Tear secretion and tear film function in insulin dependent diabetics

Editor,—I read the article by Goebbels1 with interest and would like to share a few opinions with you.2 Goebbels stated in his paper that BUT results did not differ between diabetics and controls and also pointed out that BUT is a very rough test for the detection of tear film stability. He found low Schirmer test values and conjunctival squamous metaplasia in diabetics compared with controls. He hypothesised that a decrease in reflex tearing inducing conjunctival surface damage, disturbance of the trophic function of the tear film, or metabolic alterations might be responsible. First of all, we believe with many others that BUT is an unreliable and incorrect test for tear film stability when performed carefully.3,4 In addition, a keen observation of the breaking tear film provides a lot of information on the minute changes on the ocular surfaces. We found in our study that BUT scores, Schirmer test values, central corneal sensitivity, and goblet cell density were significantly lower in NIDDM patients compared with controls. NIDDM patients also had significantly higher squamous metaplasia grades. We showed that tear film function and impression cytology variables significantly fared poorly in those patients with diabetes with peripheral neuropathy. Decreased corneal sensitivity and poor metabolic control without any correlation with duration of diabetes and status of retinopathy. We believe that the ocular surface disease in diabetes is characterised by squamous metaplasia and goblet cell loss which seems to evolve in close proximity to the status of metabolic control and peripheral neuropathy. Corneal and conjunctival epithelial damage caused by disruption of tear quantity and quality and diabetic neuropathy may be important determinants of diabetic ocular surface disease.

Our final comment and request to all researchers who carry out impression cytology. It is worth mentioning that the methodology of the procedure should be reported in each paper with photographs of the samples so that we can compare and refine our own procedures despite variability in cytological techniques and difficulties in comparing impression cytology studies with one another. Besides, no impression cytology study should be without information on figures of squamous metaplasia grade and goblet cell densities. Absence or presence of mucin pick up of filter papers must be mentioned without fail since such observations prove noteworthy; mucin being one of the major components in increasing the tear film stability and the wettability of the ocular surface.

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BOOK REVIEWS


This book includes contributions from experts in the field of ophthalmic immunology. Topics covered in the book are concisely summarised, providing relevant anatomical information, clinical presentation, and appropriate treatment of various immune mediated intraocular diseases, as well as ocular adnexal diseases and orbital pathology. To accomplish the objectives of the book, the editors imported the editors and experts who carry out impression cytology. The paper that BUT results did not differ between diabetics and controls. He hypothesised that a decrease in reflex tearing inducing conjunctival surface damage, disturbance of the trophic function of the tear film, or metabolic alterations might be responsible. First of all, we believe with many others that BUT is an unreliable and incorrect test for tear film stability when performed carefully.3,4 In addition, a keen observation of the breaking tear film provides a lot of information on the minute changes on the ocular surfaces. We found in our study that BUT scores, Schirmer test values, central corneal sensitivity, and goblet cell density were significantly lower in NIDDM patients compared with controls. NIDDM patients also had significantly higher squamous metaplasia grades. We showed that tear film function and impression cytology variables significantly fared poorly in those patients with diabetes with peripheral neuropathy. Decreased corneal sensitivity and poor metabolic control without any correlation with duration of diabetes and status of retinopathy. We believe that the ocular surface disease in diabetes is characterised by squamous metaplasia and goblet cell loss which seems to evolve in close proximity to the status of metabolic control and peripheral neuropathy. Corneal and conjunctival epithelial damage caused by disruption of tear quantity and quality and diabetic neuropathy may be important determinants of diabetic ocular surface disease.

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and immunomodulation is uninspiring and cyclosporin A is even described as a new drug. The chapters on immunological mechanisms underlying uveitis are rather disconnected as they are written by different authors so there is no common theme and they could be quite confusing to the novice. Nevertheless, the chapters on cytokines and HLA are informative but more could have been written on the latter topic and it cries out for a diagram. There are a large number of colour figures but not all of good quality; some fail to show the pathology and some are unnecessary. Diagrams explaining the taking of intraocular specimens would have been useful, as would the inclusion of treatment algorithms using corticosteroids and cyclosporin A for immunosuppression (although one does appear in the chapter on Behchet’s disease). The layout is unexciting and this is basically just another textbook on uveitis. In the age of multimedia, a more imaginative layout with shading, coloured text and diagrams, and boxes highlighting key points would have made it much more readable. The non-uveitis chapters are too brief and just as they start to whet one’s appetite for more—they end. Yet there are some good chapters, particularly on orbital inflammations (thyroid eye disease is not included), ICG, birdshot, and AIDS. The chapter on Behchet’s disease briefly mentioned the standard, recognised ISG classification but failed to document it. The most glaring omission was the topic of corneal graft rejection. Apart from a short paragraph on rejection lines it was never mentioned.

This book is overambitious as it tries to cram intraocular, conjunctival, corneal, and orbital inflammations into one text and fails to succeed. I am unsure what market the book is aimed at but it may have a place on some library shelves. Ophthalmology residents, however, should keep their credit cards firmly in their wallet.

PHILIP I MURRAY

NOTICES

Vision 2020: the cataract challenge
The latest issue of Community Eye Health (34) discusses cataract blindness and surgery with an editorial by Allen Foster. For further information please contact Community Eye Health, International Centre for Eye Health, Institute of Ophthalmology, 11–43 Bath Street, London EC1V 9EL. (Tel: (+44) (0) 20-7608 6909/6910/6923; fax: (+44) (0) 7250 3207; email: eyeresource@ucl.ac.uk) Annual subscription £25. Free to workers in developing countries.

Residents’ Foreign Exchange Programme
Any resident interested in spending a period of up to one month in departments of ophthalmology in the Netherlands, Finland, Ireland, Germany, Denmark, France, Austria or Portugal should apply to: Mr Robert Acheson, Secretary of the Foreign Exchange Committee, European Board of Ophthalmology, Institute of Ophthalmology, University College Dublin, 60 Eccles Street, Dublin 7, Ireland.