: 15 December 2009

direction and incidence of light rays within the implanted prostheses inside the eye should be undertaken as this may give a clearer indication of the degree of neuronal stimulation within the optic nerve.

CONFLICT OF INTEREST

None declared.

REFERENCES

1. Alward WLM: A new angle on ocular development. *Science* 2003, **299**, 1527-1528.
2. Ganong WF: Review of Medical Physiology (21st edn). McGraw Hill, Boston 2003.
3. Margalit E, Sadda SR: Retinal and optic nerve diseases. *Artif Organs* 2003, **27**(11), 963-974.
4. Carelli V, Ross-Cisneros FN, Sadun AA: Optic nerve degeneration and mitochondrial dysfunction: genetic and acquired optic neuropathies. *Neurochem Int* 2002, **40**(6), 573-584.
5. Brelén ME, De Potter P, Gersdorff M, Cosnard G, Veraart C, Delbeke J: Intraorbital implantation of a stimulating electrode for an optic nerve visual prosthesis. Case report. *J Neurosurg* 2006, **104**(4), 593-597.
6. Fabricius EM: [Why are AIDS patients frequently visually impaired?]. [Article in German]. *Ther Umsch* 1996 **53**(1), 49-57.
7. Korsholm K, Madsen KH, Frederiksen JL, Skimminge A, Lund TE: Recovery from optic neuritis: an ROI-based analysis of LGN and visual cortical areas. *Brain* 2007, **130**(Pt 5), 1244-1253.
8. Mets MB, Holfels E, Boyer KM, Swisher CN, Roizen N, Stein L, Stein M, Hopkins J, Withers S, Mack D, Luciano R, Patel D, Remington JS, Meier P, McLeod R.: Eye manifestations of congenital toxoplasmosis. *Am J Ophthalmol* 1996, **122**(3), 309-324.
9. Tabbara KF: Ocular toxoplasmosis. *Int Ophthalmol* 1990, **14**, 349-351.
10. Cull RE, Will RG: Diseases of the nervous system. **In:** Edwards CRW, Bouchier IAD, Haslett C, Chilvers ER editors. Davidson's Principles and Practice of Medicine (17th edn). Churchill Livingstone, London 1995, 1021-1115.
11. Schanze T, Hesse L, Lau C, Greve N, Haberer W, Kammer S, Doerge T, Rentzos A, Stieglitz T: An optically powered single-channel stimulation implant as test system for chronic biocompatibility and biostability of miniaturized retinal vision prostheses. *IEEE Trans Biomed Eng* 2007, **54**(6), Part 1, 983-992.
12. Plant GW, Harvey AR, Chirila TV: Axonal growth within poly (2-hydroxyethyl methacrylate) sponges infiltrated with Schwann cells and implanted into the lesioned rat optic tract. *Brain Res* 1995, **671**(1), 119-130.