obtained in Peak II caused the agglutinated cells to adhere to glass. Thus, the property of sticking to glass appears uniquely associated with the 7S fraction of chicken serum.

An additional observation, in agreement with that reported for rabbit antibody(11), revealed that the incubation period necessary for agglutination is related to antibody molecular weight. For the macroglobulin fraction, strong agglutination took place in about 45 minutes, while for the low molecular weight antibodies having the same specificity, 10 minutes was sufficient.

With regard to the molecular weight of the isoantibodies, no clear-cut distinction between the histocompatibility (*B* and *C*) and the non-histocompatibility (*A*, *D* and *L*) blood group systems was found. However, the rate of 7S antibody production may be related to the antigenicity of the particular system. With our stock, nearly all individuals produce *B* antibodies upon immunization so that the *B* antigens are typically strong antigens. Relatively few individuals produce detectable *D* or *L* antibodies to the antigens we have tested. The *A* and *C* antigens in our population are intermediate in antigenicity.

Summary. Eighteen chicken blood typing antisera were fractionated into different molecular weight classes by passing through a column of Sephadex G-200 gel. These antisera, prepared by isoimmunization, were specific for antigens of 5 blood group systems. Antibody activity in sera specific for two systems (*D* and *L*) was present in the high but not the low molecular weight fraction. With *A*, *B* and *C* sera, antibody activity was usually present in both fractions although *B* antibodies were primarily of low molecular weight. The property of the agglutinated erythrocytes to stick to glass was associated with the low molecular weight chicken antibodies. Differences in the time required for strong agglutination were also associated with molecular weight. With the macroglobulins the incubation period required for agglutination was about 4 times longer than with low molecular weight antibodies.

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Effect of Plasmin on Outflow Resistance in the Primate Eye.* (30719)

MAURIZIO PANDOLFI AND TAGE ASTRUP
James F. Mitchell Foundation, Institute for Medical Research, Washington, D.C.

The mechanism regulating outflow of aqueous humor from the eye is incompletely understood. Hyaluronic acid, present in tissues of the angle of the anterior chamber of the eye, has been suggested as a regulator of filter resistance since application of hyaluronidase increases the outflow rate(1). However, hyaluronidase is active in some animal species and not in others(2). In the human eye its effect has been reported as equivocal(3). In a histochemical study of fibrinolytic active sites in the eye, the canal of Schlemm was

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TABLE I. Mean Outflow Facility (C) of 10 Pairs of Rhesus Monkey Eyes Perfused with Isotonic Saline After the Infusion of Progressively Increasing Amounts of Liquid.

ml infused	.05	.10	.25	.50	.90-1.0
Eye 1	.50 ± .07*	.45 ± .10	.55 ± .13	.66 ± .10	.86 ± .19
(Saline)	.42 – .62†	.30 – .58	.39 – .83	.55 – .85	.65 – 1.15
Eye 2	.52 ± .10	.47 ± .11	.55 ± .14	.67 ± .13	.78 ± .13
(Saline)	.42 – .76	.35 – .66	.35 – .76	.48 – .88	.60 – 1.00
Mean ratio: Eye 1 / Eye 2	.96	.96	1.0	.98	1.10

* Mean ± S.D. † Range.

found active(4). The activity is caused by an activator of plasminogen. It was suggested that this activity might aid in preventing clot formation obstructing drainage of aqueous humor. The presence of fibrinolytic activity in structures of the angle of the anterior chamber, and the presence of fibrinolytic activity in aqueous humor(4,5,6), could also suggest that fibrinolysis plays a regulating role in the outflow of aqueous humor. However, observations by Grant(3), using Actase® (a preparation consisting of an activator of plasminogen produced by addition of streptokinase to human euglobulin) or streptokinase (Varidase®), gave inconsistent results. Hence, to elucidate the possible role of fibrinolysis as a flow regulator, it was decided to investigate the effect of a genuine plasmin preparation.

Materials and method. Pairs of eyes of Rhesus monkey (*Macaca mulatta*), enucleated immediately after killing, were perfused *in vitro* according to the constant pressure method(1), using a 27 gauge needle attached to a one-ml pipet graduated in 1/100 ml. No regard was given to which eye was removed or cannulated first. Perfusion pressure was 27 cm of H_2O (20 mm Hg). The perfusion fluid was 0.15 M NaCl, either alone or containing plasmin (2 NOVO units/ml). The plasmin preparation was a trypsin-activated, porcine plasmin (NOVO Laboratories, Copenhagen, Batch 1-14, containing lysine, 25 NOVO units per vial) diluted in 0.15 M NaCl and adjusted to pH 7. The time required for 0.05 ml of the liquid to enter the eye was measured at intervals and the flow rate (facility of outflow, C) calculated as μl/minute per mm Hg. Readings began 1 to 3 minutes after cannulation. Each eye was perfused with 0.9 to 1.0 ml of liquid. Considering the deteriorating effect of the flow of saline on filter resistance(2,7), it was thought more appropriate to relate the perfusion to the amount of liquid perfused rather than to the time elapsed. For this reason, outflow facilities (C) were compared at points where equal volumes of perfusion fluid had entered the eye. Ten pairs of eyes were perfused with saline alone. Of 12 pairs one eye was perfused with saline containing plasmin, while the contralateral eye served as a control and received saline alone. Further, in 3 pairs of eyes after 0.2 ml saline had entered the eye, plasmin, yielding a calculated final concentration of 2 units per ml, was injected through the rubber tube connecting the needle with the pipet. Similarly, saline alone was added to the perfusion fluid of the contralateral eye.

Results and discussion. An experiment, typical for the perfusion of both eyes of a pair with saline, is shown in Fig. 1. There is good agreement between the measurements obtained. The average values of outflow facility, with range and estimated standard deviation, are presented in Table I. All comparisons were made on pairs, because there are differences between the eyes from separate individuals. The short flow time observed in the first recording in Fig. 1 (corresponding to the slightly raised calculated value for the outflow facility, Table I) does not represent actual outflow but is caused by the stretching of the eyeball under the pressure of the perfusion liquid. After equilibrium has been reached, the flow rate progressively increases approaching a level. When the ratios between the averages are calculated for each point of

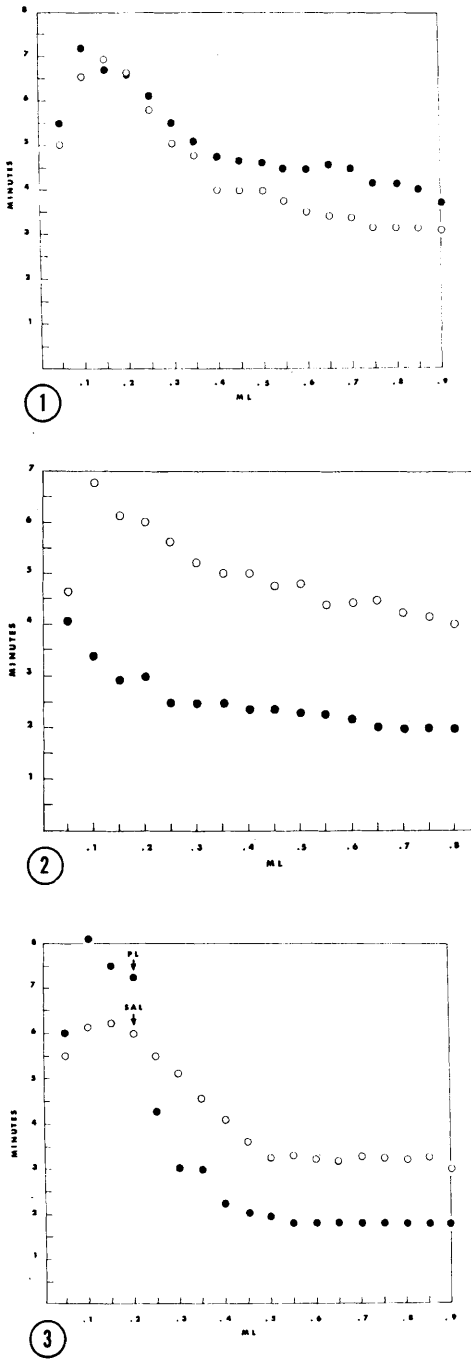

FIG. 1. Perfusion of a pair of eyes with saline. Abscissa: volume of perfusion fluid (in ml) entering the eye. Ordinate: time in min required for 0.05 ml of perfusion fluid to enter the eye.

FIG. 2. Influence of plasmin on flow resistance in a pair of eyes. Abscissa and ordinate as in Fig. 1. Eye 1 (full circles) perfused with saline containing plasmin. Eye 2 (empty circles) perfused with saline alone.

FIG. 3. Influence of plasmin added during perfusion. Abscissa and ordinate as in Fig. 1. Both eyes originally perfused with saline. After perfusion of 0.20 ml saline, plasmin was added (at arrow) to the perfusion fluid of eye 1. A similar amount of saline was added to the perfusion fluid of eye 2.

determination (Table I), the deviation between the eyes in a series of pairs does not exceed 10%.

A perfusion with plasmin is shown in Fig. 2. There is an immediate decrease in flow resistance even concealing the effect of the stretching of the eye. The flow times decrease progressively and level off at a much lower value than in the saline control. The average values of flow facility of a series of 12 pairs of eyes, with range and estimated standard deviation, are presented in Table II. The difference between the plasmin-treated and the control eyes is pronounced yielding a ratio of approximately 1.6 over the whole range of perfusion, excepting the first determination. When plasmin was added during the perfusion, the immediate effect of plasmin on the flow resistance is particularly striking (Fig. 3). It is apparent that the effect of plasmin is rapid, and that a much lower level of resistance is reached than after saline alone.

The present findings show that analogous to the effect of hyaluronidase in certain animal species, the plasmin preparation used has an immediate and striking effect on the outflow rate of the monkey eye. Though the cause of this effect is not exactly known, results of experiments with protease inhibitors indicate that the effect is produced by plasmin proper. Hence, it is believed that there is in the eye of the primate a plasmin-sensitive barrier to the outflow of aqueous humor. Taken together with the recent observation of fibrinolytic activity related to the canal of Schlemm and the presence of fibrinolytic activity in aqueous humor, this suggests that the fibrinolytic system could be involved in the regulation of flow resistance.

Summary. In vitro perfusion with plasmin (porcine, trypsin-activated) decreases the flow resistance for aqueous humor in the eye

TABLE II. Mean Outflow Facility (C) of 12 Pairs of Rhesus Monkey Eyes After Infusion of Progessively Increasing Amounts of Liquid. One eye of each pair was perfused with saline containing plasmin (Eye 1), while the contralateral eye was perfused with saline alone (Eye 2).

ml infused	.05	.10	.25	.50	.90-1.0
Eye 1	.58 ± .16*	.61 ± .16	.83 ± .20	1.01 ± .23	1.14 ± .27
(Plasmin)	.34 – .81†	.38 – .83	.48 – 1.07	.62 – 1.25	.63 – 1.36
Eye 2	.45 ± .15	.38 ± .12	.50 ± .13	.63 ± .17	.67 ± .15
(Saline)	.21 – .58	.18 – .63	.31 – .76	.41 – .88	.45 – .93
Mean ratio: Eye 1 / Eye 2	1.29	1.60	1.66	1.60	1.70

* Mean ± S.D. † Range.

of monkey (*Macaca mulatta*). This effect is immediate and striking and levels off at a lower value than reached with saline alone. The effect resembles that produced by hyaluronidase in some animal species.

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Effect of Sodium State on Reactivity of Renal Juxtaglomerular Cells And Adrenal Zona Glomerulosa. (30720)

EDWIN R. FISHER AND HARVEY Z. KLEIN

Departments of Pathology, University of Pittsburgh and VA Hospital, Pittsburgh, Pa.

Sodium deficiency increases the degree of granulation of renal juxtaglomerular cells (JGI) in several species, including man, whereas the converse has been observed in situations characterized by sodium retention. These alterations of JGI have been considered to result from changes in blood volume and pressure attendant with these Na states; and the juxtaglomerular apparatus has been visualized as a stretch receptor(1). Recent studies in our laboratory with hypertensive and nephrotic rats subjected to sodium depletion or salt overload delineated pressor and electrolytic effects on the juxtaglomerular apparatus. The results suggested that this morphologic unit might also represent an osmoreceptor(2-4). These, as well as contemplated studies of the juxtaglomerular apparatus, disclosed the need for information concerning the rate and degree of change in JGI following sodium depletion or salt overload and the recovery from these states. Miller and Hartroft(5) briefly noted the occurrence of prompt degranulation followed by hypergranulation of juxtaglomerular cells after administration of a sodium deficient diet. A similar reaction has been observed following partial renal artery occlusion(6).

Lack of a systematic study of the responsiveness of juxtaglomerular cells to sodium depletion or salt overload and the recovery phases from these states has prompted this report. The changes in JGI, which reflect renal renin content, have also been correlated with the width of the zona glomerulosa of adrenal cortices (ZGI), a morphologic index of adrenal cortical aldosterone production, be-