It is known that inositol hexaphosphate (IHP), the stronger effector, changes the redox potential of ferricferrous hemoglobin[7] and also changes the conformation of methemoglobin[8]. Since 2,3-DPG, though weaker than IHP as an effector, is also shown to change the redox potential of the same system[9], it is likely that the conformation of methemoglobin is changed by 2,3-DPG. A plausible explanation for the acceleration may be attributed to the increase in reactivity of ascorbic acid with methemoglobin, which is facilitated by the change of conformation of the latter due to the binding of 2,3-DPG. However, detailed mechanism of the reaction remains to be clarified.

These results suggest that 2,3-DPG affects the rate of methemoglobin reduction in intact red cells, especially when ascorbic acid is applied to patients suffering from hereditary methemoglobinemia. This is consistent with the report[10] that the highly increased content of 2,3-DPG in red cells of hereditary methemoglobinemia.

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The Effect of Renal Hydrodynamics on Immune Complex Deposition[1]

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Summary. The role of renal hydrodynamics on renal deposition of immune complexes was evaluated in acute serum sickness. Using i.v. radiolabelled antigen in rabbits under a variety of hydrodynamic alterations, these studies suggested that although intrarenal hydrodynamics influence renal deposition of immune complexes factors other than intrarenal hydrostatic pressure may be important.

It is well recognized that circulating antigen-antibody immune complexes deposit in tissues, particularly vascular membranes, resulting in an inflammatory injury through activation of complement[4]. Immune complex glomerulonephritis has been studied extensively by means of the acute serum sickness model[5]; however the effedts of renal hydrodynamics on immune complex deposition have not been well defined. GERMUTH et al.[6] reported that renal hydrodynamics were a determinant of the site of deposition and injury caused by complexes. These investigators suggested that clamping of the renal artery or ligation of the ureter induced the morphological and immunohistological differences in the kidneys in serum sickness, and they postulated that these differences are secondary to the differences of immune complex deposition; however, they did not quantitate the amount of immune complexes which were deposited.

Materials and methods. Human serum albumin (HSA) was labelled with I^{125} by the chloramine-T methods as described by MCCONAHEY and DIXON[7]. Ten 3 kg rabbits were injected i.v. with I*HSA 250 mg/kg. On the same day 10 ml/kg HSA in Freund's complete adjuvant was injected s.c. to ensure an immune response. Blood samples were taken at frequent intervals from the central ear artery using butterfly scalp vein needles and aseptic technique. Blood was allowed to clot at 37 °C for 2 h. The clotted blood was centrifuged at 2,000 rpm and the serum was decanted. Protein from 1 ml of serum was precipitated in 10% trichloracetic acid (TCA), washed by centrifugation and counted to determine total serum I*HSA.I*HSA complexed with antibody was determined by the ammonium sulfate precipitation technique of FARR[8] using fresh non-refrigerated serum. 2 days after administration of antigen the animals were divided into 5 groups with 2 animals in each group. Group I: controls which received no hydrodynamic manipulations. Group II: the right renal vein was ligated. Group III: the left renal vein was ligated. Group IV: the right ureter was ligated. Group V: the left ureter was ligated. Blood was obtained for detection of I*HSA or I*HSA immune complexes. The animals were sacrificed on the 16th day, at which time the kidneys were removed and studied by light and immunofluorescent microscopy after their radioactivity had been measured. The method of immunofluorescent microscopy has been described previously[9].

Results. Free I*HSA was detected in the serum until day 8. Following this time I*HSA immune complexes were detected in the serum. On the 15th day, the day prior to sacrifice, no free I*HSA or I*HSA immune complexes were detected in the serum.

Deposits of rabbit IgG, C3, and HSA were detected in both kidneys of all animals. Kidneys subjected to ureteral ligation were hydronephrotic whereas those subjected to renal vein ligation were sclerotic.

Discussion. Renal hydrodynamic forces have been suggested to play a role in deposition of circulating immune complexes[10]. Increase arterial blood pressure pre-

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Radioactivity of kidneys

Group		Right	Left
I	Control	1.430/min	1.574/min
	Control	1.569/min	1.594/min
II	R. renal vein ligation	17.193/min	2.947/min
	R. renal vein ligation	11.534/min	6.097/min
III	L. renal vein ligation	2.003/min	17.662/min
	L. renal vein ligation	Died	
IV	R. ureter ligation	2.137/min	2.080/min
	R. ureter ligation	Died	
V	L. ureter ligation	8.425/min	8.700/min
	L. ureter ligation	4.227/min	4.786/min

sumably facilitates glomerular deposition of immune complexes. Immune complexes localize in areas of greatest turbulence. The capillaries of the glomerular tuft located between 2 arterioles are subject to 4 times higher blood pressures than other capillary beds, being unopposed by any pressure from surrounding tissues since the glomerular tuft lies within Bowman's space. The blood flow to the kidney represents a large percentage of the cardiac output. Decrease in renal arterial pressure as in coarctation of the aorta or increase in pressure in Bowman's space as in hydronephrosis, has reportedly been associated with inhibition of deposition of circulating immune complexes and inhibition of deposition of circulating immune complexes and arteritis. Conversly, hypertension has been suggested to increase the severity of these lesions due to the increase pressure in glomerular capillaries. However, evidence for an active process of deposition of immune complexes related to vasoactive amines, and several mediators of inflammation has been well documented[10].

It has been suggested by GERMUTH et al.[6] that the high hydrostatic pressure within the arterial system and glomerular capillary bed may be responsible for the unique susceptibility of these tissues to the penetration and localization of pathogenic soluble immune complexes and the development of glomerulonephritis. If this hypothesis were correct, reduction in renal hydrostatic pressure produced by constricting the main renal artery or interference with renal filtration produced by ligation of a ureter should inhibit the glomerular localization of complexes and should reduce or abolish the development of glomerulonephritis. Our study demonstrated that renal vein ligation is associated with increased deposition of immune complexes, probably due to increased hydrostatic pressure on the glomerular capillaries; however, ureteral ligation failed to produce significant differences in immune complex deposition between the two kidneys.

The use of radiolabelled antigen is a sensitive method of quantitating localization of immune complexes. Factors other than renal capillary hypertension should be considered in analyzing the reasons for preferential deposition of immune complexes in the renal glomerular capillaries.

Subcellular Steroid Distribution in the Rat Adrenal Cortex[1]

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Summary. The subcellular distribution of main steroids (pregnenolone, progesterone, corticosterone) and of cholesterol – this last after correction according to exchanges occuring during differential centrifugation – is presented in the rat adrenal cortex. The distribution patterns are not necessarily the same as established according to the localization of hydroxylating enzymes.

The present accepted metabolic pathway of steroidogenesis in the mammalian adrenal cortex[2] suggests a continuous movement of the steroid molecule between different intracellular compartments. This scheme was established according to the subcellular localisation of different hydroxylating enzymes. Apparently there is little information concerning the corticosurrenal subcellular distribution of the different steroids. However it is as important in this metabolic scheme as the different enzymatic systems.

The purpose of the present paper is to establish the subcellular distribution of 3 important steroids (pregnenolone, progesterone and corticosterone) and of cholesterol in the rat adrenal cortex. These results are compared with the subcellular distribution of the organelles as defined morphologically, morphometrically and chemically after differential centrifugation.

HOLZBAUER[3] was the first to approach this problem. We agree with her conclusions, but nevertheless obtained some different results. This may be due to mainly technical differences: we distinguished an intermediate (IM) fraction between mitochondrial (MITO) and microsomal (Micr) fraction; the absence of high esterified cholesterol level in the postmicrosomal supernatant (Snt) indicates good liposomal (Lip) recovery; systematical morphological and morphometric control of subcellular fractions was made. Lastly we give results obtained only from rats, the cholesterol content and metabolism being different in adrenals which contain large amounts of esterified cholesterol (rat, guinea-pig, etc.) in comparison with those containing exclusively free cholesterol (beef, ewe, etc.)[4].

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