# Modeling of the Organ of Corti Stimulated by Cochlear Implant Electrodes

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*Abstract* – Cochlear implants are used by deaf people to recover partial hearing. The electrode array inserted inside the cochlea is an extensive area of research. The aim of the electrodes array is to directly stimulate the nerve fibers inside the Organ of Corti. The electrical model of the physical system consisting of Organ of Corti and the electrodes is presented in this paper. This model allowed to run Spice simulations in order to theoretically detect the minimal voltage sufficient for nerve fiber stimulation as well as the impact of the electrode voltage on the duration of nerve fibers stimulation. Besides, the perturbations introduced by one electrode on the neighboring electrodes were theoretically studied. Finally, the study of power consumption was performed, which is of great importance in such embedded system.

Keywords: cochlear implant, electrical analog, transient simulations

## I. INTRODUCTION

Cochlear implants are an electrical device used by severely deaf people to gain or recover partial audition. They allow direct stimulation of the auditory fibers using an electrodes array designed to reproduce the stimulus that would be generated by a healthy cochlea.

To do so, an external part of the hearing devices is located outside the ear and contains a microphone that captures the acoustic waves and transforms them into an electrical signal used by the data processing unit. Then, this signal is transmitted to the receiver, located within the patient's head, close the skull. The receiver is composed of a demodulator and a set of electrodes driven by electrical signals that will contract the cochlea and stimulate the auditory nerve [1] [2].

Cochlear implants directly stimulate the nerve fibers inside the cochlea, and requires surgery to pull the electrodes array inside the scala tympani (Figure 1).

The connection between the electrodes in the scala tympani and the auditory nerve fibers is critical for efficient nerve fibers stimulation.

In an healthy ear, when a sound wave is produced, it strikes the eardrum and this vibration is reported in the oval window using ossicles. The oval window is the very first part of the cochlea. This oval window vibration creates a wave propagating inside the scala vestibuli, which is filled with perilymph.





According to biophysical theories [3] [4] when a mechanical wave propagates inside the cochlea, the Basilar Membrane (BM) distorts to absorb the wave energy, resulting in a height variation of the BM which compresses the organ of Corti. As shown in Figure 2, the organ of Corti is composed of Hair Cells (HC) (Outer Hair Cells (OHC) and Inner Hair Cells (IHC)), which have stereocilia at their end. When BM vibrates, stereocilia position change allowing potassium channels to open [5] [6]. Opening of the potassium channels creates the depolarization of the HC allowing complex mechanisms to take place (reviewed in [7] [8] [9]), and finally, resulting in neurotransmitter released in the synapse. Once released, these neurotransmitters travel to the post synaptic cell (the nerve fiber) and creates the depolarization of the nerve fiber. This depolarization, if sufficiently important, generates an Action Potential (AP) running through the nerve cell membrane [10] [11].



Figure 2. Organ of Corti

The aim of the electrodes array of the cochlear implant is to generate an AP once a sound is perceived.

Consequently to obtain the same Action Potential at the nerve fiber using only electrode stimulation, two possibilities exist. First the direct nerve fiber stimulation can be made by changing the nerve membrane potential in order to produce a membrane depolarization above the threshold of Voltage Sensitive Na+ Channels (Na<sub>v</sub>) to create an AP [12]. The second solution consists in opening the potassium channels of the stereocilia to recreate the complete stimulation process. As HC or stereocilia are disfunctionning in the vast majority of implanted patients, only the first mechanism is considered in this paper.

Electrical model of electrodes inserted within the cochlea have been proposed by Hartmann et al. [13], where the spatial distribution of electrical potential was measured for intracochlear stimulation. In addition, electronic model of electrode/neuron coupling is available in [14] in order to reveal the most efficient coupling conditions. However, both models lack of physical connection with AP generation. In this paper, we present an electrical description of the electrode and organ of Corti in order to obtain theoretical minimal stimulation voltage sent to the electrodes for AP generation. Furthermore, this model allowed us to link the stimulation voltage with the duration of the nerve fibers stimulation. Finally the impact of surrounding electrodes were theoretically investigated.

The next section presents the theoretical model developed for the Organ of Corti associated with the electrodes. Then, simulation results from Spice software are presented. Finally, the conclusion and future work direction are presented.

#### II. MATERIALS AND METHODS

The electrical equivalent circuit of human tissue used in this paper is the one presented in figure 3 and extracted from Cole and Cole impedance model [15], which has been shown to fit experimental data. The human tissues considered were the one present in Figure 2. The value of Rs, Rp and Cp were obtained using a gain response extraction over frequencies analysis. As the maximum hearing frequency is 22khz, it was considered in this paper that Rp could be neglected as it models the energy loss in high frequencies.



Figure 3. Human tissue electrical analog [15]

To obtain the numerical values for Rs and Cp for all the tissues or interfaces, we use the physical equations for the capacitance (parallel-plate capacitor) and for the resistance computation (cylindrical resistance model) [16] [17].

The values of the relative permeability and electrical conductivity for the nerves were extracted from [18] or from [19] for platinum as electrodes are mainly composed of it. However as far as the authors know, no relative permeability or electrical conductivity was available for Deiter cells or Basilar Membrane tissue. As Deiter cells are mainly composed of microtubules [20], which are involved in mechanical transport as actin proteins found in muscles cells and because the width of the BM is negligible compared to the Deiter cell height, we chose to take the relative permeability and electrical conductivity of muscle cells to characterize those two tissues.

On the other hand, the computation of the capacitance and the resistances ( $C_{patch}$ ,  $Rp_{patch}$ ,  $Rs_{patch}$ ) between two electrodes is more complex, as highlighted Figure 4, those variables depend on the distance between the two electrodes. The Cable Model Theory was used to compute those variables as the space between two electrodes is composed of various tissues rendering the Cole and Cole model implementation difficult. We simplified the tissue between two electrodes as only made of Deiter cells, then we implemented the Cable Model Theory in order to obtain a general impedance depending on the electrodes distance.



Figure 4. Two surrounding electrodes influences the nerve fibers targeted.

To compute  $Rs_{patch}$ , the cylindrical model of resistance was considered. The cylinder going from the first electrode to the second electrode, as defined in Figure 5.a, was used to compute  $Rs_{patch}$  (expressed in (1)):

$$R_{spatch} = \frac{1}{\sigma_M} * \frac{l_{spatch}}{S_{spatch}}$$

with 
$$l_{spatch}(y) = \int_{0}^{Y_{tot}} y * dy$$
  
and  $S_{spatch}(y) = \int_{0}^{2*\pi} \int_{-\frac{X1}{2}}^{\frac{X1}{2}} \rho * d\rho * d\theta$  (1)

where  $Y_{tot}$  is the distance between the two electrodes,  $X_1$  is the distance between one electrode and the corresponding nerve fibers. Those values were respectively extracted from [22] and [23]. y is the variable shown in Figures 5.a, 5.b and 5.c.  $\sigma_M$  is the electrical conductivity of muscle cells.

 $Rp_{patch}$  models the resistance between the two longitudinal edges of the cylinder defined previously. Hence, this computation changes as expressed in (2), as it models all the losses through the ground from one electrode to another one.

$$R_{ppatch} = \frac{1}{\sigma_M} * \frac{l_{ppatch}}{S_{ppatch}}$$
with  $l_{ppatch}(y) = \int_0^{Z_1} dz$ 
nd  $S_{spatch}(y) = \int_0^{2*\pi} \int_0^y \rho * d\rho * d\theta$  (2)

where we supposed  $Z_1$  equal to  $X_1$  for simplification purposes. We defined  $Cp_{patch}$  as a squared parallel plate capacity (Figure 5.c) (developed in (3)):

$$C_{p} = \mathcal{E}_{0}\mathcal{E}_{M} \frac{A_{patch}}{d_{patch}}$$
with  $d_{patch}(y) = \int_{0}^{Z_{1}} dz$ 
and  $A_{patch}(y) = \int_{0}^{y} dy_{1} \int_{0}^{X_{1}} dx$  (3)

where  $\mathcal{E}_{M}$  is the muscle relative permeability.

а

For reader's convenience, the value of the capacitance and resistance described previously are summarized in Table 1.





Figure 5. Physical model of Rs<sub>patch</sub> (5.a), Rp<sub>patch</sub> (5.b) and Cp<sub>patch</sub> (5.c)

The electrical description containing only a single electrode is shown Figure 6.a. The input voltage generator is directly connected to the electrodes analog model (low frequencies model), which can be eventually considered as a perfect conductor compared to the other resistance values. Then the current can flow to the nerve cell or can go back to the ground. The current loss through the physical isolation between the electrode and the ground is neglected as the insulator has a low loss tangent (high resistivity). The membrane rest potential of a nerve cell is around -70mV, explaining the two -70mV voltage generators, in figure 6.a. We defined the analog equivalent circuit of a nerve cell using a resistance ( $R_n$ ) in parallel with a capacitor ( $C_n$ ) (this electrical description should not be confused with the Hodgkin-Huxley model [24], which is used to model ions flow through the nerve cell membrane and not the electron flow).

In addition, the electrical description of the system starting from the nerve and going through all the body to the earth was not considered because very little electrical current is going through this pathway.

TABLE 1. RESISTANCES AND CAPA	ACITANCES USED IN THE
ELECTRICAL MODEL	

Electrodes	Re= 1.5 Ω, Ce=11 fF
Basilar Membrane and Deiter cells	$R_{bc} = 933 \Omega, C_{bc} = 300 nF$
Nerve fibers	$R_n = 1076\Omega, C_n = 3\mu F$
Cable Model Theory	$Rs_{patch} = 8 M\Omega$ , $Rp_{patch} = 1265$ $\Omega Cp_{patch} = 92.6 nF$

Figure 6.b exhibits the electrical description of the overall system with two surrounding electrodes added. They are composed of a voltage generator, the platinum electrode equivalent circuit and the cable model ( $Rs_{patch}$ ,  $Rp_{patch}$  and  $C_{patch}$ ), to connect the peripheral electrodes with the nerve fiber that we want to activate.

y

5.b



Figure 6.a. Electrical analog of the electrode and nerve. Figure 6.b. Electrical analog with three electrodes

The main goal of the addition of the two surrounding electrodes was to study theoretically the influence of these on the stimulation of selected nerve fibers (or more precisely of the packet of nerve fibers that should only be stimulated by the central electrode). These perturbations, if significant, could make the sound reconstitution inaccurate.

### III. RESULTS

The membrane potential (Vm) (which corresponds to the difference of potential between point A and point B in figure 6.b) had to vary of 30mV to generate an AP. The electrode stimulation (Velec) was made using a DC source. Neglecting the effect of the capacitors, Vm varied linearly with Velec and the variation of 30mV was reached for an electrode stimulus around 0.9V.

When a nerve fiber is stimulated constantly, it will not produce an AP indefinitely but rather produce a succession of randomly spaced AP called spike trains. The spike train length that could be produced by a sound of given intensity has to be reproduced with the electrodes of the cochlear implant. We performed transient simulation including the capacitors effects by injecting a square voltage with a period of 150ms. This experiment was repeated for input square voltages varying from 1V to 5V (Figure 7.a). The aim of this simulation was to study if the voltage amplitude sent to the electrode would affect the spike train duration and starting time. Figure 7.b reveals that the delays for Vm potential to reach its maximum value were around  $0.1\mu$ s, which were small compared to the duration of a nerve AP (few ms). This result pointed out that theoretically the electrode voltage magnitude had a very insignificant effect on the spike train duration. In addition, the recreated spike train starting time has negligible delay with the electrode stimulation starting time.



Figure 7.a. Transient simulation with different electrode voltage as input and Vm voltage as output. Figure 7.b. Time before AP generation depending on the electrode voltage

A general overview of the spike train related to the Vm amplitude is presented in Figure 8. The AP generated were obtained from basic mathematical functions in order to model the nerve fiber AP created after square voltage electrode stimulation. The interspike time was taken randomly and greatly depends on the amplitude of the stimulus [25]. However, the electrical analog presented in this paper does not account for this effect.



Figure 8. Spike train generated by the electrode input voltage

We performed also a parametric simulation using the electrical description of Figure 6.b, where the surrounding electrodes are added. The central electrode had a DC voltage of 1V and we varied the voltage of the surrounding electrodes between 0.9 and 5V. According to RC values used in figure 6.b, analytical computation showed that when the voltage of the surrounding electrodes was maximum (5V), the nerve fibers (above the central electrode) membrane potential Vm variation was 0.5mV, which was not high enough to stimulate these nerve fibers (the ones that should be stimulated only by the central electrode).

The overall system consumption is a great significance as cochlear implants are not convenient for the user to recharge. The study of the power consumption is presented Figure 9.



Figure 9. Current consumption during one stimulation period

The current peaks during each input signal transitions could reach 1A. Consequently, the maximum power consumed during a square input signal generation by the electrodes was around 1W (peak value), whereas the mean power consumed per period was around 50mW. These results may be used for the electrode array design to define battery size as well as electrode minimum width.

#### IV. CONCLUSION

The theoretical electrical description presented in this paper was used to carry out simulations allowing the detection of the minimum voltage needed to ensure nerve fibers stimulation. This voltage was found around 0.9 V. Furthermore, the current peaks during each input signal transitions could reach 1A (peak value), and the mean power consumed per period was around 50mW. These results may be used as requirements for the electrode array design and corresponding control electronics.

It has also been suggested that two consecutive electrodes were not disturbing one another and that the duration of the stimulation did not depend on the input electrode voltage. A more complex model, including the spike trains frequency (which is the number of spikes generated per second) related to the electrode input voltage is being currently developed.

Besides physical tests are ongoing to ensure that the theoretical results obtained match the measurements. Deaf people using cochlear implants were asked to kindly submit themselves to cochlear implant reprogramming in order to test if the threshold of 0.9 V was sufficient; if not, it would greatly affect the perturbation between electrodes.

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