REPTILE EARS AND MAMMALIAN EARS: HEARING WITHOUT A TRAVELLING WAVE

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Abstract

This paper takes a closer look at the functional similarities between reptile ears and mammalian ears. The ears of the first class of animal are generally acknowledged to lack travelling waves – because the sensing cells sit upon a stiff support – whereas the ears of the second group are commonly thought to act differently, having hair cells arranged upon a compliant basilar membrane that moves under the action of a travelling wave (created by a pressure difference across the membrane) so that the wave bends the cells' stereocilia. However, recent work suggests that the mammalian case can be explained without reliance upon a travelling wave as a causal stimulus and that the responses observed can be interpreted as local resonances driven by a fast pressure wave. In this light, reptiles and mammals may have more in common than currently appreciated – they might both be forced resonant systems – and this paper explores such a possibility.

OÍDOS DE REPTILES Y OÍDOS DE MAMÍFEROS: OÍR SIN ONDAS PROGRESIVAS

Extracto

El presente artículo examina con detenimiento las similitudes funcionales entre los oídos de los reptiles y los oídos de los mamíferos. En general, se considera que los oídos de los primeros no poseen ondas progresivas, porque las células sensoriales se encuentran sobre un soporte rígido, mientras que comúnmente se cree que los oídos de los segundos actúan de modo diferente, puesto que tienen células pilosas dispuestas sobre una membrana basilar elástica que se mueve bajo la acción de la onda progresiva (creada por la diferencia de presión a lo largo de la membrana), de modo que la onda curva los estereocilios de las células. Sin embargo, obras recientes sugieren que el caso de los mamíferos puede ser explicado independientemente de la onda progresiva como un estímulo casual y que las respuestas observadas pueden ser interpretadas como resonancias locales dirigidas por una onda de presión rápida. A la luz de esto, reptiles y mamíferos pueden tener más en común de lo que se creía hasta ahora: ambos pueden funcionar con sistemas resonantes, y el presente artículo explora tal posibilidad.

УШИ ПРЕСМЫКАЮЩИХСЯ И УШИ МЛЕКОПИТАЮЩИХ: СЛУХ БЕЗ БЕГУЩЕЙ ВОЛНЫ

Резюме

Настоящая научная работа приделяет глубокое внимание функциональным сходствам между ушами пресмыкающихся и ушами млекопитающих. Уши первой группы животных общепризнанно считаются не имеющими бегущих волн, потому что рецепторные клетки размещены на жесткой опоре, в то время как уши второй группы общепризнанно считаются действующими по-другому, имеющими волосковые клетки, размещенные на эластичной базилярной мембране, которая движется под действием бегущей волны (вызванной разницей в давлении в мембране), таким образом волна сгибает стереоцилий клеток. Однако, в настоящей работе предполагается, что случай с млекопитающими можно объяснить, не опираясь на бегущую волну как на причинный стимул, и что замеченные ответы могут быть интепретированы как локальные резонансы, вызванные быстрой волной давления. В этом свете, пресмыкающиеся и млекопитающие могут иметь больше общего, чем продполагается. Эти две группы могут быть принудительными резонирующими системами, а настоящая работа исследует такую возможность.

Introduction

It has been noted that there are similarities between the "travelling wave" delays in the hearing organs of mammals on one hand, and delays in the ears of frogs and lizards on the other (which do not have travelling waves in the sense of a wave generated by a serially coupled stimulus) [1–3]. The sensing surface of the ears of frogs and lizards sits upon a stiff basilar papilla that does not sustain a travelling wave [4,5]. How then do they hear? What is the adequate stimulus to their hair cells, which, like the mammalian ear, bear stereocilia that are surmounted by a gelatinous tectorial membrane? [6,7]. Given the possibility raised in a recent paper [8] that the mechanics of both mammalian and non-mammalian ears might be explicable largely in terms of fast pressure waves and resonance, a closer comparison between these classes of animals might be productive.

Bergevin and Shera [1] remark how the responses of lizard ears are "strikingly reminiscent" of those in mammals, despite major differences in inner ear morphology and function, and that lizards "evidently lack traveling waves". These authors modelled the gecko ear as an array of coupled harmonic oscillators subject to a parallel stimulus and found, in line with simple resonance, that the quality factor, or Q, of the oscillators governed the build-up time of stimulus frequency otoacoustic emissions (more specifically, their model produced delays of ~2/3 Q). The results were that the mathematics of the lizard system was virtually the same as that of the mammalian one, although that similarity was left as a curiosity and the underlying "adequate stimulus" in the two cases was considered fundamentally different. In the lizard case, the adequate stimulus was considered to be a rocking motion of the hinge-like papilla (even though such an arrangement appears to be inefficient in transferring the stimulus – the deflection angle θ of the hinge – to the hair cells, since the effective component, sin θ, approaches zero).

In looking at similarities between the reptile and the mammal, it is noteworthy that in both animals there is an arrangement where hair cells and their stereocilia are covered by a gelatinous tectorial membrane which couples neighbouring cells together. There is a more general resemblance too: in reptiles there are two clusters of hair cells of opposite "polarity" separated by a dividing line or striola [9], whereas in the mammal there are three precise rows of outer hair cells which, it has been conjectured [10,11], may also act with opposite polarity between the rows (meaning in this case that the length of the cell bodies extends in opposite directions as a result of electromotility differences). In both cases, such an arrangement could lead to standing waves and tune the system.

The calculations made in a recent paper [8] invite the suggestion that the adequate stimulus in the two cases might also be the same: a fast pressure wave detected by the body of the hair cell which then creates motion in response. If the motion is communicated to neighbouring cells, a feedback loop is created and a standing wave will result. Diagrams of the lizard ear's anatomy [1,6] are all compatible with the idea that the system receives a pressure wave directly through the cochlear fluids. The sensing cells are strategically placed between where sound enters the otic capsule and where it leaves.

Bergevin et al. [12] found that otoacoustic delays in 12 species of lizards were all well described by the build-up time of filters of defined Q, so that, again, $N_{SF} \approx 2/3\ Q$ (or $2Q_{10\ dB}$), where N_{SF} is the phase delay gradient. In other related reptile work, Bergevin (2010) [13] examined otoacoustic emissions from a wide range of gecko species and again found that the build-up time of the underlying sharply-tuned auditory filters accounted for the emission latencies. The conclusion of this work was that, given the lack of basilar membrane travelling waves in geckos, propagation of otoacoustic emissions could be via fast compression waves. While acceptable in lizards, the idea that fast waves underlie otoacoustic emissions in the mammalian cochlea has remained controversial since it was first suggested by Wilson in 1980 [14,15]. One reason is its incompatibility with the standard coherent reflection model [16] in which the circulation of slow forward and backward travelling waves is fundamental.

Ruggero and Temchin [17] compared the ears of human and non-mammalian animals, and found that the response delays were similar in both cases. These researchers viewed the human case as one in which ripples conveyed energy along the basilar membrane before exciting the cells (causing a wave front delay), and the interpretation was extended to lizards and frogs, although in this case the suggestion was that the ripples propagated serially along the tectorial membrane. In their view, the underlying Q values were low, meaning it became necessary to ascribe the observed additional delay to wave-front delay (the time taken for a travelling wave to propagate to the hair cells).

In all the above research, the results are broadly compatible with the delays in the hearing organs of mammals or reptiles being due to filter build-up time, in which case it can be rather misleading to call it travelling wave delay. The difficulty in trying to give a coherent account of what is going on is that travelling wave delays cannot be separated from build-up times: if wave front delay is zero, then the two concepts are identical, as demonstrated by the calculations set out in [8]. This paper takes the view that most of the existing literature can be well explained without recourse to travelling waves – in the sense of them being causal chains involving a ripple propagating serially from one coupled element to the next. Clearly, this diverges from the standard view, which is set out, for example, in [18]. However, as study of [18] and [19] conveys, the issue is complicated and depends on using particular models and assuming certain types of interactions. Resolving the issue calls for more investigation and measured assessment, but this paper is part of a more general program [8,20,21] to see if a basic resonance approach might be able to simplify matters.

The fast wave and an explanatory model

Conventionally, the effective stimulus to the mammalian cochlea is taken to be the pressure difference across the basilar membrane; this gives rise, via hydrodynamic and basilar membrane coupling, to a propagating ripple known as a travelling wave [22]. Given that this process

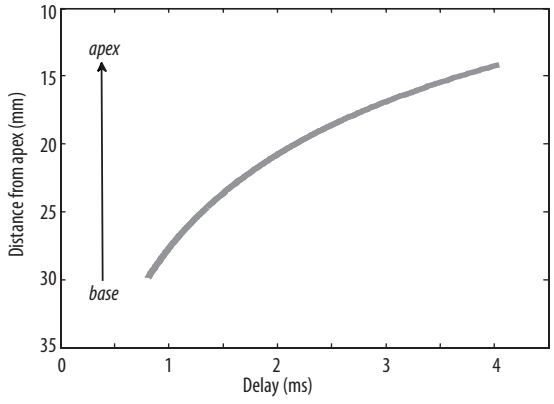

Figure 1. The delay of the cochlear resonators based on their *Q* values (from [8]). The *Q* values are derived from Shera et al. (2002) [29] and the distances are based on the Greenwood frequency –place map [30].

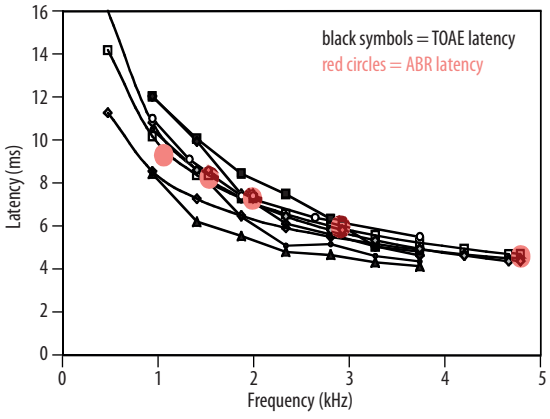

Figure 2. Matching of ABR latencies and transient OAE latencies at low sound levels (data from [32] and [33]). The ratios between the two latencies can vary depending on sound level, but the ratio approaches 1 at 20 dB SPL. This equality of delays suggests the two measures might reflect one and the same mechanism (perhaps a common resonating system).

does not happen in reptiles and amphibians, the hypothesis made here is that the fast pressure wave is in fact the effective stimulus in all ears, reptilian and mammalian. The fast wave enters the cochlea as a result of the stapes pressing on the confined fluids of the cochlea (the controlling factor then being the degree of compliance of the round window membrane), and the "common mode pressure" so generated might be detectable if the hair cells themselves embodied some pressure-sensing mechanism. That is, although the hair cells show projecting stereocilia, the cells might also, *if they contained a compressible element*, be able to react to the pressure wave as it passed through the cochlear fluids at the speed of sound in water [23–25]. The purpose of the stereocilia may then be to detect ripples generated locally and provide feedback to the system.

In this alternative to the conventional picture, the fast pressure wave signal causes *standing wave resonance* between rows of outer hair cells, creating a set of nodes and antinodes, in many ways like its electronic equivalent, the surface acoustic wave (SAW) resonator [10,11,20,26]. In this SAW model, which relies on the slow propagation and cycle-by-cycle feedback of fluid–structure waves, outer hair cells themselves are stimulated by the fast pressure wave (and also by feedback, via their stereocilia, from OHCs in neighbouring rows). Reciprocally, otoacoustic energy also propagates away from the outer hair cells via a fast pressure wave. That is, as the resonance grows cycle by cycle (due to positive feedback), a build-up in the associated otoacoustic emission occurs, which simply reflects the state of the oscillating volume of the active OHCs. This new perspective sees the cochlear amplifier as a device which sets up a standing wave between the rows of OHCs, and this standing wave can be observed almost instantaneously as an otoacoustic emission in the ear canal. In this picture, the individual resonating elements in the cochlea are both local and observable, just like hearing a plucked guitar string or "underwater piano" [20], an interpretation that means otoacoustic emissions give a direct, non-delayed window into cochlear mechanics.

Furthermore, while OHCs generate the actual otoacoustic emissions, the basilar membrane on which they sit simply moves in reaction to them, integrating the motion of the resonating elements and providing a broad delayed envelope, the peak of which apparently "moves" along the basilar membrane [8]. Thus, although the basilar membrane is, by itself, a low-*Q* structure, it can show many cycles of delay, and can appear to be as sharply tuned as the auditory nerve [27] because of the active resonators that sit upon it.

Naturally, confirmation of this resonance model of the cochlea is needed, but as the calculations in [8] show, this alternative view could be worth exploring. Simple resonance not only explains how a travelling wave is generated in the cochlea, but also supplies an explanation of why a "reverse travelling wave" [28] cannot occur. In the resonance model, a travelling wave arises only because delay increases from base to apex (Figure 1), and therefore, in a uniformly tonotopic cochlea, a reverse travelling wave, with delay increasing in the opposite direction, is theoretically impossible.

To explain more fully, it helps to see that the positive gradient seen in Figure 1 equates to a negative gradient in delay from low frequency (apex) to high frequency (base). In more familiar terms, this corresponds to the typical negative phase slope (NPS) seen in otoacoustic emissions [31], and simply requires that resonator group delays decrease with frequency [i.e., $(Q_1/f_1) > (Q_2/f_2)$, where Q_1 is the quality factor at f_1 and Q_2 is that at f_2]. This is usually the case because *Q* changes more slowly than frequency – for example, ref. [29] finds that $Q=12.7\ (f/1000)^{0.3}$ – and this offers a simpler explanation of NPS than that offered by Sisto et al. (p.3142 of [31]), which calls on backward travelling waves. Their argument that OAE latencies are too long for fast pressure wave involvement can be countered by questioning some basic assumptions. For example, the factor of 2 between ABR measures and OAE measures can be accounted for by noting that the ABR is generated by the *onset* of the stimulus whereas OAE delays are referenced to the center of the stimulus (its group delay) (p. 655 of [32]). In confirmation of this, when the *longest*

set of delays in Figure 2 of ref. [32] are plotted (i.e., those from the 20 dB ABR stimulus when the system is pushed to its limit and must use the full length of the signal in order to detect it), the ABR delays line up in the middle of the range of OAE delays (Figure 1 of [33]). This match is displayed in Figure 2, where it is seen that the ABR/OAE ratio is in this case 1, not 2. The unitary ratio is reinforced by the calculations of group delays in the cochlea and the absence of appreciable front delays [8].

The reverse travelling wave is central to the theory of coherent reflection filtering (CRF) [28]. In this widely accepted theory [34], reverse travelling waves are required to sustain a reverberant loop that takes energy back from the travelling wave to the stapes, at which place it generates another forward travelling wave. This reverse wave is elusive, and a number of researchers have failed to detect its presence [14,35], although others claim evidence for it [18,36]. This is the reason some researchers have therefore proposed that otoacoustic energy detected in the ear canal emerges via a fast pressure wave, not a reverse travelling wave [37]. However at the present time the fast pressure wave proposal has gathered little support [31,38–40]. The issue is complex [41] and the evidence for and against is not elaborated here; however, given that there seems to be a large number of uncertainties and the issues surrounding it do not seem to have been settled, the question is perhaps best left open.

At the same time, it is worth noting that recently some workers have appeared to place less reliance on travelling waves as causal entities. These investigators have shown how an array of critical oscillators could supplement, or perhaps even replace, the tuning due to a travelling wave on the basilar membrane [42,43]. The ratchet model of Reichenbach and Hudspeth [42] posits that hair-bundle resonance could be more effective than motion of the basilar membrane itself, and in fact the latter is just a stimulus "without strong variation in amplitude, wavelength, and velocity" (ref. [42], p. 4974) meant to stimulate a more finely tuned system. More recently, these workers have shifted their focus from the basilar membrane to Reissner's membrane [44].

A model of the cochlea as a surface acoustic wave resonator

In order to demonstrate that a resonance model of hearing is theoretically possible, a description of a candidate for the resonating elements has been given in previous work where a model is used that does not rely on the basilar membrane and pressure differences across it [10,11,25,26]. In this model the outer hair cells are assumed to be pressure sensitive, and each triplet of outer hair cells, one from each row, forms a positive feedback loop, creating a full-wavelength standing wave between the rows which increases in amplitude cycle by cycle (Figure 3). The fundamentals of resonance mean that amplification to the half-power criterion only appears after Q/π cycles (so if Q is 12, for example, the signal needs to circulate through the loop about 4 times). It is possible that this local back-and-forth reverberation could form the basis of the cochlear amplifier.

In summary, the input could be a pressure wave acting on the body of the outer hair cells and the output could then be deflection of the inner hair cell stereocilia. The three

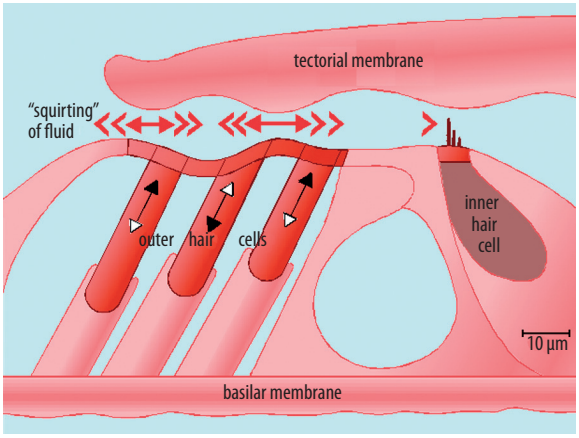

Figure 3. A candidate for the human cochlea's resonating elements. The model involves a triplet of outer hair cells and to-and-fro motion of fluid in the subtectorial space. In response to a pressure stimulus the outer hair cells change length, with the middle row moving in antiphase to the flanking rows (black and white arrowheads). This causes pumping of fluid, bending of stereocilia, and creation of squirting waves (red arrows) in the gap. The amplified fluid motion is directly communicated to the inner hair cells (red arrowhead at right). For clarity, the waves are greatly exaggerated. Adapted from [11] and used with permission of the Acoustical Society of America.

rows of OHCs, which are in continuous hydraulic connection with the stapes, are assumed to react to oscillating pressure in the cochlear fluids. In analogy with SAW devices, the polarity of response of the middle row of OHCs is assumed to be in antiphase to the surrounding rows, an analogy described schematically in Figure 4. The resulting motion causes pumping of fluid and "squirting waves" [11] in the subtectorial space which deflect stereocilia in neighbouring rows. This creates a positive feedback loop, the end result of which is a sharply tuned standing wave between the rows. The amplified signal is finally communicated to the inner hair cells via fluid motion.

Standing waves in the lizard cochlea

It is remarkable that the hair cells in the lizard cochlea often lie in two distinct regions, separated by a region of demarcation called the striola, each with opposite "polarity" – meaning that each region is occupied by cells that face 180° to the opposing set (see Figure 5). Thus, Wever [6] notes that in all skinks studied he found the ciliary tufts were "bidirectional, and very regularly so", and that the cells faced each other "with the dividing line usually in the middle of the array" (p.640).

If stereocilia face each other in this way it means that when a shearing motion is applied to the overlying tectorial membrane, each set will be receive either an excitatory or inhibitory stimulus. Given the coupling of the cells via the tectorial membrane, in which a slow shearing wave of some form is expected to occur, this arrangement is one that will again allow a whole-wavelength standing wave to form across the dividing line**.** This standing wave resembles

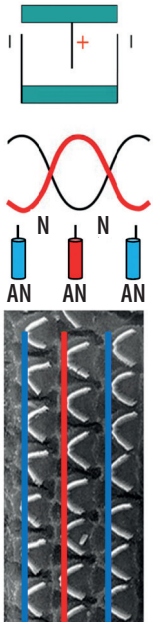

Figure 4. The parallel between the interdigital fingers of a surface acoustic resonator device (top) and the three rows of outer hair cells (bottom). Standing waves form between the fingers (middle) with a set of nodes (N) and antinodes (AN).

those in an organ pipe open at both ends. In cases where all the hair cells face in the one direction, which is usually the case at low frequencies, induced wave motion could be reflected at the edge of the tectorial membrane and again cause a standing wave, this time like an organ pipe open at one end and closed at the other.

How to detect a fast wave

If the idea of fast waves in the cochlea is to be sustained, then there has to be a physical mechanism by which these waves are detected. Essentially, a fast pressure wave is a variation in hydraulic pressure, so this means looking for a type of pressure sensor. Here, piezoelectric properties may hold the key, and this idea has been put forward in different contexts [45–49]. Sometimes researchers mean electrical sensitivity to differential pressure; other investigators mean a response to common mode pressure.

However, if high transduction efficiency is the key, then having a highly compressible material, perhaps even an air bubble, residing at the sensing point would provide a particularly effective solution: the stimulus energy would then naturally travel from the source – the stapes – through the incompressible cochlear fluids and be deposited at the compressible element (where sensing occurs). In this context, there is Bekesy's demonstration [23,50] of what happens within a confined volume of fluid subject to oscillating pressure. He inserted his finger into a capsule of paraffin that was subject to the effects of a vibrator, and when he touched a small piece of foam rubber immersed within it he reported feeling a sensation of "strong vibrations" (p. 424 of [50]). A possibility is that the outer hair cells are pressure sensors and that Hensens bodies are

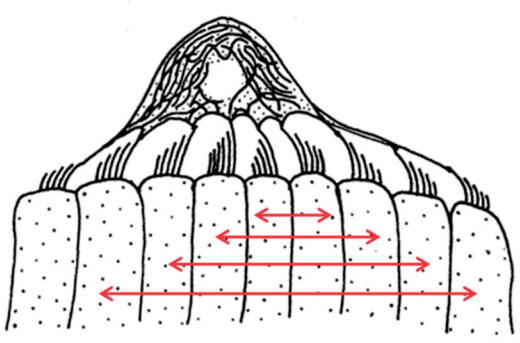

Figure 5. An example of how hair cells in the lizard ear are arranged bidirectionally, so that stereociliary bundles on the left face in an opposite direction to those on the right. In this arrangement, waves in the gelatinous sallet could propagate to and fro (arrows) and give rise to a whole-wavelength standing wave, like in an organ pipe open at both ends. The shortest arrows support the highest frequencies; the longest support the lowest frequencies. Adapted from Figure 4-19 of [6] and reproduced with permission of Princeton University Press.

compressible elements within them [23,25]. On grounds of comparative anatomy, a similar proposal has been made that the macula neglecta of sharks embodies a similar arrangement of pressure sensors [23].

Returning to reptiles, there is a remarkable observation reported in an electron microscopic study of the gecko cochlea [51]. The authors took micrographs of the sensing cells and found that the cells contained "vacuoles" (Figure 6). The idea raised here is that these vacuoles could in fact be the locus of the cells' compressibility, so that the cells detect sound pressure by sensing the change in internal cellular volume caused by pressure [24]. Vacuoles are hard to study because of fixation difficulties, but it is noteworthy that similar vacuoles have been seen in the gas gland cells of fish swim bladders [52]. The general hypothesis is that pressure detection occurs via compression of intracellular spaces.

The papilla of some lizard species is surmounted not only by a tectorial sallet, but also by a peculiar blob called a culmen, a sponge-like structure (Figure 7) that Wever [6] describes as having a "frothy appearance" (p. 105) and being endowed with "numerous vacuities both large and small" (p. 664). Such a sponge-like material is an ideal candidate for compressibility. It is also in just the right location for picking up vibrations and passing them to the stereocilia immediately beneath.

A model for a pressure wave detector and amplifier

It is strange that the otic capsule of the lizard sometimes shows a "supernumerary papilla" (Figure 25.1 of [6]), a perfectly formed arrangement of sensory cells which sits on the solid limbus and is covered with tectorial membrane (Figure 8). Remarkably, this peculiar structure is not innervated, lacking both afferents and efferents, in which case it is apparently functionless, as Wever thought. Or is it?

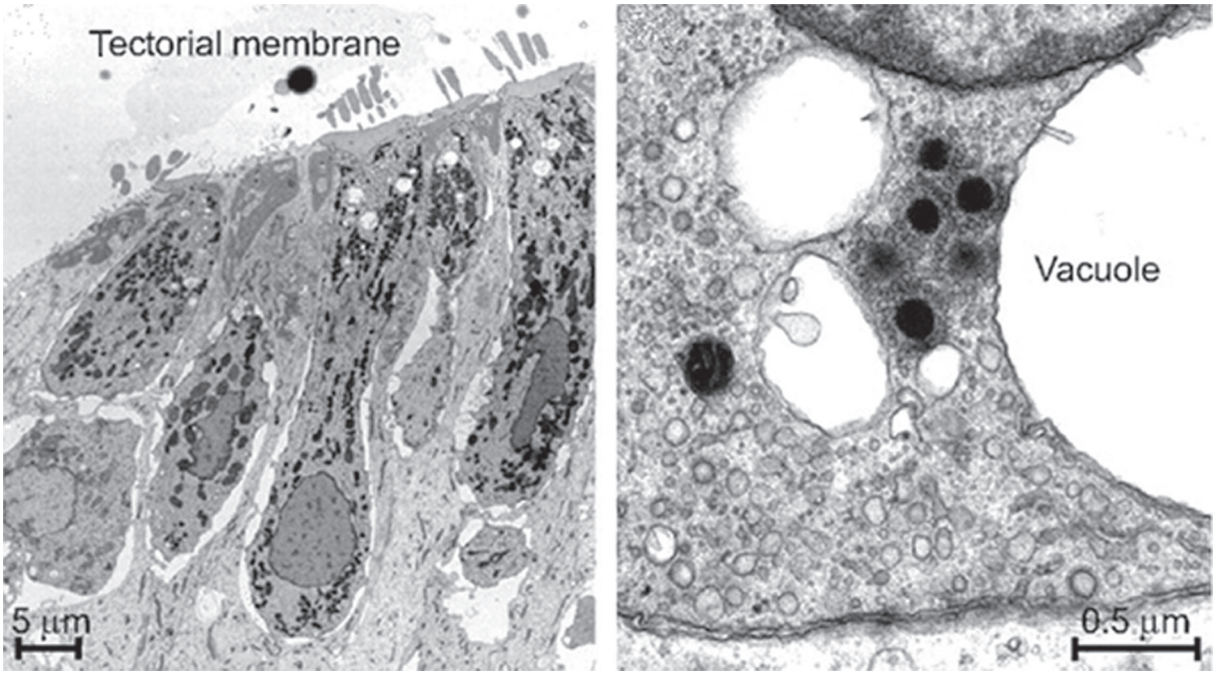

Figure 6. Vacuoles in the hair cells of the gecko, which could be compressible elements that change volume in response to pressure, leading to opening of ion channels in the wall of the cell. From [51] and used with the permission of the authors.

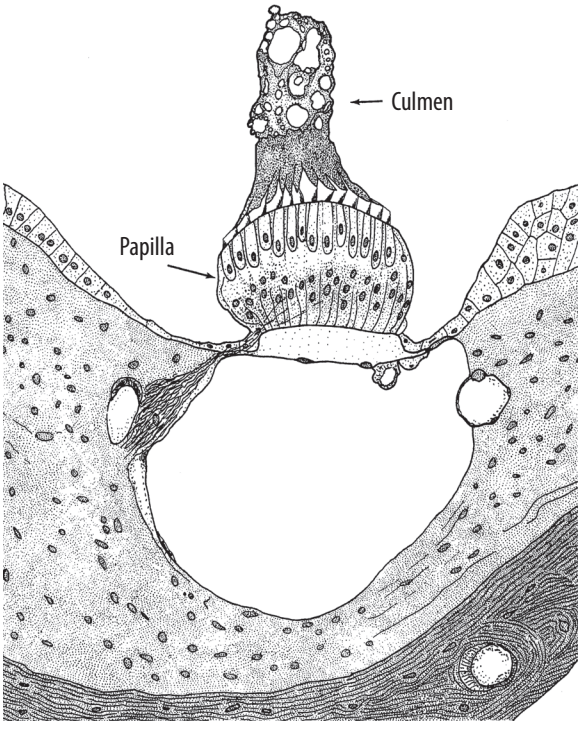

Figure 7. The papilla of the girdle-tailed lizard topped with a sponge-like structure called a culmen (Figure 19-7 of [6]). The frothy nature of the culmen, if it contained air or other compressible material, makes it an ideal pressure receptor. Note that hair cells on either side of the sallet often exhibit opposite spatial polarity (see Figure 5 and other examples in [6]). Reproduced with permission from Princeton University Press.

The gecko paper mentioned previously [51] is noteworthy for making a nice distinction between cells that are largely innervated by afferents and cells that are largely innervated by efferents. The first group are described as belonging to a class of transducers relying on direct transduction by stereocilia; the second class belong to a set of feedback amplifiers that increase auditory sensitivity. This is a useful distinction, but then the case of the supernumary papilla presents a remarkable paradox.

What could be the function of such an isolated collection of sensing cells sitting on an inflexible foundation? Wever dismisses the supernumary papilla as an aberration, with no functional utility. But if the pressure detection idea is taken seriously, a clear function becomes apparent: the cells could comprise a *pressure preamplifier* in which they both detect intracochlear pressure (using some compressible material) and *amplify* it. In brief, the idea is that, because of tectorial membrane coupling, there will be positive feedback carried to and fro between the facing cells of opposite polarity. The analogy can be made to a swing given a push at each end of its travel so that it progressively builds up amplitude.

In more detail, consider what might happen if the individual cells were to react electrophysiologically to a small pressure signal (small red arrow in Figure 8). A positive feedback mechanism between the coupled cells could then lead to pressure amplification. Positive feedback would cause increased oscillation in the volume of the vacuoles, which are presumed to contain air or other compressible fluid. In turn, these pressure changes would be coupled by the incompressible cochlear fluids directly to the normal papilla (large red arrow), where a similar compressibility-mediated detection scheme might operate.

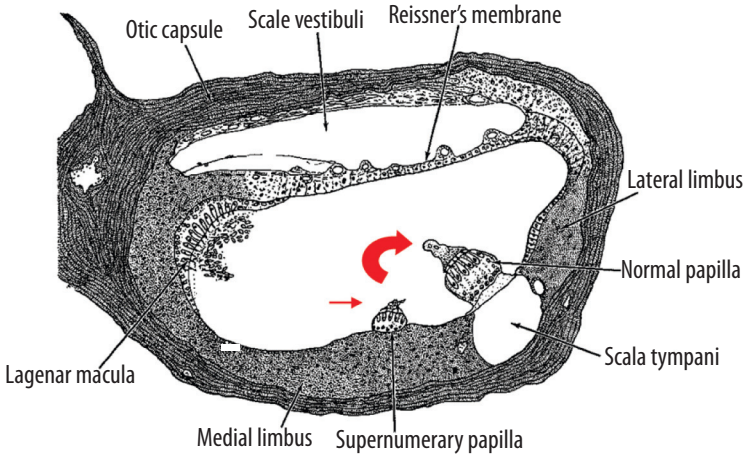

Figure 8. Section through the otic capsule of a lizard showing a "supernumerary papilla" which lacks both afferents and efferents. Although it appears functionless, it could, as explained in the text, form a preamplifier of pressure that is then conveyed to the normal papilla through the surrounding incompressible fluid. Figure 25-1 of [6] and reproduced with permission of Princeton University Press.

In summary, the supernumerary papilla might be considered a pressure preamplifier for the normal papilla. The normal papilla senses the amplified pressure via the pressure-to-displacement converter of its compressible culmen. Otoacoustic emissions, in lizards and perhaps humans, can then be seen as pressure oscillations communicated through the fluids and which leak out through the stapedial footplate and round window. This pressure-detection scheme also explains why some of the reptiles (turtles) examined by Wever did not have the papilla sitting upon the basilar membrane or fundus, but on the solid base of the limbus (p. 110, p. 853).

This reptilian arrangement is a further example of the SAW mechanism, and it operates in a similar way to the mammalian cochlear amplifier outlined above. It is an arrangement that again meets the description of a resonant system driven by a fast pressure wave, and it operates along the same lines as Gold's regenerative receiver [53,54]. It is interesting to note that outer hair cells largely lack afferents, although they do have a rich efferent supply [55], so they are well placed to act as gain-controlled receivers of acoustic energy propagating through the cochlear fluids.

Finally, it is worth noting several other schemes in which useful fine-tuning properties emerge when sensing cells cooperate. Barral and colleagues coupled a hair cell from a bullfrog to two electronic (virtual) hair bundles and found that this increased vibration sensitivity [56]. More generally, they went on to suggest that the harnessing outer hair cells together (via the tectorial membrane) could provide a way of boosting hearing sensitivity in mammals. The work of Gelfand et al. [57] involved simulating elastic connections between adjacent hair cells in the gecko's cochlea; this produced a comb-like frequency response that resembled the animal's actual spontaneous otoacoustic emission spectra. When considering how the coupling between cells might be achieved, it is notable that multiple resonances resulting from an arrangement like that in Figure 5 would also give rise to a comb-like spectrum. Incidentally, there are lizard species that lack tectorial membranes [58], but in this case coupling might be achieved via fluid viscosity.

Conclusions

This paper has explored the possibility that the ears of reptiles and mammals operate along similar lines. The prime stimulus could be a fast pressure wave and resonance might occur in defined positive feedback loops between active hair cells. The general principle is that the spacing of the hair cells sets the resonance frequency of the loop. In this way there seems to be a remarkable convergence of structure and function, and this should encourage further investigation of the intricate anatomy evident in the literature, particularly the fine tectorial membrane structures revealed by Wever [6] in reptiles. In the case of humans, the spacing between outer hair cells might again define the resonance frequency, suggesting that otoacoustic emissions open a direct window into local hair cell interactions.

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