Moreover, the number of amino-acid residues per molecule calculated from the present amino-acid analysis (Table 2) is greater than the number calculated by Tristram[7]. The present figures were obtained from 24-hr. and 96-hr. hydrolysates (6 *N* hydrochloric acid under reflux) and corrections were made for the decomposition of certain amino-acids. When compared with Tristram's figures, it is seen that there is an increase in the number of residues of leucine – *iso*leucine (4), phenylalanine (2), glycine (2), valine (1) and histidine (1) and a decrease of one residue of serine.

A full account of these experiments will be published later.

While this work was in progress, Theorell and Åkeson[8] recorded an electrophoretic diagram for myoglobin showing three components. The main component, Mb 1, had an equivalent weight of 18,800, calculated from iron analyses, nearly equal to the molecular weight of our fraction MbCO*A*.

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An Extrarenal Effect of Pitressin

It is generally believed that the action of pitressin, and more specifically, antidiuretic hormone, is entirely referable to its effects on the kidney. Although an extrarenal site of action has been suggested, the evidence has not been convincing[1]. Our recent studies in the rat suggested that pitressin might have an extrarenal action in this mammal[2]. This would agree with the findings in other vertebrate classes[3].

The problem was studied in the rat using inulin, as modified from the method of Ledingham[4], to measure extracellular fluid volume. The renal pedicles were first ligated. One hour later, a measured amount of inulin was injected into the right femoral vein and two hours allowed for equilibration. At this time (0 min.), using light ether anæsthesia, a 0·5-ml. sample of blood was obtained from the left femoral artery and analysed for inulin and sodium. This was followed immediately by the injection of 1 I.U. of pitressin (Parke, Davis) in 0·5 ml. of saline intraperitoneally in a group of ten animals. A control group of ten received the saline only. A second blood sample was obtained 40 min. later from the same artery. The procedure was repeated in fresh groups to cover intervals of 60, 90 and 120 min. after injection of pitressin.

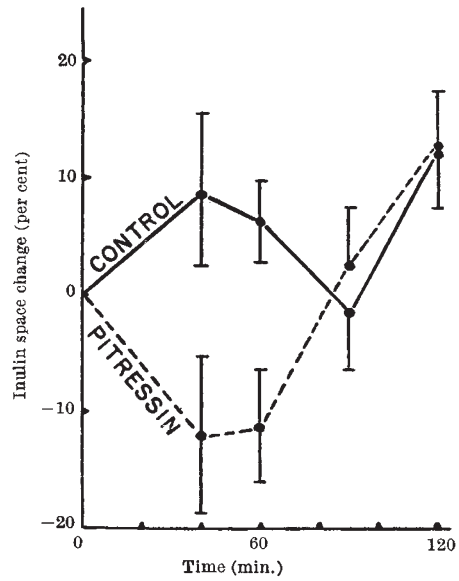

Fig. 1. Effect of 1 I.U. of pitressin intraperitoneally on inulin space in nephrectomized rats. Time of injection of pitressin is taken as 0, inulin having been injected 2 hr. previously to allow for equilibration

As shown in Fig. 1, pitressin caused a significant decrease in inulin space which can only signify a movement of water into the intracellular compartment. This was apparently accompanied isosmotically by sodium, since the plasma concentration of this ion remained unchanged throughout despite the decline in inulin space. In Table 1 the change in inulin space in two separate experiments is shown for the period of 60 min. after injection of pitressin.

Table 1. Effect of 1 I.U. of Pitressin on Inulin Space in Nephrectomized Rats

The first space determination, at 0 min., was carried out 2 hr. after inulin injection and was followed immediately by the injection of pitressin in the treated animals and saline in the control animals

	0 min.	60 min.	Change (per cent)	
Experiment 1				
Control average	26·64 ml.	27·86 ml.	+6·46	
Standard error	±1·93	±1·67	±3·51	
Pitressin average	26·63	23·59	−11·05	$p < 0·01$
Standard error	±1·37	±1·71	±5·02	
Experiment 2				
Control average	23·66 ml.	22·33 ml.	−5·53	
Standard error	±0·75	±0·93	±2·70	
Pitressin average	23·21	19·48	−15·95	$p < 0·02$
Standard error	±0·98	±1·12	±3·51	

The details of this shift as studied using smaller doses of pitressin intravenously, together with the effects of sodium and potassium, will be reported separately.

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