CHANGES IN REFRACTION IN DIABETES

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—Dr. Reid's letter in your last issue gives me the opportunity in referring to this subject again to thank correspondents from different countries for their courtesy in supplying me with additional bibliography of reported cases of this condition. With my thanks I must also tender my apologies for overlooking their work. As the additional bibliography may be of interest I have appended it.

Dr. Reid refers to the group I classed as anomalous cases. In reality it is probable that no such group exists; it was made up of those which were impossible to classify at all, from lack of accurate information or control. As I understood them, Mr. Laws's cases were not anomalous; they were examples of hypermetropia occurring "soon after the institution of (diabetic) treatment"—*i.e.*, after a decrease in blood sugar content.

From the information available Dr. Reid's case cannot scientifically be classed at all, since sugar-data at the time of change were unknown; in this sense it is anomalous. The patient, however, seemed to have had a decreasing sugar-content before she was admitted to hospital, which Dr. Reid admits "seemed to have been a variable quantity, and easily controlled." I should suggest that it might well have been so at the time when hypermetropia occurred, and that, while with the information given no one can dogmatize, it might have varied "according to rule" as probably as "contrary to rule." The recovery under treatment is certainly what one expects.

Dr. Reid wonders if these changes may be due to some "disturbance" of the endocrine glands: the mechanism of the change he does not enlarge upon. The only way I can, if I may so express it, coerce these into a share in the phenomenon is this. All vital processes are, ultimately, energy transformations whose rate of interchange is biologically adapted by an extremely complex mechanism. It has been shown experimentally that iodine increases the conductivity of tissues by increasing the permeability of the inter- and intra-cellular membranes, and it is probable that, by thus acting as a sensitizer, the katabolic group of glands (of which the thyroid is the chief) act as controllers of permeability, and, through this, of reactivity and metabolism generally. Thus is produced the dull irresponsive cretin in contradistinction to the exquisitely responsive sufferer from Graves' disease. It would appear to follow that by an increase in the permeability of the epithelium of the lens capsule through overaction of these glands, any osmotic change would be facilitated.

Whether Dr. Reid has this in his mind I do not know; but it is evident that, even so, any influence of these glands is, in the present case as in metabolic processes generally, merely that of catalysers—they cannot be credited with responsibility for the fundamental cause. In the present state of our knowledge, I prefer to view the appeal to the endocrine glands with a large amount of suspicion. They tend to fulfil to the physician that very comfortable rôle of Ever-Available Last Resort that does the appendix to the surgeon and the endometrium to the gynaecologist.

Yours, etc.,

W. S. DUKE-ELDER.

LONDON, W.

May, 1925.

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[Schiötz (*Klin. Monatsbl. f. Augenheilk.*, Vol. LXVII, Beilageheft, 1921) reports a case of the sudden development of -4.5 D.S. of myopia in pregnancy with chronic nephritis and uraemic symptoms: recovery one week after delivery; diabetes excluded. This is interesting. In drawing a parallel between the aetiology of diabetic and albuminuric retinitis, I have already noted the osmotic derangement that occurs in nephritis.—D.E.]

OBITUARY NOTICE

JOHN FERGUSON CARRUTHERS, M.D.

WE regret to announce the death of John Ferguson Carruthers, who took up ophthalmology somewhat late in life. After an adventurous career in many parts of the world, Carruthers qualified as M.D. with honours at Edinburgh. He practised for several years at Revelstoke in British Columbia, where he was Medical Officer of Health. From 1903-5 he studied at Moorfields Eye Hospital, and then took up ophthalmic practice at St. Peter Port, Guernsey. Soon after the outbreak of the war he was appointed Ophthalmic Specialist at the Royal Herbert Hospital, Woolwich, where he remained till 1922. His duties in this post were extremely arduous, and it cannot be doubted that his unremitting work undermined his health. In 1922, he was appointed Ophthalmic