Part II: Respiratory Failure

Chapter 19: Acute Respiratory Failure in Chronic Obstructive Airways Disease

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The term chronic obstructive airways disease (COAD) usually applies to chronic bronchitis and/or emphysema, but may also apply to largely irreversible airflow obstruction due to asthma, bronchiectasis or cystic fibrosis. Respiratory reserve is reduced, and superimposed acute respiratory failure (ARF) has significant morbidity and mortality. However, most precipitating factors of ARF are reversible, and the prognosis of patients who recover are good.

Aetiology

A number of factors may precipitate ARF in patients with COAD, including:

1. Acute Infection

This may be acute bronchitis or pneumonia. Acute bronchitis is precipitated by viral infection (with secondary bacterial invasion) or by primary bacterial infection. *S. pneumoniae* and *H. influenzae* are most common, but *S. viridans* and *Branhamella catarrhalis* may be found. Pneumonia is most commonly caused by *S. pneumonia* and *H. influenzae,* but mycoplasma, legionella and viral pneumonia are occasional causes.

2. Sputum Retention

Sputum retention may complicate COAD or ARF. Common causes are surgery, trauma, and depressed conscious level. The primary problem is failure of coughing to remove the increased secretion in COAD.

3. Bronchospasm

The relationship between bronchospasm in COAD and asthma is not always clear. Asthma itself may cause or coexist with COAD. In some patients, increased bronchial reactivity may result from COAD. Precipitants of bronchospasm are similat to those of asthma.

4. Pneumothorax

The risk of pneumothorax is increased in COAD. ARF may be rapidly precipitated, depending on the size of the pneumothorax and the severity of the underlying lung disease.

5. Bullae

Large lung bullae may expand and precipitate ARF in a manner similar to pneumothorax. Occasionally, subpleural bullae may be mistaken for pneumothoraces.

6. Left Ventricular (LV) Failure

LV failure may result from coexisting ischaemic heart disease, fluid overload or biventricular failure secondary to cor pulmonale. COAD lungs are very sensitive to LV failure, and ARF may be precipitated even in the absence of obvious pulmonary oedema on chest X-ray.

7. Pulmonary Embolism

Pulmonary embolism is not commonly recognized as a cause of ARF in COAD, although it is found in 20-50% of autopsies. If it is not a primary cause of ARF, it may complicate other causes. It is often hard to diagnose, because of the pre-existing lung disease and the numerous possible causes of ARF in COAD. Interpretation of lung scans is difficult when COAD is present. A high index of suspicion is required, and pulmonary angiography may be necessary.

8. Uncontrolled Oxygen Administration

Uncontrolled oxygen therapy may precipitate acute hypercarbia in a small proportion of patients with more severe COAD, especially those with chronic hypercarbia. The consequent removal of the hypoxic drive to respiration is only a partial explanation. Major factors appear to be dissociation of CO_2 from haemoglobin by oxygen (Haldane effect), and worsening of ventilation-perfusion (V/Q) mismatch. The latter is probably due to reduction of hypoxic vasoconstriction in areas of shunt, allowing more CO_2 rich venous blood into the arterial circulation.

9. Sedation

Over-sedation can readily precipitate hypoventilation in severe COAD. Although this should reverse when the sedative effect wears off, sputum retention beforehand may result in persistent respiratory failure.

10. Endstage Lung Disease

Most precipitants of ARF in COAD are reversible, but COAD itself is commonly progressive, and may ultimately lead to death. The patient presents with apparent ARF from absent or trivial precipitating factors, and a long history of severe lifestyle limitation and refractory dyspnoea at rest.

Pathophysiology

Factors obstructing airflow in COAD include mucosal oedema and hypertrophy, secretions, bronchospasm, airway tortuosity and airflow turbulence, and loss of lung elastic recoil (due to loss of lung elastin and alveolar surface tension from alveolar wall destruction). Loss of lung elastic recoil leads to decreased expiratory airflow, because alveolar pressure (which drives expiratory airflow) and intraluminal airway pressure (which distends small airways during respiration) are reduced. Airflow obstruction results in prolonged expiration,

pulmonary hyperinflation, increased work of breathing and the sensation of dyspnoea, all worsened during an exacerbation of COAD.

The pulmonary circulation is also abnormal in COAD. Alveolar distortion and destruction results in capillary bed loss, and hypoxia causes pulmonary artery vasoconstriction. These lead to pulmonary hypertension, secondary vascular changes, and ultimately, cor pulmonale. Increased hypoxia during ARF increases pulmonary artery pressure and may precipitate acute right heart failure.

The combination of airway obstruction, pulmonary parenchymal disease and circulatory disturbance leads to extensive V/Q mismatch. Underventilated lung areas act as partial or complete shunts. This results in arterial hypoxaemia, which when chronic, may cause secondary polycythaemia and increase pulmonary hypertension. Under-perfused or over-ventilated areas increase dead space. Thus, as a result of severe V/Q mismatch, ventilatory requirements for normocarbia are increased. Increased minute ventilation results in further increases in the work of breathing.

Since expiration is incomplete in airflow obstruction, this, with increased minute ventilation, result in permanent dynamic elevation of the functional residual capacity (FRC) or pulmonary hyperinflation. As lung volume is increased, the respiratory muscles (diaphragmatic and intercostal) become less efficient as a result of shortened fibre length and mechanical disadvantage, and the work of breathing is further increased. When the compromised inspiratory muscle capacity fails to meet the increased ventilatory requirements, chronic hypercarbia ensues.

Chronic hypercarbia is uncommon in COAD, and tends to occur late in the disease course, associated with renal acid-base compensation. It is usually seen in COAD dominated by chronic bronchitis with an FEV1 under 1 L, and is associated with polycythaemia, cor pulmonarle, and further CO_2 retention with uncontrolled oxygen administration. However, hypercarbic respiratory failure is readily precipitated by increased lung shunt. Patients with COAD have a poor response to increasing shunt. They have a limited capacity to increase minute ventilation, which may be decreased by the respiratory failure itself.

Clinical Features

ARF in COAD can present with two distinct clinical patterns.

1. Increasing Dyspnoea (the "can't breath" pattern)

This is more common, and results from impairment of airflow and gas exchange with no impaired respiratory drive. There is inability to achieve adequate ventilation despite a maximum ventilatory effort. Dyspnoea is accompanied by increased sputum, cough, wheezing, and reduced exercise tolerance. Hyperpnoea, use of accessory respiratory muscles, and pursed-lip breathing are usually present. Cyanosis and pulmonary hyperinflation may be evident. Rhonchi, prolonged expiration, and expiratory wheeze are usually auscultated. Signs of consolidation, usually at the lung bases, may be present if pneumonia or sputum retention are precipitating events. A loud pulmonary component of the second heart sound, sight ventricular (RV) heave (if not negated by pulmonary hyperinflation), elevated jugular venous pressure

and peripheral oedema indicate pulmonary hypertension and cor pulmonale. In severe cases, right heart failure may be accompanied by tricuspid incompetence.

Respiratory muscle fatigue is initially manifested by impaited coughing and clearance of secretion, reduced tidal volume, and increasing respiratory rate. As fatigue progresses, abnormal breatinhing patterns may emerge, i.e. abdominal, alternating with rib cage breathing (respiratory alternans) and paradoxical indrawing of the abdomen on inspiration (abdominal paradox). Both fatigue and the consequent CO_2 retention may obtund conscious level. Features of CO_2 retention may be evident - warm dilated periphery, bounding pulse and sweating. Fatigue, abnormal breathing patterns and increasing CO_2 retention herald impending respiratory collapse.

LV failure or fluid overload may be indicated by cardiomegaly and upper lobe venous diversion on chest X-ray, audible chest crepitations, and a history of ischaemic heart disease. However, COAD can effectively mask the symptoms and signs of ischaemic heart disease and LV failure. Conversely, chest crepitations may occur in the absence of LV failure.

Pneumothorax or pulmonary embolism are suggested by sudden marked increases in dyspnoea. Asymmetrical respiration, abnormal percussion note and breath sounds, and tracheal and apex beat displacement suggest pneumothorax. Haemoptysis, pleuritic chest pain, pleural rub, an opacity on chest X-ray and evidence of a deep vein thrombosis in the legs point to pulmonary embolism.

2. Decreasing Dyspnoea (the "won't breath" pattern)

This less common form of ARF in COAD occurs when conscious level is depressed by drugs, illness, or excess oxygen administration. It is mimicked in the advanced stages of the "can't breath" group, when respiratory muscle fatigue and CO_2 narcosis have taken over. The primary problem is increasing hypercarbia and respiratory acidosis, with no increase in dyspnoea. A reduced central respiratory drive underlies the absence of increasing dyspnoea in response to a rising CO_2. However, an increased V/Q mismatch also contributes to the hypercarbia.

The sole initial feature may be the blood gas abnormalities; increased $PaCO_2$, acidaemia, and small fall in PaO_2. As failure worsens, clinical features become apparent. These include reduced respiration and dyspnoea, cyanosis, sputum retention, decreasing conscious level, and signs of hypercarbia. This is a more subtle presentation and a high index of suspicion and early blood gas measurement is required in these patients.

"Blue Bloaters" and "Pink Puffers"

The value of labelling patients as "blue bloaters" (mainly chronic bronchitis) and "pink puffers" (mainly emphysema) is uncertain, as the two disease processes usually co-exist, and the principles of management are similar. However, categorization of a patient, if clearly applicable, can be useful as the two types may have different clinical patterns and prognoses.

"Blue bloaters" - COAD patients dominated by chronic bronchitis have chronic cough, large sputum production, wheeze, fluctuating levels of dyspnoea, and severe V/Q mismatch.

As a result, such patients are more prone to hypercarbia, CO_2 retention with oxygen administration, cyanosis, secondary polycythaemia, pulmonary hypertension and cor pulmonale. The tendency to cyanosis and peripheral oedema has resulted in the term "blue bloaters". The overall couse is often downhill, but recovery usually occurs from infection induced exacerbations.

"Pink Puffers" - Patients with COAD dominated by emphysema have a more constant level of dyspnoea with less cough, sputum, and wheeze. This disease process primarily affects pulmonary parenchyma with alveolar wall loss and little primary airway involvement. V/Q relationships tend to be better preserved, and less shunting occurs. Work of breathing and ventilatory requirements are increased, but with less shunting, increased respiratory effort (puffing) can normalize blood gases - hence the term "pink puffer". The course is characterized by gradually increasing dyspnoea until near terminal stages of the disease are reached. Thus, patients with emphysema presenting with respiratory failure are often closer to end-stage lung disease than those with chronic bronchitis.

Diagnosis and Assessment

Diagnosis of COAD

The diagnosis of COAD is usually established prior to patient presentation with respiratory failure. It is based on the following:

1. History of smoking or other causative factors.

2. Chronic cough and sputum production.

3. Longstanding dyspnoea with or without wheeze.

4. Lung function tests demonstrating largely irreversible airflow obstruction.

(a) Spirometry - shows a reduced FEV1/forced vital capacity (VC) ratio compared with predicted values. These values may improve to a small degree but not normalize with bronchodilators. VC is initially normal and becomes reduced later in the course of the disease, but to a lesser degree than the FEV1.

(b) Flow-volume curves - demonstrate reduced expiratory flow rates at various lung volumes.

(c) Lung volumes - measured by helium dilution or plethysmography show elevated total lung volume, FRC, and residual volume.

(d) Carbon monoxide uptake - is a measurement of alveolar surface area, and its reduction approximates the amount of emphysema present. It may be normal or near normal in pure chronic bronchitis.

(e) Bronchial reactivity tests - such as histamine, methacholine, cold air or antigen challenge are occasionally used to see if there is a component of "asthma" present. A positive test suggests benefit from aggressive bronchodilator or steroid therapy.

Diagnosis of Respiratory Failure

An exacerbation of COAD is usually clinically obvious. However, it is important to diagnose the extent of deterioration. The following assessments may be useful:

1. Arterial Blood Gases (ABGs)

ABGs are mandatory to assess hypoxia, hypercarbia and acid-base status. The large majority of COAD patients are not chronically hypercarbic and will only develop hypercarbia when compensatory mechanisms are exhausted. Such patients will have normal bicarbonate levels, and the occurrence of hypercarbia signals possible respiratory collapse. Chronic hypercarbia may be recognized by a high bicarbonate level (over 30 mmol/L) and a base excess over 4 mmol/L (indicating renal compensation). However, other causes of raised serum bicarbonate need to be excluded (i.e. diuretic and high-dose steroid therapy or gastric fluid loss). Renal compensation will increase the serum bicarbonate by approximately 4 mmol/L for each 10 mmHg (1.33 kPa) of chronic PCO_2 rise above 40 mmHg (5.3 kPa).

2. Spirometry

This should be performed if possible, as it will indicate the severity of illness and deterioration, and provide a baseline measurement for subsequent assessments.

3. Chest X-ray

Chest X-ray is mandatory to diagnose or exclude pneumothorax, lobar or segmental collapse, pneumonia or obvious LV failure. The film will commonly show hyperinflated lung fields, low flattened diaphragms, and paucity of lung markings. Pulmonary hypertension is manifest by enlarged proximal and attenuated distal vascular markings, and by RV and atrial enlargement. Lung bullae may be evident.

4. Electrocardiogram

ECG is commonly normal, but may show features of right atrial or RV hypertrophy and RV strain, including P pulmonale, right axis deviation, dominant R waves in V1-2, right bundle branch block, and ST depression and T wave flattening/inversion in V1-3. These changes may be chronic or develop acutely. The ECG may also show co-existant ischaemic heart disease.

5. Sputum Microscopy and Culture

Although expectorated samples do not always reliably identify lower respiratory pathogens, sputum samples in COAD will usually culture the causative organism and guide antibiotic therapy.

6. Full Blood Count

Full blood examination may show polycythaemia. An elevated white cell count may indicate infection.

7. Theophylline Assays

Serum theophylline levels should be performed on patients receiving theophylline derivatives.

Differential Diagnoses

It is important to recognize or exclude less common causes of ARF in COAD which require specific therapy.

1. Left Ventricular Failure

LV failure may be clinically subtle. Minor pulmonary oedema and pulmonary venous congestion may precipitate respiratory failure in a severely compromised lung without clinical or radiological features of pulmonary oedema. Comparison with previous chest X-rays is important.

2. Pulmonary Embolism (PE)

PE should be suspected in any unexplained deterioration, especially where factors predisposing to deep venous thrombosis exist. Investigative lung scans and anticoagulant therapy follow conventional practice.

3. Pneumothorax

This may be difficult to detect clinically but is usually obvious on chest X-ray. However, careful X-ray inspection is required. A lung edge may not always be visible, and with a small pneumothorax, asymmetry of lung markings or apical density may be the only clues. When dount exists, repeat chest X-rays with both inspiratory and expiratory views should be obtained.

4. Upper Airway Obstruction

Upper airway obstruction from any cause may mask the usual presenting symptoms of ARF. Unexplained deterioration, stridor, or voice alteration are important clues. Upper airway X-rays and tomograms, inspiratory/expiratory spirometry, flow-volume graphs, and direct or indirect laryngoscopy are performed when indicated.

Management

A. Conservative Measures

1. Oxygen

Most patients with COAD will not develop CO_2 retention from oxygen administration. It is more common in patients with severe COAD who are in respiratory failure. However, reversal of hypoxia is important, and oxygen should not be withheld if hypercarbia is present, nor withdrawn if it worsens. Oxygen is given initially by low-flow intranasal cannulae of 24% or 28% venturi mask. Oxygen therapy is best controlled by titrating delivery to achieve a saturation (SaO_2) of 90-92% measured by pulse oximeter and frequent ABGs. A rise in $PaCO_2$ with oxygen administration is common, and should be expected. If the rise is excessive (i.e. over 10 mmHg or 1.3 kPa), delivered oxygen is reduced, titrating SaO_2 to 2-3% below the previous value, and ABGs repeated. If no $PaCO_2$ rise occurs with oxygen therapy, a higher SaO_2 may be targeted with repeat ABGs. If hypoxia is inadequately reversed (i.e. SaO_2 <85%) higher oxygen delivery systems should be used.

2. Bronchodilators

Bronchodilators are routinely given in all exacerbations of COAD, because a small reversible component to airflow obstruction is common, and bronchodilators improve mucociliary clearance of secretions. Nebulized beta-2 sympathomimetics (i.e. salbutamol, terbutaline, or fenoterol) are given 2-4 hourly. Combination with ipratropium bromide augments efficacy. Aminophylline (loading dose 5.6 mg/kg IV over 30 min, followed by infusion 0.5 mg.kg/h) is also commonly given, despite doubts on its overall benefits. Theophyline has the additional benefit of improving diaphragm contractility, although the clinical importance of this is not yet clear. Parenteral sympathomimetic agents are rarely indicated and not recommended for routine use.

3. Steroids

Short term steroids may improve airflow obstruction in an exacerbation of COAD, but not in every patient. They should be avoided if ARF is due to bacterial pneumonia or bronchitis. Doses used are similar to those for acute asthma (i.e. IV hydrocortisone 3 mg/kg or methylprednisolone 0.5 mg/kg given 6 hourly for 72 hours).

4. Antibiotics

Benefits of antibiotic therapy are conflicting. Antibiotics are likely to benefit only those exacerbations due to bacterial infection. Nonetheless, it is reasonable to initially administer antibiotics until bacterial infection is confirmed or excluded. Suitable antibiotics to cover common bacterial pathogens are amoxicillin, erythromycin or a third cephalosporin.

5. Non-Invasive Secretion Clearance

Clearance of lower respiratory secretions is of crucial importance.

(a) *Chest Physiotherapy* is the primary technique, and should be initiated and regularly repeated as both a curative and preventative measure. Encouragement of coughing and deep breathing are the two most important factors.

(b) *Nebulized Mucolytic Afents* such as acetylcysteine have been proposed as adjuncts to bronchodilators, but their benefits are uncertain.

6. Other Measures

Other adjunctive measures are applicable to some patients.

(a) *Hydration, Diuretics, Digoxin and Vasodilators.* COAD patients are sensitive to changes in fluid status, and intravenous rehydration should be undertaken cautiously. Diuretics and digoxin are beneficial in LV failure. Even if evidence of LV failure is minimal, a trial of diuretic is worthwhile in refractory cases. Diuretics will reduce fluid overload in cor pulmonale. However, they should be used with care in those with severe pulmonary hypertension. Excessive decrease in preload will decrease RV filling pressure and may result in a low output state. Digoxin is of no established benefit in cor pulmonale, as the primary problem is one of afterload. Pulonary vasodilators have a more rational basis, but clinical results are conflicting, without clear benefits. They may even be detrimental, and are not generally used.

(b) *Heparin* given subcutaneously in low doses (i.e. 5000 units SC bid) is recommended as a prophylactic measure against venous thrombosis and pulmonary embolism.

(c) *Electrolyte Correction* is undertaken. Hypophosphataemia is common, and hypomagnesaemia, hypocalcaemia, and hypokalaemia may also present and also impair respiratory muscle function. Hyponatraemia may occur with inappropriate antidiuretic hormone release or with excess use of diuretics.

(d) *Intercostal Drainage* is indicated for pneumothorax and pleural effusions of sufficient volume to compromise respiratory function. Instead of conventional drains, simple aspiration using a fine cannula, 3-way tap and syringe, have resulted in reduced complications, admission rate, and hospital stay.

(e) *Respiratory Stimulants* increase respiratory drive and lower $PaCO_2$. These include acetazolamide, medroxyprogesterone, naloxone, doxapram and almitrine. The principle of using such drugs is doubtful in COAD, where respiratory drive is already increased (the "can't breathe" group). These drugs also significantly increase respiratory work, respiratory distress and risk of fatigue. Sustained benefits have not been demonstrated for most agents, and they are not generally recommended. They may have limited application in the "won't breathe" group, especially those patients associated with sedation or anaesthesia. Narcotic induced respiratory depression is most appropriately managed with naloxone. Other forms of respiratory depression may be managed with doxapram, which acts both on peripheral carotid chemoreceptors and respiratory centre. However, the V/Q mismatch which contributes significantly to hypercarbia, will greatly limit the capacity of a respiratory stimulant to reduce $PaCO_2$.

Almitrine is believed to act by sensitizing peripheral chemoreceptors to both hypoxia and hypercarbia. It is believed to reduce $PaCO_2$ both by stimulating respiration and improving V/Q mismatching (by augmenting hypoxic vasoconstriction in hypoxic areas and redistributing pulmonary blood flow). Some studies have shown benefit in exacerbation of COAD but use of almitrine is yet to be established.

(f) *Nutrition* is important as malnutrition is associated with decreased respiratory muscle mass and strength, and increased risk of fatigue, ARF, and death. Enteral feeding is preferred, but parenteral nutrition is given in gastrointestinal dysfunction. Excessive calories and glucose increase CO_2 production, and should be avoided.

B. Non-Conservative Measures

1. Invasive Techniques for Sputum Clearance

Most of these are temporizing techniques applied to patients who have failed or are expected to fail with non-invasive sputum clearance techniques. The aim is to avoid intubation and mechanical ventilation if possible.

(a) *Oropharyngeal/Nasopharyngeal Suctioning.* It is uncommon for the tip to reach the trachea, but the procedure is useful in clearing pharyngeal secretions, stimulating coughing and clearing lower respiratory secretions coughed only as far as the hypopharynx.

(b) *Nasopharyngeal Airway* allows the passage of the suction catheter through the nose and upped pharynx towards the larynx.

(c) *Fibreoptic Bronchoscopy* guarantees lower airway entry and access to all major subsegments. Although effective in clearing sputum, it is very labour intensive. It is usually indicated by focal collapse or consolidation due to obstruction by sputum plug.

(d) *Minitracheostomy* involves insertion of a small diameter (4.0 mm) tracheostomy tube through the crico-thyroid under loca anaesthesia, using the Seldinger technique. It allows suctioning using a fibe bore (10 FG) catheter. The tracheostomy is left patent at all times, to allow ventilation through the tube as well as the mouth and nose.

(e) *Endotracheal Intubation* solely for lower airway suction, (without ventilatory support) may be questioned, but has been used. It is less well tolerated than mini-tracheostomy, but provides wide bore suction access and better control of inspired oxygen and humidity. Ventilatory support is easily instituted if required.

(f) *Formal Tracheostomy* provides the best, most comfortable and most stable form of wide bore lower airway access.

2. Mechanical Ventilatory Support

When respiratory failure progresses or fails to resolve despite aggressive conservative treatment, intubation and mechanical ventilatory support becomes necessary. The decision to

ventilate requires careful consideration. Mechanical ventilation is associated with weaning difficulties and ventilator dependence. Hypercarbia or acidosis alone are not indications for mechanical ventilation, as they can be sustained for some time without impending respiratory collapse. As with acute asthma, the decision is based on a number of criteria:

(a) The clinical appearance of fatigue and impending respiratory collapse.

(b) Increasing $PaCO_2$ despite adequate conservative treatment, and not due to oxygen administration.

(c) Deteriorating conscious state due to fatigue, hypercarbia or both.

(d) Hypoxia, refractory to high levels of inspired oxygen.

(e) Deterioration due to failure of sputum clearance.

(f) Respiratory arrest.

Mechanical ventilation is withheld in endstage disease, when permanent ventilator dependence is likely. This decision should be based on certain criteria. In general, patients must fulfil all criteria below for ventilation to be withheld.

(a) Known severe, fully assessed COAD which has failed to respond to adequate therapy.

(b) Known or suspected chronic respiratory failure.

(c) Severe limitation by dyspnoea with a poor "quality of life".

(d) No identifiable reversible factors.

Mechanical Ventilation Technique

Ventilatory strategies aim to minimize pulmonary hyperinflation by use of low tidal volumes, low minute ventilation, and long expiratory times. Tidal volumes of 10-12 mL/kg are commonly recommended, but 8-10 mL/kg may be better. Although controversial, a high inspiratory flow rate is recommended as it results in a short inspiratory time, and hence a longer expiratory time for the given ventilatory rate. It has been shown to reduce pulmonary hyperinflation and alveolar pressure and to improve gas exchange. Ventilator rate must be adjusted to achieve a low minute ventilation. Minute ventilations of less than 200 mL/kg are guidelines, and if used with small tidal volumes (8-10 mL/kg), allow relatively high ventilator rates (up to 20-25/min). If high minute ventilations are required to normalize pH, the degree of pulmonary hyperinflation and its effects should be determined beforehand. If pulmonary hyperinflation is excessive, minute ventilation should not be increased, and hypercarbic acidosis is accepted. Positive end expiratory pressure (PEEP) increases pulmonary hyperinflation and risks hypotension and should not be applied.

If controlled hypoventilation is not required, then a ventilator support mode must be chosen. This has been the subject of much debate. Intermittent mandatory ventilation (IMV) is preferred by many, although it has no advantage over controlled ventilation in terms of duration of ventilation or outcome. It has the theoretical advantage of promoting ongoing respiratory muscle activity to minimize wasting, and it does not allow large, patient-induced increases in minute ventilation.

Continuous positive airway pressure (CPAP) either by face mask or endotracheal tube has been proposed to overcome intrinsic PEEP. This has the theoretical advantage of reducing work of breathing, and may be tried either during partial ventilator support (i.e. IMV) or as an alternative to mechanical ventilation. Its role in COAD ARF is yet to be established. Jet ventilation at 100 breaths/min can achieve satisfactory gas exchange in patients with COAD, but advantages over standard ventilatory techniques have not been shown. Newer modes of flow-by and pressure support also have theoretical advantages of reducing work of breathing and promoting a better ventilatory pattern, but have not yet been fully investigated.

Table 1. Indicators for Weaning from Mechanical Ventilation in COAD

Sahn et al

Minute ventilation requirement < 10 L/min
Ability to double this on command
Spontaneous respiration rate < 20 breaths/min
Maximum inspiratory pressure > 20 cm water

Tahvanainer et al

Maximum inspiratory pressure > 25 cm water
Vital capacity > 10 mL/kg
SaO_2 > 90% on 40% inspired oxygen

Weaning

Weaning a patient with severe COAD from ventilatory support can be difficult and prolonged. Numerous criteria have been proposed to assess the capacity of the patient for weaning (Table 1). None are absolute in their predictive accuracy, and weaning nearly always depends on clinical assessment as ventilatory support is reduced. Various modes have been proposed to facilitate weaning, including IMV, pressure support, CPAP, and flow-by ventilation. No method has been consistently shown to accelerate the weaning process. It must be suspected that the major determinant weaning is recovery from the precipitating illness, and not the ventilatory technique.

Prognosis

Most patients with COAD who present with ARF do not have end stage disease. Although their acute illness may be life threatening, their short and long term outcome is often good. Martin reported 36 patients with exacerbation of COAD, and found 96% hospital survival and 72% 2-year survival; only 1 patient required mechanical ventilation. Although

most patients do not require mechanical ventilation, of those ventilated, the hospital survival rate is 80%, and 2-year survival significantly lower at 30%.