Obituary

E. H. Cameron, MB, ChB, FRCSEd

Ernest Hugh Cameron, honorary consultant in ophthalmology to the Royal Infirmary, Edinburgh, died on 26 February at his home in Lasswade, Midlothian, at the age of 94.

Born in 1890, and educated at George Watson's College and at the University of Edinburgh, he graduated in medicine in 1910 and obtained the fellowship of the Royal College of Surgeons of Edinburgh in 1914. He was appointed house-surgeon in the eye wards of Dr George Mackay at the Royal Infirmary, and then spent a short time in general surgery. In January 1916 he joined the RAMC, serving mainly in Salonika and India. Returning to Edinburgh he became clinical tutor and later assistant ophthalmic surgeon in the Eye Department of the Royal Infirmary. In 1932 he was appointed surgeon in charge of wards.

Elected a member of the Ophthalmological Society of the United Kingdom in 1924, he served on the council of the society in 1931–5 and again ex officio in 1949–51, when he was President of the Scottish Ophthalmological Club. He also much enjoyed the meetings of the Oxford Ophthalmological Congress.

An excellent clinician, with no inclination towards research, he devoted his great talents to the care of patients and the teaching of undergraduates, an art in which he excelled. Ernest was also a very accomplished musician, particularly on the clarinet, and he played the organ at Roslyn Chapel and later at Lasswade.

He married relatively late in life, and his marriage was a supremely happy one. He is survived by his wife, two daughters, and two sons, one of the latter being a doctor.

G. I. S.

Correspondence

Effect of continuous subcutaneous insulin infusion on retinopathy

Sir, I read with interest the article by Lawson et al. on the failure of continuous subcutaneous insulin infusion (CSII) to prevent the progression of proliferative and preproliferative diabetic retinopathy.1 The hopes of many physicians and their diabetic patients with florid retinopathy could be dashed after reading it. I don’t think this is right. The metabolic control (HbA1) of the described patients were insufficient in 5 cases (cases 2, 6, 7, 8, 9) and borderline in 2 cases (cases 3 and 11) with mean blood-sugar values all in the high range of normal or elevated. Nevertheless this could be considered as a fair result in otherwise difficult cases of diabetes mellitus. The outcome appeared worst in cases 2, 8, 10, and 11; the level of HbA1 of case 10 is unknown, that of the 3 others was too high or borderline, indicating a moderate control, instead of the reported good control in 3 of them in Table 1.

But this is not the point. What would have been the outcome without CSII in these patients, including microangiopathy elsewhere in the body? The authors raise the possibility of a detrimental effect of CSII on the retinopathy and point mainly to 2 possible causes. The first, the so-called induced imbalance of somatomedin and growth hormone levels, is in fact a restoration of balance with very beneficial potential effect on growth, in spite of the fall in growth hormone concentration.2 The effect on new vessel growth is not known, but uncontrolled growth can have a deleterious effect on the retinopathy, as is known in adolescent diabetics without CSII treatment. In this series only case 3 could have had this problem, but his deterioration was probably related to temporary loss of metabolic control because of an abscess.

The second point which possibly could account, according to the authors, for the worsening of the retinopathy during CSII treatment is the ‘probable' reduction in retinal blood flow by CSII. In fact it is not the flow which is important, but tissue hypoxia, and this causes secondarily increased blood flow. Normalisation of flow indicates normalisation of tissue oxygenation.

The primary cause of microangiopathy in diabetes is chronic hyperglycaemia. The mere fact that there is a progressive retinopathy points to insufficient metabolic control. It is probably too late when connective tissue strands appear on and in the retina, but before this stage is reached, every effort should be directed to normalise the blood sugar, including CSII, together with further photo-coagulations (diminishing retinal areas with presumed production of 'angiogenic factors' and leakage).

The introduction of CSII has brought new hopes for badly manageable diabetics with deteriorating retinopathy,3,4 and this is right. It cannot always totally prevent progression of retinopathy, so other measures remain important, including photo-coagulation. False hopes should not be raised by CSII, as the authors state, but surely it can be an excellent new tool in preventing or slowing down progression of diabetic microangiopathy.

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References