signs regularly observed after successful injection of the stellate ganglion were uniformly present.

The subjects were recumbent and the fingers or toes were within 6 inches of the level of the heart. None of the patients knew when the anesthetic agent was injected. Records were obtained in the absence of surgical trauma or hemorrhage.

Observations. Before anesthesia, the needles being in place, the tracings were normal, the pulse waves being 6 to 7 cu mm‡ and the alpha waves up to 60 cu mm. Two minutes afterward, in the case of spinal anesthesia, the pulse waves in the toe became progressively larger (up to 15 cu mm) while the alpha waves became smaller and almost disappeared. Concurrently, in the fingers, the size of the pulse waves progressively decreased to one-half or even one-quarter their former size, while the size of the alpha waves decreased, as in the toes.

Similar effects were noticed on injection of one stellate ganglion. Pulse waves of the ipsilateral fingers increased and alpha waves decreased, while in the opposite fingers both waves, pulse and alpha, decreased to one-half or one-quarter the original size. There were no marked alterations in blood pressure or pulse rate during the period of spinal or regional anesthesia.

Such striking changes were not observed when occlusive vascular diseases were present (3 cases). Cardiac output and volume of visceral flow were not measured.

Comments. These observations indicate that when marked dilatation occurs in one peripheral vascular bed, owing to paralysis of the sympathetic supply, concomitant vasoconstriction occurs in certain other remote peripheral beds. The mechanism involved seems to be one of adjustment, possibly compensation, through efferent pathways of the sympathetic system. Paralysis of these nerves permits maximal expansion of the small blood vessels (large pulse waves) and interrupts the pathway of stimuli necessary for the formation of prominent alpha waves; at the same time, increased sympathetic outflow to certain other vascular fields results in vasoconstriction intense and persistent enough to produce small pulse waves continuously and to inhibit the relaxation apparently necessary for the development of alpha waves. It seems that such alterations in the patterns of alpha and pulse waves reflect changes in the activity of certain sympathetic pathways. The method may be useful, then, for clinical studies of the effects of numerous physiological stresses, such as those preceding shock, upon such pathways and upon small peripheral blood vessels.

‡ All changes in volume are calculated for finger tips or toe tips of 5 cc.

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Tubular Resorption of Chloride in Essential Arterial Hypertension: Intensive Study of One Case.*

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In a previous study[1] we have compared a group of individuals with essential hypertension and a control group of normal subjects, with respect to tubular resorption of chlorides. In the present study we have taken 2 male subjects of comparable age and investigated them extensively by the same technique, namely chloride excretion considered in terms of the urine concentration of inulin divided by the plasma concentration, or the U/P inulin ratio.

Methods. The experimental subject was a retired business man, 55 years of age, who

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1 Farnsworth, E. B., and Barker, M. H., PROC. SOC. EXP. BIOL. AND MED., 1943, **52**, 74.

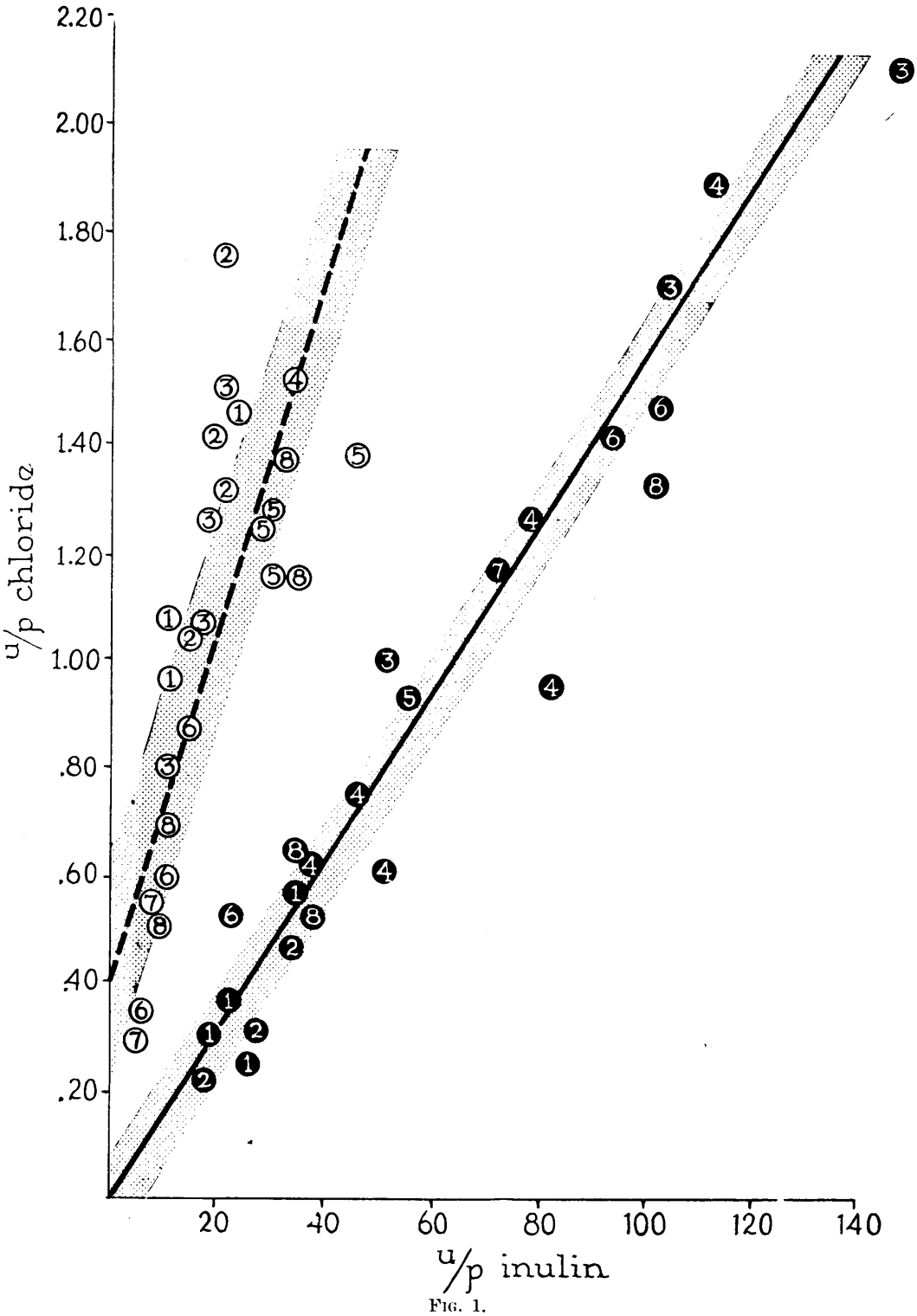

FIG. 1.

Black symbols represent the normal subject; white symbols represent the patient with essential arterial hypertension. Symbols containing the same number indicate successive clearance period performed on the same day.

The coefficient of linear correlation for the normal subject is .989 ± .0045 with a standard error of estimate of .09.

The coefficient of linear correlation for the hypertensive patient is .894 ± .0261 with a standard error of estimate of .18.

gave a history of hypertension of long standing. His complaints were of failing vision, disturbed memory and headaches. Urine analysis showed no albumin, sugar or cells. The erythrocyte count was 4,850,000, the hemoglobin 13.0 g. He was without evidence of edema. Ophthalmoscopic examination of the eye-grounds showed diffuse angiospasm, without retinopathy. The blood urea nitrogen, non-protein nitrogen, cholesterol and proteins were within normal limits. The systolic blood pressure ranged from 200 to 234, the diastolic from 106 to 134.

The normal subject was a 53-year old railroad employee who was hospitalized because of a fracture. He had no complaints and gave no evidence of other pathology. His personal and family history was negative for cardiovascular or renal disease. The urine was without albumin, sugar or cells. The erythrocyte count was 5,100,000, the hemoglobin 15.0 g. The blood pressure was 140/84.

These 2 individuals were studied by a method consisting of concomitant clearances of inulin, diodrast, and chloride. The U/P chloride was plotted against the U/P inulin. Both subjects were on the standard ward diet and received similar preparation. As in the previous study, the blood was taken under oil, and estimations were made on serum and quoted in terms of sodium chloride. Analysis of chloride concentration was carried out by the method of Sendroy.[2] Determinations on inulin were made by the method of Corcoran and Page.[3] Clearance periods ranged from 15 to 30 minutes in length. The normal subject is represented by a total of 24 periods, the hypertensive subject by 27.

Results. Multiple tests of these 2 individuals corroborated our experience in the group study previously undertaken. The hypertensive subject was found to reabsorb substantially less chloride than did the normal subject.

Discussion. The significance of this deviation of the hypertensive kidney from the normal is far from clear. In order to determine whether or not the phenomenon was specific to chloride, we ran simultaneous tests on phosphate and subjected them to similar analysis. It was found that phosphate excretion in the hypertensive subject showed only an insignificant and irregular increase over the normal. This fact seems to suggest that chloride resorption is affected by certain factors which do not govern the fate of other metabolites similarly filtered.[4]

The average clearances of the 2 subjects are given in the accompanying table.

TABLE 1.

	Hypertension (C.V.)	Normal (C.P.)
Inulin	58 cc/min	79 cc/min
Diodrast	249	430
Chloride	3.3	1.2
Phosphate	16.3	12.3

Conclusions. Repeated clearance periods taken on an individual with essential arterial hypertension and compared with those taken on a normal subject demonstrated that there was substantially less reabsorption of chloride in the hypertensive individual. This diminished resorption of chloride in the tubules of the hypertensive subject, at all rates of water resorption, is offered as evidence suggestive of a specific modification of chloride treatment in essential arterial hypertension.

[2] Sendroy, J., *J. Biol. Chem.*, 1937, **120**, 2.

[3] Corcoran, A. C., and Page, I. H., *J. Biol. Chem.*, 1939, **127**, 601.

[4] Walker, A. M., *J. Biol. Chem.*, 1933, **101**, 239.