Controversial Questions in Anesthesia

A Forum

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CAPT. O'CARROLL: The purpose of this forum is to discuss controversial questions in anesthesia from a physiologic standpoint. There are differences of opinion even among the experts on many problems in the field of anesthesia. These differences involve certain technics, indications and methods for the use of drugs, and causes and proper treatment of complications. It is the purpose of this forum to explore some of these problems, with the object of arriving at a better understanding of the points in question and, where possible, to effect a compromise.

Subjects selected for discussion are: controlled respiration, balanced anesthesia, atropine, respiratory obstruction and laryngospasm, and muscle relaxants.

If any of you have any questions, we would like you to present your questions at the end of the program.

The first subject we are going to take up this morning is that of controlled respiration. Miss Miriam Shupp will be the first speaker. Miss Shupp.

MISS SHUPP: During intrathoracic operations the major concerns are adequate ventilation and the least possible interference with circulation. Beliefs concerning the best technic of anesthesia for transthoracic operations are at variance. Therefore a number of technics are in current use, both with respect to anesthetic and method, but today we are primarily concerned with method.

Briefly, these technics are: (1) no change in method either before or after the pleura is opened, the patient being permitted to breathe at atmospheric pressure; (2) spontaneous breathing by the patient with pressure in the rebreathing

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circuit increased above atmospheric without any assistance to respiration; (3) maintenance of atmospheric pressure in the rebreathing circuit with respirations being assisted occasionally or continuously; (4) maintenance of pressures above atmospheric in the rebreathing circuit with respirations being assisted occasionally or continuously; (5) deliberate abolition of spontaneous breathing and complete control of breathing with respect to the rate, the volume, and the rhythm.

With the technic of controlled breathing the anesthetist may use either the hand or hand-knee to maintain respiratory movement (manual control) or use a mechanical respirator (mechanical control).

While it is my opinion, based on personal clinical experience and the work of other anesthetists, that a patient can safely be carried through a trans-thoracic procedure by allowing him to breathe spontaneously and by assisting respirations continuously with intermittent positive pressure, I prefer the technic of controlled breathing with the use of a mechanical respirator.

To the first question, "Is controlled breathing physiologically sound?" I would say it can be, and this I shall try to show.

When the pleural cavity is opened, if the pressure in the rebreathing circuit is kept at atmospheric and no measures are taken to aid the patient's respirations, so-called paradoxical breathing occurs, the mediastinum flaps back and forth with the patient's respiratory movements, and hypoxia and apnea result. The shifting and flapping of the mediastinum as well as the altered intrapleural and intrapulmonic pressure affect the mechanics of circulation, and the patient's condition rapidly deteriorates.

When the pleural cavity is opened, if continuous positive pressure is used and no assistance given to the respiration, the mediastinum may be stabilized, both lungs may be kept expanded, and oxygenation may be adequate (partially by diffusion respiration), but carbon dioxide is not adequately eliminated. Respiratory acidosis develops, and also cardiac output is reduced.

If continuous positive pressure is maintained within the rebreathing circuit and the respirations are assisted, tidal volume is increased, but there is an increased possibility of retained carbon dioxide and increased interference with circulation in reduced cardiac output.

The next technic to be considered is that of intermittent positive pressure assistance with the patient breathing spontaneously. This technic produces more normal variations in intrapulmonic pressures, which should result in more adequate oxygenation and more efficient elimination of carbon dioxide—and does, according to Beecher, Motley, Courand and others—and also produces the least harmful effects upon circulation.

Watrous and others have gone further than saying assisted breathing should be used only in transthoracic procedures. According to Watrous, "controlled respiration or its modification, assisted respiration, . . . should be employed constantly in almost all inhalation anesthesias because it effectively counteracts the tendency of respiratory depressant and premedicant anesthetic agents to cause hypoxia or respiratory acidosis or both."
Motley, Courand and others found that intermittent positive pressure interfered less with cardiac output than continuous positive pressure, and that certain types of respirators employing intermittent positive pressure interfered less than others, and that the difference was on the basis of the pressure curves. They found that the "ideal mask pressure curve should increase gradually during inspiration to a peak not higher than about 25 centimeters of water. The pressure should drop rapidly after reversal, in the beginning of expiration decreasing quickly to or near atmospheric with a mean mask pressure during the expiratory period as near as possible to atmospheric. Inspiratory time should not exceed expiratory time . . . this pressure curve permits the right side of the heart to compensate during expiration from the reduction in filling during inspiration."

If this is true, it would seem that if a patient were allowed to breathe spontaneously and the respirations were assisted by intermittent positive pressure, the mask pressure curve for optimal cardiac output could be altered and probably would be altered, depending upon the rate and rhythm of the patient's respirations. In assisted breathing the anesthetist can control volume only, not rate or rhythm.

Therefore, if the ideal mask pressure curve for maximal cardiac output is to be maintained, it would seem that control of rate, volume, and rhythm would be necessary. And the control of volume and rhythm as well as rate means the use of so-called controlled breathing.

If we use controlled breathing, shall we use mechanical or manual control? I have no idea what the average time for transthoracic operations might be, but in my experience they have lasted from several hours to ten hours. Can any anesthetist "squeeze" the bag, take the pulse and blood pressure and keep a chart, perhaps give drugs intravenously and manage fluid therapy for three or four or ten hours, and maintain the precision of the cycling curve, which has been shown to produce the most efficient cardiac output, efficient oxygenation, and as seemingly effective elimination of carbon dioxide as is possible with any other known technic?

In my opinion, the use of the mechanical respirator in transthoracic operations is a boon to the surgeon and the anesthetist as well as a benefit to the patient, but not many surgeons or anesthetists are aware of it yet.

As to the question, "Is controlled breathing essential to satisfactory operating conditions?" I feel that you would need to go to the surgeons and ask that question of them. But I am quite certain that the answer would be "Yes" from the surgeons who do use the controlled breathing technic and "No" from the surgeons who do not, and I think that those who answered "Yes" might also add that it depends upon the type of operation. I know that some of the delicate blood vessel and cardiac surgery that is done by Dr. Claude Beck of the University Hospitals of Cleveland would be extremely difficult without the quiet field produced with controlled breathing, either manual or mechanical.

"Does controlled respiration by manual or mechanical means alter the
carbon dioxide tension and content of the blood stream?" Beecher and associates reported a series of transthoracic cases in which the patient's respirations were unassisted except occasionally, and there was a greater incidence of respiratory acidosis in these cases than in the series of cases, reported by him and his associates a year later, in which respirations were assisted.

Gibbons reported a series of transthoracic cases with mechanically controlled breathing. His figures for arterial carbon dioxide tensions in the pneumonectomy group were comparable to Beecher's. The figures for arterial carbon dioxide tension were higher in the lobectomy group reported by Gibbons. Gibbons pointed out, however, that these figures might not be valid because of the small series and also stated that respiratory acidosis will develop in certain patients regardless of the technic of ventilation. Both men mentioned the need for more extensive research in this area.

"Does the complete apnea in controlled breathing (manual or mechanical) abolish the important prodromes of circulatory failure by disguising signs of overdosage?" The patient under controlled breathing, for some reason or reasons yet unknown—one, perhaps, being lessened metabolic rate because breathing is effortless on his part—requires much less anesthetic than the patient who is allowed to breathe spontaneously.

With the use of controlled breathing a new set of signs of anesthesia must be learned. The neophyte may very well overdose the first several patients when using controlled breathing technics. In my opinion, an anesthetist, even an experienced one, should not begin to use this technic without some practice under guidance or a sufficient number of observations of the work of others.

Our patients, unless there has been a cerebral accident, such as may occur in some types of cardiovascular operations, breathe within a minute or two after being taken off the respirator and are responding in the operating room. They are usually awake before they leave the operating room.

"Does mechanically controlled respiration increase the hazard of explosion? If so, how does the hazard compare with that in assisted or manually controlled respiration?" So far as I know, there are only two makes of respirators currently in use in the United States that can be used as part of the anesthesia equipment. One is the Mautz, which runs on air pressure, and from the standpoint of the explosion hazard—despite the fact that Beecher said mechanical respirators increase the explosion hazard—it presents no more of a hazard than the squeezing of the bag by hand.

The other respirator is the Rand-Wolfe, which is operated by an explosion-proof Fire Underwriters-approved motor. An electrical motor, even of the approved type, does increase the hazard, but no more so, I should think, than a motor-driven suction or ether-air machine.

"Does controlled respiration increase the incidence of pulmonary complications?" Not to my knowledge, although we have not done any studies on this. I believe the incidence is extremely low. Any technic that provides adequate ventilation, that prevents atelectatic areas from developing or remain-
ing, and that provides efficient circulation would, I should think, reduce the incidence.

Capt. O’Carroll: Dr. Hrant Stone will now give his point of view on the subject of controlled respiration. Dr. Stone.

Dr. Stone: The problem of controlled respiration has certainly changed in the past five or six years. Previously, there was a great deal of controversy whether or not controlled respiration was of value in the operating room under any circumstance. I believe today the feeling has gone to the other extreme where almost everyone feels that there are definite indications for using controlled respiration under certain circumstances.

Miss Shupp has certainly covered most of the important features of controlled respiration. I think, first of all, we should say something about the methods by which controlled respiration can be instituted.

During the course of an anesthesia the drugs that are used are certainly depressant to the respiratory center. The normal physiologic stimulus to respiration is carbon dioxide. Therefore, controlled respiration can be instituted, first, by increasing the resistance, or the threshold, of the respiratory center to carbon dioxide. This we do by the various anesthetic drugs we use, that is, it takes more carbon dioxide to stimulate the respiratory center than it did previously. Secondly, we may produce controlled respiration by washing out carbon dioxide, that is, by removing the physiologic stimulus, and hence the patient will become apneic, either until anoxia sets in or certainly until an active amount of carbon dioxide is allowed to accumulate. Third, controlled respiration of a different sort, a peripheral type of controlled respiration, can be instituted by the use of muscle relaxants.

During the course of clinical anesthesia one, two, or three of these technics are employed for producing controlled respiration. Probably, the most commonly used technic is to give a patient a depressing drug. We use morphine for medication, give cyclopropane, pentothal sodium, or other drugs that depress the respiratory center, and wash out carbon dioxide by compressing the rebreathing bag.

What are the possibilities of harm from controlled respiration? First of all, we must think of the rupture of an emphysematous bleb or the rupture of alveoli, which would allow air to escape outside of the lung perivascularly to produce mediastinal emphysema. Immediately, I should say it takes a great deal of pressure to cause such an accident, and usually during the course of clinical anesthesia such a pressure is not attained. The actual amount of pressure needed to rupture the lung is debatable, and no one has ever come up with a suitable explanation of the pressure needed to do it. It is possible, however, that in a patient with emphysema, an elderly patient, the use of positive pressure is attended by a greater hazard because of degenerative pulmonary changes, so that the lung has lost its distensibility, and rupture of the alveoli is a greater possibility.

Also, with a great deal of positive pressure that increases intrapulmonary pressure, you can drive oxygen into the arterial blood and produce arterial
embolism. Again, the amount of pressure needed to do such a thing is far greater than the pressure used during the course of clinical anesthesia.

So I believe you can immediately mark off the first two contraindications for, or dangers of, controlled respiration.

The third disadvantage to controlled respiration is the effect on the circulation. The first work that was done on the effect of positive pressure on circulation used constant positive pressure, that is, there was no phase during the course of the respiratory cycle when the intrapulmonary pressure returned to normal. At all times intrapulmonary pressure was above atmospheric pressure. If you use a technic of constant positive pressure, it is true that you can produce depression of circulation by positive pressure breathing. However, the technics in current use do not employ constant positive pressure. The technic that is used today is intermittent positive pressure with a relatively short phase of positive pressure; within a minute the amount of time given to positive pressure is much less than the amount of time during which relaxation takes place and atmospheric pressure exists in the lung. So if intermittent positive pressure is used with a long respiratory phase during which atmospheric pressure is reached within the intrapulmonary space, there is no significant effect on circulation.

Immediately I make an exception. If a patient with low blood pressure from shock or a decreased blood volume is put on positive pressure breathing, the circulation can be severely depressed. At the present time I do not know of anyone who has determined how much the systolic pressure must decrease before positive pressure breathing will produce detrimental effects. That certainly should be worked out. But let us say that a patient who is in fairly good condition will tolerate controlled respiration without any significant danger.

Miss Shupp covered the other features of controlled respiration, with the advantages and the disadvantages of using it. May I add that if a long intrapulmonary operation is undertaken without the use of controlled respiration, or at least semicontrolled respiration, the patient will have severe respiratory acidosis.

Miss Shupp pointed out that Gibbons and his group and also Beecher showed that while a patient can be adequately oxygenated—in fact, the oxygen saturation of hemoglobin increases during the course of an operation—carbon dioxide cannot be removed sufficiently for the prevention of respiratory acidosis unless some form of controlled respiration is used.

Capt. O’Carroll: From where I was sitting there didn’t seem to be much controversy on that question. The next subject for the forum is balanced anesthesia, and I now introduce to you Mrs. Helen Lamb Powell.

Mrs. Powell: Just what is balanced anesthesia? I have not been able to find any satisfactory explanation of balanced anesthesia. Gwathmey, in the 1920’s, combined the synergistic effects of morphine and magnesium sulfate with ether administered rectally for obstetrics. He called it synergistic analgesia. In other words, by the administration of drugs with synergistic actions, a less toxic dosage of any one drug was used.
Dr. Thomas, when I asked him what balanced anesthesia was, said, "Well, I always tell my people that balanced anesthesia is the anesthesia that the surgeon does not complain about."

Dr. Lundy was the first to call the administration of several drugs for premedication and anesthesia balanced anesthesia. His purpose was to gain an effect through a combination of agents that if produced by one agent would be undesirable.

We all know that ether is still regarded as one of the safest anesthetics. However, ether without the administration of premedication can be dangerous, with profound depression of respiration and circulation, anoxia, and other complications.

Nitrous oxide is also regarded as one of the safest anesthetics at our disposal, and no death has occurred from nitrous oxide if the patient received proper oxygenation. We all know the administration of nitrous oxide for the production of anesthesia without supplementing it with another anesthetic is very dangerous, but nitrous oxide in combination with another anesthetic drug is regarded as an ideal anesthetic in many clinics. The same may be said of ethylene.

Pentothal sodium like nitrous oxide is not a true anesthetic. It does not produce the relaxation necessary for intra-abdominal surgery. But in combination with one or more drugs it may be used to produce the degree of relaxation that will please the surgeon without profound depression. On the other hand, if we attempt to control the anesthesia with just pentothal sodium —without supplementing it with an inhalation anesthetic or some other drug—so much of the pentothal sodium must be given that the patient is profoundly depressed for days; there is likely to be deterioration.

Curare is one of the most valuable drugs that have been introduced into anesthesia in recent years. On the other hand, in some clinics today its use is forbidden, because of its misuse—its administration in profound doses. It has no direct effect upon circulation, but when a state of anoxia is brought about, that naturally depresses circulation.

With respect to current practices in regard to balanced anesthesia, I don’t know that it could be called balanced anesthesia. You probably have heard the terms “garbage anesthesia” and “crash anesthesia.” "Garbage anesthesia" and "crash anesthesia" are one and the same thing. It constitutes the administration of great dosages of premedication, then bringing the patient to the operating room and perhaps giving him a heavy dose of demerol, after which pentothal sodium is administered until he has not only lost consciousness but is in a profound state of respiratory and circulatory depression, with severe cyanosis before an inhalation anesthetic and/or curare is given.

So what is balanced anesthesia? It is the administration of a combination of drugs for anesthesia that will bring about as nearly a normal physiologic state during the administration of anesthesia as it is possible to achieve. Anesthesia is not a normal state. There are varying degrees of depression of both circulation and respiration and the danger of limitation of oxygen. So the
drugs we have at our disposal we should administer as carefully and as safely as possible. Profound depression of respiration should certainly not be countenanced by any anesthetist, nor should circulatory depression primarily due to the depressing action of the anesthetics. It behooves us all to have a thorough knowledge of the pharmacologic action of drugs used and avoid toxic drugs and dosages of drugs that will bring about depression.

Capt. O'Carroll: We will now hear from Dr. Krumperman, anesthesiologist at Temple University here in Philadelphia. Dr. Krumperman.

Dr. Krumperman: It is very difficult to define balanced anesthesia. To me balanced anesthesia means a combination of anesthetic agents or anesthetic technics that will produce ideal operating conditions while the patient is maintained in as near a normal physiologic state as possible.

Balanced anesthesia has been abused, and abused badly, throughout the country, and I think when Beecher condemned "shotgun" anesthesia he had a perfect right to do so, because it has become a tendency everywhere to use dosages of drugs that are detrimental to the patient's well-being unless he is observed carefully. I do think that if this so-called shotgun anesthesia can be handled properly, there is no great damage done to the patient, but dosages and technics are all out of line in a fair number of places throughout the country.

When I think of balanced anesthesia, I usually do not think of the combination of various inhalation anesthetic agents but of the combination of general anesthesia with regional anesthesia. I also think balanced anesthesia should be reserved for the poor risk patient. For instance, a patient in the older age group is scheduled for a radical intra-abdominal operation for carcinoma. You feel that the best operating field would be produced by spinal anesthesia or epidural anesthesia, yet you know the patient's physiology would be greatly disturbed if the level were adequate to permit the operative procedure to be performed.

Therefore, if you could obtain abdominal relaxation and still take care of abnormal reflex disturbances and discomfort from traction that the patient would have if regional alone were used, this is the type of patient on whom to use a combination of regional and general anesthesia, whether it be with pentothal sodium, nitrous oxide, cyclopropane, ether, or ethylene.

A fair number of people have misused pentothal sodium with regional anesthesia as a form of balanced anesthesia. Pentothal sodium, as you know, is classified as an anesthetic agent, but its anesthetic properties are nil unless used in rather large doses. Therefore if a spinal or epidural block is poor and the surgeon happens to get out of the operative field and causes pain to the patient, the situation is rather difficult to control with pentothal sodium alone. If you have poor regional anesthesia, the proper technic to use to balance it would be some form of inhalation anesthesia. It could be pentothal sodium-nitrous oxide but not pentothal sodium alone. Or it could be any of the other general anesthetic agents that are available.

Curare is a wonderful adjunct to general anesthesia. But there again it is
an agent that has been misused and is now being abused by the surgical profession. Surgeons are blaming curare for a lot of the complications that are occurring not only in the immediate operative period but also in the post-operative period. We must use the muscle relaxants cautiously and not use them in too large doses. With the skill that most anesthetists should be able to attain after a few years of administering anesthesia, the dosages in which and the frequency with which these curare preparations are used can be minimized. They are needed, but I do think they should not be used to excess.

Surgical procedures for which I feel combined anesthesia is essential are those in the upper abdominal field, such as gastrectomies. For example, a surgeon may be unable to determine whether or not he is going to perform a subtotal or total gastrectomy until he has opened the abdominal cavity. If you have prepared for the major portion of the operation, which may be total gastrectomy, by administering spinal or another type of regional anesthesia and supplementing or complementing it with inhalation anesthesia, then there is no delay in the operation. The surgeon can progress from the abdomen into the chest, if necessary, without a lot of fuss, and it reduces the operative time for the surgeon and the patient, and therefore postoperative morbidity and possibly mortality will be lessened.

We should continue to use balanced anesthesia but be careful in the choice of the agents used and be careful to use it only on patients for whom we feel it is indicated. I don't feel that all patients need balanced anesthesia. I think it is a technic to be reserved for the poor risk patient.

**Capt. O'Carroll:** The next subject that we are going to take up is respiratory obstruction and laryngospasm. I will now introduce to you Miss Margaret Sullivan from the Roosevelt Hospital, New York City.

**Miss Sullivan:** Respiratory obstruction is the plague of all anesthesia, and I am certain that it is a condition that every person in this audience has not only encountered but has contributed to its production along with the accompanying complications.

Technic must be considered as a contributing factor in the production of respiratory obstruction. One of the questions submitted for discussion reads as follows: “Is any technic available that disguises the signs of respiratory obstruction?” To this question I must answer that some opponents of the mechanically controlled respiration technic cite it as being the one method in which the anesthetist is not aware of respiratory obstruction. From my own experience with mechanical control of respiration, which is by no means so extensive or varied as that of Miss Shupp, I think it is perhaps the one technic that presents the earliest signs or indications of respiratory obstruction. The anesthetist must very closely observe the operative field, or rather the inflation and deflation of the lungs. This technique offers the opportunity to note the lack of respiratory obstruction or, if such a condition should develop, to observe the first indication that all is not well within the lungs. Evidence of obstructing material, such as a tiny broncholith, is immediate with this technic.

I think that any anesthetic agent or adjuvant to anesthesia can be a contributing factor in disguising the signs of respiratory obstruction. Chloroform in
the lower planes of anesthesia, pentothal sodium, avertin, and curare and curare-like preparations all produce a diminished minute volume. This tends to invite respiratory obstruction.

The next question deals with the anesthetist. "In what way or ways does she or he contribute to the production of respiratory obstruction?" I think we could devote the greater part of this morning to a discussion of the role of the anesthetist in the production of this condition. The anesthetist who permits a high concentration of ether during induction, a concentration of cyclopropane that is too high, who fails to establish a patent airway with either a pharyngeal airway or an intratracheal catheter, who fails to remove secretions from the pharynx, trachea, or bronchus, who fails to adjust the head and neck to support the jaw after the insertion of an airway is solely responsible for the obstruction that develops. These are but a few ways in which the anesthetist violates the principles of safe anesthesia.

Failure to institute prompt treatment is perhaps the greatest sin that can be committed in the presence of respiratory obstruction. We are well aware of the steps to be taken to right this condition. Some anesthetists resort to the use of drugs. It is my feeling that providing a patent airway, administering oxygen, and/or altering the level of anesthesia are the best methods to be employed. Atropine has been used for the treatment of laryngospasm that resulted in respiratory obstruction. Curare has no place in the treatment of respiratory obstruction, although this drug has been employed for the relief of laryngospasm.

To the question, "Is intratracheal intubation necessary to prevent respiratory obstruction?" I state with emphasis, "No."

The next question for consideration should be read in full: "How do agents, technics, the anesthetist, and the surgeon contribute to the production of laryngospasm? Are certain drugs, such as barbiturates, curare, atropine, and high concentrations of inhalation agents, or methods more prone than others to produce laryngospasm?"

Let us first consider the agents. It is an accepted fact that pentothal sodium does tend toward the production of laryngospasm, and it is likewise an accepted fact that laryngospasm usually occurs during the induction period. Why? Because this time finds the anesthetist involved in attending to so many details that his or her attention may be momentarily diverted, or the exact level of anesthesia may not be fully evaluated. Laryngospasm may occur during other periods of anesthesia, but only if the level of anesthesia is not sufficiently deep for the operation to be performed.

Atropine and curare are employed by some anesthetists for the relief of laryngospasm; thus we cannot consider them as drugs that aggravate the larynx.

It is my feeling that one of the greatest contributing factors in the production of laryngospasm is the pharyngeal airway. All too frequently the airway is inserted when the level of anesthesia is not sufficiently deep. Is there any anesthetist who has not been witness to the sequence of events that follows the insertion of a pharyngeal airway under light pentothal sodium anesthesia?

The surgeon contributes to the production of laryngospasm only insofar as
the anesthetist permits him to work under light anesthesia. I again refer to the
gent pentothal sodium, for it is so frequently employed when laryngospasm does
occur. We all have observed an attempted dilation of the rectal sphincter in the
presence of light anesthesia, particularly when a barbiturate and some of the
less potent agents have been employed. We all are aware of what follows. The
same may be said for upper abdominal operations in the presence of light anes-
thesia. The agent, the technic, or the surgeon cannot be blamed; the anesthetist
is entirely responsible. He or she is the person who should have full knowledge
of the sequelae that will develop with each agent and technic.

The signs of oncoming laryngospasm may be overlooked. The level of anes-
thesia may not be correctly ascertained. Who or what is responsible? The
anesthetist.

The anesthetist who fails to recognize the signs of obstruction or laryngos-
asm, who fails to appreciate the cause of either condition, who fails to institute
immediate and adequate corrective treatment, be it the administration of oxygen
or drugs, must assume full responsibility for all that ensues.

In conclusion, permit me to repeat the thought which I have been attempting
to bring to you; not the agent, not the technic, not the surgeon, but the anesthetist
is the one who either produces or prevents respiratory obstruction or laryngos-
asm.

CAPT. O’CARROLL: We are now going to hear from Dr. George R. Bright-
ton, the attending endoscopist at the Roosevelt Hospital, New York City, and
he will probably be able to tell us all about laryngospasm.

DR. BRIGHTON: I feel somewhat like a Taft supporter at an Eisenhower
rally. I want of you and demand something of you. I am not only an endoscopist
but also an otolaryngologist. That speaks for itself when you speak of laryngo-
asm.

Most of you may think of the larynx as the organ of phonation. Actually
that is only one small part of its history. It goes back a long way in evolution.
At the opening of the swim bladder of the fish are a group of muscles that act
as a sphincter. That sphincter enables the fish to get away from its enemies
because he can swim longer than his enemies can in water without oxygen.
So remember that there is a psychologic reaction in laryngospasm. It is not all
due to the anesthetist, as we have just heard. When you anesthetize a patient,
you have a decerebrate animal, and that animal has a subconscious. In that sub-
conscious is fear, and many times you will have a patient get laryngospasm in
spite of the fact that you have been extremely careful in your induction and
have used the most perfect technic and all of the drugs at your command. You
still have laryngospasm. And I think to a large extent it goes back to the fear
complex connected with the operation of the swim bladder of the fish. It may
seem farfetched, but I do think there is something in that philosophy. We have
never been able to prove the hookup with the nervous system, but it certainly
does occur.

Laryngospasm is also due to mechanical obstruction. You get mechanical
obstruction first, and then you get laryngospasm. We have made a revolutionary
change in the naming of some of our instruments. One of them is a tongue depressor. We now call that a tongue elevator, and you would be surprised what a difference it has made in laryngeal obstruction. I think you should go back to your operating rooms and simply tell your operating room supervisors that you no longer have tongue depressors; you have tongue elevators. It has made a tremendous difference in our technic both in anesthesia and in surgery.

Now, as Miss Sullivan asked, is there any technic of anesthesia that is a guarantee against respiratory obstruction? I say “Yes,” there is a technic, and that technic is very careful induction. So many of us are in a hurry. We push our anesthetists and say “Come on, let’s get this patient under. We don’t have all day.” This makes the anesthetist push the induction, which causes respiratory obstruction, which causes laryngospasm.

Miss Sullivan, I think, has touched on the way the anesthetist can help in removing the material that causes respiratory obstruction. The surgeon to a great extent can also help, because respiratory obstruction often occurs after the patient has been prepared by the anesthetist and the surgeon takes over, particularly the otolaryngologist. We are in the way most of the time, and we are apt to push the chin down or change the position of the head without paying much attention to the patient’s respiratory efforts. It is difficult for the anesthetist to say to the surgeon, “You are causing obstruction.”

As to the use of intubation to control laryngeal obstruction, I, for one, am completely opposed to routine intubation. I think anesthetists who say, “We use it on every patient we operate on,” should be suspect.

The simpler technics in anesthesia can do away with laryngospasm and laryngeal obstruction only if they are properly applied and the proper anesthetic agents used.

There was one question, “Are drugs effective in the relief of respiratory obstruction or laryngospasm?” We have had some rather interesting experiences in doing laryngeal examinations under general anesthesia. The patient would be in third plane anesthesia, but the minute the laryngoscope was put into the larynx, the larynx would go into spasm. In cases of that sort topical anesthesia before general anesthesia will give you an anesthetized larynx with no reflex, so that you can have a fairly light general anesthesia and still be able to carry out the examination properly.

The problem of laryngospasm and laryngeal obstruction, particularly to us in otolaryngology and bronchoesophagology, is extremely important, and I appreciate very much the opportunity of talking to you this morning, because many times you and I have the same problem and have to work it out together.

**CAPT. O’CARROLL:** I am sure that all the people on the forum with me will agree with Dr. Brighton that the nervous system is hooked up with laryngospasm. I am sure we have all had some laryngeal irritation this morning even before we started to approach this microphone.

We will now hear something on the subject of atropine, and Miss Margherita Powers, from Johns Hopkins Hospital, will be the first speaker.

**MISS POWERS:** “What are the effects of atropine on the respiratory func-
tions, circulation, the vagus nerve, and the heat-regulating mechanisms?"

Atropine has two separate actions affecting most of the organs of the body. The centers in the brain are stimulated, and the transmission of impulses from postganglionic craniosacral nerves to smooth muscle is interrupted. The specific effect on any organ or tissue is the function of the dose.

When administered as part of preanesthetic preparation, the dose of atropine is usually about 0.5 mg. In this dosage the respiratory center is stimulated, and the depressing effects of morphine or similar drugs are counteracted. In addition, the transmission of impulses from the vagus nerve to the bronchial muscles and the secretory glands of the respiratory tract is interrupted. Through these three effects, central stimulation, bronchial dilatation, and suppression of secretions, respiratory minute volume is increased.

Atropine has little direct action on the myocardium but causes cardiac slowing in doses of 1 mg. or less, by stimulating the nucleus of the vagus nerve. In persons in the extremes of age and in Negroes the cardiac slowing is less noticeable. With larger doses vagal inhibition is masked by the blocking action of atropine between the vagal terminations and the effector cells. Blood vessels, other than those in the splanchnic area, are dilated. The blood pressure is not usually altered by average doses.

Although peripheral vascular dilatation, under other circumstances, encourages heat loss, with atropine body temperature tends to increase because of the reduction in secretion of sweat and an increase in metabolism. Little evidence exists of direct effect on the temperature-regulating center.

"Is atropine effective against laryngospasm?" This has been covered fairly carefully in the previous discussion. The use of atropine to prevent and treat laryngospasm is widespread if one believes the literature. By reducing secretions atropine can be helpful in preventing spasms caused from local irritation of secretions. But as a treatment for laryngospasm there is no pharmacologic basis for its use. Atropine in the usually recommended doses has no effect on striated muscles. The muscles of the vocal cords are striated, even though they are innervated by the vagus nerve.

"In what types of cardiac disturbances is atropine indicated?" If atropine is to be used to treat cardiac disturbances, two points should be kept in mind: (1) Atropine prevents vagal inhibition of the heart whether reflex or direct. (2) Small doses (less than 1 mg.) will not interrupt completely impulses from the vagus. Effective treatment of sinus arrhythmias, ectopic beats, A-V blocks, premature systoles, and bradycardias, regardless of origin, is reported. However, some difference of opinion exists as to whether atropine is the best drug for any of these. Procaine, digitalis, quinidine, and epinephrine are also recommended. Internists and cardiologists have more faith in digitalis and quinidine usually.

"Does atropine as premedication disguise the signs of asphyxia?" Alterations in pulse rate, blood pressure, and respirations are generally considered the best signs of asphyxia. All of the drugs used as preanesthetic medication cause some variation in one or more of these. With atropine the onset of bradycardia may be delayed, and the blood pressure and respiratory changes may be abrupt
and slow in appearing. Little experimental work has been reported on this question. Reports of clinical observations are not very convincing, particularly when we remember that deep anesthesia also masks the same signs.

"Does the use of atropine contribute to the occurrence of convulsions?" By suppressing the secretion of sweat and increasing metabolism, atropine is possibly a factor in the production of convulsions. But it is only one of many factors. Inadequate exchange of respiratory gases, fever, high environmental temperature, malfunction of the temperature-regulating center, toxic disease, electrolyte and fluid imbalance, and the technic of anesthesia are all considered contributing factors. Some of these are beyond our control; others we can control to some degree. Before indicting atropine, we should, as far as we are able in every anesthesia, insure adequate exchange of oxygen and carbon dioxide, provide proper fluid replacement, and discourage heat retention.

"Does the use of atropine contribute to postoperative pulmonary complications?" We occasionally hear of a patient who has received too little or no atropine as premedication. In this patient the respiratory secretions were so profuse as to interfere with respiratory exchange and the establishment or maintenance of anesthesia. Whether the patient should be given atropine in such a situation is a controversial question. It has been stated that atropine will thicken the mucus, and that this will lead to atelectasis. It seems unreasonable that anyone would believe that atropine could have direct action on mucus bubbling in the lungs. However, as a result of inhibition of further secretion and bronchial dilatation, the increased airflow in the bronchial tree may lead to the evaporation of water from the mucus making it viscid. Deepening the anesthesia, if this can be accomplished, will produce the same effects. Aspiration of the pharynx or tracheobronchial tree is usually of very temporary or no help. If atropine is given, good postoperative care will probably prevent atelectasis.

"Should atropine be used routinely as premedication? If not, are there specific indications for its use?" The advisability of inhibiting the secretion of mucus and saliva during general anesthesia is probably unquestioned. It should be remembered, however, that scopolamine is a better drug for this purpose, although many consider it more toxic. Furthermore, the idea that the usual preanesthetic dose of atropine will reduce cardiac irregularities is unproved. On this basis, scopolamine may be substituted.

The use of either atropine or scopolamine before regional anesthesia causes the patient unnecessary discomfort in the form of an objectionably dry mouth. If general anesthesia becomes a necessity, atropine or scopolamine may be administered intravenously before beginning the induction.

**Capt. O'Carroll:** Our second speaker on the subject of atropine is Dr. Duncan A. Holaday, from the Presbyterian Hospital, New York City. Dr. Holaday.

**Dr. Holaday:** In 1940 Goodman and Gilman in their textbook outlined the experimental and clinical information on atropine. Its clinical use dates back to very ancient times when it was used for its mydriatic effects. Pharmacologic studies on atropine date back about a hundred years or more. And yet
there is a great deal that we don't know about it. Miss Powers has just reviewed for you much of what we do know about it, and I am going to take a minute to discuss some of the things that we don't know about it.

First, in brief recapitulation of what we do know. Atropine has two sites of action, central and peripheral. Centrally it stimulates most of the medullary centers and many higher centers. It may have some depressant action, as is indicated by its use in the treatment of parkinsonism, and yet this may not be a directly depressant action but a stimulating action having its effect by stimulating centers that inhibit the gross movements characteristic of parkinsonism.

Peripherally, it acts as if it paralyzed the postganglionic craniosacral nerves to smooth muscles and glands. It does not interfere with the production of acetylcholine. Apparently, as was indicated by the work of Clark in 1926, it competes with acetylcholine. Moe and collaborators in 1950 disproved the displacement theory of its action against acetylcholine in working with the pressor response to acetylcholine.

Atropine may have some direct effects upon myocardial tissue, although this is in dispute at the moment. It has some mild anticholinesterase activity, as was indicated by the work of Scholler in 1942. Marazzi in 1938 showed that it had some ganglionic blocking action. This has been confirmed by work that Koelle, Kamijo and I have done in recent months. So much for what we know about it.

We will consider for a moment what we don't know about it. In the first place, we have no idea as to what its mechanism of action is on the central nervous system. It has been the consensus until recently that synaptic transmission in the central nervous system was almost certainly not humoral or chemical and most probably electrical. If this theory obtains, there is little rational basis for believing that atropine has any effect centrally. However, di-isopropyl fluorophosphate and other anticholinesterases have been shown in recent years to have striking effects upon the central nervous system, and the evidence that a humoral mechanism may obtain is increasing. Atropine is useful in depressing the undesirable actions of di-isopropyl fluorophosphate, acetylcholine and eserine on the central nervous system. It may well be that it has a mechanism of action similar to that which obtains peripherally.

Regarding its peripheral mechanism of action, it would appear to act as curare does, that is to block the effects of liberated acetylcholine upon the receptor cells. That atropine primarily affects smooth muscles and glands, that curare blocks skeletal muscle, and that tetracethylene ammonium blocks ganglionic transmission is said to be due to the specificity of the receptor substance in the reacting end-organ.

That atropine efficiently blocks the action of acetylcholine at all muscarinic points of action but has a rather poor action upon the effects of vagal stimulation on the small intestine has been laid to the fact that the nerve endings going to the smooth muscle of the intestine are inside the cell at a site where atropine does not have access. This is mostly opinion, and there is little direct proof for it. Histologic studies of the smooth muscle of the intestine do little to clarify the point, the usual methods not being adequate to study these small structures.
However, certain histologic studies, including the work of Cajal, indicate that the nerve endings of the postganglionic nerves in the plexuses of the intestine do exhibit end plates that appear to be extracellular.

Another piece of evidence that may eventually help to clarify this matter has come from a French worker who has isolated buteryl choline from the smooth muscle of the bowel of the ox. It is perhaps that the normal transmitter of the postganglionic impulse in the intestine is not acetylcholine but some other choline ester, perhaps buteryl choline, and atropine may not be nearly so effective in competing with this transmitter as with acetylcholine.

Atropine is generally conceded to have no effect upon skeletal muscle. However, extremely large doses can be shown to modify the response to electrical stimulation of efferent motor nerves to skeletal muscle.

Regarding the problem of the efficiency of atropine in overcoming laryngospasm, I have no good opinion at all upon its clinical efficacy. I have tried it only once or twice. In one instance I thought it got me out of a rather difficult situation. I used two or three 1 mg. doses administered in rapid succession. I was using every other remedy at the same time, so I could not prove that atropine had any effect. Most people who say that atropine may have a beneficial effect upon laryngospasm have to rely upon the same kind of evidence. Nevertheless, the possibility has been raised, and there may be some basis in fact for it.

Two new facts have been uncovered recently that may eventually lead to an understanding of this question. Youmans at the University of Wisconsin described some very interesting experiments at the recent Pharmacology Society meeting. Stimulating the motor nerves directly in the severed sciatic, he was able to show that ether has a curareform action on a rather special preparation. He administered curare by slow drip to anesthetized dogs just to the point where there was slight interference with neuromuscular transmission. In such a preparation he could then administer ether and show a striking block of neuromuscular transmission to skeletal muscle at a time when the respiratory effects from the ether were just becoming apparent. This did not show up in every experiment. The one common denominator that he found in the study was atropine. In those 10 to 12 kg. dogs that had received premedication of 1/100 gr. atropine, he found an exquisite effect of this small dose of atropine upon the neuromuscular junction.

There may be effects, important effects, upon the larynx, which, although it is a striated muscle group, is not a muscle group having the same phylogenetic or ontogenetic derivations as most of the peripheral voluntary muscles, arising as it does from the branchiomieric muscle groups.

A second fact that may eventually help to clear up this particular problem concerns studies having to do with a rather obscure system going to skeletal muscles. Kuffler and his associates in recent years described the small fiber system going to skeletal muscles. Very briefly, in the frog this consists of nerve fibers half or less than half the diameter of the usual large alpha motor fibers that innervate skeletal muscles. Stimulation of these small efferent fibers in the frog's rectus produces a tonic response, not a twitch response such as one gets from stimulation of alpha motor fibers.
In mammals, the species we are primarily interested in, Kuffler was not able to demonstrate any motor responses to stimulation of these small nerves. They are present. He has described them in large representation in the sciatic nerve of the cat. The function that he ascribed to them is that they send motor impulses to one of the two primary proprioceptive organs in the muscles, namely, the muscle spindles. Impulses coming from muscle spindles which by monosynaptic pathways modify the response of the anterior horn cell to impulses playing on the anterior horn cell. An article by Tasaki and his associates raised the point that these may actually be motor fibers in the same sense that they are in the frog. Again I call your attention to the fact that the striated muscle of the larynx is not an ordinary group of musculature. Aside from its ontogenetic derivation, its function is considerably different from that of most of the skeletal muscles. It is an organ in which tonus plays a great role in its ordinary work. That normal tonus has anything to do with the tonus that we see in laryngospasm I don’t necessarily mean to imply. However, the possibility certainly exists.

One more fact to bring out the point of this story. In the frog the motor fibers from anterior horn cells to the skeletal muscle of the rectus are exquisitely sensitive to blockade by curare. Atropine has very little effect upon neuromuscular transmission from these fibers. However, atropine does have a much greater effect in blocking the effects of stimulation of the small fiber system to this muscle.

I have not been able to find any information upon the effects of atropine on the small fiber system in mammals. I am not sure any work has been done.

This is a very provocative problem, and one that I hope more work will soon clear up. In the meantime there is good reason to keep open the question as to the efficacy of atropine on laryngospasm from a theoretical if not a clinical point of view.

**Capt. O’Carroll:** Our next subject will be muscle relaxants, and Mrs. Opal M. Schram, from Wesley Memorial Hospital, Chicago, will be the next speaker. Mrs. Schram.

**Mrs. Schram:** The problem of the use and misuse of the muscle relaxants never fails to bring forth innumerable varying opinions. The committee has offered eight questions concerning curare, and I shall introduce the discussion of this subject by presenting my answers to these questions.

The first question: “Under what conditions are muscle relaxants used as a substitute for adequate anesthesia?”

All of us are aware that muscle relaxants do not produce either narcosis or relief from pain, and hence that anesthesia is necessary. The difference of opinion apparently lies in the degree of anesthesia believed to be necessary. In our hospital we are accustomed to using upper second plane anesthesia for abdominal surgery. I am told that curare is used by some to keep the patient on the table with anesthesia so light that there is imminent danger of returning consciousness. This would constitute using the drug as a substitute for adequate anesthesia and would, in my opinion, be quite improper. On the other hand, to use curare to quiet a vigorous, muscular, young patient who is requiring an excessive amount of pentothal sodium seems to me justifiable.
The second question: “Does the respiratory depression following adequate curarization have ultimate ill effect?”

It is the responsibility of the anesthetist to maintain adequate respiratory exchange throughout anesthesia, and if this is done no harm will result during this period. It is the anesthetist’s further responsibility to make as certain as possible that the respiratory exchange will be adequate during the immediate postanesthesia period, which means active, functioning intercostal muscles at the completion of the operation. Inadequate ventilation after operation should not be permitted. The literature reports postoperative atelectasis and death following curarization, but in most of the reports that have come to my attention curare was used in a more radical way than we are accustomed to using it. However, in some of these case reports the technic and dosage did not seem radical, so there may be occasional unusual delayed effects for which we should be on the alert. The article to which I refer is in The Journal of the American Medical Association, April 29, 1950, Foyger’s article on “Fatalities following the use of curare.”

“Are the adverse effects of muscle relaxants always reversible?”

One of the requirements of a good anesthetic drug is that its effects be reversible. Muscle relaxants qualify in this regard to about the same extent that anesthetic drugs do, with the exception that any drug given intravenously is attended by greater hazards because it is so easy to administer, and a gross error in dosage may be made. I would say that the adverse effects of muscle relaxants are reversible if the dosage is carefully individualized, and if the patient is not hypersensitive as is the patient with myasthenia gravis.

“Are the known antidotes to muscle relaxants always effective? Are the antidotes in themselves dangerous?”

The antidotes for muscle relaxants, as is true of antidotes for other drugs, may be ineffective and may be dangerous. While the careful anesthetist has little use for antidotes of any kind, the antidotes for muscle relaxants are comparatively safe and effective.

“What ill effects may result from repeated doses of muscle relaxants?”

d-Tubocurarine chloride, d-tubocurarine dimethyl ether, and flaxedil all have cumulative effects, and prolonged depression of the muscles of respiration may follow repeated doses. The greatest danger is present when the patient has left the care of the anesthetist and the reduced respiratory exchange may not be recognized by the attendant in charge.

Syncurine is not cumulative in action; on the contrary it becomes tachyphylactic, and repeated doses are ineffective in producing muscle relaxation. However syncurine has such pronounced depressing effects on respiration that this is a frequent complication following its use.

Judging from the reports in the literature, postoperative atelectasis is the complication we have to fear. In our hospital we use a muscle relaxant in approximately 1,800 out of 6,500 surgical cases per year and have been doing so for a number of years and so far have had no such complication.
"Are there dangers in the wide use of combined solutions of muscle relaxants and other agents?"

It is probably safe to assume that this question refers primarily to the combination of a muscle relaxant with pentothal sodium, such as the Baird solution or some modification of it. I cannot answer this question fairly as I am not accustomed to using a combined solution. If the patient's need for curare was always in direct proportion to his need for the sleep-producing drug, and if the two drugs were mixed in the proper proportion, the method should be effective. But if these variables were not all in balance, it would seem that there would be danger of inadequate or excessive effects of one or the other of the drugs. I know that those are fighting words, and I know many anesthetists use the combined solutions very successfully. As I say, I haven't had that experience.

"What are the dangers of histamine-like reactions of muscle relaxants? Which muscle relaxants are less prone to produce histamine-like actions?"

The histamine-like effects that are reported to attend the administration of muscle relaxants are decrease in blood pressure, bronchospasm, and bradycardia. In the experience of our anesthesia department such complications have been so rare as to be almost nonexistent. The antihistaminics, isuprel 1 cc. of 1:50,000 or aminophylline 250-300 mg., may be used to treat such reactions. Every new muscle relaxant that is introduced is said to have less histamine-like effects than its predecessor, hence syncurine, mytolon chloride, and succinyl choline are at present reported to have the least of such effects.

"What can be considered safe practice in the use of curare?"

The muscle relaxants should not be used by an anesthetist who is not sufficiently experienced to recognize inadequate respiratory exchange and compensate for it in any way that is necessary. Cautious, individualized dosage should be strictly adhered to, and the anesthetist should always be willing to change her plan if the reaction of the patient is unfavorable. The anesthetist should remember that curare is an adjunct to anesthesia, not a substitute for it.

If good judgment and common sense are employed in the use of curare, I believe that it can make anesthesia safer and more pleasant for the patient, more satisfactory for the surgeon, and easier and more satisfying to the anesthetist.

Capt. O'Carroll: I have a question here for Dr. Holaday: "Does atropine stimulate or depress the vagus?"

Dr. Holaday: Atropine depresses transmission of nerve impulses from the postganglionic craniosacral fibers of the vagus; it does nothing but depress at that site.

It has been said that small doses of atropine can stimulate the dorsal motor nucleus of the vagus. One may see bradycardia in some individuals. It is very difficult or impossible to demonstrate this in experimental animals. But in man it has been seen. The medullary-stimulating action on the dorsal motor nucleus of the vagus is overcome very easily by the peripheral blocking action. In any event it is usually very transitory, and this action disappears shortly after the dose has been given.
Dr. Stone: There is a question: “Do you use controlled respiration in thoracic surgery?” We do in almost every case that we do, yes.

“In your hospital, do you weigh sponges during operations? How is actual blood loss calculated by this method?” We weigh sponges in some instances. We do not do it routinely. Dry sponges are weighed, and the average weight of one dry sponge is calculated. Dry sponges are used during the operation, and each sponge is weighed after its use. The total weight of the wet sponge represents the sum of the weights of the dry sponge plus the blood it contains. The weight of the dry sponge, therefore, is subtracted from the total weight, which gives the weight of the blood. One gram of blood equals about 1 cc. Thus the volume of blood loss can be calculated.

Capt. O’Carroll: Question for Miss Shupp: “Is there not a real need at present for a positive-negative mechanical respirator that will respond to the needs of the patient when controlled breathing is indicated?”

Miss Shupp: There is a respirator, the E & J, that will deliver positive and negative pressure or positive pressure without the negative pressure. This respirator also will act as a demand or semi-demand type in cases where spontaneous respiration is resumed.

I may also state that research work is going on at the present time with negative-positive pressure anesthesia.

“If you were using an explosive gas along with a respirator with a motor—especially in pulmonary operations where cyclopropane is used—is using the machine advisable?” The respirator that uses air pressure should not be, and in our experience has not proved to be, any more an explosion hazard than manual pressure on the rebreathing bag. Any respirator that uses a motor, even though it is an approved motor, would, I should think, increase the explosion hazard. And if you had a leak in the rebreathing bag and had explosive mixtures of ether or cyclopropane or ethylene, you would increase the hazard. We are very, very careful that we use rebreathing bags that have no leaks in them.

Dr. Holaday: At Presbyterian Hospital we have a Rand-Wolfe respirator, which is motor driven. We use it all the time for thoracic operations with explosive mixtures, both cyclopropane and ether. We feel it is a very reliable machine as regards explosion hazard.

Dr. Stone: “Do you feel controlled respiration has a future place in upper abdominal operations without an open chest?” Yes. If you ever used controlled respiration for an upper abdominal operation and compared the conditions with those when a patient is breathing voluntarily, you would find a vast difference in the operating conditions. In one situation you have passive breathing, and in the other you have muscles that are actively contracting. There is also a lot more activity of the upper abdominal viscera. Controlled respiration gives a very quiet, relaxed operating field.

“What do you feel is the anesthetic of choice in most cases of thoracic surgery in adults and children?” I believe, and this is my own personal opinion, that cyclopropane is probably one of the best drugs to use. But of course you can use most any drug, and I should not say it is the best method. It certainly is a method which has a great deal of merit.