cover. It turns red on standing and absorbs the oxygen from the air in the container.

The principal object of this report is to bring carbitolic caustic to the attention of laboratory workers as a possible replacement for the well-known alcoholic caustic. There are probably many circumstances under which it should be a more desirable reagent. It is relatively nonflammable and can be used at temperatures considerably higher than the boiling point of ethanol if required to decompose a compound under reflux conditions.

Changes in Urinary Steroid Excretion and Correlated Metabolic Effects During Prolonged Administration of Adrenocorticotropic Hormone in Man[1]

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Adrenocorticotropic hormone was administered to a 46-year-old male, known to have an expanding lesion involving the sella turcica which had been arrested by roentgen therapy. This subject was chosen because of the presence of signs and symptoms of moderate secondary adrenal-cortical insufficiency, which could be expected to accentuate any adrenotropic response to the pituitary hormone.

The adrenocorticotropic hormone was prepared from hog pituitary glands.[3] Two gamma of this preparation was found to give an adrenotropic response in the assay method of Sayers and Sayers (*1*). Electrophoretic analysis revealed the presence of more than one component. The material contains the equivalent of 0.12 unit of oxytocic activity/mg.; growth-promoting, gonadotropic, and thyrotropic factors are not present in significant amounts.

The patient was maintained on a constant diet and, after reaching a steady state, was given intramuscular injections of 10 mg. of A.C.T.H. every 6 hours for 6 days (40 mg. daily).

Some of the metabolic effects observed are shown in Fig. 1, in which the following changes should be noted: A 7-fold increase in the daily excretion of "11-oxysteroids," as estimated by a modification of the method of Talbot, *et al.* (*2*); an accompanying 5-fold increase in the 17-ketosteroid excretion; an increase of 58 per cent in the daily urinary output of total nitrogen, with a concurrent rise in the excretion of uric acid. Urinary creatinine remained constant from day to day. The excretion of sodium was markedly decreased throughout the period of administration.

The marked increase in the excretion of "11-oxysteroids" may be taken as definite evidence of increased adrenal-cortical activity under the action of the A.C.T.H. used. The rise in 17-ketosteroid output to as high a figure as 12.2 mg./day would indicate that the major part of this material was, in fact, derived from the adrenal cortex, since the maximum output by

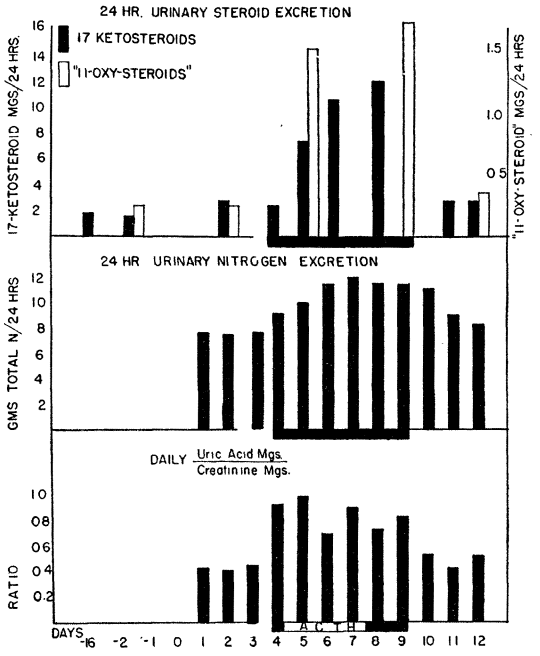

FIG. 1

even normal testes is only of the order of 5 mg./day. The simultaneous changes observed in the excretion of both types of urinary steroid may be taken in support of either of two hypotheses: (1) that 17-ketosteroids derived from the adrenal cortex are formed to a considerable extent by the oxidative removal of the side chains of C_{21} steroids, the production of the latter being controlled by A.C.T.H.; or (2) that the anterior pituitary gland, through its A.C.T.H., stimulates the independent production of both C_{21} steroids and 17-ketosteroids, or related androgens, by the adrenal cortex.

The increased nitrogen excretion parallels that of the "11-oxysteroids," which are known to promote the catabolism of body protein, whereas the sodium retention suggests the elaboration of a desoxycorticosterone-like substance. From the close correlation between the observed changes in the urinary excretion of steroids, nitrogen, and uric acid, it appears useful to regard the ratio of uric acid to creatinine as a measure of increased activity of the adrenal cortex during a period of stimulation. This has been confirmed by experiments, being reported elsewhere, which include the demonstration that patients with primary adrenal-cortical insufficiency fail to show this response.

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