of the enzyme into the circulation due to anoxia (Highman and Altland, 1960).

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Biochemical Findings in Suprarenal Adenoma

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It is often believed that in cases of Cushing's syndrome due to suprarenal adenoma the levels of excretion of adrenal steroids are unaffected by the administration both of corticotrophin and dexamethasone. Exceptions to this rule have been reported but are extremely rare.

CASE REPORT

A woman aged 47 was referred to the out-patient department by her general practitioner as a case of Cushing's syndrome. She complained of lack of energy for three years, gain of a stone (6.3 kg.) in weight, and increasing fine hirsutism of the face. She had noticed that she bruised easily, and a recent wound on her leg had had to be skin-grafted as there was delayed healing. In 1958 she had had a hysterectomy for menorrhagia, and in 1960 she was treated by a psychiatrist for depression. For one year she had had numbness and tingling in the thumb and index finger of the right hand.

On examination she presented the classical features of Cushing's syndrome, with moon facies, high colour, and fine hirsutism. There was marked increase in body fat with slim arms and legs. There were no striae, but she had several bruises on her legs.

Her blood-pressure was 170/100 mm. Hg, and the only other abnormal finding was sensory loss of the distribution found in the carpal-tunnel syndrome. Visual fields were probably normal.

Investigations gave the following results: Hb 94%, W.B.C. 9,400/c.mm., eosinophils 60/c.mm., P.C.V. 51%. Bleeding and clotting times were normal. Glucose-tolerance test showed a "lag" curve. Serum sodium 139 mEq/l. Potassium 3.76 mEq/l.

A radiograph of the chest showed slight cardiac enlargement, but a radiograph of the spine did not show osteoporosis. A radiograph of the pituitary fossa was normal.

Twenty-four Hour Urinary Output of 17-Oxysteroids and 17-Oxygenic Steroids

Treatment		Volume of Specimen (ml.)	17-Oxysteroids		17-Oxygenic Steroids		Total Creatinine
			mg./24 Hours	mg/g. Creatinine	mg./24 Hours	mg/g. Creatinine	
None	Basal	1	2,235	29.5	19.9	32.9	1.48
	levels 2	1,550	12.9	9.6	19.2	14.2	1.35
40 Units of A.C.T.H. for 2 days	1st day	1,670	25.5	18.6	85.0	62.0	1.37
	3rd "	2,300	33.8	20.7	114.0	68.8	1.66
	4th "	1,460	14.2	12.5	33.0	29.0	1.15
Dexamethasone	1st day	1,400	12.8	10.8	23.7	20.1	1.18
	2nd "	1,660	11.2	9.8	24.4	21.2	1.15
	3rd "	1,390	13.6	10.9	42.1	34.9	1.25
	4th "	1,800	15.5	11.5	20.2	14.9	1.35

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A 24-hour urinary output of 17-oxysteroids and 17-oxygenic steroids are seen in the Table. Basal levels were estimated and repeated after (1) an infusion of 40 units of adrenocorticotrophic hormone (A.C.T.H.) given over a period of eight hours on two consecutive days, and (2) dexamethasone 2 mg. six-hourly for four days.

A report on radiography with presacral air insufflation (CO₂) read as follows: "Gas passed almost entirely into the left peri-renal region. Tomography showed good outlining of the left kidney and suprarenal, and the latter was considered to be within normal limits. Repeat examination demonstrated only the right kidney. The gas did not appear to pass between the latter and the suprarenal" (Dr. A. H. Isaacson).

On 5 May 1964 a right adrenalectomy was performed by Mr. A. J. Heriot through the bed of the eleventh rib. He found a small tumour of the suprarenal gland and a small accessory suprarenal which he also removed.

The pathology report was as follows: "Adrenal gland measures 6 by 3 by 2 cm. within which is a small tumour 1 cm. diameter. Gland weighs 12 g. Sections show the presence of a cortical adenoma composed of cells of the middle zone of the cortex zona fasciculata. There may be some hyperplasia also of the inner zona reticularis" (Dr. F. E. Dische).

COMMENT

Stimulation with A.C.T.H. in cases of adrenal hyperplasia causes considerable rise in glucocorticoid excretion. It has been suggested that stimulation of adrenal tumours with A.C.T.H. causes little or no rise (Bayliss, 1957).

However, Soffer *et al.* (1957) reviewed the literature and found six cases of adenoma, in four of which the response to A.C.T.H. was indistinguishable from that seen in adrenal hyperplasia. In the other two cases and in their own two cases there was little or no response to A.C.T.H. They concluded that a failure of the plasma 17-hydroxycorticoid level to rise strongly suggested a tumour, but the presence of an augmented response was not of diagnostic significance. Since then other cases have been described (Scott *et al.*, 1962); these included four cases of adenomas that gave no response to A.C.T.H. and one in which there was a good response. The case here reported is a further example of the latter.

I am grateful to Dr. B. J. Freedman for permission to publish this case, and to Dr. Raymond Greene, who first suggested that I should do so.

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