dition during operation is one of the duties of the anaesthetist. Intravenous fluids, newer analeptics, new methods of blood pressure determination and better records all aid in this objective. The ability of the anaesthetist is more important than new agents and techniques.

F. A. M.


Many of the forerunners of the discovery of anaesthesia were British. October 16, 1946 is the centenary of the advent of surgical anaesthesia as a practical measure. The word anaesthesia was first used by Bailey in 1721. In 1829 it was used by Reid as synonymous with “loss of sensation.” The New English Dictionary (Oxford) gives the earliest use of the word “anaesthetic” as by J. Y. Simpson in 1847. Oliver Wendell Holmes wrote to Morton to suggest that the state should be called “anaesthesia,” from which the adjective would be “anaesthetic.” Knowledge of prehistoric attempts to produce anaesthesia is speculative. Early civilizations have left some evidence that methods for producing insensitivity to pain were being sought. Early pioneers of inhalation anaesthesia include Humphry Davy who suggested that nitrous oxide might “probably be used with advantage during surgical operations in which no great effusion of blood takes place.” His suggestion was not followed up. Henry Hill Hickman suggested the use of “suspendedanimation” in surgical operations. He experimented on animals, after inducing a “torpid state,” by allowing them to rebreathe their own exhaled air or by passing carbon dioxide into the bell-jars from which air was excluded. In the United States

W. E. Clarke, Crawford W. Long, Horace Wells, W. T. G. Morton, and C. T. Jackson all contributed to the early use of anaesthetics for surgical operations. In England Robert Liston, John Snow, Joseph Clover, and James Young Simpson were pioneers in the development of anaesthesia. An exhibition at the Wellcome Historical Medical Museum, illustrating the whole history of anaesthesia, was opened on October 16, 1946. 33 references.

F. A. M.


The purpose of the investigation was to determine the changes in the water and chloride content of the skin and musculature during a period of chronic dehydration and recovery, and to compare these changes with those occurring in these organs with acute dehydration as a result of hemorrhage. Thirteen dogs were used in this study.

Dehydration by withholding food and water was followed by a greater loss of water from the skin than from the muscles of the body. Acute dehydration resulted in a similar but smaller loss of water from the skin. The chloride content of the skin increased from an average of 297 mg. sodium chloride per 100 cc. water to 440 mg. after chronic dehydration. The chloride content of the muscle deviated much less. In acute dehydration a slight increase in the chloride content of the skin occurred, while muscle tissue showed a decrease in chlorides. Acute hemorrhage in two dogs at the height of chronic dehydration produced slight deviation of the water content of the skin and muscle and a decreased chloride content of the muscle.

The results indicate that during chronic dehydration muscle tissue
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shifts isotonic extracellular fluid to the circulatory system, whereas the skin loses chloride-free water and actually increases its chloride content. After hemorrhage, however, the isotonicity of the blood is maintained by obtaining water chiefly from the skin and chlorides from the musculature. The experiments involving acute dehydration at the height of chronic dehydration suggest that the conservation of fluid had reached a degree where tissues did not yield fluid even under stress of rapid hemorrhage.

M. F. P.


The striking and dramatic relief of painful muscle spasm and arthritic pain through anesthetization of the sphenopalatine ganglion by topical treatment presented a challenge for its interpretation. The problem was approached on the thesis that the underlying biochemistry of contractile elements which would characterize all muscle spasm would tend to be the same.

The chemistry of muscle contraction underlies the basic physiology of nutrition. The keystone of muscle metabolism is adenylic nucleotide. It phosphorylates thiamin to ecarboxylase, thus making the biologically active coenzyme. Similarly, it phosphorylates riboflavin and combined with nicotinamide, it goes to form Coenzyme I and II. These are the factors that control cell respiration, for the coenzyme together with the amino acids of the protein portion form the respiratory enzymes (the enzymatic means whereby carbohydrate is gradually broken down by the stepwise removal of hydrogen and the liberation of energy). It is the deficiencies of the elements of the respiratory enzymes that produce the classical picture of vitamin deficiency.

Muscle metabolism requires, in addition to this energy releasing enzymatic setup, a substance that is uniquely capable of changing its molecular structure so as to alternately contract and return to its original form and utilizing for this purpose the energy released by tissue respiration. Such a substance is myosin, composed of a protein that is also bound to adenylic acid to form the enzyme adenosintriphosphatase.

The basic idea that develops from the chemical study of muscle contraction is the uniformity of the reaction of all types of muscle. The clinical implication of this conception is the intimate relation between the spasm of large voluntary muscles and those of the heart and blood vessels, and the possible unity of the etiologic factor.

Confirmation lies in the success of the therapeutic use of adenylic nucleotide as the iron salt. The factors that tend to throw the balance of the chemical reaction in muscle metabolism toward the maintenance of the contracted state with incomplete recovery are the keys to the therapy of muscle spasm.

Our next consideration is the reflex neurogenic factor in muscle spasm. The idea of interrupting reflex autonomic factors by surgical attack on the adrenal sympathetic system demonstrated the ability to control muscular vasospasm in a lasting manner. Similar results may be obtained through blocking at the sympathetic sphenopalatine ganglion. The neurological connections of the nasal ganglion are pointed out.

We still have to consider what constitutes muscle spasm. A muscle