phenylalanine, however, led to a large increase of hydroxytyramine not only in the cell sap, but also, and to a similar extent, in the mitochrondria in the brain of rabbits treated with reserpine. The concentrations of adrenaline and noradrenaline were restored to the level observed in the controls, but did not rise above it. Small amounts of dihydroxyphenylalanine were found in the mitochrondria 0·5 hr. after injection, but none after 5 hr.

It thus appears that, if the theory of reserpine action is correct, it is not permissible to equate the bound and free forms of amines with the fractions contained in mitochondria and cellular sap respectively.

The techniques used in this investigation were similar to those used in a previous study[3]. They will be described fully elsewhere.

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Micro Estimation of Urea

A qualitative test for the recognition of urine contamination of fabrics has been described by Ishler *et al.*[1]. With the aid of urease the ammonia evolved was detected with Feigl's reagent[2]. This reagent (silver manganese nitrate solution) appears to be rather more sensitive to ammonia than is Nessler's reagent (alkaline potassium–mercuri-iodide solution), having its lower limit of detection in the order of 0·5 μgm. ammonia. Using Feigl's reagent and a filter paper spot test technique we have devised a rapid and economic method of determining the concentration of urea in 5–20 μl. of biological fluid.

Approximately 20 μl. fresh blood, cerebrospinal fluid, diluted urine, etc., are accurately pipetted into a small (50 mm. × 8 mm.) citrated tube to which is added one drop of 10 per cent w/v soya bean meal suspension (prepared in 0·1 per cent citrate and buffered with sodium hydroxide so that the *p*H falls within the range 6·8–7·2). A washed filter paper (Whatman No. 3 *MM*) is then clamped over the open end of the tube and moistened with 1 drop of Feigl's reagent. At the end of 6 min. incubation in an oven at 120° C. the intensity of the manganese dioxide/free silver deposit is compared with those obtained by similarly treated standard urea solutions. Under these conditions the urease suspension is capable of converting 30 μgm. urea quantitatively to ammonia within 1 min. (Table 1). No ammonia has been detected as a result of heating either human blood or urease suspension alone for 10 min. at 130° C.

Blood analyses are frequently performed in all hospital biochemistry laboratories in order to assess the extent of urea retention. In view of this, and the fact that the classical methods of Van Slyke and Cullen[3] and Archer and Robb[4] require about an hour to complete 12 estimations, a simple apparatus has been designed which deals successfully with 24 analyses in less than 15 min. In pædiatric units our method has obvious advantages, and furthermore, may be used to screen a larger number of patients for hyperuræmia than would otherwise be possible.

Table 1. Recovery of Urea added to Blood

Urea added (mgm./ml.)	Urea found (mgm./ml.)	Urea recovered (mgm./ml.)
0·0	0·15	—
0·3	0·35	0·2
0·51	0·6	0·45
0·78	0·9	0·75
1·26	1·4	1·25
1·72	1·7	1·55
2·15	2·2	2·05
2·46	2·8	2·65

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Audiogenic Epileptic Seizures evoked in Rats by Artificial Epileptogenic Foci

We consider the so-called audiogenic epilepsy in rats to be epileptic seizures of the procursive or procursively convulsive type which can be produced as a reflex to a strong acoustic stimulus.

Hitherto, seizures of audiogenic epilepsy have been evoked either in initially sensitive animals or in rats the sensitivity of which had been increased pharmacologically or metabolically. In our experiments we tried to produce a predisposition to audiogenic seizures in non-sensitive animals by creating an artificial epileptogenic focus by the local application of alumina-cream—a method introduced into experimental epileptology by Kopeloff, Barrera and Kopeloff[1].

Experiments were performed on rats, approximately two months old, the sensitivity of which to acoustic epileptogenic stimuli was determined by repeated testing. Six tests, consisting of a powerful electric bell sounding for two minutes in a cylindrical, 50 cm. × 50 cm., sound-proof box, were given in the course of two weeks. A thin gel of aluminium hydroxide was applied by micro-injection, with the aid of the stereotaxic apparatus, into three different regions of the brain. Rats were then observed and tested weekly for 5–12 months.

2–6 mm.^{3} of the cream were injected subdurally or intracortically into one or both motor areas (area 4) or into the acoustic area (area 41 according to Krieg[2]) either uni- or bi-laterally in 75 rats. Audiogenic seizures after a latent period of 2–77 days were observed in 25 rats (33 per cent). The dependence of this artificial audiogenic epilepsy on the position of the focus is evident from Fig. 1. Foci in the optic zone (area 17) in control experiments, and in the cerebellar cortex, did not evoke audiogenic seizures.

In 35 rats, 1–3 mm.^{3} of alumina-cream was injected into the inferior colliculo of the lamina quadrigemina. Audiogenic seizures were observed in 34 per cent of animals after a latent period of 9–219 days. Seizures