Diabetes is a complex multisystem disease that requires routine monitoring for known complications affecting the renal, visual, and peripheral nervous systems. Research has hinted at an increased risk for hearing loss in diabetic patients, but confounders of noise exposure, ototoxic drug exposure, presbycusis, and known syndromes that affect both glucose metabolism and cochlear function make it difficult to establish this association (1–5). Currently, there are no formal recommendations for screening for hearing loss in diabetic patients (6).

The article by Bainbridge and colleagues (7) in this issue reports on the risk for hearing loss in persons with self-reported diabetes. In a cross-sectional study, they used the National Health and Nutrition Examination Survey to determine the relative risk for sensorineural hearing loss in a community-based random sample of patients who reported a history of diabetes. They found that persons with diabetes were at increased risk for hearing loss (adjusted odds ratio, 2.2 to 2.4). The degree of hearing loss ranged from mild to moderate, causing deficits that would be difficult to detect without screening but would pose substantial impairment for communicating.

One previous study (2) provided evidence that diabetes affects hearing. Cullen and Cinnamond (2), who used a large population-based data set, reported an odds ratio for hearing loss of 1.41 in non–insulin-dependent diabetic patients. A possible explanation for the divergent results between this study and Bainbridge and colleagues’ (7) is that Cullen and Cinnamond (2) excluded patients younger than age 48 years and patients with non–insulin-dependent diabetes. The exclusion of young persons is important because Bainbridge and colleagues found the strongest association between diabetes and hearing loss in the youngest age group, in which other causes of hearing loss (which would dilute the effect of diabetes on the incidence of hearing loss) are uncommon.

The pathophysiology underlying diabetes-associated hearing loss is unclear, which allows for speculation. A leading candidate explanation is the effect of diabetes-related microvascular disease on the cochlea (8). Unlike the retina, the cochlea is virtually impossible to examine visually, and its microcirculation is embedded in the temporal bone, which cannot be effectively examined, even under surgical conditions. Previous postmortem studies have invoked microvascular disease affecting the stria vascularis, the organ responsible for generating endolymph, which serves as the driving force for mechanotransduction of hair cells. Temporal bone studies have demonstrated pathology in the stria vascularis and in hair cells of postmortem studies of the inner ear in diabetic adults (9, 10). Despite these findings, the pathophysiology of impaired hearing in diabetes remains uncertain. First, pathologic change in the stria vascularis and the outer hair cells is a known consequent of aging. Second, some investigators have looked for these changes in diabetic patients and failed to find them. Third, studies in diabetic animals have shown decreased hearing function but no abnormality in hair cells or the stria vascularis (11, 12).

Although hearing loss researchers have a large battery of physiologic tests to help localize a lesion—including studies of outer hair cell function, eighth nerve function, behavioral response to sound, localization of sound, detection of words in noise, and central auditory processing—we do not have the cochlear equivalent of a fundoscopic examination. Thus, we have a very limited ability to confirm the enticing hypothesis of impaired microcirculation of the ear (8). Two previous studies (13, 14) indicate that distortion product otoacoustic emissions can detect early hearing loss in diabetic patients. Distortion products are an objective physiologic test of outer hair cells; they are measured through microphone recordings of the sounds reflected back from the ear in response to 2 pure tone stimuli. This form of hearing test is commonly used to evaluate neonates for universal newborn hearing screening. The 2 studies indicate that distortion product otoacoustic emissions are more sensitive than routine behavioral audiometry in detecting cochlear dysfunction in diabetic patients (13, 14). Downstream effects of hyperglycemia, such as elevated glucose levels in the cerebrospinal fluid or in the perilymph, could also contribute to cochlear dysfunction in diabetic patients. Metabolic studies in animals have demonstrated that glucose can pass from serum to the perilymph and endolymph, but no one has convincingly shown elevated glucose levels in these fluids in animals with hyperglycemia (15).

This study reveals some potential directions for future research. Can we develop medical or surgical therapies that could halt the progression of diabetic cochleopathy? The possible relationship of glucose levels in the serum, perilymph, and endolymph suggests other potential avenues of investigation that could prove helpful in managing diabetes-associated hearing loss. Evidence has shown that close monitoring and regulation of blood glucose decreases the likelihood of renal and retinal complications of diabetes. Would tighter glycemic control have a similar protective effect against the development of hearing loss? The existence of diabetes-related hearing loss increases the possibility of a corresponding condition: diabetic vestibulopathy (16). Physicians have attributed impaired balance and gait in diabetic patients to impaired proprioception. It is possible that diabetic patients also have impaired vestibular input because the microcirculation that maintains cochlear function is entangled with the circulation of the vestibular organs.

What are the clinical implications of diabetes-related hearing loss? On the basis of Bainbridge and colleagues’
study, the American Diabetes Association could recommend that physicians include routine audiometry in the annual test battery for diabetic patients. Bainbridge and colleagues provide level B evidence from a large cohort in a well-conducted cross-sectional study to support this recommendation. However, their study cannot tell us whether the severity of diabetes is related to the likelihood of hearing loss. Its cross-sectional design means that we cannot be sure that hearing loss in diabetic persons was permanent. Although unlikely, some of the patients with hearing loss may have had a temporary shift in their hearing threshold. Nevertheless, pure tone audiometry is a relatively low-cost test that can lead to interventions—specifically, hearing amplification—that can improve an individual’s productivity and quality of life. However, at this time, we do not have therapies to restore hearing in patients with progressive hearing loss, and this study does not provide evidence that hearing loss can be either prevented or stopped as a result of early detection.

Counseling patients with mild to moderate hearing loss to avoid loud, prolonged noise exposure and ototoxic medications can be helpful in eliminating other sources of progressive hearing loss. We have few current therapeutic options for progressive hearing loss from any cause, and the study of hearing loss in diabetic patients could lead to important progress in new techniques of studying and treating microvascular disease of the inner ear. In many cases of mild to moderate hearing loss, patients are not aware of what they cannot hear; thus, screening for hearing loss in individuals at risk could lead to interventions that would affect their ability to communicate, their productivity, and their safety.

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References