determination of the tubular excretory mass.

If greater accuracy is desired, the calculation should take "delay time" into account.‡ Two samples of blood should be collected during the clearance period and the plasma concentration taken 150 sec before the midpoint of the urine period. This was done in five of our subjects and the difference between the value given in the table and the corrected value averaged 7.78% (5.66 to 9.9%). This difference has very little clinical significance, and becomes negligible when the rate of urine flow is 15-20 cc/min or more.

Results. Effective renal blood flow and tubular excretory mass were determined in 11 normal subjects (6 males and 5 females). In 5 subjects the determinations were repeated after an interval of a few days. As shown in Table I, the values obtained are well within the normal range as determined by Smith's method[1,2] and the determination can be repeated in the same patient with good agreement.

Summary. A simple method for the determination of effective renal blood flow and tubular excretory mass in human subjects is described. Continuous intravenous infusion and catheterization are avoided. The method is suitable for routine clinical use.

‡ for the significance of "delay time" see Smith *et al.*[1]

The diodrast used in this study was generously supplied by the Winthrop Chemical Co.

13983 P

Effect of Testosterone Therapy on Concentration of Potassium in Serum.*

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In his pioneer investigations on perfusion fluids, Ringer observed that while the addition of potassium was not essential for the maintenance of the frog or turtle heart beat, a concentration of approximately 4 milliequivalents per liter was one of the requisites of a solution providing optimal contractions. For the mammalian heart Locke designated the optimal potassium concentration of perfusion fluids at approximately 5.5 meq. per liter.

The observation that potassium salts relieved the paralysis of so-called familial periodic paralysis[1-6] and the more recent observation that a fall in serum potassium concentration was associated with the attacks of paralysis suggested a correlation between muscle weakness and serum potassium concentration.[2-6] During such attacks bradycardia, hypotension, arrhythmia and cardiac dilatation have been observed. However, though a fall in the serum potassium concentration occurs with such attacks, the specific concentration at which paralysis appears varies greatly. A somewhat similar concomitant decrease in serum potassium and in muscle strength has been observed following the administration of desoxycorticosterone acetate[7-13] and the skeletal mus-

* This investigation was aided by a grant from the Commonwealth Fund, New York City.

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TABLE I.
Effect of Testosterone Therapy on the Concentration of Serum Potassium.

Subject	Diagnosis	Date	Serum K Meq. per liter	Daily therapy NaCl g	DOCA* mg	Me-T† mg	Test-P‡ mg
M.M.	Addison's disease ♀	5-11	5.3	9	3	—	—
		6-15	3.8	9	3	—	—
		6-29	2.9	9	3	90	—
		7-6	1.9	9	3	—	25
		7-9	1.9	9	3	—	25
		7-14	3.8	9	3	—	—
		7-17	4.8	9	3	—	—
		7-25	5.6	9	—	—	—
		7-29	2.6	9	—	—	50
		8-10	0.7	9	—	—	50
		8-13	4.7	9	—	—	—
R.Y.	Dwarf ♂	8-21	0.6	—	—	30	—
D.W.	Dwarf ♂	8-12	4.2	—	—	—	—
		9-9	4.1	—	—	—	25
		9-14	0.4	—	—	—	50
		10-16	3.5	—	—	20	—
Le.B.	Hyperthyroid ♀	8-26	5.0	—	—	—	—
		9-9	3.6	—	—	—	25
		9-24	4.2	—	—	50	—
		10-7	1.5	—	—	100	—

*DOCA equals desoxycorticosterone acetate administered I.M. 4-29 to 7-17.

†Me-T equals methyl-testosterone administered orally for patient M.M. from 6-16 to 6-29; for patient R.Y. from 7-17 to 8-21; for patient D.W. from 9-16 to 10-16; for patient Le.B. 50 mg from 9-11 to 9-24; 100 mg from 10-3 to 10-7.

‡Test-P equals testosterone propionate administered I.M. for patient M.M., 25 mg from 6-30 to 7-8; 50 mg from 7-25 to 7-31, 8-4 to 8-10; for D.W. 25 mg from 8-30 to 9-9 and 50 mg from 9-10 to 9-14; and for patient Le.B. 25 mg from 8-30 to 9-10.

cle weakness and the heart failure that may occur with such therapy has been ascribed to these low serum concentrations.[9,11,13,14] On the other hand, low serum potassium levels may occur without paralysis or heart failure following the injection of insulin.[15,16]

This paper reports the striking fall in serum potassium which we occasionally have observed in patients following the daily administration of either methyl testosterone by mouth or testosterone propionate intramuscularly. The analyses were carried out on 4 to 8 cc of non-hemolyzed serum by the method of Fiske and Litarczek.[17] So far as we are aware the serum potassium concentrations presented in the table include the lowest values recorded in medical literature.

The subjects during the periods of these low values were up and about, suffered no muscular weakness or alterations in their electrocardiograms. Detailed metabolic studies of subject M.M., will be reported elsewhere.[18]

From the previous observations referred

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to above and the data reported here, it would appear that the circumstances associated with the low serum potassium concentrations encountered in clinical medicine may be outlined as follows:

1. A fall in serum potassium with testosterone therapy appears to reflect an increase in total tissue potassium associated with an increase in tissue nitrogen consistent with an increase in tissue mass.[18] It is not associated with weakness or paralysis or marked alterations in blood sugar.

2. A fall in serum potassium and decrease in blood sugar following the injection of insulin appears to be associated with a decreased urinary potassium and phosphorus excretion and an increased intracellular potassium and phosphorus (possibly without increase in muscle mass). It is not associated with loss of muscle contractility.

3. A fall in serum potassium during attacks of periodic paralysis is associated with a decreased urinary excretion and increased tissue retention of potassium and phosphorus similar to that observed following insulin but is not associated with alterations in blood sugar.

4. A fall in serum potassium that follows excessive administration of desoxycorticosterone is associated with no change in blood sugar, an increased urinary excretion of potassium, a decrease in muscle potassium concentration, an increase in muscle sodium concentration, and a decrease in muscle strength.

Finally in experiments on rats[12] it has been shown that within wide limits neither the amounts of potassium in muscle cells nor abnormally low concentrations of potassium in the serum limit the capacity of rats to swim.

Thus our observations on the fall in serum potassium concentration and alterations in intracellular potassium balance during testosterone therapy supplement the existing evidence indicating that such changes in potassium distribution do not *per se* determine muscle contractility.

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Comparison of pH and Population of *Trichomonas Foetus*.*

BANNER BILL MORGAN. (Introduced by M. R. Irwin.)

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Witte[1] first showed that *Trichomonas foetus* could tolerate a pH range of 5.5 to 8.5. This was later confirmed by Riedmuller[2] who also reported that the optimal pH for *T. foetus* in pure culture was 6.5 to 7.5. Lyford[3] demonstrated the adaptability of *T. foetus* in pure cultures to various hydrogen-ion concentrations with final pH readings of 4.8 and 5.2. Morisita[4] stated the optimum pH for *T. foetus* ranged from 6.6 to 7.8; the flagellate could survive between 5.6 and 8.4, and the range during maximum growth was 5.4 to 5.6. Johnson[5] reported the population in relation to pH with a pure culture of *T. vaginalis*. Prior to this report, the relationship had not been completely established with *T. foetus*.

This paper is concerned with the correlation between pH and population of several pure culture strains and 2 strains of *T. foetus* in association with an atypical strain of *Corynebacterium renalis*.† This strain is unable to ferment dextrose.

Strain A,‡ originally isolated by Glaser and

* Published with the approval of the Director of the Wisconsin Agricultural Experiment Station. Project No. 622-V; Trichomoniasis and other reproductive diseases of cattle.

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