A similar phosphaturic effect as seen with L-alanine has been shown with the neutral amino-acids L-valine, L-tryptophan, and glycine. The basic amino-acids, L-lysine and L-arginine, in doses of 0·05 mole, did not cause an increased excretion of phosphate.

Investigations on human beings have been carried out in our laboratory which demonstrated an identical inhibition of phosphate reabsorption by L-alanine.

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Phosgene Formation in Uræmia

In a previous communication, I reported that the formation of carbonyl chloride in a state of high blood urea-level associated with acidæmia is responsible for uræmic coma. It was also claimed that phosgene is not formed under normal conditions in blood due to the absence of biologically available urea and acidæmia. Since then, investigations from different aspects have been pursued to confirm that uræmia is attributable to the formation of phosgene as claimed by the author. The results have lead to some additional direct evidences which are reported here.

A state of asthenic uræmia was induced in a set of three dogs (each weighing about 10 kg) by tying both the ureters at operation. The dogs developed uræmic coma with all the associated symptoms and died 80–84 h following the operation. Immediately on death, a sample of blood was collected from the heart. The sample was then laked. Another sample of blood was taken from a normal animal serving as the control. The hæmoglobin of the treated animal was matched against that of the normal animal and both the samples were subjected to test for phosgene only when the concentration of hæmoglobin was identical in both. The phosgene test was performed by the standard method involving the use of 10 per cent alcoholic solution of *p*-dimethyl aminobenzaldehyde and colourless diphenylamine[2]. Positive reaction was obtained in each case of the uræmic animal while the control yielded no reaction whatsoever, thus confirming the cause of uræmia as claimed previously[1]. It may be noted that these experimental animals did not receive any urea or ammonium chloride solution intravenously.

Since carbonyl chloride reacts vigorously with water, producing carbon dioxide and hydrochloric acid, recovery experiments following dialysis of human uræmic subjects were not performed. Furthermore, positive indication of presence of carbonyl chloride in uræmic cases has been obtained from samples of blood taken from human uræmic subjects in coma[1].

In two more cases of coma recently investigated where the patients had high blood urea-level but no acidæmia (as proved by the alkaline reaction of the urine), phosgene could not be detected in their blood. This result serves as further evidence of the fact that uræmic coma is only attributable to the formation of phosgene.

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Inhibitory Effect of Pre-optic Stimulation on Adrenal 17-Hydroxycorticosteroid Secretion Rate in the Cat

In diencephalic stimulation experiments of Suzuki *et al.*[1] a definite decrease in 17-hydroxycorticosteroid (17-OHCS) secretion rate was observed in one dog after stimulation of the pre-optic area. Similar observation was made in one cat with éncephale isolé by Slusher and Hyde[2]. These observations suggested a possible inhibitory effect of pre-optic stimulation on the pituitary-adrenocortical function. However, since they were made only on one dog and on one cat, many more experiments seem to be necessary before this problem can be settled. The present investigation was undertaken in order to settle this problem.

Cats were used: they were anæsthetized with intraperitoneal 'Nembutal' (25 mg/kg body-wt.). The adrenal venous blood was collected through the lumbar route by a slight modification of the procedure described in the paper of Suzuki and Arai[3]. Steel bipolar electrodes, 0·25 mm in diameter, were placed in the pre-optic area with the aid of the electrode holder of the Hess-type fixed on the skull. Interpolar distance of the electrodes was 1·5 mm; 30 min after making these arrangements, a control adrenal venous blood sample was collected. 15 min after collection of the control blood sample, electrical stimulation of the pre-optic area was performed. The rectangular currents, 2 V, 20/sec and 1 msec in duration were supplied by an electronic stimulator. A 45-sec stimulus was applied in 2 cats (cats 6 and 7) and a 30-sec stimulus every minute for 5 min in 3 cats (cats 8, 9, and 10). At 15, 30, 60, and 90 min after the start of stimulation, adrenal venous blood samples were collected. On 5 other cats, control experiments were performed without stimulating the pre-optic area. As a fall in body temperature seriously affects the adrenocortical function[4], a heat lamp was carefully applied so as to maintain constant body temperature. The adrenal venous blood samples were centrifuged immediately after collection. The 17-OHCS content of the adrenal venous plasma was measured by the method of Nelson and Samuels[5]. The rate of secretion of 17-OHCS was calculated by multiplying the plasma 17-OHCS by the adrenal plasma flow. After experiments the brain was fixed in formalin solution and serially sectioned. Stimulated sites were defined microscopically.

Results are presented in Table 1. Inasmuch as the levels of pre-stimulation values are variable from cat to cat, they are taken in each cat as 100 per cent and all the secretion rates are expressed as a percentage of the

Table 1. Effect of Pre-optic Stimulation on Adrenal 17-Hydroxycorticosteroid Secretion Rate in the Cat

17-Hydroxycorticosteroid secretion rate (expressed as percentage of the pre-stimulation value)

Cat No.	Before stimulation	Min after stimulation 15	30	60	90
(Control experiments)					
1	100	119	121	117	97
2	100	106	106	96	94
3	100	104	113	125	124
4	100	98	106	112	95
5	100	105	99	97	97
Mean ± *S.E.*	100	106 ± 3·3	109 ± 3·6	109 ± 5·7	101 ± 5·7
(Pre-optic stimulation experiments)					
6	100	59	68	64	51
7	100	73	70	66	50
8	100	73	79	80	85
9	100	96	67	71	82
10	100	78	89	59	84
Mean ± *S.E.*	100	76 ± 6·0	75 ± 4·2	68 ± 3·6	70 ± 8·1