Non-Ventilator Management of Respiratory Failure: The Ventimask*

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The Problem—Acute Respiratory Failure in Chronic Obstructive Pulmonary Disease. In a recent review the authors comment on acute respiratory failure complicating chronic obstructive pulmonary disease. They state that because of the tolerance to chronic hypoxemia, the recurrent nature of the lung failure, and the increased number of complications during mechanical ventilation, "artificial ventilation is not indicated until all other attempts have failed to reverse hypoxemia without causing hypocapnia" (2).

This paper will detail the conservative management of acute respiratory failure in patients with chronic respiratory failure due to chronic bronchitis and emphysema. It is important to recognize that this is a very specific group of patients. They have had chronic hypoxia and hypercarbia for months or years preceding their current episode of acute respiratory failure. There occurs a further drop in arterial P_{O_2} (Pa_{O_2}) and usually a further rise in arterial P_{CO_2} (Pa_{CO_2}). The baseline arterial Pa_{O_2}, Pa_{CO_2}, and pH in such patients are usually as follows:

Pa_{O_2} = 50 mm Hg
Pa_{CO_2} = 45 mm Hg
pH = 7.4

Because of infection, sedation, heart failure, associated asthma, and so forth, they usually present to their physician with increasing dyspnea, cyanosis, disorientation, and commonly the following arterial gases and pH:

Pa_{O_2} = 40 mm Hg
Pa_{CO_2} = 55 mm Hg
pH = 7.35

Remember that the statements in this paper *do not apply to asthma*, only to chronic bronchitis and emphysema.

The Therapeutic Goal. The physician must increase the Pa_{O_2} "significantly" with "little increase" in Pa_{CO_2} and "little decrease" in pH. While this stopgap measure keeps the patient alive, therapeutic efforts are directed toward complicating diseases responsible for the acute respiratory failure. An attempt will be made to define the vague term "significant decrease" in Pa_{O_2}, and "little decrease" in pH.

The Tool—The Ventimask.[1] Since this group of patients has had an elevated Pa_{CO_2} for varying periods of time, ventilation is keyed to the low arterial oxygen, rather than to the CO_2 partial pressure. The usual methods of oxygen delivery, the nasal catheter and face mask, deliver 30% to 40% oxygen, enough to lethally depress ventilation.

The ventimask represents a method by which the physician can deliver low flow oxygen inexpensively and with safety. Because of the Venturi effect, the 100% oxygen that flows into the mask will entrain room air (21% O_2) through the side ports of the mask (fig. 1). Four liters of oxygen flow per minute is necessary to achieve the 24% O_2 concentration in the mask. However, further increases in flow will merely draw more 21% oxygen into the mask, and by clever engineering the oxygen concentration in the mask is therefore kept constant at 24%. The 28%

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* Presented by Dr. Hunt at the Symposium on Respiratory Failure, May 26, 1972, at Richmond, Virginia.

[1] The ventimask can be obtained from The Bethlehem Corporation, 225 W. Second Street, Bethlehem, Pennsylvania.

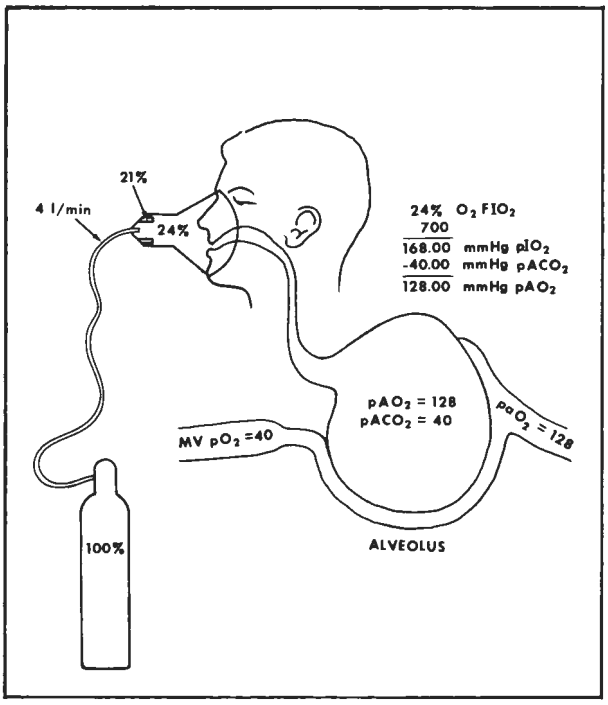

Fig. 1—The 24% ventimask.

ventimask is similar in all respects except that the final FI_{O_2} is 28%.

Guidelines for Arterial Gases.

Arterial Oxygen. When the Pa_{O_2} falls *below 50 mm Hg*, pulmonary capillary vasoconstriction occurs, and pulmonary hypertension occurs or is accentuated. Pa_{O_2} *below 40* will decrease sodium and free water excretion and may depress ventilation. In this range (40 or below) digitalis intoxication is markedly accentuated and ventricular arrhythmias occur.

In practice then, the immediate goal of therapy should be to increase the Pa_{O_2} from 30–40 mm Hg to the 45–55 mm Hg range. This frequently can be done with the 24% ventimask, remembering that an increase in inspired oxygen concentration (FI_{O_2}) from 21% (room air) to 24% is really 3% of an atmosphere (760 mm Hg) or approximately 21 mm Hg. Since in patients with chronic bronchitis and emphysema ventilation is usually reduced to many alveoli, the alveolar O_2 (Pa_{O_2}) of 128 mm Hg (as in fig. 1) is usually reduced so that the increase in Pa_{O_2} is more like 10 mm Hg than 21 mm Hg.

But it is very important to note that this 10 mm Hg change in Pa_{O_2} is on the steep portion of the oxyhemoglobin dissociation curve (fig. 2) and will actually nearly double the oxygen delivered at the tissue level.

Guidelines for Pa_{CO_2} and pH. Except for its narcotic properties, Pa_{CO_2} elevation *per se* is probably not important but may be used as a guide to oxygen delivery. If the 24% ventimask therapy results in a 10 mm Hg increase in Pa_{O_2} and at the same time elevates the Pa_{CO_2} greater than 5 mm Hg, further increase in oxygen concentration should not be attempted. If the Pa_{CO_2} remains the same or is reduced then one can safely go on to a 28% ventimask and another 4% (28 mm Hg) increase in FI_{O_2}. A Pa_{CO_2} greater than 65 mm Hg may decrease salt and free water excretion, but this can usually be overcome with the use of diuretics (Lasix® not ethacrynic acid).

The pH, however, is critical. Many vital enzyme systems do not work as the pH falls below 7.2, and hydrogen ion elevation will also cause pulmonary capillary vasoconstriction.

If the pH can be kept between 7.3 and 7.4 and does not remain consistently below 7.2, and if the patient remains conscious and *able to cough,* then conservative therapy should be continued.

General Measures. Dr. E. J. M. Campbell has said, "controlled oxygen therapy plus ineffectual nursing is better than uncontrolled oxygen plus ineffectual nursing only because the patient gets into less trouble" (1). The most important factor immediately is nursing care.

The patient should be:

1. In a chair, not flat in bed.
2. In a lighted room or corridor for the first 24 hours for observation and stimulation.

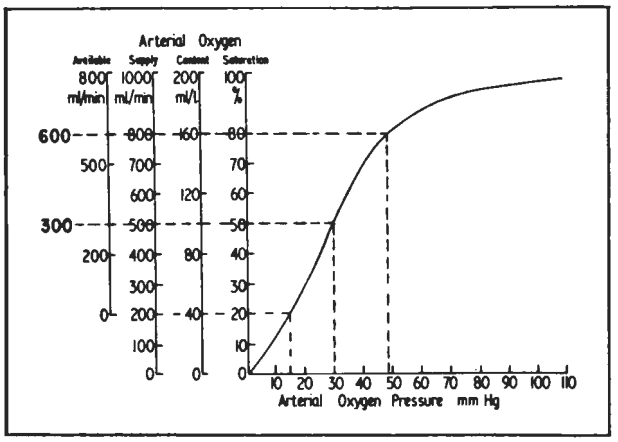

Fig. 2—Increase in Pa_{O_2} on 24% ventimask. "Available" means available at the tissue level (modified after Campbell).

3. Receiving chest physiotherapy each hour from the floor nurse or relatives or at least encouraged to cough and breathe deeply every 15 minutes.
4. Encouraged to use a pressure regulated IPPB machine (Bird or Bennett) five minutes out of every 30 or 60 minutes (without Isuprel®). This is for "stir-up," cough, and stimulation not specifically for ventilation.

Specific Therapy. In general, there is no "specific therapy" for chronic bronchitis and emphysema, so that complications or associated diseases must be diligently searched for and vigorously treated if present. One must not blindly and mindlessly treat each patient with chronic bronchitis and emphysema with digitalis, antibiotics, diuretics, steroids, and broncholytic agents.

Mucolytics. There are none that are effective. Acetyl cysteine (Mycomyst®) may actually aggravate any bronchospasm that may be present. Robitussin® should be reserved for your grandmother who has a "tickle in her throat."

Look for and treat:

1. *Left ventricular failure.* This is most easily diagnosed by an upright chest x-ray showing pulmonary venous engorgement in the upper lobes or Kerley B lines (fig. 3). Obvious pulmonary edema, whether unilateral or mild, should be suspected and searched for. Treat vigorously with Lasix® and replace K losses with KCl not K-Triplex. Digoxin should be used judiciously. The right ventricle will respond to digitalis, but toxicity may occur at half the usual digoxin level when the Pa_{O_2} is below 50 mm Hg.

2. *Asthma.* Eosinophils in the blood or on Hansel stain[2] of the sputum suggest a diagnosis of asthma. Suspect this diagnosis if the patient does not smoke, is under 50, or has a childhood history of asthma. If asthma is not present, Isuprel® can accentuate hypoxemia and in the already hypoxemic individual, aminophylline, adrenalin, and ephedrine may cause dangerous arrhythmias. In the patient with chronic bronchitis and emphysema in respiratory failure without prior evidence of a good response, certainly Isuprel® and probably the other bronchodilator drugs are contraindicated.

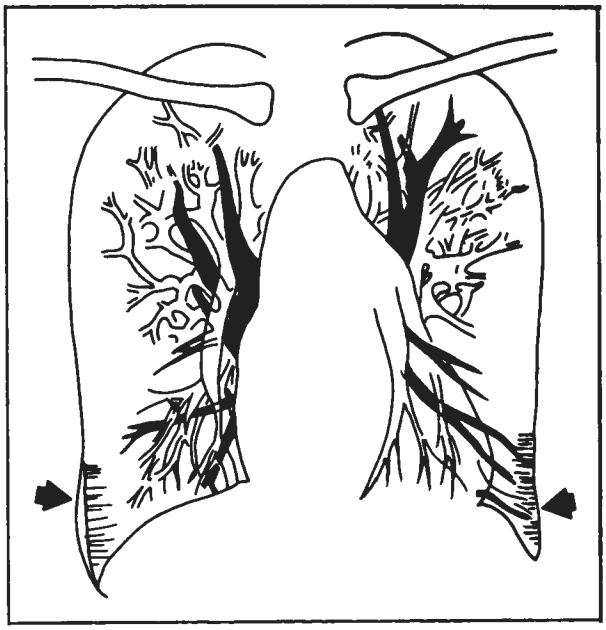

Fig. 3—The arrows indicate the position of Kerley B lines. The pulmonary veins are shown in black.

Steroid therapy (prednisone 40–60 mg daily) in patients with associated asthma may be life saving. An intermediate PPD should be done on every patient on admission prior to the institution of steroids and, if positive, isoniazid therapy should be considered, depending on the duration of the steroid therapy and the appearance of the chest x-ray.

3. *Infection.* The sputum should be observed, stained (both Gram and Hansel), and cultured if grossly purulent. Infrequently, grossly purulent sputum can be due to the presence of eosinophils alone. Usually acute purulent bronchitis is the the cause. The most common pathogens found are the pneumococcus or hemophilus influenza. Since probably 10% of all strains of streptococcus pneumonia are resistant to tetracycline, ampicillin 2 g daily is the treatment of choice (unless penicillin sensitivity is present).

4. *Pneumonia: A chest x-ray must be obtained.* The use of a stethoscope alone in the examination of the emphysematous chest is grossly inadequate. The presence of pneumonia in the patient with acute respiratory failure constitutes a medical emergency. Unless the overwhelmingly predominant organism can be identified by gram

[2] Hansel stain may be obtained from Lide Laboratories, 6828 Oakland Avenue, St. Louis, Mo. 63139—$7.50 for 8 ounces.

stain and quellung[3] as a pneumococcus, cephalothin and gentamycin should be used for 48 hours until the results of the sputum culture are available. You do not have time to be wrong, then switch.

5. *Cor pulmonale.* Rapid, effective, specific therapy for the right heart failure of cor pulmonale in these patients is not available. The patient has right heart failure primarily because he is hypoxemic, and you cannot rapidly relieve his hypoxemia or he will underventilate, become unconscious, needing intubation and mechanical ventilation, and this is what you are trying to avoid. As mentioned above, digitalis will improve the function of the right ventricle but must be used cautiously. If the patient has not been given digitalis in the past, digoxin 0.25 mg daily without a loading dose is the preferable technique. A reduction in pulmonary blood volume, ascites, and pleural effusion all may improve pulmonary function so that aggressive diuretic therapy may be indicated. Ethacrynic acid may produce underventilation and should be avoided. Currently Lasix® is the drug of choice. *Thoracentesis may produce a lethal pneumothorax and should be avoided unless a massive effusion is present.* There is no evidence that phlebotomy is an effective method of therapy under these circumstances.

Sedation. If over sedation with morphine or pentazocine is suspected, naloxone (narcan) is specific therapy and can be given without risk of further sedation. If other drugs (Valium®, meprobamate, and so forth) are known to have been primarily responsible for the respiratory depression, then it becomes even more imperative that vigorous attempts be made to keep the patient awake for the first 24 hours of therapy. It should be noted here that it is self-defeating to keep patients awake continuously for more than 24 hours. At the end of that time, alternating naps with arousal every 30 minutes to one hour becomes necessary.

[3] Quellung reagent may be obtained as "Omniserum" from Statens Seruminstitute, Amager Boulevard 80, DK-2300, Copenhagen S, Denmark.

When to Intubate. Reduced consciousness and an inability to cough are the major indications for intubation and ventilation. Inability to achieve an arterial Pa_{O_2} greater than 30 mm Hg or maintain a pH greater than 7.2 after 24 hours of therapy are relative indications for intubation. But, one should never act precipitously on a single number obtained from the laboratory that does not fit the clinical course that has been observed.

Emergency tracheostomy should never be done without prior intubation. Ideally a period of mechanical ventilation with achievement of good oxygenation and reversal of the respiratory acidosis should be accomplished prior to tracheostomy.

Summary. Low flow oxygen therapy should be given a trial in all conscious patients presenting with acute respiratory failure due to chronic bronchitis and emphysema. The tolerance of these patients to hypoxemia and hypercarbia make this means of therapy possible. The ventimask is an effective, inexpensive method of administering low flow oxygen, with the added attraction that the oxygen concentration cannot be inadvertently increased. Nursing care and aggressive therapy of complicating or associated medical problems are the key to successful management.

REFERENCES

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(A more complete bibliography may be obtained by writing Dr. William Hunt.)