this. That the correlation is far from perfect is an indication that the equivalent circuit in Fig. 1 is at best an approximation.

So far as the action of thorium is concerned, it is well known that glass-electrolyte interfacial potentials can be eliminated by trace concentrations of heavy metal ions while electrochemical junction potential differences are not affected[5]. It should be noted in Table 1 that thorium chloride produces a reversal of sign.

We therefore suggest that tip potentials are related to interfacial potentials, perhaps according to the scheme shown in Fig. 1, but that in any case they can be controlled by appropriate concentrations of thorium chloride in the extracellular solution. A constant error still remains because of the tip potential associated with the intracellular solution, but for experiments involving variation of extracellular ion concentrations, this error is eliminated by treating differences rather than absolute magnitudes.

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3,4-Dimethoxyphenylethylamine in Schizophrenia

REPORTS of the occurrence of 3,4-dimethoxyphenylethylamine (DMPE) in the urine of schizophrenic patients are attracting increasing attention[1,2], although some of the chromatographic procedures used to detect this amine have been criticized as being insufficiently definitive. This communication reports an investigation in which, using two-way thin-layer chromatography, we have failed to detect urinary DMPE in any of twenty-two schizophrenic patients. In seventeen of the patients the illness was of recent acute onset, the remaining five having been ill for several years. Of the seventeen acute cases, fourteen had received no previous medication at all for their condition, while the other three had been treated with barbiturates only. None of the five chronically ill patients had received drug therapy for at least 3 years. No dietary restrictions were imposed.

Twenty-four hour urine collections were made from each patient and a volume of 400–500 ml. was adjusted to *p*H 9 with sodium hydroxide solution and extracted with chloroform (2 × 200 ml.). The dried extract was shaken thoroughly with N/1 hydrochloric acid (2 × 10 ml.), which was then evaporated to dryness *in vacuo*. The solid residue was dissolved in a small volume of aqueous methanol and chromatographed in two dimensions (15 cm each way) on silica-gel plates, using the solvents described by Kuehl *et al.*[3]. Primary amines were located with ninhydrin reagent. As little as 3 μg of DMPE in each 400 ml. of urine could be detected by this method, as shown by adding appropriate amounts of authentic amine to urine.

Initially, in sixteen cases the presence of a purple spot in very close proximity to the position indicated for DMPE was interpreted as resulting from the latter. Subsequent work, in which two chromatograms were run (one with added DMPE) for each patient, however, indicated that this spot was not exactly coincident with DMPE, and in no case were we able to locate a pink spot corresponding to the DMPE on the duplicate chromatograms. Furthermore, overspraying the chromatograms with Ehrlich's reagent, as described by Friedhoff and Van Winkle[4], failed to demonstrate an orange spot in the DMPE position, except when this amine had been deliberately added. It would thus seem that, in the cases we have studied, either excretion of DMPE did not occur or that the amounts excreted were below the limit of detection of 10–15 μg/24 h.

This result is surprising in view of the large proportion of acute subjects in our sample, a fact which other workers have indicated as being favourable for detection of DMPE. We would tentatively suggest that our use of two-dimensional thin-layer chromatography, with its excellent resolution and thus reduced probability of detecting false positives, plus the definite absence of drug metabolites which are known to interfere with detection of DMPE, might be important factors in the results obtained in this study.

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Recoveries of Iodine-131-labelled Iodo-amino-acids in the Plasma Protein-bound Iodine Assay

IT is the orthodox view that circulating thyroid hormone consists mainly of thyroxine (T_4), together with small amounts of triiodothyronine (T_3), while the iodotyrosines account for only 0–4 per cent of the total organic iodine[1–4]. There have been reports[5–8], however, that the iodotyrosines account for 30–50 per cent of the iodine in normal human blood. In the condition of thyrotoxicosis, quite large amounts of diiodotyrosine (DIT) are detected in the plasma, although the amount has not yet been precisely quantitated[3,4,9].

Most of the reports on the recovery of organic iodine compounds by means of the various protein-bound iodine (PBI) procedures refer only to the recovery of iodothyronines. However, there are two reports[10,11] which suggest that there are marked differences in the extents of recovery of the iodotyrosines by the two commonly used protein precipitation procedures, namely, the zinc sulphate-sodium hydroxide (Somogyi) system and the trichloroacetic acid (TCA) system.

In view of the increased attention which has been given to the possibility of finding greater amounts of iodotyrosines in the blood of normal people than were previously estimated, we decided to investigate the recovery of iodine-131-labelled monoiodotyrosine (MIT) and diiodotyrosine using both the Somogyi and trichloroacetic acid systems throughout the PBI procedure. At the same time, the opportunity was taken to re-investigate the recovery of iodine-131-labelled T_3 and T_4. The recovery of each iodo-amino-acid was studied after addition to plasma samples of diverse ability to bind thyroid hormone.

Blood was withdrawn from a healthy volunteer, heparinized and the plasma separated under conditions free of iodine. Plasma containing increased amounts of thyroid binding proteins (TBP) was obtained by pooling specimens from four euthyroid, pregnant patients.