13.9, 7.3 and 3.5, respectively. Based on body weight of the fish, this elimination amounts to 0.0047, 0.0047, 0.0054, 0.0057 and 0.0056 mg. per kg. Through this series, increasing the injected dose up to 16 times, increases the mg. per kg. elimination by not more than one-fifth. In other words the excretion remains practically constant.

6762

Modification of Skin Sensitivity to Neoarsphenamin in Rabbits Treated with Potassium Iodide and Bromide.

J. W. MU. (Introduced by C. N. Frazier.)

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It has been demonstrated[1, 2, 3] that a nonspecific positive luetin reaction can be produced in the human subject and animals by the administration of potassium iodide. The object of the present study was to ascertain whether or not potassium iodide and bromide are capable of exerting a similar influence on the skin hypersensitiveness to neoarsphenamin in rabbits.

This work included 2 experiments, involving 76 adult male albino rabbits. Twenty-four of these animals which had been inoculated with *Treponema pallidum* from 5 to 8 months previously, were distributed in both experiments and were all in the latent stage of the infection at the time of the experiments. The neoarsphenamin solution was prepared and the rabbits were shaved according to the methods described previously.[4] The brand of neoarsphenamin used was that prepared by Hoechst Company, Germany, and the ampoules employed were always from the same batch. The rabbits were sensitized and tested with 0.2 cc. of freshly prepared 0.15% neoarsphenamin in normal salt solution. The right flank of the animal was used for the sensitizing injection and the left for the testing injections, all being intradermal. The hyper-

[1] Sherrick, J. W., *J. Am. Med. Assn.*, 1915, **65**, 404.

[2] Kolmer, J. A., Matsunami, T., and Broadwell, S., Jr., *J. Am. Med. Assn.*, 1916, **67**, 718.

[3] Kolmer, J. A., Immerman, S. L., Matsunami, T., and Montgomery, C. M., *J. Lab. and Clin. Med.*, 1917, **2**, 401.

[4] Mu, J. W., PROC. SOC. EXP. BIOL. AND MED., 1932, **29**, 781.

sensitive reaction, which was manifested as redness and infiltration, was read 24 hours after injection, the size of the involved skin area ranging from 1 to 3 cm. in diameter.

In the first experiment, 42 rabbits were used and in the second one, 34 rabbits. Five weeks after the animals had been sensitized, they were tested for hypersensitiveness (first test). As shown in the accompanying table, the animals of each experiment were then

TABLE I.

Skin Reactions in Rabbits Receiving Sensitizing Doses of Neoarsphenamin and then Treated with Potassium Iodide, Sodium Iodide or Potassium Bromide.

Exp. No.	Group No.	No. animals	Hypersensitive animals on first test	Treatment	Animals showing increase of skin reaction	Non-sensitive animals becoming hypersensitive
I	1	14	4	No drug (control)	0	0
	2	14	5	Potassium iodide	3	7
	3	14	4	Potassium bromide	1	3
II	1	10	2	No drug (control)	0	0
	2	12	2	Potassium iodide	1	6
	3	12	4	Sodium iodide	2	5

divided into 3 groups, each of which included both hypersensitive and non-sensitive rabbits of about equal number. The first group of each experiment was used as control and the others as test groups. The test groups in the first experiment were injected intravenously with 10% aqueous solutions of potassium iodide and potassium bromide respectively. To determine the relative action of the individual ions of the drugs used in the first experiment, the test groups in the second experiment were treated intravenously with 10% aqueous solutions of potassium iodide and sodium iodide respectively. The chemicals employed in both experiments were given once a day for 3 days in daily doses of 0.02 gm. per kilo of body weight. Twenty-four hours after the final administration of the drugs, the animals of all groups in both experiments were tested again for hypersensitiveness (second test). The skin reactions were measured 24 hours after the second set of tests, and the animals were also observed for flare-up of the reactions from the first series of tests.

As recorded in the table, the results indicate that potassium and

sodium iodides intensified the skin hypersensitiveness to neoarsphenamin in 54% of the animals showing positive skin reactions on the first test, and produced well-marked cutaneous reactions in 66% of the animals which had not reacted to this arsenical before the administration of the iodides. The potassium and sodium iodides were about equally capable of exerting these influences. Potassium bromide was found to have a similar but much less effect. There was no appreciable difference in the response to the tests for sensitization after treatment with the iodides and bromide between the normal and syphilitic rabbits. No flare-up was noted at the site of the test conducted before the injection of the drugs under study. The control animals did not show any significant variation in their skin reactions after the first and second tests, thus ruling out the possibility that the changes observed in the rabbits treated with iodides and bromides were attributable to their skin reactivity being influenced by the preliminary testing injections. The observations reported here seem to support the conclusion that the modification of skin reactivity to neoarsphenamin in the animals of these experiments is to be ascribed to the action of the iodide and bromide ions and not to the potassium and sodium ions of the drugs used.

6763

An Optimal Diet in Promoting Nitrogen Gain in Nephrosis.

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In the treatment of nephrosis Epstein[1] advocates a high protein diet, 150 to 200 gm., with low caloric intake. Peters and Bulger[2] from an investigation of the nitrogen metabolism in albuminuric, edematous patients conclude that it is possible to promote the storage of food protein and to cover the protein lost in the urine by allowing a moderate amount of protein in a high calory diet. The present study involves the determination of nitrogen balance and plasma proteins on 2 patients with nephrosis on diets which were varied in (1) total calories, (2) protein content and (3) proportion of animal and vegetable protein.

[1] Epstein, A. A., *Am. J. Med. Sci.*, 1917, **154**, 638, and 1922, **163**, 167.

[2] Peters, J. P., and Bulger, H. A., *Arch. Int. Med.*, 1926, **37**, 153.