The Management of Acute Respiratory Failure

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Respiratory failure implies that the respiratory system is unable to maintain an alveolar ventilation which is capable of coping with the amount of carbon dioxide being produced by the body (i.e., alveolar hypoventilation). Thus the amount of oxygen added to the arterial blood and carbon dioxide eliminated is reduced with resultant arterial hypoxia and hypercapnia. As is seen in Table 1, alveolar ventilation may develop in the three groups of conditions: pulmonary disease, extrapulmonary disease, and central respiratory depression.

Respiratory failure develops most commonly in patients suffering from chronic airway obstruction, such as chronic bronchitis and/or emphysema, and in patients suffering from neuromuscular diseases, severe obesity and disorders of the chest cage such as kyphoscoliosis, or in severe chest injuries.

Alveolar hypoventilation also results when the central control of respiration by the medullary respiratory centers is depressed by drugs such as barbiturates, narcotics and tranquilizers, or anesthesia. In addition, the medullary respiratory center may become relatively insensitive to increases in arterial pCO₂ in patients who are suffering from chronic hypoxia and hypercapnia.

In diseases involving the pulmonary parenchyma, the pulmonary vasculature, or pleura, hypoxia frequently develops because there is a mismatching of the distribution of blood and gas, but the alveolar ventilation is usually adequate to eliminate carbon dioxide. On the other hand, when there is superimposed bronchial obstruction, infection or pulmonary congestion, the alveolar ventilation may be inadequate relative to the carbon dioxide production, so that severe hypoxia and carbon dioxide retention may develop.

When hypoxia and hypercapnia develop acutely, it is a true emergency which requires immediate therapy. The most important factor in establishing the diagnosis of respiratory failure is a high index of suspicion. This is particularly true if an acute infection, thromboembolism or other catastrophe, such as spontaneous pneumothorax, develops in any of the above underlying conditions, and if oxygen or a depressant medication has been administered.

The clinical manifestations of respiratory failure depend upon the underlying disease process, the precipitating factors and the severity of the hypoxia and hypercapnia. The clinical findings due to hypoxia and hypercapnia are predominantly neurologic, e.g., disturbances of consciousness, headache, and muscular movements; and cardiovascular, e.g., tachycardia, a bounding pulse, and usually an elevated blood pressure. However, in severe cases, the blood pressure often falls and is associated with generalized vasodilatation and profuse sweating. Physical examination is of little benefit in establishing the diagnosis of inadequate alveolar ventilation unless the condition is extremely obvious.

The definitive assessment of the adequacy of the alveolar ventilation can only be obtained by measurement of the arterial blood gas tensions and pH. Table 2 indicates the arterial blood findings in acute and chronic respiratory failure. In both situations, analysis of the arterial blood reveals hypoxia (unless the patient is receiving O₂), and an elevated carbon dioxide tension. In acute respiratory failure, the carbon dioxide content is normal and the pH low; while in chronic respiratory failure, because bicarbonate has been retained to compensate for the elevated pCO₂, the carbon dioxide content is elevated and serum chloride low. Under normal circumstances, the compensatory retention of bicarbonate is sufficient to bring the pH to a low normal value. On the other hand, a metabolic alkalosis is occasionally present because of potassium depletion.

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There are probably several reasons for this. Potassium is released from the cells into the serum when acidosis is present, and if renal function is adequate, the excess potassium is lost in the urine. Since many of these patients are treated with diuretics for heart failure, or steroids, the potassium deficiency and alkalosis may be further aggravated. In addition, the dietary potassium intake is often considerably less than the normal, while if nausea and vomiting are present, additional potassium is lost.

**Management**

Although there are specific adjuncts to therapy in certain underlying conditions, the management of acute respiratory failure is similar under most circumstances, and is designed to reverse the physiologic disturbances which are present.

The alveolar (and thus the arterial) pCO₂ at any given time, is related to the amount of carbon dioxide being produced in the body (VCO₂) and the amount being eliminated in the alveolar ventilation (VA).

\[
P_{A, CO_2} = \frac{V_{CO_2}}{V_A} \times 0.863
\]

Thus, hypercapnia (and hypoxia) will develop whenever the alveolar ventilation is inadequate for the amount of carbon dioxide being produced in the body.

The therapy of this condition is designed to provide adequate oxygenation, to decrease the numerator of the equation (CO₂ production) through reduction of the work of breathing, and to increase the denominator of the equation (alveolar ventilation) so that there is adequate elimination of carbon dioxide.

**Adequate Oxygenation**

The hypoxia which develops in acute respiratory insufficiency may be extremely severe. While reduction of the arterial pCO₂ can be brought about slowly, it is imperative that the hypoxia be alleviated immediately through the administration of oxygen. When the cause of the hypoxia is purely a reduction in alveolar ventilation, provision of an adequate alveolar ventilation alone will raise the arterial pO₂ to normal levels. However, in most instances, the hypoxia is also the result of an alteration in ventilation/perfusion ratios, so that oxygen enrichment of the inspired air is necessary. Unless there is circulatory failure, adequate oxygenation can be achieved by the administration of oxygen at low flow rates through nasal cannulae even when hypoxia is severe in the majority of patients with respiratory disease.

In the past, many physicians have been extremely wary of administering oxygen to patients with chronic respiratory disease when they become acutely ill. This was because of the concern that oxygen administration would eliminate the hypoxic stimulus to breathing, and, as a result, cause a further fall in alveolar ventilation and aggravation of the hypoxemia. However, the hazards of the administration of oxygen to patients who are unable to maintain an adequate alveolar ventilation have been, if anything, overemphasized. A slight rise in arterial pCO₂ under conditions of adequate oxygenation is not associated with significant deleterious effects, and is, in no way, comparable to the potential danger of severe hypoxia. A rise in arterial pCO₂ when oxygen is administered is not an indication for cessation of oxygen therapy, but rather an indication for intensification of concomitant therapy directed at reducing the work of breathing and increasing the alveolar ventilation. Thus, if increasing mental stupor or confusion, a drop in ventilation, or a rise in arterial carbon dioxide tension occurs during oxygen therapy, intensive therapeutic measures directed at reducing carbon dioxide retention should be instituted.

**Reduction of Carbon Dioxide Retention**

Reduction of carbon dioxide production is brought about by therapy directed at reduction in the work of breathing through relief of bronchial obstruction or pulmonary congestion.

**Relief of Bronchial Obstruction**

Reduction of bronchial obstruction is brought about through measures directed at reducing the production and increasing the elimination of secretions, and alleviating bronchospasm.

**Reduction of Secretions**

A reduced production of secretions requires elimination of all irritants and eradication of infection. Infection, with thick viscid secretions which may be difficult to raise, and inflammatory swelling of the bronchial mucosa cause an increase in airway resistance, and are important factors in the development of severe hypoxia and carbon dioxide retention in patients with chronic respiratory disease. Whenever possible, an effort should be made to identify the offending organisms by smear and culture of the secretions and to assess their sensitivity to antibiotics, so that the appropriate antibiotic can be administered. Since most of the gram-positive organisms are sensitive to penicillin, this is usually the antibiotic of choice. If gram-negative bacilli predominate, a
broad spectrum antibiotic is indicated. Control of infection with the appropriate antibiotic should be achieved early and carried on until the sputum shows sustained reduction of pus cells and bacteria, and the patient is clinically improved.

THINNING OF SECRETIONS

Thinning of bronchial secretions and prevention of crusting are integral components of the management of airway disease. Intake of 3 liters of fluids per day should be provided in order to ensure adequate hydration of the patient. In some cases, warm humidification of the inspired air will help liquefy secretions; so that they are easier to raise, with resultant reduction in airway resistance. The administration of oral liquefying agents such as potassium iodide, and nebulized liquefying agents, enzymes or detergents have been recommended by many, but they have not shown to be more effective than water or saline mists, or good hydration.

PHYSIOTHERAPY

The role of physiotherapy cannot be overemphasized. Postural drainage is often useful to expedite elimination of secretions and should be undertaken as often as possible. The foot of the bed should be elevated about 12 inches, and the patient turned from side to side every half hour to facilitate drainage of bronchial secretions. While this is being carried out, the chest should be pummelled with rapid repetitive strokes and the patient then encouraged to cough and expectorate secretions.

ENDOTRACHEAL SUCTION

In severe cases of respiratory failure, particularly if the cough is ineffective, nasotracheal suction, and in some cases, bronchoscopy (preferably under local anesthesia) may be necessary to clear the tree of accumulated secretions.

RELIEF OF BRONCHOSPASM

The administration of bronchodilating agents is frequently of considerable benefit in the management of airway obstruction. Bronchodilators such as racemic epinephrine, epinephrine, isoproterenol, or orciprenaline, either alone, or diluted with saline, should be administered by means of a nebulizer which delivers small droplets.

It is essential that the nebulizer be used properly. If the patient is unable to coordinate the squeezing of the nebulizer with a slow maximum inhalation, the bronchodilator aerosol should be nebulized with a compressed air or oxygen source. In this case a Y-tube between the pressure source and the nebulizer allows the patient to control the nebulization. Following a maximal expiration, the nebulizer is placed in the mouth, and the bulb squeezed repeatedly, or the Y-tube occluded, so that airflow is directed through the nebulizer, while the patient makes a slow maximal inspiration, as though he is "sipping" hot soup. Following this, the breath is held for a few seconds, and then the air is slowly expired through pursed lips. After each inhalation of bronchodilator the patient should attempt to cough up secretions so that the fine droplets delivered by the ensuing nebulization can exert their effect in the smaller bronchi and bronchioi. The entire procedure should be repeated until the patient feels subjective relief in the lower lateral thoracic areas, or until he notes tachycardia or shakiness. The number of inhalations necessary will depend upon the severity of the obstruction, so that it will vary from patient to patient, and, in a given patient, from day to day.

If a patient is not able to take a big breath, the nebulized bronchodilator can be administered by a positive pressure breathing machine. Once again slow deep inspirations should be encouraged. It is debatable whether the routine use of IPPB is of any benefit in the average case of respiratory insufficiency. If the patient is capable of taking a deep breath voluntarily, the beneficial results claimed in patients can, for the most part, be attributed to the bronchodilators which are used in conjunction with the apparatus. There is considerable evidence that the proper administration of bronchodilating agents with a nebulizer is just as beneficial as bronchodilator delivered with a positive pressure apparatus. Nevertheless, it must be pointed out that the use of IPPB to deliver nebulized bronchodilator is of marked benefit if the patient is unable to take a big breath or, if he cannot coordinate his breathing to allow the inhalation of bronchodilator agents which are delivered by nebulizer with either a handbell or a pressure source.

Oral bronchodilating agents such as ephedrine sulfate, aminophylline or a combination of these, should be taken 4 to 1 hour before meals and at bedtime. Aminophylline preparations are best taken on an empty stomach because the absorption of aminophylline is slowed in an alkaline medium. For the patient who has considerable difficulty during the night, the sustained action oral bronchodilators or a rectal aminophylline suppository taken at bedtime are often of great benefit.

In emergencies, aminophylline (500 mg) in 200 to 500 ml of glucose in water given slowly, by the
in intravenous route, often brings about relief. In very severe bronchial obstruction, it may be necessary to administer steroids. Occasionally, particularly in status asthmaticus, beneficial results are obtained by the intravenous administration of hydrocortisone.

**Relief of Pulmonary Congestion**

Right ventricular failure is a major complication of chronic respiratory insufficiency and may develop acutely in severe respiratory failure. This is frequently accompanied by fluid retention and pulmonary congestion, particularly in elderly patients who are suffering from arteriosclerotic cardiovascular disease. The pulmonary congestion further increases the work of breathing and aggravates the disturbances of gas exchange. The administration of diuretics, digitalis and a salt-free diet in such circumstances frequently results in improvement in ventilatory function and arterial blood gases, presumably because of a reduction in pulmonary congestion.

**Provision of Adequate Alveolar Ventilation**

**Patent Airway**

In order to provide adequate alveolar ventilation, it is essential to maintain a patent airway. In acute situations, the introduction of a bronchoscope will not only provide an airway but will also permit examination of the tracheobronchial tree, and aspiration of secretions or application of epinephrine (adrenalin) to edematous mucous membranes. Instillation of an endotracheal tube will permit aspiration of secretions and, in addition, will facilitate assisted or controlled ventilation. The duration of endotracheal intubation is a matter of debate and depends upon the underlying condition, accumulation of secretions, and consideration of the probable duration of the acute problem.

When it has been decided that prolonged therapy will be necessary, a tracheostomy is indicated. However, it must be stressed that intensive physician and nursing care directed at the tracheobronchial tree, with repeated nasotracheal suction and physiotherapy may frequently obviate the necessity for tracheostomy. It is important to point out that a tracheostomy is not without complications, and should not be rushed into indiscriminately. Tracheostomy is not an emergency, rather it is the need for a patent airway which is the true emergency, and, as has been pointed out, this emergency can be corrected by the installation of an endotracheal tube. In most reported series, the majority of complications occur when the tracheostomy is done as an emergency. Tracheostomy should be respected as an operative procedure with inherent hazards and should be carried out under sterile aseptic conditions, in an operating room where it can be done without haste, and with the advantage of good assistants, light and position.

The largest possible endotracheal or tracheostomy tube which fits easily into the trachea should be used. In addition, the tube should have an inflatable cuff at its inferior end. A large tube facilitates aspiration of secretions and prolongs the life of the cuff. In addition to effectively preventing aspiration the cuffed tube makes it possible to assist or control the ventilation. Metal tracheostomy tubes are largely being replaced by plastic, nylon and rubber tubes, for these usually have a cuff built into their wall. These tubes are preferable to the metal tube because the possibility of life-threatening airway obstruction due to a cuff which has slipped off the end of the tube no longer exists.

**Humidity**

When dry gas is inhaled, approximately 650 ml of water per day is added to the inspired gas by the upper respiratory tract, particularly the nasal mucosa. Whenever an endotracheal or tracheostomy tube is in place, the inspired air bypasses the upper respiratory tract. Unless evaporation of water from the tracheobronchial tree is prevented, bronchial secretions will become viscous and thick, and crusting may develop.

Increasing the moisture content of the inspired air and adequate hydration of the patient will minimize complications after tracheostomy. By saturating the air being inspired at or above body temperature, water will be added to the tracheobronchial secretions. This is accomplished by nebulization of oxygen or compressed air through heated water or saline into a perforated plastic adapter which is placed over the tracheostomy opening. Another apparatus, which fits into the tracheostomy tube, traps warm water vapor from each expiration so that the ensuing inspiration is humidified and warmed.

**Prevention of Trauma**

By far the majority of complications of tracheostomy or an endotracheal tube result from improper care and attention to details. The mode of inflation of the cuff is very important for an overinflated cuff can cause necrosis of the tracheal mucosa. With proper precautions, pressure necrosis of the tracheal wall can be avoided, and patients can be ventilated indefinitely without tracheal complications. The cuff...
should be inflated while a positive pressure breathing machine is attached to the tube and only that amount of air which will prevent air leakage around the cuff during inspiration is introduced into the cuff.

Suctioning

Suctioning of the tracheobronchial tree is frequently necessary because the patient cannot develop an explosive expiration so that his ability to eliminate secretions is reduced. However, suction catheters, per se, can introduce pathogenic bacteria and produce tracheal damage. An aseptic technique and avoidance of trauma to the tracheobronchial mucosa during aspiration is essential. While the wearing of masks, gown and donning of sterile gloves would be ideal, it is frequently difficult to ensure this, particularly in emergency situations. Nevertheless, frequent hand washing and the donning of gloves while handling any form of excreta is essential.

Complications frequently develop because of excessive suctioning which may decrease the ciliary activity in the trachea even though the most rigid aseptic technique is used. The airways should be aspirated only when necessary and the catheter should not be left in the tracheobronchial tree for longer than a few seconds.

Suctioning is indicated when there is stethoscopic evidence of rales, rhonchi or diminished air entry on one or both sides; or if the respiratory pattern or the pressure necessary to deliver a given tidal volume changes while the patient is being ventilated. The catheter should be directed as far down the tracheobronchial tree as possible, and into the appropriate bronchus. A Y-tube at the proximal end of the catheter will ensure that the negative pressure is not exerted during instillation of the catheter. Suction should be applied while the catheter is being withdrawn quickly in a twisting motion by occlusion of the open limb of the Y-tube.

A separate sterile catheter should be used each time the patient requires suctioning. It is preferable to use a curved catheter which has a single opening at its tip. The curve facilitates passage into the left
main bronchus, and the single opening may prevent mucosal damage. If there are multiple side openings, the tracheal mucosa may be sucked into these openings, particularly if a plug is attached to the distal opening.

The aspirate from the tracheobronchial tree should be examined regularly. If the secretions are purulent, a smear and culture as well as antibiotic sensitivity tests should be carried out and the appropriate antibiotic administered.

In addition it must be remembered that inhalation therapy equipment of all kinds are frequent sources of nosocomial pulmonary infection. Frequent gas sterilization of all equipment or nebulization of weak acetic acid through all nebulizers is therefore necessary whenever inhalation equipment is being used.

Mechanical Aids to Respiration

When severe hypercapnia persists, despite intensive therapy directed at reduction of the work of breathing, or if the patient is becoming excessively fatigued, a mechanical ventilator may be necessary. In addition to increasing the alveolar ventilation, the ventilator also reduces the work of breathing.

Ventilators can be assistants, controllers, or assistant-controllers. The assistant inflates the lungs in response to an inspiratory effort by the patient, usually being tripped by the inspiratory reduction in airway pressure. The controller cycles automatically and controls the patient’s respirations at a preset pattern, and is not affected by inspiratory efforts by the patient. The assistant-controller is capable of controlling the respiratory pattern, and is also capable of assisting a breath if the patient makes an inspiratory effort out of phase with the ventilator. This type of ventilator acts as an assistant as long as the patient’s breathing efforts are at a higher rate than the set rate, but it will control breathing if the respiratory rate drops below the preset value.

Assisted Ventilation

When assisted respiration is all that is necessary, the patient-triggered intermittent positive pressure...
MANAGEMENT OF ACUTE RESPIRATORY FAILURE

breathing apparatus (IPPB) which is a convenient and effective means of administering nebulized bronchodilators and humidity, is an exceedingly useful adjunct to the management of the patient with respiratory failure. In such cases, constant supervision is necessary to ensure that the patient takes deep breaths. In addition, it is important to recognize that the patient who is extremely fatigued or whose respiratory muscles are weak or paralyzed, may be unable to activate the machine.

Controlled Ventilation

If the patient is apneic, excessively fatigued, working hard to breathe, or unable to achieve an adequate alveolar ventilation with assisted respiration, some form of automatic ventilator which controls the ventilation is necessary to restore the alveolar ventilation to normal levels. Most ventilators are either pressure-limited (ie the delivery pressure is preset) or volume-limited (ie the volume of air delivered with each breath is preset).

Monitoring of Patient with Respiratory Failure

The effectiveness of the therapy of acute respiratory failure is assessed by monitoring of arterial blood gas tensions and pH (Fig 1) as well as other parameters at regular intervals.

Monitoring Arterial pCO₂

Since the respirator is being used to provide an adequate alveolar ventilation, the arterial pCO₂ should be monitored regularly, and the ventilator adjusted to provide an alveolar ventilation which will maintain the pCO₂ at normal levels.

Monitoring Arterial pO₂

As long as the oxygen concentration of the gas being inspired is constant, the arterial pO₂ is a sensitive indicator of minute pathologic changes in the lung (Fig 2). In patients who have severe respiratory failure, particularly if associated with circulatory shock and acidemia, the pulmonary capillary perfusion may diminish. This "pulmonary hypoperfusion syndrome," or "shocked lung" is associated with altered alveolar cell function and available surfactant, increased capillary permeability and pulmonary edema. The deficiency in functional surfactant results in increased instability so that there is a tendency towards collapse. Similarly, small areas of focal atelectasis may develop in patients who are being ventilated at a constant tidal volume. In these situations perfusion of the atelectatic areas persists (ie venous admixture-like perfusion increases) and the arterial oxygen tension falls.

Thus a fall in arterial pO₂, while the inspired oxygen concentration is constant, particularly

Table 1—Respiratory Failure

<table>
<thead>
<tr>
<th>Increased Work of Breathing</th>
<th>Reduced Tidal Volume</th>
<th>Increased Carbon Dioxide Production</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased Oxygen Consumption</td>
<td>CENTRAL RESPIRATORY DEPRESSION</td>
<td>ALVEOLAR HYPOVENTILATION</td>
</tr>
<tr>
<td>PULMONARY DISEASE</td>
<td>HYPOXIA</td>
<td>HYPERCAPNIA</td>
</tr>
<tr>
<td>ALVEOLAR HYPOVENTILATION</td>
<td>Uneven Ventilation and Perfusion</td>
<td></td>
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</tbody>
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CHEST, Vol. 58, Supplement No. 2, October 1970
Table 2

<table>
<thead>
<tr>
<th>Arterial Blood</th>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td>pO₂</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pCO₂</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HCO₃⁻ content</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
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*Unless otherwise indicated.

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if the arterial pCO₂ is unchanged, should be taken to indicate the development of poorly or nonventilated areas of lung which are still being perfused. This frequently precedes clinical signs or X-ray findings by several days. When pO₂ falls under these circumstances the attending personnel should provide sufficient oxygen to meet the patient’s physiologic needs and institute vigorous efforts to expand the lungs in order to open up small areas of atelectasis. This can be achieved by large-volume ventilation and in addition, continuous positive pressure breathing (ie the application of an expiratory retard which keeps the pressure in the thorax positive during expiration). In addition to the above measures, bronchoscopy to remove accumulated secretions may be necessary in some cases.

Monitoring Hydrogen Ion

Alterations in acid-base balance exert important effects on the cardiorespiratory system which influence the therapeutic regimen. The presence of acidosis is particularly significant, for an increased hydrogen ion concentration, particularly when there is an associated hypoxia, causes constriction of the pulmonary vasculature and increases the work of the right ventricle, and in addition may precipitate severe cardiac arrhythmias. In addition, it has also been shown that an increase in hydrogen ion concentration reduces the therapeutic effect of bronchodilating agents. The acidosis can be particularly alarming even though carbon dioxide retention is not marked, for severe hypoxia is associated with considerable anaerobic metabolism and lactic acid production by the tissues.

On the other hand, as has been pointed out previously respiratory failure is occasionally associated with a concomitant metabolic alkalosis. In addition, excessive artificial ventilation, particularly in the patient who has chronic carbon dioxide retention and compensatory increase in serum bicarbonate may produce a marked metabolic alkalosis, and lead to convulsions. The excessive artificial ventilation results in a sharp fall of arterial pCO₂, but bicarbonate excretion in the urine is slow, so that a metabolic alkalosis will be produced.

Thus it is important to stress that the urgency in the management of respiratory failure should be directed not at the arterial carbon dioxide tension but rather at the degree of hypoxia and hydrogen...
Monitoring the Ventilator

Once an adequate alveolar ventilation is ensured (as evidenced by a normal arterial pCO₂), the airway pressure, tidal volume and respiratory rate should be monitored at frequent intervals. Since the force generated by any ventilator during inspiration is expended against the resistance offered by the ventilator, and the elastic and nonelastic resistances of the lung and chest wall, more force will be required to provide the same tidal volume if any of these resistances increase. Conversely, less force will be required if there is a decrease in resistance.

So long as there is no change in resistance to inflation or a leak in the airway, both the pressure-limited and volume-limited ventilators will deliver a constant tidal volume. If the resistance of the lung rises, the volume-limited ventilator will continue to deliver the preset tidal volume to the patient by generating a higher pressure in order to overcome the increased resistance to inflation. The pressure-limited ventilator, on the other hand will be unable to deliver the same tidal volume because the pressure cutoff has been preset. As a result, the amount of gas forced into the patient will fall reciprocally. Thus different parameters will be affected by an increase in lung resistances depending on whether a volume-limited or a pressure-limited ventilator is in use (Fig 3). When a volume-limited ventilator is being utilized, an increased resistance to inflation or a leak in the airway is reflected by a change in the airway or monitoring pressure. On the other hand, if a pressure-limited ventilator is being utilized, an increased resistance to inflation or a leak in the airway is reflected by a change in tidal volume and respiratory rate. In addition, the pressure-lim-

![Figure 4. Monitor sheet for serum electrolytes, sodium, potassium and fluid balance.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21506/ on 04/05/2017)
itted ventilator will chatter in the face of complete obstruction, or hiss and fail to terminate inspiration if there is a leak in the airway.

OTHER CONSIDERATIONS IN MANAGEMENT

Cardiac Monitoring

As a result of many factors such as hypoxia, acidosis, alkalosis and hypokalemia, severe cardiac arrhythmias may develop in patients with respiratory insufficiency. Thus monitoring of the electrocardiogram and heart rate is essential when acute respiratory failure is being treated. When acidosis is severe, bicarbonate should be administered. Conversely, it is important to point out that quite dangerous degrees of hyperventilation can be encountered when techniques that will effectively maintain the mechanical integrity of lung and gas exchange are utilized. Under such circumstances it may be necessary to add a mechanical dead space in order to maintain CO$_2$ homeostasis and to prevent the development of hypocapnia and alkalosis.

Fluid and Electrolyte Monitoring

Since it is necessary to ensure good hydration in the management of respiratory failure, it is essential to monitor the intake and elimination of fluids, and particularly the urinary excretion. In addition the serum potassium, sodium, and chloride levels, and the sodium and potassium balance should be monitored daily. An example of the type of monitor sheet which can be used to follow the trends in serum electrolytes, sodium, potassium and fluid balance is shown in Figure 4.

As has been described above, the potassium stores of the body may become severely depleted in patients with respiratory insufficiency. In addition when the acidosis of respiratory failure is corrected, there is a tendency for sodium to move preferentially into the cells. In order to prevent or correct a chronic hypochloremic alkalosis, the administration of potassium must be prompt and aggressive so long as there is good urinary output. In addition, the administration of an adequate amount of chloride containing solution is frequently necessary for the potassium balance may not be corrected until chloride has also been administered.

THE RESPIRATORY CARE UNIT

Respiratory failure, cardiac arrhythmias, circulatory collapse and severe fluid and acid-base imbalance may develop in a large variety of patients on the medical or surgical wards of a general hospital, where proper management of these critical situations on a 24-hour basis is almost impossible. Because of the added responsibility of caring for other patients on these wards, residents and nurses are unable to pay sufficient attention to minute details, which often make the difference between life and death in critically ill patients. In addition, because the staff in the general wards is constantly changing, it is difficult to ensure continuity in treatment and sufficient background knowledge to provide the necessarily high quality of care which is required.

Although there is a marked trend towards the development of specialized "respiratory care units," it is apparent that the management of respiratory failure requires staff who can also recognize and manage acid-base, fluid and electrolyte problems, and cardiovascular failure. The converse is also true—diseases primarily affecting other systems are often complicated by respiratory insufficiency. For these reasons, we do not favor a unit which focuses solely on patients suffering from acute respiratory failure, or on any other system.

In addition, it is important to point out that the respiratory and circulatory failure which develop in medical and surgical patients is the same. The establishment of separate medical and surgical units requires considerable duplication of equipment and trained personnel.

On the other hand, there is a need for the concentration of critically ill patients in a special area, where they can receive special attention and care directed to respiratory and cardiac malfunction and the maintenance of an adequate circulating blood volume and urinary output. The necessity for concentration of the care of the critically ill patient in a special ward area of a hospital is dictated by the overriding importance of having a trained nursing and resident staff which is capable of understanding basic pathophysiology and the equipment being used to monitor or manage these patients. The unit must be organized in such a way as to be capable of recognition of all emergencies and be equipped to handle them for 24 hours per day. Unfortunately this standard of resident and nursing care cannot be achieved on every ward of a hospital. In addition, by having the highly trained personnel and specialized equipment concentrated in one area, it is possible to initiate clinical investigation of disordered function in respiratory and circulatory insufficiency and shock, and the application of newer ideas in management.