THE EFFECT OF POTASSIUM DEFICIENCY ON INTESTINAL MOTILITY AND GASTRIC SECRETION*

D. R. WEBSTER, M.D., PH.D., H. WINTER HENRIKSON, PH.D., AND D. J. CURRIE, M.D.

MONTREAL, CANADA

FROM THE EXPERIMENTAL SURGICAL LABORATORIES, MCGILL UNIVERSITY, MONTREAL

THE EFFECTS produced by a loss of body potassium have been studied in a variety of clinical conditions such as diarrhea, intestinal obstruction, Cushing's syndrome and diabetic acidosis. Potassium deficiency has been produced experimentally by feeding a low potassium diet or by injecting desoxycorticosterone. It was shown that potassium deficiency may lead to muscle weakness and, in dogs, to eventual paralysis. In addition to this skeletal muscle change, pathologic lesions in cardiac muscle occur which are associated with characteristic electrocardiographic changes. In man and animals given desoxycorticosterone, the water exchange is increased.

In order to investigate certain aspects of potassium deficiency, it was decided to study the effects on organs containing smooth muscle and the effect on gastric secretion. In addition, the effects due to loss of digestive juices has been studied in dogs and in several cases with gastro-intestinal dysfunction in man.

To produce potassium deficiency experimentally, animals were fed a low potassium diet (0.01 per cent potassium). In *rats* the tone and movements of the whole digestive tract decreased progressively as the deficiency continued (Fig. 1B, 2B, 3A). In an average of two months, this resulted in severe distension which eventually was the cause of death. The hypotonic intestine was usually filled with large amounts of digestive-secretions, even before symptoms became severe. In many animals in the severe stage of deficiency, large amounts of gas accumulated, so that the condition resembled that of paralytic ileus. These signs rapidly disappeared upon the administration of potassium. The bladder of deficient animals was also atonic and was usually found filled with urine (Fig. 1B).

In potassium deficient adult *dogs,* little change in motility of the digestive tract occurred, although there was a reduction in the strength and rate of rhythmic contractions. In these dogs, water exchange was increased early in the deficiency.

Other studies have been carried out in dogs with gastric-pouches without loss of gastric juice. In potassium deficiency produced by a low potassium diet, the dogs showed an increase in volume of gastric secretion which paralleled the increase in water exchange (Fig. 4). Muscle weakness and eventual

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paralysis developed in these animals and was relieved by the administration of potassium.

In other dogs with gastric pouches which were fed a normal diet of meat, the secretion by the gastric pouch was discarded. Potassium deficiency resulted and the water exchange and volume of gastric secretions increased markedly (Fig. 5). Loss of body potassium due to loss of gastric juice has also been observed by other investigators clinically and in rats.

These studies have shown that simple potassium deficiency in rats greatly reduces motility in the smooth muscle organs of the gastro-intestinal tract and bladder. Because of the accumulation of fluid and gas, there is either an in-

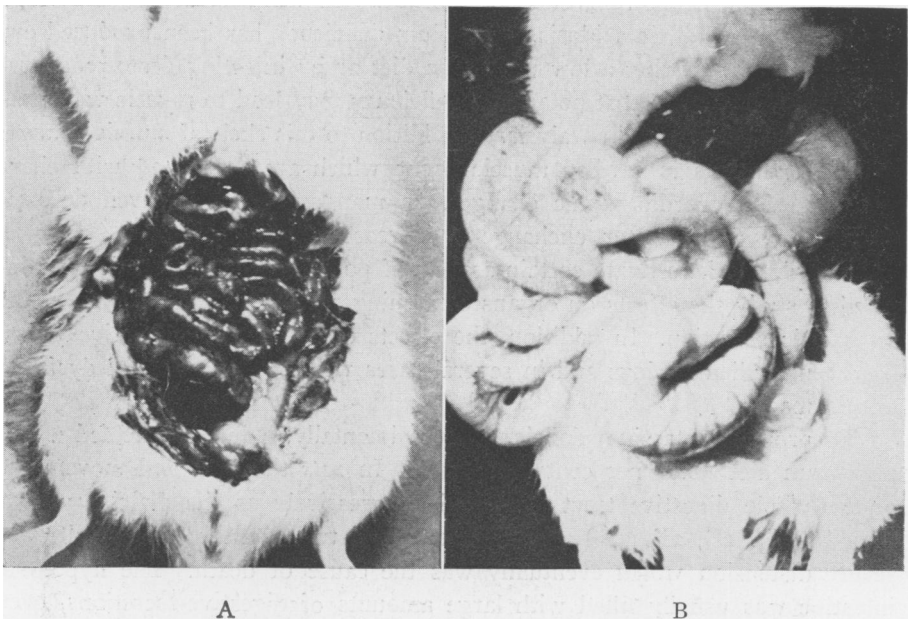
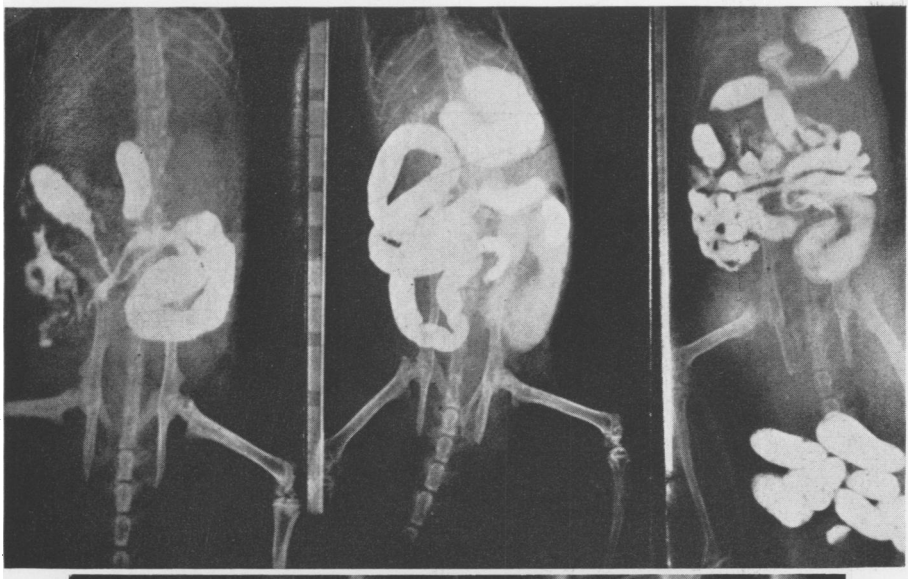


FIG. 1.— (A) Gastro-intestinal tract of normal rat. (B) Hypotonic, distended tract of rat in severe potassium deficiency. Fluid and gas accumulated throughout.

FIG. 2.—Roentgen-ray of same rat. (A) Normal—showing that at five hours much barium was excreted, and the remainder was mainly in cecum and colon, with traces in small bowel. (B) Severe potassium deficiency—14 hours after barium. Barium was in stasis for many hours in the hypotonic, distended stomach and small bowel. No barium was excreted for 48 hours. (C) Recovery on a normal diet five days. Five hour roentgenogram shows the marked improvement in tone, movements and transport. Transport was about two hours slower than normal. Complete recovery was obtained later.

FIG. 3.—Roentgen ray on same rat. (A) Very severe potassium deficiency. Twenty-four hours after a first barium administration, a second dose of barium was given. This roentgenogram taken one-half hour after a second barium feeding shows the marked distention and hypomotility of the whole tract. Complete retention of first meal, mainly in ileum and cecum, occurred. There was practically no evacuation of second barium from stomach in one-half hour or even in four hours. (B) This shows the rapid stimulatory effect of potassium on gastro-intestinal motility and transport of contents. Two meq. of potassium were injected subcutaneously four hours after second meal. This roentgenogram, taken 20 hours later, shows that most of the two barium doses was excreted along with much fluid and gas. No feces had been excreted for a total of three and a half days prior to this.

FIG. 2



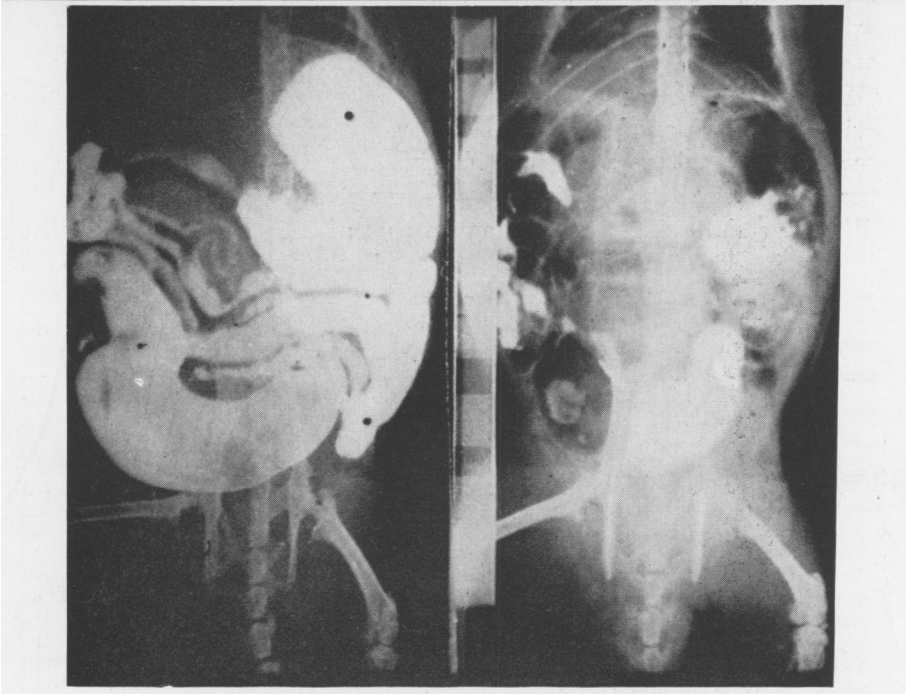
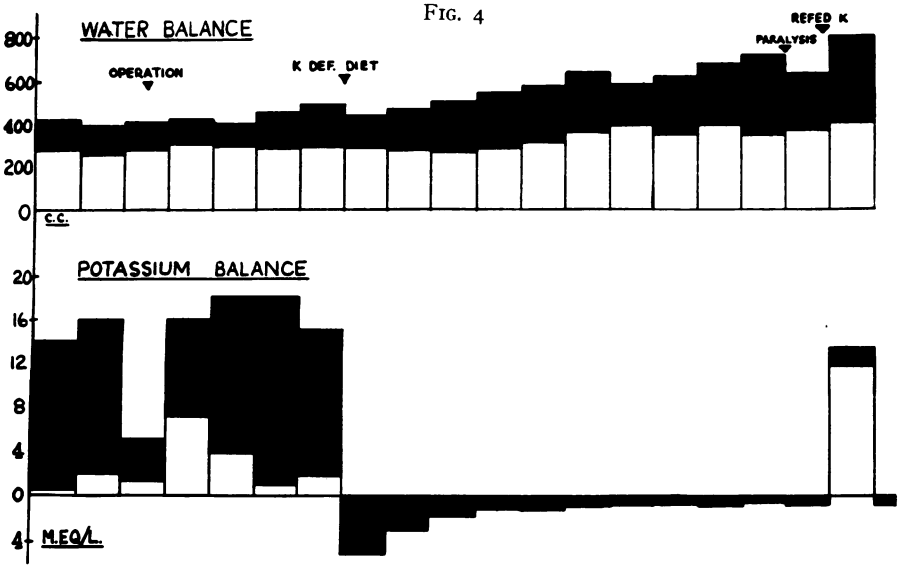


FIG. 3

(Legends on opposite page)

FIG. 4



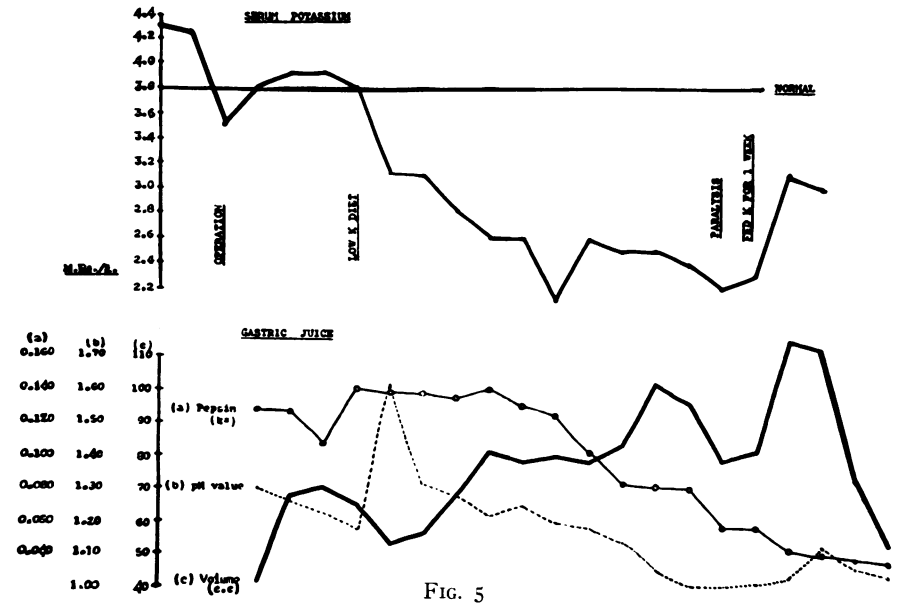


FIG. 5

FIG. 4.—*Water and Potassium Balance in Dog with a Gastric Pouch.* Intake in each case is shown by the upper limit of the black block above the base line. The output is measured downward from the upper limit of the black block and is indicated by the lower margin of the block. Therefore positive balance is shown by a clear area above the baseline, a negative balance by a black block below. Note the steady increase in water exchange with potassium deficiency and the negative potassium balance postoperatively for several days. When potassium was fed, there was an effort to conserve potassium as shown by the positive balance.

FIG. 5.—This chart shows that as the serum potassium decreases from feeding a low potassium diet, the volume of gastric juice increases. Note also that the gastric pouch dog pepsin tends to decrease in concentration and the juice becomes more acidic.

creased secretion of digestive juices and/or a decreased absorption. In potassium deficient dogs, the water exchange and volume of gastric secretions is increased.

A clinical illustration is provided by a patient who had a perforation of a diverticulum of the cecum. A cecostomy was performed by the surgeon, Dr. Harry Ballon, and continuous gastric suction was used postoperatively. The cecostomy did not function for three weeks. The patient lost up to eight litres of gastric secretion each day. Parenteral glucose, physiologic saline, and plasma were given intravenously. At the end of this time, although the patient was not paralyzed, the peripheral reflexes were absent. The serum potassium was 9 mg. per 100 cc., the serum chlorides were 290 mg. per 100 cc. and the carbon dioxide combining power was 100 volumes per 100 cc. of serum. Ammonium chloride was administered with little or no improvement. When potassium chloride was administered, the cecostomy began to function and the patient made an uneventful recovery.

So it would seem that when gastric and intestinal secretions are lost from the body, a relatively large amount of potassium is likewise lost, as these fluids contain two to three times as much potassium as an equivalent amount of serum. Two effects may ensue:

1. There may be inhibition of the intestinal musculature aggravating an ileus already established from some other cause.

2. There appears to be a stimulus to increase the volume of gastric secretion. The stomach continues to secrete potassium, and because of the increased volume of secretion, the potassium deficiency develops. Hydration and electrolyte balance can only be restored if potassium is administered.

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DISCUSSION.—DR. ROBERT ELMAN: I would like to congratulate the speaker, Dr. Webster, and his co-workers on a very important contribution to our knowledge of this complicated and very interesting subject of potassium deficiency.

We have all seen patients with gastric retention, in which there has seemed to be insufficient organic evidence of obstruction. We have been accustomed to say that part of the mechanism is due to an atony of the stomach. It may well be that a deficit in potassium plays a significant part in the pathogenesis.

Although we have studied the plasma level of potassium in many of these cases, a correlation with clinical manifestations has seemed to be rather inconstant. Nevertheless, we have recently added potassium chloride to the saline solution in the treat-