PROBLEMS IN VENTILATION

A PANEL DISCUSSION

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Henry K. Beecher, M.D., Boston, Massachusetts
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This Panel Discussion was held during the annual A.S.A. meeting in Philadelphia, Pa., in November 1952. Stuart C. Cullen, M.D., Chairman, Division of Anesthesiology, State University of Iowa presided as Moderator.

Moderator Cullen: We are particularly fortunate this morning in having four men who are as well versed in the problems of ventilation as any we could gather together. They are going to present four short discussions on problems of particular interest to them and to you. After that, they will answer questions.

Dr. Julins H. Comroe, Jr., of the Department of Physiology and Pharmacology, of the Graduate School of Medicine of the University of Pennsylvania, is speaking first, on Bronchial Obstruction.

Dr. Comroe: Pulmonary ventilation, whether accomplished by natural breathing, by positive pressure applied to the airway or by negative pressure around the thorax, requires the application of certain forces to overcome certain resistances. The latter include the elastic (viscous) resistance of tissues and the resistance to the flow of air through the airways.

The elastic resistance can be measured by constructing a static pressure-volume curve. This requires inflation of the lungs by known volumes to known alveolar pressures. For example, in a deeply anesthetized or curarized patient with a cuffed endotracheal tube in place, air can be pushed into the lung, the airway clamped temporarily while the static lung pressure is measured and the lung then allowed to empty into a spirometer to measure the volume of gas that had previously been pushed into the lungs. If this is repeated at four different volumes on four successive breaths, a static pressure-volume curve can be constructed.

These volumes and pressures are more difficult to obtain accurately in an anesthetized patient who resists inflation or in an unanesthetized patient. The anesthesiologist, working as he does with deeply narcotized patients, is much better able to make these measurements
accurately and routinely than is the physiologist working with unanesthetized subjects.

In addition to measuring the elastic resistance, it is important to determine whether there is airway obstruction. The latter can be estimated readily by recording the emptying of the lungs from a known alveolar pressure into a spirometer which records on a rapidly moving drum. If there is little expiratory resistance, the lung empties quickly; if there is considerable expiratory resistance, the lung empties slowly. This simple clinical method also permits the anesthesiologist to determine what agents produce or relieve bronchial obstruction. When bronchoconstriction develops, considerable amounts of positive pressure may be required to overcome this resistance and inflate the lungs; this is particularly true if the patient has been apneic for a few breaths. In our experience, when bronchiolar obstruction is present, better ventilation is obtained with positive pressure alone than with positive and negative pressure, even though the total pressure difference is the same for the two methods.

MODERATOR CULLEN: We are fortunate in having with us also today, Dr. E. B. Brown, Jr., of the Department of Physiology of the Medical School of the University of Minnesota.

DR. BROWN: Until the last few years the physiological effects produced by administration of high concentrations of carbon dioxide to animals was largely of academic interest only. These "unphysiological" concentrations of carbon dioxide were of little clinical interest because it was assumed that a reduction of respiratory ventilation sufficient to allow alveolar air and arterial blood carbon dioxide to increase appreciably would result in an intolerable degree of hypoxia. Certainly it is true that in simple asphyxia the animal dies of hypoxia long before an accumulation of carbon dioxide to dangerous levels has taken place. Evidence of cyanosis, then, has been the danger signal to warn of inadequate ventilation.

Recently, however, a renewed interest in this problem has been awakened particularly among anesthesiologists and surgeons by the demonstrations of so-called diffusion respiration by the group of investigators at the University of Colorado, and secondly by reports from several hospitals of elevated carbon dioxide tensions occurring in patients during general anesthesia. The experiments with diffusion respiration have demonstrated that under proper conditions adequate blood oxygenation can be maintained for 30 minutes or more in dogs during complete respiratory arrest. In this period of time alveolar air carbon dioxide may increase to concentrations of 40 to 50 per cent. Several factors favor this peculiar situation in which inadequate ventilation of the alveoli produces no embarrassment of blood oxygenation but allows a marked increase in carbon dioxide tension. Some of these are the following:
1. Inadequate alveolar ventilation
2. Replacement of the nitrogen of the body with oxygen
3. Low alveolar respiratory quotient
4. High oxygen concentration in air available for movement into lungs
5. Patent airway

In the first place, alveolar air carbon dioxide concentration is a function primarily of the rate of alveolar ventilation when other factors are constant. Increasing the alveolar ventilation rate reduces alveolar carbon dioxide tension and vice versa. By removing nitrogen from the body, much higher oxygen partial pressures in the alveoli and body fluids will be present at the onset of reduced ventilation, and by having a high oxygen tension mixture present as the ambient air, any gas moved into the lungs as a result of the low respiratory quotient will help keep the alveolar oxygen tension high.

In order to visualize how these factors act to produce the end result, consider the normal situation in the lungs. Here we see oxygen diffusing across the pulmonary epithelium under a partial pressure gradient of about 60 mm. of mercury while carbon dioxide is moving in the opposite direction under a gradient of only 6 mm. of mercury. Breathing 100 per cent oxygen for a few minutes will leave the carbon dioxide situation unchanged but the gradient for oxygen will be markedly elevated—probably to a value around 600 mm. of mercury. On the basis of a functional residual volume of 2,500 cc., some 1,800 cc. of oxygen could be absorbed from the lungs before the oxygen tension in the alveoli would fall to 100 mm. of mercury if no additional ventilation took place. Such a volume plus the extra oxygen dissolved in body fluids might meet the metabolic needs of the body for 10 to 20 minutes. On the other hand delivery of 20 cc. of carbon dioxide from blood to alveolar air would increase the alveolar carbon dioxide tension to that of venous blood. This would be accomplished in a few seconds; about the length of time necessary for one breath.

With the abolition of the usual carbon dioxide gradient and the resulting decreased delivery of carbon dioxide to the lungs, accumulation of carbon dioxide in the tissues begins with an essentially simultaneous rise in carbon dioxide tension of tissues, blood, and alveolar air. When this tension has reached a value of 300 mm. of mercury the lungs of a human being would contain approximately 1 liter of carbon dioxide while several liters, the remainder of that produced during this time, would be dissolved in and combined in body fluids. During this interval of time about 5 to 10 liters of oxygen would be taken up from the lungs under a still adequate pressure head.

Under these conditions 5 to 10 volumes of oxygen are being removed from the lungs for each volume of carbon dioxide added to the alveolar air, and a volume of ambient air equal to the difference is moved into
the lungs. With this ambient air containing a high percentage of oxygen the alveolar oxygen tension is maintained at an adequate level for good blood oxygenation while the hypercapnia becomes more and more severe.

In many cases of general anesthesia in which the patient has breathed a high oxygen mixture and then been subjected to a decreasing respiratory ventilation, the stage is set for respiratory acidosis in a well oxygenated patient.

**Moderator Cullen:** I would like to introduce at this time Dr. James V. Maloney, Jr., who will speak on “Physiologic Measurements During Anesthesia.”

**Dr. Maloney:** The specialty of anesthesia is unique in that the welfare and the life of the patient depend very largely on the exercise of instantaneous and accurate clinical judgment. Anesthetic emergencies do not permit recourse to laboratory findings, to consultation with colleagues, or to the policy of “watchful waiting”; which is common elsewhere in the field of medicine. For this reason, the training of the anesthetist is concerned largely with the development of the physician’s ability to employ his own senses to evaluate a clinical situation, to make an immediate decision, and to take appropriate action without delay.

Since our physical senses are so often and so easily misled, it is inescapable that much empiricism should creep into the teaching and practice of anesthesia. An illustrative example would perhaps serve to clarify what is meant by “empiricism.” The technique of administering spinal anesthesia is a case in point. It is said that, following the introduction of procaine into the subarachnoid space, the positioning of the patient (for example, in the Trendelenberg position) should be determined by the “fixation time” of procaine in the nervous tissue of the spinal cord. Thus, we teach anesthesia residents to avoid accidental high spinal anesthesia by leaving the patient horizontal for at least several minutes. The fact of the matter, however, is that physiologists and biochemists have found little evidence to indicate that procaine is “fixed” in nervous tissue. Thus, the concept of fixation time is an empiricism which we have invented to explain certain clinical observations. Although the concept has little basis in fact, it should not be too severely criticized since it has for many years served a useful purpose in the teaching of anesthesia.

Other empiricisms which have crept into clinical practice are more pernicious. Most of us have always placed great reliance on our ability to detect reduced arterial oxygen saturation by clinical observation of the color of the patient’s skin. And yet, Dr. Comroe, in a carefully controlled study, tested the ability of a group of physicians and medical students to detect unsaturation by observing skin color. Their estimations were compared to an objective, simultaneous measure of arterial oxygen saturation. The medical students did as well as the
practiced clinicians, and the results of both groups were little more than guesswork even with moderately severe hypoxemia.

Dr. Beecher has recently developed means of objectively measuring the analgesic power of drugs. The studies by him and his co-workers have upset some of our most cherished empiricisms regarding the value of certain so-called analgesics.

The third member of this panel, Dr. Brown, demonstrated some years ago the limitations of the physician’s ability to estimate by visual observation the adequacy of a patient’s pulmonary ventilation. His studies indicate that poliomyelitis patients being treated in the

body-type respirator are usually greatly hyperventilated with resultant severe alterations in acid-base equilibrium.

It appears that our clinical judgment is sometimes susceptible to error, most often in a direction dictated by our own desires. Therefore, it is becoming increasingly necessary for the anesthetist to “calibrate” his clinical judgment by actual measurements with the modern techniques now available.

In recent years, my colleagues and I have been interested in studying the effects of positive pressure breathing on the circulation, the adequacy of pulmonary ventilation during thoracic surgery, the air flow resistance of endotracheal tubes and anesthesia machines, the
results of intra-arterial transfusion, and the circulatory and respiratory effects of certain drugs and anesthetic agents. During the course of these studies, it became progressively more apparent that our powers of clinical observation were inadequate to make an exact and scientific evaluation of our methods of anesthesia. It was therefore necessary to develop means of measuring and recording physiologic changes as they occurred in the operating room during anesthesia. Figure 1 illustrates the type of equipment which has permitted an objective evaluation of the patient’s physiologic condition during surgery. It is possible to measure continuously in various combinations for hours at a time such factors as arterial blood pressure, central venous pressure, cardiac output, electrocardiogram, changes in oxygen saturation, airway pressure, respiratory minute volume, the pneumotachogram, and the pO₂ and pCO₂ of the alveolar air. As might have been predicted, the objectivity of these measurements soon revealed many empiricisms and frank errors in the viewpoints which we had established on the basis of clinical impressions.

Many of these techniques are too complicated for general use. They therefore do not supplant, but serve only as an aid to, clinical observation. For the present time, at least, no amount of complex equipment will replace the value of sound clinical judgment. Such methodology, however, does permit the establishment of a correlation between the clinical appearance of the patient and his actual physiologic status. It is of further value in the teaching of anesthesia residents and in the objective evaluation of new methods of therapy.

The specific results of our studies will be left to the open discussion which follows these introductory remarks by the members of this panel. My own answers to questions put to the panel should be taken with some reservation, since they are the viewpoints, not of an anesthetist, but of a physiologist and surgeon.

Moderator Cullen: We have with us also today, Dr. Henry K. Beecher, with whom all of you are familiar. He will speak on the subject, “The Influence of Position on Ventilation.” Dr. Beecher is of the Department of Anesthesia, Harvard Medical School.

Dr. Beecher: One of the most important areas in which anesthetists are confronted with the carbon dioxide problem is in thoracic surgery. One of the features of thoracic surgery is abnormal position and there is a relationship between position of the patient and the excretion of carbon dioxide.

It is our thesis that the lateral position is not the optimum position for the excretion of carbon dioxide. Lobectomies, where the respiration is not assisted, result in an elevated pCO₂. If respiration is assisted, the pCO₂ tension can be kept low.

If lobectomy is done in the prone position, without assistance, we find that there is much less tendency of the pCO₂ to be elevated than there is where the lateral position is used.
Our thesis is that the lateral position is not the optimum position for the excretion of CO₂ without assistance. The lobectomies, though, do not really sharpen the focus as much as we would like to have it. The pneumonectomies do. In the pneumonectomy carried out in the lateral position with respiration not assisted, the pCO₂ becomes elevated to approximately 100 mm. of mercury, or around 14 volumes per cent of CO₂ in the alveolae. That is a very worrisome level. If pneumonectomies are done in the lateral position, with assisted respiration, we can keep that tension at something not normal, but still not too far from normal. 

In the Annals of Surgery, Dr. Eastwood and others have shown that one can even in the pneumonectomy, produce a respiratory alkalosis. The surgeon who will submit to the vigorous artificial respiration which is necessary to bring the pCO₂ down below normal levels, is a rather rare bird in our world. He doesn't want that much activity going on in the thorax, so we don't have alkalosis as a common problem in open chest surgery. 

Again, supporting our thesis that the ventilation is crippled by the lateral position with the pleura open, studies of Dr. Quinn and others in our group revealed that the pCO₂ can remain at relatively normal levels where we are dealing with pneumonectomies—a very different situation from the lobectomy—where the patients are prone or supine, that is, even without any assistance at all. We also find in the supine position it is just as good, as far as we can tell, from these limited data, as in the prone position.

There remains just one thing which I should like to mention, and that is, whether the educated hand is as good or better than the machine?

I think one can say with a great deal of conviction, that our published data indicate that the educated hand is quite adequate to overcome the impairment of CO₂ excretion, which is imposed by the lateral position.

Moderator Cullen: Dr. Beecher has brought to our attention some very pertinent questions. To put one of them in the vernacular, he says if there is an accumulation of CO₂, so what?

Another pertinent question is, how much of a compromise should there be between the surgeon who wants room to work and the patient who needs ventilation?

To initiate the discussion, we have a very cogent question; one which I think will initiate a very lively series of discussions by our panel members. The first question I will direct to Dr. Beecher. The question is:

Granted that the educated hand is efficient in producing adequate ventilation, what constitutes an "educated hand"?—and, how does one educate it?

Dr. Beecher: Well, I think there is only one way to acquire a well-
rounded education, and that is by experience. I think that a laboratory is always a very useful thing. I realize that one can’t have a good gas laboratory in every hospital in the country, but it is a very good thing to have in training centers. One can find out simply by trial and error whether or not his ventilation is being adequate, indicated by the level of pCO₂. That’s the only way I know.

**Moderator Cullen:** I think perhaps the questioner had in mind what objective means one might employ to develop this education, this “educated hand.”

**Dr. Beecher:** I don’t think there is any objective means other than what one can obtain from arterial blood samples studied in the laboratory. You find that a certain amount of ventilation will, on the average, give you a reasonable level of pCO₂. One can simply use that information. I don’t believe that one can judge it.

**Dr. Brown:** I wonder if an instrument such as Dr. Maloney described, his thermal conductivity CO₂ Meter or an instrument such as we are using (mass spectrometer) might substitute for blood gas information? In this case you would have a continuous indication of alveolar CO₂, rather than having to wait for the answer some time later.

**Moderator Cullen:** Dr. Maloney made some trips around the country and made some observations on this matter of the educated hand and the degree of ventilation, and I wonder if he has any ideas now that he might impart to the practicing anesthesiologists who do not have a gas spectrometer and the usual gas analysis apparatus.

**Dr. Maloney:** I can’t answer the question, but I can say that it is not adequate to tell whether the hand is adequately educated by visual observation of the breathing bag.

It always hurts when somebody tells us that our clinical judgment is not quite as accurate as we think it is. If one considers that the normal tidal volume might be 280 cc. per breath, the dead space 180, then the difference, 100 cc., gets to the alveolae, and may represent with a rate of 30 respirations per minute, adequate ventilation.

With 100 cc. in each breath, and with a rate of 30 times a minute, 3 liters, or 3,000 cc. of gas will reach the alveolae. That’s adequate respiration for most resting adults. If the patient’s tidal volume is only 180 cc., that only fills the dead space and is inadequate ventilation. The 100 cc. difference in tidal exchange is very important. The standard anesthesia bag holds 5,000 cc. Are there many among us who can estimate, by visual observation, 100 parts in 5,000 or 2 per cent of volume change—2 per cent by visual observation? I would be inclined to doubt it, because 47 out of 50 anesthetists whom we studied were unable to do so.

**Dr. Beecher:** I think that makes it sound almost impossibly difficult. I do believe that one can by experience learn what his patients can tolerate and won’t tolerate.
Dr. Comroe: Dr. Beecher showed that in the lateral position, with assisted respiration by the "educated hand," the mean values were a pH 7.26 and arterial pCO₂, 58 mm. of mercury. Those were closer to normal than mean values attained by others. Nevertheless, if I had a patient like that in my pulmonary section, I'd call for a doctor.

The other thing that I wanted to call to your attention is that Dr. Maloney said that, if you want to know whether you are ventilating your patient well, the tidal volume minus the dead space gives you that information.

However, the process of ventilation of the lungs is more complex than just a matter of the volume of air going in and out of the lungs. The problem of oxygenating the blood consists of a series of processes which includes ventilation, distribution and circulation. Since this discussion deals with ventilation, let me remind you that the distribution of the inspired air is of great importance. This distribution factor has been largely neglected until the last several years.

In the lateral position, the individual does not have normal distribution of gas to his alveoli, a major part of the tidal exchange may go to areas of the lung where there is not the major part of the pulmonary capillary blood flow, and a smaller part of the gas may go into part of the lung where there is a great deal of pulmonary capillary blood flow. This matter of uneven distribution of the inspired gas in relation to uneven distribution of the pulmonary capillary blood flow makes a great deal of difference in the final result.

It is true that the greater the tidal volume the better the ventilation is going to be, but you can have a situation in which most of the tidal volume is going to areas that don't have good circulation, and very little may be going to areas with better circulation. This may be the situation in patients with emphysema. They may have very serious pulmonary insufficiency even though their tidal volume is normal, even though their anatomic dead space is normal, their minute volume is normal, and their so-called alveolar ventilation is normal. They may die because of uneven distribution of gas and blood.

Moderator Cullen: I don't know that we have actually answered this question as to how we can educate the hand. What sort of observations can the ordinary clinical anesthesiologist make, who is confronted with this problem daily, to arrive at this "education" of the hand? Are we committed to use mechanical respirators rather than the "educated hand"? Are we going to be more assured with the mechanical respirator of adequate ventilation in the absence of the uneven distribution of which Dr. Comroe spoke?

Dr. Maloney: The alveolar ventilation-perfusion ratio is not affected by whether one uses mechanical squeezing of the bag or whether one uses manual squeezing of the bag.

I certainly agree with Dr. Comroe when he says that air going to unventilated areas of the lung, as in emphysema, can cause abnormal
Blood gas tensions. This is very important in the presence of an open chest with a collapsed lung. Blood can be flowing to an area that is not ventilated, and air can be ventilating an area of lung that is not perfused.

I agree with Dr. Beecher that it is important that we calibrate our clinical judgment. I don’t know as yet whether we must necessarily use some mechanical or complex device to measure ventilation, but it is certainly apparent in studies made in many clinics that all of us need to calibrate our clinical judgment against some objective means of measuring ventilation, at least occasionally.

I suspect your question, Dr. Cullen, leads to the point that I had been concerned with, along with Dr. Derrick and Dr. Whittenberger, in the use of the device we call an assistor, to give automatically assisted respiration. We found that we were able to maintain an adequate alveolar ventilation with the assistor, which we were not doing before. In theory, adequate ventilation can be maintained manually by squeezing the bag, or with the machine. From the practical point of view, I think it is extremely difficult to do it manually, because the anesthetist must take blood pressure, watch fluids, and tend to many other tasks. By visual observation I don’t believe he is able to determine adequate ventilation at all times.

Dr. Beecher, I believe, made the statement that manually assisted respiration is at least as good as, or better than, the machine respiration. I would like to make that more specific by saying that his manual respiration is better than Dr. Gibbon’s mechanical respiration, with the particular machine that Dr. Gibbon used, and the manual respiration that Dr. Beecher uses. I don’t think that we can generalize about all anesthetists, from those two observations.

It seems not worth while at this point in the development of physiology, to prove that alveolar ventilation, other things being constant, determines pCO₂. The alveolar gas equation used by physiologists demonstrates this beyond doubt.

There is much confusion in this field because we who are interested in the problem repeatedly refer to factors affecting pCO₂—depressant drugs, anesthetic agents, and position on the operating table. We should emphasize that these factors affect arterial pCO₂ only insofar as they affect pulmonary ventilation.

If the pressure in the patient’s lung is raised 10 mm. of mercury in the average normal human being, about 1,700 cc. of air will flow into the lung. As the pressure is released, the lung volume will return to its resting point. The anesthetist thus has under his control the amount of air entering the lungs. He can either effect that manually or with a mechanical device. The amount of air in turn determines the alveolar pCO₂, and the alveolar pCO₂ determines in turn arterial pCO₂, at a given ventilation perfusion ratio.

Let’s dispel the confusion and say that only one thing, under a
given set of circumstances, determines the arterial gas tensions, and that is lung ventilation.

Dr. Comroe: May I just add one word, and that is, again I want to stress the importance of the distribution of gas to various alveoli. We have a very simple method by which during a single breath of oxygen one can get a fairly quantitative idea of distribution of gas to the various alveoli in the lung and the lungs. In an individual who is quite normal, sitting or lying on his back, the record shows normal distribution. Turn the individual on his side and you will find that the normal curve is converted into a fairly abnormal one. To look at the curve without knowing that the subject is a normal individual on his side, you would say that he had definitely abnormal gas distribution as in a patient with mild pulmonary emphysema. Bearing this in mind, I think that I would be willing to say that it is unlikely that patients with any sort of thoracic operations, with any abnormal position, would have even ventilation and therefore it is rather hard to check on alveolar ventilation simply by knowing the tidal volume and dead space.

Moderator Cullen: We have another question: Will you please make some remarks on the optimal size of intratracheal catheters, both directly connected to a machine and placed under a mask?

Dr. Beecher: I would just like to say that I wish that the enthusiasts for the enormous tubes would take a bronchoscope and look at the airway after they have used them. In certain cases you will find sloughs. If you have sloughs in some cases, you must also have a good deal of lesser damage, such as elimination of ciliary action. I assume the cilia are very important in emptying the lungs. There are a certain number of cases, reported in the literature, of this type of damage. I think we sometimes forget that there is this side of the problem—not only the ventilation side.

Moderator Cullen: Do you feel that the use of the Carlens intrabronchial catheter, with collapse of the operated lung is likely to reduce the problem of CO₂ retention?

Dr. Maloney: The chief purpose of intrabronchial anesthesia, as I understand it, is the presence of lung abscess, to prevent spillage from the bad lung to the good lung. Our studies with Dr. Gaensler and Dr. Björk show the Carlens catheter to be the only bronchospirometric catheter presently manufactured which is satisfactory from a physiologic point of view for pulmonary function studies; it is my feeling that it is also the only satisfactory one for use in anesthesia. The other tubes have intolerably high air flow resistance, and in addition, they impose this burden unequally on the two lungs.

Dr. Beecher: There is a side to this question of catheters other than ventilation, important as that is. I think that we had better face the fact that with endobronchial tubes there is a problem in addition to the ventilation problem. In the first case, an endobronchial tube to function, has to be tight. How much damage that is going to
do to the mucosa, I don’t know, but I suspect it is going to do a great deal. If the cilia in a good lung are no longer functioning for a matter of hours to days, and even if there is no slough in this tight pressure, we still may be in a rather serious predicament.

We all know that in the course of surgery these tubes can fairly easily become dislodged. With an endobronchial tube into the good lung, one is not able to keep the trachea as clean as you would like to. The trachea may have filled up around this type of bronchial tube. If it becomes dislodged, there is spillage and a sudden gross contamination of the good lung. So I think these are points that one has to keep in mind in addition to the ventilation.

Moderator Cullen: I am just wondering, if in trying to arrive at some satisfactory solution to the problem we have under discussion, whether we have to elect the lesser of two evils. The determination of which is the lesser of the two evils presents a difficult problem.

In other words, if one uses smaller intratracheal catheters, there is less possibility of interference with the cilia; less possibility for tracheal granuloma, less possibility for ulceration of the trachea, and so forth. But there is also at the same time an increased possibility for resistance to ventilation and impaired ventilation, in addition to the increased expenditure of energy on the part of the patient.

I am wondering if any of the panel would like to make any remarks as to which possibility is the lesser of the two evils; the impairment to ventilation, or the possibility of damage to trachea and larynx?

I presume, Dr. Beecher, you have arrived at the conclusion that the smaller tube results in the lesser of two evils.

Dr. Beecher: I believe it is, but I am not unwilling to entertain the idea that I might be wrong. We do have blood-gas data; we do know that the ventilation is adequate with the tubes in the systems that we use. We hardly ever use larger than a size 32 French tube, occasionally a 34; but we don’t go under size 29 for adults, which is small in comparison with the size tubes used by a good many individuals.

Dr. Maloney: My interest in the past several years has been in pulmonary ventilation, so I would say that ventilation is the more important—but as soon as I have a patient with an eroded trachea, I may change my mind.

Moderator Cullen: I would like to know if the other members of the panel feel that the use of small catheters probably does not increase resistance to any significant degree?

Dr. Brown: It seems to me that if the patient can breathe through the catheter and through the mask around the catheter, the only additional resistance he should get would be simply by reduction of the cross sectional area of the trachea by virtue of the area taken up by the catheter wall. Unless you had a very thick wall catheter I shouldn’t think there would be any great increase in resistance by that system.
DR. COMROE: There is one little part of the anatomy that has not been mentioned yet, and that’s the glottis. When you put a tube through the glottis, doesn’t the glottis contract over the tube and hinder flow from the lung that is breathing around the tube?

DR. BEECHER: No, under the type of anesthesia we like I don’t think the glottis does contract.

MODERATOR CULLEN: There is another question here which I think is pertinent to a question initiated by Dr. Comroe earlier in the meeting, regarding pressure volume relationship. The question reads:

"Do you have any data concerning the dissipation of positive pressure from the breathing bag to alveoli? In other words, if 15 mm. is applied to the bag, what pressure is transmitted to the alveolus in the normal lung?"—in an unobstructed area, I presume.

DR. COMROE: I would like to answer the question as follows: If one has an unlimited volume of gas at constant pressure (this might be achieved by pushing on a large bag which has a blow-off valve so that the pressure will always attain but never exceed a certain value) and one attaches this to the airway for 1 to 2 seconds, when gas ceases to flow all the pressure in the bag will be transmitted to the alveoli. In other words, a plateau is reached and the alveolar pressure is equal to the bag pressure.

Now, in cases of bronchial obstruction, such as I discussed earlier, you notice that when you did this little trick of inflating at constant pressure, with unlimited volume either a plateau was never attained or a bag time was required. This must mean that a fair amount of the pressure was dissipated over the bronchiolar resistance. If the airway were completely occluded, either at the trachea or in the bronchi or bronchioles, then none of the pressure of the bag would get to the alveoli. If the airway was wide open, then within a very short period of time the alveolar pressure would be equal to the bag pressure, if you are operating at constant pressure and unlimited volume.

The same theory holds, of course, for dissipation of pressure across the alveolar wall. If one had an absolutely cast iron system of alveoli, none of that pressure would be transmitted to the intrapleural space. However, the alveolar walls are not cast iron and therefore there is variable transmission, depending upon the elasticity of the lung and the absorption of energy in stretching that lung.

DR. BROWN: I had the impression from textbook information that in bronchial and bronchiolar constriction, there was more difficulty in moving air out of the lungs than into the lungs and I was wondering if Dr. Comroe found that to be true.

DR. COMROE: I think that certainly the first sign of the usual types of bronchial obstruction is difficulty in expiration, and certainly in all the observations we have made on asthmatic patients, we have found that they may have little difficulty in inspiring and a great deal of difficulty in expiring. However, as the bronchial constriction becomes
more and more severe, then one gets more difficulty in inspiration, as well as expiration.

Dr. Beecher: I would like to ask Dr. Comroe if he has seen a situation where an intratracheal tube is placed too soon, or in a too lightly anesthetized patient, with the result that it is difficult to ventilate that individual. Can one guess from that that probably when one has laryngeal spasm, he also has bronchiolar spasm?  

Dr. Comroe: If there is severe trauma to an artery, there is generally peripheral spasm in the arterioles peripheral to the point of injury. Theoretically the same response may occur in the tracheobronchial system.

I feel the same as Dr. Beecher, and that is, that if there is trauma to any part of the respiratory tract, there may very well be constriction in the peripheral part; at the moment I don't have any objective information on this point.

Moderator Cullen: Another question has to do with the hazards of continuous positive pressure, utilized for inflating the lung on the occasion of closing the chest. There are many circumstances in clinical anesthesia in which it seems advisable from a surgeon's standpoint, at least, to hold the lung inflated; at the moment of closing the chest, or on those occasions when the pleural cavity is opened inadvertently, for resection, or any such procedure as that.

Dr. Maloney: The hazards of continuous positive procedure are two, one related to ventilation, the second related to circulation.

In 12 trials in 7 patients who were getting continuous positive pressure during closure of the chest or during times of continuous positive pressure to help the surgeon in segmental resection to outline the area to which the bronchus had been occluded, we measured respiratory rate, tidal volume and minute volume. When 5 cm. H₂O of continuous pressure was given, tidal volume dropped from 280 cc. to a value which is about the estimated dead space of these patients (150 cc.).

When 10 cm. H₂O continuous pressure was given—this is without any assisted respiration—tidal volume fell to an average of 50 cc. The reason for this is a simple mechanical one. When the patient inspires, the breathing bag collapses; but when he tries to expire, he must work against the elastic resistance of the bag and the ventilation decreases. This impairment of ventilation can be avoided by adding proper respiratory assistance during the inspiratory phase.

The second danger is that of circulatory depression due to positive pressure.

In by far the large majority of cases any reasonable degree of positive pressure administered in any reasonable manner, is not a danger in any patient whose circulation is adequate. We have previously demonstrated that in emergency situations positive pressure may cause profound circulatory depression and death, whereas alternating positive-negative pressure may permit successful resuscitation.
Moderator Cullen: Dr. Brown of Minnesota has been investigating retention of CO₂ by objective means and a question has been brought to our attention here. We are going to direct it to Dr. Brown, first, so that he may enumerate and discuss the clinically reliable signs of respiratory acidosis during anesthesia for those that don't have the mass spectrometer in their hands.

Dr. Brown: I'm afraid that there are no thoroughly reliable signs that you can use, as far as I know. You may find signs that arise from blood pressure and pulse rate, as the CO₂ begins to accumulate.

In work we have done in dogs, in which we have raised CO₂ to high levels, we find the blood pressure invariably rises above normal as the CO₂ accumulates and remains elevated until the CO₂ reaches levels above 40 per cent. However, whether or not these differences are sufficiently large in the clinical situations of anesthesia to be infallible, I would hesitate to say. I doubt very much if they are, as a matter of fact.

Moderator Cullen: I have one specific question here that naturally arises as a result of our use of intermittent positive pressure, positive and negative pressure, and that is, "What pressure is necessary to rupture normal adult alveoli?"

Dr. Comroe: The pressure which is required to rupture alveoli is the pressure which is required to distend them to a volume greater than maximal, such as those attained during determinations. Alveoli don't rupture because of the pressure within them; they rupture because their volume gets to be too large at which point they stretch and they tear. If you have the same pressure on the outside as you have on the inside, no matter how high you go, you don't rupture alveoli.

The next question is whether the pressure in the airway is a necessary indication of the volume of all the alveoli. If an individual has obstruction in the airways leading to certain parts of his lung, while the airways to other parts of his lungs are open, an abnormal distention of the open alveoli may occur if you inflate these lungs to the same volume that you would if all the airways were open. So the matter of uneven distribution comes into play there, again, and I think that all you can do is to set an arbitrary limit. I think that the present limit of 20 cm. of water is satisfactory. You have to say to yourself that there are probably conditions in which you can do good to the patient by going above that limit, but that when that limit is exceeded you take a certain risk of overinflating some alveoli and rupturing them.

Dr. Beechen: I tried some time ago to find out how often the ordinary population has blebs of the lungs, blebs presumably arising from old tuberculous foci. The nearest I could find out was that it is about 10 per cent of the population. I would suppose that blebs might blow out a good deal easier than a normal lung tissue. It is a point to bear in mind when we are attempting to go too much beyond a reasonable limit.
MODERATOR CULLEN: We find that fairly regularly in open chest surgery, particularly in those circumstances in which the lung is completely compressed on the open side, or partially compressed, with one of the hands retracting the heart, it takes more than 20 cm. of water to effect adequate exchange. One of the tricks for bringing this to the attention of the resident is to connect a manometer into the circuit and regularly, if they are doing it according to our clinical estimation of it, adequate ventilation may blow the water out of this manometer. I wonder if we are doing something that is wrong?

Dr. Comroe: In the ordinary lateral position, the "down" lung has more blood in it because of gravity. In other words, because of gravity, there would be greater pulmonary blood volume which may increase the resistance to inflation of that lung. The other possibility that still has to be explored is the one that Dr. Beecher asked me a little while ago, and that is whether injury to any part of the pulmonary system leads to bronchoconstriction throughout the lung. Another possibility that always has to be considered in addition is, how many surgeons are leaning on the poor man's chest?

MODERATOR CULLEN: I would like to bring into the discussion of this group also the very controversial question of the matter of assisted versus the so-called "controlled" ventilation. There are exponents of both here, I believe, and I would like to have their comments on that.

Dr. Beecher: I have been a staunch advocate over the years of assisted ventilation as opposed to controlled ventilation. I think there are several reasons for that stand.

The only data we can get, blood-gas data, seem to indicate that assisted respiration is adequate. I am always a little frightened of completely controlled respiration because it seems to me that the greatest safeguard the expert has as well as the novice, as to whether or not he is over-anesthetizing the individual is the state of the respiration. Of course that is not as good a sign under cyclopropane as ether, but still, if a man has a good volume exchange, you can be pretty sure that he is not at a dangerously deep level, at least not for a short period of time. I think once you have taken away the respiration entirely that you are perhaps asking for trouble. Now, it is much easier to take the respiration away entirely with cyclopropane than it is with ether. Many people still use ether, then turn to other means of knocking out the respiration, such as muscle relaxants. Those agents bring a problem in themselves. I steer a middle course on this, and feel that so long as assisted respiration will apparently do the job, that there is no need to go to controlled respiration.

There are exceptions to this. When our surgeons are anastomosing blood vessels in the chest, it is necessary that they have complete quiet, as quiet as you can get it, for a short period of time. We can produce apnea, a period of apnea, very easily, by the injection of a small intra-
venous dose of morphine, which will last just about the right length of time for the surgeon to carry out the anastomosis.

Moderator Cullen: "Suck and blow" type of artificial ventilation has been recommended as a means of better ventilation. In the polio patient with a tracheotomy or possibly decreased bronchial lumen, with secretions, might not better ventilation be apparent?

Dr. Malonez: The written question states that I have recommended positive-negative pressure ventilation because it gives better ventilation. I don't recommend it for that reason. It does not give better ventilation. All the mechanical respirators presently available provide hyperventilation for the ordinary cases of respiratory rest. And that is natural, because you don't want to have a respirator ready for an emergency situation if you only breathe half the population adequately. So, therefore, the mechanical respirator manufacturer has set these respirators so that they give tidal volumes of over 1,000 cc. 15 or 20 times a minute.

Dr. Comroe pointed out that during bronchoconstriction a certain number of cm. H₂O of positive pressure, do give more ventilation than the same number of cm. of water, divided between positive and negative pressure. Our point of view is that all mechanical respirators, in emergency situations in which we have studied them, give more than adequate ventilation. Then our attention was turned to emergencies. In an emergency situation, with a patient's circulation inadequate, we found that the positive pressure respirators depress the circulation, whereas the positive-negative respirators do not depress the circulation. So, our recommendation is based on the fact that both types of respirators ventilate more than adequately. We recommend positive-negative respirators because they will not cause the death of some patients by circulatory depression.

Moderator Cullen: We have overworked our Panel perhaps, but there are two final questions that were brought to our attention by Dr. Beecher's opening remarks. One is this: "Is it bad for CO₂ to accumulate, and what sort of upper limit should one set to the accumulation of CO₂?" Is it all right, in other words, for there to be a little bit of carbon dioxide accumulation?

Another question is this: "How much compromise can there be between a surgeon's demand for a relatively workable field and the anesthetist's demand for sufficient ventilation of the patient?"

Dr. Brown: We have made observations over the last two or three years, both in human beings and in dogs in our laboratory, on the influence of high CO₂, and it was during some experiments in which we had dogs breathing successively 15, 20, 25 and 30 per cent CO₂ for about 40 or 45 minutes each, that we first discovered what appeared to be a rather significant finding. This observation was the following: although the dogs with a pH of 6.7, approximately, and a pCO₂ of above 200 mm. of mercury, still had an adequate circulation and an adequate
cardiac function as indicated by their blood pressure and their ECG, some of them died in a matter of 2 to 5 minutes when we suddenly returned the animal to breathing air.

It was a purely accidental observation that we made the first two or three times; then when we followed it with continuous blood pressure recording during this interval we found that the dogs were going into a severe circulation collapse, with ventricular fibrillation. This was not really a result of the high CO₂, per se, apparently, but of the rapid loss of CO₂ or rather the rapid rise in pH and the rapid fall in pCO₂. As far as the effects of the high pCO₂ per se are concerned, we know that the pH falling below 7 does not produce cardiac block, as is described in some of our physiology texts. There is plenty of evidence to indicate that this is not the case, either in experimental animals or in human beings. There are some deleterious effects or possibly such effects that we know something about. Some of these are isolated observations.

In the first place, we find a rather severe hemococoncentration during the high CO₂. Possibly that is or is not bad. At any rate, the blood pressure is still adequate. We don’t know precisely what the cardiac output is. We are going to study some of these things next year.

As far as human beings are concerned, I think experiments in Colorado would indicate that accumulation of CO₂, along with all of the other things that go into the diffusion-respiration procedure, is not a safe procedure because in three patients treated one death occurred.

As far as the general impression is concerned, our feeling is, at the moment, that the high CO₂ per se, or the increased CO₂, with the respiratory acidosis, along with a concomitant metabolic acidosis which we have also found with some regularity, evidently is not too serious in many cases. As far as our animals are concerned, it would appear that if they survive the 2 to 15-minute interval during which cardiac arrest may occur, the animal is perfectly all right, as near as we can tell, the following day. He might have a hypotension that continues for several hours, and he may appear to be in rather severe shock, for a few hours, but the next day he usually appears to be in fairly good condition and after 48 hours the animal is in good condition.

Dr. Maloney: I would like to make a comment from the surgeon’s point of view about assisted versus controlled respiration.

In Baltimore, we do considerable surgery about the great vessels and heart. A quiet operative field is very desirable for this type of work. What makes things difficult is when the pCO₂ becomes greater than 42 or 43 mm. of mercury. The patient’s diaphragm descends in an effort to get in more air and this causes the mediastinum to swing in an extremely disturbing manner, and we have to put the sutures in the vessels “on the fly.” The patient should be ventilated to keep arterial pCO₂ normal. If you do that, the patient does not have vigorous
diaphragmatic movement stimulated by his chemoreceptors, and surgery is relatively easy.

Dr. Comroe: I think there is no doubt that one can very rapidly, by hyper- or hypo-ventilation, reverse the high or low alveolar pCO₂, and as alveolar pCO₂ is in equilibrium with arterial pCO₂, we can undoubtedly reverse quickly the arterial pCO₂.

But I am not equally sure that we can reverse rapidly high tissue CO₂ concentration, such as the situation that Dr. Brown spoke of, in which there is diffusion respiration, and the alveolar, arterial, and tissue CO₂ tensions get up around 200 mm. of mercury. I think in 3 good breaths you can get the alveolar pCO₂ way down, but I don't think you can get the tissue back to normal quite that readily.

With respect to the harmful effects of CO₂, I would like to summarize my own views on this, which are not backed up by evidence.

First of all, I think I would like to consider what happens when CO₂ accumulates in a normal unanesthetized individual. This is a problem that the submarine service is very much concerned with, since personnel might work in an enclosed submarine for quite long periods of time. There it is not a matter of life or death in the physiological sense though it might be a matter of life or death if a man's psychomotor performance is altered—for example if he turns a valve the wrong way, the submarine goes to the bottom of the ocean. There, I suppose a few per cent CO₂ would make a great deal of difference in efficiency.

When one really increases the concentration of CO₂ to which normal individuals are exposed various things happen. We know that high concentrations lead to narcosis and sometimes convulsions, and an increase in blood pressure and respiration. We also know that an abrupt withdrawal of CO₂ and substitution of air will often lead to a very abrupt lowering of diastolic blood pressure which may well go below normal levels within a matter of 5 or 10 seconds. That may either be because of the sudden reduction in alveolar CO₂, or in arterial pCO₂, acting through chemoreceptors.

There is another effect that I ought to call to your attention. Patients with pulmonary disease who develop CO₂ retention over a long period of time seem to get along pretty well. Dr. Francis Wood has a patient at the University Hospital who was kept alive for a year or two by the use of nasal oxygen. We measured his arterial pCO₂ at different times during that period of 2 years; it increased from 60 mm. of mercury to 85, and then to 140 mm. of mercury and stayed there for a year or more. During all this time he was alert, jolly and even quite witty, so he certainly was not narcotized.

Therefore, over long periods of time, there must be some compensation so that over long periods of time people can tolerate very high tensions without any gross damage.
I think we also must bear in mind that the CO₂ has a dual role, so far as the circulation is concerned; it has a direct action on the vasomotor center which tends to raise the blood pressure and a direct action on arterioles which tends to dilate and lower the blood pressure. In an individual whose vasomotor center is not functioning, CO₂ cannot affect it but can still lower blood pressure by the direct dilator action on arterioles. We have studied a patient who was not breathing, who had a pCO₂ as high as 330 mm. of mercury. That fellow wasn’t healthy; he had a blood pressure which was down to a shock level. I think that the severe acidosis and the very high CO₂ pressure probably contributed to his very low blood pressure.

In summary, in the anesthetized person who may have a high CO₂ for a matter of a couple of hours, there will probably be no serious irreversible damage any more than there is with ether. We don’t like to go round etherizing everybody just for the fun of it, but the ether effect is reversible.

I also think that the most serious effect that has been reported so far, both in our own work and that of Dr. Brown, is the very abrupt lowering of a very high CO₂ tension. I am wondering if we should not lower arterial pCO₂ a little more gradually to see if these dangers might be circumvented in that fashion.

Dr. Cullen: I think it is time that we conclude this extremely interesting session. We are very grateful, as I know all of you are, to the members of the panel for their contributions of knowledge and their making us aware of the very difficult problems associated with ventilation, and particularly during anesthesia.

We have discussed a number of things, such as the “educated hand,” the mechanical respirator, intratracheal size, pressure volume relationships, assisted versus controlled respiration, the influence of these things upon the circulation, and so forth.

We have left out a great many things that are of equal interest, but I think we have learned a great deal.

I want to take this opportunity to thank Dr. Brown, Dr. Beecher, Dr. Comroe and Dr. Maloney for their generosity and for their excellent contributions to this session.