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THE DECOMPOSITION OF SOLUTIONS OF SODIUM SULFIDE.*

BY G. BULFER, A. J. BOYLE AND L. H. BALDINGER.[1]

INTRODUCTION.

The decomposition of sodium sulfide in aqueous solution has long been a subject of interest. Thomsen (1) has reported that a solution of sodium sulfide is almost completely hydrolyzed into sodium hydrosulfide and sodium hydroxide. This agrees with the reports of Knox, Bauer, Kolbe and others (2). Küster and Heberlein (3) showed that the hydrolysis of sodium sulfide in a tenth-normal solution occurs to the extent of 86.4% and results in the formation of sodium hydrosulfide and sodium hydroxide. They further reported the hydrolysis of sodium hydrosulfide into hydrogen sulfide and sodium hydroxide to the extent of 0.14 per cent.

Aqueous solutions of sodium sulfide are also oxidized in air with the formation of sodium thiosulfate and sodium bicarbonate. Mitscherlich (2) states that half of the sulfide is converted to the thiosulfate and half to the bicarbonate in the oxidation. Mellor (2) reported that carbon dioxide reacts with aqueous solutions of sodium sulfide converting it to sodium hydrosulfide and sodium bicarbonate. Borntrager (4), while investigating the analysis for copper using sodium sulfide solution, found that increasingly large amounts of sulfide solution were necessary as the solution became older. One sample showed 66.6% decomposition in a period of one month.

Sodium sulfide in varying concentrations, both in water solution and in paste form, can be used as a depilatory. The effectiveness of the preparations, attributed to the sulfide ion, is impaired by the decomposition of the sulfide salt in aqueous solution. Organic compounds such as glycerol and carbitol (diethyl ether of ethylene glycol)[2] have been added to depilatory preparations to inhibit decomposition. The present problem was undertaken to study briefly the effectiveness of these two compounds as inhibitors of the decomposition of sodium sulfide solutions.

ANALYTICAL PROCEDURE AND DATA.

A rapid method of analysis of alkali sulfides was desirable for the proposed work. The standard and approved method of oxidation of the sulfide to the sulfate, precipitation and determination as barium sulfate was ruled out because of the time involved. Calcott, English and Downing (5) found that a mixture of ammonium chloride and sodium chloride readily evolves hydrogen sulfide from sodium sulfide while other substances which may be present in the sodium

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magnesium citrate. It was furthermore noted that those samples in which the magnesium oxide content was higher showed considerably more precipitation. This is in accordance with the findings of Oakley and Krantz (3) who showed that when citric acid in solution is treated with magnesium oxide tribasic magnesium citrate will be formed in solution before all the hydrogen ions arising from the primary and secondary dissociations of citric acid have been neutralized and that, consequently, an excess of citric acid would act in such a manner as to stabilize the solution against precipitation by virtue of its shifting the equilibrium in the direction of forming an acid citrate.

Realizing the importance of the free (titratable) acidity of solution of magnesium citrate the free acidities of the various experimental lots were determined, expressed in terms of cc. of $N/2$ NaOH required to neutralize 10 cc. of the solution in the presence of phenolphthalein indicator. It was found that the actual values obtained corresponded quite closely with those obtained theoretically by calculation, based on the magnesium oxide, potassium bicarbonate and citric acid content. This substantiated the results of Mayer (4) who found such a calculation to give a result very close to that obtained by actual titration.

As an illustration of this close conformity between actual and calculated free acidity values the following are given as examples:

	Actual Value.	Calculated Value.
Sample No. 2	10.13 cc.	10.10 cc.
Sample No. 4	8.76 cc.	8.66 cc.

In no case was the difference between the calculated and actual values greater than 0.3 cc.

Inasmuch as all the samples of solution of magnesium citrate containing potassium bicarbonate prepared in the laboratory showed evidence of precipitation, the authors were led to examine several of the commercial products on the market. Five samples were purchased from retail pharmacies in Philadelphia. Each sample was manufactured by a different large scale producer of this product, and was labeled with the official title.

All five samples were found to be perfectly clear without the slightest trace of precipitate being present. Each was then assayed for magnesium oxide, free acidity and total citric acid. The results were as follows: (Slide II.)

RESULTS OF ANALYSES OF COMMERCIAL SAMPLES OF SOLUTION OF MAGNESIUM CITRATE U. S. P. XI.

Sample No.	MgO Gm./ 100 Cc.	Free Acidity Cc. $N/2$ NaOH per 10-Cc. Solution.	Free Citric Acid (Calculated from Free Acidity Titration) Gm./350 Cc.	Total Citric Acid Gm./350 Cc.
1	1.676	10.92	13.38	33.46
2	1.680	8.89	10.89	31.15
3	1.633	9.18	11.26	30.91
4	1.461	9.58	11.74	29.28
5	1.652	10.92	13.38	33.64

From these figures it will be seen that all were of U. S. P. quality in regard to magnesium oxide with the exception of No. 4 which was 0.139 Gm./100 cc. lower than the U. S. P. XI minimum of 1.6 Gm./100 cc. Samples No. 2, No. 3 and No. 4 were below the U. S. P. XI minimum for total citric acid which may be shown by calculation to be 31.85 Gm. per 350 cc.

In regard to total citric acid deficiency it may be said here that, in the opinion of the authors, the requirement of the U. S. P. XI seems a little too rigid.

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COMPARISON OF THE TOXICITY OF NEOARSPHENAMINE TO DIFFERENT STRAINS OF RATS.*

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That individual variation with a group of test animals plays an important rôle in the determination of the toxicity of neoarsphenamine has long been recognized. Voegtlin, in his many papers on the arsphenamines, repeatedly states that "due to the great individual difference in the susceptibility of animals it is difficult to obtain a sharply defined M. L. D." Hooper, Kolls and Wright (1) in their paper on the influence of diet on the toxicity of arsphenamines direct attention to the considerable variation in individual animals to arsphenamine poisoning. Heyle, Hart and Payne (2) have shown that the M. L. D. of a single lot of neoarsphenamine varied from 320 mg. per Kg. to 440 mg. per Kg. when determined on different groups of animals from their colony. Recently Morrell and Chapman (3) in their work on the determination of the characteristic toxicity curve of neoarsphenamine for rats have found that the L. D. 50 of a single lot of neoarsphenamine when tested at intervals over a period of eight months on rats from a carefully controlled colony varied from 380 mg. per Kg. to 500 mg. per Kg.

Except for a note in a report by Hart and Payne (4), in which they mention a difference of more than 100 mg. in the M. L. D. of a single lot of neoarsphenamine as determined in two different laboratories, there appears no direct evidence of a consistent difference in the tolerance to neoarsphenamine of rats from different colonies. Inasmuch as the official method for the determination of the toxicity of neoarsphenamine as recommended by the National Institute of Health, U. S. Public Health Service (5) calls for the use of healthy albino rats without specification as to strain, it occurred to us that an investigation of albino rats from different sources might prove of interest.

Young male albino rats from three different breeding colonies, hereafter referred to as colony I, colony II and colony III, were obtained for this investigation. We elected to use only male rats to eliminate any variation due to sex. Upon arrival in the laboratory the young animals were placed on an adequate diet similar to that recommended in the official method and were maintained on this diet for one week to minimize the possible influence of sudden change of diet on the resistance of the rat. Only obviously healthy animals weighing from 90 Gm. to 110 Gm. were used in the test.

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