made of the concentration and amount of bromsulfalein in each sample. The following criteria were used as a basis of comparison of the two groups of animals: (1) completeness of removal of dye from the blood; (2) rate of entrance of dye into the bile; (3) time of attainment of maximum concentration of dye in the bile; (4) curve of excretion of dye in the bile; (5) range of concentration of dye in the bile and (6) total dye excretion in the bile within one- and 2-hour periods following injection.

Prior to initiating a series of liver function studies the two types of permanent biliary fistula dogs were compared for suitability by observing their prompt convalescence from operation, maintenance of good general physical condition and liver function. The liver function was tested by estimating the quantity and rate of excretion in the bile of intravenously injected bromsulfalein and, in some animals, estimating the quantity of dye retained in the blood according to a method previously described.[2]

Results. The pertinent data are summarized in the table and figure. No abnormal retention of dye in the blood was observed in the Thomas-type fistula dogs; abnormal retention occurred in 5 of 6 standard fistula dogs. Similary, the unselected dogs with the Thomas-type fistula were superior to unselected dogs with the standard type fistula when judged by the other criteria enumerated above. Dogs in the latter group selected on the basis of satisfactory bromsulfalein excretion approached the former in this regard.

Summary and Conclusions. Evidence has been presented to indicate that dogs with the Thomas-type bile fistula are much more satisfactory than those with the Rous-McMaster-type fistula for studies of liver function, particularly those involving collection and examination of bile. Data are presented regarding the normal biliary excretion of bromsulfalein following intravenous injection of 2 mg and 5 mg of the dye per kilogram of body weight.

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Urticarial Hypersensitivity to Sunlight.

PETER L. BEAL. (Introduced by S. Rothman.)

From the Division of Dermatology, Department of Medicine, University of Chicago, Chicago, Ill.

Urticarial hypersensitivity to sunlight or solar urticaria is a well recognized disease entity.[1] The reaction is analogous to the urticarial hypersensitivity reactions to foods, drugs, and such physical agents as mechanical, cold or heat stimuli. These reactions have all the characteristics of Thomas Lewis' "triple response".[2] It is generally assumed that in this type of hypersensitivity the antigen-antibody reaction leads to wheal formation via the liberation of histamine. Solar urticaria has been regarded as one form of the "physical allergies" in which an antigen or allergen is produced by action of a physical agent. Recently two types of solar urticaria have been described.[3,4] In one type the sensitivity spectrum is between λ 4000-5000 Å and in the other type it is below λ 3700 Å.

Experimental. In this clinic 2 patients were studied who responded to sunlight exposure with severe urticarial reaction. In both cases

1 Blum, H. F., *Photodynamic Action and Diseases Caused by Light*, New York, Reinhold Publ. Corp., 1941.

2 Lewis, T., *Blood Vessels of the Human Skin and Their Response*, London, Shaw and Sons, 1927.

3 Abramson, H. A., PROC. SOC. EXP. BIOL. AND MED., 1940, **43**, 410.

4 Blum, H. F., Baer, R. L., and Sulzberger, M. B., *J. Invest. Derm.*, 1946, **7**, 99.

it was possible to establish the spectral sensitivity: (1) by radiating the skin of the patients through a set of Corning glass filters and (2) by use of a monochromator which made it possible to project single lines of the mercury arc spectrum on the skin. In both cases the range of sensitivity was found to be between 2967 and 3341 Å with a maximum sensitivity at 3131 Å. Thus these 2 patients belong to the type $< \lambda$ 3700 Å.[4]

The urticarial sensitivity could be passively transferred. By injecting the patients' serum into the skin of normal individuals and irradiating the injection site with the active wavelengths, a "triple response" could be elicited. The transfer site began to manifest sensitivity within 30 minutes after injection and remained sensitive for several days although the sensitivity decreased with time. Normal control sera never caused such reactions.

The incidence of successful passive transfers was much higher with the serum of the more sensitive patient than with the serum of the less sensitive one. When large surfaces of the body were exposed to ultraviolet light prior to withdrawal of blood for passive transfer experiments, the incidence of positive transfers was greatly increased.

Studies of the properties of the transferable sensitizing agent in the serum revealed that it decreased in potency with storage at ice-box temperature. After 8 days cold storage of otherwise potent serum, samples gave only weak or questionable passive transfer tests. The sensitizing agent proved to be heat labile and was inactivated at 56°C in ½ hour. Also, the agent was inactivated by irradiation with ultraviolet light. Evidence that the photosensitizing agent is a large molecule, probably of colloid nature, is suggestive since it failed to dialyze through a semi-permeable membrane.

Early morning gastric analysis following radiation of large areas of the patients' skin showed a rise in total acidity in both patients.

A number of substances with absorption spectra similar to the sensitivity spectrum of the patients were incorporated into ointments and tested for protective action against the active rays. Some protection was obtained by using an ointment containing 30% G-Salt (sodium salt of 2 - naphthol - 6, 8 - disulfonic acid).

Greater protection was achieved by the oral administration of antihistaminic drugs, benadryl and pyribenzamine. By virtue of the protective action of these "antihistamines" it was possible to subject the patients to gradually increasing generalized ultraviolet light exposures.[5] By this treatment the skin acquired such tolerance that the administration of "antihistamines" could be discontinued. This gradual increase in tolerance to the active wavelengths was purely a local effect since no protection was achieved on skin areas which were kept covered during the course of ultraviolet therapy. The protection may have resulted from pigmentation and/or the thickening of the horny layer of the skin. Both of these physiologic reactions to sunshine hinder the penetration of the active wavelengths.

Discussion. The narrow and highly specific range of the active wavelengths in this condition together with the passive transferability of the sensitivity to normal individuals with the patients' serum strongly supports the theory of Sulzberger and Baer.[6] According to these authors, the absorption of the specific wavelengths causes the formation of a photochemical product in everybody's skin; however, patients with solar urticaria develop a sensitivity by forming antibodies against this normal metabolite.

Summary. In 2 cases of solar urticaria the maximum sensitivity was found to be at 3131 Å. Studies of the photosensitizing agent in the patients' serum support the conception that this condition is due to sensitization to a physiological radiation product in the skin.

5 Rubin, L., Beal, P. L., and Rothman, S., *J. Invest. Derm.*, 1947, **8**, 189.

6 Sulzberger, M. B., and Baer, R. L., *J. Invest. Derm.*, 1945, **6**, 345.