ADRENAL FUNCTION AND DIABETIC RETINOPATHY*

C. P. DANCASTER, B.Sc., M.D., M.R.C.P. and W. P. U. JACKSON, M.A., M.D., F.R.C.P.
From the Endocrine Research Group, University of Cape Town

Some characteristics of the retinopathy complicating diabetes suggest that adrenal hyperfunction might be concerned in its aetiology. Thus, diabetic retinopathy may develop or become aggravated during pregnancy, especially during the last trimester, when adrenal activity is probably increased, and the retinopathy may improve after delivery.[1,2] Postpartum pituitary necrosis, hypophysectomy and adrenalectomy have all been followed by a great improvement in diabetic retinopathy.[3-7] Retinal changes resembling those complicating diabetes have followed steroid administration,[2,8] although they have not been described in Cushing's syndrome. Diabetics with retinal disease have had heavier adrenal glands than uncomplicated diabetics.[9]

Studies of urinary and blood corticosteroids, however, have not proved that an association exists between adrenal function and retinopathy. Results have been conflicting; both increased and normal hormonal levels have been reported among retinopaths.[10-12] In the most comprehensive report of this nature, Jakobson could find no significant difference in the adrenal function of diabetics with and without retinopathy.[13] In fact his study appeared so decisive that we have been dissuaded from our earlier plan of enlarging our own work by including corticotrophin stimulation tests.

Material

Urinary steroids have been assayed in 81 diabetics — mostly hospital inpatients. 28 of these had retinopathy, which was graded as severe, moderate or mild, according to the numbers of haemorrhages and exudates present. No patient had retinitis proliferans.

The two groups, retinopaths and non-retinopaths, were age-matched, and an approximately equal number in each group were treated with insulin, oral hypoglycaemic agents, and diet only. Patients who had recently been in diabetic ketosis or suffered myocardial infarction were excluded. No patient had an elevated level of blood urea and all had normal chest radiographs. An attempt was thus made to eliminate any factors that might have affected adrenal function, or at least to distribute these factors equally among the 2 groups.

The diabetics belonged to 3 racial groups — European, Bantu and Coloured. As we had previously found no significant difference in the excretion of urinary steroids among diabetics of the 3 races,[14] they have been combined in the present study.

Methods

Two consecutive full 24-hour urine collections were made in each case. 17-hydroxycorticoids and 17-ketosteroids were measured on each sample, by the methods of Norymberski *et al.*[15] (1953), and Appleby *et al.*[16] (1954).

Results (Tables I and II)

In considering 17-ketosteroid excretion males and females are separated. The sexes are combined when comparing hydroxycorticoid excretion. 21 diabetics with retinopathy had a mean hydroxycorticoid excretion of 13·8 mg. per 24 hours, compared to a mean level of 11·3 mg. per 24 hours among 23 diabetics without retinopathy. The wide range in both groups is reflected by the high standard deviations (the range among non-retinopaths was 4·9 to

TABLE I. URINARY STEROIDS IN DIABETES
(*mg. per 24 hours*)

	Retinopaths			*Non-retinopaths*		
	Number	*Mean*	*S.D.*	*Number*	*Mean*	*S.D.*
17-hydroxycorticoids	21	13·8	7·5	23	11·3	5·6
17-ketosteroids						
Males	9	14·5	4·5	28	12·3	4·5
Females	19	9·4	4·0	25	13·6	4·2

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23·2). As a result, the slightly higher hydroxycorticoid excretion among retinopaths is not significant.

Likewise, although the mean ketosteroid excretion was slightly higher in 9 male diabetics with retinopathy (14·5 mg. per 24 hours), when compared to 28 diabetics without retinopathy (12·3 mg. per 24 hours), this difference is not significant. 25 female diabetics without retinopathy actually had a slightly greater mean 17-ketosteroid excretion (13·6 mg. per 24 hours) when compared to 19 females with retinopathy (9·4 mg. per 24 hours), but again, owing to the wide range, this difference is not significant.

Five diabetics with severe retinopathy had a slightly, but not significantly, higher hydroxycorticoid excretion than the milder retinopaths (14·7 mg. per 24 hours compared to 12·5 mg. per 24 hours). However, 7 retinopaths in whom the condition was graded as 'moderate' actually had a lower hydroxycorticoid excretion than a group of 9 diabetics who had less severe retinopathy (12·5 mg. per 24 hours compared to 13·5 mg. per 24 hours)

TABLE II. 17-HYDROXYCORTICOID EXCRETION IN DIABETIC RETINOPATHY

(*mg. per 24 hours*)

	Number	*Mean*
Severe retinopathy	5	14·7
Moderate retinopathy	7	12·5
Mild retinopathy	9	13·5

Conclusion

We found no biochemical evidence to incriminate excessive adrenal function as a factor in the aetiology of diabetic retinopathy.

We present these findings in partial confirmation of the conclusions of Jakobson.[13]

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