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A NEW THEORY OF SHOCK

Notwithstanding the rapid advance made in some of the fundamental problems confronting the surgeon, such as infection and hemorrhage, shock has remained more or less of an enigma. Indeed, most of the large amount of investigative work which has been done in connection with this subject has served only to enrich our knowledge of the phenomena occurring in association with it; so that although we have at present a better understanding of its symptomatology, yet almost nothing has been done which convinces us of its fundamental nature. Thus Crile[1] and Romberg and Pässler[2] have independently observed that during shock the arterial pressure falls; and from this observation they concluded that this drop of pressure was the "essential phenomenon," and that shock was fundamentally due to a vasomotor failure.

Hardly had this conclusion been reached, however, when Seelig and Lyon and others disproved it by demonstrating conclusively that instead of a failure of the vasomotor system there is actually a great activity, at least up to the last stages of shock. So no sooner has one theory been advanced to explain it than further work has served to disprove or at least to minimize the importance of that theory.

During the past two years, however, a series of articles has appeared by Yandell Henderson, of New Haven,[3] dealing with an elaborate experimental investigation of shock from an entirely new basis. Briefly, Henderson believes that the condition primarily is due to a diminished amount of carbon dioxid in the blood and tissues of the body. This lack of the normal amount of carbon dioxid is termed acapnia. His experimental results are most interesting; and they are full of much practical suggestion to the surgeon because they seem to show that some of the technic, commonly practiced, is based on erroneous ideas. In a short summary of this kind necessarily only a few of the most interesting features of the work can be cited.

Henderson reviews the literature of carbon dioxid as a hormone, or chemical regulator, of respiration to show that the failure of circulation and of the nervous system in shock and the cessation of respiration in true apnea must logically be referred to the same cause—acapnia.

Experiments on dogs, which were devised to bring about a sudden great diminution in the carbon dioxid content of arterial blood, resulted in an increase of the heart-rate up to the point of cardiac tetanus and death. Voluntarily forced breathing in man induces symptoms resembling shock, probably because the increased pulmonary ventilation results in an abnormally great exhalation of carbon dioxid. Pain, ether, excitement, sorrow, fear and other conditions inducing shock involve excessive respiration. Because a marked diminution in the amount of carbon dioxid in the arterial blood creates a lack of the normal stimulation of respiration, excessive artificial respiration in dogs for twenty-five to thirty minutes, by causing greatly increased pulmonary ventilation, is followed by a cessation of breathing so prolonged that the heart fails for lack of oxygen. During this anoxemia an acidosis occurs owing to an accumulation in the blood of products of incomplete tissue combustion. The administration of carbon dioxid gas during the period of cessation of respiration induces an immediate return of natural breathing; and the administration of oxygen affords ideal conditions for the prevention of the asphyxial acidosis. Anesthetics tend to prevent shock because they diminish the excessive respiration caused by pain. The respiratory excitement during the initial stages of anesthesia diminishes the carbon dioxid content of the blood, and thus tends to induce a subsequent failure of respiration. Similarly, ether, unless neutralized by morphin, often causes (in dogs) hyperpnea, acapnia and a consequent fatal apnea. Of particular interest in this connection are observations by W. D. Gatch[4] in several hundred anesthesias during which patients were allowed to rebreathe some of their own carbon dioxid. In no instance was there any evidence of shock. These observations seem to confirm Henderson's theory to a greater or less degree; and they lend evidence to his statement that "skilful anesthesia consists in maintaining the threshold of the respiratory center for carbon dioxid at a nearly normal level, and in avoiding the development of either acapnia or hypercapnia."

The section of the work dealing more particularly with the relation of shock to abdominal operations is of special importance because of its bearing on many cases of hitherto inexplicable postoperative ileus. It is shown that the loss of tonus of the abdominal viscera during and after laparotomy is almost certainly due to the direct exhalation of carbon dioxid from the organs during the time of exposure. Indeed, Henderson states that the common practice among surgeons of wrapping viscera in moist hot compresses repeatedly changed is an effective means of producing shock instead of pre-

1. Surgical Shock, 1899.
2. Deutsch. Arch. f. klin. Med., 1899, lxiv, 652.
3. Am. Jour. Phys.

4. THE JOURNAL, March 5, 1910, p. 775.