A Small Analog VLSI Inner Hair Cell Model

André van Schaik
School of Electrical and Information Engineering
University of Sydney
Sydney, NSW 2006, Australia

ABSTRACT

In this paper we present a simplified analog VLSI inner hair cell model, which models the main characteristics of the biological inner hair cell, i.e., 1) soft half-wave rectification, 2) low-pass filtering at 1kHz, and 3) adaptation to sustained input. The main challenge lies in the creation of the long time constant associated with the 1kHz low-pass filter and the adaptation. A modified current mirror structure has been used to create a nonlinear low-pass filter which has a cut-off frequency of 1kHz or lower with reasonable component and bias values. Comparison of measurements of the biological inner hair cell and the circuit show a good agreement between the two.

1. INTRODUCTION

In the last decade, several computer models of low-level auditory processing in the brain have been developed (see for instance [1],[2],[3],[4]). As our understanding of the actual processes in the brain increases, our models will become more and more detailed. Furthermore, while early attempts at modeling focused on the peripheral (cochlear) mechanisms of hearing, more recent modeling efforts are attempting to characterize higher levels of auditory processing taking place in the auditory brainstem. A serious problem with this development is that the computer models are becoming more and more computationally intensive and memory demanding. Current trends threaten to take the simulation of these models beyond the range of even the most powerful digital computers.

An alternative is to build auditory models using analogue electronic circuits [5]. Analogues VLSI enables one to create small, but imprecise building blocks, corresponding to, for example, individual neurons [6]. These can then be replicated many thousands of times and put on a single chip. The precision of the building blocks is compensated by the large number of units used, and is therefore not an issue in neural architectures. Over the last few years, we have developed and implemented several such analogue VLSI building blocks that allow us to model parts of the auditory pathway [7],[8]. As the building blocks for these models, we have designed a passive and an active version of a silicon cochlea [9],[10] which both model the mechanical filter function of the basilar membrane. We have also presented a generic silicon spiking neuron [11], which can be used to model the different spiking neurons in the auditory pathway. Finally we have presented a detailed VLSI implementation of an Inner Hair Cell [12],[13], which models the transduction from basilar membrane motion to spiking neuron stimulation performed by the biological Inner Hair Cell. The circuit in [12],[13] implements the Meddis Inner Hair Cell model [14], which is the most widely accepted computational model of the biological Inner Hair Cell. A disadvantage of this model is that it is rather large. In this paper we present a smaller, but less accurate, analog VLSI Inner Hair Cell model. We compare this circuit with its biological original, which we will briefly present in the next section.

2. INNER HAIR CELL FUNCTION

There are two types of hair cells in the human cochlea, the Inner Hair Cells (IHCs) and the Outer Hair Cells (OHCs). Both type of hair cells are embedded in the so called Organ of Corti, which resides on top of the basilar membrane. There are three rows of OHCs and only one row of IHCs. However, since 95% of the afferent connections of the cochlea contact the inner hair cells, we must presume that it is their task to convey information concerning the basilar membrane movement to the central nervous system. The OHCs receive most of the efferent connections and are probably involved in active feedback which controls the gain and selectivity of the cochlear filter.

The organ of Corti is covered by the tectorial membrane, a gelatious and fibrous flab. When the basilar membrane moves, the organ of Corti is displaced. The stereocilia (or hairs) of the outer hair cells, which are attached to the tectorial membrane, are displaced due to the shear between the organ of Corti and the tectorial membrane. The stereocilia of the inner hair cells do not touch the tectorial membrane, but fit loosely into a raised groove known as Hensen's stripe on the under-surface of the tectorial membrane. When the organ of Corti moves with the basilar membrane, forces are exerted on the stereocilia, due to the viscous drag of the endolymph. The displacement of the inner hair cell stereocilia is thus proportional to the velocity of the basilar membrane motion.

The stereocilia of the hair cells pass through rigid upper surface of the organ of Corti, which is called reticular lamina. This reticular lamina forms a boundary between two kinds of fluids. The stereocilia are emerged in endolymph, which is rich in potassium ions, whereas the body of the hair cells are surrounded by perilymph, a fluid poor in potassium ions. The current opinion on the mechano-electrical transduction by the hair cells deals with the ion channels in the tips of the stereocilia, which are permeable to potassium ions, as variable resistances [15]. Potassium ions flow into the cell, driven by the battery of the positive (+80mV) endolymphatic potential and the negative (-45 mV) intracellular potential. Increased potassium flow leads to intracellular depolarization, which causes release of transmitter, and activation of the auditory nerve fibers via the spiral ganglion cells. Because the basal end of the hair cells are in contact with perilymph, which has a low potassium concentration, potassium...
entering the cell at the stereocilia will automatically diffuse out of the cell at the basal end. The hair cells thus modulate the potassium current flow through the reticular lamina. The high potassium concentration of the endolymph is maintained by the potassium pumps of the stria vascularis, which acts as a quiet power supply for the transduction system.

Figure 1. Hair cell response as function of hair bundle deflection. Adapted from [16].

Both types of hair cells have three rows of stereocilia, the stereocilia in the inner row being shortest and those in the outer row being tallest. The tip of a hair is attached by a thin fiber to a taller hair in the next row. The transducer channels are assumed to open and close randomly due to thermal motion, but the amount of time spent in the open state will depend on the elastic force generated by this tip-link. In equilibrium, the ion channels are open about 20% of the time, which implies a small tension on the tip links. If the hair bundle is flexed towards the smallest stereocilia, tension on the tip links is reduced and the channels will spend less time in the open state. Flexion of the hair bundle towards the tallest stereocilia increases tension on the tip links and therefore increase the time the ion channels spend in the open state. Because the relation between the hair bundle deflection and the intracellular voltage change is asymmetric around its resting point, as shown in Fig. 1, the excursions of the intracellular voltages in the positive direction are larger than the excursions in the negative direction. It is therefore possible to divide the intracellular potential changes into an a.c. response at the stimulus frequency and a sustained d.c. depolarization. This can be clearly seen in Fig. 2. Because the cell membrane has a certain capacitance, the a.c. component will decline relative to the d.c. component as the stimulus frequency is raised, i.e., the inner hair cell has a low-pass behavior, with a cut-off frequency of about 1 kHz. The response of the hair cells will therefore be dominated by the d.c. response at stimulus frequencies above a few kHz.

Each inner hair cell synapses with about 20 spiral ganglion cells in humans, and the probability of firing of these spiral ganglion cells is proportional to probability of neuro-transmitter release by the inner hair cell. The probability of neuro-transmitter release depends on the intracellular voltage change and the amount of
neuro-transmitter available. Because the neuro-transmitter is used when the inner hair cell is stimulated, this leads to a reduced response of the spiral ganglion cells to a continuous stimulation of the inner hair cell. Typical post stimulus time histograms of the response of an auditory nerve fiber, i.e., the axon of a spiral ganglion cell, to a high frequency stimulus (3kHz) as measured on a single auditory nerve fiber in a cat is shown in Fig. 3a. This clearly shows the adaptation to the stimulus. Adaptation effects of different origins than the consumption of neuro-transmitter have also been shown in hair cells. However, since an adaptation effect is hardly visible in the intracellular voltage changes shown in Fig. 2, the adaptation seen on the auditory nerve signal must be dominated by the conversion of the intracellular voltage into a neural signal, i.e. by the transmitter release. Fig. 3b. shows the measured response of the circuit which will be discussed in the next section.

3. THE CIRCUIT MODEL

Although the fine details of the operation of the inner hair cell are still not fully understood, we know enough of the input/output relation of the inner hair cell to construct an electrical model. Fig. 1. shows us that the relation between bundle deflection and the percentage of open ion channels has a sigmoid form with a certain offset, so that 20% of the channels are open at equilibrium. This can be easily modeled using a differential pair (see Fig. 4.) for which the output current is a sigmoid function of the input voltage. By using a differential pair with one transistor four times as large as the other, only 20% of the bias current will flow through the smaller transistor when \( V_{in} \) equals zero. The current level, and the input voltage range can be controlled with the bias current of the differential pair. Furthermore, as we can see in Fig. 2., the inner hair cell itself functions as a low-pass filter with a cut-off frequency of about 1kHz. Obtaining such large time constants is one of the main challenges in modeling comparatively slow brain elements with analog VLSI circuits. If the linearity of the filter is not an important issue, then large time constants can be realized using the current mirror shown in the low-pass filter blocks in Fig. 4. This current mirror creates a non-linear low-pass filter for which \( I_{op} \) sets the maximum rise speed and \( I_{down} \) sets the maximum fall speed of the voltage on the capacitor \( C_i \). This construction allows us to obtain programmable time constants in the order of several milliseconds using capacitors in the pico-Farad range while using bias currents in the 100 pA range.

Finally, Fig. 3a. shows that the conversion of the intracellular voltage into neuro-transmitter release shows adaptation to the stimulus. Fig. 3a. actually shows the spike rate of a single auditory nerve fiber, but since the spiking probability of a spiral ganglion cell is directly proportional to the amount of neurotransmitter released by the inner hair cell it contacts, we can use this figure to model neuro-transmitter release. A form of adaptation similar to the one shown in Fig. 3a. can be obtained by taking the weighted difference between two low-pass filtered versions of the signal, with the second low pass filter having a longer time constant than the first. This longer time constant is obtained by making \( C_2 \) four times larger than \( C_1 \). If postfabrication independent control of the two time constants is needed, a separate \( I_{op} \) and \( I_{down} \) can be used for the second mirror.

The final circuit, modeling the input/output relation of the inner hair cell is given in Fig. 4. The actual output current will be created with an additional current mirror, not shown in this figure for clarity, yielding \( I_{out} = I_e - A I_1 \), where \( A \) is the gain of the mirror, and controls the ratio between the peak response and the sustained response of the circuit.

4. MEASUREMENTS

The circuit of Figure 4 was implemented in a 1µm technology. The cell size was 60x130µm including the two capacitors. With this circuit we have tried to reproduce both the relation between basilar membrane motion to intracellular voltage in the inner hair cell, and the relation between this intracellular voltage and the neuro-transmitter release. Fig. 5. and 6. show the measurements of the current \( I_e \), which is the output of the first low-pass filter, and which is the equivalent of the intracellular voltage in the real inner hair cell. Comparison of Fig. 5. and 6. with Fig. 1. and 2. shows the similarity that can be obtained between the response of the circuit and the IHC by choosing the correct bias values. The difference in the onset and offset of the response between Fig. 2. and Fig. 6., is due to the difference in the stimulus tone. In our measurements we used a tone burst with a square envelope, whereas Palmer and Russel [17] used a stimulus with a gentle onset and offset. Finally, comparison of Fig. 3a. with Fig. 3b. shows that by subtracting a weighted version of the current \( I_2 \) from \( I_1 \), a temporal adaptation can be obtained similar to the one seen on the auditory nerve.

5. CONCLUSIONS

In this article we have presented a simple analog VLSI circuit which captures both the transduction from basilar membrane motion to intracellular voltage and from intracellular voltage to neuro-transmitter release of the biological inner hair cells. Forfeiting linearity of the low-pass filter, a modified current mirror structure can be applied which uses reasonable component and bias values to obtain time constants in the 10ms range. Comparison between the measurements of the circuit and the biological inner hair cells show that the circuit approximates the output of the biological original well.
Figure 5. Measured output current as function of input voltage from the IHC circuit.

Figure 6. Measured $I_1$ changes from the IHC circuit for different frequencies of stimulation. Note the change of scale for the lower five traces.

6. REFERENCES


