Amplitude Modulation Revisited

DOI: 10.3766/jaaa.20.10.1

“It’s déjà vu all over again.” —Yogi Berra

If the alternate binaural loudness balance (ABLB) test launched the era of diagnostic audiologic evaluation, the difference limen (DL) for amplitude modulated tones was responsible for its global spread. The now classic 1948 paper by Dix, Hallpike, and Hood in England generated a good deal of interest in the use of the ABLB test in differentiating Ménière’s disease from acoustic tumor, but the test was inherently limited to persons with unilateral loss. There remained a pressing need to devise a diagnostic test that could be applied to persons with bilateral losses. In Switzerland, Ernst Lüscher, an otolaryngologist, reasoned that, if loudness recruitment, as measured by the ABLB procedure, differentiated cochlear from retrocochlear sites, then one need only devise an indirect measure of whether loudness was growing more rapidly than normal as intensity increased. He thought that, if this were the case, then the just noticeable difference in sound intensity, the difference limen (DL), should be smaller than normal, since the number of DLs corresponding to a given range of loudness must be compressed into a smaller range of intensities. Although subsequent experience has revealed that the situation in cochlear disorders is considerably more complicated, the concept did enjoy some clinical success. Lüscher assigned his laboratory assistant, a young engineer named Jozef Zwislocki, to fabricate a device with which to measure the intensity DL for pure tones and to carry out DL measures on patients at the ENT clinic in Basel. Their 1948 paper stirred worldwide interest in the intensity DL as an indirect measure of loudness recruitment, ergo cochlear site of disorder.

Shortly thereafter, the Amplivox company in England produced a commercial DL unit that could be coupled to a clinical audiometer in order to carry out DL testing. At Northwestern University (NU), Raymond Carhart, intrigued by the Lüscher and Zwislocki paper, bought one of the Amplivox units and installed it in the audiology clinic at the NU Medical School in downtown Chicago. I became involved in this area when Carhart “suggested” that an undergraduate student, such as myself, might want to carry out some research using the new Amplivox device. It must be said that measuring DLs with this gadget would give any modern psychoacoustician an apoplectic fit. The depth of modulation was varied by manually turning a potentiometer at a “slow” rate until the patient indicated that the previously steady tone now “wobbled.” Nevertheless, this experience ultimately led to the SISI test and many subsequent years of personal interest in diagnostic audiological evaluation. Over the years I became convinced that abnormally small DLs were related not so much to loudness abnormalities as to the unique distortion consequent on the cochlear pathology. But interest in such matters waned as the ABR and otoacoustic emissions effectively upstaged their venerable behavioral antecedents.

In this issue of JAAA, investigators Brian C.J. Moore, of the University of Cambridge, and Vinay, of the All India Institute of Speech and Hearing, have revisited amplitude modulation (AM) in the ear with cochlear loss, evoking a long-suppressed sense of déjà vu in this aging investigator. Using a sophisticated psychometric procedure several orders of magnitude superior to those early efforts in the 1950s, Moore and Vinay asked how the DL for AM is affected by dead regions in the cochlea. Briefly, they found that for frequencies below the edge frequency of the dead region, AM-detection thresholds were lower for ears with dead regions than for ears without dead regions, but for frequencies above the edge frequency of the dead region, AM-detection thresholds were higher for ears with dead regions than for ears without dead regions.

These findings highlight the complexity of the situation in the diseased cochlea and the importance of basic research in understanding the fundamental nature of the disorder for which a diagnostic test is contrived. Understanding the key role of dead regions in the cochlea can help to put many previous observations and contradictions in diagnostic test results into proper perspective. Perhaps they explain, for example, why the old DL measures did not seem necessarily related to degree of loss as one would have expected. In any event, Moore and Vinay have reopened and illuminated a long and almost forgotten chapter in the history of our profession.

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REFERENCES


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