

Problems of Anesthesia  
3<sup>rd</sup> Hartmann Memorial Lecture

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It is a great pleasure and a real privilege for me to have the honor of delivering the 3<sup>rd</sup> Hartmann Memorial Lecture. The honor you confer on me is multiple in nature. First is the opportunity of bringing to you some of the problems of anesthesia and a few of their solutions; second is the gratification of being invited by my good friends Professor Nissen and Dr. Hugin; and third is the fact that I am permitted to follow two of the outstanding and gifted leaders in the world of modern anesthesia, Sir Robert R. MacIntosh and Professor Henry K. Beecher.

Professor MacIntosh told you, with his customary wit and brilliance, of Teaching and Learning in Anesthesia and of the importance of a long period of effective training required for specialization in Anesthesia. Professor Beecher discussed with his usual vitality his important studies on the problems of pain. The first cycle of the Hartmann Memorial Lectures should logically be completed with a discussion of the application of scientific knowledge to the art of anesthetic practice. It will be my task to attempt the achievement of this purpose in the humble realization that my talents are limited and the objective a vital one for the public welfare.

It is necessary to remind you, for a moment, that the Hartmann Foundation in Anesthesia is an extraordinary tribute to the impetus and energy for progress of the Department of Anesthesia of the University of Basle. There are few endowed lectureships in anesthesia and relatively few permanent chairs of anesthesia in Europe or

the United States. One should, therefore, pay one's profound respects to the vision of Jenny Hartmann in wishing to bring the practice of anesthesia into mid Twentieth Century level in her country on a par with the practice of surgery or medicine.

It is an extraordinary thing to contemplate the tremendous recent growth of knowledge in anesthesia and its related sciences unaccompanied by the widespread training of skilled physicians able to translate this knowledge (even though far from complete) into the care of the surgical risk in daily practice.

The need for closing this gap between knowledge and clinical practice is a function of education, research, and most important of all – the awareness by surgeons that modern, skillful anesthesia is essential to their daily tasks, and that the future of the development of surgery is in part dependent on the free intellectual growth of anesthesia. Mutual affection and respect between surgery and anesthesia flourish to the greatest degree in the atmosphere of that equality accorded to accomplishment and merit. If these do not exist, anesthesia becomes a monstrous mockery of science and surgery a tyrannical despot whose brilliant accomplishments eventually are limited by the hardening of mind and spirit that seem to be shared equally by oppressor and oppressed. This presentation will have success if it helps increase mutual confidence between surgeon and anesthetist by outlining in some detail a few of the important problems the anesthetist must face and solve if the patient is to receive safe benefit from surgical treatment.

It will be my purpose to examine several of the important and common circulatory disorders secondary to impaired ventilation which occur during anesthesia and to comment on their practical consequences. No attempt can be made to relate these

directly to mortality from anesthesia since such statistical knowledge is not available. It is hoped, however, that the inferences will be clear and that there will be a relationship observed between the subtleties of derangement and the potential damages which may result in death and morbidity.

#### Problems of Ventilation and the Circulation.

The establishment of the anesthetized state brings about either an actual or a potential change in the pattern of total pulmonary and alveolar ventilation. Depression of ventilation occurs with many of the potent anesthetic and sedative drugs by virtue of their central depression of respiratory exchange. The commonly used narcotics are excellent examples of drugs producing this type of response. The anesthetics thiopental sodium and cyclopropane also produce actual depression of ventilation. Other anesthetic agents can cause a depression of breathing as a consequence of developing adequate anesthetic depth for a given surgical procedure. Ether provides a good example of this action. The muscle relaxants which are employed to provide effective muscular paralysis in the surgical field can produce depression of breathing by causing an associated paralysis of the muscles of respiration. Depression of ventilation can also occur as a direct consequence of the surgical procedure itself. For example, the opening of the chest or the employment of a posture not ideally suited to satisfactory respiratory exchange may also lead to hypoventilation.

Hypoventilation causes respiratory acidosis as well as anoxia. Since the circulatory effects of retention of carbon dioxide are not so well known as those of anoxia, a detailed description of these changes should be useful. Increases of carbon dioxide in the blood can produce local vasodilatation presumably by direct action upon

local vessels. This is certainly true in the brain. (1) On the other hand, carbon dioxide retention can effect a reflex peripheral vasoconstriction because of stimulation of the carotid and aortic chemoreceptors. (2) There are other circulatory consequences which occur during the acidotic state. A state of acidosis can produce electrocardiographic changes particularly in the T wave and S-T segments. (3) Hypercapnea also appears to potentiate certain vagal reflexes which may actually result in cessation of heart action. (4) Traction on the stomach, the suction of the tracheo-bronchial tree, and manipulations about the hilum of the lung in the presence of hypercapnea may produce either severe bradycardia or possibly cardiac standstill. These reflex changes are also associated with hypotension which may be dangerous because of the subsequent diminished perfusion of vital organs.

As though this were not problem enough, the period of recovery from carbon dioxide retention is also fraught with danger and difficulty. It has now been well established that the sudden withdrawal of carbon dioxide from an acidotic patient can, in the instance of cyclopropane, lead to a severe and profound hypotension. This syndrome is known as "cyclopropane shock". (5) Even more drastic than this circulatory derangement following the rapid correction of hypercapnea has been the development of ventricular tachycardia and fatal ventricular fibrillation. It is necessary to reduce the carbon dioxide tension slowly over a period of 15 to 30 minutes to avoid death in the dog. (6)

It does not seem necessary to belabor further the point that retention of carbon dioxide during any form of general anesthesia is an ever present hazard, that it is extraordinarily difficult to detect by ordinary clinical means, and that its repercussions

upon the circulation in the form of irregularities of cardiac rate and rhythm, hypotension, and sudden death from cardiac standstill are potential threats. Logically one could surmount this type of difficulty by providing for efficient and adequate ventilation at all times during anesthesia. This is ideal but, like other ideals, it is not always a practicable reality. Furthermore, the production of vigorous hyperventilation, which is sometimes necessary to maintain normal blood carbon dioxide tensions, may produce an effect upon the circulation which is also deleterious. More work must be done to define the precise nature of these changes. For the present at least, it is sufficient to state that the imposition of positive pressures upon the airway of a patient whose circulation or respiration is already compromised from other causes may prepare the way to circulatory failure. It is difficult to escape the judgment that the effects of hypoventilation are so frequent, so difficult to diagnose, and sometimes so difficult to treat that the establishment of a state of general anesthesia should be one that is undertaken with due and mature consideration for the maintenance of effective ventilation to offset the development of serious circulatory effects.

#### Manipulation within the airway.

It is necessary to work with the pharynx, the larynx, and the trachea of the patients subjected to general anesthesia. This purpose is one that is commonly exercised and is one that is necessary to assist in the establishment of a free airway. It does no good to move gases in the proper volume and under the proper pressure through an airway that is not open. Early studies in this aspect of anesthetic manipulation suggested that certain circulatory disturbances could originate from mechanical stimulation within the air passages. These reflexes were termed vago-vagal since the afferent and efferent

paths of the reflex were assumed to be the vagus nerve. (7) Subsequent electrocardiographic studies by Burnstein et al. found that the common cardiac changes following such stimulation as endotracheal intubation consisted of increases in rate and variety of arrhythmias; the most frequent being sinus tachycardia. It was concluded from these observations that circulatory changes incident to the manipulation of the airway were very frequent, occurred in the majority of patients, and were probably associated with increases in cardio-accelerator or sympathetic tone, rather than vagal tone. (8,9) King and his associates demonstrated that laryngoscopy usually produced an increase in blood pressure and less frequently an increase in heart rate in light anesthesia. Intubation of the trachea uniformly produced an increase in blood pressure and an increase in heart rate. As anesthesia was deepened the changes in blood pressure and heart rate associated with laryngoscopy and endotracheal intubation were less important. These circulatory changes did not appear to be related to a specific anesthetic agent but occurred with all agents. Depth of anesthesia minimized the changes observed. (10)

These changes in light anesthesia suggest that the performance of this type of manipulation during light general anesthesia may pose a strain upon the myocardium that will be poorly tolerated by patients with diminished myocardial reserve. On the other hand, a vexing problem is posed since a weakened myocardium may also not tolerate anesthesia of sufficient depth to minimize the reflex responses from endotracheal intubation without producing hypotension. It has also been shown that cough, strain, or reaction upon the tube in light general anesthesia will impose circulatory disorders due to the physical transmission of increased intrathoracic pressure to the heart and the great vessels. (10) However, the production of cough and reaction on the tube during general

anesthesia in patients with severe heart disease may produce a serious circulatory collapse which cannot be relieved with ease or even with certainty. On the other hand, the production of cough during the instillation of topical anesthetic agents into the respiratory tree in these patients, prior to endotracheal intubation, is not associated with the circulatory depression that occurs during general anesthesia. (11) It appears certain, therefore, that airway manipulations may result in changes of cardiac rate and rhythm and in a pressor response in patients with healthy hearts. These developments are more noteworthy in light anesthesia than they are in deep anesthesia and they are not related to the type of anesthetic agent used, but rather to the depth.

#### The Influence of Anesthesia on the Renal Circulation.

It is of interest to examine briefly the behavior of the kidney circulation in the anesthetized state. There are at least three critically important aspects of renal function which have a bearing on the ultimate welfare of the surgical patient. The first of these is the kidney as a circulatory organ; secondly, is the kidney as a regulator of acid base balance; and finally, there is the role of the kidney in the control of water and electrolyte metabolism. Although all these aspects of renal activity are of interest and important to the welfare of the surgical patient, only a few aspects of these derangements of renal activity can be mentioned.

It is not always appreciated that the kidney is a circulatory organ of considerable importance. Approximately 25 per cent of the cardiac output normally goes through the kidneys in each unit of time. During general anesthesia, regardless of the anesthetic agents used, the renal blood flow is reduced significantly. (12, 13) The greatest reductions are associated with the greatest depths of anesthesia. The diminution of blood

flow is apt to be more profound with cyclopropane than it is with the other inhalation agents. This is but one illustration of the tremendous alteration in circulatory dynamics which goes on even with well conducted general anesthesia. It implies an extensive redistribution of blood flow throughout the body. At the present time neither the quantitative nor the qualitative nature of this readjustment of blood flow is well understood. Again, one must point out that this rather tremendous change perfusion of blood through a major organ (the kidney) is not detectable by any of the clinical means available for operating room measurements.

There is only fragmentary evidence at this time as to the possible part that the kidney may play in the regulation of acid base balance during general anesthesia. The information which is at hand suggests that the kidney's ability to compensate for the respiratory changes in acid base balance which were described previously is greatly impaired. The well recognized inability of the kidneys to tolerate abnormal loads of salt in the postoperative period may be related indirectly to the influence of anesthesia. This relationship is, at best, tenuous and may consist in a "pre-conditioning" of renal tubular cellular activity in some manner which interferes with normal function in the postoperative period.

### **SUMMARY**

In this essay an attempt has been made to describe important physiological adjustments of the circulation secondary to disturbances in respiration during the anesthetized state. Comment has included the influence of inadequate ventilation, manipulations within the airway, and changes in renal circulation. Some of these circulatory derangements are obvious and grossly important. Others are more subtle but



may very well be the precursors for some of the circulatory catastrophes which have occurred during anesthesia.

The solution of these problems will not be easy. It depends largely upon the acquisition of more quantitative data on the nature of the changes produced by anesthesia and the application of this knowledge by skillful clinicians to the care of the surgical patient in the operating room.

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