Few new tools have generated as much interest during the past decade as the measurement of otoacoustic emissions (OAEs). Because of their sensitivity to the status of outer hair cells, many people had the initial thought that OAEs might prove to be the elusive “objective test of hearing” that would permit prediction of threshold sensitivity loss without recourse to the more time-consuming, and often frustrating, behavioral measures. It is becoming clear, however, that the goal of a truly independent objective test of the magnitude of hearing loss remains elusive. The very property of exquisite sensitivity to outer hair cell status, which at first blush seemed so appealing, appears to be the source of so much intersubject variability that OAE amplitude does not bear a strong predictive relation to degree of loss.

Paradoxically, one of the growing clinical applications of OAEs appears to be its value in predicting not how much loss a person has, but how much loss he/she does not have. While the presence of an attenuated or absent emission is an imperfect predictor of how much loss exists, the presence of a robust emission effectively rules out (with the relatively rare exceptions noted below) virtually any degree of sensory hearing loss. In this issue of JAAA, authors Musiek, Bornstein, and Rintelmann, in their paper “Transient Evoked Otoacoustic Emissions and Pseudohypacusis,” show how this principle can be applied to the clinical problems of malingering and psychogenic loss. In five cases, they illustrate the important point that, when pseudohypacusis is suspected, OAE testing can provide valuable supplementary information pointing toward better sensitivity than is revealed behaviorally. Thus, the OAE test should play an increasingly important role in the evaluation of patients suspected of pseudohypacusis.

Musiek et al make the equally important point, moreover, that, even as an indicator of normal hearing, OAE is imperfect. Retrocochlear disorder, especially eighth nerve disorder, and perhaps even selective inner hair cell damage, can produce significant hearing loss without concomitant change in OAEs. Thus, it is possible to see, both in theory and in practice, (1) reduced hearing and reduced OAEs, (2) reduced hearing and normal OAEs, (3) normal hearing and normal OAEs, and (4) normal hearing and reduced OAEs.

The key to the successful use of OAE as a diagnostic tool resides, therefore, in the careful interpretation not of OAEs alone, but of the combination of OAEs and other audiometric information. In this sense, the fruitful clinical application of OAEs is not unlike the clinical application of immittance data. A particular outcome of the measure, per se, may be ambiguous, but combining the outcome with the results of other audiometric measures can lead to relatively precise prediction. For example, the presence of normal emissions is, in isolation, not entirely definitive. It could mean (a) normal hearing, (b) pseudohypacusis, or (c) a retrocochlear disorder. Nor is the combination of normal OAE and depressed sensitivity entirely unambiguous. Both pseudohypacusis and retrocochlear site are still possibilities. But a battery consisting of OAEs, the pure-tone audiogram, and the auditory brainstem response (ABR) can differentiate among the three possibilities quite precisely. If all three components of the three-test battery are normal, one can predict, with some confidence, normal auditory function. If OAEs are normal, audiometric sensitivity is depressed, and ABR is normal, there is a strong case for the presumption of pseudohypacusis. Finally, if OAEs are normal, the audiometric level is depressed, and ABR is abnormal, a suspicion of retrocochlear site should be entertained.

Musiek et al put it succinctly: “Otoacoustic emissions must be used judiciously by the experienced clinician in conjunction with other clinical tools...”

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