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## USEFULNESS OF CPAP IN INTENSIVE PULMONARY CARE- A REVIEW

Respiratory failure produces low arterial oxygen tension, lung failure, pump failure, respiratory muscle dysfunction, pulmonary hyperinflation and intrinsic PEEP depending on the etiological factors. Acute respiratory failure is the main indication for the mechanical ventilation and when conservative treatment fails, mechanical ventilation through an endotracheal or tracheostomy tube is given. The indications of such ventilation will be when the consciousness level of the patient deteriorates, patient goes in for a cardiac or respiratory arrest and when we see impending respiratory muscle fatigue. The goals of mechanical ventilation will be to support the blood gases and to unload respiratory muscles. The other aim is to give time to the cause of acute respiratory failure to subside. But these are not without complications. The complications may occur due to intubation itself, during or throughout the course of mechanical ventilation and even after removing the endotracheal tube or tracheostomy. The recent methods of non invasive ventilation are mainly to reduce the complications ensuring the same level of efficacy. This article is to just review the use of CPAP in an intensive pulmonary care setting and the plus or minus points regarding its use in such a setting in various disease patterns in acute care.

In COPD, it is well documented that CPAP improves the efficacy of ventilation by preventing bronchiolar collapse during expiration thus reducing the work of breathing. It has also been shown that the sensation of dyspnoea is reduced,  $PCO_2$  reduction is gradual and is

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generally well tolerated. 15 minutes of CPAP through nasal mask with 10cm H<sub>2</sub>O, in COPD, improved PaO<sub>2</sub> & reduced PCO<sub>2</sub> in acute exacerbation. It is also proven that in patients with COPD having hypercapnia and respiratory insufficiency, CPAP gave fairly good results.

Role of CPAP in asthma is mainly to produce a back distending pressure which will keep open the bronchial and bronchiolar structures, that physiologically constrict, during the expiratory cycle, inducing more emptying of the alveoli.

CPAP can be used in hypoxemic respiratory failure in intubated infants with hyaline membrane disease and intubated patients with acute lung injury. Basically, CPAP increases the abnormally low FRC with consequent effects like 1) recruitment of collapsed alveoli 2) improved PaO<sub>2</sub> 3) improved lung compliance 4) decreased the work of breathing and 5) prevention of atelectasis.

Community acquired pneumonias (CAP) lead to acute respiratory failure in 58-88% patients leading to ventilatory support. Rapid improvement with CPAP in respiratory function is noted in these cases. Intermittent application of 5-10cm H<sub>2</sub>O CPAP avoided intubation in 47% of patients with CAP and respiratory failure. The beneficial effects of CPAP in CAP are well documented.

The beneficial effects of CPAP in overcoming weaning difficulties and also to increase the ventilation during weaning,

reducing the work of breathing, improving the sensation of dyspnoea by reducing peripheral airway dynamic collapse, and use in ALI after extubation are well established.

In trauma, CPAP had very good results in post traumatic hypoxemic respiratory failure. In trauma patients, decreased FRC, compliance and restrictive defects precipitate gas exchange derangements and CPAP is ideal for treatment.

CPAP can be used in post operative respiratory failure especially in thoracic and upper abdominal surgery patients, who will have reduction in FRC, PaO<sub>2</sub> and FVC. Significant good results were documented when CPAP of 7.5 - 10 cm H<sub>2</sub>O was used early after prolonged surgery. Mask CPAP is now used to treat post operative hypoxia at many centers. It improves FVC, FRC and gas exchange in upper abdominal surgery cases. It is also shown that nasal CPAP is well tolerated after surgery and the beneficial effects are seen.

The rationale of using CPAP in ARDS is to prevent lung damage resulting from over - distension of the preserved regions of lungs and requires only 5cm H<sub>2</sub>O of CPAP. The improvement is seen within one hour and ET intubation is needed only in about 15% of patients.

CPAP can be used in patients with cardiogenic pulmonary oedema who have increased work of breathing due to reduced lung compliance and increased airway resistance and oxygen delivery to respiratory muscles is

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compromised. After treatment with CPAP of 10cm H<sub>2</sub>O, the PaO<sub>2</sub> increased, there was a decrease in respiratory rate and PaCO<sub>2</sub> and the need for mechanical ventilation and hospital costs were reduced.

It is worth noting that in AIDS, respiratory failure due to pneumocystis carinii pneumonia is common and non invasive ventilation with mask reduces the hospital cost with good results.

In conclusion, CPAP can be used in most of the ICU patients with potential benefits. CPAP in patients with acute respiratory failure work through two different mechanisms : 1) by increasing lung volumes in patients with low FRC and 2) by counterbalancing PEEPi in patients

with dynamic hyperinflation due to flow limitation. CPAP care in most cases improves oxygenation and reduces work of breathing. The advantage of CPAP over PEEP is the lower mean intrathoracic pressure and less depression of the cardiac output and it can be used non invasively. Thus CPAP can be tried in ARDS, AIDS, cardiogenic pulmonary oedema, CAP, in difficult weaning, trauma, airway diseases, asthma and also in hypoxemic respiratory failure with good results.

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## LUNG FUNCTION TESTING : (Part II)

### Interpretative strategies

The normal range gives information about the distribution of test results in the healthy population from which they were derived. It says nothing about the true positive rate, the false negative rate, or the predictive power of a positive test.

### Obstructive defect

An obstructive defect is defined as a disproportionate reduction of maximal air flow from the lung with respect to the maximal volume (vital capacity) that can be displaced from the lung. It indicates air flow limitation and implies airway narrowing during expiration. The earliest change is slowing in the terminal portion of the spirogram reflected in a proportionally greater

reduction in FEF<sub>75%</sub> or in FEF<sub>25-75%</sub> than in FEV<sub>1</sub>.

### Restrictive Defect

A restrictive defect is characterized physiologically by a reduction in total lung capacity. Vital capacity is reduced, FEV<sub>1</sub>/FVC is normal or increased.

### Bronchodilator response

20-30% of responsive subjects will respond to one type of agent but not to another. Also, the correlation between bronchoconstriction and bronchodilator responses is imperfect. There is a tendency for the calculated response to increase with the decreasing baseline VC or FEV<sub>1</sub>.

There is no consensus on how a

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bronchodilator response should be expressed. The three most common methods are:

1. Percent of initial spirometric value
2. Percent of initial predicted baseline value
3. Absolute change

Of these, the percent of initial predicted baseline value is most accepted. It requires at least 12-15% increase in FEV<sub>1</sub> from baseline value to define a meaningful response. Increments less than 8% or less than 150 ml are within measurement variability. It is also important to remember that a patient may respond to long term bronchodilator therapy even though a bronchodilator response is not seen in a single laboratory testing session.

### **Assessing severity**

Severity scores are most appropriately derived from studies that relate lung function test values to independent indices of performance such as the ability to work and function in daily life, morbidity and prognosis. The severity of the spirometric abnormality is based on the actual or percent predicted FEV<sub>1</sub> in the case of obstructive disorders or on the VC in nonobstructive disorders. Physicians should view arbitrary scoring systems with caution.

### **Changes in spirometry over time**

To interpret changes in spirometric values over time, reliance should be placed on FEV<sub>1</sub> and VC. It is important to decide whether a change is real or only a result of test variability. It is more likely that a real change has occurred when series of tests show a consistent trend. For FVC and VC in healthy subjects, within day

changes of  $\geq 5\%$ , between week changes of  $\geq 11-12\%$  and yearly changes of  $\geq 15\%$  are clinically important.

### **Recommendations for interpretation**

1. Clinical questions should be included in the requisition.
2. Interpreters should be conservative in suggesting a specific diagnosis based only on pulmonary function abnormalities.
3. Borderline normal values should be interpreted with caution.
4. The first step in interpretation is to evaluate and comment on the quality of the tests.
5. The number of test indices used in interpretation should be limited to avoid an excessive number of false positive results.
6. The primary guides for spirometry evaluation should be VC, FEV<sub>1</sub> and FEV<sub>1</sub>/VC.

### **Concerning airway obstruction**

7. FEV<sub>1</sub>/VC should be the primary guide for distinguishing obstructive from nonobstructive patterns.
8. Instantaneous or midflows may be used to confirm the presence of airway obstruction in the presence of borderline FEV<sub>1</sub>/VC.
9. FEF<sub>25-75%</sub> and instantaneous flows should not be used to diagnose small airway disease in individual patients.
10. Low FEV<sub>1</sub>/VC ratio, greater than average FEV<sub>1</sub> and VC may occur in healthy individuals.

11. Severity of airway obstruction should be based on FEV<sub>1</sub> rather than FEV<sub>1</sub>/VC.
12. Abnormalities in instantaneous flows and FEF<sub>25-75%</sub> should not be graded as to severity when FEV<sub>1</sub> and FEV<sub>1</sub>/VC are within normal range.

### Concerning bronchodilator response

13. VC and FEV<sub>1</sub> are primary indices to judge response. Total expiratory time should be considered when using FVC to assess bronchodilator response.
14. A 12% increase, calculated from pre bronchodilator value, and a 200 ml increase in either FVC or FEV<sub>1</sub> are reasonable criteria for a positive bronchodilator response in adults.

15. FEF<sub>25-75%</sub> and instantaneous flows are to be considered secondarily.
16. Ratios such as FEV<sub>1</sub>/VC should not be used to judge response.

### Concerning restriction

17. The diagnosis is based on reduced total lung capacity. Reduced VC in the presence of a normal FEV<sub>1</sub>/VC may be used to suggest but not diagnose restriction.
18. The severity of restriction should be based on total lung capacity. If VC is used to infer the presence of restriction, severity may be based on VC.

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## MUCOCILIARY DYSFUNCTION (PART - I)

### Definition

Any defect in the ciliary and secretory component of mucociliary interaction that disrupts the normal physical, chemical and histological defence functions of the airway epithelium.

### Functions of mucociliary apparatus

1. Mechanical barrier : Traps particulates in inhaled air in the surface liquid covering the airway epithelium and clearing them from the tracheobronchial tree by ciliary action.
2. Chemical screen : Airway mucus has anti-oxidant properties
3. Biological barrier : Interacts with micro-

organisms and luminal inflammatory cells, thereby preventing them from adhering to and migrating through airway epithelium.

### Ciliary epithelium

- \* Extends from proximal trachea to terminal bronchioles.
- \* Contains ciliated columnar cells and goblet secretory cells
- \* Mucous velocity 4-20 mm/min.

This can be measured by two methods :

- a. Teflon disc method - Teflon discs are deposited in airways and viewed directly with the bronchoscope.

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- b. Radioaerosol technique -  $^{99}\text{Tc}$  labeled particles are inhaled and then viewed externally by gamma camera.

Mucous velocity is highest in the trachea and decreases down the tracheobronchial tree.

### **Surface mucous**

- \* 10-100 ml liquid is transported through trachea each day
- \* Contains : i) IgA- forms immunological barrier, ii) Lysozyme - bactericidal, iii) Lactoferrin - bactericidal to Gram negative bacteria by trapping iron ions.
- \* Contains periciliary fluid layer (sol phase) and mucus layer (gel phase)

### **Mucociliary dysfunction in airway diseases :**

#### **1. Asthma**

Asthma - Inflammatory mediators (PAF, hydrogen peroxide, protease, eosinophilic major protein, neutrophil peroxidase) cause ciliary inhibition and mucus secretion.

Mucociliary clearance is impaired during severe exacerbations of asthma and improves markedly during convalescence. Patients with less severe asthma also have impaired clearance, notably in central airways. In milder asthma, the impairment is more subtle, again mainly in central airways. Bronchial clearance may actually increase in these patients.

#### **2. Bronchitis**

Infection worsens mucociliary clearance by direct cytotoxic effect and epithelial shedding. Bacterial products and Inflammatory mediators impair mucociliary clearance.

Acute exposure to cigarette smoke reduces ciliary activity in vitro. But in vivo studies are contradictory, suggesting that short term exposure to cigarette smoke increases, has no effect or decreases mucociliary clearance. Cigarette smoke consists of a gas phase and particulate phase, both of which are potentially ciliotoxic.

Chronic exposure to cigarette smoke impairs mucociliary clearance in asymptomatic smokers, smokers with productive cough or airflow limitation.

#### **3. Cystic fibrosis and Bronchiectasis**

Due to absorption of  $\text{Na}^+$  from the secretions, the viscosity of sputum is increased. Increased volume of secretions and altered rheology of sputum causes slower mucociliary clearance.

#### **4. Primary ciliary dyskinesia**

Commonest defect is absence of outer diene arms.

Mucociliary clearance is greatly reduced and mucus is cleared from the airway primarily by cough.

### **Consequences of mucociliary dysfunction**

1. Productive cough
2. Respiratory infection
3. Airflow obstruction

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## LTRAs AND ASTHMA

Leukotriene - receptor antagonists (LTRAs) represent the first new therapeutic class to be introduced for the treatment of asthma in the last 25 years. Cysteinyl-leukotrienes ( $LTC_4$ ,  $LTD_4$  and  $LTE_4$ ) are derivatives of arachidonic acid synthesized by 5-lipoxygenase (5-LO) pathway in the mast cells, eosinophils, macrophages and basophils. They act at Cys-LT receptors and are potent mediators of asthma. LTRAs like zafirlukast, montelukast and pranlukast have been extensively evaluated in clinical trials in patients with persistent asthma. The exact role of LTRAs in asthma treatment guidelines is being evaluated.

Compliance is an important issue in asthma management and there are three advantages when using LTRAs : they are orally effective, well tolerated and do not cause steroid-phobia. LTRAs are effective in children also where compliance to inhalers is rather poor due to technical reasons. LTRAs also reduce rhino-conjunctival symptoms.

LTRAs are indicated in patients with mild to moderate persistent asthma whose symptoms are not well controlled with existing medication (Add-on therapy). In some countries, LTRAs have been recommended for certain mild asthmatics (instead of inhaled steroids). Clinical evidence also suggest that LTRAs can benefit patients with severe persistent asthma. LTRAs are particularly effective in patients with aspirin intolerant asthma and exercise-induced asthma.

LTRAs may have an additive effect with inhaled  $B_2$  agonists on lung function. In mild

asthmatics, zafirlukast is as effective as cromolyn sodium and theophylline (with better compliance and fewer side effects). There is evidence that the combination of LTRAs and inhaled steroids is more effective than either drug alone, and that adding LTRAs to the treatment regimen is better than increasing the dose of inhaled steroids. Clear evidence is available which shows that LTRAs reduce the need for short courses of oral prednisolone. The combination of inhaled steroids and LTRAs may be particularly effective because steroids reduce the number of Cysteinyl - LT- generating leucocytes infiltrating the lung and LTRAs block the activity of Cys- LT by resident and infiltrating cells.

An important aspect of LTRA therapy is variability in individual response to treatment- some patients do not respond, while others can improve dramatically, Nearly 35% experience little benefit, 50% experience significant clinical improvement and other 15% experience dramatic clinical improvement.

In view of the above mentioned facts, it is expected that the exact place of LTRAs in step-wise management of asthma will become clear very soon.

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