Introducing the Compression Wave Cochlear Amplifier

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Abstract

The compression wave cochlear amplifier (CW-CA) model is introduced for the first time. This is the first cochlear amplifier model that is soundly based on the physiology of the inner ear and neural centres. The CW-CA, which is assumed to be driven by the basilar travelling wave (TW), includes outer hair cell (OHC) motility and neural signal transmissions between the hair cells and the central nervous system as key elements. The OHC motility sets up a pressure wave in the cochlear fluids, which is modulated by the neural feedback. The CW-CA is represented mathematically as a delay-differential equation (DDE). Even the simplest model based on this concept is capable of explaining a wide range of hearing phenomena, including various types of otoacoustic emissions, distortion products, two-tone suppression, amplifier gain and compression, frequency specificity, neural spontaneous rates and many other phenomena including various pathological conditions.

1. Introduction

To understand how speech and audio are perceived by the ear, it is pertinent to consider how the inner ear transduces sound to the brain. Central to the current understanding of inner ear function is the concept of the travelling wave on the basilar membrane:

“With the stapes set into sinusoidal vibration, the phase relations, measured along the length of the cochlear partition, show that a travelling wave has been set up. The form of resonance which appears does not correspond at all with that to be found in a simple vibrating system.” [4]

It has been found that this simplistic model of the inner ear only matches the behaviour of the cochlea of dead animals [8, 7, 19]. Further, in accordance with an earlier prediction of cochlear activity [21], emissions from the ear were finally captured [25]. For these and other reasons, more than a passive travelling wave model is required to capture the nature of the active live cochlea. These extended active models are known by the generic term ‘cochlear amplifier’.

It is not clear whether the active processes of hearing are due to nonlinear mechanics [2] or the cochlear amplifier (CA) [9]. This point is discussed with respect to both nonlinear mechanics and the CA in [3]. The present article and its companion [17] attempt to lay the foundation for a physiologically based model that embodies both nonlinear mechanics and the CA.

The most popular CA model currently available is the active travelling wave (TW) model [7, 32, 18]. Other models also exist, such as oscillator models [24, 42, 39, 38], the squirting wave model [5], tuned hair cell models [6], the feedback amplifier model [43] and Hopf bifurcation models [15, 30, 13]. Oscillator models of the CA lack any close physiological correspondence and are derived purely from a mathematical basis.

The squirting wave model is physiologically based, but requires further alignment with known auditory phenomena such as two tone suppression. The tuned hair cell model can only be valid for mammals with hair cell tuning [26]. Finally the feedback amplifier models (which include Hopf bifurcations) also have a limited physiological basis, and the Hopf bifurcations have static features that don’t correspond with known phenomena.

As for the other CA models, the active travelling wave model also lacks a good physiological basis. For example, the active pressure in one popular model [31, 32] is functionally derived from the relative velocity between the tectorial and basilar membranes, but this is not related to any known physiological mechanism. Common to all active travelling wave CA models is the point of view that the CA generates an active pressure difference across the scala vestibuli and scala tympani [10]. Exactly how this pressure difference is generated is still a mystery, and as these pressure difference models are at least 25 years old, the continuing lack of a physiological explanation is troublesome.

Other factors also contradict the concept of an active pressure difference across the basilar membrane (BM). For example, CA emissions have been found in animals that don’t possess a basilar membrane [20, 35, 40], but the active travelling wave model requires a BM to explain most emissions [7].

This article will introduce for the first time the compression wave CA (CW-CA) model. Such a model has previously been hypothesised to exist due to experimental evidence [41, 36, 33, 34, 22], but so far it has been nothing more than a suggestion. Even before its definition (in this article) some authors have argued against its existence [11, 37]. The CW-CA comprises cochlear mechanics, hair cell activity and nonlinearity, and neural feedback.

This article begins by introducing the CW-CA, then briefly touches on what the CW-CA is capable of modelling, and concludes by showing an example output from the model.

2. The compression wave CA

An auditory vibration which enters the outer ear is transduced from air compression to a cochlear fluid compression wave by the middle ear [7]. This fluid compression wave is filtered by passive cochlear mechanics, most popularly modelled by the travelling wave (TW) [7]. At cochleotopic points of reception of the TW, the stereocilia are mechanically moved and the electrical state of the hair cells is altered through transconductance modulation. This foundation for transconductance modulation is treated in the companion paper [17] by examining the electrophysiology of the hair cell, which is the foundation for nonlinear mechanics.

The transconductance and electrophysiological changes experienced by the hair cells generate two significant effects: neurological signalling and active mechanical signalling.
The active mechanical signalling is generated by outer hair cell (OHC) motility [28, 14, 7]. The compression of the OHCs generates fluid-borne pressure waves which propagate through the mechanical cochlea and are retained by the compliance of the cochlear windows. In this first presentation of CW-CA mechanics, simple macromechanics are assumed, and the unbalanced nature of the cochlear windows, as well as the micromechanics of the Organ of Corti (OoC), are not modelled.

In contrast to the active TW models, the physiological basis of the active compression wave is simple in this CW-CA model. The OHC basolateral membrane experiences electrophysiological changes, due to transconductance changes in the stereocilia, which result in potential changes across the membrane. These potential changes generate a force centred at a reference point at the middle of the OHC. This force generates equal and opposite forces at the apex and base of the OHCs. The generated forces act on the mechanical impedances of the cochlear scala tubes and windows. The mechanical signals which are filtered by the mechanical impedances again alter the stereocilia and the hair cell basolateral membrane conditions. Due to the changes in the basolateral membrane conditions, the inner hair cells excite the afferent neurons. These neural signals affect the central nervous system and the resulting delayed signals are fed back to the periphery through the efferent neurons. The efferents innervate both the type-I afferents and the OHC basolateral membrane resulting in mechanical actuation and neural modulation. The cycle is complete and the CW-CA loop is defined.

Two lumped mechanical impedances in the new model represent the fluid damping, fluid mass and window compliance between the active OHC compression and the middle ear, as shown in Figure 1. The two mechanical pathways are from the apex and base of the OHC through the scala vestibuli and scala tympani, represented by the mechanical impedances $H_{L,O}$ and $H_{L,R}$ respectively (the subscripts $O$ and $R$ refer to the oval and round windows). The windows (and stapes) are not attributed mass, as their mass is more than one hundred times less than that of the rest of the cochlea [29]. Hence the macromechanical mass is assumed to model the lumped masses of the windows and the OoC micromechanics as well as the fluid masses. Similarly, the compliances of the windows are assumed to be lumped with those of the micromechanics of the OoC into one macromechanical element. This lumping is not unreasonable as the windows are attributed a significant compliance in relation to the rest of the cochlea [29]. These mechanical impedances are taken to be second order in the simplest model.

The hair cell neurological signalling is best modelled by the transient fluctuations in basolateral hair cell currents, whose foundation is discussed in the companion paper [17]. The neurological signals are conducted to the central nervous system (CNS) through the afferent type-I and type-II neurons, which innervate the inner hair cells (IHCs) and OHCs respectively [7]. The superior olivary complex (SOC) returns the neural signals from the CNS to the cochlea through the efferent neurons [7]. The efferents innervate the type-I neurons and the OHC basolateral membranes through the lateral and medial efferents, respectively. These afferents and efferents represent a neural feedback through the CNS, which was first hypothesised in [27], through this reference did not attribute any significance to the neural signalling. Due to the delay in propagation, the two efferent systems are modelled by a lumped delay and system gain. The signals propagating in the neural system are treated as continuous time signals in the model (neural smoothing and signal degradation [23] and regeneration are not presently allowed for).

This neural feedback completes the olivocochlear feedback

Figure 1: OHC actuation and mechanics. The image shows the cochlea with apex on the right hand side. The force of the OHC ($F$), which resides inside the Organ of Corti (OoC), shifts fluid, which is bounded by the round (R) and oval (O) cochlear windows. The force acts through the transfer function $H_{L,O}$ and $Z_o$. As there is no compliance apically (to the right), their loads are simply assumed to be infinite and neglected in the computation. $Z_t$ is the scala tympani and round window impedance (transfer function $H_{L,R}$) and $Z_o$ the scala vestibuli and oval window impedance (transfer function $H_{L,O}$).

Figure 2: An expanded view of the physiological CW-CA (taken from [16]). The structure in the middle of the image represents the OoC with the IHC on the left and the three OHCs on the right. The support cells are also shown, as well as other OoC-related structures. Sound from the external world is transformed into the inner ear through the ‘passive mechanics’ block, which includes the outer, middle and inner ear (the passive travelling wave). The result is a signal which mechanically actuates the hair cell stereocilia. The actuation of the stereocilia generates both neural transduction and OHC pressure. The neural transduction is channelled to the CNS through the afferent neurons and fed back to the periphery through the efferent neurons. The MOC efferents alter the OHC state and this affects the generated pressure by expanding and contracting the OHCs. The generated OHC pressure creates compression waves in the fluid and these affect the CW-CA mechanics accordingly. The compression waves generate both stereocilia actuation and otoacoustic emissions (on occasions) at the oval window. A significant amount of overlap exists between the passive and active cochlear mechanics, as indicated by the dotted line.
loop. The complete physiological CW-CA model is shown in Figure 2. This physiological model can be simplified for analysis by lumping the various signal pathways. For example in the simplest model the mechanical impedances can be lumped into a single second order transfer function $H_L$, which is a parallel combination of $H_{L,R}$ and $H_{L,O}$. The neural subsystem may be attributed a linear gain and delay denoted by $H_N$. The nonlinearities represented by the hair cells are attributed instantaneous nonlinearities $f_1(·)$ and $f_2(·)$ respectively. This then generates a lumped signal loop shown in Figure 3(a). The lumped system can be simplified by merging the two hair cells nonlinearities into one limiter as shown in Figure 3(b). For convenience the entire system loop gain is also represented by the gain block $k$ and the system delay by the delay block $T$.

The lumped system equation may now be written as the nonlinear delay-differential equation (DDE)

$$k \left( \frac{b_0}{v(t)} + \frac{d}{v(t)} + b_2 \right) u(t) = \left( a_0 \frac{d^2}{dt^2} + a_1 \frac{d}{dt} + a_2 \right) v(t)$$

where the coefficients $\{a_0, a_1, a_2\}$ and $\{b_0, b_1, b_2\}$ represent the numerator and denominator of the mechanical impedance $H_L$ in the second order case (the simplest possible case). For simplicity we assume a tanh function for the limiter, so that we can eliminate $u(t)$ from this equation using $u(t) = \tanh(v(t-T)) + w'$. The equation can then be solved for $v(t)$. An infinite number of Hopf bifurcations are possible in this system, and it is capable of an infinity of different limit cycles, depending on the value of the system gain $k$.

3. Successes of the model

The lumped and simplified model (Figures 3(a) and (b) and Equation 1) of the physiological system shown in Figure 2 is capable of modelling a very large number of observed physiological phenomena [16]. These include an active gain of 30 dB SPL for forcing signals below 60 dB SPL, saturation and limiting for signals above 60 dB SPL, the sharpening of the passive cochlear response by at least 10 times, otoacoustic emissions such as SOAEs and DPOAEs, two-tone suppression and distortion products (including DPOAEs) and many other phenomena not listed here.

Figure 3: (a) Block system representing Figure 2. Block $H_L$ represents the mechanical filter that actuates the stereocilia, $f_1$ represents the nonlinear HHC transduction, $H_N$ the total nerve gain and delay, and $f_2$ represents the nonlinear OHC transduction, which generates the mechanical force. The different afferent feeds are lumped into one pathway. The LOC neural split is modelled as a gain in the $H_N$ block. (b) The lumped CW-CA is further simplified by shifting the two nonlinearities $f_1$ and $f_2$ into the same limiter (lim). The neural delay ($T$) and gain are linearly separated, with $k$ the total system gain. The forcing of the stereocilia by a travelling wave is denoted $w'$.

Figure 4: Result of ramping the level of the input signal to a stable CW-CA. A constant gain of roughly 30 dB SPL is apparent for small to mid-range input signal levels. At a forcing signal level of around 60 dB SPL, a compressive nonlinearity emerges, which becomes a limiter at around 90 dB SPL.

The model presented in this article represents only one cochlear segment. For this reason, certain phenomena aren’t explained by the single segment CW-CA. These phenomena include TEOAEs [25] and the Allen-Fahey results [1, 12]. Expansion of the CW-CA model to account for the many neural pathways as well as the modelling of many cochlear segments is expected to account for these missing phenomena.

As an example, the output from the CW-CA model in the stable case (gain $k$ small enough) is shown in Figure 4. The system demonstrates a constant 30 dB SPL gain for forcing signals below 60 dB SPL. When the forcing level is between 60 and 90 dB SPL, the response begins to saturate. For forcing levels above 90 dB SPL, the response is limited.

If the gain $k$ is increased so that the system becomes unstable, it may enter a limit cycle, which is hypothesised here to correspond to an SOAE. (The system signals remain finite in the unstable case because of the limiter.) Other phenomena can also be explained by an unstable system [16].

4. Conclusion

For the first time, an active CA model is presented that is based on the firm foundation of the electrophysiology of the hair cells. This is the first model to postulate a physiological feedback mechanism of the CA without making any unfounded assumptions or simply adopting ‘mathematical conveniences’.

This article proposes that the OHCs generate equal pressures at their apices and bases due to OHC motility, and this pressure is the active force of the CW-CA. This force propagates through the scala tubes and the macromechanical model from the OHC apex and base to the middle ear (through the cochlear windows) and thus leads to emissions. The complete CW-CA model is a combination of the three major systems present in the cochlea: the cochlear mechanics, the hair cell physiology and the neural feedback. The complete physiological system can be simplified to give a mathematically tractable model, which is best described by a delay differential equation.

The behaviour of the CW-CA varies, depending on the values of the system parameters. The system could be either stable or unstable (but still with finite-valued signals). The sta-
ble system exhibits gain for low signal levels, and compression and ultimately limiting at higher signal levels, just like the real ear. The unstable system can have many different possible states, and these are capable of explaining a large majority of the known hearing phenomena, including spontaneous and distortion product emissions, more complex multi-tonal responses, pathological conditions and emissions of various types [16].

5. References


