THE RELATION OF LIGHT OR DEEP ANESTHESIA TO SHOCK*

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In their efforts to prolong the life expectancy of man, surgeons in the last few years have become bolder and more expressive with the scalpel. Their horizons have broadened to include the newborn and the tottering among their candidates for corrective surgery. Such attitudes are most commendable, but they have served to intensify the problems of the anesthetist in his care of the patient.

Not the least of the hazards which the anesthetist must contemplate is the problem of shock. The etiology of the syndrome of shock is related fundamentally to the cardiovascular system. To paraphrase an over-simplification, the condition develops when "there just ain't enough blood going around." The volume of circulating blood will diminish with hemorrhage; it will become less if the capacity of the blood vascular bed enlarges, and it will be reduced if the heart itself loses its efficiency.

Looking to his own bailiwick, the anesthetist wonders to what extent the potentially toxic agents he employs may contribute to the establishment of shock. Does the concentration of agent he introduces into the blood stream, or the depth of narcosis he maintains, act in a salutary or contributory fashion toward the pathogenesis of shock?

The introduction of curare by Griffith and Johnson (1) has necessitated a reappraisal of this question. Prior to 1942 the anesthetic agents were used to produce hypnosis, analgesia and relaxation to a greater or lesser degree in the patient. This latter requisite could be obtained only by subjecting the patient to deeper planes of anesthesia. Today our narcotic agents need to supply only hypnosis and analgesia, and these may be provided apparently in light planes of anesthesia. Is this trend a boon to the patient, or are we failing to protect him sufficiently from the noxious stimuli of the surgeon?

It has been recognized for a number of years (2) that the experimental animal, when awake, will tolerate about twice the amount of hemorrhage that he will when anesthetized. Clinical experience corroborates that shock will often be precipitated in the borderline patient by administration of general anesthesia. What specific effects do an-

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esthetic agents exert to produce these undesirable sequelae? Zweifach
and his associates (3) have visualized directly the peripheral vascular
network and indicated that in dogs subjected to hemorrhage under
anesthesia there is only a partial and inadequate vasoconstrictive
response to the demand for a smaller blood vascular bed. Beecher
and his group (4), experimenting with dogs, noted similar reactions
under anesthesia and, more important, indicated that the changes be-
came more profound as the depth of anesthesia increased.

Not all anesthetic agents produce this loss in vasomotor tone to the
same degree. There is general agreement among investigators that
ethyl ether is the worst offender, animals under its influence showing
the least response to resuscitative measures. Animals bled under barb-
biturate anesthesia show greater reflex vasomotor tone, although a
point is reached when deterioration appears rapidly. Cyclopropane
produces the least change in the functional activity of the peripheral
vascular bed (5). Reactions under it are similar to those which occur
in unanesthetized animals.

When consideration is given to the heart itself, recent work by
Fisher et al. (6) indicates that anesthetic agents in the heart-lung prep-
paration of the dog impair the efficiency of the myocardium. Again,
ether is the worst offender and cardiac function decreases hand in
hand with depth of anesthesia. In earlier work Cattell (7) found that
ether decreased the functional capacity of the heart and hence its
output. Investigators at Harvard (4) found that ether, barbiturates
and cyclopropane all produced a rise in central venous pressure. It is
reasonable to deduce that this increase in venous pressure may be an
indication of cardiac incompetence (8).

With regard to circulating blood volume, it is known that ether
causes a hemoconcentration. Two independent investigators (9) (10)
concluded that the reason shock was delayed in animals under amytal
anesthesia, as opposed to ether anesthesia, was that hemoconcentration
appeared much later in the former group.

These observations tend to indicate that some general anesthetic
agents exert an untoward influence on the peripheral vascular bed, on
the heart and on the circulating blood volume, all of which may be
implicated in the pathogenesis of shock. Moreover, these effects be-
come more profound and deleterious as the depth of narcosis increases.

Corroboration of these findings is not lacking in our clinical impres-
sions. The use of practically all general anesthetic agents, and ether
perhaps most convincingly, results in dilatation of the peripheral veins.
The reasons for this are obscure, but conceivably are related to an in-
crease in venous pressure or to a disturbance of the function of the
peripheral vascular bed. It is certain that ether is almost as effective
in producing vasodilatation as sympathectomy (11). Anesthetists
hesitate to utilize large doses of pentothal over long periods of time, be-
cause of the common occurrence of tachycardia with narrowing pulse
pressure after a two hour period. Such signs are believed to be a manifestation of cardiac inefficiency (12). We are wary of employing ether or pentothal in patients with impending shock. This evidence strengthens the belief that these patients "do better" under cyclopropane anesthesia. The agents that are employed are used in small amounts, so that the planes of anesthesia may be kept light, thus avoiding further predisposition to shock.

On the other side, the question arises as to whether the present tendency to maintain the patient in light planes of anesthesia is predisposing him to a greater likelihood of shock. Are surgical stimuli more likely to exert a deleterious effect under light anesthesia than under deeper planes? There is little objective evidence to assist us with this problem. In his theory of anoci-association, Crile (13) in 1914 stated that shock is the result of the excessive conversion of potential into kinetic energy in response to adequate stimuli. Such stimuli, he thought, could originate from the field of operation, and could be intercepted only by nerve block with local anesthetic agents between the site of operation and the sensory receptors in the central nervous system. There are those who subscribe to this theory today, and who believe that surgical trauma, if severe and prolonged enough, will produce the syndrome of shock unless adequate nerve block is instituted by administration of local anesthetics or deep general anesthesia. From time to time articles appear in the literature to support such thought (14). Conversely, much clinical evidence has accumulated since World War II to indicate that patients withstand prolonged operative procedures well under light planes of anesthesia associated with muscle relaxants. The problem is far from settled, but clinical impressions indicate that painful surgical stimuli in light planes of anesthesia do not necessarily contribute to the syndrome of shock.

There is little doubt that autonomic reflex responses are more active and effective under light planes of anesthesia than in deeper planes. These reflex responses may work to the patient's detriment, and precipitate a so-called neurogenic shock. The carotid sinus reflex, vagal reflexes from the mediastinum, the celiac reflex and the pelvic reflex are examples. Fortunately, there are pharmacologic antidotes for such reactions, and it is probable that d-tubocurarine chloride (15), atropine, or some of the vasopressor drugs may protect against or reverse the situation without any generalized adverse effects. Of course, such light planes of anesthesia permit activity of reflexes which are beneficial to the patient and which aid in maintaining his physiologic equilibrium. In particular, the integrity of the cardiovascular system is preserved, so that adaptations can be made when anoxia, hypercapnia or blood loss supervenes. Such reflex mechanisms are depressed when deep planes of anesthesia are present (8).

With the introduction of muscle relaxant drugs, the need for deeper planes of anesthesia to produce relaxation no longer existed. The
commonest agent used to produce relaxation was ether, and in the concentrations required for this purpose, the normal physiologic protections against the development of shock were paralyzed. For the same reasons large doses of barbiturates were contraindicated, and it has perhaps rightly been recommended that they be employed alone only for short procedures (16). Of the several drugs which could be used before curare was introduced, cyclopropane appeared the safest, so far as protection against shock was concerned, but in deep planes, its potential devastating effect on the conducting mechanism of the heart limited its employment by many anesthetists to circumstances in which low concentrations were adequate. From such considerations has emerged the concept that the anesthetist will serve the patient best in his efforts to overcome the strains and stresses incidental to surgical interference by not overloading the patient with any one potentially toxic anesthetic agent. To stretch the theory of Selye (17), the anesthetist can aid the general-adaptation-syndrome response of the patient by allowing him as full use as possible of his normal physiologic protective mechanisms.

The physiology of unconsciousness and narcosis is a secret which has as yet not been revealed to us. However, it would appear that what we are creating with anesthesia is pathologic physiology, which must upset to some extent the normal functions of the patient. If the individual is considered as a closely integrated, ever-changing collection of interdependent cells, it would seem that the less profound the narcosis, the fewer adaptations he would have to make on its account. With this idea in mind, it would appear that the present trend of employing small amounts of several agents, each to achieve its own purpose, be it hypnosis, analgesia or relaxation, and each interfering with normal physiologic processes to a minor degree, would tend to allow the patient to utilize his defenses to the greatest advantage against the surgical insults to which he is exposed.

REFERENCES


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Wednesday, November 12, 1952

MORNING: FIRST SESSION, HOUSE OF DELEGATES
REFRESHER COURSES
ROUND TABLE LUNCHEON

AFTERNOON: REFRESHER COURSES

Thursday, November 13, 1952

MORNING: GENERAL SCIENTIFIC SESSION:
Problems in Ventilation:
Moderator: Stuart C. Cullen, M.D.
Panel Participants: Julius H. Conroe, Jr., M.D.
E. B. Brown, Jr., Ph.D.
Henry K. Beecher, M.D.
James V. Maloney, Jr., M.D.

AFTERNOON: SECOND SESSION, HOUSE OF DELEGATES
REFRESHER COURSES

EVENING: COCKTAIL PARTY AND DINNER DANCE

Friday, November 14, 1952

MORNING: REFRESHER COURSES
Panel Luncheon
AFTERNOON: REFRESHER COURSES