OXYGEN THERAPY AND RESUSCITATION

ERNEST H. WARNock, M.D., Resident Anesthetist
AND
RALPH M. TOVELL, M.D., Attending Anesthetist
Hartford Hospital, Hartford, Connecticut

In the time of Harvey, the function of respiration was considered to be that of cooling the blood. Conflicting ideas existed concerning the physiology of respiration but in 1666 Boyle showed conclusively that in the absence of air, life was impossible. The true conception of the physiology of respiration as we recognize it today was suggested by Mayow in 1673. He stated that the air contained a certain “nitroaerial” spirit which was absorbed by the blood in the lungs, eventually reaching the muscles where it initiated muscular contraction through its union with sulphur. In 1774 this same substance was isolated in pure form by Priestley and was called “dephlogisticated air.” During his experiments, Priestley showed that this respirable gas was necessary for the conversion of venous into arterial blood (1). Lavoisier in 1777 also isolated the gas and showed that it combined with carbon to form carbon dioxide and that in this combination, animal heat was produced. The results of these and other findings soon led to the use of oxygen by inhalation as a treatment for a wide variety of conditions in a great number of which its use was unsuccessful. Hence, oxygen soon became discredited as a therapeutic agent.

The status of oxygen as a therapeutic agent remained questionable until the time of the World War when Haldane, Barcroft, Hunt, Dufton, and others used it successfully in the treatment of soldiers suffering from exposure to poisonous gases (2). Since that time oxygen therapy has been scientifically investigated. At present it is gaining recognition as a therapeutic agent for an increasingly large number of conditions.

The delayed recognition of the therapeutic value of oxygen is a result of misconceptions. When it was first used, it was thought to be a panacea. This idea is substantiated by the fact that in 1798 Thomas Beddoes established a pneumatic institution at Clifton where oxygen was administered to patients, regardless of the cause of their illness. Such unscientific use of oxygen led to its failure in many instances. The early clinical signs of oxygen want were frequently not recognized and oxygen was not administered until after irreparable damage had been done. Fear of overdosage and inefficiency of apparatus has in the

*Read before the joint meeting of the American Society of Anesthetists, Inc. and the Pacific Coast Association of Anesthetists, December 14, 1939, Los Angeles, Calif.
past prevented the use of oxygen in sufficient concentration to relieve the patient of anoxemia. There are those who have used oxygen, not as a therapeutic aid to the patient, but as a means of convincing the patient's relatives that everything possible was being done. In recent years clinicians have learned to recognize the symptoms of oxygen want and they have become aware of the often irreparable damage which lack of oxygen may produce (3). As a result, an increasing demand is being made for more efficient equipment and a more thorough understanding of the requirements for oxygen in various diseases. Cohen states, "Oxygen to be efficacious must be used freely, frequently, fearlessly, and almost constantly, nor must its use be postponed until the patient is moribund for it will not revive the dead" (4).

Anoxemia may be subdivided into two classes, namely, chronic and acute. Chronic anoxemia is that form which occurs constantly in varying degrees. It is the type seen in individuals suffering from congenital heart lesions. In this type of anoxemia, oxygen therapy is less beneficial than for the acute type. Although the concentration of oxygen in the blood is below normal, the chronicity of the condition permits the body to adjust itself to a reduced content of oxygen in the blood. After this adjustment has been accomplished, increase of the content of oxygen in the blood will not be needed unless some acute condition is superimposed on the chronic one (5).

Acute anoxemia (6) may be subdivided into three types, namely, anoxic, anemic, and stagnant. Anoxic anoxemia exists when the arterial blood does not contain the normal amount of oxygen because of alteration in the oxygen content of the surrounding atmosphere or because of an impaired exchange of gases in the respiratory system. This type of anoxemia is found in mountain sickness and in pneumonia. Anemic anoxemia exists when there is a deficiency or alteration of the hemoglobin making it impossible for the blood to carry sufficient oxygen from the alveoli to the tissues. This is the condition found in cases of anemia. Stagnant anoxemia results when, owing to local or general circulatory causes, the rate of transfer of gas from the lungs to the tissues may be so slow that the hemoglobin becomes "emptied" of oxygen before the journey's end. This type is encountered in cases of cardiac failure. Peters (7) and Van Slyke add a fourth classification, histotoxic anoxia in which the supply of O₂ may be perfectly normal but the cells are poisoned and unable to utilize it properly. This is true of carbon monoxide poisoning.

Depletion of oxygen in the gastro-intestinal system is indicated by the occurrence of nausea, diarrhea and vomiting. The symptoms of lack of oxygen in its early stages that are referable to the respiratory system are increased rate and depth of respiration. Subsequently they are rapid and shallow. In the circulatory system deficiency of oxygen manifests itself by a constant and progressive increase in
Oxygen Therapy and Resuscitation

If the deficiency is prolonged, there is a decrease in diastolic blood pressure with concurrent cardiac failure. Oxygen want within the central nervous system is manifested by headache, visual disturbances, irrational states, delirium, hyperpyrexia coma, and finally death.

Krogh (8) found that a lack of oxygen in the system increased the permeability of the capillaries. With this increase in permeability there ensues a loss of blood plasma into the tissues. This in turn leads to a concentration of the corpuscles in the capillaries and a decreased blood volume. The increased concentration of the blood leads to a reduced volume flow which further reduces the amount of oxygen which is delivered to the tissues. With deficient oxygenation there is an accumulation of products (9) which tend to produce atony and dilation of the capillaries. This likewise reduces blood volume flow. This vicious circle is present in a large number of acute infections and has often been misinterpreted as a toxemic reaction. If hyperpyrexia is present, this sequence of events may vary somewhat. Alkalosis (10) develops and the slightly alkaline hemoglobin compound gives up its oxygen less readily than normal hemoglobin. With the resulting decrease in arterial oxygen tension there is also associated another factor that tends to increase the severity of this condition. With each increase in temperature of 1° F, the basal metabolic rate increases 5.5 per cent. and with it the demand for oxygen increases (11). The velocity of the blood flow through the capillaries increases, but after the heart has exceeded its maximal effectiveness, and as blood pressure falls, the accelerated velocity may be replaced by comparative stagnation in the dilated vessels. The inhalation of oxygen is a logical procedure in many conditions where this vicious circle is present. Krogh found that the capillary stasis resulting from deficiency of oxygen became irreversible after approximately fifteen minutes. Hence it is essential that remedial measures to be effective must be instituted early.

Since oxygen is becoming recognized as a valuable therapeutic agent in certain diseases and since the medical profession is realizing that unsatisfactory results have in many instances been due to errors in administration, many are becoming interested in improving the efficiency and technique of oxygen therapy. Various methods have been advocated, each of which has been satisfactory in the hands of a few but none has met with universal approval. Oxygen has been administered by mouth in the form of a souffle, made by bubbling oxygen through water to which foam extract has been added (12). It has also been used intravenously. However, the general method of administration has been by inhalation. One of the first methods of administration was by means of the inverted funnel to which was attached a tube and bubble bottle containing water. This method has been found useless in that it was wasteful of oxygen, unpleasant to
wear, and it increased the concentration of oxygen in the inspired air by about two per cent.

The method (13) whereby an intranasal catheter was employed was devised by Adrian Stokes during the World War I. Its chief advantage was in the fact that several patients could be treated simultaneously from one tank of oxygen. However, since the World War I the catheter method has been widely employed, and it is thought by some to be the method of choice.

![Image of oxygen equipment](image)

**Fig. 1.** Oxygen that has been adequately humidified is being administered intranasally.

One of the chief objections to the administration of oxygen by the intranasal method had been the occurrence of drying of the mucous membrane of the nasopharynx as a result of insufficient moisture in the oxygen. This caused the patient discomfort and resulted in infection of the naso- and oro-pharynx. In order to meet this objection, a type of apparatus has been developed, in which conventional types of reducing valves and gauges to indicate tank pressure and oxygen flow are utilized. Attached to this set of valves by means of a metal tube is a closed cylinder, the wall of which is an alloy, which, due to its porous texture, is permeable to gases. This cylinder is immersed in water,
which is contained in an ordinary fruit jar of quart size. The lid of the jar, which fits tightly, contains an outlet through which the oxygen is delivered to the patient. The essential feature of this apparatus (Fig. 1) is that the oxygen in passing through the porous wall of the cylinder becomes finely dispersed, and a larger surface is exposed for the absorption of moisture as the bubbles rise to the surface of the water. Its chief advantage is that oxygen carrying sufficient moisture to insure the comfort of the patient can be administered. When the apparatus is in operation over a twenty-four hour period there is an appreciable lowering of water level in the container, thereby demonstrating the fact that the oxygen in passing through the water does absorb moisture freely.

There are certain precautions which must be taken in order to insure satisfactory performance of this type of outfit. First the tip of the catheter must be accurately placed in the oro-pharynx. Faulty placement will result in marked reduction of the concentration of oxygen in the lung. If the catheter is inserted too far into the pharynx the patient will swallow oxygen which will produce a dilatation of the stomach. It is also necessary that the water level in the container be kept within proper limits. Too low a level reduces the absorption of moisture, while with too high a level, water will flow into the intranasal catheter. The proper level can be indicated on the jar.

It is felt that this apparatus possesses advantages over those of similar type. Its simplicity of construction and ease of installation are worthwhile factors. It does permit adequate humidification of the oxygen. The parts of the apparatus which may be broken are freely available and they are inexpensive.

The same apparatus may be used to deliver oxygen or oxygen and helium mixtures to a B.L.B. mask of either the nasal or oro-nasal types (Fig. 2). These masks are particularly useful for the administration of high concentrations of oxygen for the relief of abdominal distention, or for the relief of severe anoxia associated with pneumonia or cardiac decompensation. For the administration of mixtures of helium and oxygen in the treatment of status asthmaticus two regulators and humidifiers are employed. One is attached to a tank containing helium 80 per cent. and oxygen 20 per cent. and the other is attached to a tank containing only oxygen. The delivery tubes are connected through a Y to a B.L.B. mask of the oro-nasal type. Helium may be given in 80 per cent. concentration or additional oxygen may be added if signs of deficiency of oxygen are present.

An oxygen tent of standard make operated under proper supervision furnishes an excellent and economical means of administering oxygen. In the selection of a tent there are certain fundamental principles which should be recognized. The motor should be mechanically efficient and readily controllable. There should be sufficient space so that the patient will not feel restricted. Means should be provided for
variation of the rate of circulation and height of temperature in the tent. Adequate means for controlling humidity and for controlling the concentration of carbon dioxide should be provided. In those tents employing ice for cooling and soda lime for absorption of carbon dioxide the containers for these agents should be insulated. Soda lime is not an efficient absorber unless it is warm.

![Image](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931178/)

**Fig. 2.** A mixture of oxygen and helium is being administered to the patient through a B.L.B. mask.

The tent, as it is run in the average hospital, is a most extravagant and inefficient method for the administration of oxygen. Unless the concentration of oxygen and carbon dioxide in a tent is controlled by actual frequent determination, the health of the patient may be jeopardized. A check of the concentration of oxygen in three separate hospitals revealed that the average concentration of oxygen was from 23 to 28 per cent. This low concentration was in most cases the result of leakage of oxygen. The same apparatus when properly supervised was efficient. The oxygen tent, to the average attendant, is only an obstacle which makes the routine care of the patient more difficult. Until attendants are taught the refinements of management and certain special procedures to be employed for those in tents, many patients treated by this method will suffer from anoxemia.

The oxygen chamber gave good results in the experience of many. The chief objections to this type of administration are the expense of installation, the hazard of fire and the limited number of patients who can be treated by this method simultaneously.
OXYGEN THERAPY AND RESUSCITATION

OXYGEN IN THE TREATMENT OF DISEASE

Experience has shown that in addition to the primary pathological changes occurring in diseases there are often secondary changes which occur as a result of anoxemia. By relieving the anoxemia the patient is often able to survive until such time as he has developed sufficient resistance to overcome the primary infection. This is the basis for the use of oxygen in the treatment of pneumonia. In order for the cells of the body to live they must receive a sufficient quantity of oxygen but of more importance is the fact that they must receive it at a definite partial pressure. At sea level this partial pressure is approximately 160 mm. of mercury. Any condition which interferes with the maintenance of this partial pressure in the cells produces serious results. In pneumonia the engorgement and consolidation in the lungs result in a decrease in the transfer of gases to and from the blood. At the same time there is a systemic reaction to the changes in the lungs. This is manifested by an increased temperature. For each degree of rise in temperature there is an increase in metabolism (11) of from 5 per cent. to 7 per cent. The anxiety and pleuritic pain experienced by the patient in this stage further increases the metabolism. As the metabolism increases the requirement for food and for oxygen increases. With the requirement for oxygen increased and the supply of oxygen in the blood decreased, the body attempts to stabilize itself by increasing cardiac output.

In the production of energy dextrose is converted into lactic acid. Oxygen is required for this conversion as well as for re-conversion of four fifths of the lactic acid into dextrose. The fifth part of lactic acid remaining is united with oxygen to form carbon dioxide and water. With a depletion of oxygen, as is found in pneumonia, this process is inhibited. Hence there is a collection of lactic acid in the cells. The cells cannot assimilate food because of the acidosis and the acidosis cannot be relieved without oxygen. Consequently the demand for oxygen increases as the supply of oxygen decreases. A constantly increasing demand is made upon the heart whose cells are reacting similarly to those of other parts of the body. Therefore, a pneumonic condition consists of three factors, namely: infection by pneumococci or other bacteria, intoxication, and cellular malnutrition. Recognition of these factors is of utmost importance because the intoxication and cellular malnutrition may be prevented by the early and the adequate administration of oxygen. Once these conditions have become established, oxygen therapy will not always correct them. Lack of oxygen exists long before the first sign of cyanosis appears. Cyanosis of the fingers tips and lips that can just be seen represents desaturation of oxygen to the extent of ten per cent. When cyanosis is definite it represents a desaturation of fifteen per cent., and when it is marked it indicates a desaturation of twenty per cent. The chief factors which
may operate to produce anoxemia are: failure of adequate ventilation as a result of rapid shallow breathing, intrinsic alveolar disturbance, intrinsic changes in the circulating blood, and intrinsic disturbance of cellular tissue (14). Although all these play a part, the essential factors are those which retard access of oxygen to the blood in the pulmonary area.

Considerable diversity of opinion exists concerning the proper safe and beneficial concentrations of oxygen which patients should receive. It is generally agreed that a concentration of under thirty per cent. is of no appreciable value. Barach (15) has stated that a concentration of over seventy per cent. for longer than four days has harmful effects. Karsner (16) has shown that with laboratory animals concentrations of over eighty per cent. result in definite cardiac damage, fibrous pneumonia, cloudy swelling and congestion of the other organs of the body. Beecher (17) in summarizing the work of Shaw, Behnke and others states that pure oxygen at a pressure of one atmosphere can be breathed by man for about six hours before toxic organic effects are seen. Some of these effects are impaired vision, peripheral constriction, abrupt rise of systolic and diastolic blood pressures, pulmonary edema, extreme pallor, dizziness, a feeling of impending collapse, slowed mental responses, and convulsions. However, Evans (18) contends that anoxicemic subjects react differently than normal subjects and that the higher concentrations of oxygen are beneficial in these cases. He followed carefully the course of 409 patients who received oxygen in high concentrations over various intervals of time, and concluded that 100 per cent. oxygen can be given not only without harmful effects to the patient but with excellent therapeutic results. Treatment with oxygen should be started as soon as diagnosis of pneumonia is made and continued throughout the course of the disease. Oxygen should be administered in concentration sufficient to abolish unsaturation of oxygen in arterial blood at all times. The efficacy of oxygen in the treatment of pneumonia is practically in direct proportion to the day of the disease on which treatment is begun.

The pulse rate is the most reliable criterion by which the benefit or failure of therapy of oxygen can be judged. Although numerous other changes are seen, a reduction of the pulse rate is the only factor in the metabolism of man which shows consistent change following exposure to oxygen-rich atmospheres. According to Barach (19) the symptoms of pneumonia may be classified according to their possible mode of origin. The manifestations that are pulmonary in origin are: cough, sputum, pleuritic pain, expiratory grunt, edema of lungs, and shallow breathing. Those that are of toxic origin are: delirium, weakness, rapid pulse, rapid breathing, chill, fever, dyspnea, prostration, and jaundice. The presence of headache, nausea, vomiting, delirium, weakness, rapid pulse, and rapid breathing is indicative of existing toxicity and anoxemia. Of these symptoms, the outstanding ones are those
pertaining to respiratory difficulty. If the respiratory difficulty can be relieved by the administration of oxygen, the patient does not become exhausted and his ability to combat toxemia and bacterial invasion is thereby increased. Discussion of the role of specific chemo-
therapy used in conjunction with oxygen therapy is beyond the scope of this paper.

**Cardiac Disease**

It has been found that the normal saturation of arterial blood was from 95 per cent. to 98 per cent. and of venous blood from 65 per cent. to 75 per cent. In cases of cardiac insufficiency Barach (5) found the arterial oxygen saturation to be between 95 per cent. and 75 per cent. and the venous oxygen saturation to be from 65 per cent. to 30 per cent. Cardiac conditions in which oxygen has been helpful may be divided into four groups, namely: congestive heart failure due to primary cardiac disease, cardiac insufficiency as a sequel to chronic pulmonary disease, acute coronary thrombosis, and coronary arteriosclerosis with chronic cardiac pain. The physiologic action of oxygen in the treatment of cardiac conditions has not been fully explained but various investigators have demonstrated definite changes following its use. In congestive heart failure due to primary cardiac disease it has been found (20) that by using oxygen in a concentration of 45 per cent. there was a relief of dyspnea, diuresis was promoted, edema was reduced, and there was a marked rise in the content of carbon dioxide in the arterial blood. Other changes noted were increase in arterial saturation with oxygen, decreased pulmonary ventilation, decrease in pulse rate, decrease in lactic acid in the blood, and diminution of cyanosis. Similar results were encountered when oxygen was used in cases of cardiac insufficiency which developed as a sequel to chronic pulmonary disease. Oxygen therapy, however, had no influence on the pulmonary disease. The syndrome seen in acute coronary thrombosis is one of acute lack of oxygen due to myocardial insufficiency. Wiggers (21), Ulrich (22), and Barach (20) are of the opinion that the pain experienced in acute coronary thrombosis is a result of an ischemia of the cardiac musculature which can often be relieved by the administration of oxygen. Barach and Levy (23) noted these results following oxygen therapy in acute coronary occlusion; relief of pain and restlessness, improvement in volume of the pulse with slowing and strengthening of cardiac contraction, decrease in pulmonary congestion, lowering of temperature, improvement in respiration, and increase in arterial pressure with decrease in venous pressure. Similar improvement was noted when patients suffering from coronary arteriosclerosis were treated with oxygen. As a therapeutic agent in cardiac diseases, oxygen is of more value in those who exhibit acute anoxemia. Cases of chronic cardiac disturbance such as those showing congenital defects apparently develop a compensatory mechanism which enables them to
withstand their deficiency of oxygen without serious embarrassment unless an infective process develops.

In summarizing it may be stated that oxygen has a definite though limited place in the treatment of cardiac disease. It is especially indicated in acute forms. It should not be used as a substitute for other accepted methods of treatment but it should be considered as an aid which will support the circulation until the heart has at least partially recovered. Even in the cases in which recovery is doubtful, oxygen is often warranted for the symptomatic relief which it affords the patient. Administration by means of an intranasal catheter is usually the most satisfactory method, but the B.L.B. mask may be employed to advantage for those exhibiting marked cyanosis.

Resuscitation

The Bureau of Vital Statistics, New York City, for the years 1931 and 1934 reports a total of 6110 deaths due to general asphyxia as compared to 2443 deaths from automobile accidents during the same two years. The Division of Vital Statistics, Bureau of Census, reported 18,432 suicidal and accidental deaths from asphyxia occurring in the United States in 1933. Through increased knowledge of preventative medicine and therapeutics, the death rate from many diseases is decreasing. However, as living conditions become more complex the possibility of death from some form of asphyxia increases. Conditions producing asphyxia usually occur without warning and unless proper treatment is immediately instituted, death is certain.

Many communities maintain rescue squads whose duty it is to be prepared at all times to meet asphyxial emergencies. Their work has been very commendable and society owes them a debt for the saving of many lives annually. The same degree of preparedness is not maintained in many hospitals. The one institution where the asphyxiated patient should have the best chance of survival is too frequently poorly equipped. Various machines are relied upon as the sole resuscitating agent. When the emergency occurs the machine may be loaded with empty tanks or it is so well stored that it cannot be found for a time, and there may be no one at hand who is sufficiently skilled in the operation of the machine to make its application efficient. The reason for this situation is that resuscitation has not been definitely recognized as a specialized procedure and hence no one has been made responsible for the purchase, care, maintenance, and operation of proper equipment. Because of the close correlation between the two fields of endeavor, it seems a wise arrangement to make members of the department of anesthesia of a hospital responsible for the maintenance and operation of apparatus used for resuscitation. Workers in different specialties may have their own ideas regarding the treatment of asphyxia. The internist may rely chiefly on stimulating drugs and manual methods of artificial respiration, the surgeon may advocate a
tracheotomy while the obstetrician may suggest tubbing and external stimulation. All of these procedures have their place in the treatment of asphyxia, but none alone is sufficiently comprehensive. Resuscitation should become the duty of a specialist who makes a study of the processes involved and who is able to apply all forms of treatment as they are indicated.

Asphyxia may be induced by a wide variety of causes. Regardless of the cause, the events which follow are similar. The problem of resuscitation therefore resolves itself into one of promptly breaking the vicious circle by relieving cellular anoxia. If it cannot be broken before the damage becomes irreversible, the patient will die. Patients, in whom resuscitation has apparently been successful but only after some delay, may die within a short time as a result of these irreversible changes.

Coryllos (24) has classified asphyxia according to degree into slight, moderate and severe. Anesthetists are familiar with the signs and symptoms of the severe and moderate phases but it is the manifestations of slight degrees of asphyxia with which we must be familiar because, if unrecognized and untreated, patients will progress toward the more severe stages of the syndrome. In slight asphyxia the symptoms often resemble those following an overdose of alcohol. There may be headache, depression, apathy, drowsiness or excitement, and a general loss of self control. The subject may become quarrelsome and insolent. He is often reckless in the face of danger and he is quite confident that his mind is clear. Evaluation of time is altered and memory and understanding are impaired. The subject may see without knowing what he sees. Pain is dulled and judgment of position is altered. There is considerable muscular weakness and easy fatigability. Nausea, loss of appetite, and perhaps vomiting may be exhibited. At first, there may be a slight rise in blood pressure with an increase in frequency and apparent force of cardiac contraction. This is due to a stimulation of the vasomotor and cardiac centers of the medulla as a result of anoxia. The increased force of the heart beat later is diminished as the rate further increases. Breathing is increased in rate and may be shallow and periodic in character. The appearance of these symptoms may first manifest themselves several hours after the onset of asphyxia if the asphyxia is mild in degree.

In those conditions in which asphyxia is slow in developing, the body may accustom itself in some degree to the change and in this way compensate for lack of oxygen. This acclimatization is brought about by the kidney, the lung and the blood itself. With the rapid breathing of asphyxia, the carbon dioxide is washed out of the system, and results in an alkalemia (25). The kidney compensates for this by excreting an alkaline urine with a low content of ammonia. The pulmonary volume increases, thinning the alveolar epithelium, and thus permitting a more rapid gaseous exchange. The spleen contracts, the
red marrow proliferates and the value for hemoglobin rises thereby increasing the carrying capacity of the blood for oxygen. The individual under these circumstances may be quite capable of supplying his requirement for oxygen under ordinary conditions. However infection, exertion or any other condition requiring oxygen in excess may overpower this compensatory mechanism and cause rapid, lethal asphyxia.

With the increase in the armamentarium of anesthetic agents now in use, the problem of anesthetic asphyxia increases. Many of the regional anesthetic agents now in use act as convulsants when given in overdose or when accidentally introduced into the circulation. The convulsive action of these drugs produces spasm of the respiratory muscles which results in asphyxia. One should therefore administer oxygen under pressure until an anti-convulsant, such as a barbiturate, can be administered to relieve the spasm. Death from the administration of barbiturates may likewise be the result of asphyxia. Two factors are involved. The drug may depress the respiratory center to such an extent that the demand for oxygen by the tissues is not satisfied or the blood pressure may be diminished to the point where the coronary circulation is not sufficient to provide the cardiac musculature with an adequate supply of oxygen. In either case oxygen should be administered under positive pressure until the effects of the drug may be counteracted by a convulsant drug or until the drug is detoxicated. Many untoward effects accompanying spinal anesthesia are the result of anoxia, due to depression of the respiratory center by action of the drug, or it may be associated with stagnation of circulation. In either case the treatment consists of increasing the content of oxygen in the tissues, especially of the brain by administering oxygen by inhalation and fluids intravenously. Since the advent of cyclopropane as an inhalation anesthetic agent, asphyxia during inhalation anesthesia occurs less frequently than formerly. As the use of helium as a diluent becomes more prevalent one may predict that some cases of asphyxia will result from administration of too high concentrations of this agent. Kaye (26) in reviewing a series of 105 anesthetic fatalities concluded that asphyxia from respiratory obstruction was responsible for ten per cent. of the deaths. The treatment of asphyxia from respiratory obstruction is evident: establish an efficient airway and administer oxygen in adequate concentration and under sufficient pressure. The authors believe that the best method of supplying oxygen is through an endotracheal catheter fitted with an inflatable balloon. Sufficient pressure may be employed to rhythmically inflate the lungs without the danger of distending the stomach.

Patients not infrequently arrive in hospital accident rooms in a state of moderate or severe asphyxia. Ruth has (27) outlined a generalized plan of action to be followed by specific treatment, varying with the etiological factor, and by certain supportive measures. All
mechanical interference to normal respiration must first be removed. A foreign body should be removed if that is possible. If removal is delayed, oxygen or oxygen and helium mixtures should be administered under sufficient pressure to force oxygen past the obstruction, if possible, while preparations are being made for a tracheotomy or bronchosopic removal of the foreign body. Permanent patency of the airway must be assured. An intratracheal tube of large bore may be introduced and fluids aspirated through a smaller catheter inserted through the larger tube. Oxygen may be supplied by mouth-to-mouth or mouth-to-nose insufflation. The accepted manual methods of resuscitation may be employed but mechanical methods are usually more efficient. A mask and a bag (Fig. 3) filled with oxygen may be used to inflate intermittently the lungs. If resuscitation must be maintained for some hours, a mechanical resuscitator (McKesson type), providing alternately positive and negative pressure, may be used. If the intratracheal tube to which this type of apparatus is attached is not equipped with an inflatable balloon, a Levine tube should be introduced into the stomach to prevent its distention. If resuscitation must be continued longer than 12 hours, one must consider the necessity of placing the patient in a resuscitator (Emerson or Drinker), providing intermittent negative pressure. Prolonged intubation may be conducive to production of a tracheitis. With the patient in this type of apparatus, the intratracheal tube can usually be removed. If edema
of the larynx develops following the original trauma, a tracheal stoma should be established before the intratracheal tube is removed. If the patient's original injury involves the deep structures of the neck, tongue, or of the pharynx the danger of the subsequent occurrence of emphysema is very real. Mature consideration must be given regarding the advisability of performing a tracheotomy to provide an airway, should this unfortunate complication seem likely to occur. It seems to be a rather radical preventative measure but it has been a lifesaving one on occasion.

**Asphyxia Neonatorum**

Approximately 80,000 infants die annually at birth in this country and 30,000 more die during the first day of life (28). Many of these are respiratory in origin. Henderson (29) states, "If measures to insure expansion of the lungs were as much a part of the routine treatment of the new-born as is now the disinfection of the eyes, lives would be saved as effectively as blindness is now prevented." Asphyxia in the new-born may be divided into four types, namely: asphyxia livida, asphyxia pallida, asphyxia due to intracranial hemorrhage, and asphyxia due to inspiration of foreign material during delivery. Asphyxia livida results from obstruction of the circulation between the child and the placenta or obstruction of the circulation to the child's head. This obstruction usually occurs late in labor and is of short duration. The baby is born with extreme cyanosis evident. If the infant is not deeply narcotized and if the heart is still beating, resuscitation can usually be effected by inhalational methods. Asphyxia pallida is of far more serious import. A baby in this condition exhibits evidences of shock. Respiration is greatly depressed, cardiac contractions are slow and feeble, muscular tonus is lost, circulation fails, and the capillaries and veins of the skin are deficiently supplied. Similar methods in treatment should be applied.

Asphyxia livida and asphyxia pallida develop previous to delivery while asphyxia due to intracranial hemorrhage develops after birth. Babies suffering from asphyxia following intracranial hemorrhage usually breathe at birth but as intracranial pressure is increased due to hemorrhage, respiration gradually fails. Asphyxia due to aspiration exhibits the classical signs of an obstructed airway.

Considerable diversity of opinion has existed concerning the explanation of the initiation of the first gasping inspiration. Normally respiration begins in utero. Snyder and Rosenfeld have confirmed this both experimentally and clinically. Asphyxia of the new born therefore becomes a suppression of a previously existing respiration rather than a failure of a new mechanism to begin its function. The former procedure of rough handling has recently been superseded by more rational methods. Babies suffering from asphyxia are now treated as are individuals in shock. Extreme gentleness is practiced. The cold water
in the tub has been replaced by warm water, thus conserving the body heat. Efforts are made to restore the normal relations between oxygen and carbon dioxide in the centers in the brain and in this way allow the child to breathe rather than produce respiration by force. The first procedure in the resuscitation of the new-born is the establishment of an unobstructed airway. This can usually be done by lowering the head and gently wiping the mucus from the nostrils and pharynx. If the

![Image of a resuscitator](Fig. 4. A resuscitator providing controlled intermittent positive pressure mounted on a convenient stand, equipped with a drawer which may be used as an oxygen chamber for a new born infant.)

obstructive material cannot be reached by this means, a catheter may be inserted into the pharynx and the material may be aspirated, or the catheter may be directed into the trachea either manually or with the aid of a laryngoscope and the mucus may be aspirated. The baby then may breathe somewhat inefficiently. In order to help, initiate or aid in this movement it is our practice to employ a mechanical resuscitator (Fig. 4) which provides intermittent controlled positive pressure. If inspiration remains inefficient after it has been initiated the baby is placed in an oxygen chamber. For this purpose the stand on which the resuscitator
is mounted is equipped with a drawer (Fig. 4). This drawer may be removed and placed on top of the table. A mattress, sheet and blanket are constantly kept in this container. The baby is placed in it and an oil silk covering is employed to convert the drawer into an oxygen chamber (Fig. 5). The delivery tube from the constant flow meter of the resuscitator terminates in a perforated rubber stopper. This rubber stopper is inserted in a hole in the side of the oxygen chamber. With a flow of five liters per minute, the baby may be maintained in an atmosphere containing 60 to 80 per cent. oxygen. When the infant's condition warrants it, the perforated rubber stopper may be replaced by a solid rubber stopper and the oxygen chamber may be carried to the nursery where the infant is transferred to a Hess bed that can be readily converted into an oxygen chamber (Fig. 6). This oxygen chamber offers the added advantage of having the temperature thermostatically controlled. The infant may be kept in this chamber until it is considered safe to remove him to an ordinary crib. We are confident that this type of treatment has been responsible for a reduction in the mortality rate of infants, particularly those classified as prematures.

Final Summary.—Problems in oxygen therapy and resuscitation are of vital interest to the anesthetist. A thoroughgoing understanding of
the fundamentals involved is an essential requirement for successful treatment. Certain fairly well standardized methods have been developed. If they are employed wisely, one's percentage of successes should be fairly high. Ideal methods have not been achieved, however, and further investigation of these problems is warranted.

BIBLIOGRAPHY

29. Heubner, Youell: Adventures in Respiration, Williams & Wilkins Co., Baltimore (June) 1938.

* 

A Section on Anesthesiology will be held in the Scientific Assembly of the American Medical Association in Cleveland, Ohio in June, 1941.