Hi Dick (CC Jont),

Thanks for the info; I downloaded the CARFAC code and will look into running it with our two-tone stimuli.
Nigel and I will present the AGC story at the ISH meeting; when submitting the final draft of the Chapter for the Proceedings, I will send you a copy, too.

I will try to give a point-wise answer to your question on amplification, I hope we will find an opportunity to talk about this.

1. Experimental evidence against
Just yesterday JARO accepted our paper that presents evidence against power amplification. It reports our experimental and analytical work on cochlear energy flux. I am attaching a pdf. From the Discussion:

Our key experimental finding is that the energy transport by the traveling wave slows down abruptly at a frequency-dependent location. As in a traffic jam, this results in a densification of the energy, which boosts the amplitude. To a casual observer his may look like amplification but in reality it is mere spatial focusing of the available acoustic energy.

2. Wrenched physics: "negative impedance"
The literature contains claims of "negative damping". The first kind is based on inverse models (e.g. de Boer), in which a basic hydrodynamic model (e.g. 2-dimensional waves) is combined with an unknown point impedance of the partition. This point impedance function z(freq, x) is then estimated by fitting the model to basilar membrane data. Apart from the mathematical ill-posedness of inverse models, there is a grave physical problem: given the dimensions of the organ of Corti relative to the wavelength, the interaction of the partition with the fluid cannot be captured by point impedance. It is not even close to being a reasonable approximation (see the JARO MS, Discussion). Thus the inverse method fits a completely unphysical model to the data, and it is not surprising that this yields nonsensical outcomes like negative damping. The same is true for the second kind of claim, which is based on attempts to derive point impedance from intracochlear pressure measurements (Olson). There is no such thing as point impedance of the partition.

3. Physiological implausibility
There has been 30 years of spin talk since the dicovery of OHC "motility", but Ashmore's finding that it is voltage driven, and thus subject to shunting of the AC transduction potential by the cell membrane, has been lethal from the start. Figure 1 of our 2014 MoH chapter (also attached) shows estimates of the amplitude of this receptor potentlal near hearing threshold, based on a straightforward combination of published data in the literature:

(Only after submitting the chapter did I realize that these estimates are still too optimistic. They are derived from peak motion at best place, i.e. after the alleged amplification.) Any cell physiologist will tell you that such tiny voltages are too low to do anything coherent in a living cell. Even worse (see the MoH Chapter), due to the low number of MET channels per OHC, the S/N ratio of the receptor currents is so poor that, even if motility were sensitive to such tiny voltages, that would completely ruin detection of soft tones instead of helping it. Motility would be hijacked by the random opening and closing of channels in their rest position, and this would swamp soft sounds.

4. Amplfication solves no known problem
From stapes to characteristic place the traveling wave spans ~2 cycles. In order to impose major dissipative losses during such a short trip, the amount of damping needs to be excessive. To illustrate, waves on a canal (e.g. generated by a boat) are very similar to cochlear waves (both are surface waves in an incompressible fluid), and they travel tens of cycles before decaying. The textbook story that amplification is needed "to overcome friction" makes no sense; there is no phsyics behind that statement. Are we to believe that, on the one hand, nature is unable to build a passive waveguide that mediates waves without excessive loss over a distance of one or two millimeters, but on the other hand has solved this problem, not by a more sensible choice of materials, but by constructing a costly and vulnerable, ultra-high-frequency, mechanical amplifier? You don't have to be a creationist to find this ridiculous. Let me translate it to the eye: nature was unable to produce a transparant material for constructing eyeballs to guide the light to the retina. Therefore our eyeballs, which are opaque by themselves, are spiked with little light-sensitive glow-in-the-dark cells. When they receive light, they glow, and that's how our eyeballs pass the light to the retina. More seriously, the obsession of many of our colleagues by the cochlea's sensitivity to soft sounds (a rather trivial problem for a nanotechologist like mother nature) has distracted the attention from the true engineering challenges of the cochlea. In the very last paragraph of the Discussion of the attached JARO paper, we argue that dissipation is a blessing and not a curse (& we give Jont the credit he deserves).

5. Wrenched logic
"When you damage OHCs, the vibrations of the BM in response to soft sounds are strongly reduced. This proves that OHC mechanically drive these vibrations. Moreover, spontaneous emissions prove that OHCs can generate energy." Sure.
"Cutting nerves that enervate muscles causes paralysis. This proves that these nerves deliver the energy that moves our limbs. Moreover, the recording of action potentials from these nerves confirm that they produce energy."

6. Alternative ideas
It is not easy to understand the peaking of the traveling wave in a way that is consistent with BM data, but to invoke the magic of little motile cells feels like cheating. It is a deus ex machina -- fine for the theater, but too cheap for science. To quote Ray Meddis, who mailed me a while ago: "The idea that the system is detecting a signal in order to amplify it in order to detect it has always seemed odd. However, people are very impressed by movies of dancing OHCs and move quickly to conclusions that are not logically supported by what they have seen." (You see, Jont and I are not alone.) Inspired by our experimental finding of the deceleration of the energy transport (see JARO paper), I constructed an alterative model that carries traveling waves and acts as a spectral analyzer, yet is completely passive and linear (http://www.pnas.org/content/111/40/14548.short). I am not claiming this is how the cochlea works, but it certainly shows that with a bit of imagination and basic physics, it is possible to obtain cochlea-like behavior without invoking unphysical concepts like negatve damping or unphysiological concepts like phase-locked mechanical feedback at 150 kHz (in a dolphin).

There are always more arguments, but these are the main ones. In my opinion, the amplifier story has caused a deplorable stagnation (and even retreat) in the field. Over the last 30 years there has been no progress whatsoever in answering the real questions: (1) How does he cochlea realize frequency selectivity? (2) How does it realize dynamic range control? And by "how" I mean: mechanisms, physics. Regarding these basic questions, there is no trace of consensus. There are as many theories and models as there are authors. In a healthy scientific field, a hypothesis failing to produce any tangible result in 35 years would be dumped -- well, people would at least welcome alternatives. Not so in cochlear mechanics, where amplification is regarded as an semi-religious dogma and defended in ways that sometimes scare me. (I admire Jont Allen's persistence.) The field is in a bad shape. There is virtually no interaction between theoreticians and experimentalists. Most current theoreticians and model builders have a poor understanding (if any) of the basic physics of waves in fluids. All of this makes me hesitant to encourage young people to enter cochlear mechanics. This a sad state of affairs, because the big questions are still open, so it should really be an exciting field! My main hope is the advent of new experimental techniques like OCT.

Well, this is probably more than you were asking for, but I could't help myself :-)

Best regards,

m.

From: richard.f.lyon@gmail.com [richard.f.lyon@gmail.com] on behalf of Richard F. Lyon [dicklyon@acm.org]
Sent: Saturday, May 23, 2015 7:03 PM
To: M.L. van der Heyden
Subject: Re: cochlear AGC and tuning

Marcel,

My model description is in the book draft at http://www.dicklyon.com/hmh/
and open-source code is on github at https://github.com/google/carfac

Lacking, data, I picked AGC loop filter time constants of 2, 8, 32, and 128 ms (representing a range of mechanisms from very fast to nearly syllable); book section 19.4 AGC Filter Temporal Response; the actual AGC response times are faster than these filter time constants, by a level-dependent "speedup factor" that I discuss in section 11.6 AGC Dynamics. I'd love to have some actual data, and try to tie time constants to actual mechanisms eventually.

I had a good chat with Jont Allen at the ASA meeting this week in Pittsburgh. As usual, he's trying to convince me that the cochlear has no active amplifier. It seems that you are he are the prime voices for this point of view; I'd like to understand better your reasoning and evidence on this.

Dick