

methods,¹ and it was found that the corresponding intracellular acidities, in the tissues of 3 species of sponges, one echinoderm, and a nudibranch mollusk, apparently must lie within the limits 5.6–8.0 (P_H); the actual range is probably narrower than this. The intracellular reactions in the different tissues are probably not all identical. In the case of the nudibranch *Chromodoris*,¹ containing integumentary pigment particularly sensitive over the physiological range, the intracellular acidity (of the fluids associated with the pigment) appears to vary from <6.0 to >7.0 (P_H). In every instance, however, the intracellular reaction is more acid than that of the sea-water medium ($P_H = 8.05$ – 8.15), and perhaps more acid than that of the blood. These acidities, however, all lie within the limits of non-significant protein swelling.²

Individuals of the nudibranch *Chromodoris* undergo natural death after having attained a length of about 16 cms.; during this death process the integumentary tissues containing the intracellular indicator become more acid ($P_H < 5.6$), and seem to undergo a certain amount of swelling ("edema"); *these individuals do not recover, but invariably die*. Quantitative estimations of the amount of tissue swelling are difficult, for technical reasons (owing to the presence of macroscopic, fluid-containing, extracellular spaces), but this amount seems to be relatively small.

These observations consequently increase the difficulties in the way of accepting Fischer's conception of water metabolism, since they indicate a range of intracellular acidities, in animal tissues, within which it is known that no significant protein swelling occurs, and since they show that an intracellular acidity even remotely approaching that at which significant swelling might be possible is irreversibly associated with natural death. The importance of these observations lies in the fact that direct investigation of intracellular acidities is least liable to error when based upon the behavior of appropriate pigments of natural occurrence.

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CORRECTIONS.

On page 1259 of the August number the reference to Abegg's *Anorganische Chem.* in the fourth line from the bottom of the page should be omitted.

On the Quinone-Phenolate Theory of Indicators.—On account of failure to read page proof for the article in the July number of THIS JOURNAL for this year, we have published a number of proof-errors which are obvious to anyone following the work closely, but which should be corrected as a matter of record. In discussing the application of the idea advanced in 1907 that a dibasic acid has *two* primary

¹ Crozier, *Loc. cit.*

² *J. Pharmacol.*, 5, 449 (1914).

and *two* secondary ionization constants (see application of this idea to urazoles and indicators, *Am. Chem. J.*, **38**, 45-6 (1907); **39**, 541-3 (1908), and later papers) expressed by the equations (see footnote 5, page 1092)

$$\begin{aligned} (\text{HAn} + \text{HAn}') \times \text{H}/\text{H}_2\text{An} \times \text{H} \times \text{An}''/(\text{HAn} + \text{HAn}') &= \\ (\text{K}_1 + \text{K}_1') \times \text{K}_2\text{K}_2'(\text{K}_2 + \text{K}_2') = \text{H}^2 \times \text{An}''/\text{H}_2\text{An} &= \\ \text{K}_2\text{K}_2'(\text{K}_1 + \text{K}_1')/(\text{K}_2 + \text{K}_2') = \text{K}_1\text{K}_1'(\text{K}_1 + \text{K}_1')/(\text{K}_2 + \text{K}_2') & \end{aligned}$$

the K_1H in the last member of the equation on page 1093 and the K_1 in the latter part of the footnote should obviously be replaced by $\text{K}_1'\text{H}$ and K_1' , respectively. The "—4" in the graphic formula in the first line on page 1094 should be omitted. The formula in line 20, page 1094, should clearly be $-\text{C}:(\text{C}_6\text{H}_4:\text{O}) (\text{C}_6\text{H}_4\text{OH})$. The typographical errors are obvious.

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NEW BOOKS.

Organic Compounds of Arsenic and Antimony. By GILBERT T. MORGAN. Longmans, Green and Company, London. 375 pages.

During the last ten years, the remarkable achievements in chemotherapy have drawn attention to the importance of the arsenical drugs. The gratifying results which have attended the clinical use of several such substances have only been possible because of an enormous amount of pioneer research. As a result of the chemical investigations, we now have a surprisingly complete knowledge of the behavior of the organic compounds of arsenic. The appearance of an English book on this subject is most welcome, particularly at this time, when the manufacture of certain drugs has become a matter of national importance.

A discussion of the aromatic arsenic compounds occupies two-thirds of the book. This allotment of space is entirely in proportion to the importance of these substances. The aliphatic arsenicals are considered in two chapters, and the aromatic antimony compounds in one. The general methods of preparing each class of compounds are presented in considerable detail, and in many cases specific laboratory directions are also given. A chapter is devoted to each of the therapeutically important substances—atoxyl, salvarsan, neosalvarsan and the metallic coordination compounds of salvarsan. A fuller discussion of the physiological action and clinical value of the various drugs would have added to the general interest of the book. However, the brief consideration given to this phase of the subject is an excellent résumé and includes the most recent advances. From a chemical point of view the monograph will be of the greatest value as a reference book. The chemical and more important physical properties of practically all the organic com-