INTRODUCTION

Patients with chronic obstructive pulmonary disease (COPD), have an increased chance of developing complications perioperatively. The causes of these problems are usually secondary to shallow breathing, poor lung expansion, basal lung collapse and subsequent infection.

In the general surgical population, thoracic wall and upper abdominal procedures are associated with the highest risk (10-40%) of pulmonary complications *(Brown et al., 1990)*.

After upper abdominal or thoracic wall surgery lung volumes are reduced in a restrictive pattern with severely reduced inspiratory capacity (IC) and vital capacity (VC) and a smaller, but important, reduction in functional residual capacity (FRC). A decrease in FRC contributes to atelectasis and ventilation-perfusion abnormalities resulting in hypoxemia. The reductions in VC and especially IC, limit the patients' ability to cough effectively, leading to mucus retention, airways obstruction, atelectasis and an increased risk of infection *(Liu et al., 1995)*.

Because general anesthesia with tracheal intubation can elicit life-threatening bronchospasm in patients with bronchial hyperactivity; epidural anesthesia is often preferred. Thoracic epidural anesthesia (TEA) improves pulmonary dynamics after thoracic wall and upper abdominal surgery.

This occurs due to its indirect effect on the vital capacity through the analgesia produced, which permits better spontaneous ventilation

Despite sympathetic blockade, thoracic epidural anesthesia (TEA) does not increase airway obstruction and evokes only a mild respiratory motor blockade *(Groeben et al., 2002)*.

Ropivacaine is a new local anesthetic with a greater therapeutic ratio than other long-acting local anesthetics.

Ropivacaine has both anesthetic and analgesic effects. At high doses it produces surgical anesthesia, while at lower doses it produces sensory block (analgesia) with limited and non-progressive motor block (*Casati et al., 1999)*.

AIM OF THE WORK

The aim of this study is to compare the impact of general anesthesia, and thoracic epidural anesthesia (TEA) on lung function in patients with chronic obstructive pulmonary disease (COPD), undergoing thoracic wall and upper abdominal surgery.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Definitions:

Chronic obstructive pulmonary disease (COPD) is defined as a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema; the airflow obstruction is generally progressive, may be accompanied by airway hyperreactivity, and may be partially reversible.

Chronic bronchitis is defined as the presence of chronic productive cough for 3 months in each of two successive years in a patient in whom other causes of chronic cough have been excluded.

Emphysema is defined as abnormal permanent enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis. Chronic bronchitis is defined in clinical terms and emphysema in terms of anatomic pathology (*Snider et al., 1985)*.

Epidemiology of COPD

Knowledge of the prevalence of COPD is incomplete. It is estimated that approximately 14

million persons in the United States suffer from COPD - about 12.5 million from chronic bronchitis and about 1.65 million from emphysema. The estimated number of those with COPD has increased 41.5% since 1982. Estimates of diagnosed emphysema or chronic airflow obstruction in population-based studies in the United States range from 4 to 6% of adult white males and from 1 to 3% of adult white females *(Higgins, and Thorn 1990)*.

Risk factors for COPD

The primary cause of COPD is without question exposure to tobacco smoke.

Cigarette Smoking:

The major risk factor is cigarette smoking. Smokers have higher death rates for chronic bronchitis and emphysema. Cigarette smokers also have a greater annual rate of decline in FEV. Age of starting, total pack-years, and current smoking status are predictive of COPD mortality. For unknown reasons, presumably related to constitutional differences, only about 15% of cigarette smokers develop clinically significant COPD *(Sherrill, et al 1990).*

Secondhand smoke, or environmental tobacco smoke, increases the risk of respiratory infections, augments asthma symptoms, and causes a measurable reduction in pulmonary function.

Air pollution:

Although the role of air pollution in the etiology of COPD is unclear, the effect is small when compared to cigarette smoking. The use of solid fuels for cooking and heating may result in high levels of indoor air pollution and the development of COPD.

Airway hyperresponsiveness:

Airway hyperresponsiveness stipulates that patients who have nonspecific airway hyperreactivity and who smoke are at increased risk of developing COPD with an accelerated decline in lung function. Nonspecific airway hyperreactivity is inversely related to FEV_1 and may predict a decline in lung function. The possible role of airway hyperresponsiveness as a risk factor for the development of COPD in people who smoke is unclear. Moreover, bronchial hyperreactivity may result from airway inflammation observed with the development of smoking-related chronic bronchitis. *(Burrows, 1990)*

Alpha1-antitrypsin deficiency (AAT):

AAT deficiency is the only known genetic risk factor for developing COPD and accounts for less than 1% of all cases in the United States. AAT is a protease inhibitor produced by the liver that acts predominantly by inhibiting neutrophil elastase in the lungs. Severe AAT deficiency leads to premature emphysema at the average age of 53 years for nonsmokers and 40 years for smokers *(Davis and Novotny, 1989)*.

Pathophysiology:

Pathological changes in COPD occur in the large (central) airways, the small (peripheral) bronchioles, and the lung parenchyma. The pathogenic mechanisms are not clear but most likely involve diverse mechanisms. The increased number of activated polymorphonuclear leukocytes and macrophages release elastases in a manner that cannot be counteracted effectively by antiproteases, resulting in lung destruction. The primary offender has been human leukocyte elastase, with a possible synergistic role suggested for proteinase 3 and macrophage-derived matrix proteinases, cysteine proteinases, and a plasminogen activator. Additio-

nally, increased oxidative stress caused by free radicals in cigarette smoke, the oxidants released by phagocytes, and polymorphonuclear leukocytes all may lead to apoptosis or necrosis of exposed cells.

Airflow obstruction in COPD cannot be explained entirely on a structural basis; broncho-constriction is another mechanism.

A significant increase in FEV_1, after an inhaled beta-adrenergic agonist has been observed in up to one third of COPD patients during single testing sessions and in up to two thirds during serial testing *(Antoine, 1986)*.

Diagnosis of COPD

1. History

Most patients with COPD have smoked at least 20 cigarettes per day for 20 or more years before the onset of the common symptoms of cough, sputum, and dyspnea. Presentation commonly occurs in the fifth decade of life.

A productive cough or an acute chest illness is common. The cough usually is worse in the mornings and produces a small amount of colorless sputum.

Breathlessness is the most significant symptom, but it usually does not occur until the sixth decade of life. By the time the forced expiratory volume in 1 second FEV_1 has fallen to 30% of predicted, the patient is breathless after minimal exertion.

Wheezing may occur in some patients, particularly during exertion and exacerbations.

With disease progression, intervals between acute exacerbations become shorter; cyanosis and right heart failure may occur. Anorexia and weight loss often develop and suggest a worse prognosis.

2. Physical Examination:

The sensitivity of a physical evaluation for detecting mild-to-moderate COPD is relatively poor; however, the physical signs are quite specific and sensitive for severe disease. Patients with severe disease experience tachypnea and respiratory distress with simple activities.

The respiratory rate increases proportionally to disease severity. Use of accessory respiratory muscles and paradoxical indrawing of lower intercostal spaces is evident. In advanced disease, cyanosis, elevated

jugular venous pulse (JVP), and peripheral edema are observed.

Measurement of forced expiratory time (FET) maneuver is a simple bedside test; FET of more than 6 seconds indicates considerable expiratory flow obstruction (i.e., FEV_1/forced vital capacity (FVC) <50%).

Thoracic examination reveals hyperinflation (barrel chest), wheezing, diffusely decreased breath sounds, hyperresonance on percussion, and prolonged expiration. Coarse crackles beginning with inspiration may be heard, and wheezes frequently are heard on forced and unforced expiration *(Am J Respir Crit Care Med, 1995)*.

3. Lab Studies:

Secondary polycythemia due to chronic hypoxemia may develop in severe COPD or in those patients who smoke excessively. A hematocrit of more than 52% in males and more than 47% in female indicates disease.

Measure the AAT levels in all patients younger than 40 years or in those with a family history of

emphysema at an early age. If the AAT level is low, then phenotyping should be obtained.

Sputum examination:

In stable chronic bronchitis, sputum is mucoid, and macrophages are the predominant cell. With an exacerbation, sputum becomes purulent due to the presence of neutrophils. A mixture of organisms often is visible using a Gram stain.

The pathogens most frequently cultured during exacerbation are Streptococcus pneumoniae and Haemophilus influenzae.

4. Imaging Studies:

- *Chest radiograph*

Frontal and lateral chest radiographs reveal signs of hyperinflation, including a flattening of the diaphragm, increased retrosternal air space, and a long narrow heart shadow. Rapid tapering vascular shadows accompanied by hyperlucency of the lungs are signs of emphysema. With complicating pulmonary hypertension, the hilar vascular shadows are prominent, with possible right ventricular enlargement and opacity in the lower retrosternal air space.

- *Computed tomography scan*

High-resolution CT (HRCT) scan is more sensitive than the standard chest radiograph. HRCT scan is highly specific for diagnosing emphysema, and the outlined bullae are not always visible on a radiograph. This information does not alter therapy; therefore, a CT scan is not useful in the routine care of patients with COPD *(Sanders, 1991)*.

5. Pulmonary function tests

These measurements are essential for the diagnosis and assessment of the severity of disease, and they are helpful in following its progress.FEV_1 is a reproducible test and is the most common index of airflow obstruction. Lung volume measurements may document an increase in total lung capacity, functional residual capacity, and residual volume. The vital capacity decreases.

Carbon monoxide diffusing capacity is decreased in proportion to the severity of emphysema.

Arterial blood gases reveal mild-to-moderate hypoxemia without hypercapnia in the early stages. As the disease progresses, hypoxemia becomes more severe and hypercapnia supervenes. Hypercapnia

commonly is observed as the FEV_1 falls below 1L/s or 30% of the predicted value. The lung mechanics and gas exchange worsen during acute exacerbations.

As many as 30% of patients have an increase in FEV_1 by 15% or more after inhalation of a bronchodilator. However, the absence of bronchodilator response does not justify withholding therapy *(Donohue et al 2002)*.

Treatement of COPD:

- *Medical Care:*

The goal of management is to improve daily living and the quality of life by preventing symptoms and the recurrence of exacerbations by preserving optimal lung function. Once the diagnosis of COPD is established, educate the patient about the disease. Encourage the patient to participate actively in therapy.

Smoking cessation continues to be the most important therapeutic intervention. Most patients with COPD have a history of smoking or are currently smoking tobacco products. A smoking cessation plan is an essential part of a comprehensive management plan. The success rates are low because of the

addictive power of nicotine, the conditioned response to smoking-associated stimuli, and psychological problems, including depression, poor education, and forceful promotional campaigns by the tobacco industry. The process of smoking cessation must involve multiple interventions.

Oral and inhaled medications are used for patients with stable disease to reduce dyspnea and improve exercise tolerance. Most of the medications employed are directed at 4 potentially reversible causes of airflow limitation in a disease state that has largely fixed obstruction. The following factors may be present: (1) bronchial smooth muscle contraction, (2) bronchial mucosal congestion and edema, (3) airway inflammation, and (4) increased airway secretions *(Chapman, 1991)*.

Oxygen therapy

COPD commonly is associated with progressive hypoxemia. Oxygen administration reduces mortality rates in patients with advanced COPD because of the favorable effects on pulmonary hemodynamics.

Indications for long-term oxygen therapy:

Absolute

- PaO2 < 55 mm Hg or SaO2 < 88%
- In presence of cor pulmonale:
  - PaO2 55-59mmHg or SaO2 89%.
  - ECG evidence of "P"" pulmonale, hematocrit >55%, congestive heart failure

Only in specific situations:

- PaO2 >60mmHg or SaO2 >90%.
- With lung disease and other clinical needs, such as sleep apnea with nocturnal desaturation not corrected by CPAP.

If the patient meets criteria at rest, O2 should also be prescribed during sleep and exercise, appropriately titrated.

If the patient is normoxemic at rest but desaturates during exercise or sleep (Pao2 <55mmHg), O_2 should be prescribed for these indications. Also consider nasal CPAP or BiPAP ***(Dunn et al., 1991)***.