tion during operation is one of the duties of the anaesthetist. Intravenous fluids, newer analectics, 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 insensibility 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 "suspended animation" 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 29 g. 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
shifts isotonic extracellular fluid to
the circulatory system, whereas the
skin loses chloride-free water and ac-
tually increases its chloride content.
After hemorrhage, however, the iso-
tonicity of the blood is maintained by
obtaining water chiefly from the skin
and chlorides from the musculature.
The experiments involving acute de-
hydration at the height of chronic de-
hydration suggest that the conserva-
tion of fluid had reached a degree
where tissues did not yield fluid even
under stress of rapid hemorrhage.

M. F. P.

RUSKIN, SIMON: The Control of Muscle
Spasm and Arthritic Pain through
Sympathetic Block at the Nasal
Ganglion and the Use of Adenylic
Nucleotide; Contributions to the
Physiology of Muscle Metabolism.
Part II. J. Digest. Dis. 13: 311,
1946.

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 nu-
trition. The keystone of muscle me-
tabolism is adenylic nucleotide. It
phosphorylates thiamin to cocarbox-
ylase, thus making the biologically
active coenzyme. Similarly, it phos-
phorylates riboflavin and combined
with nicotinamide, it goes to form
Coenzyme I and II. These are the fac-
tors 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 gradu-
ally broken down by the stepwise re-
moval of hydrogen and the liberation
of energy). It is the deficiencies of
the elements of the respiratory en-
zymes that produce the classical pic-
ture of vitamin deficiency.

Muscle metabolism requires, in addi-
tion 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 adenosintri-
phosphatase.

The basic idea that develops from
the chemical study of muscle contra-
sion 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 nu-
cleotide as the iron salt. The factors
that tend to throw the balance of the
chemical reaction in muscle metabo-
lism toward the maintenance of the
contracted state with incomplete re-
covery 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 au-
tonomic factors by surgical attack on
the adrenal sympathetic system demon-
strated the ability to control muscular
vasospasm in a lasting manner.
Similar results may be obtained
through blocking at the sympathetic
sphenopalatine ganglion. The neuro-
logical connections of the nasal gang-
lion are pointed out.

We still have to consider what con-
stitutes muscle spasm. A muscle