M. R. BOND AND LIONEL WOLMAN: NEUROTOXICITY OF ETHOGLUCID

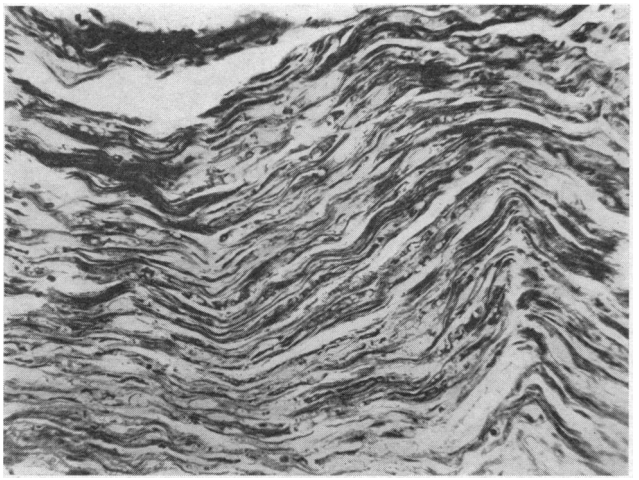


FIG. 6.—Distal part of same nerve as in Fig. 5 eight days after injection, showing severe degeneration and fragmentation of axons. (Silver. ×180.)

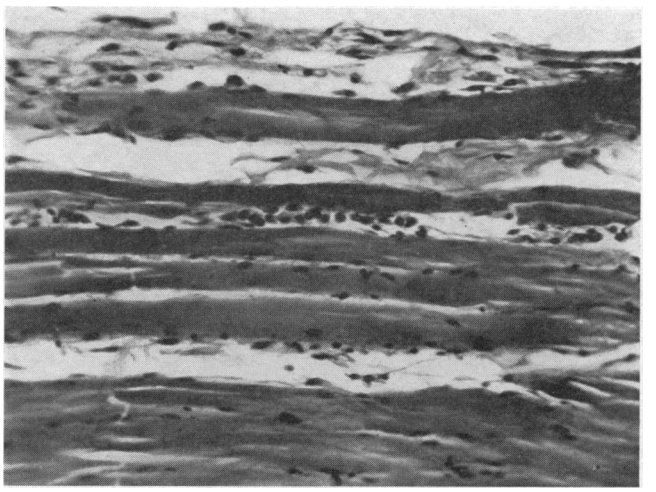


FIG. 7.—Focal necrotic myositis 10 days after injection. (H. and E. ×186.)

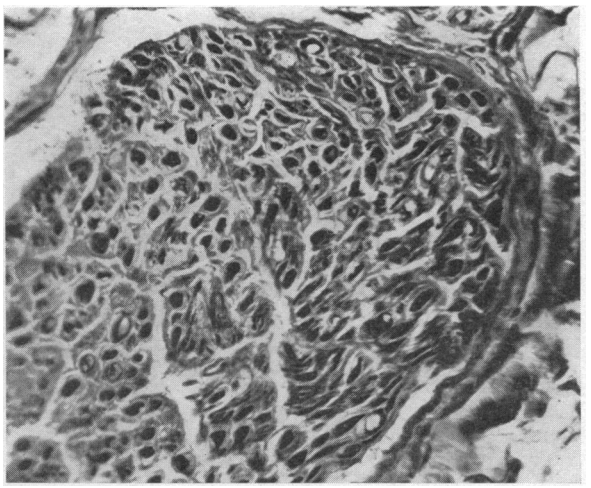


FIG. 8.—Transverse section of femoral nerve 10 days after injection, showing swollen vacuolated axis cylinders. (Silver. ×248.)

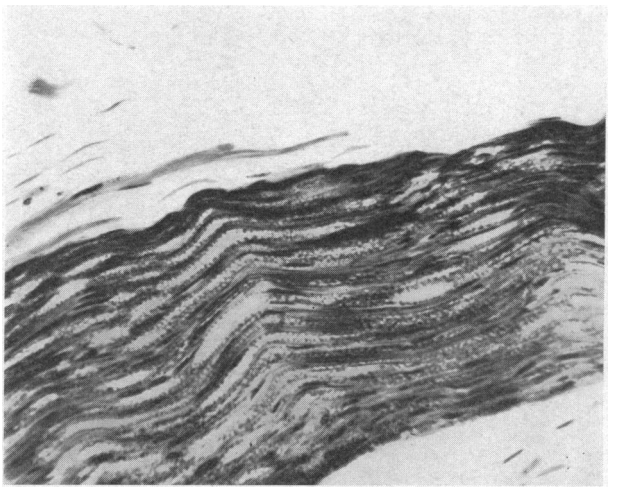
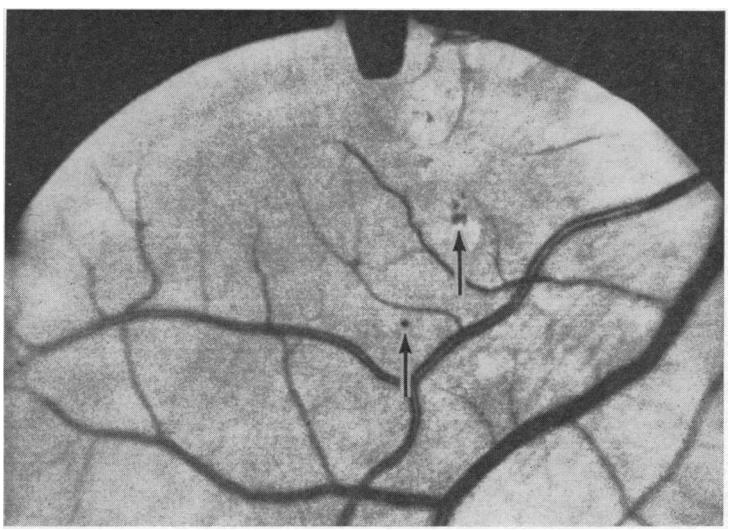


FIG. 9.—Longitudinal section of femoral nerve 14 days after injection, showing vacuolated myelin sheaths. (Myelin. ×245.)

D. J. GALTON: DIABETIC RETINOPATHY AND HAEMOCHROMATOSIS



Enlargement of right inferior temporal quadrant of retinal photograph showing several micro-aneurysms (arrowed).

Anterior mediastinal tumours cannot be safely approached by this method because of their relation to the large vessels in the anterior mediastinum.

I wish to thank Dr. L. H. Capel and Dr. A. H. James for helpful criticism, and Miss K. J. Graham, medical artist, Hillingdon Hospital, for the diagram.

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Medical Memoranda

Diabetic Retinopathy and Haemochromatosis

[WITH SPECIAL PLATE]

Brit. med. J., 1965, 1, 1169

It has been stated that cases of diabetes caused by haemochromatosis rarely develop diabetic retinopathy (Finch and Finch, 1955). Moreover, important reviews on haemochromatosis fail to mention that a retinopathy can occur (Sheldon, 1935 ; Beutler, Fairbanks, and Fahey, 1963). The following report shows the development of a retinopathy, indistinguishable from diabetic retinopathy, in a case of haemochromatosis.

CASE REPORT

The patient, a 57-year-old white man, has no family history of diabetes or haemochromatosis. His illness began in 1936, when he lost weight from 11 st. 7 lb. (73 kg.) to 7 st. 8 lb. (48 kg.). At the same time he became pigmented and noticed loss of hair from the arms, chest, and genitalia.

In 1950 he was referred to Hammersmith Hospital complaining of polyuria and polydipsia. The signs at this time were loss of hair over the axilla, chest, and pubis. He had pigmentation of the skin and testicular atrophy, and the prostate gland was small on palpation. The liver was enlarged but no spleen was palpable. Biopsy specimens were taken from the liver, skin, and testicle ; and each specimen contained an excess of haemosiderin ; in addition the liver showed cirrhosis. As the patient had glycosuria a glucose-tolerance test was performed ; this showed a diabetic curve (fasting blood sugar 237 mg./100 ml. ; highest blood sugar after 50 g. of oral glucose, 506 mg./100 ml.). A diagnosis of haemochromatosis was therefore made. It was noted at this time that he had no retinopathy.

In March 1963 it was observed in the out-patient department that he had signs of a diabetic neuropathy, with absent ankle reflexes and impaired sensation to touch, vibration, and positional movements below the knees. In addition he had a retinopathy.

The photograph (Special Plate) of this is of the right inferior temporal quadrant. It shows a cluster of microaneurysms in the upper segment. These changes did not interfere with his vision and he could read Jaeger 6 with both eyes.

The serum iron values and serum iron-binding capacities throughout this illness were:

	Serum Fe (mg./100 ml.)	Iron-binding Capacity as % Fe Saturation
Normal	120–175	... <30
March 1955	303	... 76
October 1956	252	... —
March 1963	198	... 74

An E.C.G. in 1963 showed low-voltage QRS complexes with flattened T waves. This suggested there was myocardial haemochromatosis.

DISCUSSION

This patient showed the classical tetrad of haemochromatosis —namely, skin pigmentation, diabetes, cirrhosis, and myocardial involvement (Finch and Finch, 1955). He also had a retinopathy which showed microaneurysms.

The most characteristic feature of diabetic retinopathy is the microaneurysm (Ashton, 1959 ; Dollery, Hodge, and Scott, 1963). However, fluorescein studies of the retina have revealed microaneurysms in cases of both benign and malignant hypertension, though these features are usually invisible with the ophthalmoscope (Dollery and Hodge, 1963). In this patient no blood-pressure has been recorded over 150/80 mm. Hg since his first attendance at hospital in 1950. Moreover, his microaneurysms were visible by ophthalmoscopy. His retinopathy was therefore almost certainly diabetic in type.

It remains to show that the patient's diabetes was due to haemochromatosis. From the clinical history the time relations would suggest this. The haemochromatosis began in 1936 and diabetes developed suddenly 14 years later. By this time there was sufficient iron deposition in his testes to cause hypogonadism ; his liver biopsy showed cirrhosis and excessive deposition of haemosiderin, though there was no laboratory evidence for impaired liver function (a usual finding, see Finch and Finch, 1955). Assuming that iron deposition occurred at the same rate in the pancreas as in the other tissues (Pollycove, 1961), it is likely that the sudden onset of diabetes in 1950 was in fact due to haemochromatosis. This being so, the diabetic retinopathy developed within 14 years from the onset of diabetes, the diabetes occurring as a complication of haemochromatosis.

The probable reason why this has rarely been described before is that in most instances of haemochromatosis the diabetes is present for less than one decade before death, so that the late degenerative sequelae of diabetes are rare.

I am indebted to Professor R. Fraser for permission to publish this case ; I also wish to thank Dr. C. T. Dollery for permission to use the retinal photograph.

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