servations: first, the toxicity of NaCl solution, and second, the occasional deaths that occur after a short period of clinical improvement after intravenous administration of hypertonic solution in cases of head injuries with symptoms of increased intracranial pressure.

Our experiments were carried out on a series of 10 dogs and one man. We used various concentrations of NaCl and of glucose. The results were uniform. First, it was demonstrated that hypertonic saline has a definitely toxic effect, which in concentrations of 10% is manifested after immediate injection by a primary drop in blood pressure, associated with a rise in cerebrospinal fluid pressure; and in concentrations of 30% uniformly resulted in the death of the animal in doses of 75 cc.

Glucose under no circumstances show toxic effect. From the standpoint of spinal fluid pressure, the primary drop was demonstrated varying in extent, depending on the concentration and volume of the injected material, and persisting for periods varying with same factors. With 10% saline the total period of depression of the spinal fluid pressure was about 50 minutes. With 50% glucose the total period of depression was as long as 4 hours. In every instance, however, a secondary rise followed the primary drop which reached the levels from 20 to 100 mm. of water above the control level. On the basis of this secondary rise which was seen in every instance, we feel that hypertonic solutions of either salt or glucose carry a very great hazard in the presence of intracranial pressure.

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Excretion of Organic Phosphorus in Urine.

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Surprisingly large quantities of organic phosphorus in the urine have been recorded in certain pathological conditions. Amounts approaching 1 gm. in 24 hours have been reported and numerous determinations varying from 0.1 to 0.3 gm. may be found. The excretion of so much organically combined phosphorus would constitute a striking abnormality and would bare careful investigation in the light of recent advances in our knowledge of the rôle played

by phosphorus compounds in nerve, muscle, and bone physiology. Several investigators did not note these great quantities of urinary organic phosphorus. However, their studies did not include many pathological conditions. A more extended search was, therefore, made to find instances of such an abnormal state.

The organic phosphorus was separated from the inorganic by treating the urine with Mathison's[1] magnesium citrate mixture. The filtrate was tested for complete precipitation by Scott's[2] reagent and an aliquot digested with sulphuric and nitric acids. The digest was neutralized and prepared for colorimetric readings according to Benedict and Theis. It was unnecessary for the subjects of study to be placed on a specific dietary regimen since it has been found that ingestion of various kinds of organic phosphorus compounds does not increase the organic phosphorus excretion.

The following diseases were included in this study: hyperparathyroidism, hypoparathyroidism, hyperthyroidism, diabetes insipidus, diabetes mellitus, chronic arthritis, Paget's disease, amyotrophic lateral sclerosis, syphilis of central nervous system, tuberculous meningitis, epidemic encephalitis, cardiac decompensation, lobar pneumonia, pulmonary emphysema, psoriasis, dermatitis herpetiformis, obstructive jaundice, carcinomatosis, and Hodgkin's disease. The effects of general anaesthesia and the febrile reactions following intravenous typhoid vaccine were also included. The amount of organic phosphorus excreted in the urine was invariably quite small. The average was 13.4 mg. in 24 hours; the maximum 49 mg.

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Increase in Cholesterol Content of Gallbladder Bile Following Ligature of Cystic Duct.

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The action of the gallbladder wall on the cholesterol content of the bile within its lumen has long been the subject of much dispute. To study this problem experimentally a number of different procedures were carried out. One of these consisted in ligating the cystic duct,

[1] Mathison, G. C., *Biochem. J.*, 1909, **4**, 233.

[2] Scott, F. H., *J. Physiol.*, 1906-97, **25**, 119.