potassium may prove fatal if an absolute or even relative hyperpotassemia develops. The readjustment of the serum electrolyte abnormality should be made promptly but not too rapidly and preferably by the oral route (chart 2).

Patients treated with ACTH and cortisone tend to retain sodium and water, so that edema develops. This is particularly undesirable in situations where congestive heart failure is imminent or already exists. To a large extent it may be prevented by restricting the patient to a low sodium diet (less than 1.5 Gm. daily) and limiting fluids to 1,000 to 1,500 ml. daily. In spite of these measures, it is occasionally necessary to administer a mercurial in order to force diuresis. Since this diuresis increases potassium excretion, it can prove serious to the edematous patient who may already be hypopotassemic. It therefore is extremely important before using diuretics to have a nearly normal serum potassium level or to protect the potassium level with supplementary potassium.

Finally, it should be stated that a spontaneous diuresis occasionally occurs during prolonged ACTH or cortisone therapy and that one invariably follows 48-72 hours after withdrawal of treatment. At the end of the period of diuresis the patient may complain of headache, weakness, drowsiness and a "washed-out" feeling. Additional fluids by mouth, such as broths or fruit juices, will correct this situation if it is not too severe.

In summary, the most important electrolytic change associated with the use of ACTH and cortisone is the reduction in serum potassium. This is easily detected chemically or with the electrocardiogram and is readily prevented and corrected with potassium chloride. A varying degree of sodium and water retention is observed in some patients receiving these hormones. Any edema which develops clears when treatment is withdrawn.

Metabolic Changes.—Characteristically, ACTH and cortisone significantly affect the intermediary metabolism of protein, carbohydrate and fat (table 2). The effect most commonly observed clinically is on carbohydrate metabolism. Hyperglycemia and glycosuria accompany treatment with these hormones, but it rarely becomes necessary to institute insulin therapy. The glycosuria is in a large measure due to a reduction of renal threshold and alone is not sufficient reason for giving insulin. If the fasting blood sugar is increased more than twofold and the treatment is to be prolonged, it may be wise to start insulin using the blood sugar to determine the dosage needed. Thus far, permanent diabetes following ACTH or cortisone has not been reported. In the diabetic patient who must be treated with ACTH or cortisone, the change in insulin requirement is most easily handled by the addition of small increments of crystalline insulin to the daily insulin dosage. The amount of crystalline insulin required may be determined by following the blood sugar. This is a much more accurate procedure to follow, but the increase in glycosuria may be used. It is important to emphasize the necessity of administering larger amounts of crystalline insulin than are customarily given to reduce glycosuria. This is in part due to the antagonism which exists between adrenal cortical steroids and insulin. Any increase in the severity of the diabetes never reaches alarming proportions. In the diabetic patient receiving ACTH or cortisone it is most important to reduce the insulin dosage immediately on discontinuing hormone treatment. Otherwise, severe hypoglycemic episodes may develop, due to temporary adrenal cortical insufficiency and increased insulin sensitivity. It is much better to be safe and cut back the insulin to the previous maintenance level immediately rather than make graded reductions.