Management of Pulmonary Ventilation under Anesthesia

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Today there is an evidenced increasing interest in the incidence of cardiac arrests due to an interruption of the normal physiology of respiration on the operating table, and in the immediate postoperative period.

How can the anesthetist provide a fast induction of anesthesia, adequate relaxation for the surgeon and still maintain good pulmonary ventilation? All too often it is impossible for an anesthetist to administer what might be termed a “good anesthetic” from a surgical point of view, without either knowingly or unknowingly compromising the respiratory physiology of the patient. The ability of many anesthetists is often judged by the rapidity with which he is able to induce anesthesia, have the patient intubated and ready for the surgeon to make the incision. There is only one way to accomplish this speed and that is by the use of excessive amounts of anesthetic drugs which produce sudden depression of the vital centers in the medulla to a point that normal physiological reflexes are abolished. The most vital function that may be lost, or severely depressed, is that of respiration. Gone are the days when the anesthetist could be guided by observation of the patient’s progress through the stages of anesthesia. In those days cessation of respiration was the danger signal of Stage IV. Now we think nothing of inducing this state of affairs in the first five minutes of an anesthetic. Every drug that we use to induce anesthesia is a respiratory depressant in some degree—many to a severe degree.

Just what is the harm in depressing the respiration? The greatest bugaboo that the anesthetist has to face is the retention of carbon dioxide with associated hypoxia. Since carbon dioxide retention is always the result of respiratory depression it is the anesthetist’s responsibility to make certain that hypoxia is not present. This combination of oxygen lack and carbon dioxide retention is the cause of more cardiac arrests than any other factor associated with anesthesia.

**Prevention of Oxygen Lack**

What means can be used to assure adequate oxygenation, or ventilation? First, the patient can be maintained in a light plane of anesthesia so that there is no impairment of intercostal function; if this is not feasible, assisted or reinforced respirations may be employed to supplement the patient’s voluntary but depressed respiratory efforts.
In the event that this latter method should still be inadequate to maintain the vital signs within normal limits, or, should the surgical procedure contraindicate excessive movement of the diaphragm and mediastinum, respirations must then be controlled. This involves the abolition of the Hering-Breuer reflex and can be carried out by either manual compression of the breathing bag or by the use of a mechanical ventilator. There are two schools of thought regarding controlled respirations: 1. That which contends that it is impossible, even for a skilled anesthetist, to adequately ventilate a patient’s lungs by manual compression of the breathing bag, relying solely on the elastic recoil of the lungs for the elimination of carbon dioxide. Proponents of this contention maintain that sufficient ventilation can only be provided by use of a mechanical device which delivers gases to the patient under positive pressure and removes carbon dioxide by negative pressure. 2. That which holds to the belief that mechanical means are not necessary and that an experienced anesthetist can effectively perform the same function by manual means. At the Cincinnati General Hospital, we have used both methods and have clinical evidence to substantiate the value of each technic.

**Factors Affecting Respiration**

Let us briefly review the basic physiology with which we are concerned. Respiration, which supplies oxygen to the tissues by way of the lungs, and removes an end-product of metabolism, carbon dioxide, through the same route, is controlled by a center in the medulla. The center controls respiration through nerves to the muscles of respiration, e.g., the phrenic nerve to the diaphragm, and those to the intercostal muscles. Many factors influence the center but those with which we are most concerned are the following: (1) oxygen tension of arterial blood, (2) carbon dioxide tension of arterial blood, (3) blood pressure changes, and (4) pH or hydrogen ion concentration of the blood. Anything, such as anesthetic drugs, which depresses the activity of the respiratory center will cause alterations in the normal arterial oxygen and carbon dioxide concentrations. This, in turn, produces changes in blood pressure and pH of the blood. If the oxygen supply to the lungs falls below normal requirements from any cause, and this is allowed to persist over a period of time, a condition of hypoxia will result. Likewise, if normal tidal volume is decreased from any cause over a period of time, carbon dioxide will be retained and respiratory acidosis will ensue from a lowering of the pH of the blood. In the conscious patient a rise in blood carbon dioxide tension will stimulate the respiratory center and produce hyperventilation. By this mechanism the excess carbon dioxide can be eliminated. In the anesthetized patient respiration is no longer under the control of normal mechanisms so that respiratory acidosis is an ever present danger. In lighter planes of anesthesia, hyperpnea and elevation of blood pressure, without tachycardia, will reflect hypercapnia. However, without repeated blood samples it is difficult to assess the degree of acidosis. In the deeper planes of anesthesia the usual responses to increased blood carbon dioxide tension are absent. The stimulation to respiration may be lacking despite a high carbon dioxide tension, and an elevation of blood pressure may not be present. Patients who
have remained in an acidotic state during anesthesia will frequently have postoperative hypotension, rapid, thready pulse, facial pallor, but a warm, dry skin unlike the cold, clammy skin characteristic of surgical shock. They may also recover slowly from anesthesia since carbon dioxide in excess acts as a narcotizing agent. This condition cannot be explained on the basis of blood loss or on other operative factors.

**Tests for Respiratory Acidosis**

The only accurate means of determining whether or not an anesthetized patient is in respiratory acidosis is by repeatedly drawing arterial blood samples during the course of an operative procedure and by getting a laboratory analysis for carbon dioxide tension. This, of course, is impractical and cannot be done as a routine. In our Clinic, however, studies have been done both in the animal laboratory and in the operating room to try to ascertain just how much respiratory acidosis is present during anesthesia. In the laboratory, both dogs and monkeys were used as subjects, and in the operating room, patients were used. Blood samples were drawn for CO₂ analysis at frequent intervals during operative procedures. Ether-oxygen or cyclopropane-oxygen were the anesthetic agents administered. Closed absorption and semi-closed technics were employed, using both reinforced and controlled respiration. In the group in which controlled respiration was the technic of choice, the majority of cases was controlled using the Jefferson Ventilator. This ventilator has a positive-negative pressure mechanism. The findings of this experiment, to date, have revealed a frightening result.

**Results of Study**

Of seven patients undergoing intra-thoracic procedures, six were in moderate to severe acidosis throughout the operation; the seventh was in slight alkalosis. None of these patients had elevation of blood pressure or pulse rate to indicate accumulation of CO₂ and none showed cyanosis.

These findings indicate that respiratory acidosis, in more or less degree, is more frequently the rule than the exception. The question of how much harm this does to the patient is a debatable one. I am sure we will all agree that it is not a desirable situation and should be overcome if possible. However, the real danger lies when this acidosis becomes associated with hypoxia—when we have allowed the patient's arterial oxygenation to fall short of normal demands. It is then that carbon dioxide excess becomes toxic to the cardiac mechanism and sets the stage for ventricular fibrillation or cardiac standstill. This catastrophe is fortunately relatively infrequent.

**Causes of Poor Ventilation**

But it is a documented fact that most of the fatalities occur either during induction of anesthesia or at the close of an operative procedure. During induction the anesthetist is hurriedly trying to get the patient into a state of flaccid relaxation to meet the demands of the surgeon. Too often time is not allowed for a safe induction with security in the knowledge enough oxygen is being provided and that the patient is breathing it in a natural way. There is nothing more satisfying to an anesthetist than to have a patient with good, full respirations from start to finish of anesthesia. One of the most
frequent criticisms aimed by the surgeon at the anesthetist of today is that they use “shot-gun anesthesia” and too frequently inject drugs into intravenous tubing. What the surgeon often does not realize is that a safe induction is impossible without time. Again anesthetists are many times required to carry patients to profound depths of anesthesia in order to produce sufficient relaxation, and in the lower depths of anesthesia normal physiology is not possible. Much attention is focused on the legal responsibility of the anesthetist and surgeon, but very little is written or said about their moral responsibility. There should be an understanding and a compromise between the surgeon and the anesthetist so that the patient can be protected from the hazards of “shot-gun anesthesia”. Anesthetists have actually made the statement to me that they have no qualms about administering any drugs to a patient as long as the surgeon “O.K.’s” it and takes the responsibility. If a surgeon requests, for example, that you give a patient an intravenous anesthetic, usually because it is quicker, and the anesthetist knows that it may be contraindicated for the patient, I feel that the anesthetist is morally wrong if he goes ahead just to please the surgeon.

Please do not get the idea that I am advocating that every patient be anesthetized with gas-oxygen-ether or that syringes of Pentothal, Surital and Curare, be put away because that is certainly not my intention. But I am advocating that when a patient needs an induction with gas-oxygen-ether he should have it no matter how long it takes. Also, that when we use the faster, more depressing agents we should be able to counteract the associated respiratory depression by our skill in maintaining adequate ventilation, thereby minimizing acidosis and preventing hypoxia or anoxia.

I should like to quote, in part, from a paper by M.D. Nosworthy, aptly entitled “A Return to Simplicity”: “whereas a patient may be able to limit what he takes into his lungs, he has no control whatever over what may be light-heartedly injected directly into his blood stream... There are various pitfalls—often of his own making—lying in wait for the inexperienced or unthinking anesthetist—Pulmonary inflation of a paralyzed patient is such a common practice during maintenance today that apnoea now holds few terrors, and consequently is produced at once by thiopentone without seemingly a thought to the cardiovascular slump which is the common sequel to injecting too much of this drug too quickly. Although this circulatory depression is fortunately only transitory, as a rule, it may progress and be long-lasting... In years gone by... most anesthetists were—and some still are—often content with a marked reduction in respiratory exchange: in fact, part of the toxicity ascribed to general anesthetic agents may well be due to the respiratory acidosis thus occasioned and the apnoeic technic was developed as an honest attempt to insure an efficient minute-volume exchange... Because of the belief that muscle relaxants are harmless, and because the apnoeic technic is so simple to those who understand it, active respiration is often abolished when the degree of peripheral suppression demanded by the operative procedure does not itself entail inadequate spontaneous breathing. The modern craze for speed is an added

incentive to knock out the respiratory center with thiopentone, to paralyze the patient completely for the routine intubation, and then to hyperventilate with nitrous oxide-oxygen so that the patient can be ready forthwith for the knife.

PREVENTION OF ACIDOSIS

Today there is more and more emphasis being placed on the use of lighter planes of anesthesia, adequate pulmonary ventilation and prevention of respiratory acidosis. Toward this end we, in our Clinic, are getting away from the use of the closed system, with necessarily low liter flow of gases. We have adopted the technic of using high liter flows, as much as 5 to 10 liters of oxygen in combination with nitrous oxide or helium, on a semi-closed technic, to carry the anesthetic agent of choice to the patient. In this way we feel that adequate oxygen is available to the patient and carbon dioxide is being continually “washed out”. We emphasize the use of the bag pressure manometer to regulate the resistance to the expiratory phase of respiration, maintaining as little resistance as feasible to keep the breathing bag in a state of partial distention. It is our contention that over-distention of the breathing bag, with consequent resistance to exhalation, is one of the most frequent causes of carbon dioxide retention. If muscle relaxants must be used, depressed respirations are assisted by increasing the amount of bag pressure so that it can be manually compressed. It is true that in using this technic it is necessary to use more of the anesthetic agent but the results are gratifying enough to warrant it. Respirations are, almost without exception, spontaneous and adequate; the patient’s skin remains pink, warm and dry; vital signs are maintained within normal limits; and at the end of the procedure the patient is responsive to the extent that he can reply to questioning. Another means which we employ to cut down on the amount of anesthetic agent needed and on reflex irritability is the frequent intravenous injection of small amounts (10-25 mgm.) of Demerol. With the judicious use of this drug we do not encounter depression of respiration or fall in blood pressure. By the above method the patient’s sympathetic tone can be maintained throughout an operative procedure with concomitant maintenance of blood pressure, pulse and respiration, without the excessive central depression inherent in deeper phases of anesthesia.

SUMMARY AND CONCLUSIONS

It is hard to make definitive conclusions or propound a formula for the maintenance of good pulmonary ventilation; however, I would like to summarize with the following recommendations. 1. If we are to be able to practice anesthesia, conscientiously keeping the welfare of the patient first in order of importance, it is mandatory that we have the surgeon’s complete confidence in our judgment. This can only be obtained by demonstration of our knowledge and our ability to make correct decisions and exercise good judgment over a period of time. No anesthetist who makes a practice of changing jobs once a year can ever hope to attain this respect. 2. We must keep informed about new drugs and technics and current literature on anesthesia. We must be good enough in our field
so that we are prepared to cope with any anesthetic problem with which we may be confronted without having to "call for help" because we feel inadequate. No surgeon can be expected to respect our abilities if we do not have confidence in ourselves.

3. To return to the original subject, I know that the apnoeic technic is necessary in the management of some patients and that it gives very satisfactory results in the hands of those skilled in its use; however, it is my firm belief that the good anesthetist is the safe anesthetist, and that the "safest" signs of good anesthesia are the maintenance of blood pressure, pulse and respiration within the limits of normal physiology by adequate pulmonary ventilation with oxygen and elimination of the toxic effects of carbon dioxide retention.