Multi-neuronal auditory coding for frequency resolution beyond the refractory threshold

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Abstract

We propose a new mechanism for high-pitch perception by a system of multiple neurons capable of resolving frequencies higher than the frequency associated with the mean refractory period up to a multiple thereof.

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The cochlea works as transducer of minor fluctuations in the atmospheric pressure (sound) into a train of action potentials along the auditory nerve. The properties of sound are represented in spatial (tonotopic) and temporal patterns of the neuronal spike trains. There is still controversy about significance and interrelationship of both coding strategies [1, 2].

Defects on the gap junction protein Connexin 26 are predominantly responsible for severe-to-profound non-syndromic hearing loss. Temporal bone histopathology has evaluated near-total degeneration of cochlear hair cells, but a preservation of spiral ganglion neurons, in Connexin 26–related deafness [3]. Under convenient conditions like these, for the propagation of acoustic information, cochlear implant techniques, bypassing the cochlea and stimulating auditory neurons directly, are clinically useful.

Cochlear implants attempt to mimic tonotopy and time resolution of the normal ear with varying degrees of success (NIH: Consensus statement 1995). In multichannel cochlear implants, low frequency information is delivered to apical cochlear locations while high frequency information is delivered to more basal locations. This technical mimicry of cochlea's tonotopy is sophisticated but cannot guarantee persistent speech discrimination. Recent findings in human cochlear pathology qualify the implication of spatial stimulation techniques for speech discrimination [4]. Up to now, all the different temporal coding strategies work on the basis of stimulus-correlated modulations of predefined carrier-frequencies [5, 6]. Therefore, cochlear implant stimuli elicit deterministic response patterns, whereas natural spike trains in auditory neurons show stochastic temporal distributions [7, 8].

The modulation or replacement of implant carrier rates with noise is a practicable approach to mimic Nature [9, 10]. Noise is the basic requirement for the manifest stochastic resonance phenomena in natural auditory signal transduction [11]. Already 1996, Gstoettner et al. [12] tested the validity of fractally coded signal transmission in man. A small cohort of six patients recognized intensity, frequency and some nonlinear temporal cues of the electrical signal trains feeded into a single intracochlear electrode. The fractal dimensions of the used electrical random patterns corresponded to the predicted values, necessary for a safe information transfer in mammalian auditory circuits [13, 14].

In what follows, we present a novel mechanism utilizing stochasticity for the transduction of sound into neural signals by considering the correlated effect of such signals on groups of neurons, rather than considering the spike activity resulting from a single auditory neuron. We consider several neurons whose refractory phases are not exactly identical but

vary stochastically. Initially, the offsets of the spiking activity of these neurons also vary stochastically.

For the sake of demonstration, suppose these neurons are confronted with a mono-frequency signal whose pitch would require an effective absolute refractory period of r/n, where r is the mean refractory period of a single neuron, and n is the number of such neurons. We will show that through the coherent stimulation of neurons, a collective pattern of neural activity forms which would properly contain the frequency information of the signal otherwise unattainable by single auditory neurons.

To obtain a first feeling for this mechanism, consider a signal whose frequency is $1/r$, identical to the associated refractory period r of a single neuron. Ideally, in such a case, the temporal resolution renders the neuron to immediately fire after each refractory period. That is, the signal of frequency $1/r$ gets temporally resolved as $1/r$ spikes per second.

Now suppose that the frequency of the input signal is doubled, or mulztiplied. In such a case, with only one neuron, this signal gets still temporally resolved by merely $1/r$ spikes per second. However, if multiple neurons are involved, multiple wave crests could activate different neurons of the group, thereby contributing to a higher spiking activity. For instance, if we add all spiking activity of a group of k neurons, the resulting activity could result in k/r spikes per second. In this way, the magnitude of the spiking activity is directly proportional to the frequency *even beyond the refractory threshold.*.

This effect is based on the assumption that the neurons either have a different offset of the refractory period, or have different absolute refractory periods (within a certain frequency range). In such cases, different neurons are stimulated by successive signal peaks. The sum over the neural activity of this group of neurons then properly represents information about the high-pitch signal, even if its frequency is too high to be resolved by a single neuron alone.

We demonstrate this effect by an elementary model of $n = 3$ neurons, all having the same absolute refractory period r, which are equidistributed over n periods of length r/n, starting from time $t = 0$. That is, these three neurons can be successively stimulated at times $0, \frac{r}{3}, \frac{2r}{3}$, and then over again with a total offset of the absolute refractory period r of each single one of these three neurons; i.e., at times $r, r + \frac{r}{3}, r + \frac{2r}{3}$, and so on.

For such a configuration, each one of the neurons can take up a signal for the successive wave trains at a frequency $\frac{3}{r}$. Fig. 1 depicts the temporal evolution of this system of neurons, stimulated by successive wave peaks.

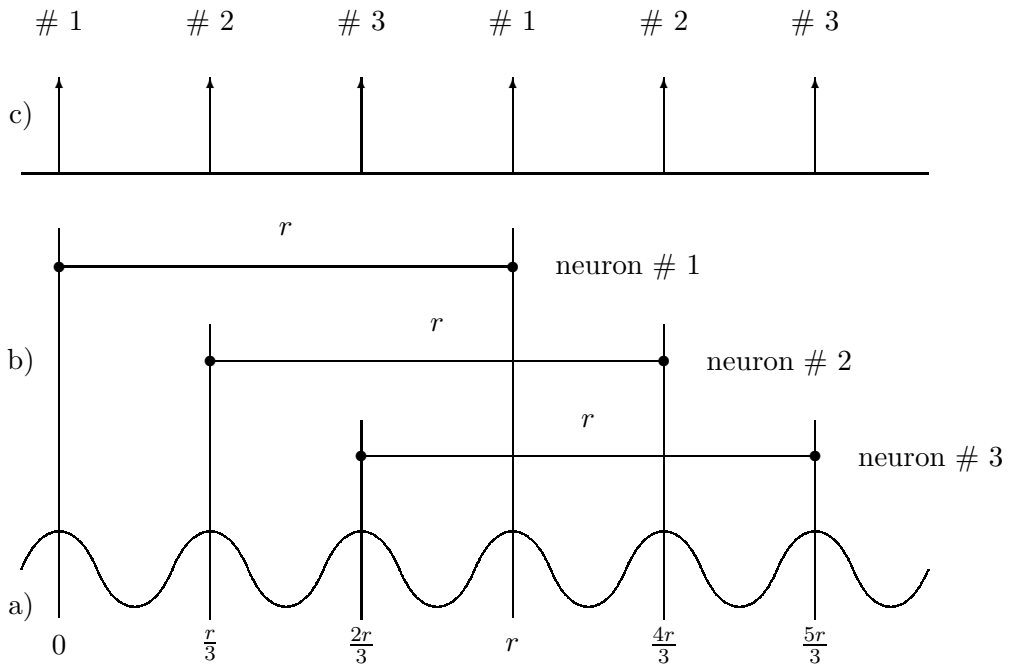

FIG. 1. Temporal evolution of a system of three neurons with equidistributed onsets of identical absolute refractory periods r, stimulated by successive wave peaks and thus being capable of resolving signals of frequency $3/r$. a) Original signal; b) neuron activation cycle; c) sum of spike trains from neuron activity.

The price to be paid for this "optimal" resolution of a mono-frequency signal is the narrow (indeed, of width zero) bandwidth which is resolved by the three neurons. This can be circumvented by considering a *stochastic* distribution of the offset phases. Stochastic offsets will be discussed below in greater detail.

Another issue is the attenuation of the signal by an effective factor of n with respect to the single, stand-alone neuron activation in the case of signals with frequencies so low that they can be resolved within the absolute refractory period. This attenuation should be compensated by either the plasticity of the auditory perception system, or by the integration of more neurons which effectively contribute to the overall signal.

In what follows we present detailed numerical studies of multi-neuron systems with a stochastic distribution of absolute refractory periods within an interval $\left[r - \frac{\Delta}{2}, r + \frac{\Delta}{2}\right]$ and initial offsets of the order of r. The driving signal is modeled by a regular spiking activity of Frequency $\omega = k/r$. In the $k > 1$ regime, coherent stimulation can be expected to contribute to high pitch perception. As for the regular case described above, the mechanism can be

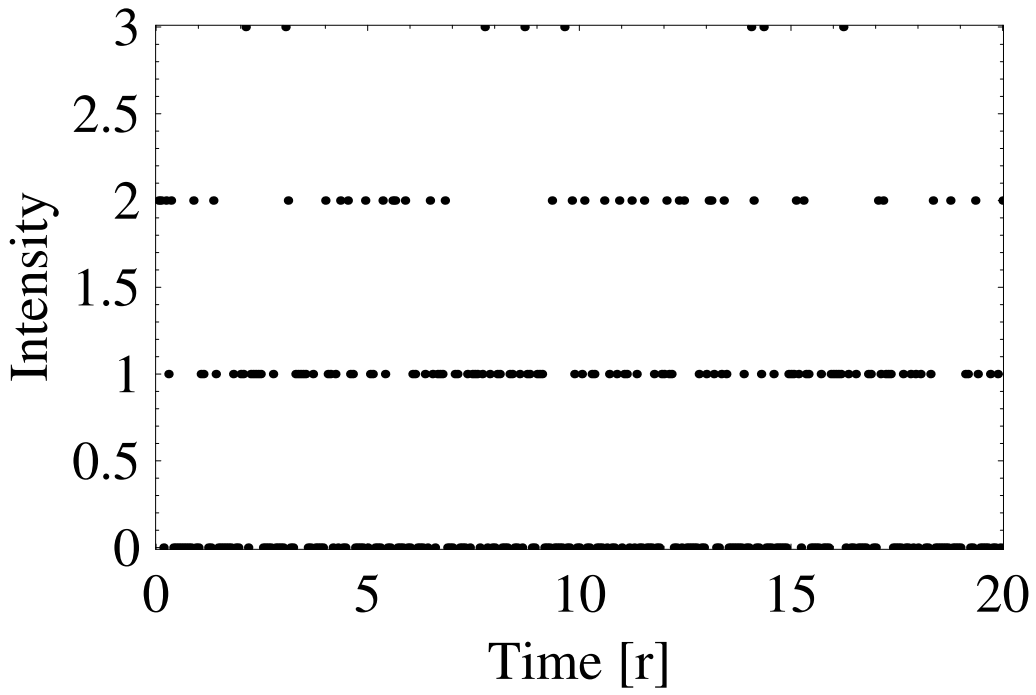

FIG. 2. Numerical simulation of the intensity of the spiking activity as a result of 11 neurons driven by a signal corresponding to 17 times the inverse mean absolute refractory period.

expected to work for $k \leq n$.

Fig. 2 depicts a numerical simulation of the intensity of the spiking activity as a result of 11 neurons driven by a signal corresponding to 17 times the inverse mean absolute refractory period. Fig. 3 depicts a numerical simulation rendering the relative error ratio ϵ of missed signal spikes to the absolute number of signal spikes as a function of frequency ω for a number of asynchronous neurons ranging from a single neuron ($n = 1$) to 30 neurons. The numerical studies indicate a reliable performance of coherent stimulation for frequencies corresponding to lower than or equal to the number of participating neurons.

In summary, we have presented one theoretical mechanism of high-pitch sound perception and one practical application thereof. (i) Theoretically, we have demonstrated a novel mechanism of high-pitch perception for the auditory transduction of sound into neural signals. This mechanism utilizes stochasticity in a system of multiple neurons, whose collective excitations resolve frequencies higher than the frequency associated with the mean refractory period up to a multiple thereof. (ii) As a practical application we suggest an economic solution for a single electrode cochlear implant which yields speech discrimination through

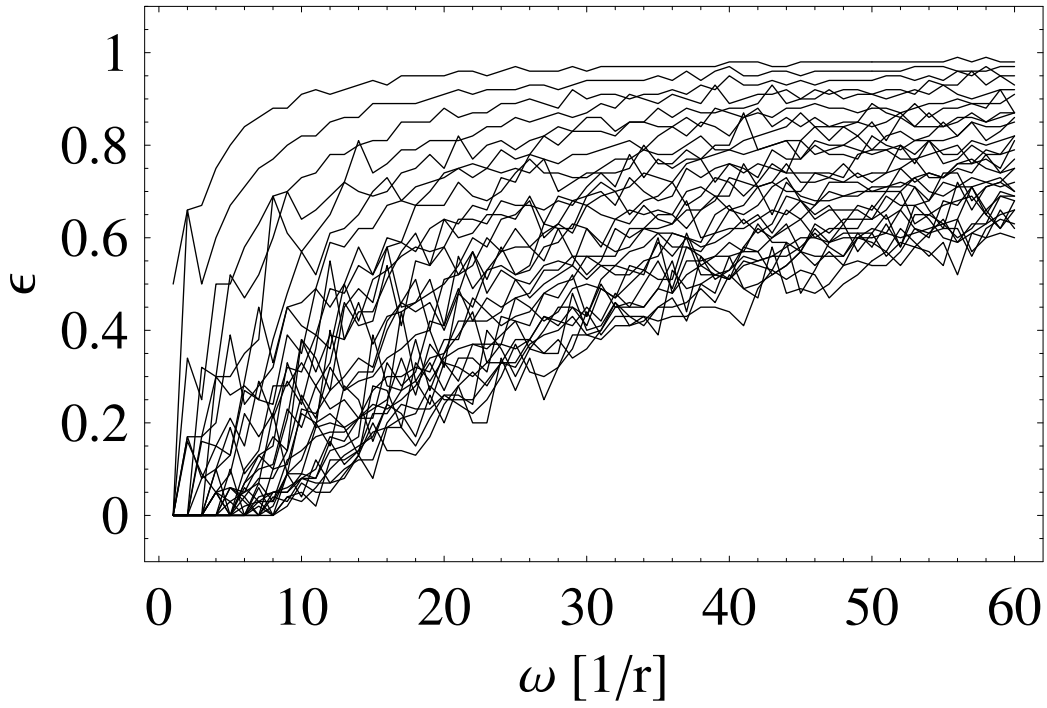

FIG. 3. Numerical simulation of the relative error ratio ϵ as a function of frequency ω for a number of asynchronous neurons ranging from a single neuron ($n = 1$) to 30 neurons.

this mechanism.

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