

HDR Sub-retinal Implant for the Vision Impaired

Heinz-Gerd Graf, Alexander Dollberg, Jan-Dirk Schulze Spüntrup
and Karsten Warkentin

10.1 Introduction

The photo-resistive cells of the human retina can degenerate by different diseases leading to about 50% of the incidence of total blindness. Retinitis Pigmentosa and age-related macular degeneration are two main examples for the progressing degeneration of the outer retina. Until now, there is no known successful medical treatment to cure the tissue and renew the visual faculty [1]. Thus, a visual prosthesis to recover sight is an important application of integrated micro-systems. The different concepts apply electrical signals to the visual cortex [2], the optic nerve [3] or to the retinal tissue.

For retinal implants, basically two different types exist. The epi-retinal implant uses the bottom layer of the retina to supply electrical impulses for stimulation. In the healthy eye the processed information is sent to the brain by the ganglion cells. The epi-retinal implant is mounted on the retina and supplies the electrical impulses to the ganglion cells/optic-nerve interface [4–6]. Therefore, a complex image processing and transmission unit is needed to generate an appropriate stimulus.

The second method is the sub-retinal implantation which is used by the implant introduced here (Fig. 10.1). The degenerated photo-receptors are in loco replaced by the implant. They directly stimulate the following bipolar cells and thus use the image processing abilities of the retina and eye movements. No image processing unit is needed in this implant. For sub-retinal implants, a high number of artificial stimulation cells is needed to replace the light-sensitive human cells in a sufficient manner. In many reports, passive photo-diode arrays for electrical stimulation have been proposed [7–9]. After a thorough investigation it appeared that the generated impulses were not strong enough to successfully stimulate the retina [10]. Thus, an active implant with analogue amplifiers for processing the photodiode signals was developed [11].

Similar to the micro-photodiode arrays the active retinal implant utilises the eye-lens image to create the electrical stimulation in the different image

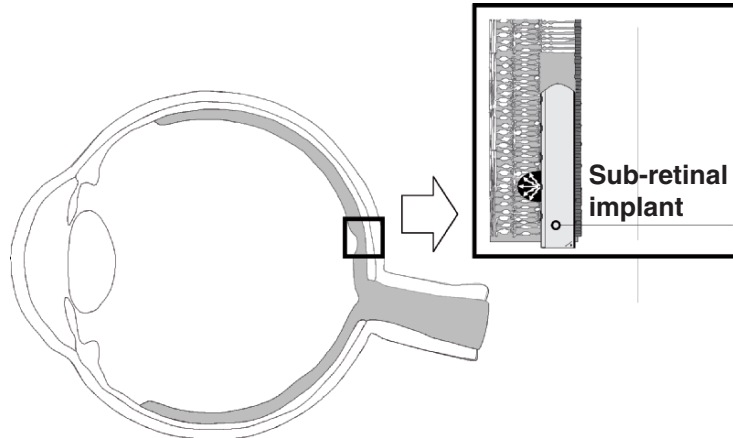


Fig. 10.1. Schematic of the human eye with the insertion of a sub-retinal implant

areas. The amplifier cells convert differences between the local and the average illumination into electrical impulses. The stimulation electrodes placed on every amplifier supply an amount of charge locally into the tissue of the retina.

The maximum height of the output pulses and also the DC level are strictly limited because higher values can cause electrolysis effects and decompose the tissue surrounding the electrode. This could permanently damage the retina and has to be avoided. Moreover, the injected charge has to be in a particular range to ensure successful stimulation of the retina [8,9]. For this reason, a very important circuit requirement is the measurability and testability of any amplifier cell on the implant.

10.2 Electronic HDR Photoreceptors

The conversion of the brightness into a voltage is performed by a logarithmic cell. A photodiode is driven in the reverse direction. Corresponding to the chip illumination, a proportional reverse current is generated in the photodiode. The photo-current is supplied to a MOS transistor which is in sub-threshold region as description in Fig. 2.6 (Fig. 10.2). The voltage drop across a MOS transistor, which works in the sub-threshold region, is exponentially dependent on the current. A logarithmic change in illumination causes a linear change in the output voltage of the logarithmic cell. This results in a logarithmic behaviour similar to the light sensitivity of the eyes. This circuit is well known to work for a very wide dynamic range of more than $1-10^6$.

Averaging the voltage of some cells, by an analogue summation and attenuation circuit, results in a signal representing the average illumination. This signal is very robust to the large contrast of a natural environment.

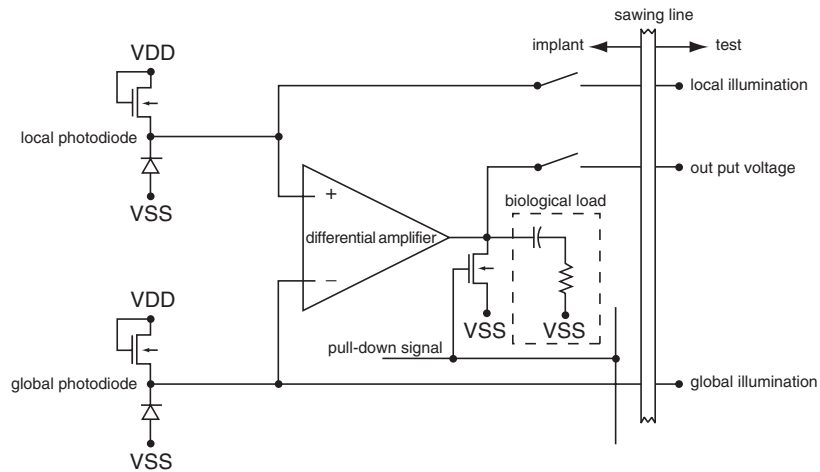


Fig. 10.2. Circuit schematic of one pixel of the implant

Bright local illumination of a small part of the “average illumination” cells network results only in small changes of the average illumination.

10.3 The Differential Principle

For the amplifier cells, a differential configuration was chosen to get differences in lightness independent of the absolute level of brightness (Fig. 10.2). In this way, patterns can be identically recognized in daylight and twilight. The negative input of the differential amplifier is the representation of the local illumination. The voltage corresponding to the global illumination is connected to the positive input. If the local brightness rises, the negative input voltage decreases and the output voltage of the amplifier increases. The output of the amplifier is not dependent on the absolute voltage level in a very wide range of illumination. The output responds only to the difference between the illuminations. The resulting signal corresponds mainly to differences in the reflectance of illuminated objects. Each pixel delivers a charge proportional to the log of the illuminance, effectively $2.5 \mu\text{C}$ per decade of illuminance and the dynamic range of each pixel is two decades of illuminance around the global illuminance. The maximum charge packet delivered at the pixel output is 30×10^{12} electrons. The two decades of sensitivity are shifted with the global illuminance level.

10.4 The Complete Amplifier Cell

The complete cell integrates the HDR photoreceptors for local and average illumination, a differential amplifier and the discharge switch. As test circuits,

there is a NAND an inverter gate for cell selection and some switches needed to connect the output of the amplifier to the test-circuit and to make the inputs accessible.

A pulsed power supply is connected to the amplifier. With each pulse, the amplifier cell emits charge between 0.5 and 10 nC to the high capacitive load connected to the tissue. The maximum height of the output pulses is set to 2 V because a higher voltage can cause electrolysis effects. For this reason, the design of the amplifier considers 2 V as the maximum output voltage of the amplifier. The electrode capacitance restricts the charge margin.

Discharging the output node to ground reference voltage during the time of inactivity secures charge balance at full pulse cycle. This is done by a pull-down circuit that forces the amplifier outputs to ground during inactivity and thus discharges the electrodes. Additionally, this implements charge balance regarding the stimulation impulses.

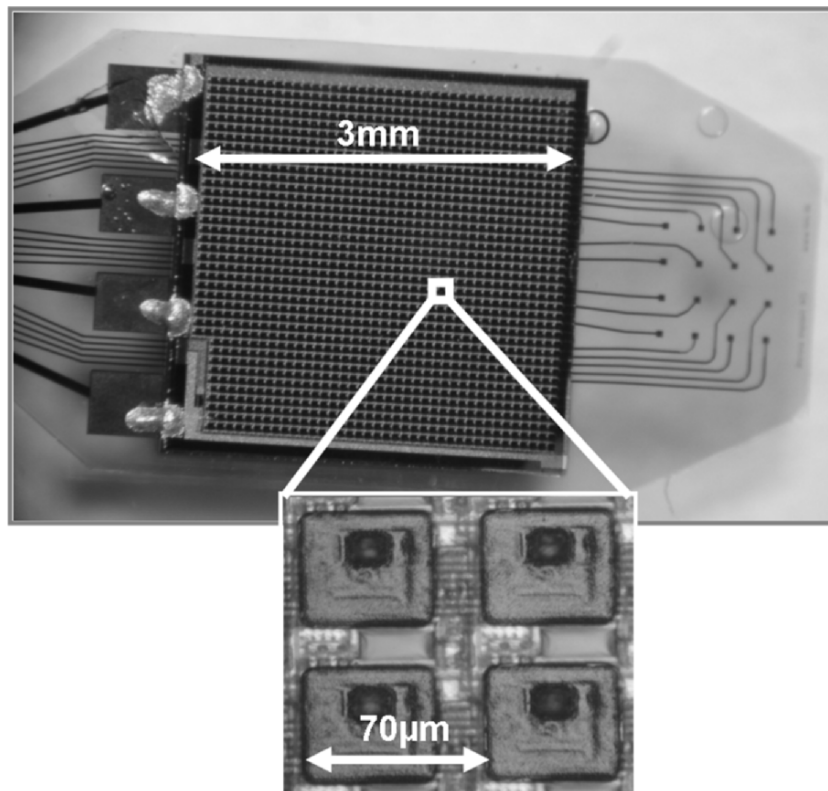


Fig. 10.3. Capsulated retinal implant. The higher magnification shows the output contacts to the tissue (photo: Retina Implant AG)

The pull-down signal is generated by an NMOS inverter. The inverter is connected to a different supply voltage to keep on working even if the pulsed supply is switched off. The dimensions of the cells are $72 \times 72 \mu\text{m}^2$.

10.5 The Retinal Implant

The complete implant consists of 38×38 cells. The space of the missing cells is needed to place the reference source, the pull-down inverter and the bonding pads to connect the final implant. The dimension of the implant chip is $3 \times 3.1\text{mm}^2$ (Fig. 10.3).

During normal operation, the implant uses a pulsed supply at about 20 Hz. The active time per period is about 500 μs . The pull-down signal is generated by means of an additional DC supply voltage.

Acknowledgement

The project, in which this implant was developed, was funded in part by BMBF (German Federal Ministry of Education and Research). The cooperation with NMI (Naturwissenschaftlich Medizinisches Institut, Reutlingen) with the Augenklinik, University Tuebingen and with Retina Implant AG, Reutlingen, is gratefully acknowledged.

References

1. E. Zrenner, Will retinal implants restore vision, *Science*, 295, 1022–1025, Feb. 2002
2. W.H. Dobelle, Artificial vision for the blind by connecting a television camera to the visual cortex, *ASAIO-J*, 46, Jan–Feb 2000, 3–9
3. C. Veraart et al., *Brain Res.* 813, 181, 1998
4. R. Eckmiller, Learning retina implants with epi-retinal contacts, *Ophthalmic Res.*, 29, 281–289, 1997; A.Y. Chow, M.T. Pardue, V.Y. Chow, G.A. Peyman, C. Liang, J.I. Perlman, N.S. Peachey, Implantation of silicon chip microphotodiode arrays into cat sub-retinal space, *IEEE J. Solid State Circuits*, 35(10), 2000
5. M. Schwarz, R. Hauschild, B.J. Hosticka, J. Huppertz, et al., Single-chip CMOS image sensors for a retina implant system, *IEEE Trans. Circuits Systems II*, 46(7), 1999
6. R.J. Greenberg, T.J. Velte, M.S. Humayun, G.N. Scarlatis, E. de Juan, A computational model of electrical stimulation of retinal ganglion cell, *IEEE J. Solid State Circuits*, 35(10), 2000
7. A.Y. Chow, Electrical stimulation of the rabbit retina with subretinal electrodes and high density microphotodiode array implants, *Invest. Ophthalmol Vis. Sci.* 34(suppl), 835, 1993

8. M. Stelzle, A. Stett, B. Brunner, M. Graf, W. Nisch, Electrical properties of micro-photodiode arrays for use as artificial retina implant, *Biomed. Microdevices*, 3(2), 133–142, 2001
9. B. Schlosshauer, A. Hoff, E. Guenther, E. Zrenner, Towards a retina prosthesis model: Neurons on microphotodiode arrays in vitro, *Biomed. Microdevices*, 2(1), 61–72, 1999
10. F. Geckler, H. Schwahn, A. Stett, K. Kohler, E. Zrenner, Sub-retinal micro-photodiodes to replace photoreceptor function. A review of the current state, *Vision, sensations et environment*, Irvin, Paris, 2001, pp. 77–95
11. A. Dollberg, H.G. Graf, B. Höflinger, W. Nisch, J.D. Schulze Spuentrup, K. Schumacher, E. Zrenner, A fully testable retinal implant, *Proceedings of IASTED Conference on Biomedical Engineering*, Salzburg 2003, pp. 255–259