For a peak resonator voltage of about 800 volts, the range of stability in phase is about 340°, with an associated radial oscillation of amplitude given by Δr in the table.

Taking an intermediate estimate for the ratio of magnetic energy in working volume to total condenser energy as 1/3, we find that 10,000 joules stored energy will produce 85 MeV. electrons with a peak force on the coil of the order of 500 lb./in.[2] directed away from the accelerating region.

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Renal Regulation of the Extracellular Fluid

THE behaviour of the kidney in relation to the electrolytes of the extracellular fluid is usually described in terms of two mechanisms operating to maintain the homœostatic state: (*a*) Regulation of the concentration of several individual electrolytes and non-electrolytes within a limited range of variability. The concept of 'renal threshold' is implied in this proposition. (*b*) Regulation of the osmotic pressure of the extracellular fluid, so as to maintain isotonicity with intracellular fluid.

A patient with a duodenal fistula was studied in some detail. He lost up to the equivalent of one quarter of the extracellular fluid of the body each day. The fluid from the fistula contained an average of 121 milli-equiv. per litre of sodium and 55 milli-equiv. per litre of chlorine. Thus there was an excess of 66 milli-equiv. per litre of sodium relative to chloride. In these circumstances, the level of sodium in the extracellular fluid would tend to fall by a greater amount per unit time than the chlorine, and the loss of this alkaline secretion would impose a severe strain on the *p*H control of the extracellular fluid.

The following observations were made: (*a*) Large amounts of chloride were present in the urine over a period of six weeks, during which time the level of chloride in the plasma was constantly below the accepted 'renal threshold'. (*b*) There appeared to be a commensurate variation between the quantity of chloride ion in the urine, and the volume of fistula secretion per day. (*c*) On the thirty-sixth day after the development of the fistula, the blood sodium and alkali reserve were restored to within normal limits by the intravenous administration of two-thirds of a gram molecule of sodium lactate, over a twelve-hour period. This caused a cessation of the large chloride excretion by the kidney. When the blood sodium fell again the chloride excretion recommenced. Over the time interval during which these variations occurred, the plasma chloride remained relatively constant at a level below the 'renal threshold'.

The most interesting feature of the findings was the relative constancy of the ratio of chlorine to sodium in the extracellular fluid, despite the continued loss through the fistula and from the body of 2–4 litres per diem of fluid containing approximately twice as much sodium as chloride. On the thirty-seventh day after the development of the fistula plasma, values were Cl = 82·7 and Na = 148·7 milli-equiv. per litre with the ratio $Cl_p/Na_p = 0{\cdot}56$. Over the following six days 11·5 litres of fluid containing 766 milli-equiv. of sodium in excess of chloride were lost from the fistula. This is equivalent to 766/14 = 55 milli-equiv. of Na per litre of extracellular fluid, assuming this latter to have a volume of approximately 14 litres[1]. However, the ratio was $Cl_p/Na_p = 80/138{\cdot}3 = 0{\cdot}58$ at the end of the period. The probable explanation was the excretion by the kidney of fluid containing a sufficient excess of chlorine over sodium to maintain homœostasis. The cation used in this latter mechanism was predominantly potassium from intracellular fluid.

It would therefore seem possible that to account for the factors affecting the renal regulation of the quantity per unit volume of extracellular ions, it is necessary to consider plasma *p*H as a major variable.

It has become apparent that the maintenance of the equilibrium state within the tissue fluids in circumstances of disease is a basic problem of medical practice. Marriott, in the Croonian Lectures[2], has suggested that the quantity of chloride in the urine, as determined clinically by the simple Fantus test[3], can be used as a diagnostic criterion of the presence or absence of chloride depletion in the extracellular fluid. A correlation of the above observations with clinical phenomena to be described elsewhere would indicate the necessity of an experimental investigation to determine in what clinical circumstances the test is valid. It is suggested that the logical first step in clinical practice of ionic replacement therapy is qualitative analysis and quantitative record of the material which the body is losing.

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Isophosphorylase and the Formation of Branched Polysaccharides

THE combined action of isophosphorylase and phosphorylase on glucose-1-phosphate results in the formation of branched polysaccharides like amylopectin and glycogen. Isophosphorylase is the enzyme which catalyses the formation of α-1,6-glucosidic links from glucose-1-phosphate as well as their phosphorolysis[1].

Whereas phosphorylase obtained from potato, yeast or muscle is strongly inhibited by phlorhizin, we have established that isophosphorylase from potato or yeast does not suffer any diminution in activity in the presence of phlorhizin (see table).

By adding phlorhizin to a mixture of phosphorylase and isophosphorylase, the activity of the former enzyme is diminished but not that of the latter.

Effect of phlorhizin on the action of potato isophosphorylase (phosphate buffer *p*H 6·6)

Concentration of phlorhizin	Degradation of residual dextrin by β-amylase	
	Isophosphorylase present	Isophosphorylase absent
0	25 per cent	0 per cent
$6{\cdot}0 \times 10^{-3}$ mol.	25 ,,	0 ,,