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Editorial Views

Speculation

UNGAR AND KEATS call our attention to anesthetic action at the cell surface in their systematic survey of the action of diverse agents on the fascinating phenomenon of homotypic aggregation. It is refreshing, in these days of biomedical pragmatism, to see speculation labelled as such and free of the final, "further studies of this phenomenon may reveal the long-sought mode of action of anesthetics." The authors make no claim to solution of this mystery, merely reporting some interesting experiments and advancing a few ideas about their possible significance.

Research into the mechanism of anesthesia began, following Claude Bernard's hypothesis of colloid aggregation, with theories of an intracellular locus of action. Hans Winterstein, L. V. Heilbrunn, Kathleen Dougherty, and many others wrote extensively of experiments favoring such a site of anesthetic action. Forty years ago Heilbrunn believed that calcium translocation within the cell resulted when diethyl ether was applied and that this caused contraction of protein structures in the cellular interior. Since his ideas were derived from studies of amebae and sea urchin eggs and published in the literature of basic biology, they did not receive much medical attention. Quite recently, the sustained muscular contraction of malignant hyperthermia was found to result from an anesthetic-induced defect in calcium uptake by sarcoplasmic reticulum of muscle cells, a finding which has been eagerly accepted owing to its relevance to a pressing clinical problem. We have long known that a rare person died quickly and with high fever when given anesthesia, but few really tried to find out why. "He took anesthesia badly" was the explanation usually offered. Could the biologists, working diligently on basic actions of anesthetics, have helped clear the mystery surrounding these cases? We'll never know.

The Ungar–Keats paper deals with anesthetic action on surface properties of single cells in suspension. This is not a new approach, even though the phenomenon they describe has not been studied previously. Fifty years ago, Traube suggested that cell surface stabilization followed anesthetic administration. This hypothesis took on more meaning when the Hodgkin–Huxley theory of action potential generation at excitable membranes achieved a degree of acceptance which led to a Nobel Prize. If this is where the action is, electrochemically speaking, then anesthetics must act here, many reasoned. Today there is a common belief that anesthetics must somehow alter cell surfaces, or cell membranes, or something of the sort, but no one really knows how.

We are allowed, from time to time, to share the speculation of creative investigators who offer only their experimental results and the notions they have about the significance of their work. Too often, editors do not allow authors to digress from their data during the discussion of their experiments. This problem is eliminated when the editor is the author, some might say. To the contrary, publication of this paper reflects a more open editorial policy than allowed in some quarters. Those who object to the appearance of this sort of article in our Journal should consider carefully the consequences of relegating such studies to relatively inaccessible biology journals. Science has grown through speculation. If anesthesiology is based in science, it must allow itself to speculate once in a while.

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