tile FSHD, these findings are commonly positive.[2] Additionally, one's serum creatinine kinase level is commonly elevated in FSHD.

In diagnosing FSHD, facial hypotonia deserves priority over bilateral eye findings. The bilaterality of Coats disease in these patients may be subtle; thus, we suggest examination under anesthesia and fluorescein angiography for these patients. Finally, we thank the authors for demonstrating that though some bilateral cases remain unsolved, vigilance and persistence can reveal a diagnosis.

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In reply

We appreciate the comments by Drs Sheth and Shapiro and their points on the importance of facial hypotonia in fascioscapulohumeral dystrophy. Indeed, our patient showed features of facial hypotonia with a flat, expressionless appearance and a sagging in her lower lip.[1] At initial examination, we were concerned about this appearance and related systemic problems, particularly muscular dystrophy. However, systemic examination by geneticists and specialists, including a battery of genetic tests and even a muscle biopsy, was unrevealing. We agree with Drs Sheth and Shapiro that, in retrospect, the facial hypotonia was truly striking, now that the diagnosis has been established. In our case, persistence in seeking a diagnosis with second neuromuscular consultation eventually led to the correct clinical diagnosis of fascioscapulohumeral dystrophy. This was confirmed by genetic testing, which showed a 4q35 deletion.

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Diabetes and Glaucoma

Dr Sommer[1] reminds us that associations found in large data sets may not be correct or causally related. He also points out the need for common sense when interpreting data from clinical trials or epidemiologic studies. He believes that investigators of the Ocular Hypertension Treatment Study (OHTS) may not have followed these admonitions.

One of the major goals of the OHTS was to understand the factors associated with the development of primary open-angle glaucoma (POAG) in individuals with ocular hypertension.[2] There is an extensive literature on diabetes mellitus and the risk of POAG. Unfortunately, the literature is contradictory, and it is difficult to draw conclusions about this association. Therefore, we thought it was important to assess this association in the OHTS. In our initial analysis published in 2002,[3] we used patient self-report of diabetes. Dr Sommer correctly points out that self-report may be inaccurate, that some participants may not have been adequately tested, and that other participants may have been misclassified by their self-report. The same objection can be raised, however, about most aspects of medical history, including hypertension, cardiovascular disease, migraine, family history of glaucoma, and race, to name a few. Ideally, investigators would be able to corroborate patient self-report with laboratory tests, review of medical records, or both; however, this is usually not feasible because of cost and lack of access to patient medical records, assuming the patient has a consolidated medical record. The OHTS used self-report of diabetes mellitus knowing that the information could be flawed. We could have chosen not to assess the relationship between diabetes mellitus and incident POAG, but that would have undermined a major goal of the study. We could have performed the analyses but not published the results; that would violate scientific integrity. We could have performed blood tests and reviewed medical records, but we did not have the personnel, funding, or access. We could have chosen not to report a finding that seemed controversial or improbable (Dr Sommer's reference to common sense), but that fails to bring potentially new information to clinicians and scientists for their review and possible additional studies.

When the original predictive model indicated that diabetes mellitus might be protective against developing POAG,[3] many of the OHTS investigators were perplexed by this finding. We tried to indicate our concern and caution in the original article. We tried to improve our data by asking the OHTS participants additional questions about diabetes mellitus and its treatment. The updated data are improved, but it is likely that they are still imperfect. With the new information published in 2008,[4] the protective effect of diabetes mellitus was not observed. We believe we had a responsibility to publish these results, which did not corroborate our previous analysis.

The true association of diabetes mellitus and POAG remains unclear. We hope our articles and Dr Sommer's

editorial will stimulate other investigators to find the true association.

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Classification of the Corneal Dystrophies

In my recent article titled "Elucidating the Molecular Genetic Basis of the Corneal Dystrophies: Are We There Yet?"[1] that was published in the February 2007 issue of the *Archives*, I presented a review of the genes and chromosomal loci that have been implicated in the pathogenesis of the corneal dystrophies, a revised framework for classifying the corneal dystrophies, and information regarding how molecular genetic analysis can be incorporated into everyday clinical practice. I should have brought to the readership's attention the International Committee for Classification of Corneal Dystrophies (IC3D), whose specific purpose is to devise a new, clinically relevant nomenclature system for the corneal dystrophies that accurately reflects our current knowledge based on genetic information. I have been a contributing member of this committee, which is supported by the Cornea Society, since it was founded in 2005. I did not mention the IC3D in my recent article to avoid the impression that my article reflected the consensus opinion of the IC3D, which it does not. However, I express regret to my colleagues on the IC3D for not acknowledging this international collaborative effort in my article, and I look forward to the publication of the IC3D report, which should become the definitive corneal dystrophy reference.

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