negative rôle in the elimination or detoxification of the solution. So, 3 days following the above injections, the cholecystectomized dogs were anesthetized with ether for from 15 to 30 minutes, and then allowed to recover. They were then given a second injection of like quantity of Dakin's solution containing 0.49% available chlorine. Forty-eight hours following this injection one dog died. The necropsy was entirely negative. The remaining 3 dogs were apparently unaffected by this injection, being in good condition 5 days later.

From these results, we conclude that the intravenous injection of Dakin's solution is somewhat more toxic in cholecystectomized dogs than in normal dogs.

[1] Mann, *Ann. Surgery*, 1921, lxxiii, 54.

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The Source of Urinary Proteins.

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It was previously reported by one of us[1] that complete picture of uremia in all its clinical, pathological and chemical details could be produced by the injection of hypertonic salt solutions into animals already made acid by the absorption of autogenous edema fluid. In these animals, although the kidneys remained histologically normal, there were evidences of degenerative changes in the liver. It was thought possible that the proteins in the urine in these experiments originated in the liver, and were the exciting cause of the renal changes which afterwards made the kidneys permeable to blood proteins. It is well known that foreign proteins injected into the blood are excreted almost quantitatively and that after a certain period the kidneys are thus rendered permeable to blood proteins as well.

Proteins from these artificial uremias and also those in the urine of dogs with ether nephritis were studied. Rabbits were sensitized to dogs' blood proteins by the subcutaneous injection of dog serum in the following series of doses: 1, 2, 4, 6, 8, and 10 cc. at 3 day intervals. The rabbits were sacrificed on the 21st day and bled. Sera thus obtained will react to dog blood proteins in dilutions of 1/1,000,000.

Urines were collected from our experimental nephritides in small 15 minute samples. Each sample was tested first for traumatic blood, which often spoiled the experiments. Next, all blood-free samples were tested for albumen and on all positive specimens, were tested against the serum. These tests were carried out both on whole urines and on urinary proteins, purified by dialysis and precipitation by ammonium sulphate saturation. From the earlier samples in a large series of experiments over 100 specimens were found in which the urinary proteins were free from serum proteins. With sera sensitive in dilutions of 1/1,000,000 they failed to react in dilutions of 1/10 and many times undiluted. Aside from the reports of Thomas[2] and a few others[3, 4, 5] these are the only blood-free urinary proteins ever studied. Previous attempts to ascertain the origin of urinary proteins by precipitin reactions have never been successful.

Next, normal dog livers were extracted, after perfusion until the perfusate no longer contained albumen. The liver was then ground in the ball-mill for 60 hours, extracted repeatedly with water, and the watery solution precipitated by saturation with ammonium sulphate. The precipitated protein was then freed of ammonium sulphate by dialysis. A serum was prepared by the above described method, which gave reactions in dilutions of 1/100,000 against liver protein. This serum was then matched against the urinary proteins (purified with urine), both from artificial uremias and ether nephritides, and precipitins reactions were again obtained in very high dilutions, at times as high as 1/100,000. This was controlled by taking samples of urinary protein which contained both blood and liver protein and mixing equal parts of urine and dog serum sensitized to dog blood. This was left over night, filtered, and then matched again against liver serum and reactions were obtained in just as high dilutions as before.

We are justified then in concluding that the albumens in the urine of certain types of nephritis originate in the liver.

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[2] Welker, Wm. H., Thomas, W. A., and Hektoen, L., *J. Am. Med. Assn.*, 1926, lxxxvi, 1333.

[3] Bramwell, B., and Paton, D. N., *Rep. of Lab. Roy. Coll. Phys. Edinburgh*, 1892, iv, 7.

[4] Bayne-Jones, S., Wilson, D. W., and Everett, H. S., *Bul. of Johns-Hopkins Hosp.*, 1923, xxxiv, 77.

[5] Hektoen, L., Kretschmer, H. L., and Welker, Wm. H., *J. Am. Med. Assn.*, 1924, lxxxiii, 1154.