A One-dimensional Frequency Map Implemented using a Network of Integrate-and-fire Neurons

Leslie S. Smith
Department of Computing Science and Mathematics, University of Stirling
Stirling FK9 4LA, Scotland. email: lss@cs.stir.ac.uk

Abstract

A network of integrate-and-fire units (consisting of five excitatory units and one inhibitory unit) is shown to implement a one dimensional frequency map over one octave (80 - 160Hz). The network has a biologically plausible structure, conforming to Dale's law, and using plausible synaptic and axonic timings.

1 Aims

The aim of this work is to demonstrate a one dimensional frequency map (i.e. a network in which an ordered sequence of neurons respond to different frequencies of input) using a biologically plausible network of integrate-and-fire neurons. Biological plausibility here means obeying Dale's law, using EPSP and IPSP timings which are plausible, and using inter-neuron delays of the correct order of magnitude.

2 Background

This work was motivated by a problem in audition. For an auditory stimulus which consists of many harmonics of a (low) fundamental frequency, the resultant auditory nerve signal from those parts of the cochlea which cannot resolve each harmonic is amplitude modulated at the fundamental frequency [6]. Amplitude modulation is amplified by stellate cells in the cochlear nucleus (choppers) [9], and there are cells in the inferior colliculus which form a map ordered by amplitude modulation frequency [11]. We sought a neural network solution to the problem of forming a map ordered by frequency. There are a wide variety of different neurons in the early auditory system [10], and one option would have been to model a system of these, based directly on the neurophysiology [3, 13]. We decided instead to attempt to produce a frequency-sensitive system using the simplest form of neuron which is sensitive to time-varying signals at all, namely the integrate-and-fire neuron [7], exploiting their parameters and interconnection to produce differential frequency selectivity.

Integrate-and-fire units have been used for grouping signals [2, 8], and for clustering [12]. Using them in a frequency mapping system emphasises their importance as a useful abstraction of real neurons. The work reported here builds on earlier work by the author's student [1] (in which pulsatile input was used, and the excitatory units directly inhibited other excitatory units).

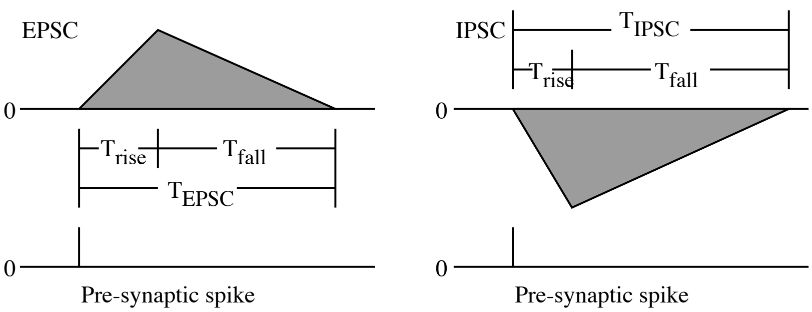

Figure 1: Excitatory and inhibitory post-synaptic currents for a single pre-synaptic spike. For a weight of 1 (-1 in the inhibitory case) the total charge transferred (over T_{EPSC} (T_{IPSC})) is 1 (-1). $T_{\text{EPSC}} = 3.7$ms, and $T_{\text{IPSC}} = 3.3$ms, with $T_{\text{rise}} < T_{\text{fall}}$ in both cases.

3 Methods

The experiments were carried out on a simulator for networks of integrate-and-fire neurons developed by the author and Chumbo [1] which runs under the NeXTStep operating system.

3.1 The neurons

All the neurons are integrate-and-fire units, characterised by their voltage-like state variable, V, between spikes:

$$\frac{dV}{dt} = -\alpha V + I(t)$$

where α is the dissipation. If $\alpha = 0$, this is simple integration, and, for $\alpha > 0$ leaky integration. When V hits some threshold (θ, initially 1 in this case), the unit fires: that is, it emits a spike. There then follows a refractory period, during which $\theta = \infty$, at the end of which V is reset to 0. This is followed by a relative refractory period, which starts with $\theta = 10$, followed by a quadratic (parabolic) decay back to the original threshold.

Input to the neurons takes two forms. External input to the network is considered to be like injected current. From an auditory perspective (i.e. where the "real" input was an amplitude modulated signal), this input would be made up from many auditory nerve fibers or outputs from primary cells in the cochlear nucleus. These will have arrived nearly simultaneously at a number of synaptic sites, resulting in (i) half-wave rectification of the amplitude modulated signal and (ii) smoothing of high frequency content of the signal [4].

Input from other neurons is modelled using simple triangular EPSPs and IPSPs as shown in figure 1. This is a simplification of real EPSP and IPSP shapes, similar to that discussed in [5].

3.2 The network

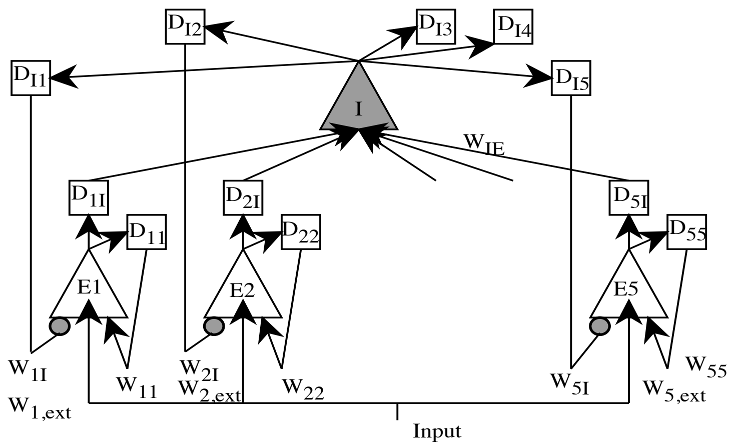

Figure 2: The network used. Only 3 of the 5 excitatory neurons are shown. Neurons Ei are excitatory, and neuron I is inhibitory. The weights (W_{ij}) and delays (D_{ij}) are discussed in the text.

The network consists of five excitatory neurons each with a recurrent excitatory connection, all exciting an inhibitory interneuron, which in turn inhibits all the excitatory neurons (see figure 2). Each has either all excitatory or all inhibitory outputs, so that the network satisfies Dale's law. There is a delay associated with each connection between neurons: this delay is crucial to network design.

3.3 Design of the frequency mapping network

We set the dissipation of each excitatory neuron so that it would be maximally sensitive to half-wave rectified input at its "best" frequency. For this to occur, the activation should peak near the end of each positive half-cycle. We chose the dissipation to be $\frac{1}{T_{\mathrm{half-cycle}}} = 2f_{\mathrm{best}}$, where f_{best} is the desired "best" frequency, then adjusted the strength of the input to each neuron ($W_{i,\mathrm{ext}}$) so that it just fired in response to input of strength 1 at its best frequency.

The basic ideas for the weights and delays in the filter were taken from digital filtering. There, bandpass filters are produced using multiple weighted delayed signals which are fed back to the input of an amplifier. However, using a neuron whose output was fed back through many synapses, each with different delays, some with positive and some with negative weights is not biologically plausible. Instead, we used one excitatory recurrent connection, with a total delay (including the EPSC delay) of one period of the "best" frequency. We set the refractory period (RP) on each of the excitatory neurons to 2ms, and the relative refractory period (RRP) to a value such that $\mathrm{RP} + \mathrm{RRP} = \frac{2}{f_{\mathrm{best}}}$. The idea was that the excitatory feedback should be needed in order to make the neuron fire for a second time. The strength of the recurrent excitatory feedback (W_{ii}) was then set so that input at f_{best} resulted is a stream of spikes. This results in each excitatory neuron being a low-pass filter (LPF), in the sense that it fires once per cycle for $f \leq f_{\mathrm{best}}$, and less often for $f > f_{\mathrm{best}}$.

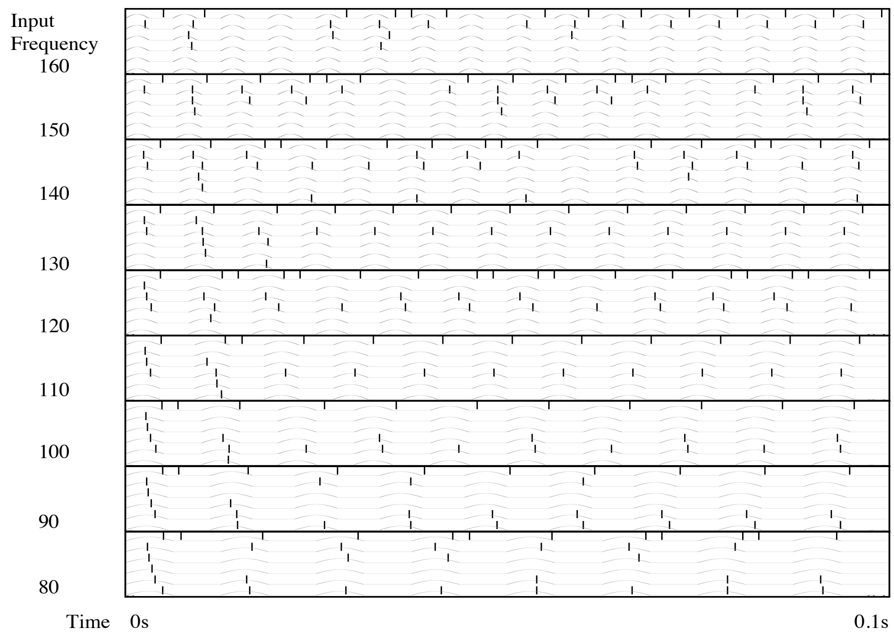

Figure 3: Network response to half-wave rectified signals from 80 to 160Hz. Grey lines are input, black vertical lines are spike outputs. In each small rectangle, the bottom 5 lines are the input and output from each of the excitatory neurons (lowest frequency at bottom, highest at top), and the top line is the inhibitory interneuron.

Turning the LPF characteristic of each neuron into a frequency map requires inhibition. To provide this without sacrificing biological plausibility, we used an inhibitory interneuron. This integrate-and-fire neuron is excited by each excitatory neuron through a fast synapse with short delay. All the weights to this neuron (W_{IE}) and all the delays (D_{iI}) are identical. The weights were set so that any single spike from an excitatory neuron will cause the inhibitory interneuron to fire. The RP and RRP were set to small values (1ms each), and the dissipation to 200 (altering this seems to have little effect). The delays from the inhibitory neuron back to the excitatory neurons (D_{Ii}) were set so that the round-trip delay from output of the excitatory neuron back to inhibition of the excitatory neuron was $1.5 * \frac{1}{f_{\mathrm{best}}}$. Inhibition occurs when the excitatory neuron should not be firing if it is responding to its f_{best}. The inhibitory weight to the excitatory neuron, (W_{iI}) was set to the same value for all the neurons.

4 Results

A network with 5 excitatory neurons, and one inhibitory neuron was designed, and set up for f_{best} of 80Hz, 100Hz, 120Hz, 140Hz, and 160Hz. It was tested with input (of strength 1) at frequencies between 80 and 160 Hz, in intervals of

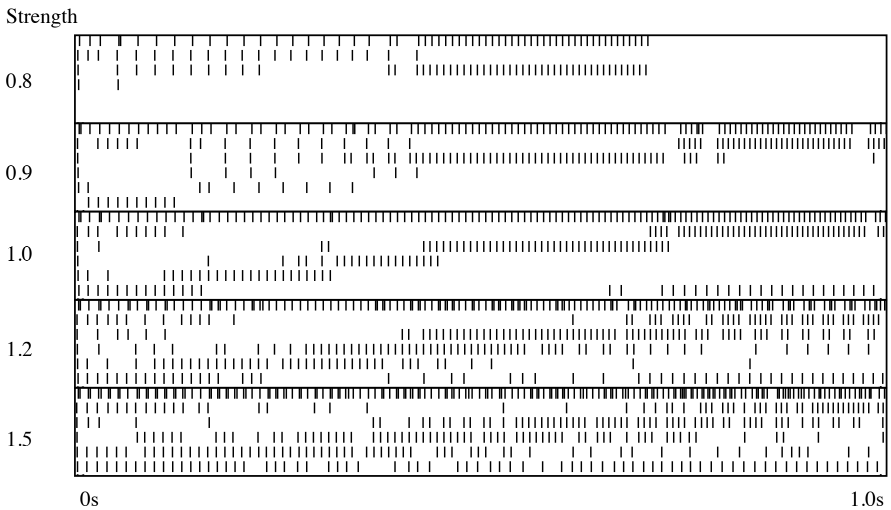

Figure 4: Network response to chirp input (see text) at different strengths. Top line is inhibitory interneuron.

10Hz, The results are shown in figure 3. Initially, each neuron works as a LPF, but then the inhibitory interneuron takes effect, and the network settles down with the appropriate neurons responding most strongly. The exception to this occurs at 80Hz, where the highest frequency neuron also responds.

The network was tested with a "chirp" input (a half-wave rectified sine wave, whose frequency varied linearly from 80Hz to 160Hz over 1 second). This was applied at varying strengths, and the results are shown in figure 4. When the signal has strength 0.8, it is not strong enough to make the network behave correctly. At higher strengths, the appropriate neurons fire. As the input strength is increased, more excitatory neurons fire, although the inhibitory neuron also fires more often. The response is centred on the correct neuron, although there is additional noise as the strength increases. The exception is near 160Hz, where the 80Hz unit also fires, though only on every second pulse.

5 Discussion

We have provided constructive proof that one can build a frequency mapping network using only integrate-and-fire neurons. Real neurons are more complex, and it may be that the neurons of the early auditory system have structures which make this task easier: however, we have demonstrated a biologically plausible neural circuit for producing a frequency map similar to that in the inferior colliculus. The network is designed, not adapted: it is not clear how one would produce an adaptive network to perform this task. We have not experimented with scaling this network up: we suspect that larger clusters of excitatory neurons controlled by one inhibitory neuron would be less stable.

References

[1] P.S.C. Chumbo. Integrate and fire neural network simulator and bandpass filter. Technical report, Centre for Cognitive and Computational Neuroscience, University of Stirling, Stirling UK, 1997.

[2] U. Ernst, K. Pawelzik, and T. Geisel. Multiple phase clustering of globally pulse coupled neurons with delay. In M. Marinaro and P.G. Morasso, editors, *ICANN94: Proceedings of the international conference on artificial neural networks, Sorrento, Italy, 26-29 May 1994*, pages 1063–1066. Springer-verlag, 1994.

[3] M.J. Hewitt and R. Meddis. A computer-model of amplitude-modulation sensitivity of single units in the inferior colliculus. *Journal of the Acoustical Society of America*, 95(4):2145–2159, 1994.

[4] M.J. Hewitt, R. Meddis, and T.M. Shackleton. A computer model of a cochlear nucleus stellate cell: responses to amplitude-modulated and pure-tone stimuli. *Journal of the Acoustical Society of America*, 91(4):2096–2109, 1992.

[5] W. Maass. Networks of spiking neurons: The third generation of neural network models. *Neural Networks*, 10(9):1659–1671, 1997.

[6] M.I. Miller and M.B. Sachs. Representation of voice pitch in discharge patterns of auditory nerve fibers. *Hearing Research*, 14:257–279, 1984.

[7] R.E. Mirollo and S.H. Strogatz. Synchronization of pulse-coupled biological oscillators. *SIAM J. Applied Mathematics*, 50(6):1645–1662, 1990.

[8] A. Nishwitz and H. Glunder. Local lateral inhibition - a key to spike synchronization. *Biological Cybernetics*, 73(5):389–400, 1995.

[9] A.R. Palmer and I.M. Winter. Cochlear nerve and cochlear nucleus responses to the fundamental frequency of voiced speech sounds and harmonic complex tones. *Advances in the Biosciences*, 83:231–239, 1992.

[10] J. O. Pickles. *An Introduction to the Physiology of Hearing*. Academic Press, 2nd edition, 1988.

[11] C.E. Schreiner and G. Langner. Periodicity coding in the inferior colliculus of the cat. ii. topographical organization. *Journal of Neurophysiology*, 60(6):1823–1840, 1988.

[12] L.S. Smith. Onset-based sound segmentation. In D.S. Touretzky, M.C. Mozer, and M.E. Hasselmo, editors, *Advances in Neural Information Processing Systems 8*, pages 729–735. MIT Press, 1996.

[13] A. van Schaik, E. Fragnière, and E. Vittoz. A silicon model of amplitude modulation detection in the auditory brainstem. In M.C. Mozer et al., editor, *Advances in Neural Information Processing Systems 9*, pages 741–747. MIT Press, 1997.