

An Analogue Electronic Model of Ventral Cochlear Nucleus Neurons

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Abstract

This paper proposes a simple analogue electronic spiking neuron circuit, which can be used to create hardware models of biological neural systems. In spite of its simplicity, the circuit is able to simulate a variety of different neuron types. Measurements of the neuron model in various settings are compared with the physiological response of certain neuron types in the Ventral Cochlear Nucleus, i.e., part of the first relay station in the brainstem of the auditory pathway. A good analogy between the response of the circuit and the different neurons has been obtained.

1: Introduction

In recent years, several computer models of low-level auditory processing in the brain have been developed [1], [2], [3], [4], [5], [6], [7]. As our understanding of the actual processes in the brain increases, our models will become more and more detailed. Furthermore, while early attempts at modelling focused on the peripheral (cochlear) mechanisms of hearing, more recent modelling efforts are attempting to characterise 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. Analogue VLSI enables one to create small, but imprecise building blocks [8], corresponding to, for example, individual neurons. These can then be replicated many thousands of times and put on a single chip. The imprecision of the building blocks is compensated by the large number of units used, and is therefore not an issue in neural architectures.

One advantage of conserving the architecture of neural

wetware in VLSI implementations, is that all processing happens in parallel and in continuous time. Since there is no time-multiplexing in order to simulate multiple neurons, the circuits can function in real time. One chip models only a small number of neurons, but multiple chips can be used in parallel, so there is no obvious limit to the number of neurons in an analogue VLSI model.

There are two main reasons for building hardware models of neural processing in the brain. Firstly, a faithful neural model will allow the study of neural processing in the brain without the need for living neural tissue and without the need for simultaneously probing hundreds or thousands of neurons in the brain. Once we have neuron models with the same transfer function as biological neurons, and knowledge of their input and output connections, we can build a hardware model that allows us to study the collective behaviour of such a neural network and the individual behaviour of the single neurons in the network. Secondly, we can hope that by using similar processing strategies as the brain, it will be possible to perform perceptive tasks with the hardware model hereby approaching the excellent performance of the brain on such tasks. In this case, the neuron model might not need to replicate the biological neuron as faithful as in the first case. However, since the actual amount of detail that has to be included in order for the model to function is unknown, the only safe way to build such a model is to reproduce the biological neuron as faithful as possible. Once a functional model is built it will allow us to distil the amount of detail needed for a certain function and thus to create a simpler model. Because of natural evolution it is very well possible though that most of the details of the processing performed by a biological neuron are important.

In either case it is not only a faithful neuron model which will allow us to build hardware implementations of neural processing. The total amount of neurons that we can use in the model will be as important. This thus leads to a trade-off between the size of the single neuron circuit and the amount of detail incorporated in it.

In this paper, we will present an analogue electronic spiking neuron model that is simple enough to be small, yet allows us to reproduce spiking behaviour of several different types of neurons. In particular, we will compare the measured responses of the actual neuron circuit with the neurophysiological responses of the main neuron types in the AnteroVentral and PosteroVentral Cochlear Nucleus (see figure 1), which are part of the first site of innervation of the Auditory Nerve.

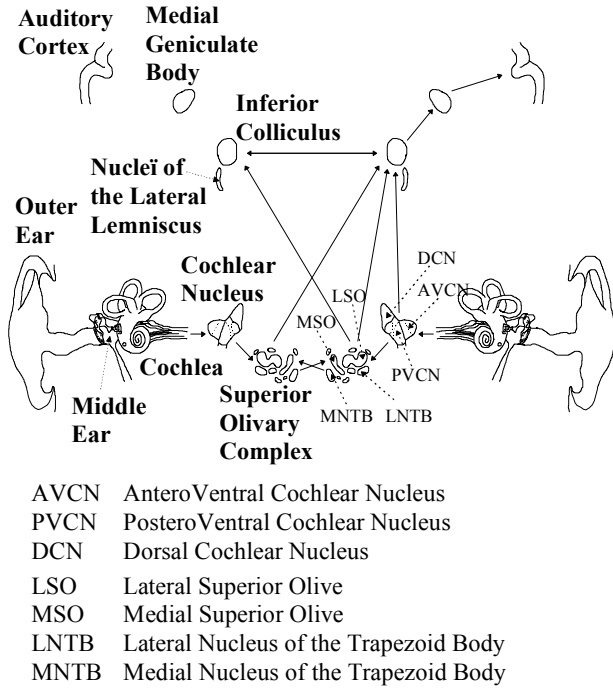


Figure 1. Simplified diagram of the auditory pathway. The major stages are named in boldface, the subdivisions of the CN and SOC in small capitals.

2: The neuron model

The proposed neuron model is shown in figure 2. The membrane of a biological neuron is modeled by a capacitance, C_{mem} , and the membrane leakage current is controlled by the gate voltage, V_{leak} , of an NMOS transistor. In the absence of any input ($I_{ex}=0$), the membrane voltage will be drawn to its resting potential (controlled by V_{rest}), by this leakage current. Excitatory inputs simply add charge to the membrane capacitance, whereas inhibitory inputs are simply modeled by a negative I_{ex} . If an excitatory current larger than the leakage current of the membrane is injected, the membrane potential will increase from its resting potential. This membrane potential, V_{mem} , is compared with a controllable threshold voltage V_{thres} , using a basic transconductance amplifier driving a high impedance load.

If V_{mem} exceeds V_{thres} , an action potential will be generated.

The generation of the action potential happens in a similar way as in the biological neuron, where an increased sodium conductance creates the upswing of the spike, and a delayed increase of the potassium conductance creates the downswing. In the circuit this is modelled as follows. If V_{mem} rises above V_{thres} , the output voltage of the comparator will rise to the positive power supply. The output of the following inverter will thus go low, thereby allowing the "sodium current" I_{Na} to pull up the membrane potential. At the same time however, a second inverter will allow the capacitance C_K to be charged at a speed which can be controlled by the current I_{Kup} . As soon as the voltage on C_K is high enough to allow conduction of the NMOS to which it is connected, the "potassium current" I_K will be able to discharge the membrane capacitance.

A third inverter, with variable threshold and V_{mem} as input, (not shown in figure 2 for clarity) generates the actual spike. Its output is high when V_{mem} is lower than the inverter's threshold, but will go low when an action potential is generated.

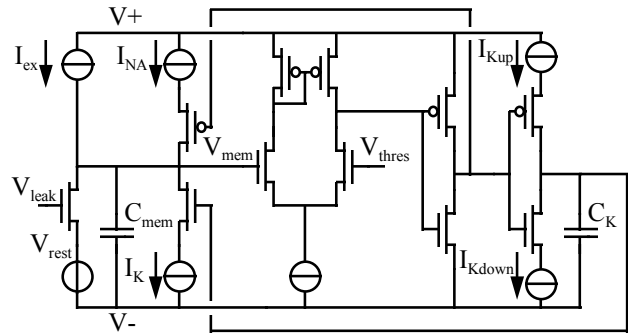


Figure 2. The proposed electronic neuron circuit.

Two different potassium channel time constants govern the opening and closing of the potassium channels. The current I_{Kup} which charges C_K will control the spike width, since the delay between the opening of the sodium channels and the opening of the potassium channels is inversely proportional to I_{Kup} . If V_{mem} now drops below V_{thres} , the output of the first inverter will become high, cutting off the current I_{Na} . Furthermore, the second inverter will then allow C_K to be discharged by the current I_{Kdown} . If I_{Kdown} is small, the voltage on C_K will decrease only slowly, and, as long as this voltage stays high enough to allow I_K to discharge the membrane, it will be impossible to stimulate the neuron if I_{ex} is smaller than I_K . Therefore I_{Kdown} can be said to control the 'refractory period' of the neuron.

32 neurons as shown in figure 2 have been realised,

together with some circuitry to facilitate communication of signals on- and off-chip on a 1mmx2.5mm die, using the ECPD10 (1 μ m) technology of ES2. All transistors are 10 μ m/10 μ m except for the switches and inverters, which are 2 μ m/10 μ m, and C_K and C_{mem} are 10pF.

3: Measurements

In this section we will compare the response of the chip in different settings with the response of Ventral Cochlear Nucleus (VCN) neurons to tone bursts. VCN neurons are often characterised by their Post Stimulus Time Histogram (PSTH) [9], which is a histogram of the neuron's output spikes as result of repeated presentations of a pure tone at a frequency that best stimulates the cell. The chip's PSTHs are created from the summed response of the 32 neuron circuits to 20 presentations of a 5kHz tone burst to the chip. If summation over multiple neurons is considered equivalent to summation over multiple presentations, this can be thought of as the result of 640 stimulations. Statistically however both types of summation are not equivalent, although they both introduce variation in the neuron's response through device mismatch and noise respectively. Alternatively, we can think of both the neurophysiological and electrical PSTH as the result of repeated stimulations of a group of neurons. The neurophysiological PSTH is always measured on a single neuron, but the summed PSTH of a group of single neurons will have the same appearance as the PSTH of a single neuron in this group, if the neurons are almost identical.

Since the pure tone is not directly presented to the cell, but to the Outer Ear, the actual input signal to the VCN neurons is not a pure tone, but the Auditory Nerve signal, which is the result of the processing of the ear. This input signal therefore needs to be created first in order to simulate the VCN neurons.

3.1: The input

For experiments with a single pure tone at a fixed amplitude, the processing relevant for the neurons which are best stimulated by this frequency consists mainly of half-wave rectification and a temporal adaptation to the tonal stimulus as performed by the Inner Hair Cells (IHC) in the Cochlea. This signal can be created using the IHC circuit shown in figure 3.

The input signal, which is a sinusoid with DC offset equal to V_{ref} is half-wave rectified and a weighted low-pass filtered copy of this rectified signal is subtracted from this signal, creating the temporal adaptation. In the biological cochlea, the output signal of the IHC is representative of the firing rate of the Spiral Ganglion cells

innervated by this IHC. The axons of these Spiral Ganglion cells constitute the Auditory Nerve, and are thus the inputs to the Cochlear Nucleus neurons.

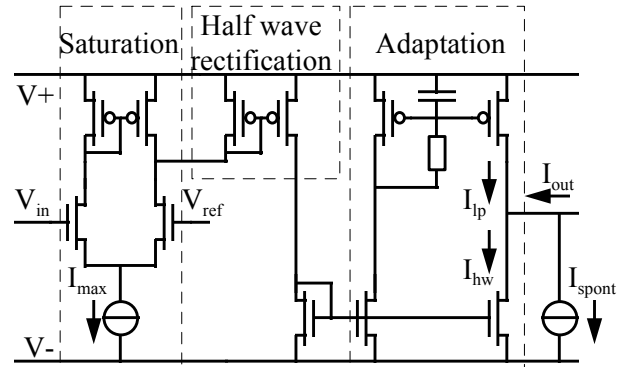


Figure 3. The Inner Hair Cell circuit.

The output current of our IHC circuit is not first converted into spikes, but is directly connected to the neuron chip. On the neuron chip, this current is mirrored, to be compatible with the sign of the excitatory input current shown in Figure 2. The spontaneous rate of the biological Auditory Nerve fibres can be simulated by adding a DC current (I_{spont}) to the IHC output current. The output of the IHC circuit when stimulated with a 1kHz, 100mVpp sinusoid for 25ms is shown in Figure 4.

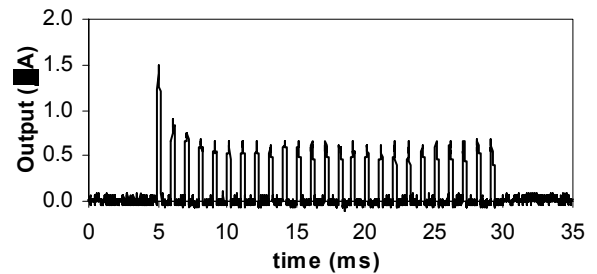


Figure 4. Measured output current of the IHC circuit.

The fact that the IHC circuit output is not first converted into spikes, but directly presented to the VCN neurons is a clear deviation from the biological auditory pathway. However, as it can be seen in the following comparisons of the PSTHs of the VCN neurons and the neuron circuit, this difference does not seem to be important for replication of the neurophysiological PSTHs. This suggests that on the Auditory Nerve the spikes are only used for communication, since they are easier to transmit over a certain distance than analogue voltages in the biological nervous system. However, there might be certain types of processing, as for instance correlation of two signals, which are more easily performed when spikes are used to carry the information. A second difference is the fact that the IHC circuit performs a form of half-wave rectification

up to frequencies well above the auditory frequencies. This means that the output current of the circuit is always zero for one half of the input waveform, over the complete auditory frequency range. In the biological auditory system this is only true up to frequencies of about 1kHz. Since the measurements will be performed at a frequency of 5kHz, there will be another difference between the input signal of the biological VCN neurons and the neuron circuit. The effect of this is that the neuron circuit will only generate spikes during one half of the input waveform, since the membrane potential will not rise if the input signal is zero, whereas the VCN neurons generate spikes independent of the input waveform's phase. This is not visible in the following PSTHs due to the limited time resolution of these images, i.e., in the PSTHs of the neuron circuit there are 'invisible' zeros between the printed values. However, this difference is not a flaw in the circuit model of the VCN neurons, but a flaw in the current IHC model.

3.2: The output

In this paragraph the PSTHs of the major cells found in the VCN of the cat are compared with the PSTHs of the neuron chip in different settings. We will briefly discuss each neuron type. Most neurophysiological and anatomical information is taken from [10] and [11].

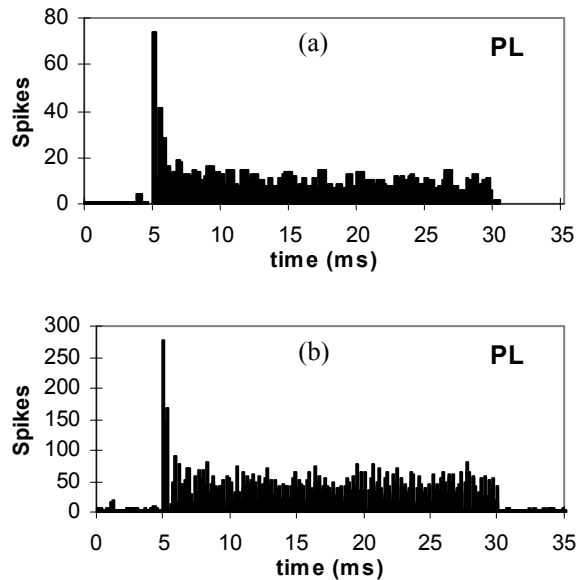


Figure 5. Primarylike PSTH.
(a) Physiological response measured in the VCN of the Cat. (Based on data from [9]).
(b) Measured response of the chip.

The Primarylike PSTH is the typical response of a

spherical bushy cell, mainly found in the AnteroVentral Cochlear Nucleus. This cell receives input from only one Auditory Nerve fibre through a large synaptic contact on its soma, called the endbulb of Held. This cell outputs one spike for every input spike, so it comes as no surprise that its PSTH is very similar to the PSTH of an Auditory Nerve fibre. The Primarylike cell is thus the least interesting to replicate, but is also traditionally the first VCN neuron presented in neurophysiological textbooks. In keeping with this custom it is also the first one to be presented in this paper. It is however not only included for completeness, it also shows that we can use this circuit to convert the output of the IHC circuit into spikes, if Auditory Nerve spikes are needed for a certain type of processing.

The spherical bushy cells do receive inhibitory inputs on their dendrites, which might be implicated in some form of spectral sharpening. The Primarylike cells project almost exclusively to the Lateral Superior Olive (LSO) and the Medial Superior Olive (MSO), which are both part of the Superior Olivary Complex (SOC). The LSO and MSO are the brain nuclei that underlie the localisation of sound based on interaural intensity difference and time difference respectively.

The chip's response is obtained by giving the neurons a medium level membrane leakage current (I_{leak}), to avoid long term integration of the input signal, and a short refractory period, so that the neurons are ready to fire, when the next input arrives.

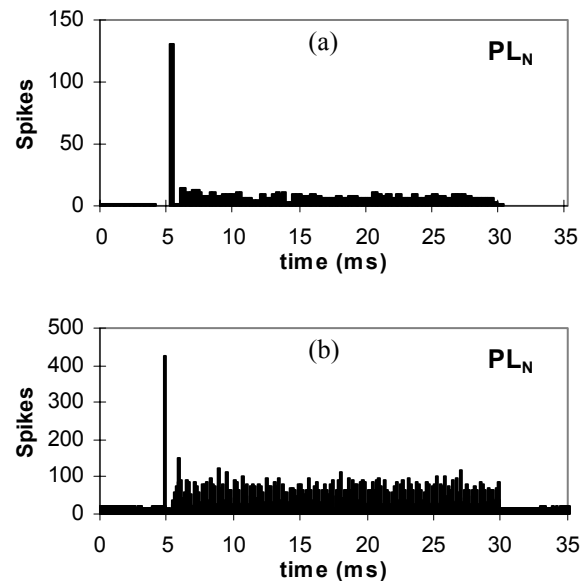


Figure 6. Primarylike with Notch PSTH.
(a) Physiological response measured in the VCN of the Cat. (Based on data from [9]).
(b) Measured response of the chip.

The Primarylike with Notch PSTH (figure 6) is very similar to the Primarylike PSTH and is the typical response of a globular bushy cell, which is also mainly found in the AnteroVentral Cochlear Nucleus. The globular bushy cell receives synaptic contacts from a few Auditory Nerve fibres that originate in the same region of the Cochlea. Therefore, its PSTH is still similar to the PSTH of an Auditory Nerve fibre, but the probability of the cell firing at the onset of the stimulus is almost one, because at least some of its input fibres will carry a spike at onset, due to the higher Auditory Nerve spiking probability at the onset of the stimulus (see figure 4). If the cell always spikes at the onset of the stimulus, it will always be in its refractory period just after it spikes. This creates the notch in the PSTH. The globular bushy cell thus enhances the onset of a stimulus. Furthermore, the cell typically needs a few simultaneous Auditory Nerve spikes in order to generate an output spike. This means that the cell only responds to correlated action potentials of several fibres, thereby suppressing the random activity present on the Auditory Nerve. The globular bushy cell therefore can be said to improve the signal to noise ratio.

The globular bushy cells project to neurons in the Medial Nucleus of the Trapezoid Body, which in turn project with inhibitory connections to neurons in the contralateral Lateral Superior Olive (LSO). These LSO neurons also receive ipsilateral contacts from the spherical bushy cells. This neural circuit is probably the substrate for auditory localisation based on interaural intensity differences. The globular bushy cells also project to the Lateral Nucleus of the Trapezoid Body (LNTB) and to other PeriOlivary nuclei (PO). Both the LNTB and several other PO nuclei have efferent projections to the Cochlear Nucleus and the Cochlea itself. Therefore, they are thought to be part of one or more feedback loops that modify the gain of the Cochlea and that inhibit several neurons in the Cochlear Nucleus. Several of these PeriOlivary nuclei also have reciprocal connections with other nuclei in the Superior Olivary Complex and also with the Inferior Colliculus (IC), which is the next stage in the auditory pathway towards the auditory cortex. The PeriOlivary nuclei thus seem to regulate the operating point of many different neurons in the auditory pathway.

The Primarylike with Notch response is obtained from the chip by using a relatively high I_{leak} , to avoid long term integration of the input signal, a threshold voltage that assures that the neuron will always fire at the onset of the stimulus, and a refractory period that matches the duration of the notch.

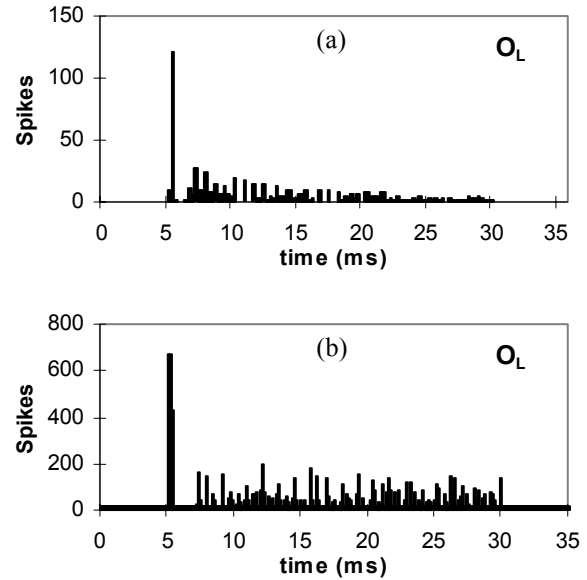


Figure 7. Onset Locker PSTH.
(a) Physiological response measured in the VCN of the Cat. (Based on data from [9]).
(b) Measured response of the chip.

The Onset Locker PSTH is similar to the Primarylike with Notch PSTH, but with a lower and less constant sustained rate. This PSTH is the typical response of the large octopus cells found in the PosteroVentral Cochlear Nucleus (PVCN). This response is obtained by the same high probability of spiking at the onset, which creates the onset peak and the notch in the PSTH. Having a higher leakage current than the globular bushy cell, and more synaptic inputs, it needs even more synchronous action potentials in order to generate a spike. Therefore, its signal to noise ratio will be even higher than the signal to noise ratio of the Primarylike with Notch response.

The Onset Inhibitory PSTH (figure 8) is a less common response of the octopus cells. It only shows an initial peak and hardly any activity after. This is probably due to an even higher threshold, so that only at the onset of the stimulus, the input is large enough to invoke firing. Alternatively, the cell might receive direct excitatory contacts from the AN and delayed inhibitory contacts. This inhibition however has never been shown in physiological experiments. Some cells have been measured that stay depolarised after the first spike, i.e., once the cell has fired, its membrane potential stays above the spiking threshold and the cell can not generate another spike as long as the input activity persists.

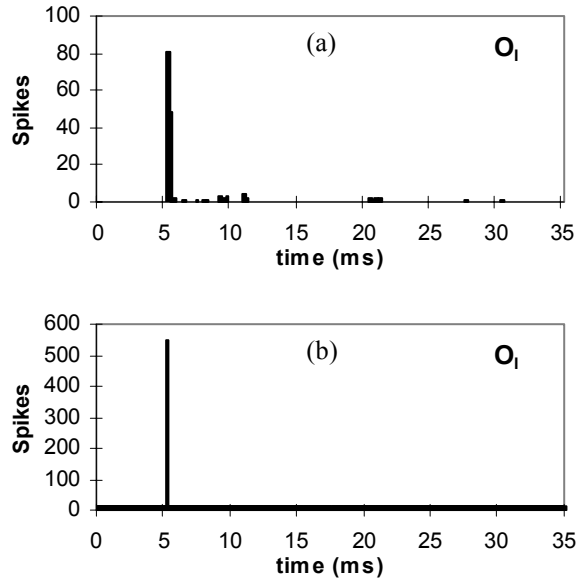


Figure 8. Onset Inhibitory PSTH.
(a) Physiological response measured in the VCN of the Cat. (Based on data from [9]).
(b) Measured response of the chip.

The onset spike of the octopus cells have a very short latency, i.e., the cell reacts very fast to the start of the stimulation, sometimes within $100\mu\text{s}$. The octopus cells project to some of the PeriOlivary nuclei, which in turn inhibit the Outer Hair Cells (OHC) in the Cochlea and also inhibit other Cochlear Nucleus neurons. They are therefore probably implicated in some fast feedback loops. They also project to the Ventral Nucleus of the Lateral Lemniscus (VNLL) (see figure 1), which is known to be part of the neural circuit that mediates the fast acoustic startle response. Some neurons of the VNLL also have inhibitory projections to the Inferior Colliculus. The octopus cells might thus provide fast inhibition within the Inferior Colliculus. In general, the octopus cells are probably part of the neural circuitry implicated in the detection of sound and the initiation of some reflexes.

The Onset Locker and Onset Inhibitory responses are obtained from the chip by progressively increasing the leakage current from the setting that produces the Primarylike with Notch PSTH. Alternatively, the Onset Inhibitory response might be obtained by having a I_K only slightly larger than I_{NA} , so that the cell can not reset as long as there is an excitatory input current.

The Onset Chopper PSTH (figure 9) is the response of a different neuron, the large, multipolar, stellate cell, which is mainly found in the PosteroVentral Cochlear Nucleus, but also relatively frequent in the AnteroVentral Cochlear Nucleus. This type of stellate cell has its soma largely

covered by synaptic contacts, and also has a very short onset latency. A spike arriving at a synapse on the cell soma creates a relatively large membrane voltage variation. The cell also receives many synapses from Auditory Nerve fibres, and has a high threshold of firing, but has a smaller leakage current than the other two onset cells. This neuron is also assumed to produce the Transient Chopper response, which is like the Sustained Chopper response (figure 10), but with a less regular form of chopping. This would also be caused by the many somatic contacts of the cell.

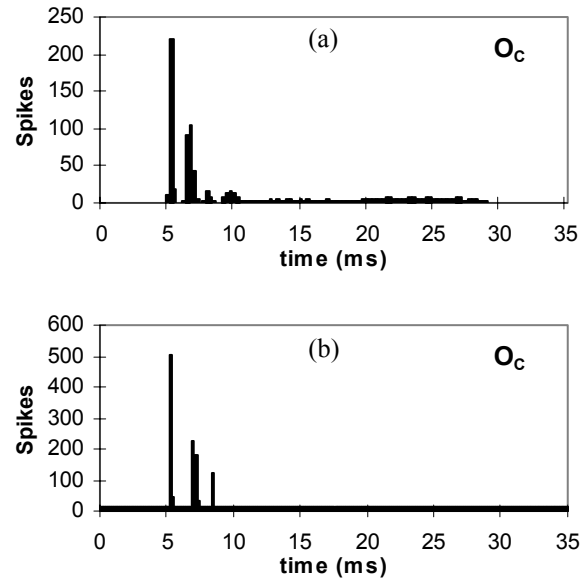


Figure 9. Onset Chopper PSTH.
(a) Physiological response measured in the VCN of the Cat. (Based on data from [9]).
(b) Measured response of the chip.

Chopping is the standard behaviour of a spiking neuron that completely resets after spiking. After an action potential is generated, such a cell cannot be activated during its refractory period. Once its refractory period is over, the cell starts to integrate the input spikes over a certain time, controlled by the leakage current of this leaky integrator. This yields a fairly constant firing rate of such a cell as response to an input signal with a constant mean spike rate, thus generating the typical chopping behaviour. If the sustained input level (from the Auditory Nerve) is too low to reach the firing threshold, the cell will only chop at the stimulus onset.

This type of stellate cell projects directly to the Inferior Colliculus, and is therefore part of the shortest and fastest pathway from the auditory periphery to the forebrain. It also projects to several other neurons in the Cochlear Nucleus and to the Superior Olivary Complex and the Lateral Lemniscus. Because of its very large dynamic

range — the output of the cell saturates only after an 80dB intensity increase of the input signal from the spiking threshold — and the fact that its chopping rate increases with the intensity of the input signal, this neuron is thought to be part of the neural substrate that codes the intensity of sound. The output of this neuron is inhibitory however, so it can only serve to inhibit other neurons to keep them working at an operating point which is within their dynamic range.

This behaviour is obtained from the chip by having a relatively low leakage current, and a high threshold voltage. Furthermore, an input signal with a high onset to sustained ratio is used, corresponding to the somatic contacts which perform less low-pass filtering than the dendritic contacts of the Sustained Chopper cells.

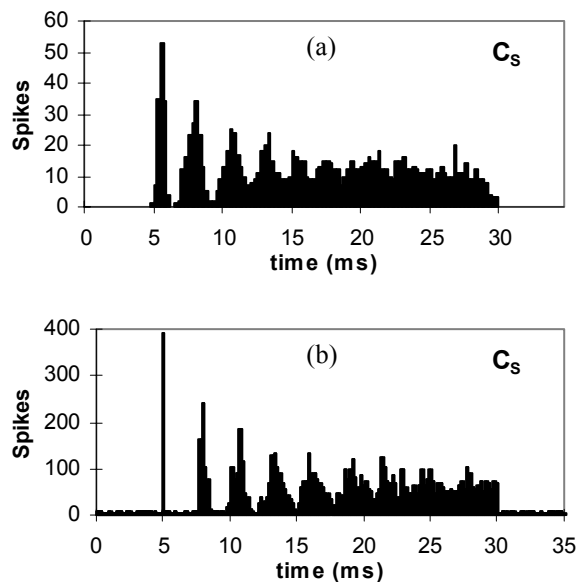


Figure 10. Sustained Chopper PSTH.
(a) Physiological response measured in the VCN of the Cat. (Based on data from [9]).
(b) Measured response of the chip.

The Sustained Chopper PSTH is the typical response of the other type of stellate cell. This stellate cell hardly has any synapses on its soma and thus receives most of its inputs on its dendrites. Because the dendrites low-pass filter the incoming spikes, the membrane potential will rise only smoothly. This explains the regularity of the Sustained Chopper cell. Regularity is however destroyed due to the integration of noise over time, as can be seen in the last 10ms of the PSTH in figure 10.

The Sustained Chopper cells have a dynamic range which is much smaller than that of the Onset Chopper cells, up to 30dB. They do receive inhibitory connections from the Onset Chopper cells, which might be used to keep the cell's operating point within its dynamic range. The

Sustained Chopper cell has excitatory projections to the Inferior Colliculus and also to several neurons in the Dorsal Cochlear Nucleus. Together with the Onset Chopper cells they might be used to code intensity. Furthermore, they lock to the modulation frequency of amplitude modulated sound if the modulation frequency is well below 1kHz. Therefore, they are probably used in the extraction of the AM frequency and of the pitch of a speech signal [3], [5]. Several Sustained Chopper cells with the same best modulation frequency project to the same coincidence detecting neuron in the Inferior Colliculus, which is thus only activated if a certain modulation frequency is present in the input signal.

4: Conclusions

Although the proposed neuron model is very simplistic, this neuron model already allows us to simulate different spiking behaviours, characteristic of different neuron types, by changing its biases. As shown in the previous section, Post Stimulus Time Histograms closely resembling the PSTHs of Ventral Cochlear Nucleus neurons can be obtained with the circuit. An advantage of the analogue VLSI implementation is that one can change the bias voltages and see the neuron model react in real time. This simplifies largely the task of determining the different settings to model the different neurons. Because of its simplicity, the neuron model is small and hundreds of neurons can be put on a reasonable size chip.

5: Future work

Measuring the PSTH of a neuron is not the only way to characterise a neuron and certainly doesn't characterise the neuron completely. One would have to measure spike rate versus frequency, spike rate versus intensity, with and without masking by another tone or noise, and one would have to generate the interspike interval histogram. The latter can be determined from the current set-up if we measure only a single neuron of the 32 on the chip. The measurement of the rate-intensity curve needs a model of the AN input which has the right output activity for a given input tone intensity. This needs a more detailed study of the current IHC model. For the rate-frequency curve measurement, the IHC model needs to be connected to our electronic model of the cochlea [12], and the proper IHCs need to be connected to the neuron chip to model its frequency response area.

The neuron chip can be used as a building block, allowing the implementation of neural architectures found in the auditory pathway. This makes it possible to study these architectures and determine their utility as auditory signal processors.

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