Adrenocortical Steroid Therapy and Pituitary-Adrenocortical Function

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The present report deals with the effect of adrenocortical steroid therapy on the pituitary-adrenocortical function in 43 patients treated with the steroids for various diseases in varying periods of time. The steroid used were chiefly dexamethasone (0.5 mg./tablet), betamethasone (0.5 mg./tablet) and paramethasone (1.0 mg./tablet), and the longest period of the therapy was 8 years. Therapeutic regimen in all cases was that with initial high suppressive dose (usually 6 to 12 tablets a day) which was gradually tapered off according to the symptoms, or decreased to maintenance dose suffice to control the symptoms.

Suppression of the pituitary-adrenocortical function occurred within a few day after institution of the treatment, which was reflected in decreased urinary 17-hydroxycorticosteroids (17-OHCS) excretion. Fashion of restoration of once suppressed adrenocortical function could be classified in 3 types according to urinary 17-OHCS; namely, those returned to normal range while they were still on steroids, those returned rather promptly, and those with persistent adrenal hypofunction after discontinuation of the treatment. In general, the less the dose administered and the less the period of treatment better recovery of the function was observed.

Approximately one-half of the cases responded well to ACTH (ACTH-Zn, 40 units for 3 consecutive days or more), however, rest of them had hypopituitary type delayed response which was indicative of suppressed ACTH secretion from the pituitary during treatment. All of these cases had low urinary 17-OHCS level after stopping therapy, however, the ACTH administration restored the pituitary-adrenocortical function in most of them. It may suggest that ACTH administration is of value in restoring once suppressed
function produced by the steroid therapy in many cases.

It is interesting to note that pituitary reserve test performed with 11-β-hydroxylase inhibitor (SU-4885; Ciba) indicated loss of pituitary reserve for ACTH in 2 cases. They were those who received the treatment for 14 months with alternate ACTH in dose of some 2,000 I.U., and for 8 years with some 1,800 tablets of various adrenocortical steroids.

When the pituitary-adrenocortical function in steroid treated cases was evaluated with urinary 17-OHCS levels after discontinuation of the treatment, type of response to ACTH and 11-β-hydroxylase inhibitor, in general, no hypoadrenocorticism was encountered in cases which received therapy less than 3 months in duration, or in cases which took 200 to 300 tablets in the same period. However, the treatment with higher doses or of longer period produced varying degree of hypoadrenocorticism; generally, the larger the dose and the longer the duration of treatment the higher the degree of the impairment was observed.