A Lumped Model of the Neural Peripheral and Central Nervous Auditory Link - Qualitative View II

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Abstract

This paper develops a simple lumped model of the auditory link between the cochlea and the central nervous system (CNS). The model is developed for one temporal (tonotopic) segment of the periphery. The model appears to approach that of a delay line resonator. The lumped model is derived from an expanded physiological model, which is still under development. The lumped model is capable of explaining many phenomena which are important aspects of hearing. The delay line re-circulates signal within the system, it is a delayed feedback mechanism. This signal is non-linearly limited by various physiological limitations. These combine to explain many auditory phenomena such as the base mechanism of the cochlea amplifier, phase locking and masking frequency spreading functions - to name a few.

1. Introduction

A treatment of the peripheral hearing mechanism of mammals is presented. It is particularly designed to model physiological constants of humans. This model is one stage in the ongoing development by the authors of a more complete model of the peripheral system. See also (Flax and Holmes 2004) for a general introduction to delay line oscillators and other aspects in this development.

The cochlea and the brain are connected together through a signal regenerative system. This regenerative system consists of an afferent neural projection to the brain from the cochlea and an efferent neural projection from the brain back to the cochlea (Jahn and Santos-Sacchi 1988). The term 'peripheral auditory system' is applied as a label to this system, between the cochlea and the CNS. The label 'peripheral auditory circuit' also applies. This article and its companion (Flax and Holmes 2004) will treat a physiological basis of the peripheral auditory system. The same system is used in the companion article and is re-introduced here to make this article complete.

These papers target phenomena relating to the nonlinear active process of a segment of the auditory periphery. Consequently they do not deal with phenomena which may only be expressed by the combination of peripheral segments. For example the cochlea microphonic potential, which is a function of the combined action of the active cochlea, is only mentioned here, but is treated in (Geisler 1998).

Some nonlinear effects still do not have their physiological basis known and will therefore not be discussed within this article either. These papers do however outline new links between the peripheral auditory model and its realisation of several previously un-explained auditory phenomena.

These papers are based on a simplified physiological description of the peripheral auditory system. A lumped model will be derived and used to explain some hearing phenomena, including.

1. Phase shift of neural firing with respect to stimulus frequency, treated in (Flax and Holmes 2004)
2. Frequency and phase locking in the auditory periphery, treated in (Flax and Holmes 2004)
3. Compression and expansion dependent on sound level (the so called cochlea amplifier), treated in (Flax and Holmes 2004)
4. Close relation to some otoacoustic emissions (OAEs)
5. Masking frequency spreading functions

The phenomena which have previously been explained (using other models) include compression with sound level (Geisler 1998).

Examples of phenomena not explained by the new model include the shift of best frequency of the cochlea amplifier with intensity (Zhang and Zwislocki 1996) and the temporal peak nonlinearity that affects the basilar membrane within less the one millisecond from the time of onset (Recio, Rich, Narayan, and Ruggero 1998). The latter of these effects may be explained by this model, but this is not yet confirmed.

This lumped model attempts to average many neural element variables into a single lumped neural element. This is done along the path from the Organ of Corti (OoC) to the brain and back. As this system is a lumped model, it will not exactly represent the intricacies of the peripheral unit. Consequently the focus is on the general nature of the lumped model. We are concerned mainly with identifying the phenomena that this type of model explains, and also with its implications for the peripheral and central nervous auditory link.

1.1. Lumped model theory

The efferent medial and lateral signals are lumped into the same neural feedback. It is the equivalent of assuming that only one type of neural family exists in the olivocochlear bundle. This is obviously not the case, as there

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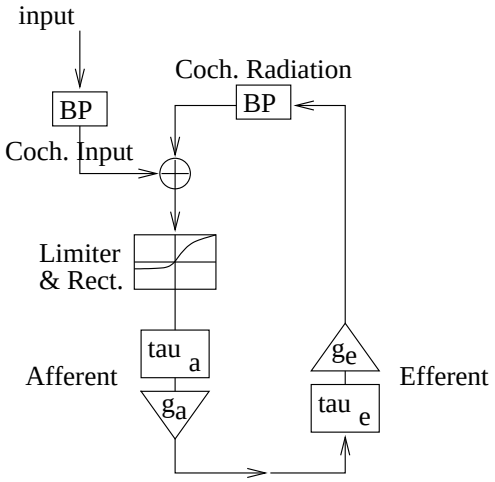


Figure 1: Lumped temporal time Domain model (one tonotopic segment). This model lumps afferent and efferent feeds into one pathway. Gains (g_a and g_e) and delays (τ_a and τ_e) model the lumped afferent and efferent feeds around the neural loop. The nonlinear limiter/rectifier represents the nonlinear (limited) range of the hair cells in transduction and transmission (Geisler 1998, Figure 8.3). The band pass filters represent the cochlea membrane (gammatone input) and motile chamber response (low order BP). Motile radiation is assumed the function of the outer hair cell group.

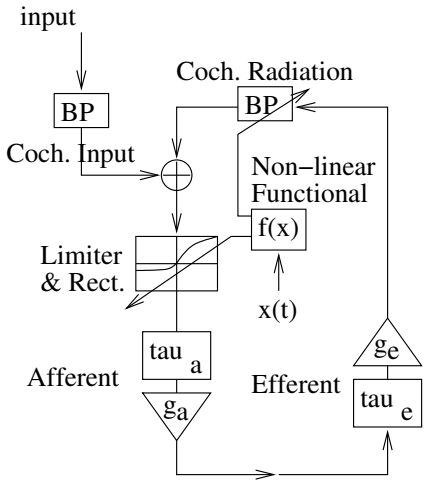


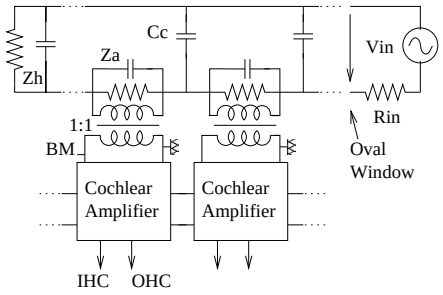
Figure 2: Lumped temporal time domain model, including the physiologically unknown non-linearity. The unknown signal source ($x(t)$) is altered by an unknown physiological element ($f(x)$). This in turn non-linearly affects the OHC BP and limiting circuit and the IHC rectification circuit. All other elements as for the lumped model in Figure 1

are two types of efferent fibres (the medial and lateral originating) (Dallos, Popper, and Fay 1996). The lateral fibres are further subdivided into intrinsic and shell neurons (Horváth, Kraus, and Illing 2000). Medial fibres are regarded as inhibitory. Lateral fibres are regarded as excitatory. One lumped segment of the neural periphery is shown in Figure 1. Still unknown nonlinear sources and elements are shown in Figure 2.

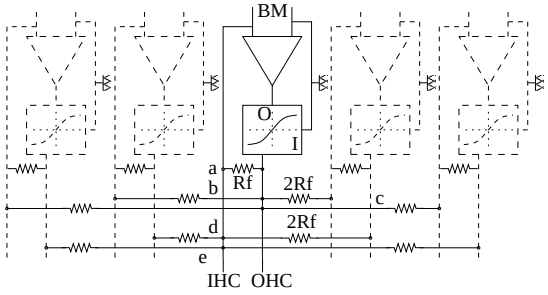
To expand further, such a model is similar to that depicted by Zwicker's lumped model (Zwicker 1986; Zwicker and Peisl 1990; Baumgarte 1997), shown in Figure 3. In this case, the cochlea input is represented by a ladder of resonant LRC circuits, Figure 3a (the 'input' band pass filter in Figure 1). These are connected to a cochlea amplifier through a transformer. This transformer feeds the cochlea amplifier and obtains feedback from the cochlea amplifier through the same transformer. This feedback from the higher centre is depicted by the 'cochlea re-radiation' BP filter in Figure 1. Each of Zwicker's higher temporal segments were coupled using simple resistances, Figure 3b. Similarities exist between the model in Figure 1 and Zwicker's hardware model. Most note-ably, are the nonlinear limiter and cochlea re-radiation (feedback).

The neural model in this article incorporates the fact that the feed back to the mechanical cochlea occurs at a basally shifted frequency (Sobkowicz, August, and Slapnick 2004), however this is not a necessity - merely a novelty - the results still hold without frequency shift.

The model turns off the OHC related nonlinearities. These nonlinearities are believed to explain the frequency



(a) Cochlea input to the higher lumped 'cochlea amplifier' is conducted through a chain of resonant BP filters



(b) Cochlea amplifier segments are resistiveley coupled

Figure 3: Zwicker's lumped model. The peripheral to CNS path is simplified to an amplifier and limiter. No specific delays of transmission exist as there is only local feedback.

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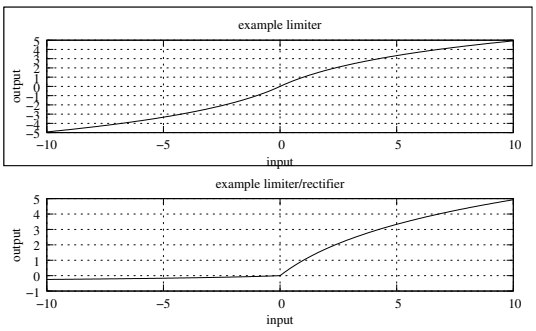

Figure 4: Example limiter and rectifier.

glide (Zhang and Zwislocki 1996) of the cochlea amplifier (CA). This nonlinearity is the block in Figure 2 which is represented by $f(x)$. For this article the physiological element which implements the CA frequency glide is unknown, consequently $x(t) = 0$, which is intended to imply no adjustment.

This article, is not interested in producing a neural spike reality. Instead the flux density of action potential movement through the lumped afferents and efferents are modelled. This flux density variance per unit time is termed 'Ionic Current units'. As it is a propagation of charge through an area of neural tissue.

We are interested in pursuing the general nature and possibility of a coarse lumped neural loop. For this reason the absolute minimum of processing affects are used. IHC rectification is used in circuit, unless otherwise noted. The rectification is believed to be a process of the ribbon synapse in the IHC mechanism (Lenzi, Crum, Ellisman, and Roberts 2002). See Figure 4 for an example limiter and rectifier. These units are actually different to those which should be used to model the limiting nature of Hair cells. Hair cells tend to compress the input at both subthreshold and large input magnitudes. The mid-magnitude region thus has a gain, which is linear in nature. A limiter would thus have a sigmoidal type of nature for both positive and negative input signals individually. As this article is a qualitative inspection of the periphery, more complex nonlinearities then those depicted in Figure 4 will not be used. A more accurate model may be derived from the experimental data of (May and Sachs 1992).

The type of delay-loop model used in this article is quite common in electrical and electro-mechanical systems. A brief overview is given in (Flax and Holmes 2004), as well as a treatment of phase/frequency locking and the cochlea amplifier. Other results of the implementation are presented here.

2. Implementation and Results

A coarse (reasonably low Q=1) filter is used to represent the cochlea chamber. The re-radiation filter representing the actuation of the OHC and re-transduction is chosen to be second order. This is classically based on the transmission line model of the passive cochlea (Ambikairajah, Black, and Linggard 1989), however this has been developed further in the literature to include a second filter (Allen 1980). This second filter is thought to approximate more closely the influence of the tectorial membrane on the OoC/basilar system and sharpens mechanical response.

As the thickness of the afferent neurons are large, their propagation speeds are greater then the efferent neurons (Davey 2002). The averaging of lateral and medial efferent delays is like mixing myelinated and non-myelinated propagation speeds. Because of this averaging, the efferent delay is approximately two orders slower then the afferent delay. The efferent delay used in this implementation is $\tau_e = 1.3\,ms$. The afferent gain is set to $g_a = 0.95$ which is directly related to the percentage of the afferent population which are type I neurons. The efferent gain is set to $g_e = 0.14$, again related to ipsilaterally lumped population percentages. Inhibitory populations are represented as negative gains when averaging to find the lumped gain parameter. The lumped gain is positive and shows that there is more ionic current in the lateral efferents then the medial efferents (ipsilaterally).

Circular buffers are used to implement the neuron delays. This conserves computer memory. The input signal is a linearly chirped sinusoid. It is chirped from 20 Hz to 6 kHz. Conceptually it is easier to think of the input signal as a train of sinusoids which gradually increase in frequency. Using this paradigm, each time point represents the response to probe frequency as well as instantaneous signal (at that point in time).

As the time domain model is trivial, it is implemented in C++ for speed of execution.

2.1. Close relation to some Oto-Acoustic Emissions (OAEs)

A review of the literature reveals that OAEs are uniform in many attributes (Kemp 1978; Wit and Ritsma 1979; Wilson 1980; Zurek 1981; Schloth 1983; Wit and Ritsma 1983; Rabinowitz and Widin 1984; Zurek 1985; Zizz and Glattke 1988; van Dijk and Wit 1990; Probst, Lonsbury-Martin, and Martin 1991; van Dijk, Wit, Tubis, Talmadge, and Long 1994). Evoked emissions generally have an onset time delay which peak within approximately ten to twenty milliseconds. Likewise a suppressed spontaneous OAE reappears within ten to twenty milliseconds. OAEs are most common and maximal in the one to two kilo Hz frequency range.

When OAEs are evoked with a chirped (sweeping) probe signal, they undulate in intensity depending on the frequency of the stimulus. These OAEs form peak and trough intensity undulations in their emissions as the probe signal is swept.

Neural afferents are re-cycling power to the IHC and OHC through the efferents. It takes a while to build power in the neural loop, this is the hypothesised delay to OAE maximum from onset. This delay may be verified by inspecting Figure 5, it is a function of loop delay. The maximal energy in the cochlea segment is a short time after the gammatone best frequency is swept. This is related to the limited bandwidth of the gammatone filter and loop delay. It is seen by a neural energy peak at $1000+\triangle f$ Hz. Further, it is again desirable to explain the OAE intensity undulations by the re-circulation system developed in this

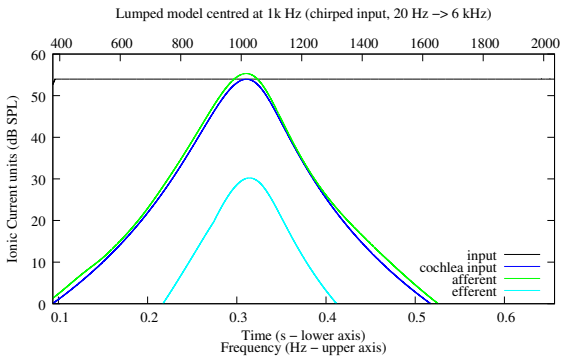

Figure 5: OAE energy can be derived from the efferent current which excites the OHCs. This efferent energy not only adds to the afferent neural response but also may be radiated out from the cochlea into the middle ear.

article. The emission maxima would match the system natural modes as they are swept through by the probing chirp. Frequencies between the maxima would cause troughs. A gradual sweep through many modes would produce such emission intensity undulations. This however would be incorrect. The undulations are an artefact of all coupled segments of the auditory periphery. Although the coupled segments would still embody some form of re-circulation system, it is unclear exactly what the nature of a coupled system would be.

Finally OAEs are phase locked to the stimuli which generate them (with the exception of distortion product OAEs) (Shera and Guinan 1999). This locking is the nature of a re-circulation system.

Experiments which show the preservation of OAEs post efferent neurectomy can easily disprove this hypothesis.

2.2. Masking frequency spreading functions.

Masking frequency spreading functions (FSFs) are the first step in masking psychological systems (Flax and Jin 2001). These frequency spreading functions are an expression of the affect of a segment of the cochlea on other segments. People who generated FSFs for the purpose of masking estimation assumed that the higher side of the best frequency varies with input level (Terhardt 1979; Beerends and Stemerdink 1992; Black and Zeytinoglu 1995). Examples of Terhardt's FSFs are shown if Figure 6. The physiologically based FSFs are shown in Figures 7 and 8.

Physiological FSFs are composed of two main affects which are dependant on input signal level. Small signal levels produce frequency spread at octaves of the filter best frequency, Figure 7. For loud signals, the spreading function has energy centred at harmonics of the filter best frequency, Figure 8. The off octave harmonics appear for loud signal levels because of the limiting and rectification stage. Likewise the 'roughing' of the frequency spectrum between peaks is due to the rectification stage. This explains the high side FSF variation with level in other masking models. A more signal dependant method also exists, (Moore and Glasberg 1983). Moore's method extends the masking FSF generation mechanism to be derived from other segment tuning curves. This may be addressed once a coupled

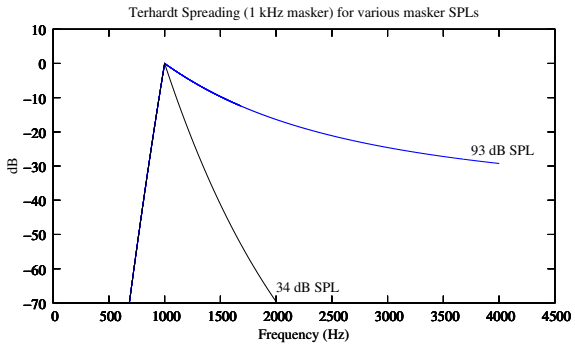

Figure 6: FSFs proposed by Terhardt. The high side frequency spread levers up with signal level. The low side stays the same.

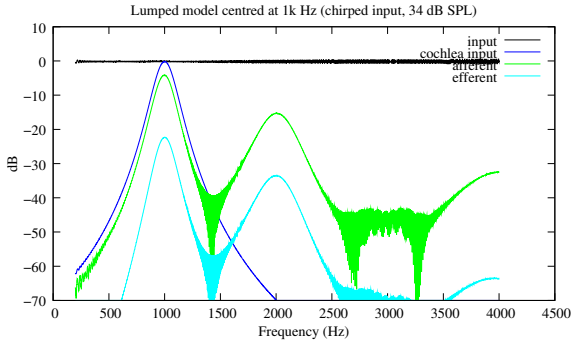

Figure 7: Frequency Spreading Function at 34 dB SPL, with rectification. In this case, the cochlea amplifier is amplifying the soft signal.

DLR model is derived from the physiological morphology.

3. Conclusion

This lumped model of the physiological auditory periphery presented here is a simplified feedback system. Each element is based on physiological units which are present in mammalian anatomy. The lumped model simplifies the periphery to the bare essentials that describe some of the known phenomena of hearing. Examples of the ele-

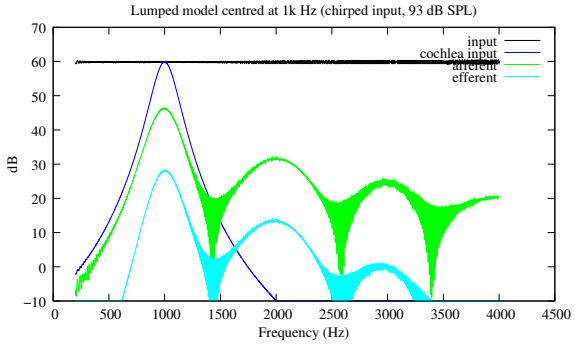

Figure 8: Frequency Spreading Function at 93 dB SPL, with rectification. In this case, the cochlea amplifier is saturated by a large signal.

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ments omitted from this model include the IHC high pass filter (Mountain and Cody 1999) and signal degradation during propagation (Johannesma 1971). This signal degeneration is thought to be re-generated by the many different types of neural morphologies in the cochlea nucleus (Brown and Benson 1992; Paolini and Clark 1999).

This model which is still being developed by the authors, does not yet account for all the phenomena in the peripheral auditory system. It does however explain the physiological basis of phase and frequency locking which occurs in this first peripheral neural circuit before contacting the higher levels of the CNS. It explains the physiological basis of masking frequency spreading functions. Frequency spreading functions are affected by nonlinear elements. Low levelled input signals have an octaval nature in the FSF contour. The contour between the octave maxima are amplified by the rectification mode of the IHC. This flattens the FSF contour. High level input signals are more drastically affected by the nonlinear mechanisms and wide band harmonic peaks appear in the spectrum. This physiological mechanism of FSF generation strengthens the importance of the FSF elements in traditional expert masking systems. A level dependant harmonic FSF generation mechanism would aid future expert masking model development.

The model behaves in line with the experimental nature of the OAEs. It is hypothesised that the auditory periphery is a part of the physiological basis of OAEs. Qualitatively, the time required to build up OAEs is explained by the delay and time required to build energy in the neural recirculation system. The phase locking nature of evoked and spontaneous OAEs is explained by the phase locking nature of the auditory peripheral circuit. It is unclear whether the swept probe emission undulations are a function of the recirculation system. This is because swept probe emissions are a function of coupled neural loops. While it is proposed that OAEs are generated by this peripheral circuit, it is most likely that OAEs are a combination of this and other generator mechanisms.

The main contribution of the new model, compared to earlier models, is to introduce time-varying nonlinearities and realistic delays associated with the neural link into the peripheral circuit. Earlier models ignored these delays, which we believe are significant and help explain several auditory effects that the earlier models did not account for. These effects include phase locking, masking frequency spreading functions and part of the basis of OAE generation. These other models include those mentioned previously (Zwicker 1986; Zwicker and Peisl 1990; Baumgarte 1997) as well as others (Stasiunas, Verikas, Kemesis, Bacauskiene, Miliauskas, Stasiuniene, and Malmqvist 2003; Stasiunas, Verikas, Kemesis, Bacauskiene, Miliauskas, Stasiuniene, and Malmqvist 2004).

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