Patients with chronic obstructive pulmonary disease pose one of the most common respiratory problems encountered by anesthetists. In this article, the author discusses the anatomic and physiologic changes that impact on the anesthetic management.

As a result of air pollution, smoking, and occupational health related diseases, chronic obstructive pulmonary disease (COPD) is one of the most common respiratory problems the anesthetist will encounter. Since COPD is such a common condition, the anesthetist must understand the physiologic changes in the patient and their implications in anesthetic management. The anatomic and physiologic changes in a COPD patient, which necessitate knowledge and skill on the part of the anesthetist, are detailed in this article.

COPD defined

In the past 20 years, COPD or COLD (chronic obstructive lung disease) has been a controversial subject among medical specialists. In the 1950's, obstructive lung diseases were not differentiated from each other, and consequently, individuals suffering from all types were lumped together under the term COPD. Since then, separate disease entities within the category COPD have been identified, but the blanket term COPD is often used today.

Four subcategories of COPD have been identified: chronic bronchitis, emphysema, asthma, and small airway disease. Obstructive disease has been defined as "a disease state in which the ability to exhale is affected." The effect of these obstructive diseases is to alter airway resistance, disturb ventilation-perfusion relationships, alter pulmonary mechanics, and interfere with lung diffusion. Treatment and anesthetic management of a COPD patient depends greatly on the type of obstructive disease the patient has and its physiology and cause.

Physiology of COPD: Emphysema

The primary physiological abnormality in emphysema is alveolar fragmentation, associated with loss of lung elasticity. Emphysema patients retain secretions and develop infections easily, which causes bronchial irritation, edema, and bronchospasm. Expiratory obstruction is mechanical. The peripheral airways collapse during forced expiration, due to disruption of alveolar walls. The airway obstruction is irreversible. The increased collapsibility of the nonrigid walls of the small airways during expiration produces an increase in expiratory airflow resistance.

An early symptom of emphysema is dyspnea on exertion, becoming worse as the disease progresses. A history of cigarette smoking is strongly linked to emphysema, although the reason is still
unknown. A careful history usually reveals loss of appetite, weight loss, and cough and sputum production as a result of expiratory obstruction. In advanced emphysema, the observer will find several physical abnormalities: decreased chest expansion, use of accessory muscles of respiration, and prolonged expiration. Breath sounds are greatly decreased. The major point which separates emphysema from the other COPD’s is loss of elastic recoil of the lungs. In emphysema, the lungs “behave like highly compliant non-elastic paper bags,” which are easily inflated.

A second important abnormality is reduction in expiratory flow rates, while inspiratory flow rates tend to remain normal. This occurs because, in emphysema, the expiratory obstruction is mechanical. The bronchi retain normal caliber, but are compressed during expiration, causing expiratory obstruction and preventing normal lung deflation. This, in turn, increases the residual volume to total lung capacity ratio (RV/TLC). Hypoxemia and hypercapnia occur late in emphysema, along with right ventricular failure. Severe ventilation perfusion inequalities exist in the lung, which cause hypoxemia. Possibly due to the accumulation of bicarbonate in the cerebral-spinal fluid, the respiratory center (via the chemoreceptors) becomes less sensitive to increases in carbon dioxide tension; and hypoxia is the main stimulus for respiration.

Bronchitis

Chronic bronchitis is characterized by mucosal swelling, inflammation, and excess secretion production in the airways. Secondary problems include infection and bronchoospasm. The usual history is one of cough and sputum production, and cigarette smoking. In bronchitis, inspiration and expiration are both prolonged. Auscultation reveals wheezing noises produced by bronchial narrowing and secretions. Patients with bronchitis have normal elastic recoil, but decreased expiratory and inspiratory flow rates, and increased airway resistance. In bronchitis, hypoxemia, hypercapnia, and right ventricular failure with edema occur early.

Asthma

Asthma patients also suffer from airway obstruction, but its cause is primarily smooth muscle contraction, which may become chronic. These patients may also have a family or personal history of hay fever, allergy, eczema, or similar problems.

Normal elastic recoil is found in the asthma patient, along with decreased airway resistance. In contrast to the other COPD’s, these abnormalities are reversible with the use of bronchodilating drugs. During an asthma attack, the lungs are hyperinflated, with greatly increased RV/TLC ratios. When a patient is in full remission, the lung volumes may be perfectly normal.

Small airway disease

Small airway disease is a fourth category of COPD. A patient with this disease exhibits inflammation of the small bronchi, producing airways less than 2 mm in diameter. Most of these patients smoke cigarettes, and often have no symptoms except dyspnea on effort. These patients have normal elastic recoil and normal static compliance, but “dynamic compliance is frequency dependent; that is, the faster the patient breathes the worse (lower) the compliance becomes.”

Blood gases

When lung ventilation is inadequate, abnormalities in arterial blood gases may arise. In COPD patients, however, measurements have shown the amount of ventilation per minute to be normal or above normal. The blood gas abnormalities cannot be attributed to hypoventilation. Now, there is evidence that maldistribution of inspired gases is the major abnormality.

Even in the normal lung, ventilation is not equally distributed. Some parts of the lung may receive twice the ventilation another section receives. Ob
struction caused by COPD produces markedly uneven ventilation, with one-fourth of the lung spaces receiving nine-tenths of the ventilation. The remaining lung spaces are poorly ventilated (these spaces are called slow spaces.) The slow spaces wash out contained nitrogen very slowly when 100% oxygen is breathed, in contrast to diseased lungs which require 15 to 30 minutes, even though the total volume of ventilation per minute in COPD is greater than normal.

The composition of alveolar gas and end capillary blood depends on two factors: (1) alveolar ventilation, and (2) the amount of blood perfusing the alveolus. In areas of the lung which are poorly ventilated, a proportionate reduction in perfusion will produce normal blood gas composition. When hypoxia occurs, this produces constriction of the pulmonary vessels, thereby reducing blood flow to poorly ventilated areas. This is beneficial because it minimizes the development of low ventilation-perfusion (V/Q) ratios and consequent hypoxemia.

In COPD, the slow space is so large that its blood flow cannot be completely redistributed in proportion to ventilation. The slow space in COPD has a V/Q ratio between 0.2 and 0.4, in contrast to the normal V/Q ratio of 1.0. Consequently, a large part of the cardiac output is poorly oxygenated in the various lung spaces, this admixture of poorly oxygenated blood from the slow space with fully saturated blood from the well-ventilated spaces causes arterial unsaturation and hypoxemia.

A normal V/Q ratio (1.0) is sufficient to saturate the perfused blood almost completely (97%). A very high V/Q ratio is some parts of the lung cannot make up for poor oxygenation due to low V/Q ratios in other parts of the lung.” The low ventilation of the slow space, coupled with its large size and hence its large blood flow, causes the arterial oxygen unsaturation of COPD.”

In practical terms then hypoxemia and resultant respiratory acidosis that occurs in COPD may be defined as an oxygen saturation less than 93%, a PCO₂ above 45 mmHg, PaO₂ less than 70 mmHg, and a pH less than 7.34. Sodium bicarbonate may be used to rapidly correct respiratory acidosis. However, since the underlying mechanisms of the acidosis (regions of lung which are poorly ventilated in relation to their perfusion), is not changed and since respiratory alkalosis may further depress the activity of the respiratory centers, this agent should not be used alone to correct acid-base imbalance.

EKG changes

“The cardiovascular complications of chronic bronchitis and emphysema are manifested chiefly by the development of pulmonary hypertension and subsequent right-sided congestive heart failure.” Consequently, these changes are reflected in EKG readings. As the severity of pulmonary disease increases, progressive cardiovascular complications set in: pulmonary hypertension and subsequent right-sided cardiac hypertrophy, and then right-sided congestive heart failure.

The term cor pulmonale has been defined as “hypertrophy of the right ventricle resulting from diseases affecting the function and/or structure of the lung, except when these pulmonary alterations are the result of diseases that primarily affect the left side of the heart or of congenital heart disease.” Then, by definition, cor pulmonale does not imply right-sided failure, although it can develop with increasing severity of cor pulmonale.

Among the EKG changes noted in COPD patients are: inversion of T-waves in V-1, V-3, V-4 (associated with marked right ventricular overload plus severe hypoxemia and hypercapnia), and abnormal right axis deviation of the QRS complex in the frontal plane (as pulmonary emphysema causes depression of the diaphragm, the heart assumes a vertical position and rotates in a clockwise direction about its longitudinal axis, sometimes with displace-
also noted in the EKG is a first-degree, right-bundle branch block, the significance of which seems controversial, some authors believing that it is good evidence for right ventricular hypertrophy and others that it is due to a conduction defect in the right bundle branch.17

Pediatric COPD

Although COPD is uncommon in the pediatric patient, COPD in the adult occasionally develops as a result of childhood respiratory illnesses. Observations suggest that chronic bronchitis and emphysema in children, as well as asthma and bronchiectasis, are related to adult COPD.18

Assessment of operative risk

In assessing postoperative risk, the anesthetist must evaluate the patient's "capacity to survive and recover previous activity following the administration of anesthesia, the surgical procedure, and any associated period of immobilization."19 The risk varies with the procedure to be done, as well as with the degree of disease present in the patient. One must also weigh the risk of non-operative treatment against the risk of actually doing the procedure.

In order to prevent postoperative respiratory complications, a COPD patient must be carefully evaluated. Common screening methods, such as physical examination and chest x-ray films, are the least reliable assessment tools. The patient's history gives some information, since chronic bronchitis is defined by history alone. Cigarette smoking and exertional dyspnea are clues to the degree of COPD which may be present. Physical signs are also clues, although they may be misleading. For example, a barrel chest and apparent cyanosis bear little relation to severity of COPD. Simple screening tests of lung function usually suffice for both detection and quantitative evaluation of significant COPD. Measurement of forced vital capacity and one-second forced expiratory volume are useful data. A standard exercise will measure the patient's physical reaction to stress.

In a patient scheduled for a non-thoracic surgical procedure, an additional important test is analysis of arterial blood gases. A PaCO₂ above 45 mm of mercury shows ventilatory failure at rest, and forecasts potential post-operative problems. A PaCO₂ above 60 mm of mercury in a COPD patient implies severely impaired gas exchange and advanced disease.20

Thoracotomy in COPD patients "implies virtual certainty of further deterioration of lung function, at least temporarily, and if lung resection is involved, permanent functional deterioration must be expected."21 In these patients, regional lung function, including bronchospirometry, ventilation and perfusion scans, and angiography may be indicated.

Anesthetic management of the patient with COPD

Patients with obstructive lung conditions, such as chronic bronchitis, severe asthma, and emphysema, exhibit an increased airway resistance and a reduced dynamic compliance. Emphysema shows marked changes in lung structure, associated with an increase in tissue rigidity leading to a complete loss of elasticity. The lack of lung elasticity greatly reduces the radial support of the peripheral airways. This means that during expiration, the small bronchioles collapse before the alveoli distal to them have emptied completely, trapping air.

Thus, the intra-thoracic pressure becomes less negative, the lung volume is increased, and there is no lung-recoil when a pneumothorax is induced. The chest wall gradually becomes fixed in a position of inspiration. Consequently, the emphysema patient must work harder to ventilate his lungs because of his fixed chest wall. Increased respiratory work leads to dyspnea and limited maximal breathing capacity.

The patient with COPD, in the conscious state, can partially compensate for loss of lung elasticity by actively con-
tracting the abdominal muscles on expiration. Since these muscles are the basic muscles of expiration and anesthesia frequently relaxes them, the COPD patient is deprived of his principal means of emptying the alveoli. If spontaneous respiration is allowed under these circumstances, the level of CO₂ in the blood rises. Therefore, when marked abdominal relaxation is produced during general anesthesia for a COPD patient, respiration should be assisted or controlled.²²

Substitution of local or regional for general anesthesia is not advised (unless the procedure is minor and of short duration) because endotracheal intubation permits the best control of ventilation.²³ Many factors may interact in COPD to produce CO₂ retention during anesthesia with spontaneous ventilation. Abnormalities in ventilation secondary to increased airway resistance may worsen as a result of a decrease in functional residual capacity. Ventilation/perfusion inequalities may prolong the uptake and distribution of inhalational anesthetics, and must be taken into consideration.

High inspired oxygen concentrations are known to depress peripheral chemoreceptor drive and ventilation in patients with CO₂ retention, especially when anesthesia accentuates depression of central chemoreceptor drive. Therefore, it would seem that a 5-liter flow (3-liters N₂O, 2-liters O₂) resulting in a maximum inspired concentration (FIO₂) of 40% would be the most beneficial for the patient with COPD, and would allow spontaneous respiration and extubation at the end of surgery. Anesthesia in normal patients is associated with increased physiologic dead space. In a COPD patient, gas exchange could be seriously impaired. On the other hand, anesthesia also could have beneficial effects on COPD patients. For example, halothane "has a direct relaxant effect on bronchial muscle and reduces pulmonary resistance in constricted airways."²⁴

Studies show that significant alveolar hypoventilation may occur during an operation (even with light anesthesia) in a COPD patient whose blood gases were normal preoperatively. It has also been found that controlled or assisted ventilation is necessary during anesthesia to prevent alveolar hypoventilation.²⁵ Those medications known to release histamine (such as, morphine sulfate, curare, Surital®), and induce bronchospasm or laryngospasm should be avoided. Fluids should be carefully calculated, and excesses avoided so as to prevent pulmonary edema.

Benefits of positive end-expiratory pressure (PEEP)

Abnormal oxygenation in patients with COPD is thought to be caused by a pronounced ventilation perfusion inequality. Using PEEP on these patients during controlled ventilation theoretically could have a positive effect on PaO₂ both by improving the distribution of ventilation in relation to perfusion, and by diminishing intrapulmonary shunting. Actually, though, the slight increase in mean PaO₂ during controlled ventilation with PEEP seems of doubtful significance. If hypoxemia is due to a large intrapulmonary shunt, PEEP will be beneficial.²⁶

Conclusion

To conclude, in patients with significant COPD and impaired pulmonary function, both the anesthesia and the operation further reduce the limited respiratory reserve. In the anesthetic management of these patients, the goal is to prevent and reduce hypoventilation, hypoxemia, hypercapnia, and retention of secretions that may lead to respiratory failure.²⁷ There are specific measures which may be utilized before, during, and after the operation on a COPD patient, depending on hospital and anesthesia department policies.

Preoperatively, the severity of COPD must be recognized. A patient who smokes should be encouraged to quit, and the obese patient should undertake a program of weight reduction.
Infection must be controlled. Intermittent positive pressure breathing (IPPB) treatments may be used along with humidification and expectorants. Excessive premedication should be avoided. Narcotics tend to depress respiration. Barbiturates may induce bronchospasm and depress respiration. Tranquilizers, such as hydroxyzine and droperidol, relieve anxiety without depressing respiration.

Intraoperatively, the patient should be intubated for general anesthesia. Arterial blood gases and EKG should be monitored whenever possible, and ventilation should be controlled or assisted. Excessive intravenous fluids and blood infusions, and hypotension should be avoided. The patient should be positioned so that the mechanics of breathing are not interfered with.

Postoperatively, the endotracheal tube should be left in place until the patient demonstrates adequate ventilatory ability, and the patient should be transferred to a respiratory care unit if possible. PO₂ and PCO₂ should be monitored along with vital signs. The patient should be encouraged to cough and turn from side to side frequently every hour. Tracheobronchial suctioning and IPPB are also useful. Excessive analgesia and sedation should be avoided, and early ambulation should be promoted for patients with any form of COPD.²⁸

REFERENCES
(6) Marks, 713.
(9) Moser, 380.
(10) Moser, 381.
(11) Moser, 382.
(13) King and Briscoe, 102.
(14) Malik, 514-515.
(21) Diener, 767.
(22) Wylie and Churchill-Davidson, 70-71.
(23) Gaensler and Weisel, 190.
(25) Pietak, 164.
(28) Gaensler and Weisel, 189.

ADDITIONAL REFERENCES
(3) Holford, Fred D. and John C. Mithofer. 1973. Cardiac Arrhythmias in Hospitalized Patients with Chronic Obstructive Pulmonary


**AUTHOR**

Mark N. Gorman, CRNA, is a 1972 graduate of Oakland Community College School of Nursing, Union Lake, Michigan, and a 1976 graduate of the University Michigan School of Anesthesia, Ann Arbor, Michigan. At the time of this study, he was a student of nurse anesthesia under the direction of David R. Ely, CRNA, MA. Mr. Gorman presently serves as an anesthetist with the Kalamazoo Anesthesiology P.C., Kalamazoo, Michigan.