7·34, 7·89, 7·89, 8·04 and 8·23. The degree of hydrolysis of the sodium salts and the acid-salt ratios at the *p*H of blood were calculated. These values are such that there is no apparent correlation between them and the rate of onset of anæsthesia. L. E. GILSON (Chem. Abstr.).

The Local Action of Eserine on the Central Nervous System. (*Journ. Physiol., vol. xci, pp.* 212–21, 1937.) *Miller, F. R.*

Experiments with cats indicate that the eserine response is evoked locally from the cerebral cortex, and is not a consequence of absorption followed by spinal or peripheral action. E. D. WALTER (Chem. Abstr.).

The Theory of Narcosis. (*Trans. Faraday Soc., vol. xxxiii, pp.* 1062–8, 1937.) *Meyer, Kurt H.*

The threshold concentration of the "indifferent" narcotics (CH_4, $CHCl_3$, CCl_4, ether, alcohols, etc.) in the surrounding medium (water or air) was determined for tadpoles and mice. Determinations were made of the corresponding distribution coefficients between the external media and albumin, olive oil and oleic alcohol (the latter was chosen as a model for the cholesterol type of compounds). No correspondence was observed between the effectiveness of a compound as a narcotic and its surface activity. On the other hand, the distribution coefficient in oleic alcohol was nearly constant, suggesting that such a distribution occurs also in the sterols of the organism. Narcosis commences when any chemically indifferent substance has attained a certain molar concentration in the cell sterols, this concentration depending on the nature of the animal or cell, but being independent of the nature of the narcotic. S. A. CORSON (Chem. Abstr.).

The Action of Narcotics on Enzymes and Cells. (*Trans. Faraday Soc., vol. xxxiii, pp.* 1057–61, 1937.) *Clark, A. J.*

There are two important rival theories which attempt to explain the action of aliphatic narcotics on living cells: (*a*) the properties of the cell surface are determined by its lipide content, and narcotics change these properties by dissolving in this surface; (*b*) narcotics are adsorbed on the cell surface, thus covering it with an inert layer. The adsorption hypothesis is supported by the following data: (1) Resemblance between the action of narcotics on living cells and on enzymes. The concentration-action curves obtained were of an adsorption, linear or intermediate type. The linear curve is usually adduced as proof for theory (*a*). But it was shown that the action of narcotics in lowering the air-water surface tension follows, over the range of concentrations of physiological interest, a nearly linear relation exactly similar to that obtained with living cells. It is suggested that if more than half the total adsorption possible is required in order to produce full inhibition of an action, then an exponential concentration-action curve is obtained. If inhibition is produced when the surfaces are less than half saturated, then the curve approximates a linear form, although it is really a portion of an exponential curve. (2) Pharmacological activity of narcotics increases with the increase in the length of the C chain, thus showing a marked parallelism between equinarcotic and isocapillary concentrations. This rule, though, often breaks down when different series of compounds are compared. However, the same divergence is obtained in the action of narcotics on enzymes. (3) Warburg showed that narcotics interfered in a similar manner with the action of cyanides on cells and on the inorganic catalyst, blood charcoal. (4) Schurmeyer showed that alcohol inhibited the action of purified invertase only after globulin was added, suggesting that inhibition depends on the active group of the enzyme being fixed to particles of colloidal dimensions. Quantitative data support the adsorption hypothesis but do not entirely exclude theory (*a*). S. A. CORSON (Chem. Abstr.).