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Bromsulfalein was retained 6 times in 18 tests. Serum bilirubin exceeded 4 mg. per cent only once. Prothrombin time was elevated markedly in 2 patients. Serum albumin was below 3.5 gm. per cent in 18 patients.

Kidney: Urea clearance was below 70 per cent in 9 of 13 patients. Phenolsulfonphthalein excretion exceeded 65 per cent (50 per cent in children) in 9 patients. The NPN exceeded 40 mg. per cent in 7 patients. Slight albuminaria was occasionally observed. RBC were found in the urine in 5 patients and casts in 8.

Liver damage, related in degree to the clinical severity of the disease, was greatest by hippuric acid test and occurred with or following the clinical peak. BSP, prothrombin, and albumin alterations occurred during the acute febrile phase. Function returned to normal with convalescence.

Kidney damage was less marked in degree, unrelated to the clinical severity, occurred before or during the clinical peak and returned to normal with convalescence. No glomerulonephritis was seen. Azotemia is probably due to fluid and circulatory disturbances rather than renal damage.

In 18 patients a high protein diet protected against changes in serum albumin and hippuric acid excretion; it did not decrease renal function but occasionally overloaded the kidney.

The mechanism responsible for the changes is obscure.

The Response to Adrenocorticotrophic Hormone in Patients with Scleroderma and the Therapeutic Use of Testosterone. SAUL HERTZ and (by invitation) PETER H. FORSHAM, Boston, Mass.

Scattered evidence has pointed to an endocrine factor in scleroderma, but none put forth to date incriminates any specific glandular defect. Suggestive features of a positive type include the marked incidence of hypercreatinuria, reduced creatine tolerance tests, low urinary 17-ketosteroid excretion and a relationship of onset of periods of exacerbation to menstruation, menopause, etc. Predominance of the disease in the female sex, and in prepubertial males, has been impressive. Disorders of calcium metabolism (ectopic calcification), negative nitrogen balance and marked wasting have been emphasized. We have confirmed these findings in our series. The creatinuria, myopathy, negative nitrogen balance, pigmentation and occasional low serum sodium and chloride levels are compatible with some degree of gonadal or adrenal hypofunction. That this might be secondary to pituitary underactivity is suggested by low FSH titres, I^{131} uptake by the thyroid and 17-ketosteroid excretion.

Seven cases have been studied from the standpoint of 17-ketosteroid excretion, creatine tolerance and responses to epinephine and ACTH.

17-Ketosteroid values on 24-hour urines in this group ranged from 1.2 mg. to 6.0. However, 48-hour tests with ACTH (40 mg. per day) led to a marked rise in 17-ketosteroid excretion and other evidences of "S" factor activation.

Since ACTH is not available for therapeutic application, we chose to observe 4 patients on high dosage of testosterone propionate intramuscularly. Dosage from 25 to 50 mgs. × 3 per week. Gradual disapp of skin lesions, as well as improvement in esc involvement (radiologic evidence) took place. weight gain and increased appetite and strength with the establishment of a positive nitrogen bal curred. Therapy was continued to the point of ment of edema; slight hirsutism and masculinizatio voice were encountered. These subsided quickl cessation of therapy and the skin lesions showed n rence four to five months after the end of therap

The Pulmonary Vascular Resistance. JOHN B. I (Introduced by Eugene A. Stead, Jr.), Durhan

Normally the pulmonary arterial pressure is l little affected by changes in blood flow. In co failure the pressure is high and may be greatly in by exercise without change in flow. It is difficul terpret these observations in terms of the state of monary vessels because the pulmonary arterial p also depends on the blood flow and the pulmonary pressure. The latter can not usually be measure present report presents data obtained from 4 patie atrial septal defect in whom it was possible to cat both pulmonary arterial and venous systems and t ure blood flow, pressure gradient, and pulmonary resistance.

In 2 subjects without apparent pulmonary vascu ease, the resistance was extremely low (0.6 to 0 Hg/1./min.). Blood flows of 15 and 20 1./min maintained by gradients of 13 and 12 mm. Hg. O ject with congestive failure and pulmonary arterial tension had a flow of 15 1./min. with a gradient of mm. Hg. This indicates that the high pulmonary pressure resulted from transmission of a high pul venous pressure back through the pulmonary vascul The low gradient suggests passive dilatation of t Exercise caused a large rise in pulmonary arteria sure, two-thirds of which resulted from an incr venous pressure and one third from an increase in v resistance. One subject had pulmonary vascular and a high resistance (80 times that of the pr subject).

The observations provide quantitative data on th potential range of pulmonary resistance and sugg means by which congestive failure produces pul arterial hypertension.

Experimental Evidence on the Mechanism of D Ketosis. LAWRENCE E. HINKLE, JR., and GEO CONGER (by invitation) and STEWART WOLF, New N. Y.

In a study of 25 human subjects with diabetes m approximately 50 instances of clinical ketosis we served to occur in a setting of emotional conflict the absence of other pertinent factors including inf Moreover, day to day observation of these subject in and out of the hospital yielded a close correlati tween life situation, emotion, and the metabolic su