CHAPTER D 8 (Discussion)

The cochlea and its response to pressure

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In order to provide a resonance model of the cochlea, this thesis has been postulating simultaneous (parallel) excitation of the cochlear resonators. As set out in Chapter I1, this translates to a requirement that the resonant elements react to the fast pressure wave (common-mode or hydrostatic pressure) rather than to differential pressure. Chapter I3 sought to show that the traveling wave fails to give a full account of cochlear mechanics – there are anomalies, and most of them appear to centre around extremely fast responses. In this context, it seems a reasonable hypothesis to assume that the compressional wave is the missing element and that it is in fact a major force in the mechanics of the active cochlea. This chapter investigates the hypothesis.

Chapter R5 has proposed that the resonant elements are the regular rows of outer hair cells coupled by squirting waves. In order for these elements to be simultaneously excited, we require that outer hair cells react directly to intracochlear pressure. Expressed simply, the hypothesis amounts to OHCs being pressure sensors.

The sequence of this chapter is first to look for evidence that the ear reacts to common-mode pressure. If it does, this leads to the corollary that, like all pressure sensors, it must display some compressibility, and evidence is assembled to show that indeed this is the case. A functional model for the pressure sensor is put forward, and an examination made of how it could operate within a cochlea possessing a large 'hole': a compliant round window.

A starting point is to consider some underwater acoustics and recognise that the problem of detecting pressure in the cochlear liquids is the same as that confronting some marine creatures in detecting underwater sound. The answer, as found by a number of aquatic animals, is to use compressible materials in order to transform pressure signals into displacements. This analogy leads us to suspect that mammalian outer hair cells also use this strategy. The hypothesis is therefore made that their characteristic Hensen bodies (and perhaps the accompanying cisternal system) enclose compressible material, most likely air, just like cells in the macula neglecta of sharks appear to do. Finally, we propose how compression of the cell leads to its excitation. The hypothesis is made that the kinocilium (and its degenerate form, the cuticular pore) are important in this process, as they contain the actual sensory apparatus: the basal body. In the light of this model, some confirmatory evidence is presented.

8.1 Evidence that the ear detects common-mode pressure

The unifying idea is that the compressional wave is the major stimulus for outer hair cells (at least at sound pressure levels below about 60 dB SPL) and that their excitation causes a squirting wave which drives the sharply tuned elements of a SAW resonator. It is proposed that outer hair cells can detect pressure because they are compressible, and their excitation leads in turn to volume changes, and pressure waves, that are detectable in the ear canal. The effect of the excitation of a bank of highly tuned resonators is, of course, an *apparent* traveling wave (§I 1.5), and it is a mistake to consider this traveling wave as having causal properties. The traveling wave produced by outer hair cell excitation might just be an epiphenomenon.

It is possible our understanding of the cochlea went awry when the common-mode pressure, p_+, was seen to be of no consequence and discarded. Just because outer hair cells bear stereocilia is not sufficient reason to ignore their potential pressure-detection capability. According to this thesis, outer hair cell stereocilia are primarily feedback devices, not sensors (although a sensory function is exhibited at high sound levels).

In this section, we will see how an active (low-level) cochlear mechanics can be constructed in which common-mode pressure takes the principal role.

8.1/a Cochlear sensitivity to pressure

The idea that hearing might result from direct detection of sound has had a long history. In the 20th century the idea was considered by Pohlman in 1933 who reviewed various opposing theories at the time[1] and was not averse to the idea that "the auditory cells react directly to the vibrations in the liquid in which they are immersed" [p. 193] rather than to transverse vibrations in the cochlear duct. The

[1] Pohlman, A. G. (1933). A reconsideration of the mechanics of the auditory apparatus. *J. Laryngol. Otol.* 48: 156-195.

advantage of this "piezo-electrical" concept, he said, is that it simplifies matters enormously and does not confuse the issue by employing mechanical structures extrinsic to the auditory cells. He cites literature that accords with this concept, some of which, like human temporal bone studies, we have already referred to (§I 3.2/e).

In the previous chapter we referred to the insights of Guild (1937), who was the first to suggest, albeit diffidently, that the outer hair cells may be directly stimulated by sound[2]. He made the leap (p. 370) from the purely anatomical observation that outer hair cells are surrounded by large fluid-filled spaces – the spaces of Nuel – to the physical interpretation that this is an ideal arrangement for detecting pressure changes in the cochlear fluids. In recent times, the peculiarity of the spaces of Nuel have also been noted, and in a popular review[3] Brownell reminds us that in no other organ of the body does one find cells surrounded on all sides by large fluid spaces. Other cells, inner hair cells included, are surrounded by supporting cells. Brownell, however, presents a different interpretation: the spaces are there to permit the outer hair cells to change length – which of course is equally true, but misses a possibility I wish to propose.

Davis and colleagues in 1934 addressed the pressure-sensitivity question when considering the origin of the cochlear microphonic[4]. They suggested that the cochlear microphonic arose from the body of hair cells being squeezed. "We venture the hypothesis that the electrical potential arises from the sensory cells themselves as a result of mechanical deformation … [and that] the difference of potential is developed between the upper and lower ends of these cells." [p. 329]. Crucially, though, they thought that the out-of-phase responses of the oval and round windows "definitely rules out the possibility that the potential is generated merely by increased or diminished pressure within the cavity of the inner ear" [loc. cit.].

The underlying idea is that if the cochlear contents are incompressible and the windows move out of phase in a shuttle-like motion, then differential pressure must be the way by which the cochlea is excited. What is wrong with that? The sections below attempt to uncover a flaw in that thinking.

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[2] Guild, S. R. (1937). Comments on the physiology of hearing and the anatomy of the inner ear. *Laryngoscope* 47: 365-372.
[3] Brownell, W. E. (1998). How the ear works: Nature's solutions for listening. *Volta Review* 99: 9–28. §12: The Organ of Corti – The Temple of Hearing.
[4] Davis, H., et al. (1934). The electric response of the cochlea. *Am. J. Physiol.* 107: 311-332.

8.1/b Phase of the two windows and compressibility of the cochlea

If the cochlea were a detector of common-mode pressure, then it would have to exhibit some compressibility. This is because any hydrophone needs to have some compressibility in order to react physically to the pressure and convert energy from one form (acoustic) into another (electrical). For optimum signal-to-noise ratio, that conversion needs to done efficiently, with well-matched impedances. One way of measuring the compressibility of the cochlea is to measure the relative phases – or better, the volume velocities – of the oval and round windows. If the volume velocities are equal and the windows move precisely out of phase, then the cochlea is incompressible. If so, the inference is that it cannot be a hydrophone, rather it is simply responding to shear of the partition generated by differential forces.

Experiments have attempted to show that differential pressure is the effective stimulus in the cochlea in two ways. The first is to directly measure the phase of motion of the two windows. The second is to apply stimuli to the two windows precisely in phase and show that the cochlea fails to respond (electrically) to this condition.

1. In Wever and Lawrence (1950), a paper[5] alluded to earlier (§D 3.1/b), sounds of independently adjustable phase were conducted (via two rubber tubes) to each of the round and oval windows of cats and, to gauge cochlear responses, an electrode picked up cochlear microphonics. They sought to dispel an 'alternative' view of cochlear mechanics – which they regarded not as a popular one but which in the absence of evidence had to be entertained as a possibility – in which the basilar membrane only served as a suspension for the hair cells and cochlear fluid pressure served as the stimulus. On this view, maximum stimulation would occur when the sounds to the two windows are in phase. Significantly, they found that the electrode response was near a minimum at this condition. Common-mode pressure did not appear to be an effective stimulus.

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[5] Wever, E. G. and M. Lawrence (1950). The acoustic pathways to the cochlea. *J. Acoust. Soc. Am.* 22: 460-467.

D 8 [6]

A major limitation[6] of this experiment now needs to be made plain: Wever and Lawrence used sound intensities well above 60 dB SPL. Intensities were not calibrated, but they set levels to give a cochlear microphonic of 10 μV, which corresponds to about 65–70 dB SPL. It is possible that at low intensities the windows moved in phase and the majority of the electrode response would then derive from the common mode signal (which drives the cochlear amplifier and which saturates at 60–80 dB SPL).

2. Offutt later[7,8] tried to replicate the Wever and Lawrence work in chinchillas and provided an interesting new dimension to the story. He measured the cochlear microphonic due to two phase-adjustable sound sources: one in the ear canal and another through a hole drilled in the cochlea[9]. He found that at times when the cochlear microphonic due to the two tone-pips was nulled, the compound action potential – the electrical signature from the acoustic nerve – reached a *maximum*. A major limitation of this work was that, again, sound stimuli were not calibrated and drilling a hole in the cochlea is likely to have disturbed the natural situation (see §D 10.1/d). Nevertheless, given a lack of data on the question, the work deserves some consideration.

3. Kringlebotn (1995) measured the volume displacements through the round and oval windows in 34 isolated inner ears of pigs and one human temporal bone[10]. The volume displacements for the pigs were "within 1 dB"[11] of being equal, whereas for the human specimen, the round window displacement was some 3 dB *smaller* than the oval window's. Inferred ratios, derived from related measurements on 20 other human temporal bones, also gave a comparable figure of 3.4 dB. Although his text stresses the *equality* of the two displacements, given the large variance in the

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[6] Another limitation is that at low frequencies – a few hundred hertz – minimum response differed considerably from the in-phase condition. According to their Fig. 5, phase offsets reached about 45° at these frequencies, meaning that a secondary response may be contributing at only several decibels below the main one. However, not too much store can be taken by these phase anomalies since it is unrealistic to expect the round window and oval window to contribute identical sound pressure.
[7] Offutt, G. (1984). *The Electromodel of the Auditory System*. (GoLo Press: Shepherdstown, WV). [pp. 162-169]
[8] Offutt, G. (1986). Wever and Lawrence revisited: effects of nulling basilar membrane movement on concomitant whole-nerve action potential. *J. Aud. Res.* 26: 43-54.
[9] ibid., Appendix 1, p. 162-169.
[10] Kringlebotn, M. (1995). The equality of volume displacements in the inner ear windows. *J. Acoust. Soc. Am.* 98: 192-196.
[11] Strictly, it is incorrect to use decibels when powers are not involved. I presume he means "within 10%".

measurements this conclusion cannot be strongly sustained[12]. He acknowledges (p. 195) that the deviation admits for a compliance[13] in the inner ear, but then only 0.2 dB inequality was sufficient for Shera and Zweig (1992) to explain the puzzling residual hearing in Békésy's "middleless" ears (§D 9.3/e). Calculations by Ravicz et al. (1996) show that Kringlebotn's inequality translates to a large inner ear compressibility: a figure for α (the ratio of stapes motion with the round window blocked to normal stapes motion) of 0.3. Perhaps it is obvious, but the comment needs to be made that these experiments are only relevant to the compressibility of dead cochleas.

4. Voss et al. (1996) approached the problem[14] by bringing modern technology to bear on the Wever and Lawrence experiments. They applied two independent sounds of controlled amplitude and phase to the round window and oval window of cats and looked at the effect on the cochlear microphonic. They expressed their results in terms of common-mode rejection ratio (CMRR) – the ratio between the cochlear microphonic in response to the out-of-phase condition compared to the response in the in-phase condition. They found that the minimum CMRR (for up to seven cats) ranged from 15 to 28 dB, while the maximum was 22 to 78 dB. Before one concludes that the results show responses to common-mode pressure can be neglected, the following factors need to be appreciated.

(i) Despite the findings of Offutt that the compound action potential (CAP), generated by the auditory nerve, could behave differently to the cochlear microphonic (CM), generated by the outer hair cells, the researchers applied tetrodotoxin (TTX) to most of the cats to eliminate CAP. This was meant to get rid of possible interference with the CM, which they focused on. While that no doubt happened, perhaps they were also eliminating a physiologically important signal. In this context, we learn (p. 1605) that TTX eliminates voltage-gated sodium channels, and these could well be the channels that are involved in generating a response between the outer hair cell rows – that is, the pressure-related signal which drives the

[12] He also quotes (p. 192) data from Nedzelnitsky (a 1974 thesis), who found that the volume flows were only equal to within ±10 dB.
[13] He uses the adjective 'small', but that is inappropriate.
[14] Voss, S. E., et al. (1996). Is the pressure difference between the oval and round windows the effective acoustic stimulus for the cochlea? *J. Acoust. Soc. Am.* 100: 1602-1616.

cochlear amplifier (see §D 9.1/j). In partial confirmation of this, we see that the CMRR values become perhaps 5 dB higher after TTX administration (their Fig. 9).

(ii) The sound pressure levels used by the experimenters were high, largely in the range 60–120 dB. Of particular interest, the linearity of the CMs (amplitude v. sound intensity) disappears and becomes quite erratic below 80 dB (their Fig. 4). This is especially noticeable for animals before TTX treatment.

My conclusion is that the Voss et al. experiments do not rule out the possibility that the ear is sensitive to common-mode pressure below about 60 dB. That is, that the cochlea is pressure-sensitive at low levels, but the sensitivity saturates at about 60 dB SPL and becomes swamped by displacement sensitivity at high levels.

5. As mentioned previously (§I 3.2/g), Ravicz et al. (1996) measured the compressibility of the cochlea of cadavers and expressed it in terms of α, the ratio between the motion of the stapes with the round window blocked to normal stapes motion. If $\alpha = 0$, then the cochlea is incompressible and no stapes motion is possible when the round window is blocked; if $\alpha = 1$, the contents are so compressible that stapes motion produces no round window motion. Values of α found by their work, and applied to the work of others, form a useful comparison, and are shown in the table below.

author	value of α	comments
Ravicz et al. (1996)	0.015–0.5	Human cadavers. Upper bound is within an order of magnitude of the compressibility of water
Shera and Zweig (1992)	0.04	Based on hearing thresholds of live humans without middle ear as measured by Békésy (1960)
Kringlebotn (1995)	0.3	Isolated temporal bones of human cadaver and pigs
Tonndorf and Tabor (1962)	0.1	Blocked round window of live cats
Harrison et al. (1964)	Not specified, but between 0.015 and 0.5	Patient with congenitally blocked round window. Hearing improved by 20–40 dB when surgically corrected

The main point to be derived from this comparison is that it only requires minute compressibility to allow a significant amount of hearing to develop (even with a poor matching of sound input to the cochlea – a missing middle ear or blocked round window). The input impedance of the cochlea is large (more than 20 MΩ[15]), and consequently the volume changes we are trying to detect are minute. So the cochlear fluids may be shuttling back and forth at *close to* 180° to each other, but at the same time the primary input to the cochlea may be the common-mode pressure generated by stapes motion. The other thing to notice is that according to the transduction model presented in this thesis, live cochleas will have a different compressibility to dead ones. Anomalies in the input impedance of the live cochlea suggest this is the case (see following section).

5. Stenfelt and colleagues examined the vibration of the temporal bones of human cadavers[16] and found that, with stimulation with air-borne sound, the volume displacement of the round window was comparable with, but generally less than, that of the oval window (some decibels less) and the phase about 180° (±20° depending on frequency). Particularly interesting was their finding[17] that the round window only moved as a whole below 1.5 kHz; at higher frequencies the vibration was multimodal, with portions moving in at the same time as others moved out. Clearly, the motion of the round window is complex, even in dead cochleas, and some compressibility of the *live* cochlear contents is compatible with current measurements (made either at high sound pressure levels or on dead specimens).

8.1/c Input impedance of the live cochlea

The input impedance of the live human ear differs from that of a non-living one[18], and there are a number of reasons that may account for that. One factor I

[15] Aibara, R., et al. (2001). Human middle-ear sound transfer function and cochlear input impedance. *Hear. Res.* 152: 100–109.
[16] Stenfelt, S., et al. (2004). Round window membrane motion with air conduction and bone conduction stimulation. *Hear. Res.* 198: 10-24.
[17] Stenfelt, S., et al. (2004). Fluid volume displacement at the oval and round windows with air and bone conduction stimulation. *J. Acoust. Soc. Am.* 115: 797-812.
[18] Goode, R. L., et al. (1993). Measurement of umbo vibration in human subjects: method and possible clinical applications. *Am. J. Otol.* 14: 247-251. See also Zwislocki (2002), Fig. 3.35.

would like to suggest is a difference in compressibility. The measurements are difficult and the scatter of data points is high, but it appears that (Goode et al., Fig. 6) a live cochlea has, beyond a few thousand hertz, a 5–10 dB smaller input impedance than a dead one[19].

In general, the live ear has a negative reactance, behaving[20] at low frequencies as if it were a volume of air of 1.4 cm^3. Beyond a couple of thousand hertz, however, the reactance goes positive (Zwislocki, p. 47), suggesting resonance at the crossover point and a more complicated process. Compliance values rise (p. 50–51) and approach infinite values – just like they would if lossless mass and spring elements were resonating. The associated acoustic resistance is the tell-tale sign: at these frequencies the resistance of the live ear falls to the resistance of air and even approaches *zero* (Zwislocki, p. 47). Zwislocki is puzzled by this result, which contrasts with the measurements on cadavers, and he suggests it is an artefact. However, it is a long-standing result, and is mentioned by Békésy (*EiH*, p. 312). It is well portrayed in Fig. 10 of Zwislocki[21] (1957), where results by a number of different experimenters are plotted on a scale where the resistance of air is unity. Data points converge towards, and even fall below, the unity line. Understanding this apparent paradox may be helped by recognising that measurements of cochlear impedance in frequency bands encompassing spontaneous emissions can return negative values[22].

8.1/d Summary

The orthodox view that the cochlear contents are incompressible is possibly incorrect, and only a small amount of compressibility is needed to allow hearing to happen. The reported experiments on animals are misleading in that they have not

[19] At 10 kHz, the gap can be as large as 17 dB [Fig. 3.35 of Zwislocki, J. J. (2002). *Auditory Sound Transmission: An Autobiographical Perspective*. (Erlbaum: Mahwah, NJ).]
[20] Zwislocki (2002), Ch. 3.
[21] Zwislocki, J. J. (1957). Some measurements of the impedance at the eardrum. *J. Acoust. Soc. Am.* 29: 349-356. See also Kostelijk, P. J. (1950). *Theories of Hearing*. (Universitaire Pers Leiden: Leiden). [pp. 141, 144]
[22] Burns, E. M., et al. (1998). Energy reflectance in the ear canal can exceed unity near spontaneous otoacoustic emission frequencies. *J. Acoust. Soc. Am.* 103: 462-474.

been done at low sound pressure levels where pressure sensitivity is highest; they reflect measurements of passive cochlear mechanics rather than active. A similar comment applies of course to measurements on non-living specimens. Finally, there are doubts that measuring the cochlear microphonic is a suitable test of hearing acuity, and the compound action potential might be a better indicator.

It therefore remains an open question as to whether outer hair cells are pressure sensors and whether intracochlear pressure drives the cochlear amplifier. The natural inclination is to suppose that the active process works with the same stimulus as the passive[23], but I suspect the effective stimulus could be different in each case. In the next section, it is shown how the presence of a compliant round window (a 'hole') is compatible with a system that senses intracochlear pressure.

8.2 The cochlea as a loudspeaker enclosure

At this point, I set out a model of how the pressure wave at the oval window may be coupled to the outer hair cells. Rather than transverse vibration of the basilar membrane, which is the standard picture, the model suggests that it is the compressibility (compliance) of the outer hair cells that form a major input impedance of the cochlea. Thus, the propagating wave in the cochlear fluids, with pressure and displacement components, interacts with a compressible element within the outer hair cell, and it is supposed that it is the *pressure* component that is the key parameter.

The effect of the round window also needs to be considered[24], since at first sight it seems inappropriate to contain the pressure sensors within an enclosure that possesses a large compliant membrane – effectively, a hole. However, consideration of the *mass* of the cochlear fluids allows for the presence of this hole to be accommodated, as we will see.

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[23] Chapter 4 of Zwislocki (2002) is headed "The Cochlea Simplified by Death" and he claims that the fundamental mode of its mechanical operation is retained post mortem, but this is not necessarily true.
[24] Anatomy suggests "compliant capabilities" (p. 44 of Goycoolea, M. V. (1992). The round window membrane under normal and pathological conditions. *Acta Otolaryngol.* 493: 43-55.)

D 8 [12]

To sketch the basic interaction, Fig. 8.1 shows the simple mechanical picture and Fig. 8.2 shows a circuit analog. Encased in bone and filled with 100–200 µL of incompressible water, the input pressure to the cochlea is applied through the oval window. This launches a wave front, with pressure p and particle velocity v, that reverberates around the cochlea and becomes absorbed by the compressible outer hair cells (maroon). Unabsorbed pressure also distends the round window and raises the intracochlear pressure.

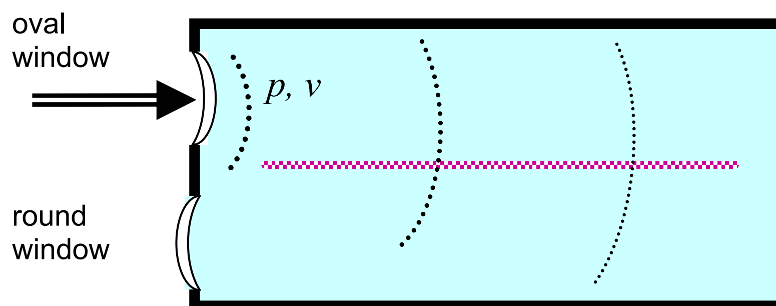

Fig. 8.1. How the pressure signal, launched from the oval window, interacts with compressible outer hair cells (maroon), becoming absorbed as it reverberates around the cochlea. Because of compressibility, the round window volume displacement need not be equal and opposite to that of the oval window. The cochlea is not a box, but a spiral, a shape that tends to prevent the formation of standing waves.

The description is complicated because the pressure sensors – the outer hair cells – are close to the pressure-forming stapes. Hence, the acoustics is near-field and we cannot properly use standard far-field equations. Nevertheless, the propagating pressure signal will encounter compressible elements and undergo many reflections in the spiral cochlea before returning to the stapes, so there should be an appreciable propagating component to the signal. It will not just form a standing wave, although there will inevitably be some. To signify the complicated mixture of propagating and standing waves we use the term propagating *signal*.

To give this propagating signal a physical underpinning, we follow the fundamentals of section 6.2 of Fletcher (1992). If any wave of frequency ω propagates through a medium of density ρ (here considered water) with amplitude ξ, then $v = j\omega\xi$ and $p = j\rho c^2 k \xi$ where c is the phase velocity and k is ω/c. This means

that the wave impedance, p/v, is simply ρc. If we consider the wave to be propagating in a pipe (a simplified cochlea), then its impedance will be $\rho c/A$, where A is its cross-sectional area. The acoustic inductance, L, of a unit length of pipe (associated with its mass) is ρ/A, and its capacitance, C (from its compressibility), is $A/\rho c^2$. Using electrical analogs of these quantities, we draw a circuit diagram, as follows (Fig. 8.2).

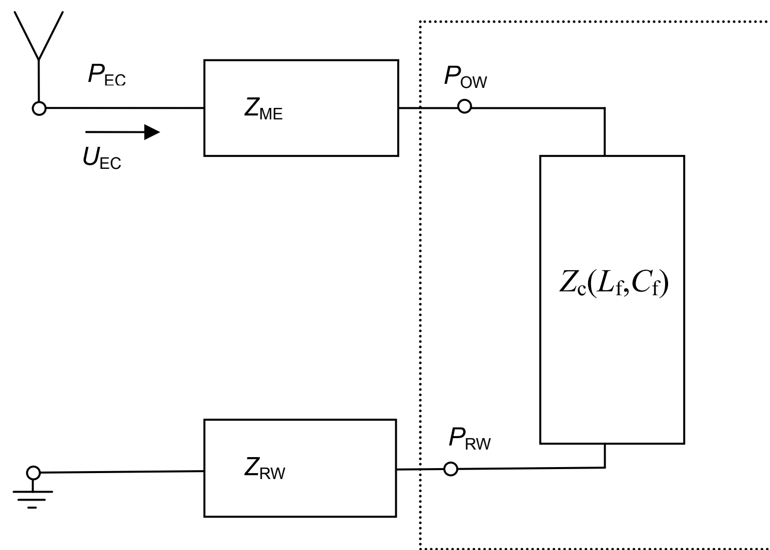

Fig. 8.2. A simplified network model of the cochlea. As well as lumped acoustic impedances, there are distributed elements (within dotted box) associated with the individual outer hair cells and their interaction, through a pressure wave, with the cochlear fluids. The lumped sections comprise outer ear (an antenna); middle ear impedance, Z_{ME} (effectively a transformer); cochlear impedance, Z_C, essentially the compressibility of all the outer hair cells (a compliance); and round window impedance Z_{RW}, a simple compliance. Z_C is comprised of distributed masses and compliances of the cochlear fluids, L_f and C_f, which interact in a complex way as the pressure signal traverses the cochlea through multiple reflections. In essence, the ear-canal pressure P_{EC} (and associated volume velocity U_{EC}) create pressure waves in the cochlear fluids, which are detected by the outer hair cells.

This circuit diagram provides a way of seeing the crucial importance of the mass and compliance of the cochlear fluids in cochlear mechanics. In effect, the system is working together like a reflex loudspeaker enclosure: the oval window is the loudspeaker driver and the round window is the port or drone cone (Fig. 8.3). In such a system, the compressibility of the enclosure interacts with the mass and

compliance of the port, and the system must be tuned for optimum performance[25]. If the resonance is placed at a suitably low frequency, the bass performance of the enclosure can be enhanced, with the radiation from the rear of the driver used to augment[26] the output of the system.

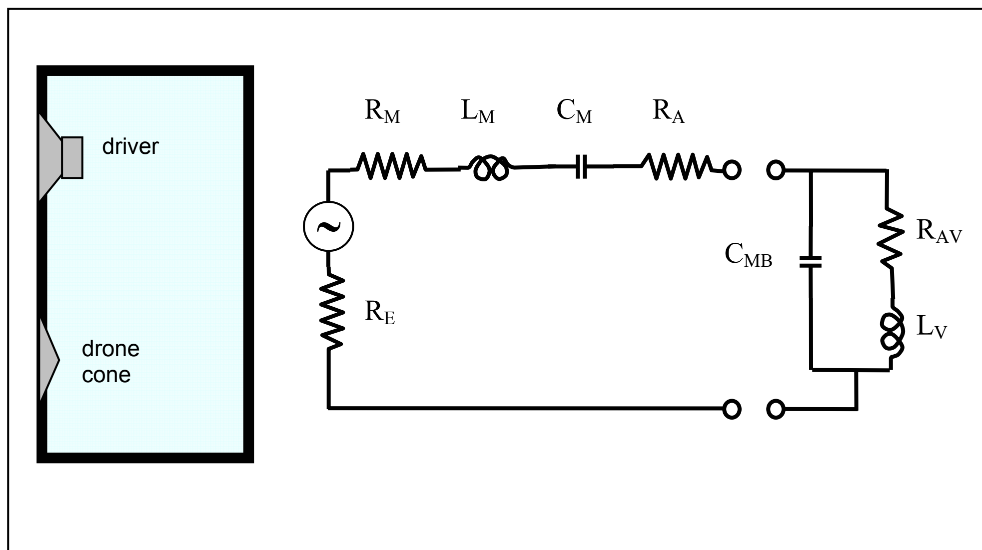

Fig. 8.3. The reflex loudspeaker enclosure. It houses an active driver (top) and a vent (below) which can comprise either a duct or passive driver. The mass of the air in the duct (or of the drone cone) creates a resonance when it bounces against the compliance of the enclosed air, extending the frequency response and output of the system at low frequencies. The circuit impedance of the system, as seen by the air – or a pressure sensor in the box – is given at right, in which the components to the left of the terminals apply to the driver and those to the far right to the enclosure and vent. R_E is the source resistance of the driver, R_M its mechanical resistance, L_M the mass of the cone, C_M its compliance, R_A the radiation resistance, C_{MB} the combined compliance of the air and vent, R_{AV} the vent resistance, and L_V the mass of the air in the vent. [Adapted from Jordan (1970, 1971)[27,28]]

Similarly, the cochlea can be considered a reflex enclosure in which the stapes is the driver and the round window the vent. Pressure sensors are located inside the enclosure, and the system is arranged so that excitation of the driver will deliver maximum pressure to the sensors inside. It seems clear that, in general, this situation will correspond with maximum amplitude of vibration of the speaker cone

[25] The system is described in terms of the Thiele–Small parameters. An overview and list of references can be found in Thiele, A. N. (2004). The Thiele–Small parameters for measuring, specifying and designing loudspeakers. *Address to Singapore Engineering Society, July 2004*: www.aes-singapore.org/LSPRMTRS604.doc.
[26] Although at certain frequencies there will also inevitably be cancellation.
[27] Jordan, E. J. (1971). Loudspeaker enclosures. Types of baffle and the acoustical laws governing their application. *Wireless World* Jan: 2-6.
[28] Jordan, E. J. (1970). The design and use of moving-coil loudspeaker units. *Wireless World* Nov: 533-537.

(the stapes); on the other hand, the presence of a nearby hole, the port or round window, need not diminish the power output (or sensitivity) of the system if the masses and compliances are chosen correctly. Hence, although the presence of the round window seems at first glance to suggest that pressure detection is not involved (see §D 9.4/b), the analogy with the speaker enclosure says this need not be the case. The resonance is expected to be at the lowest audible frequency, about 20 Hz.

In vivo, the compressibility (compliance) of the outer hair cells will be of primary importance. However, the difficulty in analysing the cochlea in terms of its equivalent circuit is that the compressibility will be frequency dependent – because outer hair cells vary systematically in length from base to apex. No single lumped circuit parameter will suffice. Another difficulty in trying to calculate the system response using the equivalent circuit is that data are insufficient. A major reason is that data that might be useful was derived from dead specimens, whereas compressibility of live outer hair cells is the crucial factor. Clearly, measurements of the impedance of live ears (§D 8.1/c) should prove useful in overcoming this lack.

Nevertheless, it is possible to make a few broad conclusions. One is that the oval and round windows will not ordinarily move out of phase with each other at equal amplitudes. Post mortem, when outer hair cells have died and lost their actively maintained compressibility, antiphase motion is almost inevitable, and in this case the radiation from the oval window is expected to be equal and opposite to that of the round window – only a small evanescent wave would then penetrate a limited distance into the cochlea.

The action of the middle ear also deserves mention. The transformer ratio of the middle ear (about 25 times) acts so as to lower the impedance of the inner ear as seen by the tympanic membrane by a factor of 500 (Zwislocki, p. 60) and in this way only about two-thirds of the acoustic energy incident of the ear drum is reflected away. The impedance of the cochlea is about the same as that of the air in the ear canal, and this impedance matching makes for efficient energy transfer. The issue has been discussed by Kostelijk[29] in 1950 and summarised by Schubert[30] in his 1978 review [pp. 46–47].

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[29] Ch. VII.2.

D 8 [16]

This leads us towards a consideration of the whole of the middle ear, including the middle ear muscles, which is beyond the scope of this thesis. It is enough for now to see that if the middle ear muscles push the stapes into the cochlea, the round window membrane will be stretched and the *static pressure* in the fluid contents will rise. Thus, the protective action of the middle ear muscles comes about not through increasing the stiffness and sound transmission of the ossicles, which is the textbook account, but by increasing intracochlear pressure, a d.c. signal that turns down the a.c. gain of the cochlear amplifier. That is, we have returned to the old *intralabyrinthine pressure theory* of middle ear muscle action, put forward by Gellé in 1885. There is evidence in its favour, but it tends to be overlooked because it does not fit in with traveling wave theory. To present just one such piece of evidence, Fig. 10 of Lynch (1982) shows the cochlear microphonic varying by 10 dB as the intracochlear pressure is varied by 7 cm of water. A synthesis of evidence supporting the intralabyrinthine pressure theory will be the subject of future work.

8.2/a Perceptible effects of the round window

The previous section sketched a simple picture of the cochlea (Fig. 8.1) in which the fast wave and the pressure it conveys was taken into account and contrasted it with the traveling wave view where the opposite is the case. In this section I will set out to show how the revised picture might explain findings that the latter view cannot.

A difficulty in trying to examine the effect of the round window is that it cannot be simply isolated from the rest of the system. For a start, the compliance of the round window is in series with the compliance of the oval window (as well as the rest of the middle ear system), so that their values cannot be determined separately by simple impedance measurements. However, we know that with bone stimulation of the cochlea[31], the volume displacement of the round window is 5–15 dB greater (say 10 dB) than that of the oval window, so we can assume that the compliance of

[30] Schubert, E. D. (1978). History of research on hearing. In: *Handbook of Perception, vol. IV, Hearing*, edited by E. C. Carterette and M. P. Friedman (Academic: New York), 41-80.
[31] Stenfelt, S. et al. (2004). Fluid volume displacement at the oval and round windows with air and bone conduction stimulation. *JASA* 115: 797-812.

the round window is about 3 times greater. This is confirmed by other studies[32], although the compliances vary from one experiment to another.

Nevertheless, it is generally agreed[33,34] that the round window membrane is a major component of cochlear input impedance at low frequencies (several hundred hertz). As shown in Fig. 10 of Lynch et al. (1982), the input impedance was approximately halved at low frequencies when the round window was removed. At higher frequencies, the magnitude of the cochlear impedance itself tends to dominate and the effect of removal is not clear. However, it is still the case that increasing pressure in the cochlea (Lynch, 1982, p. 115) reduces the cochlear microphonic to a 1 kHz tone[35], suggesting that the round window is still having an effect, but less obviously.

At higher frequencies, the round window impedance will dwindle in comparison with that of the cochlea (refs. 33, 34), but it should be pointed out the sound pressure levels used (105–130 dB) were so intense that active outer hair cell properties would have been swamped. In any case, it is noted that the input impedance of the cochlea is largely resistive at most frequencies[36].

8.2/b Effects of cochlear compressibility

Evidence put forward in §I 3.3/b was consistent with the idea that the cochlea is compressible, although probably much more so in a live cochlea – with its outer hair cell bubble – than in a dead one. There have been a handful of studies in which the effect of compressibility was considered, although the motivation was to examine the effect on the cochlea of inadvertently introduced air bubbles rather than in its natural condition, but the same physical principles apply nevertheless and the conclusions reached are useful.

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[32] Zwislocki (2002), p. 73. Stenfelt et al. (2004) report Kirikae’s 1959 result that the oval window is about 20 times stiffer than the round window (p. 809).

[33] Nedzelnitsky, V. (1980). Sound pressures in the basal turn of the cat cochlea. *J. Acoust. Soc. Am.* 68: 1676-1689.

[34] Lynch, T. J., et al. (1982). Input impedance of the cochlea in cat. *J. Acoust. Soc. Am.* 72: 108-130.

[35] The reduction is less than 3 dB, which is not as much as the 10 dB at 100 Hz, but still appreciable.

[36] Zwislocki (2002), p. 128.

D 8 [18]

Merchant et al. (1996) studied the input impedance of human temporal bones by measuring the volume displacements of the two windows[37]. In this way they could obtain a direct measure of the compressibility of their inner ears. They separate the impedance effects of any introduced bubble from those of the existing cochlea[38]. They calculate (p. 37) that any air bubble must have been less than 0.2 μL (2×10^{-10} m^3), which is about 500 smaller than the volume of the cochlear fluids (100 μL). Such a bubble would be comparable to the total volume of outer hair cell cisterns[39], although of course the specimen they measured was far from alive. They found several anomalies between their impedance figures and those of previous workers, particularly below several hundred hertz and above 4 kHz, and conclude (p. 44) that the combined impedance of the stapes and cochlea may be higher than previously thought. I surmise there may be large differences between the impedances of live and dead cochleas, a position supported by Ruggero and Temchin (2003) who surveyed the disparity between live and dead measurements and concluded that there were substantial differences[40]. Most post mortem studies of temporal bones are flawed, they suggest, and the inner ear is more wide-band and sensitive than these studies imply.

Puria et al. (1997) also studied human temporal bones and devoted some effort to looking at the effect of air bubbles[41]. First they degassed their specimens in a vacuum chamber and found that the input impedance increased, typically by a factor of about 2 (their Fig. 6). Then they introduced air bubbles of 8–20 μL and found inexplicably variable effects (Figs 7 and 8). Sometimes the effects were large (a factor of 30 or more at some frequencies) and sometimes small (negligible), but almost invariably a bubble will decrease input impedance. The authors develop a circuit model of bubble inclusion (Fig. 10) that differs depending on whether the

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[37] Merchant, S. N., et al. (1996). Acoustic input impedance of the stapes and cochlea in human temporal bones. Hear. Res. 97: 30-45.
[38] A bubble impedance Z_B would be in series with the stapes impedance Z_S but in parallel with the existing cochlear impedance Z_C. Therefore, effective $Z = Z_S + [Z_C{\cdot}Z_B/(Z_C + Z_B)]$. I think that most of Z_C is due to bubble compressibility anyway, which supports this equation.
[39] In §D 8.4/d, we calculate that the cisternal system of each outer hair cell may contain about 5×10^{-16} m^3 of air. For a total of 12 000 outer hair cells, this means a volume of about 6×10^{-12} m^3.
[40] Ruggero, M. A. and A. N. Temchin (2003). Middle-ear transmission in humans: wide-band, not frequency-tuned? *Acoustics Research Letters Online* 4: 53-58. Persistent efforts to derive live cochlea properties from dead specimens is no doubt underlain by the thought that the basilar membrane provides the tuning. See footnote 144.
[41] Puria, S., et al. (1997). Sound-pressure measurements in the cochlear vestibule of human-cadaver ears. *J. Acoust. Soc. Am.* 101: 2754-2770.

bubble is in the upper or lower gallery. They note that intracochlear pressure should depend on the cochlear and round window impedances (p. 2762), but when an air bubble is introduced into the upper gallery it will act as a shunt across the cochlear impedance, whereas one in the lower gallery will shunt the round window impedance. On my interpretation, the effect should be equivalent, but the impedances are so high that measurement errors may be responsible. In any case, the authors agree that, in general, significant volume velocity can be lost in compressing the air within a bubble (p. 2762).

Most recently, Ravicz et al. (2004) performed a study on the middle ears of human temporal bones and found some puzzling effects[42]. They were looking at the effects of otitis media with effusion (when infection causes the middle ear to fill with fluid) and could not understand why, when the middle ear (minus mastoid air cells) was filled with fluid, the tympanic membrane could still move. It was as if (p. 121) the middle ear had a hidden air bubble of between 25 and 270 µL. The authors considered compressibility in the cochlea, but dismissed it because of previous studies. They left the findings as a mystery, on a par with the curious fact (p. 127) that patients with middle ears completely filled with fluid lose only 20–30 dB of hearing sensitivity.

Whether the answer lies in cochlear compressibility or small hidden compliances in the middle ear, this work highlights how crucial small bubbles can be to the cochlea’s input impedance – and thus to our hearing sensitivity.

8.3 Are outer hair cells pressure sensors?[43]

Because outer hair cells bear stereocilia, it is natural to assume that, like inner hair cells, the unique stimulus to which they react is bending of the stereocilia. But is this long-standing assumption necessarily true?

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[42] Ravicz, M. E., et al. (2004). Mechanisms of hearing loss resulting from middle-ear fluid. *Hear. Res.* 195: 103-130.

[43] This and the following section based on Bell, A. (2003). Are outer hair cells pressure sensors? Basis of a SAW model of the cochlear amplifier. In: *Biophysics of the Cochlea: From Molecules to Models*, edited by A. W. Gummer (World Scientific: Singapore), 429-431.

Textbooks usually refer back to an experiment of Hudspeth and Corey[44] on which to justify their statement that hair cells are displacement detectors. Whereas a glass probe bending a group of stereocilia will indeed give rise to a receptor potential, this experiment does not rule out the possibility that the cell body is also being subject to pressure. Of course, I would agree that in the investigated case (a hair cell in the bull-frog's sacculus) the stimulus is likely to be stereocilia bending alone, but to generalise this experiment to include adult mammalian outer hair cells, which lack kinocilia, is perhaps an unjustifiably large leap.

The text that follows considers a number of recurring anomalies which have appeared in the literature over the years and concludes that their common underlying feature is an OHC response to pressure. Section D 8.4/e (below) examines the hypothesis that OHCs have dual sensitivity, responding not only to displacement – via deflection of the stereocilia – but, through their cell body, reacting directly to imposed pressure as well. A later section (§D 9.1) describes how this cooperative arrangement could work, but the key proposition is that OHCs react predominantly to intracochlear pressure at levels of less than about 60 dB SPL.

Of course, the stereocilia still function at these low levels, but most of the receptor potential they generate comes from responding to the standing wave of the cochlear amplifier – which is primarily initiated, it is proposed, by the pressure signal. The implication is that the traveling wave is not well suited to stimulating OHCs: up and down movement of the basilar membrane does not easily translate into bending fairly rigid V-shaped stereocilia bundles. At higher intensities, the cochlear amplifier saturates, and has no major role in IHC stimulation[45]. At these elevated levels, direct displacement of the IHC stereocilia may be the important process, and this could be a role for the traveling wave.

By using two mechanisms, one operating at low levels and the other at high, the cochlea can split its working range into two[46]. Interestingly, cancellation effects

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[44] Hudspeth, A. J. and D. P. Corey (1977). Sensitivity, polarity, and conductance change in the response of vertebrate hair cells to controlled mechanical stimuli. *Proc. Nat. Acad. Sci.* 74: 2407-2411.

[45] When people lose OHCs, their hearing threshold rises by about 60 dB. The idea that OHCs are mechanical preamplifiers that send their output to the IHCs was put forward in §M 4.2.

[46] Davis originally made the suggestion that the cochlea was composed of two sensory systems side by side. He said that the outer hair cells were sensitive and fragile while the inner hair cells were less sensitive but rugged [Davis, H. (1960). Mechanism of excitation of auditory nerve impulses. In: *Neural Mechanisms of the Auditory and Vestibular Systems*, edited by G. L. Rasmussen and W. F. Windle (Thomas: Springfield, IL), 21–39. p. 36.

become evident at about 60 dB, as we will see in Chapter D9. Range-splitting is an important way by which the ear can increase its dynamic range to 120 dB, a range which, for a single transducer, is extremely challenging. As explained later, the two systems operate independently; perhaps as protection against overload, they are even separately tuned (by half an octave or more: §D 9.1/l).

The unifying hypothesis is that there are two detection systems in the cochlea, each arranged to detect a different signal: the OHCs detect the common-mode pressure signal between 0 and 60 dB SPL (and pass it on to the IHCs); from 60 to 120 dB, the IHCs directly detect traveling wave motion. That our perception of sound is seamless, irrespective of level, is remarkable, but the cochlea is a detection system refined over millions of years of evolution. A comparison may be made with the visual system, where the rods and cones operate at low and high light levels respectively. With that perspective, let us now turn our attention to how the novel pressure-detection mechanism can work as claimed.

8.3/a Evidence for OHC pressure sensitivity

We have set out a prima facie case in Chapter I3 that outer hair cells were sensitive to oscillating hydrostatic pressure. The following sections draw parallels with other sensory systems and provide a detailed account of how this sensitivity could arise. To recap and expand the case, the following lines of evidence are set out. Additional evidence will be presented in Chapter D9.

A. Motility of OHCs is driven by the electrical potential of the cell[47,48], and this is usually thought to derive from bending of stereocilia. This is not always the case, however, and in one study[49] when the stereocilia were directly stimulated no length changes could be elicited.

B. If stereocilia are meant to detect relative shear between the basilar membrane and the tectorial membrane, their V-shaped arrangement on the cuticular

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[47] Brownell, W. E., et al. (1985). Evoked mechanical responses of isolated cochlear outer hair cells. *Science* 227: 194-196.

[48] Evans, B. N. and P. Dallos (1993). Stereocilia displacement induced somatic motility of cochlear outer hair cells. *Proc. Nat. Acad. Sci.* 90: 8347-8351.

[49] Canlon, B. and L. Brundin (1991). Mechanically induced length changes of isolated outer hair cells are metabolically dependent. *Hear. Res.* 53: 7–16.

plate seems far from optimum. The resistance of the V to deformation would appear to make bending in a radial direction more difficult.

C. OHCs give strong responses to oscillating water jets placed near the cell body[50–55]. Responses are seen both in membrane potential and length changes, and the length changes, which can be either positive or negative, depend on cell length. The ground-breaking study of Brundin et al. (1989) found that the cells were inherently tuned, with best frequency varying systematically with cell length[56]. The cells could *either lengthen or shorten* and the researchers conclude that "each outer hair cell is a tuned detector of mechanical vibrations…" (p. 816) "independent from the basilar membrane travelling wave" (p. 815). A number of reports explicitly note how difficult it is to bend stereocilia directly (which is the prime reason that water jets are used). However, in using a water jet, the observed response could be seen as deriving from the oscillating pressure component of the jet rather than its movement, although there is as yet no direct evidence for this.

D. The responses persist when the apical end of the cell is held in a pipette[57,58], so that the stereocilia are completely protected (Fig. 8.4).

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[50] ibid.
[51] Canlon, B., et al. (1988). Acoustic stimulation causes tonotopic alterations in the length of isolated outer hair cells from guinea pig hearing organ. *Proc. Nat. Acad. Sci.* 85: 7033–7035.
[52] Brundin, L., Flock, Å., and Canlon, B. (1989). Sound-induced motility of isolated cochlear outer hair cells is frequency-specific. *Nature* 342: 814–816.
[53] Brundin, L. and I. J. Russell (1993). Sound-induced movements and frequency tuning in outer hair cells isolated from the guinea pig cochlea. In: *Biophysics of Hair Cell Sensory Systems*, edited by H. Duifhuis et al. (World Scientific: Singapore), 182–191.
[54] Brundin, L. and I. J. Russell (1994). Tuned phasic and tonic motile responses of isolated outer hair cells to direct mechanical stimulation of the cell body. *Hear. Res.* 73: 35–45.
[55] Preyer, S., et al. (1995). Abolition of the receptor potential response of isolated mammalian outer hair cells by hair-bundle treatment with elastase: a test of the tip-link hypothesis. *Hear. Res.* 89: 187–193.
[56] Brundin et al. (1989), Fig. 4. The frequency increased by about an octave for every 13 µm decrease in cell length.
[57] Brundin and Russell (1993).
[58] Brundin and Russell (1994).

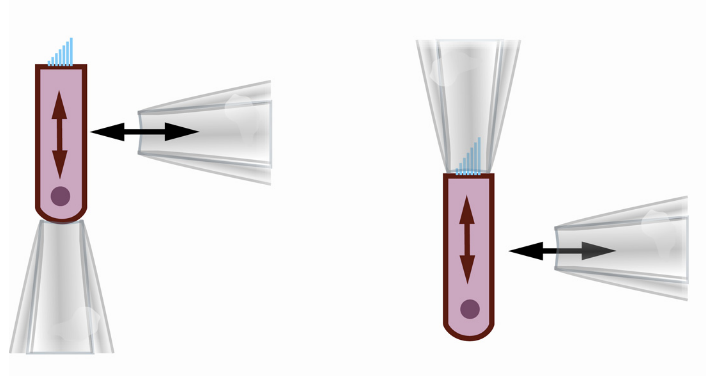

Fig. 8.4. Identical responses. Experiments by Brundin and Russell (1994) showed that an OHC responded identically whether held from above or below. [Drawing by Sharyn Wragg, RSBS Illustration]

E. Similar responses are observed when a vibrating probe is placed against the cuticular plate or on the side of the cell[59]. The vibrating probe would cause the interior pressure of the cell to vary (Fig. 8.5).

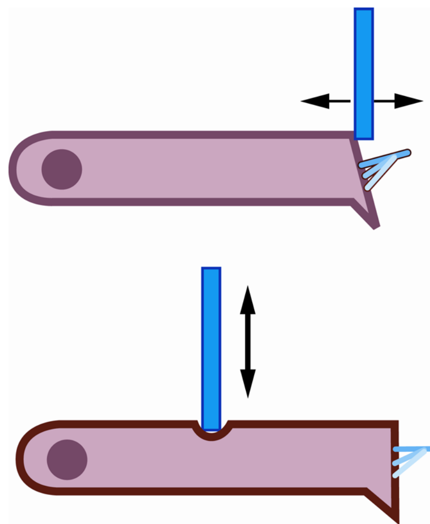

Fig. 8.5. Responses to a vibrating probe were the same irrespective of whether Chan and Ulfendahl (1999) stimulated them from the top or the side. [Drawing by Sharyn Wragg]

F. OHCs are constructed like pressure vessels: they are shaped like a test-tube with rigid walls reinforced with cross-ply belting (Fig. 8.12), and maintain an internal turgor pressure of about 1 kPa.

G. The bodies of OHCs are surrounded by perilymph, and this watery incompressible liquid is in continuous hydraulic connection with the entire fluid

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[59] Chan, E. and M. Ulfendahl (1999). Mechanically evoked shortening of outer hair cells isolated from the guinea pig organ of Corti. *Hear. Res.* 128: 166–174.

contents of the cochlea via the spaces of Nuel (see Fig. 5.1). The otic capsule is comprised of ivory-like bone, the hardest bone in the body. Such a design would allow a compressible OHC to directly and efficiently sense intracochlear pressure.

H. OHCs display piezoelectric-like properties – an electric response to pressure. In terms of length change per millivolt, the sensitivity of OHCs is more than 10 000 times greater then Rochelle salt, a common piezoelectric material[60].

Taken together, these results suggest that pressure may be the key stimulus to OHCs, not bending of their stereocilia. Of course, the above experiments were done without thought that the water jet's pressure was itself causing the observed effects, and so experimental set-up and control was far from ideal for deciding the question. It is difficult to separate a pressure stimulus from a displacement one because they go hand in hand. Ideally, we would like an experiment where the two possible stimuli were well isolated, say with the cells enclosed in a container holding a large volume of liquid and pressure applied to the container. Then we could clearly determine whether outer hair cells do respond to imposed pressure.

Nevertheless, one of the above experiments did provide a suggestive numerical result. Brundin and Russell (1993) observed that water-jet responses of OHCs could be seen down to a (roughly) calculated pressure equivalent to 49–64 dB SPL. Allowing for middle-ear gain of 30 dB, this equates to a situation of 19–34 dB SPL in the ear canal. But this measurement is on a single OHC, and so represents an open-loop gain. In the cochlea, positive feedback between the rows (the basis of the cochlear amplifier according to the SAW model) will amplify the pressure-induced signal by 40–60 dB (§M 4.3, §R 6.2). That is, the closed-loop gain of the system in response to a pressure stimulus of 0 dB SPL (threshold of hearing) would be sufficient to produce a detectable response. In other words, to evoke responses at threshold, no travelling wave and no bending of stereocilia is required.

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[60] Dong, X., et al. (2002). Piezoelectric reciprocal relationship of the membrane motor in the cochlear outer hair cell. *Biophys. J.* 82: 1254-1259.

8.3/b Revisiting the Wilson hair-cell swelling model

The Wilson hair-cell swelling model was constructed to explain how acoustic emissions could travel from outer hair cells to the ear canal (§I 3.2/k). It follows that if outer hair cells change volume and create fast pressure waves, then simple reciprocity would see fast pressure waves causing volume changes. Thus, instead of a restricting the Wilson model to one-way signal travel, it is suggested that signals (at least at low levels) travel into, and out of, the cochlea via fast waves.

If that is so, then delays measured in active cochlear mechanics must nearly all be due to filter delays – that is the time required for a sharply tuned filter to build up its oscillation. That is a simple but strong statement, and can be directly tested. The fast time constants mentioned in the second part of §I 3.2/k provide some credence that travel times in and out can be very short. The drawback with basilar membrane measurements is that they do not automatically give a measure of the cochlear oscillators; rather, they give the averaged response of many such oscillators (which, in the main, act radially). Isolating SAW oscillator behaviour from basilar membrane behaviour may not be straightforward, but it could in theory be done.

8.4 Detecting sound underwater

This section goes back to first principles. Immersed as they are in liquid, OHCs are effectively hydrophones, detecting sound propagating through water. It therefore pays to take a look at underwater acoustics and the approaches that marine creatures use to detect sound in water. In this context it is worth remembering that our hearing sense first evolved in water and only later became adapted to life in air.

A creature living in water faces a major problem in detecting sound in its environment. As pointed out by Pumphrey[61], its tissues are largely composed of water, and so it will have nearly the same compressibility and density as the medium in which it lives. In other words, sound will pass straight through it without interacting; effectively, the animal is acoustically transparent. The way around this is

[61] Pumphrey, R. J. (1950). Hearing. *Symp. Soc. Exp. Biol.* 4: 3-18.

to introduce an impedance discontinuity, but first we need to be clear in our terminology.

A basic starting point[62] is to understand that all plane propagating waves carry two different, but intimately related properties: particle displacement and sound pressure. Thus, a plane wave moving through a medium of density ρ at phase velocity c will have an rms particle velocity u and rms sound pressure p. The characteristic impedance of the medium, p/u, is given by $p/u = \rho c$. In air the acoustic impedance is close to 415 Pa s/m (rayls), whereas in water the corresponding figure is 1.5×10^6, about 3600 times greater. Since the sound intensity, I, the acoustic power per unit area, is given by the product of pressure and particle velocity, $p \times p/\rho c = p^2/\rho c$, this means that for the same acoustic power, the pressure in water is 60 times greater than it is in air and the particle velocity 60 times less. Rephrased, *in detecting sound underwater, sensing the pressure component of a wave is easier than extracting its displacement component* – provided, of course, that you have a way of introducing a compressible element to detect the pressure.

This situation, however, only applies to plane waves, far from the source. As described by van Bergeijk[63], when we are close to the source, as well as the propagated pressure wave there is an important near-field displacement effect to take into account. Van Bergeijk considers as source the case of an oscillating air bubble. As the bubble expands and contracts, it displaces water away from, and towards, itself. Because water is virtually incompressible, this radial displacement is passed on from one concentric layer to the next. The displacement amplitude with fall off as $1/r^2$, where r is the distance from the source.

If water were completely incompressible, this displacement would be, van Bergeijk observes, the only detectable phenomenon. But due to water's small compressibility, the vibrating bubble will also generate an acoustic wave, the familiar pressure wave. This wave will decrease in amplitude as $1/r$.

Therefore, close to the source, in the near-field, the displacement amplitudes are greater than expected from a plane wave; in the far-field, the displacement amplitudes are virtually all contributed by the pressure wave. The boundary between

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[62] Wartzok, D. and D. R. Ketten (1999). Marine mammal sensory systems. In: *Biology of Marine Mammals*, edited by J. E. Reynolds and S. A. Rommel (Smithsonian: Washington), 117-175.
[63] van Bergeijk, W. A. (1964). Directional and nondirectional hearing in fish. In: *Marine Bio-Acoustics*, edited by W. N. Tavolga (Pergamon: Oxford), 1, 281-299.

the two regimes, where the two displacement amplitudes are about equal, is generally taken be one-sixth of a wavelength[64]. In water at 1 kHz, this is about 25 cm.

A radially oscillating bubble gives the strongest near-field effect. Other oscillating bodies, such as solid spheres vibrating side to side, will also produce near-field effects, but the displacements will fall off faster ($1/r^3$ for the sphere) and there will be directional effects as well. A wriggling worm will only make its presence felt very close by[65].

The major conclusion to this discussion is to see that in detecting sound underwater we are better off, in the near field, with using a displacement-sensitive detector, whereas in the far field both pressure and displacement detectors are equally effective.

8.4/a Detecting pressure and displacement

The classic displacement detector is the hair cell (Fig. 8.6A), and it is usually considered that all hair cells of the acoustico-lateralis system (from which mammalian ears evolved) are displacement detectors. On the other hand, if we are to detect pressure, an impedance discontinuity is needed, and a common implementation is a diaphragm across an enclosed space so as to detect the force generated across it, as shown in Fig. 8.6B. The distinction is spelt out[66] in Ewing (1989).

[64] In the case of a source that is very much smaller than the sound wavelength. A more rigorous treatment is given in Section 6.2 of Rossing, T. D. and N. H. Fletcher (1995). *Principles of Vibration and Sound*. (Springer: New York).
[65] For analysis see Section 7.1 of Rossing and Fletcher (1995).
[66] Ewing, A. W. (1989). *Arthropod Bioacoustics: Neurobiology and Behaviour*. (Edinburgh University Press: Edinburgh). [pp. 58 ff.]

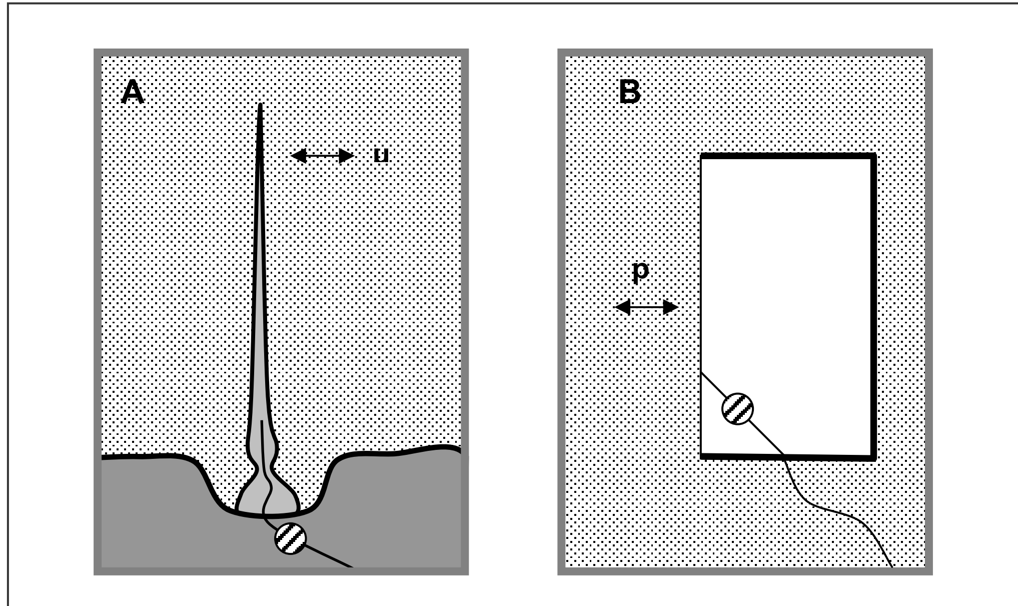

Fig. 8.6. Detecting displacement (A) and pressure (B). A hair on a hair cell responds to water displacement *u* by bending; a sensor mechanism (striped) responds neurally. To respond to a pressure wave, *p*, in the water, a diaphragm across an impedance discontinuity is required; the sensor is set to detect movement of the diaphragm. Air is commonly used to fill the space. Adapted from Ewing (1989).

Fish use both these mechanisms. Firstly, the hair cells in the lateral line organ respond to movement of fluid as the fish swims, giving important information about its nearby environment and relative motion. Secondly, most fish also detect pressure waves – they hear – by using an impedance discontinuity. One way is to introduce a material with density greater than water, such as calcium carbonate; this is the option taken by cod which have calcite otoliths sitting on top of hair cells[67]. The second way is to use a light or compressible material, and many fish species use a gas bladder filled with air[68]. The bladder is useful for buoyancy, but it also oscillates in volume and displacement as pressure waves pass through. By coupling the surface of the bladder to the ear via special bones (the Weberian ossicles), the fish can hear long-distance pressure waves[69].

The following demonstration from Békésy[70] well illustrates how a compressible volume can be very efficient in allowing vibration to be detected within nearly incompressible water. He placed his finger inside a cavity formed out of a

[67] See Ch. 4 of Fletcher (1992).
[68] Fletcher (1992), section 9.2.
[69] van Bergeijk (1964).
[70] Békésy, G. v. (1967). Some similarities in sensory perception of fish and man. In: *Lateral Line Detectors*, edited by P. H. Cahn (Indiana University Press: Bloomington), 417-435.

heavy block of lead and filled the cavity with paraffin oil (Fig. 8.7). On one face of the cavity was a flexible membrane driven by a vibrator, in this way supplying oscillating pressure to the oil.

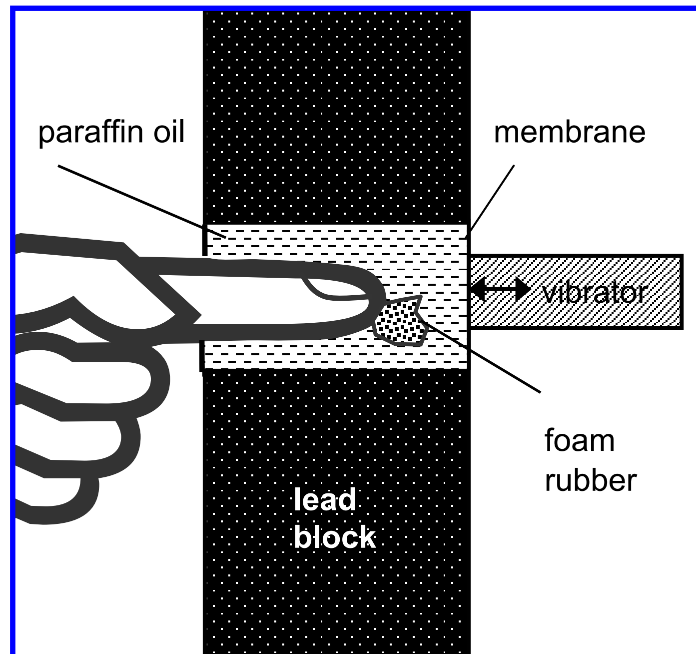

Fig. 8.7. Békésy's demonstration of an efficient way to hear underwater. Touching the foam rubber produced "the sensation of strong vibrations".

When he placed a small piece of foam rubber inside the cavity, he reported that touching the foam rubber "produced the sensation of strong vibrations" [p. 424]. His experiment was done in the context of a discussion of whether we hear by sensing pressure or displacement. Anatomy seems to suggest that with the mammalian cochlea containing hair cells, as in Fig. 8.6A, it is designed to respond to fluid displacements. If that is so, then the middle ear can be regarded as a device, like the swim bladder, for converting sound pressures into volume displacements. Such an account accords with the travelling wave theory – in which displacement of the basilar membrane is the focal stimulus. But I want to look closer at anatomy and point out that *there appears to be another pressure-to-displacement converter, Békésy's piece of foam rubber, strategically located inside the hair cell itself* – an ideal arrangement for detecting underwater sound directly which has so far been overlooked.

The material to follow therefore presents a novel extension of the underwater acoustics story. It describes a situation that neatly reflects Békésy's revealing experiment. We note that Fig. 8.7 is an arrangement in which a compressible material is completely enveloped by incompressible surrounds to create a pressure-to-displacement converter. In the ear the otic capsule is made of material even harder than a lead block – ivory-like bone, the hardest in the human body – and is filled with nearly incompressible water. Helmholtz elaborates on this arrangement by noting that "An incompressible fluid... contained between solid walls is distinguishable from a compressible one in this: that every impulse [however minute] which reaches any part of its surface communicates itself immediately throughout the whole fluid…"[71]. Now, if we introduce compressible hair cells into the system, this permits efficient detection of vibration. This possibility is explored by first considering sharks and other creatures who, without swim bladders, hear remarkably well underwater.

8.4/b The revealing case of shark hearing

How sharks hear has been a long-standing puzzle. As with all elasmobranchs – sharks, rays, and skates – they lack bone and make do with cartilage instead. Unlike the bony fish, they also lack swim bladders. While some of the cells in their labyrinth bear otoconia, others, particularly those most sensitive to vibration, do not. The standard conclusion[72] is that sharks must rely on detecting water displacement directly.

The problem is that these motions are extremely minute far from the source, and amount to molecular dimensions. Some authors have even doubted that sharks could hear over large distances, and that they converge on prey through the use of smell, but Corwin's review mentions observed acoustic responses over 250 m in some sharks. At such distances, particle displacements, he calculates, must be as small as 5×10^{-10} m, and the signal-to-noise ratio must therefore be low. If shark

[71] Helmholtz, H. L. F. v. (1874). *The Mechanism of the Ossicles and the Membrana Tympani [translated by J. Hinton]*. (New Sydenham Society: London). [p. 106]

[72] Corwin, J. T. (1981). Audition in elasmobranchs. In: *Hearing and Sound Communication in Fishes*, edited by W. N. Tavolga et al. (Springer: New York), 81-105. His Fig. 5-1 provides a good summary.

hearing is comparable to that of fish, then another perspective on the problem[73] suggests that the displacements are about 2×10^{-12} m, about 1/100th the diameter of the hydrogen atom. But the real problem is not the size of these motions but that they are theoretically undetectable because any small sensor will move in step with its surroundings as an acoustic pressure wave passes through. There is no *relative motion* to sense, a situation aggravated by wavelengths underwater being so long.

In order to hear the shark must somehow introduce an impedance discontinuity so that the pressure wave will produce differential forces. The shark has two populations of cells in its labyrinth that are involved in vibration responses: the sacculus, covered in otoliths, and the well-named macula (or papilla) neglecta[74,75] which is covered only with a gel (Fig. 8.8).

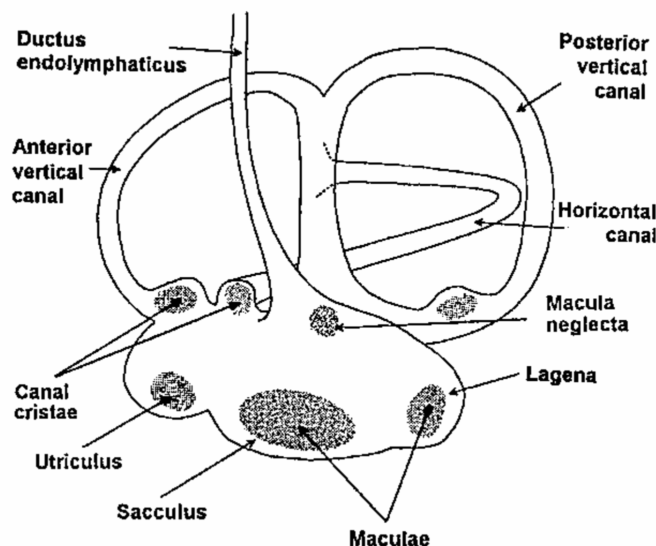

Fig. 8.8. In sharks, the macula neglecta is strongly sensitive to vibration. This extensive patch of hair cells sits inside the labyrinth but unlike the other maculae it is not covered with otoliths, only gel. What is its mode of stimulation? [From Myrberg (2001) and used with permission of Kluwer]

The otoliths, with a density some 3 times higher than water, will undergo a relative displacement of about four-sevenths that of the surrounding water, and perhaps this motion is detected. The outstanding problem relates to the neglecta and

[73] Rogers, P. H. and M. Cox (1988). Underwater sound as a biological stimulus. In: *Sensory Biology of Aquatic Animals*, edited by J. Atema et al. (Springer: New York), 131-149.
[74] Lewis, E. R., et al. (1985). *The Vertebrate Inner Ear*. (CRC Press: Boca Raton, FL). pp. 58-62.
[75] Myrberg, A. A. (2001). The acoustical biology of elasmobranchs. *Environmental Biology of Fishes* 60: 31-45.

its 200 000 or so cells, which are highly sensitive to vibration[76,77]. There should be no relative difference between the motion of the water and the motion of the covering gel (which is mainly water). One suggestion[78] has been that this sensory patch of cells may react to some pressure-to-displacement conversion in the labyrinth, but such a process seems physically unrealistic and has been criticised[79].

The answer lies, I think, in a closer study of the anatomy of the macula neglecta[80]. The microscopic study of Corwin (1981) showed that the cells of this sensory region contained both 'dark' and 'light' types, and that the light cells contained many distinctive 'vacuities' of unknown function[81]. I suggest that if these vacuities were filled with air, then, finger-like, the detection problem would be solved[82].

8.4/c Bubbles and underwater acoustics

A clear example of what I have in mind is the mechanosensor of a primitive marine polyp[83] which reacts to vibration near its tentacles by shooting out spikes (nematocysts) at high speed. Microscopic anatomy of the sensor reveals that it comprises a kinocilium, surrounded at its base by a bell-shaped basal body, sitting directly on top of a large clear vesicle (Fig. 8.9). The function of the vesicle is unknown, and its contents could not be ascertained, but the authors say it may have

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[76] Lewis (1985).
[77] Corwin, J. T. (1981). Peripheral auditory physiology in the lemon shark: evidence of parallel otolithic and non-otolithic sound detection. *J. Comp. Physiol.* 142: 379-390.
[78] van den Berg, A. V. and A. Schuijf (1983). Discrimination of sounds based on the phase difference between particle motion and acoustic pressure in the shark *Chiloscyllium griseum*. *Proc. Roy. Soc. Lond. B* 218: 127-134.
[79] Kalmijn, A. J. (1988). Hydrodynamic and acoustic field detection. In: *Sensory Biology of Aquatic Animals*, edited by J. Atema et al. (Springer: New York), 83-130.
[80] Corwin, J. T. (1977). Morphology of the macula neglecta in sharks of the genus Carcharhinus. *J. Morphol.* 152: 341-361.
[81] Like outer hair cells, the light cells appeared turgid, as if under pressure.
[82] Even though a review thought it 'highly unlikely' that, in fish at least, the inner ear could act as a pressure transducer independent of the swimbladder [p. 581 of Tavolga, W. N. (1981). Retrospect and prospect: listening through a wet filter. In: Hearing and Sound Communication in Fishes, edited by W. N. Tavolga et al. (Springer: New York), 573-587.] Of relevance, Wever (1978) points out that "the papilla neglecta is responsive to vibratory stimuli…" although "… it does not lie in a path of vibratory fluid flow and can be expected to be insensitive to aerial and aquatic sounds" (p. 974). A logical inference is that the cells are sensitive to *pressure*.
[83] Tardent, P. and V. Schmid (1972). Ultrastructure of mechanoreceptors of the polyp *Coryne pintneri* (Hydrozoa, Athecata). *Exp. Cell Res.* 72: 265-275.

functional significance. Given that the purpose of the cell is detection of vibration, it is a reasonable speculation that the vesicle is an example of a pressure-to-displacement converter and that its contents are probably air. Another candidate might be a compressible lipid[84] which is able to change phase (and volume) under pressure.

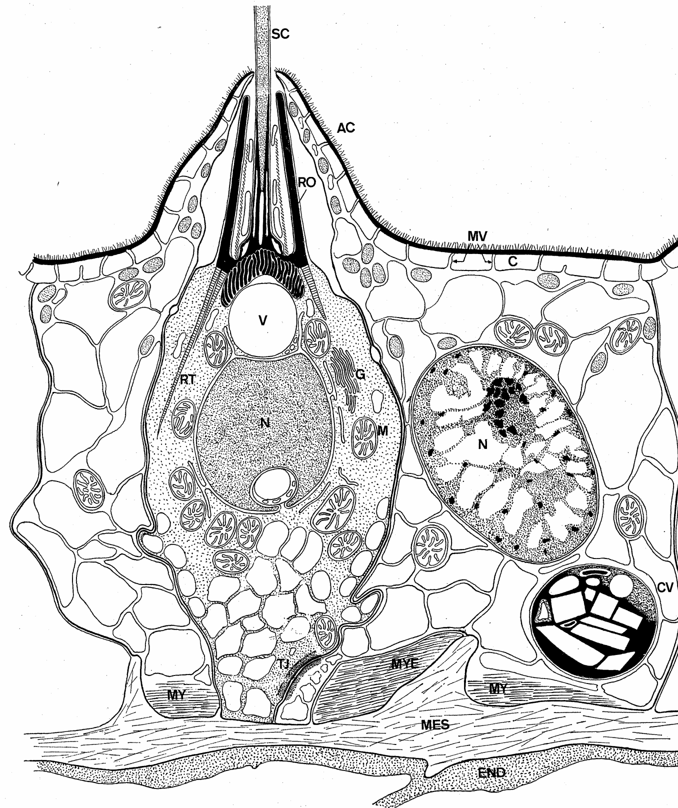

Fig. 8.9. A vibration sensor on a marine polyp. A kinocilium (SC), surrounded at its base with a basal body (black wavy vertical lines), sits on top of a vesicle (V). The contents of the vesicle are not known, but it makes sense to presume it contains air or other compressible material. [Reproduced from Tardent and Schmid (1972) with permission of Elsevier Science]

There is a distinct advantage in having an 'on-the-spot' pressure-to-displacement converter. First, it is a simple scheme, not requiring complicated anatomical structures such as the Weberian ossicles. It is also direct and robust, and each cell operates independently of the others. The problem with the swim bladder is that there's only one, an arrangement that compromises direction-finding, as each ear senses the same signal; some researchers have therefore doubted that bony fish can localise sound sources[85], a proposition which, if true, would have enormous

[84] Some biological materials are even known to have negative compressibilities: Kornblatt, J. A. (1998). Materials with negative compressibilities. *Science* 281: 143a. See also §D 8.5
[85] van Bergeijk (1964), p. 290.

evolutionary disadvantages. The suggestion made here is that many marine creatures – polyps, at least some species of fish, and sharks – have learnt to use air bubbles enclosed within hair cells as detectors of underwater sound.

Air bubbles have dramatic effects on underwater acoustics, and recognition of this opens the door to a fast-growing literature[86]. Although we do not have the space to consider the details, we note that "Gas bubbles are the most potent naturally-occurring entities that influence the acoustic environment in liquids" [Leighton (2004), p. 3267] and there is "exceptionally efficient coupling between bubbles and acoustic waves" [p. 3272], properties that nature has probably exploited in more ways than we currently appreciate. Ultrasound technologists makes use of the phenomenon to increase the contrast of the bloodstream by injecting saline in which microscopic air bubbles are suspended[87]. Whales and dolphins use it by surrounding schools of fish with cylindrical "bubble nets" into which they project intense sounds, generating a reverberating wall of sound through which the fish cannot or will not swim[88]; once corralled, the cetaceans enter for the feed.

When bubbles interact with an underwater sound field they can undergo strong resonances, or "ringing", with the stiffness coming from the bubble's compliance and the inertia from the surrounding liquid. The *Q* varies between about 5 and 30, depending on whether the bubble's radius is micrometres or millimetres (Leighton, 2004, p. 3278) and attenuations can exceed 200 dB/m (ibid., p. 3291). The resonance frequency, f_r, depends approximately on the inverse of the bubble radius, r, so that[89]

$$f_r = \frac{1}{2\pi r\sqrt{\rho}}\left(3\kappa\left(p_0 - p_v + \frac{2\sigma}{r}\right) - \frac{2\sigma}{r} + p_v - \frac{4\eta^2}{\rho r^2}\right)^{1/2} \qquad (8.3)$$

where ρ is the density of the liquid, κ is an index between 1 and 1.4 depending on the thermodynamic properties of the gas, p_0 is the static pressure surrounding the bubble, p_v is the vapour pressure inside the bubble, σ is the surface tension, and η is the viscosity. This equation holds for free-field conditions, and does not include thermal losses, acoustic radiation losses, or bubble–bubble interactions, which appreciably

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[86] Leighton, T. G. (2004). From seas to surgeries, from babbling brooks to baby scans: the acoustics of gas bubbles in liquids. *Int. J. Modern Phys. B* 18: 3267-3314.
[87] Stewart, M. J. (2003). Contrast echocardiography. *Heart* 89: 342-348.
[88] Leighton (2004), pp. 3284-3289.
[89] Eq. 1 of Leighton (2004)

lower the natural frequency. This equation was first derived in simplified form by Minnaert in 1933 by equating the kinetic and potential energies in a linear oscillating system. Then $f_r = (1/2\pi r)\cdot(3\gamma p_0/\rho)^{1/2}$, and we see that spherical bubbles will resonate in the audio range when of millimetre dimensions and in the ultrasonic range when of micrometre size. Of course, as Leighton points out, real bubbles are non-linear and the system is difficult to treat analytically. Nevertheless, the point is the strong interaction between sound and bubbles, whether by resonant or non-resonant means, and this can provide the basis of a sensitive acoustic detector.

8.4/d Cells containing air

When cells contain air bubbles, microscopically identifying the fact is difficult. Tiny bubbles tend to dissolve rapidly in water, and fixation techniques involving various stages of dehydration are bound to give unpredictable results[90]. Examining cells that most definitely contain air bubbles therefore gives some insight.

We mentioned the swim bladder of fish, which they employ for hearing, converting pressure into displacement. Fish also use their gas bladders for buoyancy control, saving them much energy by providing neutral buoyancy. The bladders are filled by a gas gland, which takes molecules of gas, mostly oxygen, out of solution from the blood. The process sounds simple, but it is physically remarkable and incompletely understood[91]. Somehow a metabolic process concentrates gas against an increasing tendency for it to dissolve in water as the pressure increases (Henry's law). Since even chemically inert gases like argon are concentrated, some physical process must be harnessed. The partial pressure of gas dissolved in sea water stays constant at about the level present at the surface (that is, about 0.2 atmosphere for oxygen). A fish living at 100 m will therefore need to concentrate oxygen from 0.2 atmospheres in the blood to 2 atmospheres in the swim bladder[92]. Nitrogen (and argon) are concentrated in a similar way and presumably by a similar mechanism.

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[90] Dr Sally Stowe, ANU Electron Microscopy Unit (personal communication).

[91] Phleger, C. F. (1991). Biochemical aspects of buoyancy in fishes. In: *Biochemistry and Molecular Biology of Fishes, Vol. 1*, edited by P. W. Hochachka and T. P. Mommsen (Elsevier: Amsterdam), 209-247.

[92] Wittenberg, J. B. (1958). The secretion of inert gas into the swim-bladder of fish. *J. Gen. Physiol.* 41: 783-804.

D 8 [36]

Even some fish that live at a depth of more than 7000 m are able fill their swim bladders with gas[93]. At that depth, the pressure is 630 atmospheres and the density of air is close to that of water, suggesting that the function of the bladder is for hearing (using its contents' compressibility) rather than for buoyancy. On the other hand, many fish species[94,95] – such as lantern fish, which migrate daily over a depth of 500 m – have their swim bladders filled with wax esters, which provides buoyancy independent of depth (a drawback of gas is that its buoyancy is a function of depth, which is not ideal for fish ranging hundreds of metres vertically). Other fish species have a swim bladder filled with gas while they are juvenile, but progressively fill it with lipids as they mature until it is completely full. In both these cases, the standard interpretation would be that these fish have compromised their hearing – an unlikely situation from an evolutionary standpoint; *alternatively, if they were to have other sites of compressibility, such as in the body of their hearing cells, the problem would not exist.*

At this point it is appropriate to draw attention to one remarkable facet of fish hearing: in contrast to the early literature that gives the hearing limit of fish at a few kilohertz, recent research shows that a number of species – notably those in a family that include the herring – can hear well into the ultrasonic range[96], up to 180 kHz. The probable reason is to enable detection, and avoidance, of predatory whales and dolphins which emit strong ultrasound signals for echolocation. The observed sensitivity is sufficient to detect cetaceans at a distance of more than 1 km. But the facility raises the question of how these fish do it, as mechanically their auditory system appears ill-suited to high-frequency operation. A simple answer may come from noting that a 10-μm air bubble possesses a resonance frequency in water of about 170 kHz, and this resonance can increase the amplitude of vibration by orders of magnitude as the mass of the water interacts with the springiness of the air.

The prediction is that the hair cells of these fish, like sharks, contain microscopic air bubbles. In terms of the SAW model, it is also noteworthy that the

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[93] Nielsen, J. G. and O. Munk (1964). A hadal fish (*Bassogigas profundissimus*) with a functional swimbladder. *Nature* 204: 594-595.
[94] Nevenzel, J. C., et al. (1969). The lipids of some lantern fishes (family Myctophidae). *Comp. Biochem. Physiol.* 31: 25-36.
[95] Nevenzel, J. C., et al. (1966). Lipids of the living coelacanth, *Latimeria chalumnae*. *Science* 152: 1753-1755.
[96] Higgs, D. M. (2004). Neuroethology and sensory ecology of teleost ultrasound detection. In: *The Senses of Fish*, edited by G. von der Emde et al. (Kluwer: Boston, MA), 173-188.

utricle of herring-like fish contains “bands of hair cells of alternating polarity” [Higgs (2004), p. 180], just like the supposed pattern of outer hair cells in the mammalian cochlea.

A prime reason for introducing a discussion of swim bladders is to look more closely at the gas gland itself. Somehow cells of the gland, typically 10–100 μm in diameter, must create tiny gas bubbles and release them into the swim bladder. Bubble formation is a tricky process in that it has to overcome surface tension between the gas and the liquid in which it forms. Because the pressure inside a gas bubble depends directly on the surface tension and inversely on the radius ($p \propto T/r$, Laplace’s law), a minute bubble faces immediate extinction because the pressure will be so high as to dissolve it away again[97]. For a 0.1 μm diameter air bubble in water, the pressure due to surface tension will be nearly 30 atmospheres. Nevertheless, it is a fact that bubbles do appear out of liquids (beer, for example) and it seems that indents in a surface (such as scratches in a glass) offer some protection from collapsing pressures. Another way of assisting bubble formation is to modify the surface tension, and so it seems no coincidence that gas glands are associated with various kinds of oily lipids. An oil will allow gas to spread on its surface at the same time as repelling water, which explains why lipid droplets are often pearly white (as are the lipid droplets secreted by Hensens cells). In the same way, the swimbladder wall of freshly captured specimens is often observed to be brilliantly white[98] and reflective due to a lipid coating.

Microscopic examination of gas gland cells confirms this picture. A general feature is the presence of vacuoles and lipid droplets[99]. Somehow the hydrophobic lipids allow the cell to generate gas bubbles. A difficulty for the microscopist is that it is hard to tell whether the observed vacuoles contain gas or lipid[100,101], due the fact that bubbles quickly dissolve once the cell dies, and preparation for microscopy induces further changes. Nevertheless, inspection of published gas gland micrographs (Fig. 8.10) shows such a profusion of vacuoles that, given the cell’s function,

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[97] Vogel, S. (2003). *Comparative Biomechanics*. (Princeton University Press: Princeton). pp. 110–111.
[98] Phleger (1991), p. 217.
[99] Pelster, B. (1995). Metabolism of the swimbladder tissue. In: *Biochemistry and Molecular Biology of Fishes, vol. 4*, edited by P. W. Hochachka and T. P. Mommsen (Elsevier: Amsterdam), 101-118. Fig. 2.
[100] Fänge, R. (1953). The mechanisms of gas transport in the euphysoclist swimbladder. *Acta Physiologica Scandinavica Suppl.* 110: 1-133. [p. 34]
[101] Fänge, R. (1966). Physiology of the swimbladder. *Physiol. Rev.* 46: 299-322. [p. 315]

there is little doubt some of the vacuoles must be air bubbles[102]. Coincidentally, in Dorn's work he identifies two types of neighbouring cells – "helle" (light, left of Fig. 8.10) and "dunkle" (dark, right), the same terminology that Corwin uses to describe the two types of interleaving cells in the neglecta of the shark.

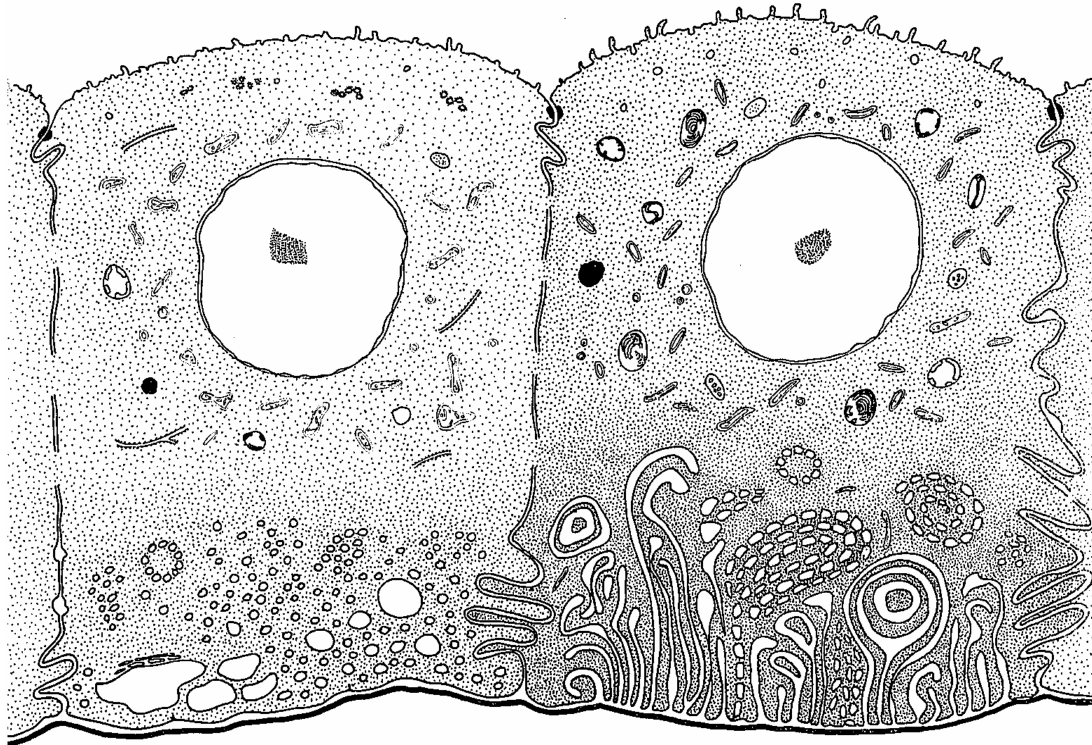

Fig. 8.10. Two types of gas gland cell, which take dissolved oxygen and other gases from the blood and pump them into the swim bladder (here, the cavity below the thick line). The cells contain many vacuoles and it is unclear whether, in vivo, they are filled with gas or lipid, since preparation for microscopy will compromise their natural appearance. Given the cells' function, it is hard to resist the conclusion that some of these vacuoles are air bubbles. [From Dorn (1961) and used with the permission of Springer Verlag]

Before concluding, it is worth noting a recent review[103] of how marine animals, including those without an obvious gas phase, are able to accurately sense depth. The field is full of uncertainties and few definite conclusions can be reached, prompting the authors to suggest that such organisms "may still contain a small gas pocket which would… [enable] stretch receptors to transduce micro-hydrostatic pressure changes" [ibid., p. 27].

[102] Dorn, E. (1961). Über den Feinbau der Swimmblase von *Anguilla vulgaris* L. *Zeitschrift fur Zellforschung* 55: 849-912.
[103] Macdonald, A. G. and P. J. Fraser (1999). The transduction of very small hydrostatic pressures. *Comp. Biochem. Physiol.* 122A: 13-36.

The conclusion is that, despite obstacles, it is possible for living cells to produce, and sustain, tiny gas bubbles within.

8.4/e An air bubble in outer hair cells?

If outer hair cells are pressure detectors, there must be some compressible material within. There are non-aqueous materials inside cells that may have compressibility higher than that of water (45 Mbar^{-1}), but the obvious candidates – conventional lipids and proteins[104] – have compressibility coefficients about the same as water (22–130 and 10–25 Mbar^{-1}, respectively). When seeking responses to micropascal pressures, it is hard to escape the conclusion that the compressible material is a gas, and air, or a component of air, is the logical choice, as consideration of §D 8.4/c, and our underwater heritage, suggests. Following Boyle's law, a gas will halve its volume for doubling of pressure, so that a threshold pressure elevation of 0.5 mPa in the cochlear liquids[105] will cause the volume of a bubble at atmospheric pressure to decrease by a factor of 1 part in 100 kPa/0.5 mPa = 2×10^8. Below I will outline a scheme by which it appears physically possible to detect such a small change.

Outer hair cells contain two distinctive multilayered structures that appear unique to these cells: Hensens body and subsurface cisternas (Fig. 8.11). These features, of unknown function, are closely related anatomically, and my suggestion is that Hensens body is a generator of gas which is distributed to the subsurface cisterns, filling the cell with a proportion of air and making it compressible. These features are now examined.

[104] Kharakoz, D. P. (2000). Protein compressibility, dynamics, and pressure. *Biophys. J.* 79: 511-525.
[105] A 20 μPa threshold pressure in air × 25 middle ear gain.

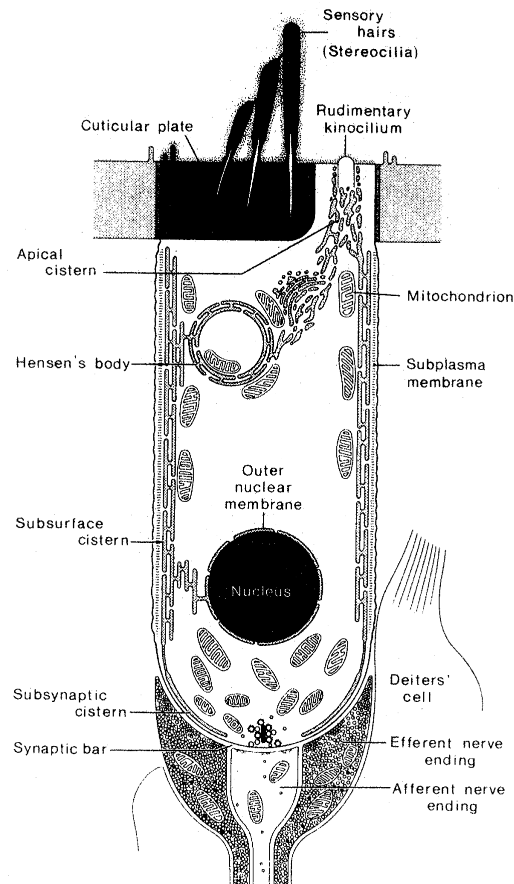

Fig. 8.11. Major organelles in an outer hair cell. Note in particular Hensens body beneath the cuticular plate and the subsurface cisterns lining the lateral cell walls. [From Lim (1986) with permission of Elsevier Science]

As described in a review by Lim (1986)[106], Hensens body is a spherical whorl of endoplasmic reticulum just below the cuticular plate connected to an elaborate cisternal system residing largely on the walls of the cell. Mitochondria lie nearby. Both organelles show a multilayered but fenestrated structure. The subsynaptic portion of the cisterns is close to efferent terminals, suggesting a susceptibility to efferent control. The number of layers and abundance of discrete bodies increases after acoustic stimulation and toxic treatments; distinct vacuoles also become more common[107]. Aspirin is one agent particularly effective in causing blistering and vacuolisation of the cisterns (and of course in reducing hearing sensitivity).

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[106] Lim, D. J. (1986). Functional structure of the organ of Corti: a review. *Hear. Res.* 22: 117-146.
[107] Leonova, E. V. and Y. Raphael (1998). Alteration of membranous structures in ototoxically damaged outer hair cells of the organ of Corti. Midwinter Meeting, Florida, Association for Research in Otolaryngology [abstract 791]

The body is named after Hensen, who first described it in 1863; it appeared to him to have a spiral arrangement[108]. The appearance of Hensens body varies from worker to worker, but clear renderings of the layered structure are seen in a TEM[109] by Engström and Ades and a freeze-fracture micrograph[110] by Mammano et al. (1999). Some revealing micrographs[111] can also be found in Harada et al. (1990). Using a different staining method[112], Spicer et al. (1998) find that the bodies appear as a cluster of vesicles and there is an abundance, and diversity of appearance, not seen before.

An ultrastructural study of the cistern system[113] of guinea pigs showed that each cell usually had between two and four Hensens bodies. Their concentric layers were connected to the cisternal system that typically had 4–7 parallel stacks, but sometimes up to 12. The lumens of the bodies and the cisterns were found to be filled with an electron opaque material, suggestive of neither water nor air; on the other hand, empty areas (caveolae) were found next to bulging and dilated cisterns (their Figs 8 and 9), and these look like remnants of aggregated air bubbles. I suspect that in vivo a thin layer of air exists next to the electron-dense generating apparatus.

Studies of the subcisternal layers with certain vital fluorescent dyes are also revealing. When the lipophilic dyes CTC and DiOC6 are applied[114–116], the whole cisternal system lights up. It appears to occupy an appreciable fraction of the cell's contents, particularly in the region of Hensens body, but also below the nucleus. A

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[108] Engström, H. and J. Wersäll (1958). The ultrastructural organization of the organ of Corti and of the vestibular sensory epithelia. *Exp. Cell Res.* Suppl. 5: 460-492. [p. 464]
[109] Engström, H. and H. W. Ades (1973). The ultrastructure of the organ of Corti. In: *The Ultrastructure of Sensory Organs*, edited by I. Friedman (North-Holland: Amsterdam), 83-151.
[110] Mammano, F., et al. (1999). ATP-induced Ca^{2+} release in cochlear outer hair cells: localization of an isositol triphosphate-gated Ca^{2+} store to the base of the sensory hair bundle. *J. Neurosci.* 19: 6918-6929. Fig. 6B.
[111] Harada, Y., et al. (1990). Three-dimensional ultrastructure of cochlea: a review. In: *Inner Ear Pathobiology (Adv. Otorhinolaryngol. v. 45)*, edited by M. Ciges and A. Campos (Karger: Basel), 49-68. Figs 5–7.
[112] Spicer, S. S., et al. (1998). Cytologic evidence for mechanisms of K^+ transport and genesis of Hensen bodies and subsurface cisternae in outer hair cells. *Anat. Rec.* 251: 97-113.
[113] Saito, K. (1983). Fine structure of the sensory epithelium of guinea-pig organ of Corti: subsurface cisternae and lamellar bodies in the outer hair cells. *Cell Tissue Res.* 229: 467-481.
[114] Forge, A., et al. (1993). Structural variability of the sub-surface cisternae in intact, isolated outer hair cells shown by fluorescent labelling of intracellular membranes and freeze-fracture. *Hear. Res.* 64: 175-183.
[115] Pollice, P. A. and W. E. Brownell (1993). Characterization of the outer hair cell's lateral wall membrane. *Hear. Res.* 70: 187-196.
[116] Ikeda, K. and T. Takasaka (1993). Confocal laser microscopical images of calcium distribution and intracellular organelles in the outer hair cell isolated from the guinea pig cochlea. *Hear. Res.* 66: 169-176.

feature revealed by these dyes is that the cisternal system is lipophilic, implying some associated lipids; in turn this suggests a role for the lipids in separating a gas from its aqueous substrate via surface tension effects, as they do in the swim bladder[117]. The lipids could originate from within the cell itself (outer hair cells are able to synthesise lipids[118]) or from the nearby lipid-rich Hensen cells.

Another fluorescent lipophilic dye, FM1-43, is indicative of cell membrane turnover, and Meyer et al. (2001) found that it strongly stained Hensens body[119] in a guinea pig OHC. The dye cannot penetrate passive cell membranes, but when turnover (endocytosis) of membrane occurs, FM1-43 can be readily taken through into the interior of the cell. In an OHC, dye molecules enter the cell through its apical end, suggesting that the dye penetrates the cuticular pore (shown as the 'rudimentary kinocilium' in Fig. 8.12) and is carried to Hensens body. This fits in with the authors' finding that the dye does not penetrate through the other possible route – the MET or mechanoelectric transducer channels of the stereocilia (at least when the MET channels are blocked)[120]. The work by Meyer et al. therefore reveals a close association between Hensens body and the cuticular pore, a relationship that will be detailed further below.

An important finding is that the cisternal system is essential for electromotility, and hence hearing. When the system is disrupted by high doses of salicylate[121], motility disappears, along with hearing sensitivity, only to return when the drug is rinsed away. Salicylate enlarged the distances between the cisternal layers, and led to increased numbers of vesicles next to them.

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[117] Pelster (1995), p. 104.

[118] Schacht, J. and H. P. Zenner (1987). Evidence that phosphoinositides mediate motility in cochlear outer hair cells. *Hear. Res.* 31: 155-160.

[119] Meyer, J., et al. (2001). Pronounced infracuticular endocytosis in mammalian outer hair cells. *Hear. Res.* 161: 10–22.

[120] The entry of FM1-43 into hair cells has been investigated by a number of workers recently, and their somewhat contradictory results can be better understood once the presumed high permeability of the cuticular pore (and low permeability of the kinocilium) is taken into account. Thus, when MET channels were blocked [Meyer et al. (2001); Griesinger, C. B., et al. (2002). FM1-43 reveals membrane recycling in adult inner hair cells of the mammalian cochlea. *J. Neurosci.* 22: 3939-3952.], these workers concluded that FM1-43 does not enter via the stereocilia because blocking did not prevent dye entry; in contrast, other work concluded the opposite [Meyers, J. R., et al. (2003). Lighting up the senses: FM1-43 loading of sensory cells through nonselective ion channels. *J. Neurosci.* 23: 4054-4065.] because blocking did stop dye entry. The anomaly is resolved when one sees that the first workers used guinea pig cochleas – that have no kinocilia – so that blocking the MET channels still leaves the fontanelle route open, whereas the later work used frogs and mice, animals that have kinocilia instead.

[121] Dieler, R., et al. (1991). Concomitant salicylate-induced alterations of outer hair cell subsurface cisternae and electromotility. *J. Neurocytol.* 20: 637-653.

A number of authors have noted the importance of the cisternal system and speculated on its function. As long back as 1955, Engström noted the distinctive regular layers, or lamellæ, beneath the outer membrane (and in Hensens body) and suggested that polarized molecules aligned within them might produce potentials when distorted, in this way explaining the origin of the cochlear microphonic. More recently, Brownell[122,123] has tried to relate the ultrastructure of the cisterns to the necessary expansions and contractions of the cell wall, and mentioned electrostatic and electroosmotic mechanisms.

While these ideas have merit in understanding electromotility, they are secondary to the main point of this thesis: what is the key initial (or 'adequate') stimulus – bending of stereocilia or compression of a bubble within the cell body? If it is the latter, then the outstanding role of the cisternal system is providing compressibility. Let us calculate the volume of the cisternal system. A single Hensens body, with a diameter of 3 μm, has a volume of 1.5×10^{-17} m^3, and there may be a handful of such organelles, but probably amounting to no more than 10^{-16} m^3. Cisterns have a typical thickness of 0.5 μm, so the volume they occupy in a cell 10 μm in diameter and 50 μm long is about 5×10^{-16} m^3. Take the volume of enclosed air to be half that, about 2×10^{-16} m. If threshold sound pressure causes a reduction in volume of 2×10^{-8}, as calculated above, then this will produce a change in the volume of the cell of 10^{-24} m^3 (which, in terms of cell-sized units, is 10^{-6} μm^3 or a cube with edges of 1/100th of micrometre – small, but not vanishingly so).

Do outer hair cells show compressibility? The common assumption is that they possess no compressibility[124,125], a move predicated on the idea that the cells are filled with water. When the cells expand and contract at acoustic frequencies, there is not enough time for water to pass in and out across the cell membrane, so the shape change must be isovolumetric – when the cell lengthens, its diameter narrows

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[122] Brownell, W. E. (1986). Outer hair cell motility and cochlear frequency selectivity. In: *Auditory Frequency Selectivity*, edited by B. C. J. Moore and R. D. Patterson (Plenum: New York).
[123] Brownell, W. E. and A. S. Popel (1998). Electrical and mechanical anatomy of the outer hair cell. In: *Psychophysical and Physiological Advances in Hearing*, edited by A. R. Palmer et al. (Whurr: London), 89-96.
[124] Iwasa, K. H. and R. S. Chadwick (1992). Elasticity and active force generation of cochlear outer hair cells. *J. Acoust. Soc. Am.* 92: 3169-3173. The measurements were made over timescales that allowed diffusion of water in and out of the cell, so they cannot be used to calculate compressibility, but they do indicate that the cell's geometry is such that volume changes will accompany length changes.
[125] Steele, C. R. (1990). Elastic behaviour of the outer hair cell wall. In: *Mechanics and Biophysics of Hearing*, edited by P. Dallos et al. (Springer: Berlin), 76-83.

accordingly. This can be expressed in terms of the Poisson ratio, σ, the ratio of the radius strain to the length strain. For an incompressible material, $\sigma = 0.5$ (so that for a cylinder the length must change twice as much as the radius), and we would expect measurements on individual OHCs to return such a value. Actual measurements[126] on an OHC gave a value of σ between 1.85 and 2.3, implying that the incompressibility assumption is wrong[127] (and that length changes must be accompanied by appreciable changes in axial and circumferential stresses and internal pressure[128]). In actual fact, not too much credence can be put on reported measurements since the in vivo changes we are looking for are, as calculated above, in the region of parts in 10^8, way below the levels detectable by standard measurement techniques. However, there are measurements on the whole cochlea which bear on the question, and these were discussed in §D 8.2/b. In §D 8.5 we look at the functional implications of a Poissons ratio larger than 0.5.

How can an OHC detect an internal volume change of 10^{-24} m^3? There are two important features.

A. The first is that the outer hair cell, test-tube like, is constructed so as to resist pressure deformations. Brownell aptly describes the OHC as a pressure vessel[129,130] or cylindrical hydrostat[131], capped by a solid plate (the cuticular plate) and encircled by strong helically wound fibres that cross clockwise and anticlockwise like a reinforced garden hose. The actin fibres are set at an angle of 9–15° to the circumference, forming a cytosketetal spring that, together with a rippled outside plasma membrane, makes it possible for the cell to undergo length

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[126] Iwasa and Chadwick (1992).
[127] Allen, J. B. (2001). Nonlinear cochlear signal processing. In: *Physiology of the Ear (2nd ed.)*, edited by A. F. Jahn and J. Santos-Sacchi (Singular Thomson Learning: San Diego, CA), 393-442. [§3.2.2].
[128] Allen calculates, for example, that the observed cyclic length changes mean that internal fluid pressure must increase and decrease by a factor of about 3 during the cycle [his Eq. 30]. Interpreted at face value, the high Poisson's ratio implies that the cell is hypercompressible and has a negative elastic modulus. This possibility is discussed further in §§D 8.4/f and 8.5.
[129] Brownell, W. E. (1990). Outer hair cell electromotility and otoacoustic emissions. *Ear Hear.* 11: 82–92.
[130] Brownell, W. E. and W. E. Shehata (1990). The effect of cytoplasmic turgor pressure on the static and dynamic mechanical properties of outer hair cells. In: *Mechanics and Biophysics of Hearing*, edited by P. Dallos et al. (Springer: Berlin), 52–59.
[131] Brownell and Popel (1998), ibid., p. 89.

changes[132]. To increase rigidity, the cells are inflated to a hydrostatic pressure (turgor pressure) of about 1 kPa[133]. This arrangement is shown in Fig. 8.12.

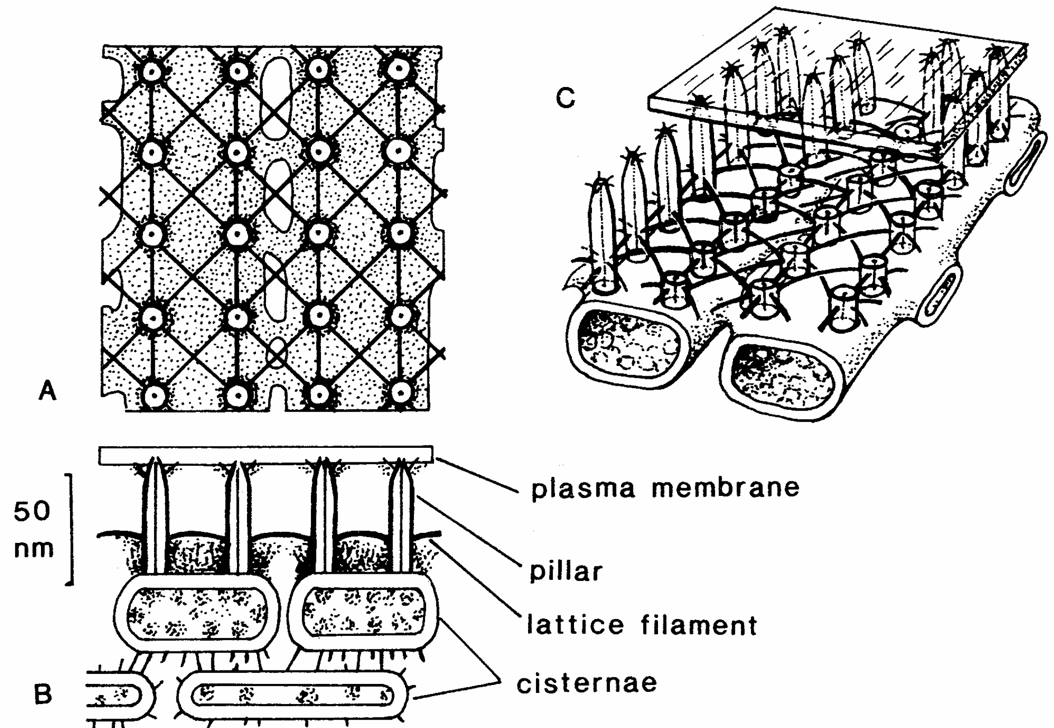

Fig. 8.12. The structure of the cell wall of an outer hair cell. The cross-ply structure, like a garden hose, adds rigidity when the cell is inflated with an internal pressure (of about 1 kPa). One place where the cell cannot resist external pressure is at the cuticular pore, which is covered only by the plasma membrane, and this is where intracochlear pressure is sensed. The unusual cisterns connect with Hensens body (both organelles are unique to outer hair cells) and may contain compressible material. [From Fig. 7 of Bannister et al. (1988)[134] and used with the permission of Elsevier]

B. The second is the presence of a small compliant spot on top of the cell, a hole in the cuticular plate anatomically associated with sensory capabilities. I suggest that this strategically placed organelle – the cuticular pore or fontanelle – is the pressure sensor.

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[132] Brownell and Popel (1998).

[133] Ratnanather, J. T., et al. (1993). Mechanical properties of the outer hair cell. In: *Biophysics of Hair Cell Sensory Systems*, edited by H. Duifhuis et al. (World Scientific: Singapore), 199–206. The pressure is metabolically controlled, and, because σ is greater than 0.5, an increase in pressure leads to a radial expansion and an axial contraction (Steele, 1990).

[134] Bannister, L. H., et al. (1988). The cortical lattice: a highly ordered system of subsurface filaments in guinea pig cochlear outer hair cells. *Prog. Brain Res.* 74: 213-219.

8.4/f The fontanelle as a pressure sensor

Whereas Fig. 8.11 shows the cuticular pore (the rudimentary kinocilium) in vertical section, a cross-section through the cuticular plate shows that it as a tiny circular hole. The cylindrical pore was first observed by Held early in the 20th century, and has been consistently seen by others[135–139], but its function is unclear. An image from Flock et al. (1962), traced in Fig. 8.13 below, shows the familiar array of stereocilia and a distinct hole, about 0.1 µm in diameter, near the vertex of the V. Flock calls it a cuticular pore; Hawkins calls it, rather aptly, a fonticulus or fontanelle[140].

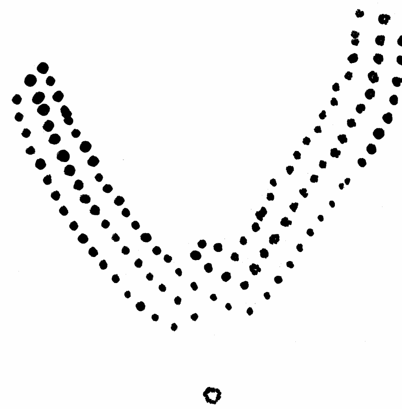

Fig. 8.13. Stereocilia and cuticular pore of an OHC (guinea pig) traced from Fig. 1 of Flock et al. (1962).

Flock and colleagues identify it as a basal body through its obvious similarity at high magnification (their Fig. 3a and shown below in Fig. 8.14A here) to the characteristic 9-fold symmetry of that familiar organelle, the basal body, at the base

[135] Engström, H., et al. (1962). Structure and functions of the sensory hairs of the inner ear. *J. Acoust. Soc. Am.* 34: 1356–1363.
[136] Hawkins, J. E. (1965). Cytoarchitectural basis of the cochlear transducer. *Symposia on Quantitative Biology* 30: 147–157.
[137] Flock, Å., et al. (1962). Morphological basis of directional sensitivity of the outer hair cells in the organ of Corti. *J. Acoust. Soc. Am.* 34: 1351–1355. Fig. 1.
[138] Wersäll, J. and P.-G. Lundquist (1966). Morphological polarization of the mechanoreceptors of the vestibular and acoustic systems. Second Symposium on the Role of the Vestibular Organs in Space Exploration, Washington, DC, NASA, SP-115, 57-72
[139] Sobkowicz, H. M., et al. (1995). The kinocilium of auditory hair cells and evidence for its morphogenetic role during the regeneration of stereocilia and cuticular plates. *J. Neurocytol.* 24: 633-653.
[140] Hawkins, J. E. (1976). Drug ototoxicity. In: *Handbook of Sensory Physiology, vol. 5.3*, edited by W. D. Keidel and W. D. Neff (Springer: Berlin), 707-748.

of kinocilia[141]. It is significant that most mammals, humans included, have a kinocilium during gestation, but it disappears at or shortly after birth, apparently becoming functionless. As its name implies, the kinocilium is a true cilium, with the well-known '9 + 2' arrangement of 9 filaments arranged around 2 central ones. (Many researchers have noted that stereo*cilia* are inappropriately named, as they are not cilia at all but modified microvilli, but the name seems too well established[142].) Kinocilia have a clear motile function in certain animals in certain places – allowing unicellular animals to move about and allowing wax-laden dirt to be transported out of the ear canal, for example – but their association with stereocilia is unclear and in any event widely considered unimportant.

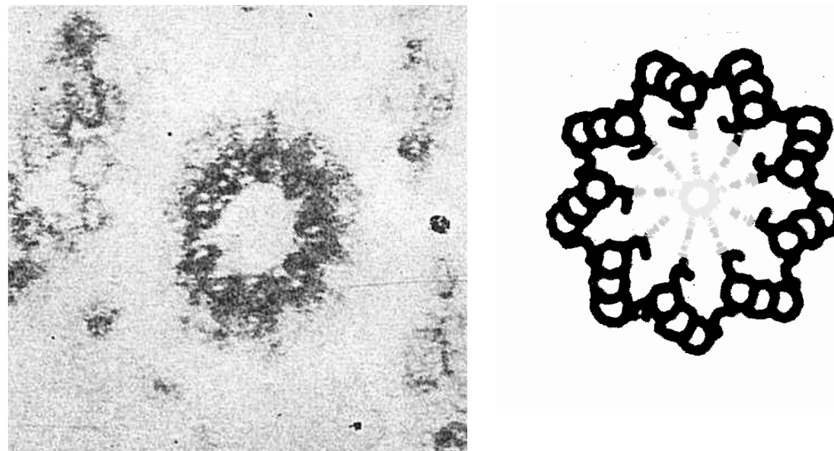

Fig. 8.14. Left: the cuticular pore, showing its 9-fold rotational symmetry; its inside diameter is about 0.1 µm. Right: image of a centriole, of similar size, enhanced to show detail. The hooks are dynein arms. [Cuticular pore from Fig. 3a of Flock et al. (1962) and reproduced with permission of the Acoustical Society of America; centriole from frontispiece of Wheatley (1982) and used with permission of Elsevier Science]

Engström, Ades, and Hawkins[143] seem to have been the only researchers who have taken the cuticular pore seriously. They point out its connection with the basal bodies of kinocilia, which in turn derive from the distinctive centrioles that all animal cells – from amœba to human – display. Because of the major organising influence of centrioles in the growth and maintenance of cells, they suggest that the basal body

[141] Clear views of the basal body in vertical section are depicted in Engstrom and Ades (1973), Figs 21 and 24.
[142] Some have suggested the name *stereovilli* (Santi, P. A. (1988). Cochlear microanatomy and ultrastructure. In: *Physiology of the Ear*, edited by A. F. Jahn and J. Santos-Sacchi (Raven Press: New York), 173–199. p. 182).
[143] Engström et al. (1962).

"should be regarded as the essential excitable structure of the hair cell." (ibid., p. 1363). Indeed, since they thought that stereocilia deflection was the adequate stimulus, the stereocilia must therefore not bend but act as stiff levers, transmitting force to the cuticular place and thereby excite the basal body. The idea received support in a review by Fex[144].

Engström and colleagues also pointed out that the pore is covered only by the plasma membrane of the cell and tends to bulge out, sometimes forming balloon-like protuberances during fixation[145]. This indicates "a high degree of compliance", and, if not artefactual, "must represent structures of great physiological importance." The basal body is surrounded by a radiating pattern of many organelles – mitochondria, small vesicles, granules, and vacuoles – suggestive of a close functional and metabolic relationship; immunolabeling for tubulin, the major component of microtubules, gives an intense fluorescent spot at this point[146]. Hillman noted the pliability of the membrane at the base of the kinocilium[147], and suggested that tilting of the kinocilium excited the cell by its plunger-like action at this point.

My proposal follows Engström and colleagues in accepting the essential importance of the basal body, but rather than having it stimulated by bending of stereocilia, I suggest it could be stimulated directly by intracochlear pressure. Although the kinocilium may be absent in humans after birth, it seems reasonable to suppose that all the machinery for operating it is still in place. That is, the cell has remodelled and refined an existing structure, not wantonly thrown one away. Specifically, I suggest this machinery has been adapted to operate in reverse, allowing it to act as a sensor. A subsequent literature search indicated a close approach to this idea, perhaps even the same: Hillman, pursuing the plunger analogy, concluded a 1971 study (with Lewis) of kinocilia movement at the cell surface of the

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[144] Fex, J. (1974). Neural excitatory processes of the inner ear. In: *Handbook of Sensory Physiology, vol 5.1*, edited by W. D. Keidel and W. D. Neff (Springer: Berlin), 585-646. "For evolutionary reasons, it would seem more likely that the site of the mechano-transducer of mammalian cochlear hair cells would be in the membrane of the cuticle free region of the hair cell top, or very close to this membrane, rather than anywhere else" [p. 596].
[145] Lavigne-Rebillard, M. and R. Pujol (1986). Development of the auditory hair cell surface in human fetuses. *Anat. Embryol.* 174: 369–377.
[146] Steyger, P. S., et al. (1989). Tubulin and microtubules in cochlear hair cells: comparative immunocytochemistry and ultrastructure. *Hear. Res.* 42: 1-16.
[147] Hillman, D. E. (1969). New ultrastructural findings regarding a vestibular ciliary apparatus and its possible functional significance. *Brain Res.* 13: 407–412.

frog labyrinth[148] by referring to the mammalian auditory system and speculating (ibid., p. 418) that the “basal kinociliary remnant” could be a “diaphragm-like, pressure-sensitive spot”. The authors do not specify exactly what the cause of such a deformation might be, and the idea was not taken further. A subsequent major review[149] by one of the pair gives only passing reference to this work and fails to even mention the speculation.

Let us revisit the figure of 10^{-24} m^3 for the volume change inside an OHC at threshold. We see in Fig. 8.15 that the bore of the cuticular pore has a diameter of 0.1 μm, which means its area is about 10^{-14} m^2. At threshold pressure, therefore, that volume change of 10^{-24} m^3 inside the cell will be accomplished by fluid flowing through this pore a distance of 10^{-10} m. Detecting such a threshold displacement seems relatively easy, as mammalian stereocilia are called on to sense a deflection of 10^{-10} to 10^{-11} m at threshold[150], and, even in crickets[151], nerve impulses are produced when their *kinocilia* are deflected *at their base* by 10^{-12} m.

The reference to crickets provides a good example that kinocilia can act as sensors as well as motors. To understand how this can happen, and to give a clearer insight into the mechanism proposed here, we need to take a general survey of kinocilia and the basal bodies at their base.

8.4/g Kinocilia, basal bodies, and centrioles

A good starting point is a wide-ranging review[152] by Flock in which he discusses sensory transduction in hair cells with a focus on the inner ear. Both

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[148] Hillman, D. E. and E. R. Lewis (1971). Morphological basis for a mechanical linkage in otolithic receptor transduction in the frog. *Science* 174: 416–419.
[149] Lewis et al. *The Vertebrate Inner Ear* (1985).
[150] When extrapolations are made from much higher levels. Dallos, P. (1996). Overview: cochlear neurobiology. In: *The Cochlea*, edited by P. Dallos et al. (Springer: New York), 1–43. I would maintain that stereocilia are part of the cochlea’s high-level sound detection system and cannot respond to deflections of 10^{-11} m and tilts of 0.01°.
[151] Thurm, U., et al. (1983). Cilia specialized for mechanoreception. *J. Submicrosc. Cytol.* 15: 151-155.
[152] Flock, Å. (1971). Sensory transduction in hair cells. In: *Handbook of Sensory Physiology*, edited by W. R. Loewenstein (Springer: Berlin), vol. 5.1, 396-441. For the reader unfamiliar with the anatomical arrangement of kinocilia and stereocilia, reference to Fig. A2 may be helpful. Stereocilia are really modified microvilli and emerge in distinct arrays from the hair cell’s solid cuticular plate. Each hair cell usually has a single kinocilium that is strategically placed next to the stereocilia. The kinocilium is a true cilium, with a complex internal structure which in some cases allows it to move (hence the name). Both OHCs and IHCs bear them. The kinocilium is a complex, distinct system that does not emerge from the cuticular plate. In mammals, the kinocilium is lost at birth, inviting the question, *what does it do*?

stereocilia and kinocilia are given treatment. He considers what the adequate stimulus may be, and comes to the conclusion that the hair cell, in all organs, is basically a directionally sensitive displacement detector. Static pressure is considered as a stimulus at one point (ibid., p. 400), but with no direct evidence in its favour, is put to one side. Engström's lever-action of stiff hairs, and excitation at the basal body, are mentioned as possibilities but a decision on the validity of these ideas must await improved knowledge of cellular mechanics since "the final mechanical transformer is probably of molecular dimensions" (p. 408). The 9-fold symmetry of the kinocilium and its basal body is set out, and its strange disappearance in adult mammals stated. The key question of the role of the kinocilia in transduction is tackled, and among some possibilities the best answer, to my mind, provided (p. 424): *that a kinocilium can act as a motile cilium in reverse*.

This idea was first proposed in 1958 by Gray and Pumphrey[153] after study of the tympanic organ of a locust[154]. A micrograph of its sensory unit shows the distinctive '9 + 2' pattern seen in kinocilia, and Gray and Pumphrey suggested that a kinocilium can, through a reversed sense, play the part of a receptor. Lowenstein and Wersäll echoed the reverse transduction idea after examining the arrangement of kinocilia in the labyrinths of guinea pigs and rays[155]. If cellular electricity can drive a motor, then all the parts exist for motion of the motor to generate an electrical signal[156,157].

Experiments leave no doubt that kinocilia can act as sensors, although this work has largely been confined to insects and molluscs. Thurm describes[158] how by slight modification of the ciliary shaft – removing dynein arms and adding some extra components – the cell can become sensory. In this way a stimulus can modulate the receptor current and depolarise the cell (producing excitation), or hyperpolarise it (producing inhibition). The outstanding common feature of this work is that the final stimulus is not bending of the hair, per se, but *pressure* (or force) exerted *at the base* of the hair, in the region of the basal body.

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[153] Pumphrey being the same person whom Gold worked with earlier.
[154] Gray, E. G. and R. J. Pumphrey (1958). Ultra-structure of the insect ear. *Nature* 181: 618.
[155] Lowenstein, O. and J. Wersäll (1959). A functional interpretation of the electron-microscopic structure of the sensory hairs in the cristæ of the elasmobranch *Raja clavata* in terms of directional sensitivity. *Nature* 184: 1807–1808.
[156] Lewis et al. (1985) p. 126.
[157] Wiederhold, M. L. (1976). Mechanosensory transduction in "sensory" amd "motile" cilia. *Annu. Rev. Biophys. Bioeng.* 5: 39–62.
[158] Thurm (1983).

Thus[159,160], if a cilium is made completely pliable, by application of a drug (chloral hydrate), they collapse and lie flat; nevertheless, pressure on the cell surface still produces a generator potential. Conversely, making the cilium completely rigid – so it is unable to bend – still allows potentials to be generated. Another relevant observation is that paramecium, who swim, and sense, using motile cilia, still show depolarising and hyperpolarising responses after removal of their cilia.

The focus therefore falls on the basal body. How can this organelle make the test-tube-like entity shown in Fig. 8.11 into a pressure transducer? There are no definite answers, but by assembling a few clues the following intriguing picture takes shape.

All cilia have at their base a characteristic 9-fold structure, the basal body, from which they grow. Unique among cell organelles, they have a constant size, shape, and ultrastructure[161]. The mystery comes from their close similarity, if not identity, to the other vital but enigmatic cell organelle, the centriole[162]. Centrioles, which come in matched pairs usually at right angles to each other, spring from the nucleus and play a major part in organisation, structure, polarity, growth, division, and death of cells[163]. They are ubiquitous throughout the animal kingdom, indicating they have some vital function to perform. A suggestive image is that, like a spider in a web, centrioles sit at microtubular centres of the cell[164], surveying activity. Both sensory and non-sensory cells have centrioles, but in a sensory cell one centriole will migrate to its surface and grow a cilium, its partner usually nearby[165]. Theories about centriole function, and the significance of the 9 arms, abound[166,167], but here I will briefly set out one model that provides a meaningful picture of what the sensory fontanelle could be doing.

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[159] Alkon, D. L. (1983). The role of statocyst sensory cilia in mechanotransduction. *J. Submicrosc. Cytol.* 15: 145-150.
[160] Alkon, D. L. (1983). Sensory function of cilia. *J. Submicrosc. Cytol.* 15: 173-176.
[161] Afzelius, B. A. (1983). Basal bodies in the immotile-cilia syndrome. *J. Submicrosc. Cytol.* 15: 111-114.
[162] Wheatley, D. N. (1982). *The Centriole: a central enigma of cell biology*. (Elsevier: Amsterdam).
[163] Impressively, so-called killer cells use miniature spears to kill target cells, and the latter can survive such barbs until struck in the centriolar region (Afzelius, 1983, p. 112).
[164] Schliwa, M. (1992). Cell polarity and centrosomes. In: *The Centrosome*, edited by V. I. Kalnins (Academic: San Diego), 331-351.
[165] Wersäll, J., et al. (1965). Structural basis for directional sensitivity in cochlear and vestibular sensory receptors. *Symposia on Quantitative Biology* 30: 115–132. Fig. 31.
[166] Albrecht-Buehler, G. (1992). Function and formation of centrioles and basal bodies. In: *The Centrosome*, edited by V. I. Kalnins (Academic: San Diego), 69-102.
[167] Wheatly highlights one scientist as saying “Biologists have long been haunted by the possibility that the primary significance of centrioles has escaped them” (p. 185).

According to Brinkley and Stubblefield[168], the entity we see in Fig. 8.14 is a motor with rotating blades. The blades are angled from one end of the centriole to the other, to give the simplified turbine-like structure seen in Fig. 8.15. In this figure, the inner dynein arms (Fig. 8.14) that presumably drive the turbine have been omitted. An attractive feature of the turbine model is the straightforward way by which flow through the core of the system might cause the blades to rotate.

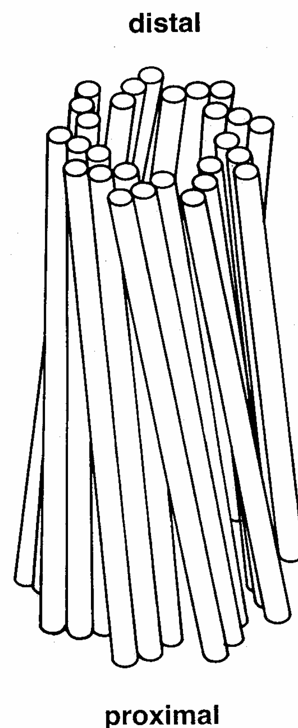

Fig. 8.15. Simplified structure of a centriole or basal body showing the turbine-like arrangement of the blades. [From Albrecht-Buehler (1992) and used with the permission of Academic Press]

A slightly different formulation was put forward by Bornens[169], who considered that the centriole may act more like a stepper motor so that portions of the device either (a) rotated or (b) oscillated backwards and forwards a number of degrees in an electrical field between the triplets. In either way, a torsional oscillator of fixed vibrational frequency might result, allowing the centriole to act as a pacemaker for the rest of the cell. Any slight change in the device's mechanical environment could affect its oscillation frequency and be detected by comparison

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[168] Brinkley, B. R. and E. Stubblefield (1970). Ultrastructure and interaction of the kinetochore and centriole in mitosis and meiosis. *Adv. Cell Biol.* 1: 119-185.
[169] Bornens, M. (1979). The centriole as a gyroscopic oscillator. Implications for cell organization and some other consequences. *Biol. Cellulaire* 35: 115-132.

with the frequency of the companion centriole[170]. The unexpected finding of silicon in centrioles may be coincidental but it highlights the possibility of electrical interactions.

The idea that the basal body of outer hair cells could be an electrical motor or, acting in reverse, a rotational sensor is given credence by consideration of a comparable structure in motile bacteria. These microorganisms propel themselves using a rotating flagellum, and ultrastructural studies show it is a miniature electric motor, complete with stator, rotor, drive shaft, bushing, universal joint, and helical propeller[171,172]. Notably, the motor can rotate well into the auditory range, at up to 1700 revolutions per second[173]. Unifying the picture we have been constructing, the rotary motor originates from the cell's basal body. A micrograph and diagram of such a rotational motor are shown in Fig. 8.16.

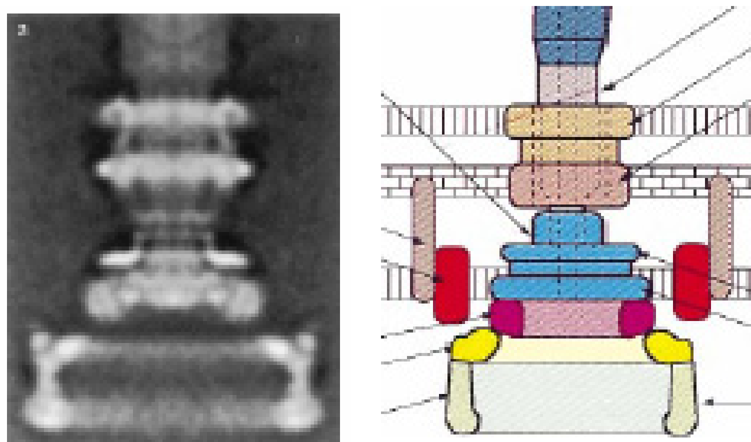

Fig. 8.16. A rotary motor, 45 nm in diameter, located at the basal body of a bacterium's flagellum. At left is a micrograph of the basal body in cross-section, showing the multiple rings of the stepper motor. At right is a schematic diagram (labels omitted) illustrating the major molecular components. A similar motor, minus the flagellum, could occur at the basal body of an outer hair cell, and might operate in reverse as a sensor of pressure: fluid flow through the pore might induce rotation. [From Thomas et al. (1999) and Namba and Vonderviszt (1997), with permission of the National Academy of Sciences USA and Cambridge University Press]

If we identify the centriole of the outer hair cell as a flagellum-less version of this motor, configured to act as a sensor, then the forces acting on it are minute. If the

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[170] In this connection, we may even consider quantum mechanical effects, as in paired detectors set at right angles which sense electron spin (Stern–Gerlach effect).
[171] Namba, K. and F. Vonderviszt (1997). Molecular architecture of bacterial flagellum. *Q. Rev. Biophys.* 30: 1-65.
[172] Thomas, D. R., et al. (1999). Rotational symmetry of the C ring and a mechanism for the flagellar rotary motor. *Proc. Natl. Acad. Sci. U. S. A.* 96: 10134-10139.
[173] Magariyama, Y., et al. (1994). Very fast flagellar rotation. *Nature* 371: 752.

intracochlear pressure at threshold is 0.5 mPa, as before, this would produce a force on the sensory pore of 5×10^{-6} pN. In comparison, the force required for activation of a typical mechanosensitive ion channel[174] is 10–20 pN, so in these terms the oscillator system must operate with orders of magnitude greater sensitivity. Is this realistic? A legitimate doubt may be raised here, although we point out that there are possibilities for resonance – outer hair cells are graded in length from base to apex[175], suggesting something like a Helmholtz resonator – and there are also two positive feedback mechanisms that might be expected to improve sensitivity.

The first is mechanical, and relates to the observation that the Poisson ratio for the OHC is more than 0.5. This means that as the electromotility mechanism is engaged and the cell changes length, the volume will tend to change accordingly. For a hyperpolarisation (say) induced by initial increase in pressure on the sensory pore, the effect is towards increasing the volume – which will decrease the internal pressure and cause an increased pressure difference across the sensory pore; the result will be increased hyperpolarisation – a positive feedback effect. Depolarisation (induced by a transient decrease in intracochlear pressure) will lead to an opposite sequence, but producing positive feedback once again. The positive feedback loop acts so as to make the cell's contents appear more compressible than under static conditions. In effect, the cell contains not air but super-compressible or rarefied air, a conclusion with which the ancient Greeks would agree (see Chapter I1). The idea is revisited below (§D 8.5).

The second positive feedback loop is electrical. Electromotility causes OHCs to change length in accordance with imposed electrical fields[176]; in the same way, the cochlear microphonic – the electrical potential generated by collective outer hair cell activity – might induce further length changes in those cells. The essential role of the cochlear microphonic in affecting the responses of hair cells (that is, it is not just an ineffectual by-product of cochlear action, an epiphenomenon) was first put forward

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[174] Morris, C. E. (2001). Mechanosensitive ion channels in eukaryotic cells. In: *Cell Physiology Sourcebook: A Molecular Approach, Third Edition*, edited by N. Sperelakis (Academic Press: New York), 745-760. (p. 749).

[175] Pujol, R., et al. (1992). Correlation between the length of outer hair cells and the frequency coding of the cochlea. In: *Auditory Physiology and Perception (Advances in the Biosciences volume 83)*, edited by Y. Cazals et al. (Pergamon: Oxford), 45-51.

[176] Brownell, W. E. (2002). On the origins of the outer hair cell electromotility. In: *Hair Cell Micromechanics and Otoacoustic Emission*, edited by C. I. Berlin et al. (Delmar Learning (Singular Publishing): Clifton Park, NY), 25-47.

by Davis[177], and was taken up by Gold and a number of others since. Here, we can see that the electrical voltages generated by sensory transduction in the hair cell could feed back to it in a way that led to additional length changes and voltages, again a positive feedback loop. Sharks and other electrosensitive fish can detect voltage gradients[178] of less than 0.5 μV/m, presumably by using this mechanism. These animals have specialised hair cells modified for electrical sensing; interestingly, these cells lack stereocilia, but retain a kinocilium[179].

Whether these refinements to the proposed scheme are sufficient to improve, to detectable levels, its sensitivity to direct pressure stimuli is a complex matter and beyond resolution here. Nevertheless, I take the position that there are a sufficiently wide range of indicators to suggest that the pressure-detection scheme may be physically possible; I hope I have set out the reasoning clearly enough for the reader to take seriously this somewhat unorthodox possibility. Further reinforcement of it is presented in the Appendix.

8.5 Towards a functional integration

We have described the outer hair cell as a pressure vessel, a proposition that provides a functional role for the cross-ply reinforcement of its cell wall as shown in Fig. 8.13. It also accommodates the finding that the cell wall supplies about 4 times greater tether force – is about 4 times stiffer – than neighbouring cells[180]. Moreover, although the cell wall is stiff, and can resist internal (and external) pressure changes, its structure allows it – like a garden hose – to change length.

Another characteristic of outer hair cells is that their Poisson ratio appears to deviate from the 0.5 value expected if their contents were incompressible. This fact (in some measurements, the ratio appears to be between 1.85 and 2.3) was used to argue that the cell in fact contains an air bubble (§D 8.4/e). Extending the argument raised in §D 8.4/f, a Poisson ratio greater than 0.5 implies that the volume gets larger

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[177] Davis et al. (1934).
[178] Zakon, H. H. (1988). The electroreceptors: diversity in structure and function. In: *Sensory Biology of Aquatic Animals*, edited by J. Atema et al. (Springer: New York), 813-850.
[179] Zakon (1988) // Kalmijn, A. J. (2000). Detection and processing of electromagnetic and near-field acoustic stimuli in elasmobranch fishes. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 355: 1135-1141.
[180] Zelenskaya, A., et al. (2005). Evidence for a highly elastic shell–core organization of cochlear outer hair cells by local membrane indentation. *Biophys. J.*

as the cell gets shorter, meaning that the air bubble appears as if it is has *negative compressibility*[181]. As flagged in §D 8.4/e, the measurements may be suspect because they were done over extended periods with pipettes penetrating the cell, a situation which may not represent the actual dynamic situation.

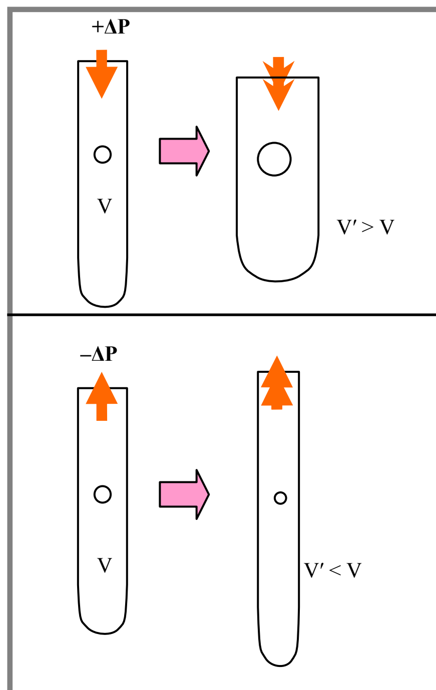

Fig. 8.17. Consequences of a Poisson ratio greater than 0.5. When positive pressure (top) is applied to the cell, inward flow occurs through the cuticular pore. This stimulus creates an electrical potential that leads, via electromotility, to contraction of the length and expansion of the radius – the volume expands. Because water in the cell is incompressible, the air bubble must expand, and the expansion will also tend to draw more flow through the pore (the pressure difference across the pore has increased), giving *positive feedback*. Conversely, when the outside pressure decreases (bottom), the flow is oppositely directed, and electromotility causes the cell to lengthen and its volume decrease, this time decreasing the size of the bubble but again *increasing the initial flow* through the pore. Overall, a pressure increase outside has led to an increase in internal volume, and a negative one to a decrease – therefore the cell displays a *negative* compressibility, and instead of absorbing energy it emits it. In response to oscillating intracochlear pressure, the cell oscillates in volume and radiates energy back into the ear canal.

Allen (2001) notes how the maximum physical value of Poissons ratio is normally 0.5, but he gives an explanation of how complex cell properties could lead to larger values. If the dynamic value exceeds 0.5, it produces interesting

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[181] Mathematically, the situation can be described, due to N. H. Fletcher, as follows. For a material with Poisson ratio > 0.5, its length necessarily decreases when external pressure is applied. Thus, for a cell of length L, it becomes $L\,(1–d)$. The Poisson ratio, s, is the increase in radius compared to decrease in length, so the radius R becomes $R\,(1+sd)$ and the cross-sectional area A becomes, neglecting second-order terms, $A\,(1+2sd)$. The volume V therefore becomes V', where $V' = V\,(1–d)\,(1+sd)^2 = V\,(1–d)\,(1+2sd) = V\,[1+ (2s–1)d]$. So if $s > 0.5$, $V' > V$ and the cell expands under external pressure.

implications. The diagram (Fig. 8.17) explains how a large Poisson ratio in an outer hair cell can lead to a *positive feedback system*.

A negative compressibility would show we are dealing with a thermodynamically unstable system. It was mentioned earlier that biological materials with negative compressibilities are known[182], but little work has been done in this field. While we cannot exclude these exotic materials in outer hair cells, it is difficult to go past air as a simple and highly compressible substance. If the actual ratio exceeds 0.5, the compressibility will exceed that of air – something that in this case could perhaps be called rarefied air. In the next chapter further synthesis will be presented that sets out how this pressure detection could work electrophysiologically and how further physical principles could be used to further increase the sensitivity of the system.

[182] See footnote 34. The reference cited is followed by a reply in which (its footnote 3) it is calculated that a negative compressibility requires a Poisson ratio exceeding unity.