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Do We Really Care Whether our Clients Have Cochlear Synaptopathy?

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In the last several issues *Canadian Audiologist* has had articles on the topic of cochlear synaptopathy: Adam Sheppard in issue 6, 2017, Colleen LePrell in issue 2, 2018, and in this issue, Martin Pienkowski in "Science Matters," and Alfarghal Mohamad. Neurological measures such as wave I amplitude, and the SP/AP ratio have been proposed as possible metrics of cochlear synaptopathy.

Four things become apparent: (1) We don't have a single (or even an established battery of) test(s). (2) We do not know the prevalence in humans; (3) We don't have a cure or treatment; and (4) "Cochlear synaptopathy" is perhaps one of the most difficult phrases to pronounce. Yet, its discussion is everywhere from research journals, to academic and professional conferences, to the popular media.

The idea of getting to the bottom of "cochlear synaptopathy" is quickly becoming the poster child of our field and whoever gets to the bottom line first will be our next Einstein.

Of course, this is hyperbole but the recent interest in the study of cochlear synaptopathy is in part related to our sloppiness in our own field. With the advent, first of evoked potentials and later brain scanning techniques, we no longer need to worry about the separation of "sensory" from "neural" in our everyday clinical analyses. Even the central auditory processing work in the 1960s was initially to help diagnose central neurological sites of lesion. The orientation of clinical audiology in the 1950s and 1960s was to distinguish at all costs, "merely sensory" hearing losses, from those with potentially fatal "neural" involvement.

Today, we boldly just use the phrase "sensori-neural."

As far as a client with a sensori-neural hearing loss is concerned, as long as there are no asymmetries in pure tone thresholds or in supra-threshold phenomena such as speech-based testing, it is assumed that the hearing loss is primarily a communication issue and not a diagnostic/medical/surgical issue. And for the vast majority of clients, this assumption is quite valid. After all, even if there was a slight neural involvement, there isn't much that we would offer our clients. Even in the case of a diagnosed acoustic schwannoma, if it was small and not causing any difficulties, a common approach would be to just monitor any progression.

We have all had clients where they appear to function more poorly than their audiogram would suggest. Clinically, assistive listening devices may be recommended along with their hearing aids. But it would be nice to get to the underlying cause(s) of why they are not performing as well as someone else with a similar pure tone audiogram. Part of the difference may be cochlear synaptopathy, and part may be neural difficulties at other parts of the central auditory system. And part may be due to undiagnosed cochlear pathology.

Audiograms use puretones that are detected by low threshold efferent fibres in the cochlea. While these may function well, it is also known that high threshold fibres can possess pathologies that are not detected by any of our commonly used clinical tests. Tests that assess the functioning of high threshold fibres that are implicated in supra-threshold stimuli such as speech and music would be quite useful. And, of course, these tests would need to be clinically efficient, taking only a small amount of time- seconds rather than minutes.

One such test I have been using for the past decade is a gross measure of JND for musical note complexes. I have a clinic piano, but really any inexpensive second hand electronic toy piano would work – just have the client play the notes in a scale going from low to high, using both the white and the black keys. If two adjacent keys do not sound different in pitch, then this is evidence of severe cochlear damage. It may not be a “cochlear dead region” but it may be a region to avoid over-amplifying with hearing aids. Frequency transposition away from this/these region(s) for speech would be helpful, and gain reduction for music would also be helpful. This keyboard/piano test takes about 30 seconds.

Such tests do not necessarily help us diagnose the site of pathology but can help us in locating the pathological frequency regions using stimuli other than potentially low-level pure tones.

The area(s) of damage may be sensory, may be at the location of the synapse, or may be neural- clinically we can do an end run around the diagnosis and still be of assistance to our clients.

Yet, it would still be interesting to be able to know the site of pathology in our clients’ auditory systems, so personally I will still continue to pour through the articles on “cochlear synaptopathy”- besides, I love saying that phrase!

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