'ganglionectomy effect'. This finding suggested that the locus of action for 'α' active adrenergic material is on the trabecular meshwork. In the work reported here no effect was noted after an intra-vitreous injection of 1·0 μg of l-noradrenaline bitartrate. By contrast, marked effects were noted with only 20 mμg after anterior chamber injection. Considerations of diffusion, protein binding, and degradation will of course necessitate high intra-vitreous dosage. The great sensitivity demonstrated with anterior chamber injection, however, would tend to exclude ciliary muscle and/or vascular effects to explain the resistance changes, and place the site of action in the trabecular meshwork.

Evidence for β or inhibitory adrenergic effects on the trabecular meshwork are inconclusive at the present time. Attempts to reproduce the elevated outflow resistance found one week after ganglionectomy using isoproterenol, a high β active material, showed that at low dose-levels isoproterenol acted more like noradrenaline, a strong α and a weak β stimulator. It is possible that at higher dose-levels, isoproterenol did have both β and α activity since the drop in outflow resistance was less than that which occurred with noradrenaline.

Parallel clinical studies of outflow resistance in patients with Horner's syndrome[12] also show a greater outflow facility on the homolateral side. The outflow resistance is further decreased after topical epinephrine bitartrate. Essentially similar results have already been reported by Krishna[13], who made a pharmacological denervation with topical guanethidine and noted a further reduction in intra-ocular pressure and outflow resistance when topical epinephrine was added.

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Distribution of Precorticotrophin in Blood

In previous communications[1-3] the existence of precorticotrophin—a precursor of corticotrophin—was demonstrated in the anterior pituitary tissue of the ox. Later, I[4] reported the presence of a material behaving like precorticotrophin in normal rabbit blood serum. Young[5] observed that the insulin-like activity of the serum or plasma from arterial blood, or from blood oxygenated *in vitro*, was higher than that of serum or plasma from comparable venous blood, or blood deoxygenated *in vitro*. In view of this, it appeared worth while to investigate the presence of precorticotrophin-like material in sera obtained individually from arterial and venous blood.

Blood collected from carotid artery and from jugular vein of normal male rabbit under 'Nembutal' anæsthesia was allowed to stand overnight in a refrigerator. Next day sera were collected and freeze-dried. Just before the experiment each serum sample was dissolved in distilled water such that 1 ml. of the solution corresponds to 1 mg of the sample. A portion of such solution was next treated with urea in such an amount that its concentration was 6 M with respect to its final volume. Corticotrophic activity of both portions of solutions, untreated and urea treated, was determined by the Sayers method as used in the previous investigation[4].

Table 1. Mean Sayers Activity of Arterial and Venous Blood Serum of Normal Rabbit

Group	Treatment	Mean adrenal ascorbic acid depletion (mg/100 g) ± S.E.
A	Saline only	35·8 ± 10·45 (15)*
B	Standard ACTH† solution (10 mμ/ml.)	77·2 ± 15·91 (6)
C	Arterial serum	37·0 ± 10·08 (20)
D	Arterial serum urea treated	75·1 ± 8·74 (20)
E	Venous serum	62·7 ± 9·81 (12)
F	Venous serum urea treated	53·3 ± 14·32 (12)

* Figures in parentheses indicate total number of observations.
† Gift of standard ACTH from Dr. D. B. Ghosh, director, Central Drug Laboratory, Calcutta, is acknowledged.

Table 1 shows the results obtained in the Sayers test when the test animals were injected intravenously with different test solutions at a dose of 0·25 ml./100 g body-weight. It is found that the response produced in the Sayers test by the arterial serum (Group *C*) is of the same order as that produced by saline only (Group *A*). But the response produced by the urea-treated arterial serum (Group *D*) is significantly higher ($0·001 < P < 0·01$) than that produced by untreated arterial serum (Group *C*). This indicates the presence of a precorticotrophin-like material in the arterial serum. On the other hand, venous serum untreated (Group *E*) compared with the group on saline only (Group *A*) appears to possess slight, although not significant ($P < 0·40$), activity in the Sayers test; such activity is not enhanced further after treatment of the venous serum with urea (Group *F*). It therefore appears that the precorticotrophin-like material which was reported earlier to be present in circulation[4] is demonstrable only in arterial but not in venous blood serum. Further, it explains, at least partly, the controversy that exists regarding the presence of corticotrophin in blood.

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Preparation of Antisera to Human Follicle-stimulating Hormone

We have previously reported the preparation of antisera to fractions of human pituitary follicle-stimulating hormone which inhibited the activity of homologous antigens in mice and agglutinated red cells coated with the antigens, but they were ineffective against human chorionic gonadotrophin in rats[1]. The antisera have since been found to cross-react with human chorionic gonadotrophin in the red cell technique and when absorbed with human chorionic gonadotrophin they no longer agglutinated red cells coated with follicle-stimulating hormone. The antibodies, therefore, are not specific for follicle-stimulating hormone and we have now prepared antisera to new fractions.

The method of fractionation was by starch-gel electrophoresis, which is described fully elsewhere[2]. The segment of gel containing the follicle-stimulating hormone activity was eluted in saline, mixed with ben-