

How to Get Patients On and Off Respirators*

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This title implies that one has already decided that one's patient needs to be artificially ventilated, that is, that he is in respiratory failure. How does one diagnose respiratory failure? Much of what I have to say in this regard is in terms of arbitrary limits, values, and guidelines. Since these guidelines are arbitrary, there may exist legitimate grounds for differences of opinion about some of them. However, we have found these guidelines to be quite helpful, and experience would indicate that they are reasonable.

Let us first define what we are talking about. Respiratory insufficiency, which is the first step in the departure from normal pulmonary function, is the failure to maintain normal blood gases without persistent tachypnea, cough, or both. Hence, it can be seen that a patient may be in respiratory insufficiency and still have normal blood gases, that is, a patient in severe status asthma may be maintaining essentially normal blood gases but only with the expenditure of considerable effort and energy manifest as tachypnea and dyspnea. When the patient is inspiring room air normal blood gases are a P_{O_2} of 90 to 95 mm of mercury, a P_{CO_2} of 40 mm of mercury, and a pH of 7.4. Mixed venous blood, as it is taken from the pulmonary artery under normal circumstances, contains a P_{O_2} of 40 mm of mercury, a P_{CO_2} of 46 mm of mercury, and a pH of approximately 7.38 to 7.39. Thus it can be seen that while venous blood may give a fairly accurate representation of the patient's acid-base balance, it is of virtually no use in determining the state of oxygenation. The main function of the lungs is to convert venous blood into arterial blood, and in order to evaluate this function one must look at the finished product, not the raw material. Respiratory insuffi-

ciency can be said to have progressed to a state of respiratory failure when the P_{O_2} is below 50 mm of mercury or the P_{CO_2} is above 50 mm of mercury, at which time the pH may or may not still be near normal. Depending upon the etiology of the respiratory failure, some patients may need the institution of artificial ventilation at this point; others may not. Regardless of the etiology of the respiratory failure, when the P_{O_2} remains below 50 or the P_{CO_2} remains above 50 in conjunction with an arterial pH at or below 7.25, mechanical ventilation is indicated. There may be a few exceptions to this degree of ventilatory impairment, but they are few indeed. In this outline, one can see a summary of the indications for respiratory support, which are: a respiratory rate of greater than 35 per minute, a vital capacity of less than 10 to 15 ml per kg of body weight, an alveolar-arterial oxygen tension gradient of greater than 400 mm of mercury (an arterial P_{O_2} of less than 200 mm of mercury on 100% oxygen), a dead space to tidal volume ratio of greater than 60%, and an arterial carbon dioxide tension in excess of 60 mm of mercury, except in the case of patients with chronic hypercapnia.

It is arterial oxygen tension or P_{O_2} that we speak of and that we think about, but in actuality, the thing that we are concerned about is the delivery of an adequate amount of oxygen to the tissues, a determination which is not directly or easily measurable. However, if by means of the oxyhemoglobin dissociation curve, one relates arterial oxygen tension to oxygen saturation and in turn relates oxygen saturation into total oxygen content, implying a normal oxygen carrying capacity, such as a hematocrit of 35% or better, and if one can then derive at least indirect evidence of an adequate cardiac output, one can then assume that oxygen delivery to the tissues is adequate. The point to be made here is to stress the fact that an adequate arterial oxygen tension may indeed exist in the

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presence of a grossly inadequate oxygen supply to the tissue, such as with either severe anemia or an inadequate cardiac output, or both. The arterial oxygen tension is what we measure, but with few exceptions it is not the arterial oxygen tension *per se* in which we are interested. Arterial oxygen saturation is quite satisfactory even down to an arterial oxygen tension of 60 mm of mercury, and indeed hemoglobin does not reach 50% saturation until a P_{O_2} of approximately 26 mm of mercury is reached.

We can therefore summarize the following factors as indications for immediate intubation and ventilation:

1. A consistently rising arterial carbon dioxide tension.
2. A pH at or below 7.25.
3. A previously alert patient who becomes somnolent and unresponsive.
4. An alveolar-arterial oxygen tension gradient on 100% oxygen of 400 mm of mercury or more.
5. A respiratory rate of above 35 per minute or more.
6. A tidal volume of less than 3 to 4 ml per kg.
7. A vital capacity of less than 10 ml per kg.

How do we measure an alveolar-arterial oxygen tension gradient? This is no more than the alveolar oxygen tension minus the arterial oxygen tension measured in millimeters of mercury. This measurement is accomplished by giving the patient 100% oxygen to breathe for a minimum of 15 minutes. In some patients with severe degrees of shunting due to pneumonia or pulmonary edema and the like, as much as 30 minutes of 100% oxygen breathing may be required to completely denitrogenate the lung because of severe ventilation/perfusion inequalities. At the end of this arbitrary 15 to 30 minutes of 100% oxygen breathing, one then measures the atmospheric pressure and draws a blood gas sample. The arterial carbon dioxide tension can be assumed to completely equilibrate with the alveolar carbon dioxide tension, and this, plus the alveolar water vapor tension, determined as a function of body temperature, is subtracted from atmospheric pressure. The difference must be the alveolar oxygen tension since one can assume that the oxygen breathing for 15 to 30 minutes has completely eliminated all alveolar nitrogen. When this maneuver is performed upon healthy patients, one

will find an arterial oxygen tension of between 575 to 600 mm of mercury, implying a shunt of about 3% of the cardiac output, which is the normal anatomic shunt. The alveolar-arterial oxygen tension gradient, or the $A-aDO_2$, can therefore be said to be 75 to 100 mm of mercury on 100% oxygen and 5 to 10 mm of mercury on 21% oxygen, that is, room air. Both of these $A-aDO_2$'s imply a difference in oxygen content or oxygen saturation of precisely the same amount, that is, 3% of the cardiac output. The difference in the numerical values of the two calculations is due to the shape of the oxyhemoglobin dissociation curve.

The initial steps in instituting mechanical ventilation in most, but not all patients, are:

1. Produce a tidal volume of approximately 12 to 15 ml per kg, realizing that in the great majority of patients in respiratory failure it is necessary to achieve an initial tidal volume of approximately 2 times that of normal, in order to compensate for the ventilation/perfusion inequalities present. (This would be excessive in patients with chronic obstructive pulmonary disease).
2. A cycling rate of approximately 12 to 15 times per minute.
3. An inspired oxygen concentration of FI_{O_2} of 100%.
4. Mechanical dead space may be added at this time if it is anticipated that such large tidal volumes will produce an abnormally low level of arterial carbon dioxide tension.
5. Sedation is often necessary especially at the outset of mechanical ventilation.
6. Arterial blood gases should be done after approximately 15 minutes of ventilation with these settings in order to determine the future needs and appropriate settings of the ventilator. Since the patient was started on 100% oxygen, the first set of blood gases will allow one to calculate the $A-aDO_2$ on 100% oxygen. This value can then be put into the following formula in order to calculate the minimal desired inspired oxygen concentration:

$$\text{Desired } FI_{O_2} = \frac{(A-aDO_2) + 100 \text{ mm Hg}}{P \text{ atmos.}} \times 100$$

In this fashion one determines the least amount of oxygen that will adequately saturate the patient's hemoglobin without running undue risk of pulmonary oxygen toxicity; one is using oxygen as a drug.

Next one must be prepared to look at certain parameters of the cardiovascular and respiratory systems to note the response to therapy. Among these observations are:

1. The electrocardiograph oscilloscope should be used in patients with arrhythmias, congestive heart failure, those critically ill or hemodynamically unstable.
2. Intra-arterial pressures may need to be measured when cuff pressures are unobtainable or unreliable.
3. Central venous pressure measurements are of value in determining the effective circulating blood volume and when myocardial efficiency is in question.
4. The $F_{I_{O_2}}$ should be measured at least daily or when altering oxygenation parameters.
5. The tidal volume, respiratory rate, and minute volume of ventilation should be measured while the patient is on the ventilator approximately every 1 to 4 hours.
6. When the patient is breathing spontaneously, the tidal volume, respiratory rate, and, in conscious patients, the vital capacity should be measured every 1 to 4 hours.
7. The $A-aDO_2$ on 100% oxygen should be measured at least daily or as necessary.
8. The VD/VT ratio—the dead space to tidal volume ratio—should be measured daily or as necessary.

What is the dead space to tidal volume ratio, and how is it measured? The VD/VT ratio is that percentage of tidal volume which is wasted in the total of the anatomic dead space and the alveolar dead space, that is, areas of lung parenchyma which are ventilated but not perfused, or at least ventilated in excess of the amount of perfusion present. This value in normal patients is 30%. The observation is performed by dividing the difference between alveolar and mixed expired carbon dioxide tensions by alveolar carbon dioxide tension.

$$VD/VT = \frac{(P_{ACO_2}) - (P_{\bar{E}CO_2})}{(P_{ACO_2})}$$

As the ratio increases, it means that the patient's ventilation/perfusion inequalities are increasing and that he is wasting more and more of each tidal volume in ventilating areas of the airway and lung which are not perfused and which therefore contribute nothing to the exchange of gas. As the VD/VT ratio increases, one must increase his minute ven-

tilation to higher and higher levels in order to maintain a normal carbon dioxide tension of 40 mm of mercury. At VD/VT ratios above 60%, the curve begins to rise in an exponential fashion, and above this level of dysfunction patients can no longer breathe enough volume to maintain normal carbon dioxide tensions, and respiratory acidosis begins to occur. The VD/VT ratio can be measured both on and off the ventilator, but measurements made while the patient is being ventilated tend to run slightly higher than those when he is breathing spontaneously, especially during high inspiratory flow rates.

Nursing routines are important for physicians to know and understand for one reason only. If the nurse is unaware of the problems attendant to the management of respiratory failure, no one other than the physician can make her knowledgeable in this respect. The physician must know what he wants in order to tell the nurse what to do and what not to do. I do not wish to under-emphasize the importance of intelligent physician care in the management of such patients; nonetheless, the successful treatment of respiratory failure is largely a nursing venture. If nursing care is less than optimal, patients are unlikely to survive regardless of the degree of sophistication and intelligence of their physicians.

The following nursing routines are therefore recommended:

1. The patient is *NEVER* left unattended. (This is virtually impossible except in an ICU setting.)
2. Sterile technique in airway care and suctioning is of utmost importance in order to prevent iatrogenic contamination and infection.
3. Endotracheal and tracheostomy tube cuffs should be pre-stretched or of the soft cuff design to avoid tracheal trauma.
4. Daily weights must be obtained and recorded.
5. Intake and output must be accurately recorded daily.
6. The arterial pressure, pulse rate, temperature, and central venous pressure are recorded at intervals.
7. The patient's position in bed should be changed hourly while he is awake or hourly around the clock if comatose.
8. Attention should be directed to the care of the skin of the dependent parts. Heels,

knees, and elbows should be padded if deemed appropriate.

9. Chest physiotherapy should be coordinated with position change, airway suctioning, and IPPB treatments, if utilized. The frequency of this is to be determined according to the severity of the disease.
10. All joints in comatose or paralyzed patients should be put through a passive full range of motion daily.
11. The color and approximate quantity of tracheal aspirate are recorded.
12. An extra sterile tracheostomy tube and cuff of the appropriate size are kept at the bedside at all times.
13. Water condensing in ventilatory tubing is drained each hour.
14. The humidifier is checked and refilled every 8 hours or more often if necessary. The volume of water required to do so is recorded. This is done in an aseptic fashion.
15. Oxygen lines and flow meters are checked hourly to be sure that they are properly connected and functioning as ordered.
16. The house officer is notified immediately in case of a change in the level of consciousness or the occurrence of tachycardia, hypotension, confusion, agitation, tarry stools, or arrhythmias.

Appropriate laboratory studies during the maintenance of artificial ventilation include:

1. A chest x-ray is taken initially, following airway placement or change, daily to every 3 days, frequency to be determined by the severity of the disease.
2. The hematocrit is determined daily.
3. All stools are examined for occult blood.
4. Electrolytes, BUN, creatinine, sugar, total protein, and albumin are measured initially and as necessary.
5. The white blood count and differential are determined initially and as necessary.
6. Tracheal aspirate obtained with sterile technique for Gram stain smear, culture, and sensitivities is accomplished initially and every 3 to 5 days as necessary.

The inability to maintain good nutrition is one of the primary problems faced by patients suffering from long-term respiratory failure. The average patient on intravenous maintenance fluids alone can maintain his own state of caloric need for only

approximately two days by means of glycogenolysis. After this the patient must utilize his own protein stores, thereby developing negative nitrogen balance. When the patient is taking nothing by mouth maintenance fluids should total 40 to 45 ml per kg per day of which one-third can be dextrose 5% in normal saline or dextrose 5% in lactated Ringer's solution, and the other two-thirds dextrose 5% in water. These figures must be adjusted upward for fever, for abnormal fluid loss, or in the presence of hypovolemia. They should be adjusted downward appropriately for patients in congestive heart failure, or those with limited renal function. Their content must be adjusted for electrolyte abnormalities. The average adult, when taking nothing by mouth, and on maintenance fluids only, should lose approximately ½ kg per day. Intake and output and daily weights must be carefully observed to make sure that this weight loss is taking place. Patients who are alert and cooperative, who exhibit normal gastrointestinal integrity, and who have audible bowel sounds, can and should eat. Patients who are comatose or paralyzed but with normal gastrointestinal integrity should have a high protein, high carbohydrate diet per nasogastric tube or gastrostomy. Patients with compromised gastrointestinal integrity lasting more than one week should receive parenteral hyperalimentation. The serum albumin should be kept above 2.0 g%, preferably above 3.0 g% in order to avoid the complication of pulmonary extravascular fluid sequestration. Starving patients cannot effectively breathe indefinitely. Successful weaning from prolonged controlled ventilation requires positive nitrogen balance.

Prolonged ventilatory support is attended by various complications which need to be anticipated and prevented if at all possible. Included in this list are the following:

1. The lowest inspired oxygen concentration possible should be used in order to prevent the adverse effects of pulmonary oxygen toxicity.
2. Sterile airway care is the hallmark of the prevention of iatrogenic pulmonary infection.
3. Soft cuffs or intermittently deflated cuffs on endotracheal and tracheostomy tubes should be used in order to prevent damage to the tracheal mucosa.
4. Prophylactic antibiotics should not be given routinely. Antibiotics should not be used

merely in the presence of positive airway cultures, but reserved for those cases where actual pulmonary or other infection exists.

5. Diuretics and serum albumin should be used as necessary for the management of interstitial pulmonary edema.
6. Anemia is not well treated with oxygen. Anemia should be corrected by the administration of additional red cells.
7. Patients who are persistently hyperventilated and thus rendered hypocapnic tend to lose potassium in the urine, and if digitalized at the same time, tend to develop arrhythmias.
8. Hypotension occurring during artificial ventilation is often due to hypovolemia.
9. Patients in chronic respiratory failure with preexistent hypercapnia should not be vigorously hyperventilated, as this form of treatment may produce dangerous degrees of metabolic alkalosis.
10. In patients with the adult respiratory distress syndrome, fluids and crystalloids should be restricted.
11. In patients with large shunts and an alveolar-arterial oxygen tension gradient in excess of 500 mm of mercury, the use of positive end expiratory pressure (PEEP) should be considered.
12. When hyperventilation and PEEP fail to lower the alveolar-arterial oxygen tension gradient, oxygen consumption must be decreased by use of morphine, curare and/or hypothermia.
13. When administered through an artificial airway, all inspired gas must be warmed and humidified appropriately.
14. Following long periods of parenteral nutrition, the resumption of oral intake must be preceded by some attempt to determine that the patient is capable of swallowing without aspirating.
15. The appearance of sudden tachypnea, cyanosis, hypotension, or arrhythmia should lead to the suspected diagnosis of either pulmonary embolism or pneumothorax.

Getting a patient off a ventilator implies that he has survived his bout of respiratory failure and is approaching that point at which he no longer needs respiratory support. When ideally ventilated

and oxygenated on a ventilator, patients may indeed appear clinically well and yet be totally unable to maintain adequate spontaneous ventilation once the ventilator is discontinued. Some appraisal must therefore be made of the patient's ability to oxygenate, to eliminate carbon dioxide, and to mechanically move air in and out of his chest for an appropriate period of time in order to insure that he can breathe spontaneously. The attempt to discontinue mechanical ventilation without obtaining this information previously may result in an otherwise "unexpected cardiac arrest." The requirements for weaning include the following 8 points:

1. The etiology of the respiratory failure must be sufficiently reversed.
2. The alveolar-arterial oxygen tension gradient on 100% oxygen must be below 350 mm of mercury.
3. The dead space to tidal volume ratio must be below 0.6.
4. The tidal volume must be above 3 ml per kg.
5. The vital capacity must be above 8 to 10 ml per kg to commence the weaning process. It must be better than 15 to 20 ml per kg to complete the weaning process.
6. The patient must be in positive nitrogen balance if he has been in negative nitrogen balance for any longer than one week previously.
7. The spontaneous respiratory rate in adults must remain below 35 per minute.
8. The hematocrit must be at least above 30%, preferably 35%.

The technique in weaning may be one of several. The following technique has been successful for us. The patient must have met the requirements for weaning already delineated.

1. The patient is in the sitting or semi-sitting position.
2. The ventilator is disconnected.
3. With the tracheostomy or endotracheal tube cuff still inflated, the tidal volume, rate, minute ventilation, and vital capacity are measured and recorded.
4. The cuff is deflated and 100% oxygen is administered for a period of 5, 10, or 15 minutes depending upon how long one expects the patient to be able to breathe spontaneously and to do so adequately.
5. At the end of this time an arterial sample

- of blood is drawn to be analyzed for P_{O_2} , P_{CO_2} , and pH.
6. The artificial airway cuff is re-inflated, and the previously mentioned ventilatory functions are remeasured and recorded.
 7. The patient is then reconnected to the ventilator.
 8. On the basis of the blood gas analysis and the ventilation studies, one then determines the duration of subsequent spontaneous trials at ventilation and the appropriate inspired oxygen concentration to be used during those spontaneous trials.
 9. The patient should progress to continuous spontaneous ventilation for one entire day before the same is attempted during sleep at night.
 10. After 24 continuous hours of spontaneous ventilation with acceptable blood gases, acceptable ventilatory function, and the patient not being excessively tired, he can be considered weaned from the ventilator.
 11. A higher inspired oxygen concentration should be utilized when the patient is breathing spontaneously than that which is used on the ventilator during the weaning process.

12. Assisted ventilation may be substituted for controlled ventilation during the weaning process if considered necessary.

In closing, I would like to remind you that the experience of an episode of ventilatory failure with the vigorous and sometimes heroic treatment that is required for successful management, and the stresses that go along with a stay in an average intensive care unit, are poorly tolerated by most patients. In addition to intelligent and sophisticated medical care and dedicated nursing care, these patients need compassion and respect for the fact that they are human beings undergoing a period of extreme stress. They will not only survive but they will survive as much more grateful patients if we all can remember to treat them as individuals with a disease rather than as disease entities alone. With this in mind, I would commend to you the following admonition which, I think, is said as beautifully as it can be said: "From inability to let well alone; from too much zeal for the new and contempt for what is old; from putting knowledge before wisdom, science before art, and cleverness before common sense; from treating patients as cases, and from making the cure of the disease more grievous than the endurance of the same; Good Lord deliver us."