Anesthesia for chest surgery has been and remains a challenge to the anesthetist. At the time of the earliest surgical invasion of the chest cavity, survival was considered to be the rare and fortunate outcome. Anesthesia now has progressed to the state where a mortality or even significant morbidity should be limited to those patients with the severest disease states prior to surgery. All of the principles concerned with good anesthetic management must be applied to the patients being subjected to chest surgery. I have limited this presentation, however, to those conditions and situations which are most common.

The four main areas in which anesthesia for chest surgery can be influenced are:

1. Preanesthetic Evaluation
2. Anesthetic Management
3. Postanesthetic Care

Anesthesiology recently has contributed much to improve patient safety during thoracic surgery. To accomplish this, the interests and efforts of the anesthetist have extended into the pre- and postoperative periods. Principles of pulmonary function have been established by respiratory physiologists and have been applied to the disease states by the medical chest physician or the thoracic surgeon. Anesthesiologists have joined this team to make application specifically to the patient who will be, is, or has been in the immobilized, anesthetized state.

PREANESTHETIC EVALUATION

To establish a diagnosis prior to suitable treatment, good medical practice requires that a history, a physical examination, and appropriate laboratory studies be completed. To select anesthesia for a thoracic surgery patient, similar evaluation must be applied.

Table II
Preanesthetic Evaluation

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. History</td>
<td></td>
</tr>
<tr>
<td>a. Known chest diseases</td>
<td></td>
</tr>
<tr>
<td>b. Persistent or recurrent symptoms</td>
<td></td>
</tr>
<tr>
<td>c. Occupational respiratory exposure</td>
<td></td>
</tr>
<tr>
<td>2. Physical Examination</td>
<td></td>
</tr>
<tr>
<td>a. Evidence of restricted breathing</td>
<td></td>
</tr>
<tr>
<td>b. Evidence of obstructed breathing</td>
<td></td>
</tr>
<tr>
<td>c. Evidence of infection</td>
<td></td>
</tr>
<tr>
<td>3. Laboratory Examination</td>
<td></td>
</tr>
<tr>
<td>a. Routine Hct., w.b.c.</td>
<td></td>
</tr>
<tr>
<td>b. Pulmonary function, vital capacity, timed vital capacity</td>
<td></td>
</tr>
<tr>
<td>c. Chest x-ray</td>
<td></td>
</tr>
<tr>
<td>d. Blood gas measurements, PaO₂, PaCO₂, pH, Base excess</td>
<td></td>
</tr>
</tbody>
</table>

A history of known chest disease such as recurrent upper respiratory infection, bronchitis, asthma, or pneumonia, the presence of a chronic or recurrent productive cough, and the
presence of wheezing or shortness of breath are important components of the general medical history. Occupation may point to respiratory disease because of exposure to fumes or dusts, but of greatest importance because of its frequency is the history related to the use of cigarettes. The smoker has chronic irritation and infection of both the upper and lower respiratory tree which will present a problem during and following anesthesia. The physical examination of the respiratory system is performed to determine three things: 1) Is there restricted breathing? 2) Are there signs of obstructed breathing? 3) Is there evidence of infection? Increased respiratory rate or effort either at rest or during exertion indicates obstructed or restricted breathing. Simple observation can detect the "barrel" chest of emphysema or inadequate chest movement due to scoliosis, kyphosis, or obesity. Palpation, percussion, and auscultation are necessary to diagnose parenchymal lung disease, pleural thickening, effusion, or diaphragmatic paralysis.

If a patient is unable to extinguish a match held eight inches away from the open mouth by exhaling without pursing the lips, this "match test" provides a warning of severe obstructive or restrictive disease. An obstructed airway can be suspected by observing the duration of the inspiration and expiration, as well as by auscultation of the chest to detect the wheezes accompanying it.

Signs of infection can be detected also in the physical examination. The "cough test" for bronchitis, particularly evident in chronic smokers is useful. The patient is asked to cough only once — no more. In the presence of chronic bronchitis he has an uncontrollable paroxysm of several coughs. The presence of fever, areas of consolidation, and rales or rhonchi with purulent sputum are confirmatory signs.

Of the routinely performed laboratory tests, the hematocrit and white blood count can be of particular value in detecting pulmonary disease. A high hematocrit may indicate the presence of pulmonary dysfunction and hypoxia producing a secondary compensatory polycythemia. An elevated white blood count may be supportive evidence of infection.

Of the pulmonary function tests available, I believe that the two of greatest help are the vital capacity and the timed vital capacity. A maximum inhalation is exhaled as rapidly as possible for this test and the expiratory curve recorded. A one-second timed vital capacity should be 80 percent or more of the vital capacity. Seventy percent is low normal, and 65 percent is found frequently in the aged individual. From the timed vital capacity it is possible to detect obstructive pulmonary disease, since the prolonged expiratory time will reduce the one second vital capacity below the 80 percent level. The vital capacity should be at least three times a tidal volume or normal breath. If it is reduced, it constitutes evidence of restrictive lung disease.

An additional laboratory study, the chest x-ray, confirms as well as complements the physical examination and many times demonstrates lesions not otherwise evident. Blood gas determinations obtained routinely as the baseline in the preanesthetic period as well as during and following anesthesia are probably the greatest
single contribution to the management of thoracic anesthesia that has occurred in the past several years.

In the preanesthetic evaluation these measurements are normal unless the patient has respiratory decompensation. On the other hand, he may have serious decrease in pulmonary function without alteration of blood oxygen or carbon dioxide levels. If there is a significant decrease in the arterial oxygen tension or elevation of the carbon dioxide tension, the reason must be established prior to surgery and compensation made for it.

Determination of the normal arterial oxygen tension level is difficult to establish for the preoperative patient, and it is not at all uncommon to find elderly patients without evidence of cardiac or respiratory disease with an arterial oxygen tension of 70 mm. of mercury, whereas we have considered normal levels to be between 85 and 100 mm. of mercury. The normal CO$_2$ tension is 40 mm. of mercury, and the pH is 7.42. The blood gas determinations in the preanesthetic period can only be confirmatory to other methods for evaluation. There are no absolute contraindications to needed surgery if thorough evaluation is made and the patient is well managed.$^6$

**PREANESTHETIC PREPARATION**

Table III

<table>
<thead>
<tr>
<th>Preanesthetic Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Respiratory physiotherapy</td>
</tr>
<tr>
<td>2. I.P.P.B.</td>
</tr>
<tr>
<td>3. Bronchodilators</td>
</tr>
<tr>
<td>4. Antibiotics</td>
</tr>
<tr>
<td>5. Rx of cardiac failure</td>
</tr>
<tr>
<td>6. Correction of anemia</td>
</tr>
<tr>
<td>7. Psychic preparation and medication</td>
</tr>
</tbody>
</table>

Respiratory physiotherapists find that they are much more effective in their efforts if they have had the opportunity to educate the patient prior to surgery rather than having to instruct the confused, postanesthetic, apprehensive, and uncomfortable patient.$^6$ Frequently the patient is taught to use intermittent positive pressure breathing equipment for the postoperative administration of bronchodilators and to encourage deep breaths. The patient with obstructive lung disease associated with chronic bronchitis or asthma may need a preoperative course of bronchodilators, antibiotics, and perhaps cortisone. All patients should be requested to stop smoking for as long a period as possible prior to thoracic surgery.

If the patient is anemic, transfusions, particularly of washed red blood cells, should be administered to provide hemoglobin for oxygen transport. If there is evidence of left heart failure, appropriate therapy with digitalis, diuretics, and diet should be instituted. For the patient with restrictive disease, there is often little than can be done. However, if there is pleural or peritoneal fluid, this should be removed shortly before surgery. In these patients it is particularly important that careful positioning on the operating table be accomplished so that the least amount of additional restriction to the ventilation will be added.$^2$

There has been much written about the use of narcotic analgesics in preanesthetic medication. Many now omit them. If they are to be used anyway in the postanesthetic period, their preanesthetic advantages may be utilized as well. I use them in moderation as well as attempting psychic preparation during the preanesthetic visit.

**CHOICE OF ANESTHESIA**

Choice of the anesthetic agent and technique for thoracic surgery is a
subject discussed extensively in the anesthesia literature. Regional anesthesia including local, spinal, and epidural, and all general anesthetic agents with and without endotracheal tubes, with and without assisted or controlled ventilation, have been praised or condemned.

The lastest in the series debating the use of nitrous oxide for thoracic anesthesia is an article by Boutrous in the journal, *Anesthesiology*, which was editorially discussed by Bendixen in that same issue. Boutrous has shown that with careful management of the ventilation, nitrous oxide can be administered during thoracic anesthesia without hypoxia to a majority of patients. However, Bendixen is quick to point out that some of these patients did develop a degree of hypoxia, even in these skillfully managed, carefully monitored circumstances. There is at present no conclusive evidence that one technique is absolutely superior or that one agent is completely contraindicated.

Table IV
Choice of Anesthesia
(Familiarity)
Flammability
Inhaled oxygen concentration
(FlO$_2$)
Recovery rate
Circulatory effect

The choice of the anesthetic agent and technique is of minor importance compared to the technical ability of the individual administering the anesthesia. In choosing the anesthesia factors to be considered in addition to the technical ability of the anesthetist are flammability, oxygen concentration supplies, speed of awakening with return of full muscle power, and degree of circulatory depression.

MANAGEMENT DURING ANESTHESIA

Table V
Management During Anesthesia
Oxygenation (FiO$_2$)
Carbon dioxide elimination (PaCO$_2$)
Infection prevention
Stabilization of the circulation (Q)
Surgical requirements
Monitoring

(See Figure 1)

PROBLEMS IN MAINTAINING OXYGEN HOMEOSTASIS

Table VI
Oxygenation
Inspired oxygen concentration (FiO$_2$)
Tidal volume ($V_T$)
Intrapulmonary shunt ($Q_S$)

Pulmonary pathology which may not be correctable by the preanesthetic management may cause decreased arterial oxygen tension prior to surgery. If there is an inadequate quan-

Figure 1. Alveolar ventilation and pulmonary blood flow distribution.

a. Normal
b. Obstructed ventilation
c. Restricted ventilation
d. Lateral position (alveoli and pulmonary capillaries)
e. Lateral position (lungs and heart)

The oxygen tension (PaO$_2$) of pulmonary capillary blood from unventilated alveoli is low. Overventilation of uninvolved alveoli allows adequate oxygenation of the blood from this portion of the lung but causes restriction of capillary flow and increases the functional dead space ($V_D$). The mixed pulmonary blood is low in oxygen. If the increase in dead space is not compensated by an increase in alveolar ventilation volume ($V_A$), an increase occurs in arterial carbon dioxide tension (PaCO$_2$).

The lateral position increases the portion of the cardiac output (Q) going to the dependent lung because of gravity (G). The pulmonary ventilation volume (V) is distributed more to the upper poorly-perfused lung and less to the lower well-perfused lung.
tity of lung tissue, if there is an al-
veolar capillary diffusion block, or if
there is severe obstructive or restric-
tive disease, the patient may have
hypoxemia while breathing 20 per-
cent oxygen. In combination with the
effects of preoperative pulmonary
pathology or even with normal lungs,
anesthesia and the surgical proce-
dure will interfere with oxygen
homeostasis.

Blood may be shunted through the
pulmonary circulation without being
exposed to alveolar oxygen (Figure
I b, I c). This shunting occurs when
the surgeon collapses a portion of the
lung while perfusion continues
through that segment (Figure I d). It
may also result from compression due
to the position or to elevation of the
diaphragm by the abdominal contents
(Figure I e). A diffuse atelectatic pro-
cess may develop during the course
of anesthesia as the result of the ad-
ministration of dry anesthetic gases
which cause thickening of secretions
and blocking of alveoli.22

If denitrogenation results from the
use of a high flow of oxygen or nitrous
oxide, and if there is a temporary
blocking of the alveolus, the rapid
absorption of the gases will pro-
duce atelectasis. Bendixen5,6 and
others17,18 have shown that rhythmic
ventilation of the lung without altera-
tion of the depth may lead to collapse
of lung units. This can be prevented,
of course, by periodic sustained hy-
perinflation. Despite ideal technique,
shunting may occur during the course
of anesthesia, since hyperinflation fills
the unrestricted superior lung and
gravity aids the increased perfusion of
the dependent lung (Figure I e).

A portion of the ventilation of the
anemic superior lung is wasted (dead
space increase), while blood exces-
ively perfusing the poorly ventilated,
dependent lung has been inadequately
oxygenated. Some degree of hemo-
globin desaturation results from the
mixing. Under circumstances of de-
creased pulmonary perfusion, as oc-
curs if there is shock, alveolar hyper-
inflation increases pulmonary capil-
lar resistance, decreases pulmonary
capillary perfusion, and leads to fur-
ther fall in the arterial oxygen
saturation.

PROBLEMS IN MAINTAINING
CARBON DIOXIDE
HOMEOSTASIS

Table VII
Carbon Dioxide Elimination

<table>
<thead>
<tr>
<th>Tidal volume ($\dot{V}_T$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dead space ($\dot{V}_D$)</td>
</tr>
<tr>
<td>Alveolar ventilation</td>
</tr>
<tr>
<td>($\dot{V}_T - \dot{V}_D = \dot{V}_A$)</td>
</tr>
</tbody>
</table>
| Unequal ventilation distri-
   bution |
| $\dot{V}_D / \dot{V}_T$   |

In the days when diethyl ether was
administered in a simple system, the
healthy patient was able to maintain
a normal carbon dioxide level by vol-
untary ventilation even when good
abdominal relaxation was provided.
This became no longer true as pa-
tients with severe respiratory insuffi-
ciency were anesthetized and the sur-
gery required prolonged relaxation or
an open thorax. Anesthetists con-
tributed to the problem by the use of
respiratory center depressing or para-
lyzing drugs or anesthetic incum-
brances such as large masks, sticky or
incompetent breathing valves, and
poorly-functioning carbon dioxide
absorbents.

The presence of inadequate venti-
lation resulting in carbon build-up in
the blood was recognized.3,19 To com-
pensate for these problems we use
endotracheal tubes to reduce dead space, we vigorously control ventilation, and we visually check breathing valves and soda lime for adequate function. However, subtle causes for carbon dioxide retention often escape detection.

An increase in anatomical dead space results from hyperinflation of the lung because of distention of the airways. Restriction of one portion of the lung in the open chest, (Figure I e), not only causes shunting and reduced oxygenation but it also increases the functional dead space by allowing hyperinflation of the unrestricted poorly perfused portion. Shock with decreased pulmonary capillary perfusion emphasizes this effect. Because of the subtle causes of dead space increase, it will be found that the minute volume must be increased by 50 to 100 percent above that measured in unanesthetized man to maintain the normal carbon dioxide homeostasis.

Accumulation of carbon dioxide for brief intervals may be of relatively small harm to the patient, and if the surgical procedure requires a quiet field, the use of reduced tidal volume but with high oxygen concentration may be permissible. If carbon dioxide is allowed to accumulate over extended period, pH changes and metabolic alterations may be seriously debilitating, particularly to the cardiovascular system.

### INFECTION PREVENTION

**Table VIII**

- Infection Prevention
- Sterile airway precautions
- Humidity
- Secretion blockage or removal

Another pulmonary problem created by the anesthetist is contamination of the airway. In circumstances where there is likelihood that contamination of the airway can occur from gastric contents or from infected secretions from diseased portions of the lung, special techniques must be considered in the management. Oral awake intubation under topical anesthesia may be necessary, particularly where debris may accumulate due to lower esophageal obstruction. The use of a Carlen's endobronchial catheter to isolate each lung is life-saving where secretions may drain into a dependent portion during surgery. The use of this technique should be encouraged where it is relatively indicated so that the anesthetists may become familiar with its use in those patients where it is life-saving.

Postoperative respiratory infection may be caused by contamination from anesthetic equipment. With increasing availability of gas sterilization, disposable suction equipment and improved sterile techniques by the anesthetist, this cause can be reduced. Humidification and warming of inspired gas are being studied and when available in a practical system may also reduce airway irritation during anesthesia.

### CIRCULATORY HOMEOSTASIS

**Table IX**

- Stabilization of the Circulation
- Cardiac rhythm
- Fluid, electrolytes, and blood
- Cardiac failure

During the course of thoracic anesthesia, a circulatory instability continues to be a problem but is not as mysterious or terrifying as in the early days. The concern over “vagovagal” reflexes is now less and is not considered to be a cause by itself for cardiac arrest. The prophylactic use of large intravenous doses of atropine or local anesthetic infiltration of the
hilum of the lung for blocking these reflexes are no longer routine, though atropine is needed if severe bradycardia develops. Hypoxia and hypercarbia as well as surgical alterations of the cardiac hemodynamics are the main causes for cardiac arrhythmia. Cardiac arrests during thoracic anesthesia are commonly associated with displacement or compression of the heart and great vessels.

The regulation of fluid therapy during thoracic anesthesia has changed. Restriction of fluids, enemas, and diuretics brought many patients to the operating room with a significant fluid deficit. The fear of overloading the circulation after resection of pulmonary tissue causes many surgeons to continue the dehydration of the patient into the postoperative period. This not only may cause circulatory insufficiency but also may lead to drying of tracheal secretions and increased incidence of atelectasis. It has now become evident that the judicious use of balanced salt solution replacement is necessary. It is also important to replace red blood cells for their oxygen carrying capacity, particularly if functioning lung tissue is removed. Buffering of metabolic acidosis developing during cardiopulmonary bypass may be necessary.

SURGICAL REQUIREMENTS

Table X

<table>
<thead>
<tr>
<th>Surgical Requirements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid induction</td>
</tr>
<tr>
<td>Cautery</td>
</tr>
<tr>
<td>Deflated lung</td>
</tr>
<tr>
<td>Quiet mediastinum and diaphragm</td>
</tr>
<tr>
<td>Lateral flexed position</td>
</tr>
<tr>
<td>Quiet, prompt awakening</td>
</tr>
</tbody>
</table>

The primary purpose of the anesthetic, to provide ideal operating circumstances with minimal physiological alteration, has not been discussed. A quiet surgical field can be produced by deep anesthesia, by narcotic blockade of the cough reflexes, by paralysis with curare or succinylcholine, or by a selected combination. There are conditions such as myasthenia gravis or myasthenia associated with carcinoma of the lung in which the use of relaxant drugs is probably contraindicated. There are circumstances in frail patients where deep anesthesia will produce profound shock. Patients with severe pulmonary insufficiency should inhale the highest oxygen concentration, and nitrous oxide is contraindicated. Application of one drug to produce the total effect may be less desirable than a combination in which each drug is used for its own specific effect. Therefore, the anesthetist must be acquainted with the pharmacology and clinical usefulness of each drug and their combined and interacting effects so that they can be adapted to the surgical procedure and the patient's clinical state. It is in this knowledge and its application that the skill of the individual anesthetist becomes evident.

MONITORING DURING CHEST SURGERY

Table XI

<table>
<thead>
<tr>
<th>Monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st Order</td>
</tr>
<tr>
<td>B.P. indirect</td>
</tr>
<tr>
<td>Pulse or heartbeat rate</td>
</tr>
<tr>
<td>Respiration rate</td>
</tr>
<tr>
<td>Venous pressure</td>
</tr>
<tr>
<td>2nd Order</td>
</tr>
<tr>
<td>Temperature</td>
</tr>
<tr>
<td>E.C.G.</td>
</tr>
<tr>
<td>Blood gases</td>
</tr>
<tr>
<td>Nerve stimulator</td>
</tr>
<tr>
<td>Ventilation volume</td>
</tr>
<tr>
<td>Urine flow</td>
</tr>
<tr>
<td>3rd Order</td>
</tr>
<tr>
<td>E.E.G.</td>
</tr>
<tr>
<td>Anesthetic gas concentration</td>
</tr>
<tr>
<td>Direct arterial pressure</td>
</tr>
<tr>
<td>Respiratory gas content</td>
</tr>
<tr>
<td>Hct.</td>
</tr>
<tr>
<td>Blood volume</td>
</tr>
<tr>
<td>Cardiac output</td>
</tr>
<tr>
<td>Blood electrolytes</td>
</tr>
<tr>
<td>Body weight</td>
</tr>
</tbody>
</table>
First order monitoring is that which should be applied to all patients for thoracic anesthesia. This includes the stethoscope and cuff method for estimation of the blood pressure, the continuous monitoring of the heartbeat by electronic pulse detector or stethoscope, the frequent recording of the respiratory rate, and the monitoring of venous pressure through a central venous catheter.

Second order monitoring which should be applied in indicated circumstances includes temperature recording, ECG monitoring, intermittent arterial blood gas determinations, and nerve stimulator detection of the degree of paralysis following the use of relaxant drugs. Temperature elevations occurring during anesthesia in children and young adults may be so tragic in their outcome that it behooves us to monitor the temperature in most circumstances. The measurement of the urine volume during anesthesia provides a measure of circulatory homeostasis of the patient.

Third order monitoring may be applied in very specific situations for research or as an adjunct to specific clinical situations. This includes the monitoring of electroencephalograms, anesthetic gas tensions, respiratory gas tensions, hematocrit, blood volume, blood electrolytes, or total body weight.

POSTANESTHETIC MANAGEMENT, 6, 7, 10, 11, 12, 17, 21, 24
Table XII
Postanesthesia Care
Monitoring
Awakening
Infection control
Ventilation

Postanesthetic management includes continuation of appropriate monitoring, prompt awakening, the use of humidity, secretion removal, and sterile technique to prevent infection, and assisted ventilation as needed. Prompt awakening with return of full muscle power is more likely when paralyzing relaxants, depressing narcotics, or long-lasting, highly-soluble anesthetics are kept to a minimum and promptly reversed or eliminated by non-rebreathing ventilation at the termination of surgery.

Post-thoracotomy respiratory care may be categorized in order of increasing respiratory dysfunction.

Table XIII
Post-thoracotomy Respiratory Care
Category I
Continuous 1st order monitoring
Pain relief
Humidified inhalation with p.r.n. oxygen
Prescribed “stir-up” routine
Category II
Intermittent ventilatory assistance
2nd order monitoring (e.g. blood gases and breathing measurements)
Respiratory physiotherapy
Pharyngo-tracheal suction
Bronchodilators, aerosols, antibiotics
Category III
Artificial ventilation
Physician supervision

The presence of respiratory dysfunction must be determined by a physician trained in recognition of these problems. Repeated physical examination supplemented by blood gas measurements and bedside breathing capacity tests will establish the degree of dysfunction and the effectiveness of therapy. The first order of care is adequate for most patients and is applied to those with minimal dysfunction. Frequent measurement of the blood pressure, pulse, venous pressure, temperature, respiratory rate and character, and continuous surveillance is essential.
Humidified air should be provided for inhalation, and if necessary, oxygen should be added to it to maintain an arterial oxygen tension near 100 mm. of mercury. The patient should be roused, stirred, and encouraged to cough by the nurses using a prescribed effective routine. Postanesthetic airway sanitation must be rigidly enforced, including the use of disposable sterile gloves and catheters when employing suction, gas sterilization of inhalation therapy equipment, and adequate separation of patients.

Analgesic drugs may benefit by relieving pain and splinting which causes depressed ventilation. Conversely they may be harmful by depressing the respiratory centers' response to carbon dioxide stimulation. Continuous nitrous oxide inhalation for postoperative analgesia is effective but complicated to administer and not without some risk, since it has been shown to depress white blood cell response. Long-lasting intercostal nerve block or thoracic epidural block are technically difficult to perform and maintain with sterile precautions and require continuous supervision. Most thoracotomy pain is managed therefore by the judicious use of analgesics. The intravenous administration of small doses in the early postoperative period is advocated as a means to avoid excessive depression.

The second category of respiratory care should be applied in those circumstances where ventilatory inadequacy is suspected. Second order monitoring should be instituted, including blood gas measurements. The ventilation should be assisted to the extent that the patient is able to maintain a normal arterial carbon dioxide and oxygen tension. Systemic antibiotics, bronchodilators, and, in some circumstances, aerosols, may be administered if needed by intermittent positive pressure breathing (IPPB). Acetylcysteine has been used as a liquifying agent for thickened secretions to aid their removal by cough and suctioning. Pulmonary physiotherapy should be applied to the patient and may be a continuation of therapy instituted before surgery.

A third category of postsurgical pulmonary care which has become more frequent in its application is that of postsurgical artificial ventilation. In patients where the removal of pulmonary tissue, the development of atelectasis, the weakening of the chest wall, and perhaps circulatory failure have combined to make the work of breathing more than the patient can manage, the use of artificial ventilation has proved to be life-saving. An oral endotracheal tube may be left in place or replaced nasally and ventilation maintained with minimal discomfort for hours or days. A tracheostomy is no longer a desperate procedure and is elected in many thoracotomy patients as a part of the postoperative care as a route for suction as well as for ventilation.

Artificial ventilation should be maintained until the patient can voluntarily and without excessive fatigue maintain normal oxygen and carbon dioxide blood levels. He should have a vital capacity at least three times his tidal volume. With improved knowledge of the mechanics of ventilators, the development of the mechanics of ventilators, the development of adequate humidification systems, and the use of strict sterility precautions, this form of therapy has received wider acceptance and increasingly frequent application.
THE FUTURE IN ANESTHESIA FOR THORACIC SURGERY

We can look forward to the certainty that there will be new procedures in thoracic surgery. Prosthetic replacements are coming into use for the great vessels. Lung transplant has been proposed, and animal experiments have demonstrated the feasibility. The esophagus is being replaced by other tubular organs, such as the colon. The extent and duration of surgery for cancer of thoracic structures are increasing. Thoracic surgery has been performed in hyperbaric chambers. Extracorporeal circulation has been applied more frequently, more successfully, and for pulmonary as well as cardiac lesions.

Routine application of more monitoring and measuring to avoid empiricism and permit more precise knowledge of the patient's functions will be made. This does not require new techniques but the intelligent application of knowledge already present. Mechanical ventilators for use during anesthesia have been developed to a degree of reliability and ease of application, so that in some cases they are more effective than manual compression of the breathing bag.

Probably the greatest advancement in thoracic anesthesia for the future is the utilization of an anesthesia "task force." This force consists of an anesthesiologist with special knowledge of lung pathology and respiratory care, an anesthetist skilled in various techniques of anesthesia, a laboratory technician, a respiratory physiotherapist, and intensive therapy nurses. This group will provide not only for adequate preanesthetic evaluation of the patient and care during anesthesia but will materially assist in providing increasing comfort and safety in the post-thoracotomy period.

Anesthesia for thoracic surgery has made demands on the anesthetist requiring improved knowledge and technique to meet the surgical needs. In turn, the anesthetic advancements have permitted continuing surgical development. The extension of activities into the pre- and postanesthetic periods has permitted better anesthetic management. Postoperative ventilation management and a "task force" approach to the patient's respiratory care have made significant advancements. New challenges can be expected.

BIBLIOGRAPHY