Fig. 1. Postulated amino-acetone cycle. The interrupted line represents a possible deamination reaction

The accumulation of amino-acetone in cultures of *Staph. aureus* is presumably due to the inability of this organism to deaminate the compound. Such a metabolic defect would be entirely in keeping with the limited nitrogen metabolism implied by the strict amino-acid requirements of *Staph. aureus*.

Experiments to test the validity of the above hypothesis are now in progress and, as with full details of the present work, will be reported later.

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Identification of Barbiturates

IN Great Britain the most common poisons at the present time are the 5 : 5' disubstituted barbiturates. The longer-acting barbiturates, barbitone and phenobarbitone, can accumulate in the body when administered in relatively low therapeutic dosage, and concentrations of these drugs in the blood of about 5 mgm./100 ml. do not necessarily imply poison-

ing. The shorter-acting barbiturates, when taken in therapeutic quantities, however, do not accumulate, and in such cases this concentration would imply that an excessive quantity had been taken.

The only reliable method for the quantitative assay of barbiturate in 5-10 ml. samples of blood is ultra-violet spectrophotometry. The optical density difference at 240 mμ of aliquots at pH 10 and pH 2 is measured, and, because the *E*(1 per cent, 1 cm.) values for the various barbiturates are very similar, the total barbiturate concentration can be obtained. In order to assess the likely effect of this concentration it is necessary to identify the ingested barbiturate or at least assign it to the long- or short-acting group. Although slight differences in spectra between the various barbiturates can be detected and used for identification purposes¹, they are of value only if the background absorption is minimal and one barbiturate is present.

It is the purpose of this communication to direct attention to a test which has proved valuable in assisting in the identification of the various barbiturates. Maynert and Washburn² showed that concentrated sulphuric acid at room temperature dealkylated some secondary alkyl groups in the 5 : 5' disubstituted barbiturates, and Brooker³, using treatment with acid at 100° for 1 hr., separated by paper chromatography the products from pentobarbitone and amylbarbitone. Combination of the techniques used by these workers has shown that if the products of the acid treatment at 100° for 1 hr. are, after dilution with water and extraction into ether, examined by ultra-violet spectrophotometry using 0.5 *N* aqueous ammonia as the solvent, then rapid and easy differentiation of several barbiturates is possible. The results of such treatment on the barbiturates commonly prescribed in the United Kingdom are shown in Table 1.

Table 1

Barbitone	Result of treatment	Maximum of product in 0.5 <i>N</i> ammonium hydroxide
Amylbarbitone (amytal)	Unchanged	240 mμ
Butobarbitone (soneryl)		
Pentobarbitone (Nembutal)	Attacked	268 mμ
Cyclobarbitone (phanodorm)	Attacked	Very weak absorption at 240, 266 and 315 mμ
Quinalbarbitone (seconal)	Attacked	Very weak absorption at 240, 266 and 315 mμ
Phenobarbitone	Completely destroyed	Nil

The products from the dealkylation of 'Tuinal' (mixed amylbarbitone and quinalbarbitone) give detectable absorption maxima at 240 mμ and 315 mμ. The absorption from the treatment of seconal is, however, very low and the 315 mμ peak is usually only observable when the extracts are reasonably pure.

The tungstic acid precipitation method followed by isolation of the weakly acidic ether-soluble fraction gives excellent products.

These techniques, coupled with those of paper chromatography⁴, have, as yet, not failed to identify unambiguously any of the barbiturates involved in more than one hundred cases submitted to this laboratory in which therapeutic or toxic doses had been ingested.

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