Advanced simulations for the differential diagnosis of sensorineural pathologies

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Abstract

The MAPsim simulator reconstructs an acoustic signal from the auditory-nerve firing pattern predicted by the Matlab Auditory Periphery (MAP, Meddis et al.) model. Stimuli reconstructed with various simulated sensorineural pathologies were presented to normally-hearing participants in psychophysical tasks. This enabled studying the impact of specific pathologies on performance in traditional tasks, and developing tasks designed to detect specific pathologies.
An emergent property of MAP was its prediction that efferent reflexes play an important role in dynamic range adaptation at the auditory-nerve level, the acoustic reflex shifting rate-level functions towards a context level and the medial olivocochlear reflex sharpening auditory-nerve sensitivity around that level. A 'normal' hearing implementation of MAPsim was validated through speech-reception thresholds (SRTs) in noise being only 1 dB above those obtained with unprocessed stimuli. Knocking out efferent reflexes significantly reduced dynamic-range adaptation and caused a 4 dB SRT inflation due to the saturation of high-spontaneous-rate fibres.

Simulating general deafferentation showed that stochastic under-sampling alone underestimates the effect of deafferentation: 90% of fibres needed to be knocked out before an appreciable effect on SRTs could be measured. Instead, the reduction of efferent signals caused by deafferentation, combined with stochastic under-sampling, yielded a 3 dB SRT elevation at 75% deafferentation. ITD discrimination was more sensitive to stochastic under-sampling alone (due to temporal fine-structure degradation) than SRTs. The combined effect of reduced efferent signals further elevated thresholds, from 20 μs with unprocessed or 'normal' processed stimuli to 60 μs at 75% deafferentation. Both tasks saw an exponential threshold elevation as the number of remaining fibres was successively quartered.

The effect of cochlear synaptopathy (selective knockout of low spontaneous rate fibres) on temporal envelope (TE) processing was assessed by comparing natural- and unvoiced-speech SRTs in modulated noise. Simulated synaptopathy elevated natural and unvoiced SRTs by 1 and 4.5 dB from the 'normal' condition, respectively. In an amplitude-modulation detection task, synaptopathy elevated thresholds more (from 3 to 5 dB) for lower than for higher modulation rates (16-64 Hz range). Overall, synaptopathy severely impaired TE processing, when the selective knockout of high spontaneous rate fibres did not.

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Gradual outer haircells (OHCs) knockout and endocochlear potential (EP) reduction were also simulated for SRTs in steady-state-noise, broadband-speech-modulated noise and speech maskers. While 75% OHC knockout led to an overall 2 dB elevation of SRTs, the benefits of dip-listening and fundamental-frequency discrimination (derived from SRT differences between masker types) did not significantly reduce. However, a total knockout of OHCs removed and halved the benefits of dip-listening and F0 segregation, respectively. The simulated reduction in EP from 100 to 12.5 mV led to similar endpoints, but via a gradual reduction in dip-listening and F0-segregation benefits.

Overall, our findings show (1) the importance of efferent reflexes in simulating normal and impaired hearing, (2) that stochastic under-sampling alone underestimates the effects of general deafferentation, (3) that synaptopathy may be easily diagnosed by probing patients' temporal envelope processing abilities and (4) that MAPsim paves the way to revealing the psychophysical signatures of sensorineural pathologies.