# *Oxygen Therapy*
# *Catheter, Mask, Hood and Tent*

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OXYGEN was discovered in 1774 by Joseph Priestley who said at that time, "My reader will not wonder that after having ascertained the superior goodness of dephlogisticated air [later named oxygen by Antoine Lavoisier] by mice living in it and by other tests above mentioned, I should have the curiosity to taste it myself. . . . I have gratified that curiosity by breathing it, drawing it through a glass siphon, and by this means I reduce a large jar full of it to the standard of common air. The feeling of it in my lungs was not different from common air, but I fancied that my breath felt peculiarly light and easy for some time to come. Who can tell but that in time this pure air may become a fashionable article in luxury? Hitherto, only two mice and myself have had the privilege of breathing it." [1]

The modern era of oxygen therapy began with the work of Barcroft [2] and Haldane [3] in England, and Barach [4] in this country. As then, the basic indication for oxygen therapy is still the lack of adequate tissue oxygenation (hypoxia). Hypoxia is most often the result of inadequate saturation of hemoglobin in the arterial blood with oxygen (hypoxemia).

## Mechanisms of Oxygen Lack

*Hypoventilation*, or uniform alveolar underventilation for any given level of metabolic carbon dioxide production which results in alveolar carbon dioxide accumulation and reduction of alveolar oxygen tension, occurs either as a result of depression of the respiratory drive or as a result of inadequacy of the ventilatory apparatus. The latter is usually a result of inadequate respiratory muscle power or a change in mechanical properties of the respiratory apparatus so that the available muscle power is inadequate in relation to the forces opposing ventilation.

Generally speaking, 30–40 per cent concentrations of oxygen in the inspired air are more than adequate for correcting the oxygen lack of hypoventilation. Thus, almost any method of administering oxygen will suffice for correcting the oxygen lack of this state. Not infrequently, however, the administration of oxygen to a patient who is hypoventilating removes the hypoxic drive to ventilation and thus may result in further depression of ventilation and further accumulation of carbon dioxide. This must not be construed as an indication for withholding oxygen but rather as an indication for the need of ventilatory assistance or stimulation of respiration.

*Uneven Ventilation with Respect to Pulmonary Capillary Blood Flow.* This disturbance is usually a result of disease of the bronchi or lung parenchyma altering the mechanical properties of the tissue. This interferes with effective ventilation while blood flow continues through the capillaries of the poorly ventilated spaces. As a result, incompletely oxygenated blood gains access to the pulmonary veins and the systemic circulation. To correct uneven distribution of ventilation, the administration of 30 to 50 per cent concentrations of oxygen is quite adequate. In the presence of even modest concentrations of oxygen, poorly ventilated spaces become well supplied with oxygen. Here again, however, in the presence of chronic hypoxia, we are confronted with the problem of hypoventilation following alleviation of the hypoxic drive to respiration and resulting in $CO_2$ retention.

*Alveolar-Capillary Diffusion Block.* This defect results from disease, of the alveolar membrane, interstitial space, or pulmonary capillaries, which alters either the thickness or the physical properties of the diffusing membrane. Again in this disturbance, even

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small concentrations of oxygen will be sufficient to elevate alveolar oxygen tension to levels which will readily overcome any abnormality in alveolar-capillary membrane diffusion. For example, assuming an alveolar air to arterial blood oxygen gradient of such magnitude that arterial blood oxygen tension approaches that of normal mixed venous blood (30–40 mm. of mercury), it is apparent that an increase in the alveolar oxygen concentration equivalent to 7 per cent added oxygen at sea level would be sufficient to increase the alveolar oxygen tension by 50 mm. of mercury, which could be expected to restore arterial oxygen tension to within normal limits (> 80 mm. of mercury).

*Veno-arterial Shunting* man occur as a result of passage of blood through either abnormal intrapulmonary or extrapulmonary vascular channels. This type of hypoxemia is most difficult and sometimes impossible to correct. The oxygen lack resulting from a right-to-left shunt of up to 33 per cent of the pulmonary blood flow usually can be corrected by 100 per cent oxygen breathing. Larger shunts will be accompanied by persistent hypoxemia in spite of 100 per cent oxygen. This situation prevails because the additional amount of oxygen that can be physically dissolved in the nonshunted blood plasma is relatively small compared to the deficiency of oxygen that results from the shunting of desaturated hemoglobin.

*Tissue Hypoxia Without Hypoxemia.* This may occur as a result of the following: (1) Reduced oxygen carrying capacity due to inadequate hemoglobin; or abnormalities of hemoglobin decreasing its oxygen-carrying capacity. (2) Reduced cardiac output or inadequate perfusion of tissues in relation to metabolic oxygen consumption. (3) Inability of tissues to utilize oxygen due to disturbances in oxygen metabolism.

These are circumstances in which arterial oxygen saturation is essentially normal but tissue hypoxia and physiological stress constitute indications for oxygen administration. Specific examples of these disturbances are: shock of any kind, but particularly blood-loss shock; coronary artery occlusion; severe cerebral vascular accidents; severe anemia; carbon monoxide and cyanide poisoning; severe methemoglobinemia or sulfhemoglobinemia; extremely high fever; severe heart failure; and postcardiac or major thoracic surgery. Another seldom recognized situation in which tissue oxygen lack exists in the presence of a normal oxygen saturation is severe alkalosis where, because of a shift of the oxygen dissociation curve to the left, hemoglobin does not give up oxygen readily to the tissues, and arterial oxygen tension is low with respect to oxygen saturation. (At *p*H 7.35 and 92 per cent saturation, the oxygen tension is 70 mm. of mercury, whereas at *p*H 7.7 and 92 per cent saturation, the oxygen tension is only 44 mm. of mercury which would be equivalent to 77 per cent saturation at a normal *p*H.) If alkalosis is due to hyperventilation, the lowered arterial carbon dioxide tension tends to compound the cerebral hypoxia since cerebral blood flow would be reduced. In these situations high concentrations of oxygen should be used unless withholding oxygen can otherwise be specifically justified. The importance of 100 per cent oxygen breathing in these states can be appreciated if one considers that in this manner, at sea level, the arterial oxygen tension can be increased to 600 mm. of mercury or more. This, as a result of physically dissolved oxygen, increases the arterial oxygen content above that which it would be on room air breathing by 1.5 to 1.7 ml./100 ml. of blood. Thus, not only is the diffusion pressure for the movement of oxygen into the tissues greatly increased but also the physically dissolved oxygen may represent a 20 to 40 per cent increase in the amount of oxygen delivered to the tissues per unit of circulating blood. The only way in which 100 per cent inspired oxygen can be approached is by use of a one-way valve system such as is part of the usual IPPB machines.

## Recognition of the Presence of Oxygen Lack

The clinical manifestations of hypoxia are known to be inconsistent, and thus the ability of physicians to detect hypoxia is variable and generally unreliable. In the past, cyanosis was considered the most useful sign but now is widely accepted as an unreliable index for the recognition of oxygen need. Moreover, cyanosis is a late manifestation of hypoxemia

Dyspnea, likewise, cannot be considered of value in recognition of hypoxia since dyspnea is a subjective disturbance which varies from person to person and is largely related to increased effort of breathing resulting from mechanical alterations of the respiratory apparatus. Thus, persons with fairly normal respiratory systems may increase ventilation many fold and be hypoxic to the point of fainting without dyspnea, whereas a person with chronic disabling pulmonary disease may experience dyspnea with the slightest increase in ventilation even without hypoxia. Increased respiratory rate is often associated with hypoxia but cannot be considered a useful index of oxygen lack since respiratory rate is influenced by many factors.

Among the most valuable clinical signs of hypoxia are tachycardia and altered mental behavior with irritability, anxiety, restlessness, inappropriate behavior, and the exhibition of poor judgment and confusion. One should avoid a natural tendency to ascribe symptoms of oxygen lack, including an obnoxious personality, to other aspects of the patient's illness. To be useful, these signs must be critically and carefully observed in relation to the presence of possible causes of hypoxia. Their value is limited, however, when, as is often true, such observations are not made with precision, in sequence, and in relation to circumstances that might be suspected to be accompanied by hypoxia. In any event, when clearly recognized, any of these signs is at best an indication of fairly severe hypoxia. Moreover, if after 100 per cent oxygen breathing for 30 to 60 minutes, there is significant improvement in all of the observable clinical parameters previously mentioned, the presence of hypoxia can be considered to be confirmed.

Regrettable as it may seem, we must acknowledge that, when in question, the only reliable method for detecting hypoxemia is to measure the arterial blood oxygen percentage saturation or oxygen tension. In addition to the time-honored but tedious Van Slyke manometric technique, a number of newer methods employing either spectrophotometric or polarographic apparatuses are readily available for more convenient measurement of arterial oxygen. When there is doubt about the presence of hypoxemia, the failure to obtain arterial blood and perform such tests is unjustifiable and results largely from lack of knowledge or because of emotional bias. In other circumstances, there is usually no reluctance to perform numerous very sophisticated and ordinarily more expensive metabolic tests that are rarely as urgent or vital as is knowledge of the presence of hypoxemia.

In situations where no apparatus for making a measurement is available, a simple bedside test can be used as an estimate of arterial oxygen saturation.

*Three Syringe Test.* The tissue down to and around the femoral artery is infiltrated with a local anesthetic if necessary. The plunger and walls of three 5 ml. syringes are wetted by heparin and the dead space is also filled with heparin. A regular 20 gauge needle is used to puncture the artery. Three milliliters of arterial blood are drawn into each of two of the syringes. The third syringe is used to collect normal venous blood from an individual with a comparable hemoglobin and hematocrit to that of the patient in question. Care must be exercised to draw the venous blood from a resting extremity while the blood is freely flowing, *i.e.*, without a tourniquet. The syringes are either capped by regular mercury-filled caps or with tapered round toothpicks. One of the samples is brought to 100 per cent saturation by adding about 2 ml. of 100 per cent oxygen to the syringe. The blood is shaken with the oxygen for about two minutes. The remaining gas is expelled so that the 100 per cent saturated sample can be compared under good natural light with the other anaerobically collected sample of the patient's arterial blood, and the normal venous blood. The oxygen saturation of normal venous blood is almost invariably about 75 per cent. In this manner, a fairly accurate estimate of the level of oxygen saturation of the patient's blood can be made between the limits of 75 and 100 per cent.

This test can be modified so as to give further information by the administration of 40 per cent oxygen to the patient. Then, another sample of arterial blood can be drawn for comparison with the fully saturated sample previously described. If after 10–15 minutes of 40 per cent oxygen breathing, the patient's arterial blood does not show essentially the same degree of saturation as the saturated sample, evidence of veno-arterial shunting as the basis for arterial hypoxia is present.

## Methods of Oxygen Administration

The method of administering oxygen should be selected on the basis of several factors, the first of which is concerned with the concentration of oxygen indicated by the par-

ticular situation to be treated. The second concern is reliability and simplicity of the method, and finally, patient comfort should be considered.

When desired, high concentrations of oxygen approaching 100 per cent can be accomplished by using intermittent positive pressure breathing devices or by using a well-fitted oronasal mask or a mouthpiece and noseclip. The mask or mouthpiece must be attached through a one-way valve to a reservoir into which oxygen is flowing continuously at a rate usually in excess of that of the patient's average minute ventilation. The latter system can provide 80 to 90 per cent oxygen in the inspired air. The exceptions may be the actively ventilating patients, particularly those with forceful, rapid inspiration, where the auxiliary inspiratory intake valves will open causing air mixing. In the case of quietly ventilating patients, these masks must be operated at flow rates (7 to 10 liters/minute) which will prevent carbon dioxide accumulation in the mask.

When lower concentrations of oxygen are indicated, the practice of using a metering device for regulating the concentration of oxygen delivered to a mask cannot be recommended. These units are difficult to use properly, since any increase in resistance downstream from the meter changes the oxygen concentration to a value higher than that indicated. Gas delivered through such devices cannot be humidified, since the resistance offered by the humidifying apparatus either would cause the oxygen to escape through air mix outlet of the dilutor or it would at least eliminate any effective air dilution of the oxygen. If lower concentrations of oxygen are desired, it is more appropriate to employ a device such as a face hood, nasal cannula, nasal catheter, or oxygen tent.

Since the plastic bag disposable mask, often referred to as the "poly mask," is frequently used, certain information relating to its properties should be emphasized. It is difficult and often impossible to fit the mask properly to the patient's face. If the patient moves about, the mask cannot be kept readily in position. When the mask is well fitted to the patient, and the flow of oxygen is sufficient to keep the bag inflated with the patient in the supine position (8 liters/minute or more), inspired oxygen will average about 75 per cent. At 4 liters/minute, the average inspired oxygen concentration will be about 45 per cent. With the patient sitting upright, it is difficult to achieve concentrations higher than 35 per cent oxygen in the inspired air, even at flow rates of 10 liters/minute. This problem exists to some extent with all disposable masks with open ports because of the tendency for the cooler oxygen to escape through the ports and leaks about the mask.

The most reliable and least expensive method of administering oxygen in low concentrations is the nasal catheter or double nasal cannula. When the catheter is used, a soft nonreactive plastic with multiple small holes which will diffuse oxygen over a wide area of the nasopharynx is preferable. Such a catheter avoids the focal irritation that occurs when oxygen issues forth from one or two holes. The catheter should be well lubricated with a water-soluble material before placement in the nose. It should be inserted with the tip extending *no further* into the throat than the uvula in order to avoid gastric overdistension which continues to occur as an accident of oxygen therapy by nasal catheter.[5] Moreover, Kory and associates[6] have shown that the differences, between alveolar oxygen concentrations achieved with the catheter and cannula, were small and insignificant. They also found that mouth breathing versus nasal breathing did not produce any significant difference in inspired or alveolar oxygen concentrations. The catheter should be removed and changed to the other nostril every 12 hours. Inspired oxygen concentrations obtained by the use of catheters vary considerably with the circumstances of use. In patients with large tidal exchange and high oxygen requirement, the concentration will be lower. Also, the resultant concentration of oxygen in the inspired air is less with the patient in the upright position, particularly when using a cannula, than with the patient in the supine position where more oxygen is retained. Under ideal circumstances of a high flow rate, that is, 6 to 8 liters/minute, with no more than average ventilation, the inspired oxygen concentration rarely exceeds 50 per cent. Nasal oxygen is often not tolerated at flow rates above 6 liters/

minute but is well tolerated even for prolonged periods at 4 to 6 liters/minute. At these flow rates, the inspired oxygen concentration varies between 30 and 40 per cent. Adequate humidification, preferably with jet-type humidifiers, is essential to the prevention of irritation and crusting in the nose when using nasal catheters or cannulas. In fact, oxygen by any route should always be adequately humidified during prolonged administration. Attention to the care of humidifiers is essential because of the danger of humidifiers acting as a source of hospital infections, as pointed out by McPherson,[7] and elsewhere in this symposium by Walter.

The open-top clear plastic face hoods, or head tents, offer no particular advantage over other forms of oxygen therapy except that they are more comfortable and often better tolerated by some patients. They can be used only when less than 50 per cent oxygen is adequate to meet the needs of the patient.

Conventional oxygen tents are troublesome to use properly because of the difficulty in controlling the oxygen concentration. These devices are costly as an initial investment and more expensive than most equipment to maintain. High oxygen flow rates are essential to maintain safe oxygen concentrations in most tents. The flow rate should never be less than 10 liters/minute. Only rarely can oxygen concentrations of 50 per cent be maintained at this flow rate. Under ideal circumstances, with a quiet patient and a flow rate of 15 liters/minute into a well-sealed tent, oxygen concentrations in excess of 60 per cent may be achieved but are rarely maintained. The indication for the use of a conventional oxygen tent is strictly a matter of patient comfort. Even so, there are some patients who prefer masks or catheters to tents. Burch and associates[8] have emphasized the principal virtue of oxygen tents as that of providing a cool, dry environment, thus avoiding the detrimental physiological effects of hot, humid environments on many patients, particularly those with heart disease who exhibit increased cardiac stress under these circumstances. For the most part, these authors could not obtain evidence to indicate that the addition of oxygen to the tent significantly improved the physiological status of a large number of patients treated in oxygen tents when these patients were individuals who were not obviously hypoxic.

Recently the usefulness of low concentrations of oxygen, administered by various methods including portable devices, to facilitate exercise or reduce the physiological stress of exercise in patients with chronic heart and lung disease has been emphasized by Barach[9, 10] and others.[11, 12] ° A discussion of standards pertaining to the use of portable apparatus was prepared by the committee on public health of the New York Academy of Medicine.[13]

Moore and Bridenbaugh[14] have demonstrated that a high concentration of oxygen is one of the proper antidotes for systemic toxic reactions from local anesthetic agents.

## Indications for Caution in Oxygen Therapy

The administration of high concentrations of oxygen (*i.e.*, over 50 per cent concentration) may be accompanied by undesirable consequences in patients with chronic obstructive pulmonary disease and long-standing hypoxia and $CO_2$ retention as a result of hypoventilation. The administration of high concentrations of oxygen may so completely supply the patient's oxygen want that further hypoventilation, further carbon dioxide retention with subsequent mental depression, stupor, coma and, in some instances, death may ensue. This is an unusual occurrence except in the presence of superimposed physical exhaustion or drug-induced sedation. This situation rarely occurs in the presence of properly administered oxygen starting with low concentrations as has been repeatedly indicated by Barach. The danger of administering oxygen to these patients has been overly exaggerated in comparison to the value of oxygen therapy. In any event, the presence of hypoxia in these patients must always be construed as a prime indication for oxygen therapy. If hypoventilation ensues, then either ventilatory assistance or appropriate stimulation of respiration by a selective agent such as ethamivan[15] should be employed.

Another situation in which prolonged administration of high concentrations of oxygen,

° See paper by Barach in this symposium.

particularly in depressed patients, may be of danger results from the washing out of nitrogen from the lungs. With complete loss of nitrogen (the only nonabsorbable gas) from the lungs, rapid absorption of oxygen from areas of temporary obstruction causes a reduction in the volume of air spaces, thus causing focal areas of atelectasis. Such a situation can be prevented by the following measures: avoiding over-sedation of the patient; avoiding the use of 100 per cent oxygen unless specifically indicated; and making sure that shallow breathing does not occur. The latter can be accomplished by stimulating the patient's respiration or by mechanically assisting ventilation. Gases containing adequate amounts of inert gas should be used in ventilation apparatuses in order to prevent the rapid reabsorption which occurs when completely reabsorbable gases are left in the lung spaces.

The question of oxygen toxicity is invariably raised, although the problem is virtually nonexistent with the therapeutic application of oxygen. I know of no instance of any untoward effects from the administration of oxygen in an appropriate concentration for the physiological needs of the patient, even if this is 100 per cent, so long as the gas is completely saturated with water vapor at a temperature approaching that of body temperature. The one apparently established example of oxygen toxicity is retrolental fibroplasia which has been shown to develop in premature infants exposed continuously over a period of several days to oxygen in concentrations in excess of 40 per cent. This disease is presumably completely preventable as long as a 40 per cent concentration of inspired oxygen is not exceeded. The possibilities of harm from high oxygen concentrations are discussed in detail by DuBois elsewhere in this symposium.

## Summary

Since man is born in and must live in an atmosphere of approximately 21 per cent oxygen, the addition of oxygen to the inspired air must be considered to be an interim procedure while the circumstances necessitating the increased oxygen concentration are elucidated and, wherever possible, eliminated.

The most important signs of hypoxia ar tachycardia and mental alterations which ma be subtle in their appearance. Confirmatio of the presence or absence of hypoxemia mus be based on measurement of arterial oxyge concentration. A convenient three syring colorimetric test has been described as a sim ple, direct method of estimating the presence of arterial oxygen lack.

Low concentrations (30 to 50 per cent) o oxygen in the inspired air will correct most causes of hypoxemia except that resulting from veno-arterial shunts. The highest possible concentrations of oxygen are often necessary to correct hypoxic states related to circulatory disturbances.

Whereas there are certain precautions that should be exercised in regard to oxygen therapy, the frequently mentioned dangers o oxygen therapy in patients with chronic hypoxia should not constitute a justification for withholding oxygen from patients suffering from severe hypoxia. Appropriate concentrations of humidified inspired oxygen necessary to correct hypoxia should always be employed, and if hypoventilation develops, ventilatory assistance or respiratory stimulation i necessary.

Portable oxygen apparatuses should be considered more frequently as a means of minimizing the physiological stress of physica activity in patients with cardiac or pulmonary disease.

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**OXYGEN THERAPY** Overall evaluation of techniques of oxygen administration to adults, based on effectiveness, comfort, ease of administration, and economy, provided the following preference, in decreasing order: nasal cannula, plastic face masks, nasal catheter, plastic face tent, rubber oronasal masks, and oxygen tent. (*Kory, R. C., and others: Comparative Evaluation of Oxygen Therapy Techniques, J.A.M.A. 179: 767 (Mar. 10) 1962.*)

**OXYGEN GRADIENTS** The mean alveolar-arterial oxygen gradient was 12 mm. of mercury during air breathing in a group of normal subjects. When hypoxic gas mixtures were substituted for air, alveolar-arterial oxygen gradients diminished and were obliterated at very low levels of oxygenation. The response was greatest during the first minute of low-oxygen breathing but change still occurred during and after the fourth minute. Simultaneous *in vivo* polarographic measurement of arterial and alveolar oxygen tensions was a reliable technique for study of alveolar-arterial oxygen gradients. (*Cosby, R. S., and others: Continuous Measurement of Alveolar-Arterial Gradients at Ambient and Anoxic Levels, J. Appl. Physiol. 17: 1 (Jan.) 1962.*)

**OXYGEN BREATHING** Significant increases in peripheral vascular resistance, mean arterial blood pressure, and both systolic and diastolic pressure occurred during oxygen breathing and persisted at least 40 minutes after oxygen was discontinued. Slight decreases in both cardiac index and heart rate also occurred during oxygen breathing. The observed changes in heart rate and cardiac index could have been initiated by either chemoreceptors or baroreceptors. No change in central blood volume was observed, but a marked increase in pulmonary blood volume may have occurred during oxygen breathing. (*Eggers, G. W. N., Jr., and others: Hemodynamic Responses to Oxygen Breathing in Man, J. Appl. Physiol. 17: 75 (Jan.) 1962.*)